The Value of Fungicides
In U.S. Crop Production

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Artichoke Research Association
California Asparagus Commission
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1.0 Overview

This report estimates the value and benefits of the use of fungicides in United States crop production. A fungicide is a type of pesticide that controls fungal disease by inhibiting or killing the pathogen causing the disease. The study consists of three parts. First, a brief introduction to plant diseases, plant pathology, and fungicides is provided. Second, a literature review is summarized for the fifty crops included in the study. The literature review summarizes information on the diseases that affect each crop and details the historical record regarding control of the diseases in the U.S. with fungicides and other methods. The third part of the study consists of the quantification in dollars of the value of fungicides used in the growing of the fifty crops on a state by state basis.

Foliar and in-furrow uses of fungicides are included; seed and postharvest treatments are not included. Fumigants that control plant diseases are not included. Plant diseases caused by fungi and bacteria are included; plant diseases caused by viruses and nematodes are not included.

2.0 Introduction

A. Plant Diseases

Most crop diseases are caused by fungi, which lack chlorophyll and therefore are unable to produce their own carbohydrate food.[144] Consequently, they must feed on other living plants or on dead organic matter. Of the 100,000 described species of fungi in the world, approximately 20,000 produce one or more diseases in various plants.[156] Many fungi produce spores in enormous numbers, which are found in the soil, air and on plant surfaces everywhere. Most fungi that cause plant disease are spread locally as a result of the dissemination of spores by wind or rain and are spread long distances in high altitude air currents.[144] When temperatures and moisture conditions in the soil or on the surface of the plant are favorable, a spore germinates producing a tube which enters the plant. The fungus grows through and between plant cells withdrawing nutrients. Some fungi produce toxins that kill plant cells. Usually, fungi produce spores on the surface of leaves, stems or fruit and these spores are then disseminated to healthy plants where the disease process is repeated.

Infected cells and tissues of diseased plants are usually weakened or destroyed by fungi or bacteria. The ability of such cells to perform their normal physiological functions is reduced or completely eliminated; as a result, plant growth is reduced or the plant dies [163].

B. History of Plant Pathology

Although the Old Testament contains many references to blights, blasts, rusts, and smuts, there seems to have been little or no effort to control them; such tribulations were accepted as an expression of God’s wrath.[156] The Romans relied on wheat as their primary crop and provided loaves of free bread to the populace. They noticed that when red spots appeared on the wheat plants, they harvested less grain. The Romans created a god of rust, Robigus, who was honored in an annual religious ceremony for over 1,700
years. A red dog was sacrificed to appease the rust god in an attempt to spare their grain. Climate change in the first century A.D. produced wetter and cooler conditions that led to frequent, severe outbreaks of wheat rust. Crop failures followed leading to famine, and social disruption that contributed to the downfall of the Roman Empire.

During the following two thousand years, little was added to the knowledge of plant diseases although references to the ravages of plant diseases appeared in the writings of contemporary historians. The invention of the microscope in the 1600’s led to the discovery of fungi and bacteria. However, for two hundred years scientists concluded that the organisms were the result, rather than the cause, of disease, which was attributed to spontaneous generation. In 1807, Prevost proved that a crop disease was caused by a fungus. However, his findings were rejected by almost all his contemporaries.

In 1844, a rot of potatoes caused the loss of 25-90% of potato production in the northeast U.S.[33][427] The rot was first reported in Europe in 1845 and it spread throughout the continent, reaching Ireland in August destroying 40% of the country’s potato crop.[425] Irish peasants were almost completely dependent on the potato for their diet and for feed for their farm animals. In 1846, the rot fungus destroyed 100% of the Irish potato crop, which led to the deaths of 1.5 million people and the emigration of a similar number of people, mainly to North America.[423] It would not be until 1861 that Anton de Bary, who is considered the father of modern plant pathology, would conclusively identified a fungus as the cause of the rotted potatoes and not until 1885 that a fungicide would be discovered that could kill the potato rot fungus.[516]

C. History of Fungicide Development

Sulfur is the oldest effective fungicide known. Elemental sulfur can be found near hot springs and volcanic regions in many parts of the world. At room temperature sulfur is a soft, bright yellow solid. Homer mentioned “pest-averting” sulfur in the 9th century BC. It was not until 1802 that sulfur, applied as a finely-ground dust, was used agriculturally to control mildew on fruit trees.[156] A major use of sulfur as a fungicide came about in France in the 1840’s to combat grape powdery mildew, which had been introduced from North America in 1845 and reduced French wine production by 80% by 1854.[601] The use of sulfur became generally widespread in vineyards, and by 1858 French wine grape production returned to its previous level.[602] The fungicidal action of sulfur is complex and functions primarily in the respiration process. When sulfur is applied to a plant, vapors are released. The powdery mildew fungus produces hydrogen, which reacts with the sulfur to form hydrogen sulfide, a gas toxic to the fungus. Attempts were made to control potato rots with sulfur, but it was not effective.

In 1878, another pathogen of grapes, downy mildew, was introduced into French wine grapes from America and, once again, French wine grape production was greatly reduced. In 1885, an effective fungicide which controlled downy mildew was accidentally found. The story goes that a French botanist, Alexis Millardet, was walking down a lane observing the grapes infected with downy mildew when he noticed that some grapes were covered with a bluish-white wash. He also noted that the leaves on these plants were healthy whereas the neighboring plants were badly diseased. When he questioned the farmer to whom the grapes belonged, he was told that the grapes along the
road had been sprayed with a mixture of lime and copper sulfate to discourage pilferers. This was the accidental discovery of the fungicide known as Bordeaux mixture, named for the area in France where it was discovered. The copper ions of the mixture were toxic to the fungus and the lime reduced the damage of the copper sulfate to plants. Bordeaux mixture proved effective in controlling grape downy mildew, potato late blight and numerous other diseases. By the early 1900’s, spraying Bordeaux mixture on potatoes was common practice in North America and Europe.

There was one more major outbreak of the potato blight which caused the death of many people as a result of famine. The famine took place in 1916 in Germany during World War I. All the copper that Germany had was being used for shell casings and electric wire; none was available for making Bordeaux mixture. A major late blight epidemic went untreated in 1916 and potatoes rotted in the fields. The resulting scarcity of potatoes led to the deaths of 700,000 German civilians from starvation during the winter of 1916-1917 and was a major factor in the demoralization of the German army.

In the early decades of the twentieth century, chemists began synthesizing thousands of new compounds that had never been found previously on earth. These synthetic chemicals found many uses throughout society. Many of the chemicals were tested for fungicidal properties and a few proved highly effective.

The first synthetic chemical fungicides (nabam, thiram, and zineb) were developed and patented in the 1930’s. Their fungicidal properties were confirmed in 1941 by researchers at the Connecticut Agricultural Research Station. Agricultural experiment stations were assigned to aid the war effort of the 1940’s by improving disease control methods and reducing preventable crop disease losses. One activity of the federal research stations was to cooperate closely with industry chemists who were synthesizing new chemicals. The experiment station personnel evaluated the chemicals in tests to determine their effectiveness and phytotoxicity in comparison to the older non-synthetic fungicides. Scientists at the Connecticut station in the early 1940’s tested 6,000 chemical compounds for possible usefulness in plant disease control.

Several synthetic chemical fungicides that were developed in the 1950 to 1970 time period proved to be effective in controlling a broad spectrum of plant disease-causing organisms (mancozeb, maneb, captan, chlorothalonil). These chemicals are similar to sulfur and copper in that they are not absorbed or translocated by the plant. They must be present as a film on the surface of a plant at or before the appearance of a pathogen in order to prevent infection. Beginning in the 1970’s, systemic fungicides were introduced. These chemicals are absorbed by a plant and translocated within. Being internally therapeutic, they can cure plant diseases several days after infection has occurred.

3.0 Crop Literature Review: Summary

The fifty crops selected for this study are listed in Table 1 and include field, vegetable, fruit, nut, berry and specialty crops. These crops were selected for the study after having been identified as major users of fungicides. Several major field crops are not
included in the study (corn, sugarcane, sorghum, canola, sunflowers) because fungicides are not generally used on these crops in any state.

For each of the fifty crops, a literature review was conducted to collect information on the diseases that are targets of fungicide use, the history of their control in the U.S., the results of control experiments, and the practices of organic growers. This literature review is summarized for each crop in Sections 6.1-6.50. A list of all the sources cited in the literature review is included in Section 7.0.

A. Target Diseases

The literature review identified 231 diseases for which growers of the fifty crops apply fungicides in the field. These diseases are listed by crop in Table 2. Most of these diseases (212) are caused by fungi while a smaller number (19) are caused by bacteria.

The pathogens attack the roots, leaves, branches, and fruit and grain of crop plants. Yields are lower due to reduced photosynthesis as well as direct damage to fruit and grain. In some cases yield is reduced due to sun scald of fruit due to loss of protective screening foliage.

Fungal spores and bacterial cells are produced in enormous quantities:

- One apple tree infected with black rot is estimated to contain eight billion spores.[709]
- Two million powdery mildew spores exist on a square inch of an infected cantaloupe.[144]
- One cucumber infected with phytophthora can produce 840 million spores.[637]
- As many as 380,000 bacterial canker cells have been counted on an unsprayed cherry tree leaf.[350]
- Up to ten million eastern filbert blight spores have been counted per square meter of trap surface per day.[216]
- A single blast infected rice plant produces 60,000 spores per night.[753]
- Every fire blight bacterial cell multiplies by dividing reaching 10 billion in 72 hours.[703]
- 500 million late blight spores can be produced on a single infected celery plant.[87]

Many of the diseases are well established in certain locations, overwintering in crop debris, in orchards or on alternate hosts prior to initiating spore release the following spring. A typical life cycle of a fungus overwintering in crop debris and infecting the next year’s crop is shown in Figure 1. Other pathogens overwinter far from infection sites and are carried by the wind to crop fields. The downy mildew fungus overwinters in Florida and spores travel up the east coast in the spring and summer reaching Massachusetts in August.[517] Figure 2 is a map showing the northern movement of the powdery mildew fungus infecting sugar beets.
Many of these diseases are estimated to infest all of the acres of susceptible crops annually. For example, apple scab is estimated to be present in 100% of eastern state apple acreage (Table 3). The early rot fungus is estimated to infect 100% of eastern cranberry beds each year (Table 4). The late and early leaf spot fungi are estimated to infest 99% and 80% of southeastern peanut acreage respectively (Table 5).

Although many of the diseases are estimated to infest a smaller percentage of acreage, they would have a very large impact on crop yields if not controlled. For example, black rot is estimated to infest 25% of eastern apple acres with a potential to reduce apple yields by 60% (Table 3). Twig rot is estimated to infest 25% of eastern cranberry beds with the potential of reducing yields by 50% (Table 4).

Many of the diseases vary in their regional infestation levels. For example, although gray mold of strawberries is a target of fungicide use throughout the U.S., anthracnose and powdery mildew are not problems in the Great Lake states (Table 6).

Some additional widespread pathogens with the potential to significantly reduce crop yields include:[13][15]

- 80% of California almond acres are infested with the shot hole fungus; potential yield loss is 50-75%.
- 100% of California’s grape acres are infested with powdery mildew; potential yield loss is 80%.
- Black rot infests 95% of the grape acres in eastern states; potential yield loss is 85%.
- 100% of U.S. peach acres are infested with brown rot; potential yield loss is 75%.
- 100% of cherry acres in western states are infected with powdery mildew; potential yield loss is 25%.
- Leaf spot of cherries is present in 80% of eastern cherry acres; potential yield loss is 100%.
- Scab infests 80% of eastern pear acres; potential yield loss is 85%.
- 100% of western raspberry acres are infested with gray mold; potential yield loss is 35%.
- 100% of U.S. strawberry acres are infested with gray mold; potential yield loss is 45-50%.

Most of the disease pathogens have been present in U.S. crop fields for a century or more. However, some of the pathogens are new arrivals in crop production fields and are the result of changes in weather patterns and long-range transport of spores:

- Anthracnose was rarely seen in California almond orchards until the 1990’s; now it is found in all orchards with the potential to reduce yields by 25%.[472]
- In the 1980’s powdery mildew infected California artichoke fields; yield losses were 30% in 1986; fungicides have been used ever since (Figure 3).
- Stripe rust of barley first appeared in western states in the 1990’s; yields were reduced 25-50%.[733]
• Prior to 1989, northern bacterial blight was not present in California celery fields; now it has spread to all celery growing regions in the state.[86]
• Bacterial fruit blotch of watermelons first appeared in 1989 and has spread throughout the southeast.[513]
• Garlic rust first appeared in California in 1998 and reduced statewide production by 50%; fungicides have been used ever since. (Figure 4)
• Panicle and shoot blight was first detected in California pistachio orchards in 1984; in 1998 the disease caused a 20 million pound reduction in pistachio production.[225]

B. History of Fungicide Use in the United States

One effect of uncontrolled plant disease epidemics in the U.S. was the elimination of the growing of certain crops in infected regions. For example, the inability to control fire blight led to the abandonment of pear production in most eastern states [13]; the inability to control bacterial spot led to the abandonment of plum production in the South [340]; devastation from black rot led to the abandonment of large acreages of grapes in eastern states in the early 1900s [613]; scab became so severe in the first half of the 1900’s that it eliminated barley production from the eastern and central corn belt.[733]

Prior to the development and adoption of effective fungicides, uncontrolled plant disease epidemics significantly reduced crop yields in years favorable for infection:

• In 1914, cedar apple rust destroyed 25% of Virginia’s apples.[728]
• In the 1870’s, bitter rot resulted in 100% apple yield loss in Illinois and Arkansas.[704]
• In 1912, in the Southeast, black rot caused 25-50% losses in apple production.[708]
• In the 1890’s, most asparagus fields in the Atlantic states were entirely destroyed by rust.[139]
• In the 1920’s, 25-50% of Michigan celery production was lost to late blight.[627]
• In the 1920’s, 60% of Florida citrus had melanose blemishes.[289]
• In the early 1900’s, diseases typically lowered New Jersey cranberry production by 33%.[104]
• In 1921, alternaria leaf blight destroyed almost the entire melon crop in Arizona.[144]
• In 1906-1912, anthracnose destroyed 25-60% of the cucumbers in Ohio, Michigan, and Indiana.[502]
• In the 1920’s, downy mildew reduced Florida cucumber production by 10-70%.[515]
• In 1926, in the Imperial Valley of California, cantaloupe production was reduced by 25-35% due to powdery mildew.[520]
• In the early 1900’s, black rot routinely reduced eastern grape production by 25% while in some years the losses were 70-100%.[612]
• In the 1930’s, rust caused 40-80% losses in green beans in Florida.[266]
• In the 1940’s, green bean losses due to gray mold were as high as 50% in the Northwest.[265]
In 1949-1950, mint losses in Oregon due to rust were 25-35%.[110]
In the 1850’s, Georgia peach production was typically reduced by 50-75% by brown rot.[356]
In 1945, defoliation caused by leaf spot resulted in the death of 25,000 cherry trees in Pennsylvania.[485]
In 1844, late blight rotted 25-90% of the potatoes in northeastern states.[427][33]
In the early 1900’s, early blight reduced Michigan potato yields by 25%.[417]
In the 1930’s, in Maine, 20% potato losses were caused by pink rot.[458]
White rust destroyed 25% of Texas spinach crop in 1937.[196]
Septoria leaf spot caused almost a complete failure of Michigan’s tomato crop in 1915.[144]
Prior to 1947, less than 10% of mid-Atlantic tomato fields were sprayed with fungicides; after a late blight epidemic destroyed 50% of the tomato crop in 1947, growers started spraying 90% of the tomato acres.[414]
In the early 1900’s, as much as 75% of California’s walnut crop was destroyed by walnut blight.[58]
In 1916, stem rust destroyed 38% of U.S. wheat production.[646]
In the 1920’s, septoria leaf spot caused an annual reduction in tomato production of 250,000 tons per year.[144]

Prior to the early 1900’s, there was little spraying of fungicides in the U.S. However, as cities expanded and markets for fruit and vegetables grew with increased storage and long range shipment, losses due to rotting produce became less tolerable as did blemishes due to pathogens.

Research demonstrated that Bordeaux mixture, copper, sulfur and lime sulfur provided effective control of many crop diseases:

- Bordeaux mixture controlled shot hole in almonds increasing yields 200%.[332]
- Bacterial leaf blight of celery was first controlled with Bordeaux mixture in 1917, reducing the number of spots per plant from 961 to 6.[87]
- 4 to 5 Bordeaux applications reduced cranberry fruit rots by 50%.[104]
- Bordeaux mixture was applied to watermelons beginning in 1919, for anthracnose control.[502]
- Sulfur applications to control powdery mildew of green beans began in the 1930’s, following research that showed a reduction in the incidence of the disease from 87% to 2%.[254]
- In the early 1900’s, powdery mildew was considered capable of destroying the entire grape crop in California if sulfur sprays were not made.[601]
- Yearly sprays with Bordeaux mixture became standard practice in California peach orchards in the early 1900’s for shot hole control.[332]
- With the development of finely powdered sulfur about 1912, southeastern peach growers began widespread spraying to control brown rot. Average losses were reduced to 13%.[352]
• A summary of twenty years of experimental data in Vermont (1890-1910) showed an average yield increase of 64% in potatoes as a result of controlling late blight with Bordeaux mixture.[428]
• In the 1920’s, experiments with copper for cercospora control increased sugarbeet yields by 20%.[676]
• In the 1930’s, experiments in Florida with Bordeaux mixture reduced the percent of tomatoes with phoma rot from 35% to 6%.[393]
• In the 1940s, experiments with walnut blight demonstrated the effectiveness of copper which has been used ever since.[58]

In a six-year study (1922-1928), it was determined that one-third of all rail shipments of peaches had 5% or more of the fruit affected by brown rot.[372] The development of brown rot in transit was directly linked to spray programs in the orchard: sound fruit from unsprayed trees developed approximately four times more brown rot in storage than sound fruit in storage from sprayed trees.

By the 1920’s, spraying fungicides (lime sulfur) became a universal practice in U.S. apple orchards and it was impossible to grow apples for market without fungicide sprays.[498] In Pennsylvania in 1938, scab incidence was 82% in 50 unsprayed apple orchards while in 107 properly sprayed orchards, scab incidence was 1.4%.[302]

There were certain problems with the early fungicides including crop damage and limited or no control of many diseases:

• Although Bordeaux mixture controlled downy mildew on cucumbers, it also damaged the crop and was not widely-used. Yield losses of 40-50% in Delaware and New York occurred in 1924.[515]
• Sulfur provided only about 50% control of cedar apple rust.[730]
• Spraying celery with Bordeaux mixture to control early blight was the accepted practice beginning in 1910. However, Bordeaux left a surplus of lime on the plants which injured the celery plants.[92]
• Three years of experiments with Bordeaux mixture did not increase onion yields; losses to leaf blight remained high.[24] Bordeaux mixture controlled pecan scab but was phytotoxic to young unfolding leaves.[35]

Research with synthetic chemical fungicides began in the 1940’s and demonstrated that crop yields were higher as a result of improved disease control efficacy and/or reduced damage to the crop.

For fruit disease control, extensive tests were made with ferbam. Researchers determined that apple trees sprayed with ferbam yielded 41 % more than trees sprayed with the standard lime sulfur treatments.[723] Much of the yield increase was attributed to reduced damage to the tree from ferbam in comparison to the lime sulfur spray. Fungicides were extensively tested for control of potato late blight. Experiments with zineb and nabam resulted in potato yields that were 23-35% higher than potato yields from plots sprayed with the standard Bordeaux mixture.[435][436]
Research demonstrated that the synthetic fungicides were effective in controlling numerous diseases:

- In 1946, ferbam increased cucumber yields by 80-100% by controlling downy mildew.[636]
- Captan and ziram increased almond yields in the 1950’s by 75-80% through control of leaf blight.[465]
- In the 1950’s, zineb provided 85% reduction in asparagus rust incidence.[147]
- EBDC fungicides reduced defoliation of carrot plants due to alternaria from 20% with copper to 3%.[234]
- Experiments substituting ferbam for Bordeaux mixture for black rot control increased grape yields by 1.6 tons per acre.[615]
- Average yield of onions were 26% higher with nabam or zineb treatments for leaf blight.[26]
- Research in the 1960’s, demonstrated that dodine or TPTH increased pecan yields by 100% in comparison to Bordeaux mixture.[36]
- Research with zineb and nabam in the 1940’s resulted in potato yield increase of 23-35% in comparison to Bordeaux mixture for late blight control.[435][436]
- Experiments with ferbam in the 1940’s, resulted in three additional tons of tomatoes per acre due to anthracnose control.[374]

Although precise use estimates are not available, it is believed that growers of apples and potatoes (and other fruit and vegetable crops) rapidly switched from older fungicides (Bordeaux mixture, lime sulfur) to the new synthetic fungicides by the early 1950’s. For example it was reported that the synthetic fungicides (zineb and nabam) were used on 75% of U.S. potato acreage in 1953.[66] There was almost universal adoption of zineb and nabam sprays by watermelon growers for anthracnose control.[495]

Yields of potatoes and apples increased dramatically in the early 1950’s (Figures 5 & 6). Much of the increase is attributable to the increased disease control effectiveness and reduced phytotoxicity of the synthetic chemical fungicides. For some diseases, the synthetic chemicals offered the first effective controls. For example, for apples, there were no effective spray materials for black rot prior to the introduction of ferbam during which time 25-50% fruit losses were common in the Southeast.[708] Ferbam reduced the incidence of black rot to 1%.[709] Other crops for which yields went up dramatically as a result of improved disease control with synthetic chemicals were peanuts and cucumbers (Figures 7 & 8).

For certain crops, effective chemical fungicides for control of key diseases were not introduced until later. For pecans, the first effective synthetic chemical fungicides for control of pecan scab were introduced in the early 1960’s and were a primary factor in significantly increasing U.S. pecan yields (Figure 9). The production of wild rice in Minnesota increased dramatically after the introduction of fungicides in the 1970’s (Figure 10).

In 1950, the American Phytopathological Society (APS) summarized the status of chemicals used to control crop diseases and concluded that:
• “Many of our fruit and vegetable crops cannot be produced economically, efficiently and in reliable volume without chemical protection from fungous, bacterial and nematode parasites. Uninhibited plant diseases would eliminate all possibility of planned production in apples, potatoes, tomatoes, peaches and many other crops.”[302]

In a 1979 report to USEPA, APS once again summarized the status of the control of plant diseases with chemicals and concluded that:

• “In the USA, many crops such as fruits, vegetables, peanuts ……could not be produced commercially without the use of chemical agents including fungicides, nematicides, and bactericides for disease control.”[370]

One result of the adoption of synthetic chemical fungicides was a significant decrease in the total poundage of fungicides used in U.S. crop production. In the 1940’s, the War Department determined the annual usage of fungicides (copper, sulfur) by U.S. growers to be 300 million pounds per year.[603] The synthetic fungicides were used at significantly lower rates per acre than copper and sulfur (3-6 pounds/acre in comparison to 10-60 pounds/acre). As a result, as growers switched to the synthetic chemicals, the aggregate poundage of fungicides used in U.S. crop production declined significantly (Table 7). As newer fungicides with even lower rates were introduced (0.1 pound/acre), the aggregate volume of fungicide use declined further so that by the late 1990’s, the aggregate use of fungicides was 131 million pounds/year; less than one-half of the annual use amount in the 1940’s (Table 7). The decline in pounds was particularly significant for apples, which saw a decline from 92 million pounds per year in the 1940’s to 5 million pounds per year in the mid 1990’s (Table 7 & Figure 11).

In recent years fungicide use has increased for certain crops following the emergence of new pathogens such as powdery mildew in artichokes and rust in garlic. A significant increase in the volume of fungicides used in potato production occurred in the 1990’s (Figure 12) due to the emergence of new harder to kill late blight spores.

As more effective fungicides have been introduced and used by growers, losses to fungal pathogens continued to decline. For example, in Georgia, as growers increased their expenditures on new fungicides for control of mummyberry in blueberries, seedling diseases of cotton, and white mold in peanuts, the reduction in crop losses far exceeded the increased costs of the fungicides (Figures 13 through 15).

C. Alternative Control Methods

One “strategy “ for disease management in the years before fungicides was simply to abandon acres in infested states and move production to areas of the country where a pathogen was not present. For certain crops, this strategy worked well for many years; however, due to long range transport of fungi, the uninfected areas have become infected, resulting in the use of fungicides to preserve continued production in the previously uninfected areas:
• In the 1920’s, eastern filbert blight destroyed all the filbert trees in eastern states. Filbert production thrived in the Northwest, which was free of the fungus. It was detected for the first time in Oregon in 1971. Without fungicide use, it is estimated that 75% of Oregon’s filbert (hazelnut) trees would be killed within 25 years.[217]

• In the 1800’s, the center of hop production was New York, where uncontrolled mildews led farmers to discontinue production. The center of hop production moved to the Northwest where the powdery mildew fungus did not occur. The powdery mildew fungus was detected for the first time in the Northwest in 1997 and currently 100% of hop acreage in the region are sprayed with fungicides.

The removal of nearby alternative hosts of pathogens has been utilized as a means of reducing pathogen pressure on susceptible crops. For example, in the early 1900’s eight states passed laws empowering the removal of cedar trees as a way of preventing infections of apple orchards by cedar apple rust spores. However, this measure proved impractical due to the large number of cedar trees and their aesthetic value.[725] In 1918, the U.S. government began a program to eradicate the barberry plant from the northern Great Plains since the wheat rust fungus could only survive by overwintering on a barberry plant. Over a half billion barberry plants were eliminated. Barberry eradication did not eliminate the threat of rust infections from the northern Great Plains since rust spores blow northward from Mexico and Texas. The effect of barberry eradication was to delay potential rust epidemics by several weeks.

One of the disease control methods that has been continuously explored as a means of managing plant diseases in the U.S. is the breeding of resistant cultivars. For several crops, the breeding of resistant crops has proven to be an effective strategy: corn, sorghum, sugarcane. However, plant pathogen populations adapt to resistant cultivars and races of pathogens that overcome the resistance become dominant. As a result, in order to sustain host plant resistance, crop breeding needs to be continuous, with new resistant varieties in development at all times.

For some crops, despite decades of breeding thousands of new varieties, disease suppression with host plant resistance has proven completely unstable because the pathogen mutates so rapidly. For example, no potato variety has been produced combining resistance genes to all late blight races with the many genetic traits needed to produce a commercial variety. Although the search for peanut cultivars resistant to white mold originated in 1917, a high degree of resistance has not been found.[543]

Some examples of the breakdown in host plant resistance that have resulted in a need to spray fungicides to control pathogens include:

• In 1906, a major asparagus breeding program was established and two cultivars with rust resistance were released and widely-planted. By 1940, 100% rust infections were occurring on these varieties.[141]

• In 1936, a powdery mildew resistant cantaloupe was released and widely planted. In 1938, a new race of the fungus appeared to which the cultivar was highly susceptible.[521]
• Prior to 1960, downy mildew was a significant disease on lettuce in California. It was brought under control with resistance from wild lettuce. New races of the fungus appeared in 1976 and overcame the resistance. In 1989, another set of resistant cultivars were introduced, but by 1992 control slipped again.[568]

• The rice variety newbonnet was released in 1983, and was resistant to the then-predominant blast disease races. By 1986, it was planted on 70% of Arkansas rice acres. During that year widespread epidemics of blast caused by two previously-minor races wiped out entire fields of newbonnet.[757]

• Commercial spinach varieties with immunity to the only two races of downy mildew were introduced in the mid 1950’s. For twenty years, downy mildew was unknown on spinach in the U.S. Race 3 appeared in the late 1970’s causing heavy losses. A gene for resistance to Race 3 was used in spinach cultivars in 1982 and provided resistance until 1989 when Race 4 appeared.[189] Since then Races 5 through 10 have been identified.

• Wheat cultivars with leaf rust resistance genes have been available since the 1940’s. Within a few years of release, virulent leaf rust strains appear that render the varietal resistance ineffective. Most recently, resistance broke down in previously resistant cultivars in the northwest. The loss in wheat yield would have been 20% without the use of fungicides.[694]

In many cases, resistant cultivars have been released but are not widely planted because of poor horticultural characteristics. For example, more than twenty apple cultivars bred with resistance to apple scab have been released, but none are widely planted since they produce fruit of small size, have a tendency to ripen unevenly, and have brownish interiors. Development of rice cultivars resistant to sheath blight has been slow because resistance is linked to undesirable traits such as tall plant stature, late maturity, and poor milling quality.[747]

Usually, there is a tradeoff between increased host plant resistance and other desirable traits that are lost in the breeding process. The tradeoff in sugarbeets is lower yield for increased resistance to cercospora (Figure 16).

Often “resistant” cultivars are not totally resistant to the pathogen. For example, resistant peanut varieties provide 20-40% control of leaf spot, rust and stem rot.[536] By contrast, fungicides provide more than 90% control. The pepper cultivar Paladin possesses excellent horticultural characteristics and exhibits excellent resistance to the crown rot phase of phytophthora; however it does not possess resistance to the foliar phase of phytophthora blight, which requires regular fungicide applications for control.[321]

D. Experimental Results

Fungicides are regularly tested for efficacy and impacts on crop yield. Some recent test results include:

• Garlic yields doubled in tests where rust was controlled.[221]
• Watermelon yields increased 61% in tests where gummy stem blight was controlled.[499]
• Fungicides reduced the incidence of citrus scab from 44% to 0.4%. [306]
• Fungicides reduced the incidence of brown rot of almonds from 44% to 4%. [463]
• Fungicides reduced purple spot losses in asparagus by 99%. [50]
• Fungicides reduced the incidence of mummyberry in blueberries by 98-99%. [276]
• Fungicide treatments produced 85% marketable cantaloupes in comparison to 20% marketable yields from plots untreated for gummosis. [493]
• Fungicide applications reduced grape powdery mildew incidence from 99% to 0.4% [609]
• Black rot of grapes was reduced from 95% to 1%. [616]
• Fungicide applications for downy mildew resulted in 98% marketable lettuce heads instead of 25% untreated. [572]
• For over forty years, the annual incidence of scab in apple trees untreated for scab has been 98-100% in experiments at Michigan State University. [717]
• Fungicides reduced peach brown rot losses from 75% to 1%. [479]
• Fungicides reduced the incidence of powdery mildew colonies on cherry trees by 96%. [346]
• Fungicides reduced the defoliation of cherry trees due to leaf spot from 80% to 0.3%. [488]
• Fungicides reduced the incidence of rhizoctonia limb rot of peanuts from 30% to 3%. [541]
• Fungicide control of white mold in peanuts increased yields by 58%. [546]
• Control of blast with fungicides increased rice yields by 45%. [751]
• Fungicides reduced the incidence of panicle and shoot blight of pistachio trees from 75-100% to 1%. [228]
• Fungicides reduced the incidence of gray mold on raspberries from 88% to 5-6%. [206]
• Fungicide treatments for phytophthora control increased yields of sweet peppers by 25,000 pounds per acre. [320]
• Fungicides reduced tomato plant defoliation due to target spot from 90% to 2%. [366]
• In field trials for rust control in wheat, fungicide applications increased yield by up to 50%. [654]

E. Organic Practices

Organic apple growers find it necessary to spray fungicides to control diseases. Organic apple growers are permitted the use of approved disease control chemicals including sulfur, lime sulfur, copper and antibiotics (streptomycin). Use of these organically-approved sprays can control the major diseases of apples but many and frequent sprays are required. [99] A recent production budget guide for organic apple growers in the northeastern United States estimated that ten gallons of lime sulfur and twelve pounds of wettable sulfur are used per organic apple acre. [100]
Other examples of the use of fungicides by organic growers include:

- Sulfur is permitted for use by organic grape growers in California who make seven applications annually totaling 66 pounds of sulfur per acre.[610]
- Organic peanut growers report that sulfur and copper are used for disease control.[598][599]
- The University of California estimates that organic strawberry growers make nine applications of sulfur, at five pounds per application per acre, to control powdery mildew.[98]
- In order to control late blight, it has been reported that organic potato growers make 9 to 15 copper applications per season.[249]
- California organic walnuts are treated with eight pounds of copper hydroxide per acre for walnut blight control.[72]

4.0 Quantification of Fungicide Benefits

A. Production Data

Table 8 displays 2002 national summary production and acreage estimates for the 50 crops included in the study. The production and acreage statistics are drawn from USDA reports.[1 through 5][44] The 50 crops total 149 million acres, with annual production of 545 billion pounds of food and fiber and a combined value of $53 billion. State-by-state production and acreage statistics for the fifty crops are presented in the Data Appendix Tables 1.1-50.1.

B. Fungicide Use and Cost

Estimates of the use of individual fungicide active ingredients on each crop in each producing state were compiled from surveys conducted by USDA [6][7][8], crop profiles assembled by USDA (available at: http://pestdata.ncsu.edu/cropprofiles/), pesticide use records compiled by the State of California [56], and from a survey of commodity organizations and extension service specialists. In all, 2,419 estimates of fungicide use are made for the fifty crops by state and active ingredient (see Tables 1.2-50.2). Forty-nine states are included (excluding Alaska). The use estimates include estimates of the percent of acres treated and the average annual rate of application with the fungicide active ingredient on a crop in a state. Forty-five active ingredients were identified as being used on one or more of the crops. These active ingredients, their trade names and manufacturers are identified in Table 9.

Table 10 contains estimates of fungicide active ingredient (pounds) used annually on each crop nationally. These national estimates are sums of the state estimates by active ingredient shown in the Data Appendix (Tables 1.2-50.2). The fifty crops total 108 million pounds of fungicide use.

An estimate of the percent of the national acreage of each crop that is treated with fungicides is included in Table 10. Nationally, it is estimated that 17.7 million acres of the fifty crops are treated with fungicides. For thirty of the fifty crops, the national
acreage treated with fungicides exceeds 70%. For three crops, (barley, wheat, soybeans) less than 10% of the national acreage is treated with fungicides since a large portion of their acreage is located in states where fungicides are not generally used. The state-by-state estimates of acres treated with fungicides for each crop are shown in the Data Appendix Tables 1.3-50.3.

Table 11 summarizes the fungicide use and cost estimates by state. The state totals are sums of the state data for each crop as shown in Tables 1.3-50.3 in the Data Appendix. Figure 17 is a national map showing the poundage of fungicides used by state.

Tables 10 & 11 also contain estimates of the cost of fungicides for each of the fifty crops and forty-nine states. The cost estimates consist of two components: the cost of the product and the cost of application.

Product costs are determined by multiplying estimates of the usage pounds of a fungicide active ingredient by an average per pound price for the ingredient. The estimates of average per pound price for each of the 45 active ingredients were calculated from survey data compiled by USDA and from estimates in extension service publications. [655 through 664] These average prices are shown in Table 12 along with estimates of the total national volume of use and the total sales value of each active ingredient.

Nationally, it is estimated that growers of the fifty crops spent $575 million on fungicide products in 2002. The state-by-state product cost estimates for each crop and active ingredient are shown in Tables 1.2-50.2 of the Data Appendix.

Application costs are calculated by assigning an average number of fungicide application trips for each crop by state and by assigning a cost of $5/acre for each application. The estimates of the number of application trips assigned by crop by state are shown in Tables 1.3-50.3 in the Data Appendix. Nationally, it is estimated that growers spent $305 million to apply fungicides in 2002.

The total cost of fungicides and their application for 2002 is estimated at $880 million. Table 10 displays these costs nationally by crop. Table 11 displays these costs by state.

C. Fungicide Value Estimation

Estimates of the value of fungicides were made in terms of the economic value to growers of the increase in production due to the application of fungicides. These estimates are based on a simulation of the nonuse of fungicides by crop and by state. Estimates of the likely impact on yields of each crop by state of not using fungicide are presented in Tables 1.4-50.4 in the Data Appendix. In all, 417 estimates of the impact of fungicides on crop yields are made by crop and state. These estimates are drawn from a series of studies conducted by USDA and the American Farm Bureau.[10 through 15][30][137][170][205][243][400]. All of these studies relied on university plant pathology specialists to specify the likely changes in yield if growers did not use fungicides. These expert opinions are based on research trials conducted by the specialists and on observations of yield losses that occurred in farmer fields when diseases were left uncontrolled. These predicted yield changes are consistent with the historical record and experimental research data collected in this study’s literature review.
Table 13 summarizes the production benefits at the national level for each crop in terms of the percent change in yield on the acres currently treated with fungicides. For 22 crops the increase in yield on treated acres is estimated to be greater than 50%. The total increase in production due to fungicides amounts to 97 billion pounds of food and fiber with a production value of $12.8 billion.

Table 14 summarizes the net economic value of fungicides for the 50 crops at the national level. The total value of the increased production due to higher yield is estimated at $12.8 billion. By subtracting the estimates of current expenditures on fungicides and their application ($880 million) a net economic value of $12 billion for fungicides is calculated. Table 14 includes a return ratio, which is the ratio of the increased production estimate to the estimate of current expenditure on fungicides. For the nation, the Return Ratio is 14.6, which means that for every dollar currently spent on fungicides, growers gain $14.60.

Table 15 summarizes the total and net economic value of fungicide use by state. Figure 18 is a national map that shows the total crop production benefits of fungicide use by state. Table 16 includes a selected list of crop yield impact estimates for each state (the complete set of crop yield impact estimates by state and crop are displayed in Tables 1.4-50.4 of the Data Appendix.)

**D. Results**

The entire national acreage of three crops (grapes, hops and papaya) are treated with fungicides annually. More than 90% of the national acres of nine additional crops are also treated annually with fungicides (raspberries, strawberries, carrots, potatoes, apples, celery, cherries, peanuts and peaches). (See table 17) Grape fungicide use is greatest in terms of pounds (44 million pounds). The next closest crop is tomatoes with 10 million pounds of national use. More than a million pounds of fungicides are applied nationally to ten additional crops: apples, potatoes, citrus, peaches, sugarbeets, peanuts, cherries, almonds, walnuts and pears (Table 17). Grape growers spend the most for fungicides and their application ($123 million/year) followed by potatoes ($100 million) and peanuts ($99 million). Growers of nine additional crops spend more than $25 million on fungicides and their application: apples, walnuts, rice, sugarbeets, tomatoes, citrus, cotton, almonds and pecans). See Table 17

California growers use the greatest aggregate amount of fungicides (61 million pounds) and spend the most on fungicides and their application ($217 million/year) (Table 18). Growers in 11 additional states use more than one million pounds of fungicides per year: Florida, Washington, Georgia, Michigan, Idaho, New York, North Dakota, Oregon, Wisconsin, South Carolina, and Virginia. Georgia ranks second to California in annual fungicide expenditures ($77 million) followed by Florida, North Dakota, Washington, Minnesota, Michigan, Arkansas, Texas, New York, Alabama, and Wisconsin (Table 18).

Grape growers gain the most as a result of fungicide application: $2.67 billion per year, which represents a 95% yield increase (Table 13). Apple growers rank second with a gain of $1.22 billion, which represents a yield increase of 86% on the 93% of acres
treated with fungicides. The value of eight other crops is increased by more than $300 million annually as a result of fungicide treatment (potatoes, citrus, lettuce, tomatoes, strawberries, almonds, peanuts, and sweet peppers) (Table 19).

Growers in California benefit the most from fungicide use ($5.5 billion/year), followed by Florida ($1.9 billion), and Washington ($1.45 billion). Seven additional states gain more than $200 million in increased crop production value as a result of fungicide use: Georgia, Arizona, Idaho, Michigan, Oregon, Texas, and New York. (See Table 19).

E. Comparison to Previous Studies

This study’s findings with regard to the value and benefits of fungicide use in U.S. crop production are consistent with numerous previously issued studies.

In a 1989 study commissioned by the National Agricultural Chemicals Association, GRC Economics simulated the impacts of the nonuse of fungicides (except for copper and sulfur) and concluded that fruit and vegetable production would be reduced by 32% and 21% respectively.[589] Production losses without fungicides were estimated to be highest for almonds (-75%), peanuts (-68%), peaches (-49%), apples (-40%), and strawberries (-38%).

In 1993, the American Farm Bureau Federation estimated the national impact of not using fungicides to be a reduction of 61-62% in fresh fruit and vegetable production.[30]

A report from Michigan State University concluded that without fungicide use in eastern states, most apple acres would be abandoned within a few years.[732]

In the 1990’s, USDA conducted a series of commodity studies that estimated the impact on U.S. production if fungicides were not used: carrots (-24%), strawberries (-58%), and tomatoes (-60% fresh; -10% processing).[243][137][400] These USDA estimates are close to the estimates in Table 13: -26% for carrots, -59% for strawberries and -19% for tomatoes.

5.0 Summary and Conclusions

Plant parasitic fungi are an implacable enemy of U.S. crop production. In the spring and summer countless numbers of spores are released by fungal organisms. If the spores land on susceptible plant tissue that is not protected by a fungicide, a germ tube grows, penetrating the plant and resulting in an infection by the fungal organism and the withdraw of plant nutrients leading to plant death and rot.

Uncontrolled plant disease epidemics have altered world history causing the deaths of millions of people (Irish potato famine), contributing to the downfall of civilizations (Roman Empire) and altering the outcome of wars (World War I).

In the U.S., before the introduction of fungicides, there were no defenses for plant diseases, which typically rotted much of the nation’s fruit and vegetable crops. Widespread use of fungicides began in the U.S. in the early 1900’s with the spraying of
sulfur and copper. Most fruit and vegetable crop acres have been sprayed with fungicides for one hundred years.

The introduction of synthetic chemical fungicides in the 1940’s led to their rapid adoption because they were more effective in controlling the fungi and did less damage to the crops than copper and sulfur. Because the synthetic fungicides are used at significantly lower use rates, the aggregate use of fungicides in U.S. crop production declined by over 50%.

For 231 diseases of fifty crops, fungicides are the primary means of defense from fungi. U.S. growers spray 108 million pounds of fungicides at a cost of $880 million. As a result of fungicide application, yields of most fruit and vegetable crops are increased by 50 to 95%. Growers gain $12.8 billion in increased production value from the control of plant diseases caused by fungi.

The benefit of fungicide use in U.S. agriculture is a significant increase in the production of fruit and vegetables, which are so important for the healthy diets of Americans.

Organic growers use fungicides too, which further indicates their indispensability.

Since fungicides are so important in United States food production, it is worth asking whether there should be policies adopted to assure that American growers have the fungicides that they need for the foreseeable future.
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## Table 2: Crop Diseases Targeted by Fungicide Use in the U.S.

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<td>Brown Rot</td>
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<td>Green Fruit Rot</td>
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<td>Pink Rot</td>
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<td></td>
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<td>Shot Hole</td>
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<td>Damping Off &amp; Pythium Seed Rot</td>
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<td>Smut</td>
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<td>Strawberries</td>
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<td>Parsley</td>
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<td>Anthracnose</td>
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<td>Black Root Rot</td>
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<td></td>
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<td>Gray Mold</td>
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<td>Leaf Spot</td>
</tr>
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<td></td>
<td>Pepper Spot</td>
<td></td>
<td>Leather Rot</td>
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<td></td>
<td>Pythium</td>
<td></td>
<td>Powdery Mildew</td>
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<td></td>
<td>Rhizoctonia</td>
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<td>Red Stele</td>
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<td>Rust</td>
<td>Sugarbeets</td>
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<td>Sclerotinia Blight</td>
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<td>Powdery Mildew</td>
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<tr>
<td></td>
<td>Web Blotch</td>
<td></td>
<td>Northern Corn Leaf Blight</td>
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<td></td>
<td>Southern Corn Leaf Blight</td>
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<td></td>
<td>Fabraea Leaf Spot</td>
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<td>Anthracnose</td>
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<td>Fire Blight*</td>
<td></td>
<td>Bacterial Spot*</td>
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<td>Mycosphaerella Leaf Spot</td>
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<td>Cercospora Leaf Spot</td>
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<td>Phytophthora Blight</td>
</tr>
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<td></td>
<td>Scab</td>
<td></td>
<td>Powdery Mildew</td>
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<tr>
<td></td>
<td>Sooty Blotch and Fly Speck</td>
<td></td>
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<tr>
<td>Pears</td>
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<td>Pistachios</td>
<td>Alternaria</td>
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<td>Panicle &amp; Shoot Blight</td>
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</table>

* indicates a particularly common disease affecting the species.
<table>
<thead>
<tr>
<th>Tomatoes</th>
<th>Walnuts</th>
<th>Walnut Blight*</th>
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<tbody>
<tr>
<td>Anthracnose</td>
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<td></td>
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<tr>
<td>Bacterial Canker*</td>
<td>Fusarium Head Blight</td>
<td></td>
</tr>
<tr>
<td>Bacterial Speck*</td>
<td>Leaf Rust</td>
<td></td>
</tr>
<tr>
<td>Bacterial Spot*</td>
<td>Powdery Mildew</td>
<td></td>
</tr>
<tr>
<td>Black Mold</td>
<td>Septoria Leaf and Glume Blotch</td>
<td></td>
</tr>
<tr>
<td>Buckeye Rot</td>
<td>Stem Rust</td>
<td></td>
</tr>
<tr>
<td>Early Blight</td>
<td>Stripe Rust</td>
<td></td>
</tr>
<tr>
<td>Gray Leaf Spot</td>
<td>Wild Rice</td>
<td>Fungal Brown Spot</td>
</tr>
<tr>
<td>Gray Mold</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Late Blight</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leaf Mold</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phoma Rot</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phytophthora Root Rot</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Powdery Mildew</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pythium</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septoria Leaf Spot</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Southern Blight</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Target Spot</td>
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* Bacterial Diseases
### Table 3: Apple Disease Incidence

<table>
<thead>
<tr>
<th>Disease</th>
<th>% Acres Infected</th>
<th>% Yield Loss w/o Control</th>
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</thead>
<tbody>
<tr>
<td><strong>Eastern States</strong></td>
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<td></td>
</tr>
<tr>
<td>Alternaria Blotch</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Bitter Rot</td>
<td>14</td>
<td>90</td>
</tr>
<tr>
<td>Black Pox</td>
<td>2</td>
<td>20</td>
</tr>
<tr>
<td>Black Rot</td>
<td>25</td>
<td>60</td>
</tr>
<tr>
<td>Blossom End Rot</td>
<td>12</td>
<td>5</td>
</tr>
<tr>
<td>Brooks Spot</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Cedar Apple Rust</td>
<td>51</td>
<td>85</td>
</tr>
<tr>
<td>Fire Blight</td>
<td>60</td>
<td>85</td>
</tr>
<tr>
<td>Fly Speck</td>
<td>65</td>
<td>42</td>
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<tr>
<td>Phytophthora Collar/Crown Rot</td>
<td>7</td>
<td>75</td>
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<tr>
<td>Powdery Mildew</td>
<td>40</td>
<td>65</td>
</tr>
<tr>
<td>Quince Rust</td>
<td>50</td>
<td>60</td>
</tr>
<tr>
<td>Scab</td>
<td>100</td>
<td>90</td>
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<tr>
<td>Sooty Blotch</td>
<td>70</td>
<td>42</td>
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<tr>
<td>White Rot</td>
<td>20</td>
<td>65</td>
</tr>
<tr>
<td><strong>Western States</strong></td>
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<td></td>
</tr>
<tr>
<td>Bulls Eye Rot</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Scab</td>
<td>52</td>
<td>25</td>
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<tr>
<td>Powdery Mildew</td>
<td>50</td>
<td>10</td>
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</tbody>
</table>

Source: [13][15]

### Table 4: Cranberry Disease Incidence:

#### New Jersey/Massachusetts

<table>
<thead>
<tr>
<th>Disease</th>
<th>% Acres Infected</th>
<th>% Yield Loss w/o Control</th>
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<tbody>
<tr>
<td>Berry Rot/Leaf Spot</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
<td>Bitter Rot</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Blotch Rot/Leaf Spot</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Early Rot</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>End Rot</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>Fairy Ring</td>
<td>10</td>
<td>30</td>
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<td>Peicillium Rot</td>
<td>10</td>
<td>15</td>
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<tr>
<td>Twig Rot/Leaf Spot</td>
<td>25</td>
<td>50</td>
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Source: [15]
### Table 5: Incidence of Peanut Diseases & Their Potential Yield Reductions

<table>
<thead>
<tr>
<th>Disease</th>
<th>Southeast</th>
<th>Southwest</th>
<th>Virginia/ N. Carolina</th>
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<tbody>
<tr>
<td></td>
<td>% Acres Infested</td>
<td>% Yield Loss Potential</td>
<td>% Acres Infested</td>
</tr>
<tr>
<td>Late Leafspot</td>
<td>99</td>
<td>50</td>
<td>80</td>
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<tr>
<td>Early Leafspot</td>
<td>80</td>
<td>50</td>
<td>75</td>
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<tr>
<td>S. rolfsi, Stem Rot</td>
<td>40</td>
<td>30</td>
<td>25</td>
</tr>
<tr>
<td>Sclerotinia Blight</td>
<td>0</td>
<td>0</td>
<td>20</td>
</tr>
<tr>
<td>Rhizoctonia Limb Rot</td>
<td>35</td>
<td>25</td>
<td>35</td>
</tr>
<tr>
<td>Rust</td>
<td>20</td>
<td>15</td>
<td>10</td>
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<tr>
<td>Web Blotch</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Pythium</td>
<td>10</td>
<td>5</td>
<td>5</td>
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<tr>
<td>Leaf Scorch/Pepper Spot</td>
<td>25</td>
<td>5</td>
<td>10</td>
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<td>Cylindrocladium Black Rot</td>
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Source: [536][537][538]

### Table 6: Strawberry Diseases Targeted by Fungicide Use

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<th>CA</th>
<th>FL</th>
<th>MI</th>
<th>NC</th>
<th>NY</th>
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<th>PA</th>
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<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
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<td>X</td>
<td>X</td>
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<td></td>
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<tr>
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<td>X</td>
<td>X</td>
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<td>X</td>
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<td>X</td>
<td></td>
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<td>Leather Rot</td>
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<td>X</td>
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<td>Red Stele</td>
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Source: [137]

### Table 7: Fungicide Use in U.S. Crop Production

<table>
<thead>
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<th>Year</th>
<th>All Crops</th>
<th>Apples</th>
<th>Potatoes</th>
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<td>1944</td>
<td>296</td>
<td>92</td>
<td>56</td>
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<tr>
<td>1971</td>
<td>154</td>
<td>8</td>
<td>4</td>
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<tr>
<td>1997</td>
<td>131</td>
<td>8</td>
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Source: [603][397][398]

1) Includes Sulfur & Copper
<table>
<thead>
<tr>
<th>Crop</th>
<th>Acreage</th>
<th>Volume (million lbs.)</th>
<th>Value ($ Thousand)</th>
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<td>Almonds</td>
<td>696,424</td>
<td>1,090</td>
<td>1,189,000</td>
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<td>Apples</td>
<td>446,491</td>
<td>8,466</td>
<td>1,550,235</td>
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<td>Artichokes</td>
<td>7,725</td>
<td>94</td>
<td>66,764</td>
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<tr>
<td>Asparagus</td>
<td>68,632</td>
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<td>173,000</td>
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<td>1,844</td>
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<td>8,385</td>
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<td>Barley</td>
<td>4,015,654</td>
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<td>525,275</td>
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<td>29,251</td>
<td>1,997</td>
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<td>44</td>
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<td></td>
<td><strong>107,960</strong></td>
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*Note: Includes the 50 crops identified in Table 10 summed by state.*
Table 12: U.S. Farm-Level Use Volume and Sales of Fungicides, 2002

<table>
<thead>
<tr>
<th>Active Ingredient</th>
<th>$/Lb. AI&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Volume Applied (Lbs. AI/Year)</th>
<th>Sales/Year ($)</th>
</tr>
</thead>
<tbody>
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<td>534,616</td>
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<td>4,783,646</td>
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<td>5.75</td>
<td>3,085,306</td>
<td>17,740,507</td>
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<td>Chlorothalonil</td>
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<td>57,462,402</td>
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<td>22,471,979</td>
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<td>121,734</td>
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<tr>
<td>DCNA</td>
<td>14.00</td>
<td>108,776</td>
<td>1,522,862</td>
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<tr>
<td>Dimethomorph</td>
<td>39.40</td>
<td>11,857</td>
<td>467,184</td>
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<td>Dodeine</td>
<td>17.86</td>
<td>65,629</td>
<td>1,172,142</td>
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<td>Etridiazole</td>
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<td>623,319</td>
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<td>Fenamidol</td>
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<td>4,709,454</td>
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<td>Fenbuconazole</td>
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<td>Fenhexamid</td>
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<td>859,542</td>
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<td>Fludioxonil</td>
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<td>Flutolanic</td>
<td>29.80</td>
<td>127,637</td>
<td>3,803,592</td>
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<td>Fosetyl-AL</td>
<td>15.75</td>
<td>543,800</td>
<td>8,558,232</td>
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<td>5,336,614</td>
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<td>Myclobutanil</td>
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<td>2,636,052</td>
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<td>Pyraclostrobin</td>
<td>59.00</td>
<td>137,322</td>
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<td>48,923</td>
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<td>Thiophanate Methyl</td>
<td>21.45</td>
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<td>7,825,126</td>
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<td>Thiram</td>
<td>4.71</td>
<td>226,772</td>
<td>1,068,098</td>
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<td><strong>Total</strong></td>
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<td><strong>575,661,639</strong></td>
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1) Source: [655]-[664]

Data includes in-field crop uses only (foliar and in-furrow). Data excludes fungicide use on ornamentals, seed treatments, and post-harvest uses.
Table 13: Benefit of Fungicide Use in U.S. Crop Production

<table>
<thead>
<tr>
<th>Crop</th>
<th>% Acres Treated</th>
<th>% Yield Attributable to Fungicides</th>
<th>Production Increase</th>
</tr>
</thead>
<tbody>
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<td></td>
<td></td>
<td></td>
<td>Million Lbs.</td>
</tr>
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<td>82</td>
<td>70</td>
<td>626</td>
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<tr>
<td>Apples</td>
<td>93</td>
<td>86</td>
<td>6,803</td>
</tr>
<tr>
<td>Artichokes</td>
<td>81</td>
<td>35</td>
<td>27</td>
</tr>
<tr>
<td>Asparagus</td>
<td>43</td>
<td>22</td>
<td>18</td>
</tr>
<tr>
<td>Bananas</td>
<td>75</td>
<td>30</td>
<td>4</td>
</tr>
<tr>
<td>Barley</td>
<td>9</td>
<td>16</td>
<td>158</td>
</tr>
<tr>
<td>Blueberries</td>
<td>75</td>
<td>63</td>
<td>116</td>
</tr>
<tr>
<td>Cabbage</td>
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<td>596</td>
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<td>Cantaloupes</td>
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<td>39</td>
<td>727</td>
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<td>526</td>
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<td>71</td>
<td>2,180</td>
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<td>Pears</td>
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<td>99</td>
<td>1,526</td>
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<td>44</td>
<td>53</td>
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<td><strong>Total</strong></td>
<td></td>
<td></td>
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</tbody>
</table>

1) National average % yield increase from fungicide use on treated acreage.
# Table 14: Summary of Fungicide Use Net Value by Crop ($ 000)

<table>
<thead>
<tr>
<th>Crop</th>
<th>Current Fungicide Use Cost (-)</th>
<th>Production Increase (+)</th>
<th>Net Value (+)</th>
<th>Return Ratio (+)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almonds</td>
<td>27,565</td>
<td>682,486</td>
<td>654,921</td>
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<tr>
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<td>1,153,399</td>
<td>17.57</td>
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<td>286</td>
<td>18,928</td>
<td>18,642</td>
<td>66.18</td>
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<td>13,907</td>
<td>14.05</td>
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<td>1,655</td>
<td>8.13</td>
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<td>4,977</td>
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<td>2,671</td>
<td>58,574</td>
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<td>21.93</td>
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<td>16.19</td>
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<td>89,191</td>
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<td>926,968</td>
<td>896,991</td>
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<tr>
<td><strong>Total</strong></td>
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<td><strong>12,849,735</strong></td>
<td><strong>11,969,382</strong></td>
<td><strong>14.60</strong></td>
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</tbody>
</table>

1) From Table 10  
2) From Table 13: Production increases attributable to fungicides.  
3) Value of production increase from fungicide use less current fungicide cost.  
4) Ratio of production increase to current fungicide cost.
### Table 15: Summary of Fungicide Use Net Value by State ($ 000)

<table>
<thead>
<tr>
<th>State</th>
<th>Current Fungicide Use Cost (-)</th>
<th>Production Increase (+)</th>
<th>Net Value (+)</th>
<th>Return Ratio (+)</th>
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<td>1.77</td>
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<td><strong>880,353</strong></td>
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<td><strong>11,969,382</strong></td>
<td><strong>14.60</strong></td>
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</tbody>
</table>

1) From Table 11
2) From Table 1.4 through 50.4 in the Data Appendix: Production increases attributable to fungicides.
3) Value of production increase from fungicide use less current fungicide cost.
4) Ratio of production increase to current fungicide cost.
<table>
<thead>
<tr>
<th>State</th>
<th>Crop Impacts</th>
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<td>Alabama</td>
<td>Collards -30%; Peaches -75%; Peanuts -78%; Tomatoes -45%; Watermelons -35%</td>
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<tr>
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<td>Cabbage -12%; Cantaloupes -20%; Citrus -25%; Cotton -20%; Lettuce -45%</td>
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<tr>
<td>Arkansas</td>
<td>Cotton -10%; Peaches -75%; Rice -30%; Soybeans -25%; Tomatoes -30%</td>
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<td>Almonds -70%; Carrots -25%; Citrus -25%; Grapes -97; Strawberries -55%</td>
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<tr>
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<td>Carrots -40; Lettuce -45%; Onions -15%; Potatoes -15%; Sugarbeets -30%</td>
</tr>
<tr>
<td>Connecticut</td>
<td>Apples -100%; Peaches -75%; Sweet Corn -15%</td>
</tr>
<tr>
<td>Delaware</td>
<td>Cantaloupes -35%; Potatoes -50%; Sweet Corn -3%; Watermelons -35%; Wheat -20%</td>
</tr>
<tr>
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<td>Citrus -50%; Cucumbers -100%; Strawberries -90%; Sweet Corn -60%; Tomatoes -100%</td>
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<tr>
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<td>Onions -25%; Peaches -100%; Peanuts -78%; Pecans -55%; Watermelons -100%</td>
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<tr>
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<td>Bananas -30%; Papaya -100%</td>
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<tr>
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<td>Barley -35%; Hops -69%; Onions -20%; Potatoes -41%; Sugarbeets -22%</td>
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<td>Apples -100%; Green Beans -25%; Peaches -75%; Potatoes -50%; Sweet Corn -17%</td>
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<td>Apples -100%; Peaches -75%; Soybeans -25%; Wheat -20%</td>
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<tr>
<td>Louisiana</td>
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<td>Apples -100%; Blueberries -60%; Potatoes -100%</td>
</tr>
<tr>
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<td>Apples -100%; Cranberries -100%; Peaches -75%; Potatoes -60%; Tomatoes -75%</td>
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<td>Potatoes -50%; Sugarbeets -30%; Sweet Corn -62%; Wheat -20%; Wild Rice -75%</td>
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<td>Mississippi</td>
<td>Cotton -10%; Rice -15%; Soybeans -25%; Watermelons -35%; Wheat -20%</td>
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<td>Missouri</td>
<td>Apples -100%; Cotton -12%; Peaches -75%; Watermelons -50%; Wheat -20%</td>
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<td>Montana</td>
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</tr>
<tr>
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<td>Apples -100%</td>
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<td>Blueberries -60%; Cranberries -100%; Peaches -75%; Spinach -81%; Sweet Peppers -80%</td>
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<td>Ohio</td>
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<td>South Carolina</td>
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<tr>
<td>Tennessee</td>
<td>Cotton -22%; Green Beans -30%; Peaches -75%; Soybeans -25%; Tomatoes -30%</td>
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<tr>
<td>Texas</td>
<td>Citrus -50%; Onions -60%; Peanuts -59%; Spinach -55%; Watermelons -50%</td>
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<tr>
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<td>Onions -3%; Potatoes -12%</td>
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<tr>
<td>Vermont</td>
<td>Apples -100%</td>
</tr>
<tr>
<td>Virginia</td>
<td>Apples -100%; Peanuts -72%; Potatoes -66%; Tomatoes -20%; Wheat -20%</td>
</tr>
<tr>
<td>Washington</td>
<td>Apples -80%; Cherries -75%; Grapes -80%; Pears -100%; Potatoes -50%</td>
</tr>
<tr>
<td>West Virginia</td>
<td>Apples -100%; Peaches -75%; Wheat -20%</td>
</tr>
<tr>
<td>Wisconsin</td>
<td>Cranberries -60%; Green Beans -20%; Potatoes -26%; Strawberries -25%; Sweet Corn -20%</td>
</tr>
<tr>
<td>Wyoming</td>
<td>Sugarbeets -30%</td>
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</table>

*Note: Selected Impacts Only, Impacts on Treated Acres Only*

See Tables 1.4 through 50.4 in the Data Appendix for a complete list.
### Table 17: National Ranking: Fungicide Use and Cost by Crop

<table>
<thead>
<tr>
<th>Rank</th>
<th>% Acres Treated</th>
<th>Lbs./Year (million lbs)</th>
<th>Cost ($ Million/Year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Grapes</td>
<td>100 Grapes</td>
<td>45 Grapes</td>
</tr>
<tr>
<td>2</td>
<td>Hops</td>
<td>100 Tomatoes</td>
<td>10 Potatoes</td>
</tr>
<tr>
<td>3</td>
<td>Papaya</td>
<td>100 Apples</td>
<td>7 Peanuts</td>
</tr>
<tr>
<td>4</td>
<td>Raspberries</td>
<td>97 Potatoes</td>
<td>7 Apples</td>
</tr>
<tr>
<td>5</td>
<td>Strawberries</td>
<td>97 Citrus</td>
<td>7 Walnuts</td>
</tr>
<tr>
<td>6</td>
<td>Carrots</td>
<td>95 Peaches</td>
<td>5 Rice</td>
</tr>
<tr>
<td>7</td>
<td>Potatoes</td>
<td>94 Sugarbeets</td>
<td>4 Sugarbeets</td>
</tr>
<tr>
<td>8</td>
<td>Apples</td>
<td>93 Peanuts</td>
<td>3 Tomatoes</td>
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<tr>
<td>9</td>
<td>Celery</td>
<td>92 Cherries</td>
<td>2 Citrus</td>
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<tr>
<td>10</td>
<td>Cherries</td>
<td>92 Almonds</td>
<td>2 Cotton</td>
</tr>
<tr>
<td>11</td>
<td>Peanuts</td>
<td>92 Walnuts</td>
<td>1 Almonds</td>
</tr>
<tr>
<td>12</td>
<td>Peaches</td>
<td>91 Pears</td>
<td>1 Pecans</td>
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</table>

From Table 10

### Table 18: National Ranking: Fungicide Use and Cost by State

<table>
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<tr>
<th>Rank</th>
<th>Lbs./Year (million lbs)</th>
<th>Cost ($ Million/Year)</th>
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<td>2</td>
<td>Florida 9</td>
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<td>3</td>
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<td>Michigan 4</td>
<td>North Dakota 58</td>
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<tr>
<td>5</td>
<td>Georgia 4</td>
<td>Washington 58</td>
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<td>6</td>
<td>Idaho 3</td>
<td>Minnesota 48</td>
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<td>7</td>
<td>New York 2</td>
<td>Michigan 40</td>
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<td>12</td>
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From Table 11

### Table 19: National Ranking: Fungicide Benefits

<table>
<thead>
<tr>
<th>Rank</th>
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<th>Rank</th>
<th>By State Million $/Year</th>
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<td>Apples</td>
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<td>Potatoes</td>
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<td>Washington 1,453</td>
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<td>4</td>
<td>Citrus</td>
<td>4</td>
<td>Georgia 406</td>
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<td>5</td>
<td>Lettuce</td>
<td>5</td>
<td>Arizona 362</td>
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<td>6</td>
<td>Tomatoes</td>
<td>6</td>
<td>Idaho 320</td>
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<tr>
<td>7</td>
<td>Strawberries</td>
<td>7</td>
<td>Michigan 294</td>
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<td>8</td>
<td>Almonds</td>
<td>8</td>
<td>Oregon 271</td>
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<tr>
<td>9</td>
<td>Peanuts</td>
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<td>Texas 235</td>
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<tr>
<td>10</td>
<td>Sweet Peppers</td>
<td>10</td>
<td>New York 224</td>
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From Tables 13 and 15
Figure 1
Disease Cycle of Cercospora Leaf Spot (C. beticola) on Sugarbeets

Source: [677]

Figure 2
Sequence of Powdery Mildew Occurrence on Sugarbeets in the Western United States

Source: [670]
Figure 3
California Artichoke Yield

Source: [169]

Figure 4
California Garlic Yield

Source: [169]
Figure 5
U.S. Apple Production

Source: [44]

Figure 6
Maine Potato Yields

Source: [44]
Figure 7  
Florida Cucumber Yield  
Source: [44]

Figure 8  
U.S. Peanut Yield  
Source: [44]
Figure 9
U.S. Pecan Yield

Lbs./Tree

Source: [44]

Figure 10
Minnesota Wild Rice Production

Million Lbs./Year

Source: [46]
Figure 11
Total Fungicide Use in U.S. Crop Production

Sources: [397][398][603]

Figure 12
Potato Fungicide Use Rates

Source: [8]
Figure 13
Impact of Mummyberry Disease on Georgia Blueberries

Source: [32]

Figure 14
Impact of Seedling Diseases on Georgia Cotton

Source: [32]
Figure 15
Impact of White Mold Disease on Georgia Peanuts

Source: [32]

Figure 16
Relative Relationship between Sugarbeet Cultivar Yield and Cercospora Resistance

Source: [695]
Figure 17: Fungicide Use in Crop Production by State

Source: Table 11

Figure 18: Crop Production Increase Due to Fungicide Use by State

Source: Table 15
## 6.0 Crop Literature Review

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</table>
6.1 Almonds

California produces three-quarters of the world’s supply of almonds. High yields enable California growers to produce five times the crop on one-third the acreage of the nearest global contender, Spain. Approximately 80% of the California crop is exported. Almonds are the top ranking United States horticultural export.

Almond trees are susceptible to nine diseases caused by fungi which are controlled with the applications of fungicides: brown rot, green fruit rot, anthracnose, shot hole, scab, leaf blight, alternaria leaf spot, leaf rust, and root/crown rot. Losses from these diseases are reflected not only in a reduction of crop yield, but in branch and root death, tree decline, and the death of young and mature trees.[464] An experiment with a combination of shot hole, brown rot, and green fruit rot demonstrated that yields in untreated controls were 70% lower than fungicide-treated almond trees.[460]

Shot hole attacks both leaves and young fruit and can result in defoliation or premature nut drop. Almost complete defoliation can occur.[461] The fungus lives in a dormant condition inside buds and twig lesions in the winter. In the spring, spores produced from the lesions infect new leaves and fruit. Small areas of leaves are killed and as the leaves grow these spots drop out. This leaves the characteristic “shot holes.”[467] Infected fruit becomes rough and corky. Under severe circumstances, it may cause the hulls to gum internally, affecting kernel quality.[468] Research in the 1930’s demonstrated that applications of Bordeaux mixture to almond trees materially reduced shot hole leaf infections and the subsequent defoliation, increasing the yield of nuts by 200%.[332] Experiments with ziram applications reduced the number of hulls exhibiting more than five infection sites per hull from 187 per 200 hulls to two per 200 hulls.[468] Research showed that two captan applications reduced the number of lesions per fruit by 96%.[460] It is estimated that 80% of California’s almond acres are infected with the shot hole fungus.[13] A four-year research study determined that severe shot hole disease resulted in production losses ranging from 50 to 75%.[464] In California, one to three fungicide applications are made to control shot hole.[39]

Brown rot first appeared on California almonds in the late 1800s and currently occurs in most almond producing areas in California.[461] The disease is worse when rains or fog occur during bloom. The fungus survives and overwinters in almond orchards in twig cankers and dead blossom parts. Brown rot cankers can enlarge each season and continue to sporulate for 4 years or more.[39] In early spring, the fungus produces spores which are wind-disseminated to blossoms. Infected flowers wither, collapse, and remain attached to twigs. The infected blossoms are covered with light brown powdery masses of fungus spores during humid weather. The fungus moves from the blossoms into adjacent twigs and kills them as well.[462] Gumming is common at the base of blighted flowers.[39] Almost complete crop loss can be experienced on susceptible cultivars when rain persists during bloom.[461] Damage is often experienced several years after a severe infection because of the loss of fruiting spurs. Uncontrolled brown rot is estimated to reduce almond yields by 20%.[13] Control of brown rot is based on the protection of flower parts by the use of fungicides through bloom.[461] Research has
shown that in untreated controls, 44% of the twigs were infected with brown rot compared to 4% where fungicides were applied.[463]

Green fruit rot, also called jacket rot, is found widespread throughout almond production regions in California.[464] The severity of green fruit rot varies greatly from year to year but can cause up to 10% yield loss when wet weather persists.[461] The fungus overwinters in decaying plant material in orchards. Spores are produced in cool, wet weather. Spores are spread by air currents and deposited on blossom parts; they can survive one to three months if the weather remains cool and humid.[462] Green fruit rot begins during the latter part of the bloom period when the fungus invades flower petals and jackets. Diseased jackets wither and stick to developing fruit. As the fruit starts to grow, a brown spot develops. The infected spot expands rapidly until the entire fruit rots. Frequently, fruit will fall off the tree, and the result is a reduction in yield.[462] The disease is usually controlled by fungicide applications made to control other bloom time fungal diseases. However, when bloom is extended by cool temperatures and moisture is present, growers are advised to make an application specifically to prevent green fruit rot.[462]

Leaf blight of almonds was first identified in 1950, when it developed in a few Sacramento Valley orchards.[462] By 1983, it spread throughout the Valley.[464] Almond leaf blight is characterized by the dying of leaves throughout the summer.[465] The fungus interferes with water conduction in the leaf. In fall and winter, dormant buds are killed by an extension of the lesion and in the spring, flowers are killed.[465] Repeated attacks of leaf blight, though rarely killing more than 20% of the leaves, ultimately reduce tree vigor. The death of flower buds causes a loss in yield during the current season. Experiments in the 1950s demonstrated that the use of protectant fungicides (captan and ziram) reduced the incidence of leaf blight by 75-80% in comparison to unsprayed trees.[465]

Rust was first detected in almond orchards in the northern Sacramento Valley and is now distributed throughout the Sacramento and San Joaquin Valleys.[464] Rust typically develops in summer and fall in almonds. The disease is characterized by angular yellow leaf spots on upper leaf surfaces and rusty brown masses of spores on lower leaf surfaces.[461] Leaf rust can cause severe defoliation in a short period of time if conditions are favorable.[461] In 1991 and 1992, observations were made of almond trees with over 50% defoliation at harvest due to rust with essentially no leaves after harvest in the month of September.[464] In California wettable sulfur is applied in the spring and early summer where early-season rust outbreaks are common.[39] Evaluations have shown that both the incidence and severity of rust were significantly reduced with maneb treatments compared to the nontreated trees.[464]

Outbreaks of almond scab were reported in 1924 and documented between 1953-1957, and again in 1983. By 1992, scab was widespread in all the major almond producing regions in California.[464] This spread occurred when the use of sprinkler irrigation became popular.[464] Scab infects leaves, fruit, and twigs in almonds causing dark spots to form. Scab lesions look greasy and oily. The major concern with almond scab is
partial or complete defoliation of the tree. In the summer of 1991, severe epidemics of scab were observed in Merced and Butte counties. Complete defoliation of certain varieties occurred before harvest.[464] If early defoliation is severe, fruit drop also occurs. Scab infections left uncontrolled for several years will weaken trees and reduce yield.[462] An annual treatment of captan or ziram reduced scab infections. Treatments must be made before scab symptoms appear.[461]

Crown and root rot of almonds is caused by at least 14 different *Phytophthora* species. Risk of root or crown infection is greatest during cool to moderate temperatures with prolonged or frequent water saturation in or on soil. The water-filled pores in a saturated soil favor reproduction of *Phytophthora* and dispersal of its swimming spores which are the principal agents of root infections. Canal and river water are frequently contaminated with *Phytophthora* and the pathogens are brought into the orchard in irrigation water.[461] The pathogen enters the tree either at the crown near the soil line, at the major roots, or at the feeder roots, depending on the species. A tree infected with *Phytophthora* can either undergo a period of slow decline that may last a few years or it can suddenly collapse and die in spring or early summer with the advent of warm weather.[462] Some *Phytophthora* species cause massive infections and decay of the entire root system that results in rapid death. The rotted root system cannot adequately supply the aboveground portions of the tree with water and minerals. Eventually, leaves drop, terminal shoots die back, followed by death of the tree. Once in the root or crown the infection, depending on the species, may extend into the crown, trunk, or branches.[462] Infected almond bark becomes discolored. Active cankers often ooze and release an amber-colored exudate in balls, strands, or puddles. In the Sacramento Valley and upper San Joaquin Valleys, tree losses to crown rot were prevalent in the 1990’s.[466] In one orchard 9% of the trees died in 1994, in another 2% died by 1996, and many remaining trees were symptomatic in 1997. In five other orchards, tree losses ranged from 1 to 10%.[466] Currently, it is estimated that crown and root infections are a problem affecting 20% of California’s almond orchards with potential yield losses of 50%.[461]

Alternaria leaf spot was first observed in California in the late 1980s and was first associated with severe defoliation of almond trees in the mid-1990s.[469] Orchards in areas with frequent dews, high humidity, and little or no air movement sustained severe defoliation resulting in yield losses often exceeding 50%.[469] Symptoms occur only on the leaves as lesions with tan spots. The center of the lesions becomes black with fungal sporulation. Damage is not only in decreased yields but also weakening of the tree, thereby decreasing the potential lifespan of the tree. Debilitation by *Alternaria* can lead to tree death within 3-4 years of the first serious outbreak. Yield losses tend to become greater each year as the tree is progressively weakened. 60-90% control of *Alternaria* is achieved with three fungicide applications.[470]

In California, anthracnose was rarely seen on almonds until the late 1980s but has since caused crop and orchard losses.[39] In 1996 anthracnose was widespread in California and the disease caused substantial losses.[471] Typical losses in 1996 were 10-15% of the crop in infected orchards with severely affected orchards incurring a yield loss of
25%. The fungus is now found in all major almond growing regions and is considered a major threat to the industry in the state. Under wet conditions, orange spore masses are produced and appear as visible droplets. Diseased fruit die and turn into mummies that remain on the tree. The pathogen overwinters in the mummies. Lesions on mature fruit are rusty orange and gum profusely. As the fungus infects the almond, toxins produced by the fungus enter the branches. The result is that branch dieback occurs. The fungus also grows through the hull and into the kernel. The pathogen has been isolated from processed almond kernels in storage. Symptoms include brown and purplish discoloration of the kernel tissue. Anthracnose continues to grow while almonds are in storage. In 1996, up to 10% of the almonds in storage tested positive for anthracnose. The disease is managed in California by applying fungicides to protect new growth before rains begin. The state of California has estimated that without the use of fungicides, anthracnose would reduce the state’s almond production by 16-30%. 80-90% control of anthracnose is achieved with proper timing of fungicide applications.

6.2 Apples

Prior to 1880, there was little spraying of apples in the United States. Apples were grown primarily for cider and fruit damage was tolerated. It was only when growing apples for dessert and commercial apple markets emerged around 1860 that attention was directed toward reducing damage to fruit. As Americans became primarily town and city dwellers, they also became more finicky and, in order to sell apples, farmers had to produce blemish free fruit. Spraying for control of fungal diseases of apples started in the U.S. somewhere between 1880 and 1905, following the discovery of Bordeaux mixture in France. In 1908, in Oregon, it was discovered that liquid lime sulfur was an effective fungicide for control of apple scab. Lime sulfur was quickly adopted as the standard remedy for apple scab control because it provided better scab control with less foliar and fruit injury than had been possible with Bordeaux mixture. By the 1920s spraying became a universal practice in U.S. apple orchards and it was impossible to grow apples for market without fungicide sprays.

In 1910 sprayers consisted of 50 gallon barrels equipped with a hand-operated pump. Some were mounted on the bed of a horse-drawn wagon with a wooden tower and a long-handled spray gun to aid in reaching tree tops. Spraying apple trees with lime sulfur was an extremely unpleasant task as testified to in childhood recollections from the 1920s:

The smell of the lime sulfur spray was fearsome and driving the sprayer as I sometimes had to do was a horrid task. The spray left a whitish-greenish surface and I remember my father coming in at night, his clothes, hat and some of his skin stained with this.

Evidence accumulated that lime sulfur decreased the productivity of apple trees and there
was no way to use it during the growing season without an undesirable degree of injury.[721] The use of elemental sulfur escalated in the 1930s as a replacement for lime sulfur. Elemental sulfur provided fair disease control. However, some growers still lost 10% or more of their crop to scab and other diseases. Sulfur injury was common and appeared to limit the yield of such apple cultivars as Stayman and Deliciousm.[722]

Far superior control of apple scab and other diseases was obtained in the 1940s after the introduction of the synthetic chemical fungicides. Ferbam controlled scab, sooty blotch, fly speck, rots, and rusts; its use allowed apple growers to observe an orchard of healthy apple trees free from injury for the first time.[721] Only 1.5 pounds of ferbam was used in comparison to the recommended 12 to 16 pounds of sulfur diluted in 100 gallons of water. Trees sprayed with ferbam showed a yield increase of 41% over similar trees sprayed with wettable sulfur.[723] Large increases in apple production occurred in the 1950s as growers switched to the new synthetic chemical fungicides due to superior disease control efficacy and reduced damage to the trees. By 1960, U.S. apple growers had a set of fungicides to prevent epidemics of the major fungal diseases.[722]

In apple production, disease control requires the use of fungicides applied in a pest control program followed year after year. Disease problems can be so severe that a delay of one or two days in application of a critical fungicide spray results in loss of the crop and possibly the trees.[721]

Fungicides are used to control sixteen major diseases of apples in the United States. These diseases and their incidence in eastern and western apple states are listed in Table 3.

Apple scab is caused by a fungus *Venturia inaqualis*, which overwinters in infected leaves on the orchard floor. Mating among different strains of the fungus occurs shortly after leaf fall and spores develop in the fallen leaves during the winter. Spring rains cause spores to be forcibly discharged; they can be carried long distances by air currents to flowers, leaves, or young fruit. In one research project 289 spores were found in a cubic foot of orchard air during spring rains.[719] Spores continue to mature and are discharged over a period of 5-9 weeks.[703] If the surface of apple tissue is wet and temperatures are suitable, the spores germinate and penetrate the cuticle and outer layers of the plant, causing an infection. The fungus grows beneath the cuticle and eventually ruptures it and forms dark green lesions. Masses of spores are produced asexually within the lesions and become detached during rain. Water splashes and redistributes these spores, causing secondary infections. The scab fungus is confined primarily to the area between the cuticle and outer cell layers. Each leaf scab lesion is capable of producing 50,000-100,000 spores.[716] The number of lesions per leaf can range from 1 or 2 to several hundred.[703] Assuming 50,000 leaves per tree have 2% scab infection, about 50 million spores would be present on a single tree. One spore can cause an infection. Infected leaves become curled, dwarfed and distorted. They often shrivel and fall from the tree. Severe leaf infections can lead to premature defoliation, which can reduce tree growth and yield for one to several years and increase susceptibility to winter injury. A solid layer of fungal growth several cells deep is formed over the area of the lesion. As
the infected fruit enlarge, the lesions become brown and corky. Infections early in the season can kill tissues near the fruit surface and the fruit develops unevenly as uninfected portions continue to grow. Cracks appear in the skin and flesh and the fruit may become deformed.[703] Heavily infected fruit fall from the tree resulting in yield losses. Scab lesions on harvested apples result in a lower price for growers since the commercial tolerance for scab damage approaches zero.

Apple scab is the most economically important disease of apples in eastern states. USDA estimates that 100% of eastern orchards are infected with the scab fungus and that 90% yield loss would occur without fungicide treatments.[13][15] An illustration of the persistent threat of loss from scab is the annual occurrence, for over 40 years, of 98-100% fruit infection on unsprayed trees in fungicide spray trials conducted at Michigan State University.[717] In Pennsylvania in 1938, scab incidence was 82% in 50 unsprayed apple orchards, while in 109 properly sprayed orchards scab incidence was 1.4%.[302] Scab is less common in semiarid regions in western states where it is limited to orchards on low ground with poor air circulation and slow drying conditions.[718] USDA estimates that 52% of apple acres in western states are infected, with the potential for uncontrolled apple yield loss of 25%.[13][15] Experiments typically show large reductions in the percent of scabby apples with the use of fungicides. For example, scab was reduced from 77% to 2% in one experiment.[724]

More than 20 apple cultivars bred with resistance to apple scab have been released. When the scab spores land on these cultivars, they penetrate the apple cuticle. However, the infection does not spread in the apple. The exact process that retards the infection is not known. The Liberty cultivar has been grown without fungicide use for scab since 1956. These scab-resistant cultivars are not widely planted. There are concerns with their small size and their tendency to ripen unevenly and develop a brownish interior after prolonged storage. Most of the scab resistant cultivars are susceptible to other major diseases. Failure to control sooty blotch and fly speck on Liberty apples resulted in a significant reduction in economic returns for the fruit.[717] Liberty was considered resistant to powdery mildew when released but is now highly susceptible.

Cedar apple rust is prevalent wherever the alternate host, eastern red cedar, occurs in proximity to apple trees. It can cause serious loss of crop and reduction in fruit grade as well as almost total defoliation.[703] The disease cannot spread from cedar to apple nor from apple to apple but must alternate between the two hosts. The fungus spends almost two years of its life on the cedar trees. Small brown galls appear during the summer of the first year but do not mature until the following spring. After a few warm spring rains, the galls increase in size and extrude long, thin, orange, gelatinous tendrils. A single cedar gall can produce over 100 billion spores.[726] After each rain, the tendrils push out farther and spores are discharged forcibly into the air. Viable spores have been collected by airplane traps several miles from red cedar trees at altitudes up to one-half mile.[725] The spores may remain viable for days even weeks. Swelling and drying of the tendrils may occur 8 to 10 times during the spring until the supply of spores is exhausted.[703] Spores that land on apple tissue can germinate and infect the host if a film of water is present. The symptoms on leaves consist of yellow to bright orange
lesions, which impede normal leaf function and may result in defoliation. Fruit lesions are superficial and cause light brown necrosis only 1 to 5 mm into the flesh.[703]

Heavily infected fruit are small, deformed, spotted with lesions, and of no market value.[727]

Small orange/brown pustules develop within the lesions, fracture and release sticky watery orange drops containing spores. Insects are attracted to this exudate and carry spores from one lesion to another. The spores are sexual and cross fertilize. After fertilization, the fungus produces a new set of spores, which are released during dry weather in late summer. Those that land on cedar trees may germinate and establish infections. During the second year after infection, mature galls are formed, thus completing the disease cycle.

In 1914, cedar apple rust was so severe that it destroyed 25% of the apples in the Winchester, Virginia growing region.[728] In 1927, 50% of the McIntosh apples were infected in some New York orchards.[729] Attempts were made to control cedar apple rust by eradicating nearby cedar trees. In 1891 in Vermont, all the cedar trees within a mile of a highly infected apple orchard were uprooted. In 1892, not a single infected leaf was found in the orchard.[731] The removal of cedars became the commonly recommended control practice. Eight states, including Virginia, Arkansas, Kansas, Nebraska, West Virginia, Pennsylvania, New York, and Missouri passed laws empowering authorities to remove cedar trees.[725] At first, in Virginia, during 1912 and 1914, good results were obtained by cutting the cedars within a half mile of orchards. Later, it was found necessary to cut down cedar trees within four miles of orchards.[728] The measure proved impractical in areas where cedar trees were abundant and apple orchards were scattered.[727] In the east many orchards were in valleys fringed by cedar bearing hills on which it was impractical to exterminate the cedar trees. The aesthetic value of the cedars in some estates and parks outweighed the value of nearby orchards.[725]

Early efforts to control cedar apple rust by spraying fungicides were futile.[728] Sulfur provided about 50% control.[730] Experiments with ferbam in the 1940s demonstrated complete control of cedar apple rust.[730] Ferbam sprays targeted at apple scab provided control of rust as well.

Quince rust is an important disease of apple in eastern states. It infects fruit, but not leaves, of apple trees. The brown spongy quince rust lesions extend to the apple core. In the fall, spores infect cedar trees giving rise to spindle-shaped or cylindrical galls from which spore horns emerge under wet conditions in the following spring. The galls are perennial and may produce spores for twenty years. Quince rust is economically important primarily when an extended wetting period (more than 48 hours) with a mean temperature above 10°C occurs between the tight cluster and late pink bud stages. Under these conditions, economic losses may occur throughout relatively large geographic areas.[703] Fungicides sprayed for cedar apple rust are also effective for quince rust.

Black pox of apple occurs primarily in the Southeastern U.S. (from New Jersey west to
Kansas and south to Texas and Georgia) on the cultivars Rome Beauty, Grimes Golden, Delicious, York Imperial, and Golden Delicious. On apple, fruit lesions are smooth, black, circular, slightly sunken spots. Severely affected leaves may abscise 2 to 3 weeks after infection. Lesions on twigs are well-defined, conical, shiny black swellings on the bark. Twigs with smooth bark remain susceptible for several years and the number of lesions increases from year to year. Heavily infected twigs and young branches grow poorly, defoliate prematurely, and eventually die. The incubation period is 3 to 6 months on fruit and 3 to 10 months on bark. Infections may remain latent on bark from midsummer until the following spring. Black pox of apple is normally controlled by the fungicide spray program used to control apple scab and summer diseases.[703]

Bull’s-eye rot is an important disease of apples in the Pacific Northwest. Bull’s-eye lesions are circular, flat to slightly sunken, and most often brown with a lighter brown or tan center. Rotted tissue is firm and does not readily separate from healthy tissue. Fruit can become infected anytime between petal fall and harvest. Fruit susceptibility increases as the growing season progresses. Bull’s-eye rot is more severe in years with frequent rains during harvest. Symptoms of fruit infections do not appear in the field and develop only after about five months in storage.[703] Infected fruit left on the orchard floor contribute to the buildup of inoculum and survival of the fungus. A preharvest fungicide spray protects fruit during the month before harvest.[703]

Brooks fruit spot is a minor disease that occurs throughout the Northeastern and Mid-Atlantic apple growing regions of the U.S. The disease first appears as irregular, slightly sunken dark green lesions on the surface of immature apple fruit. As the fruit mature, the lesions turn dark red or purple on red skin and dark green on green or yellow skin. Severe infection can result in pitted and cracked fruit.[703] Brooks fruit spot is usually controlled adequately with fungicides applied during the early cover-spray period. Wet growing seasons in northern Virginia in 1978 and 1979 promoted a dramatic increase in Brooks fruit spot incidence on unsprayed trees. Trees without fungicide treatment had 87% fruit infected while treated trees had 1% to 6% fruit infection rates.[296]

Blossom-end rot is also known as dry eye rot. The first symptom is a red discoloration that appears in late July on the calyx end of the fruit. Initially, the tissues beneath the discoloration are soft and water-soaked but the rot soon stops expanding and dries out. The affected area becomes sunken. The epidermis over the infected area turns dark brown, appears stretched and frequently separates from the surrounding healthy skin.[703] Affected fruit often drop prematurely. If harvested, about 50% of the affected fruit decay from gray mold in storage.[703] Field experiments have shown that blossom-end rot is caused by Botrytis cinera. Infection occurs during bloom, but the pathogen remains quiescent until fruit begin to mature. Sulfur has very little, if any, effect on reducing blossom end rot.[292] Experiments with ferbam showed a reduction of the percentage of fruit infected from 5% to less than 0.5% with treatment.[292]

Bitter rot was feared before the development of effective fungicides because entire crops could be lost in a few weeks during periods of warm, wet weather.[703] In 1870 in Illinois, an orchard was reported to have lost all of its apples to bitter rot.[704] Losses of
96% have been recorded in Arkansas orchards. Bitter rot is a major disease of apples in
the Southeastern U.S. from mid-June into September if the weather is hot and moist. The causal organism has a short incubation period and sporulates profusely on infected fruit. As a result, bitter rot epidemics can develop rapidly. The fungus survives from one season to the next on the tree in mummified apples and in dead wood. Spores are released during rain throughout most of the growing season. The presence of the disease is first indicated by very small light brown spots beneath the skin. The spots are watery and they become sunken because of the breaking down of the underlying tissue. The epidermis ruptures and masses of spores begin to appear in concentric rings on the surface of the lesions. Lesions extend in the shape of a cone toward the core. In cross-section, the lesions appear V-shaped. As the fungus penetrates more of the apple tissue, the area of invaded tissue becomes enlarged until the entire apple is rotted. Following the introduction of Bordeaux mixture in 1906 for bitter rot control, the disease became much less important. Growers switched to synthetic chemicals for bitter rot control in the 1940s to avoid the plant injury that was commonly associated with the use of Bordeaux mixture. No commercial apple cultivar is sufficiently resistant to bitter rot that sprays are not required. USDA estimates that 14% of eastern apple acres are infested with the bitter rot fungus and that apple yield loss would be 90% without fungicide use.

White rot is also referred to as Botryosphaeria rot or Bot Rot. In the U.S., it is most severe in the southeast where fruit losses of 50% have been reported. Lesions begin as small, often circular, slightly sunken, brown to tan spots, which may be surrounded by a halo. As the lesions expand in diameter, the rotted area extends in a cylindrical manner toward the core. In more advanced stages, the core becomes rotted and the rot extends to affect the entire fruit. Rotted fruit are soft, watery and light-colored, thus the common name: white rot. The fungus survives from one season to the next in dead bark and mummified fruit. Spores can be produced from dead wood for at least six years. Spores are produced throughout the growing season. Fruit does not become infected until the sugar content reaches 10.5%. Growers are advised not to spray for white rot until the sugar content approaches susceptible levels. USDA has estimated that 20% of eastern apple orchards are infected with the white rot fungus and that yield loss would be 65% without fungicide use.

In the U.S., black rot is most severe in the Southeast, but it occurs throughout Eastern apple growing regions. Lesions from the disease occur through fruit rot, limb cankers, and defoliation from leaf spot (known as frogeye leaf spot) which weakens trees. Black rot was a major problem in 1912 in the Southeast and resulted in 25-50% fruit loss. Fruit infection can occur early in the season. As the rot progresses, lesions may turn dark and almost black. Concentric rings may form on mature rotted fruit. The rot is usually firm, and may encompass one-half to two-thirds of the apple. The fungus survives from one season to the next in twig cankers and mummified apples. The amount and duration of rainfall are the main factors influencing spore release. Research determined that a one inch dead twig may furnish two million spores. Measurements of blighted limbs and twigs from one tree randomly selected showed 2,440 inches of dead wood, estimated to yield 4.8 billion spores during one season.
Prior to the development of ferbam in the 1940s, there were no spray materials effective on black rot. Research in the 1950s with synthetic fungicides determined that the incidence of black rot could be reduced to 1%.[709]

Sooty blotch and fly speck are two of the most common diseases of apple worldwide. The two diseases have been mentioned in the horticultural literature since the early 1800s but it was not until 1920 that it was determined that they are caused by two distinct pathogens.[703] Sooty blotch appears as dark-brown to olive green smudges on the surface of apples. Fly speck appears as well defined, slightly raised, small dark spots on the surface. The specks commonly occur in groups, seldom singly.[710] Sooty blotch and fly speck commonly occur on the same fruit, but the colonies are mutually exclusive.[703] Sooty blotch spot on apples forms a thin fungal crust. The individual fly speck spots show no connection one with another, which is visible to the unaided eye. Microscopic examination reveals the individual specks of one group to be connected by strands, however.[710] When the spores land on the waxy apple surface, they germinate and initiate growth. Both fungi utilize the waxy cuticle and penetrate only the epidermal layer spreading through the epidermal cells immediately beneath the cuticle.[710][711] Because the fungi causing sooty blotch and flyspeck grow superficially on the surface of the apple, losses are incurred primarily through lowered fruit quality. In the Southeastern U.S. virtually all of the apple crop would be affected each year if fungicides were not used.[703] USDA estimates that the two fungi infect 70% of eastern apple orchards and that losses would be 42% without fungicide use.[13][15] Research has shown that fungicide use reduces the percent of fruit showing sooty blotch from 67% to 1%.[712]

Alternaria blotch is a serious disease of apple in Japan, China, and Korea and was recently found in the United States in North Carolina. Lesions first appear on leaves in late spring. By midsummer up to 50% defoliation can occur in unsprayed plots.[703] Fruit infections are uncommon. The fungus overwinters in dead leaves on the ground and in dormant buds. Red Delicious apples are particularly susceptible to alternaria blotch.

Phytophthora species can attack the roots and trunks of apple trees and are among the most common causes of collar, crown and root rots of apple trees. Collar rot is a disease affecting bark tissues of the lower trunk at or above the soil line. Crown rot is a disease of the rootstock portion of the tree affecting tissues where the roots join the stem. Crown rot has become increasingly important since the early 1960s, probably because of the increased use of susceptible clonal rootstocks. Affected trees have sparse foliage and fruit tend to be small. Infected trees decline progressively over several years and eventually die. Tissues under the bark become dark brown as they decay.

Powdery mildew occurs wherever apples are grown. USDA estimates that 50% of the apple acreage in Western states and 40% of the apple acres in Eastern states are infected with the fungus which, if not controlled, would reduce apple yield by 65%.[13][15] The fungus overwinters on apple trees in dormant buds infected during the previous growing season. Spores are released from unfolding leaves of the infected buds, infecting young leaves, blossoms and fruit, which in turn provide inoculum for secondary infection
cycles.[703] Wind carries the spores great distances. Infections along the leaves often result in leaf curling. Severely infected leaves are narrow and folded. The fungus spreads until it covers the whole leaf surface and all the leaves in a cluster. The fungus grows down twigs, which it covers in a gray felt. When apples are infected during bloom, their growth is stunted, and their surface covered with a network pattern of cork cells (russet). When the spores germinate, the germ tube penetrates the cuticle and epidermal cells by enzymatic action. The fungus grows on the surface but it sends long suckers into the apple flesh to withdraw nutrients. Economic loss occurs in the form of aborted blossoms, reduced fruit finish quality, reduced vigor and yield of bearing trees.[713] Research has shown that the use of fungicides can reduce the percent of infected leaf area from 72% to 1% and raise yield by 50%.[714][715]

Fire blight is the most devastating bacterial disease affecting apples in the U.S. and around the world.[703] Fire blight was first described in New York in the late 1700s, and moved west with settlers, becoming established throughout North American apple production areas by the early 1900s. While fire blight has always been a concern in Eastern apple production, severe outbreaks in the West in recent years have caused growers there to adopt more consistent and vigorous monitoring and management programs as well.[444] In Washington state, severe outbreaks occurred in 5% of Washington’s apple orchards in 1993, 1997 and 1998, the latter two causing an estimated $68 million loss in trees and production.[486] In 2000, southwest Michigan was hit by a fire blight outbreak, the severity of which had never been seen before, leaving a deep and long term impact on the state’s apple industry. An estimated 400,000 trees on approximately 2,000 acres were lost, representing an investment of over $99 million. Regional apple yields were reduced by 35% with localized losses of 100% in some orchards.[487] Factors contributing to the severity of the 2000 outbreak include perfect environmental conditions for fire blight and the high proportion of acreage planted to varieties with little fire blight tolerance.

Fire blight is caused by the bacterial organism *Erwinia amylovora*, which grows readily and utilizes sugars and acids as food sources. Fire blight bacteria are microscopic: 25,000 laid side by side would not measure more than an inch. The disease can affect all parts of the tree, including blossoms and fruit, twigs and leaves, trunk and rootstock.[703] An infected tree may die within a few months of infection, or may survive with reduced yield for a few years, or until the grower pulls it out.

Fire blight was so named because the plant parts it infects look as though they have been burned, with infected tissue becoming progressively darker until it turns black. Blighted blossoms and young fruit remain attached to branches but look scorched and wilted, and blighted young shoots wilt into a characteristic shepherd’s crook shape. Within a few days, infections can move 6 to 12 inches or more into the shoot. The bacteria may live for long periods as a resident in or on apparently healthy apple tissue.

Each bacterial cell is completely independent and multiplies by dividing at a phenomenal rate, reaching 10 billion in 72 hours. As the bacterial cells multiply, they advance en masse through the tissue, giving rise to typical symptoms.[703] In addition to the
scorched appearance, tissue infected with fire blight will exude droplets of sticky ooze, particularly under humid conditions, that contain fresh inoculum. Favorable conditions lead to rapid multiplication of the bacteria, so rapid that the bacteria are forced through the diseased plant tissue, forming drops on its surface. The ooze may be clear, milky or reddish brown. Infected fruits may exude copious amounts of ooze.

Fire blight produces lesions on infected wood. When the bacteria enters an inactive, overwintering phase and lesion expansion slows, lesion margins become sunken and cracked, forming a canker. The bacteria overwinter in the canker edges, and ooze out again in the spring. Primary infection from this fresh, inoculum-filled ooze is spread to other plant tissue and other hosts by insects, wind and rain. During a fire blight epidemic, many insect species are attracted to the bacterial ooze on infected trees and help spread the pathogen. Insects are very effective inoculating agents, carrying up to 100,000 cells of the bacteria per insect. The bacteria also produce an aerosol that floats through the air like a fog and infects blossoms of nearby trees. New fire blight infections begin through existing openings in plant tissue. In the early spring, primary infections start in blossoms. In the blossoms the bacteria multiply rapidly and produce toxins, which penetrate cells and kill them. Sugars in invaded plant cells are used by the bacteria for growth and the process is continued until the whole blossom is killed and the bacteria move on.

Copper sprays have been used in commercial orchards for fire blight control since the 1930’s. Copper applied pre-bloom when green shoots are emerging may reduce inoculum on leaves, although insufficient efficacy against fire blight and potential to russet fruit currently limits its usefulness. Little progress was made in the control of fire blight until the 1950’s when antibiotics first developed for human treatment were used as a means of control. Use of streptomycin and oxytetracycline showed very high efficacy as compared to copper products. Streptomycin has been the effective treatment of choice since, applied two or three times during bloom or following rain and hail storms. Organic apple growers are allowed the use of streptomycin and copper for fire blight control.

Organic apple growers find it necessary to spray fungicides to control diseases. Organic apple growers are permitted the use of approved disease control chemicals including sulfur, lime sulfur, copper and antibiotics (streptomycin and oxytetracycline). Use of these organically-approved sprays can control the major diseases of apples but many and frequent sprays are required. A recent production budget for organic apple growers in the Northeastern U.S. estimated that ten gallons of lime sulfur and 12 pounds of wettable sulfur are used.

A recent report from Michigan State University concluded that without fungicide use: “The entire crop would be unacceptable for commercial fresh and processing markets because of almost complete severe disease damage and misshapen fruit. Within a few years, most apple acres would be removed or abandoned, if no fungicides were permitted.”
6.3 Artichokes

California produces 100% of all commercially grown artichokes in the U.S. 84% of the artichoke acreage in California is located near Monterey Bay where the cool, moist coastal climate is ideal for artichoke production.

Perennial artichokes are planted from root sections of mature plants. Artichokes are harvested for 5 to 10 seasons before replanting.[77] After full growth, the artichoke plant spreads to about 6 feet in diameter and about 3 to 4 feet high. It has long arching leaves that give the plant a fern-like appearance. Each cropping cycle is initiated by cutting off plants 2 to 3 inches below the soil surface to stimulate the development of new shoots. The timing of the cutting is staggered throughout the year, which results in continuous production in the region.[78] The vegetable that is eaten is really the plant’s immature flower bud.

In 1984, a few artichoke fields were reported to be infected with powdery mildew for the first time. The disease reappeared in the following years, each year being more severe and widespread than the previous, accompanied by a noticeable decline in fall production of artichokes.[79] It has been estimated that artichoke production was reduced by 30% in 1986 due to powdery mildew.[79] (Figure 3)

The fungus colonizes the undersides of older leaves. Spores are produced singly or in very short chains.[81] Spores are transmitted from field to field through the air. Severely infected leaves will turn yellow, then brown. The entire leaf then collapses and dries up prematurely, thus reducing the plant’s photosynthetic area. The impact of the disease is manifested in smaller buds, poor quality, and delayed production.[79] The powdery mildew fungus does not grow on the buds.

The State of California has estimated that, without control, powdery mildew would lower artichoke production by 35%.[79]

Currently, there are no registered fungicides that provide effective control of the disease in the coastal growing conditions of California. Sulfur is registered but research has shown that it is ineffective in controlling the disease. For sulfur to be effective, it is essential that the ambient air temperature be warm to facilitate its volatilization. The cool and humid climate of the coastal region results in the ineffectiveness of sulfur to control powdery mildew.[79] In addition, the thick artichoke canopy does not allow thorough coverage of the lower leaves of the plants with sulfur.

Every year since 1987, The State of California has requested that EPA grant emergency registrations for a fungicide to control powdery mildew in artichokes. EPA granted the request for the use of triadimefon for the years 1987-1997; however, the request for its use in 1998 was denied. For the years 1998-2003 EPA granted emergency registration requests for the use of myclobutanil to control powdery mildew on artichokes.
Research has shown that myclobutanil is highly effective in preventing yield losses to powdery mildew, essentially reducing losses to zero.[79] Myclobutanil is systemic in the plant and moves to the infected leaves after application.[79]

Research has shown that, in comparison to myclobutanil treatments, artichoke yields were 37% lowered without any treatment, while sulfur treatments resulted in a yield reduction of 25%.[80]

There is an organic artichoke producer in Santa Cruz County, but no information is available on the farm’s powdery mildew situation. This production may be in an area where powdery mildew is not a problem. A 20-acre organic artichoke farm began producing artichokes in Castroville late in 2002. No information is available regarding the farm’s plans for powdery mildew management. It may be that the grower is simply incurring reduced yield with no treatment but is gaining a premium price for the organic artichokes.

6.4 Asparagus

Asparagus is a perennial crop that should have a productive life of 15 or more years under ideal conditions. Asparagus spears grow upward through the soil from underground crowns. The spears are cut by hand every 2-5 days. After two to three months, spear harvest ceases and the spears are allowed to grow into ferns with a thick canopy of feathery leaves. During the fern stage, the plant produces and stores energy for the following year’s crop. In the spring, asparagus fields, including weeds and ferns, are cleared by mowing, tillage, or herbicide application to make room for new spears.

Asparagus rust was first reported in the United States in 1896, occurring in New Jersey, Massachusetts, Delaware, and New York.[138] Each season between 1896 and 1902 it progressed farther south and west until it reached California in 1902.

The severity of the disease during the first few epidemics was described as follows: “After the first few years of this epidemic, most of the older beds in the Atlantic states were entirely destroyed, the canning industry was practically abandoned, prices became about double what they had previously been, new varieties came into use and an entire readjustment of the asparagus industry took place.”[139]

Research with protective fungicides to control rust began in 1898 with Bordeaux experiments.[138] Several copper fungicides were tested but proved ineffective.[632] Sulfur dust was employed with some success.[140]

Growers in California applied over 100 tons of sulfur to asparagus fields in 1904 in an attempt to control rust.[140] An early recommended practice for asparagus rust control was to burn the ferns as soon as they became brown and lifeless which prevents any spores that may be there from overwintering and germinating the following spring.[138] Most growers abandoned the practice of burning their fields to conserve the humus layer.
Furthermore, burning is not effective in destroying all the inoculum.[146]

A major asparagus breeding project was established in 1906 and eventually two rust-tolerant varieties were released: Mary Washington and Martha Washington. However, over time the varieties no longer provided commercially acceptable rust resistance. By 1940 field investigations determined 100% rust infections were not uncommon even though the Washington varieties were grown.[141] It was not known if more aggressive strains of the pathogen had been selected or if the host had been unintentionally reselected for susceptibility.[160]

Development of the rust tolerant varieties had been considered a solution of the rust problem; consequently, little attention was paid to the control of rust by other means since the introduction of the Washington varieties.[146] The losses from rust in years of severe infection gave impetus to the search for effective fungicides.[141] Sulfur was found to not produce consistently good results and was not widely adopted by growers.[147]

Rust symptoms do not appear on the spears that are cut for market but are confined to the ferns produced after the cutting season. Good shoot growth among the ferns is essential to provide energy reserves to the crown for the next year’s crop. By interfering with carbohydrate synthesis, the rust lowers the amount of root storage metabolites and reduces plant vigor resulting in less yield the following spring.[142] Plant stress resulting from severe infection can cause death.[143]

Rust spores are produced in such great numbers that when they are disseminated by wind, they can cover the plants and ground with a reddish dust.[88] The spores germinate in the presence of moisture. Germinating spores penetrate asparagus fern tissue and a layer of spores is produced beneath the plants epidermis. This epidermal layer is ruptured by the pressure of the maturing spores which are exposed to air currents for dissemination.[141] Rust causes individual fern needles to fall.

The effects of rust are cumulative. Yield reductions are greater after two years than after one year. In a study conducted in Washington, the yields of four cultivars were evaluated following a single year of infection and following two subsequent years of infection from rust.[142] After one year of infection, total spear weight was reduced from 2 to 23% and the number of spears were reduced from 5 to 26%. After two years, total spear weight was reduced from 11 to 54% and the number of spears were reduced up to 46%.[142]

Research in the 1950s with zineb, reduced mean rust infection by 85%.[147] The use of zineb in a protective spray program in a commercial asparagus field in 1957 demonstrated only about a 10% infection in sprayed plots while the untreated asparagus was so severely infected that 50% of the plants had lost their needles.[147] Research with mancozeb resulted in a 97% reduction in rust pustules per plant while the use of sulfur resulted in a 57% reduction.[145]

Prior to 1990, Michigan asparagus growers relied on EBDC fungicides (including
mancozeb) to control rust. In December of 1989, EPA proposed to cancel 45 of the 55 registered uses of the EBDCs. While the use on asparagus was retained during this time, certain Michigan processors dictated that they would only accept asparagus that had not been treated with EBDCs. In 1992 EPA concluded its EBDC review and asparagus was retained in the final decision. Despite EPA's decision certain Michigan processors (Campbell Soup Company) continue their policy of not accepting asparagus treated with EBDCs. Without the use of EBDCs, Michigan growers were left with no registered alternative for the control of rust on asparagus. This situation has resulted in Section 18 exemption requests for the 1990-2003 seasons. EPA granted emergency exemptions for the use of triadimefon (1990-1997), myclobutanil (1998-2000), and tebuconazole (2001-2003).

In Washington, mancozeb is recommended for rust control if more than 1% of the foliage is infected by July 31, if more than 5% of the foliage is infected by September 10, or if 10% of the foliage is infected by September 30.[160]

The incidence of rust has been getting more severe in the last several years in California. Increased winter and spring rains may be contributing to the increased incidence of asparagus rust. The length of the fern growing season has been increasing in California as more growers are harvesting earlier.

Purple spot on asparagus was first reported in the U.S. in 1981. The major damage from purple spot is on fern growth. Damage to the fern results in defoliation of the needles that reduces the flow of carbohydrates to the roots and lowers next year's yield.[9] The disease can cause yield reductions of up to 52%. The disease causes small purple spots to form on emerging spears causing them to be rejected by processing companies. During severe epidemics, purple spot of spears affects quality and may result in rejection of up to 90% of the spears.[50] In spring, spores produced from last year's infected plants are spread by wind and water to newly developing plants.

Prior to the mid 1970s asparagus plantings in Michigan were subjected to annual bed renewal involving extensive tillage. Since that time the industry has converted completely to a no till system involving the use of herbicides and surface mulching of the asparagus residue.[88] This system has been effective in reducing wind erosion, and reducing damage to the underground crowns. Prior to the adoption of the no tillage system, purple spot was only an occasional problem on asparagus in Michigan. However, purple spot has become a regular and persistent disease probably due to the overwintering of spores on crop residue.[88] One attempt to burn the standing asparagus fern after it had dried down resulted in a near environmental disaster, so that option was abandoned.[88]

EBDC fungicides are effective in the control of purple spot. Since some processors reject the use of the EBDCs, Michigan has annually obtained an exemption for the use of chlorothalonil to control purple spot on the ferns since 1990. Growers apply the fungicide after the last spears are harvested. Research has shown that chlorothalonil reduces the average number of purple spot lesions/fern by 99%, with an associated yield
The increase in yield as a result of controlling purple spot translates into an additional net return of $200-400 per acre.

Crown and spear rot caused by *Phytophthora* occurs in all production areas of California. Symptoms include soft water-soaked lesions on shoots at, slightly above, or slightly below the soil surface. These lesions elongate rapidly and turn brown. Infected young storage roots become water-soaked and rotten. Crowns have yellow-orange tissue that appears slimy. As the lesions expand, they collapse and shrivel. This flattens the affected side of the spear and the spear bends. The fungus resides in the soil at or below the soil surface and is most damaging during wet periods. Disease outbreaks are sporadic due to the need for wet soil for spore germination and infection.

Yield losses in asparagus caused by *Phytophthora* were reported in California as early as 1938 when growers reported that a condition known as “slime” caused postharvest losses of 20-30%. It was not until the 1980s that the disease was extensively studied. Research demonstrated that soil treatments of metalaxyl gave almost complete control of spear rot and increased asparagus yield by 38% to 118%. Researchers concluded that asparagus yields had been lowered for many years by *Phytophthora* especially during wet harvest seasons.

Myclobutanil and mancozeb are used in California for control of rust while mefenoxam is targeted at crown/spear rot. In Washington mancozeb is targeted at rust while in Michigan mancozeb is targeted at purple spot and rust. Chlorothalonil and tebuconazole are used under emergency exemptions in Michigan for control of purple spot and rust respectively.

It has been estimated that in California, on asparagus acres treated for crown rot, asparagus yields would decline by up to 50% without fungicide treatment. For asparagus rust and purple spot it has been estimated that U.S. asparagus yields in the second year following cessation of fungicide treatment yields would decline up to 50%. A USDA assessment estimated that Michigan asparagus yield would decline by 30% without fungicide treatment for rust and purple spot.

### 6.5 Bananas

Hawaii is the primary state in the U.S. where bananas are grown. The average yield is 15,000 pounds/acre. Bananas are harvested green and unripe. Most bananas grown in Hawaii are marketed within the state. Bananas require about 80-100 inches of rainfall per year. Approximately 600-800 plants are grown per acre.

It is difficult to breed new, improved varieties because cultivated bananas are all female, do not have seeds, and are reproduced vegetatively. Banana “keiki,” which are the sucker side shoots of a mature banana plant, are cut away from the mother plant. The ‘keiki’ are then planted, producing new banana trees. Planting occurs year round. Banana bunches are ready to harvest from 12 to 15 months after initial planting. Each...
banana plant produces only one bunch of fruit.[123] On a banana plantation, plants can be seen at all stages of vegetative growth and fruit maturity year-round. Bananas can be harvested any day of the year.

There are two Sigatoka diseases, Yellow Sigatoka and Black Sigatoka. Yellow Sigatoka takes its name from the Sigatoka valley in Fiji where the disease was first recognized in 1912.[150] During the next 40 years the disease spread to all banana-growing countries. Yellow Sigatoka appeared in Central America in 1934 and in less than two years destroyed more than 22,000 acres of bananas in Honduras and Surinam.[182] By 1936, fungicide spray programs utilizing Bordeaux mixture were developed to control the disease. The disease spores can be blown by wind currents for more than 1,000 miles. The disease was first found in Hawaii in 1958.[182]

Black Sigatoka, also known as black leaf streak, was first recorded in 1964, also in Fiji. It subsequently spread throughout the world. Black Sigatoka tends to displace yellow Sigatoka. Yellow Sigatoka cannot be found in most locations in which black Sigatoka occurs.[150]

Infection occurs on the youngest leaves of the plant during and immediately after unfurling. Spores germinate on the leaf surface and the fungus undergoes a period of growth for up to six days before it penetrates the leaves.[150] After penetrating the leaf, the fungus colonizes adjacent cells for approximately 7 days without any evidence of disruption of the cells.[136] The first symptoms, small flecks, appear on the underside of leaves 10-15 days after infection. The flecks elongate into narrow streaks which enlarge and darken to give the characteristic black streaking of leaves.[150] Large areas of the leaf may become blackened and water-soaked. The affected areas collapse and may dry out in 8-10 hours. In extreme cases, all leaves can be destroyed before the bunch is mature, and the bunch may fall from the plant.[150]

Black Sigatoka disease affects the leaves of banana plants, destroying the foliage very rapidly. Both growth and yield are affected because of the reduction in the photosynthetic area.[136] The size of bunches and individual fingers are reduced. The greatest yield loss probably occurs as a result of the premature ripening of the fruit.[136] The fungus produces a complex of toxins that appear to cause premature ripening of the fruit. Premature fruit cannot be sold and is simply dumped.

Spore release requires the presence of a film of water. Spores are forcibly ejected through the leaf and dispersed by air currents.[136] Some indication of the number of spores produced in heavily diseased areas was obtained in Hawaii. From 8,000 to 33,000 spores per cubic meter of air per 24 hours were obtained in a spore trap.[183]

Under Hawaiian conditions, uncontrolled black Sigatoka disease would result in an average yield loss of 30%.[13]

Reduction of inoculum level is one of the most important cultural practice used to help manage the disease. Sporulating tissue is removed from the leaf. This tissue decomposes
once it is removed.[136] Usually, if leaves have greater than 50% diseased leaf area, they are removed. In Hawaii, banana fields are examined on a weekly basis for the purpose of removing diseased leaves.

Chemical control of black Sigatoka is achieved with the alternation of protectant (mancozeb) and systemic fungicides (azoxystrobin, fenbuconazole, tebuconazole). The use of mixtures of mancozeb and the systemic fungicides is part of a strategy to manage fungicide resistance.[136] Systemic fungicides penetrate the leaf cuticle and inhibit the pathogen inside the leaf. Protectant fungicides have to be deposited on the leaf surface prior to infection where they act by inhibiting spore germination and penetration of the pathogen into the leaves.[136]

Not all banana acres in Hawaii are sprayed with fungicides to control Sigatoka. On Oahu, bananas are grown in areas with tradewind conditions and Sigatoka does not become established under these climatic conditions.[182] Areas exposed to tradewinds remain free of the disease in spite of inoculum presence and rains because the banana leaves do not remain wet for long periods of time.[182] On the other hand, in protected high rainfall valleys not open to tradewinds, banana leaves may remain continuously wet. It is under these conditions that the disease becomes epidemic.

Fungicides are typically applied in the summer at one to two week intervals. They are typically not applied in dry months.[13]

### 6.6 Barley

Barley, a small grain cereal, is principally used in the United States for malting and brewing. Malting barley provides alcohol for beer. Barley is also sometimes used as a feed grain.

The major barely producing states are North Dakota, Montana, Minnesota, and Idaho, which account for 70% of U.S. production

Fusarium head blight (FHB), also called scab, is a fungal disease that affects barley and other small grains and grasses. Outbreaks tend to occur when high moisture conditions coincide with the flowering and grain-fill stages of barley development. The fungus overwinters in crop residue left over following corn and small grain harvests. In spring and summer, spores are carried by wind or splashing water to newly developing barley heads. The infected barley spikelets become prematurely bleached in color, contrasting with the green of healthy developing tissue. Infected barley kernels are shrunked and discolored. The fungus produces pink spore masses on the barley heads. The disease has the ability to completely destroy a barley crop within a few weeks of harvest.[696]

Economic losses due to FHB occur in several ways. Production is decreased because of kernel shriveling, some kernels being so light that they are blown from the combine.[733] In addition, the fungus produces a mycotoxin that lowers the value of barley. If the
mycotoxin is consumed at high levels, it induces vomiting. Consequently, it is commonly known as vomitoxin. The chemical name for vomitoxin is deoxynivalenol or DON. The toxin is water soluble and heat stable and passes through the malting and brewing processes into beer. Its presence in beer may cause gushing of the beer when a can or bottle of beer containing the toxin is opened. Foamy beer that gushes from bottles and cans is not commercially desirable. Barley will not be purchased for malting unless vomitoxin content is 0.5ppm or less.[733] The FDA has set advisory levels for DON in food (1 ppm) and feed (5-10 ppm). Barley producers receive severe price discounts when DON is present.

Scab became so severe in the first half of the 1900s that it essentially eliminated barley production in the eastern and central corn belt of the U.S., where barley had been grown in rotation with corn.[733] Before the early 1990s, Fusarium head blight was rarely an economic problem in central and western barley production. However, scab epidemics have been occurring in Minnesota and North Dakota since 1993. These epidemics have been associated with seasons of above average rainfall and the widespread adoption of conservation tillage practices that leave abundant crop residues on the soil surface. Incidence and severity of head blight in North Dakota in 1993 were estimated to range from 5% to 80% with an estimated 56 million bushels lost.[696] Vomitoxin levels in barley from North Dakota in 1993 were as high as 44ppm. FDA tests found average DON levels of 4.8 ppm in North Dakota barley. Most barley producers had to sell their barley for feed at a huge price discount. Many producers in 1993 decided not to harvest their barley fields due to the presence of scab and the likely low price that they would receive (500,000 acres were not harvested). Between 1992 and 1997 average North Dakota barley yields dropped from 65 bushels an acre to 45 bushels an acre. The GAO estimated that from 1993 through 1997, North Dakota barley growers suffered revenue losses from scab and vomitoxin of $200 million.[734] Maltsters and brewers have reacted to the scab and vomitoxin damage by purchasing less barley from North Dakota and Minnesota farmers and more from Canada and other western states.[734]

Improvement in FHB control by fungicide application is one of the priorities of the “U.S. National Wheat and Barley Scab Initiative,” which is a $3 million annual federal program. Results of fungicide trials on barley in North Dakota in 2004, demonstrated that fungicides reduced the field severity of FHB from 72 to 85%, reduced DON from 49 to 69% and increased yield from 10 to 14%.

Fungicides are used to control three rust diseases of barley-leaf rust, stem rust and stripe rust. These diseases cause reddish brown spots or yellow pustules that erupt through the epidermis of the aboveground portions of the barley plant.[733] The rusts increase transpiration and respiration and decrease photosynthesis in the barley plant. Overall, they reduce plant vigor, seed filling and root growth. While barley rusts are caused by three highly specialized fungi, each pathogen occurs in numerous physiologic races and new races continually surface.[733] In North Dakota and Minnesota, stem rust and leaf rust have been a problem primarily on late-planted barley, which is more likely to be damaged because the grain is not yet filled at the time that the rust spores generally arrive from their overwintering sites in the southern plains.[736] Growers are advised to use a
systemic fungicide if conditions for epidemics prevail.

Stripe rust of barley is a recent arrival in the Americas. In South America, the disease was first reported in Columbia in 1975 and was probably introduced from Europe.[733] Barley stripe rust was first reported in Texas in 1991. By 1995, it could be found in every state of the western U.S. Considerable yield losses have occurred locally as most barley cultivars lack any significant resistance to stripe rust.[733] Research demonstrated that the rust pathogen population consists of at least 31 different races.[738] Barley stripe rust is widely established in Idaho, Washington, California, and Oregon and all barley growing areas in the Pacific Northwest are susceptible to infection.[737] In Oregon in 1996-97, barley yield losses of 25 to 50% were recorded.[739] In California barley production lost to stripe rust was 15 to 20% in 1998 and 25 to 30% in 1997.[740] Research demonstrated that fungicide application protected barley late into the season throughout all its vulnerable stages.[738] The state of Idaho estimated that without fungicide application, barley losses would be 40% while fungicide application would reduce the loss to 5%.[737]

Without fungicide use barley yields are expected to be 12% lower in North Dakota and Minnesota due to scab infections and 35% lower in the Pacific Northwest and California due to stripe rust

6.7 Blueberries

Blueberries are perennial crops that fall into three categories: highbush (grown mainly in Michigan, New Jersey, North Carolina, Oregon, and Washington), rabbiteye and highbush (Florida and Georgia) and lowbush (Maine). Highbush and rabbiteye plants can live 50 years although a 20 to 30 year life is typical.[268] Lowbush blueberries are also known as wild blueberries and are produced from native stands, many of which are more than 100 years old.

Florida and Georgia production is primarily for the fresh market while Maine’s production is used primarily for processing. Blueberry production in the other five states goes to both processing and fresh markets.

Four to seven fungicide applications are made per blueberry acre for control of nine diseases: botrytis blight, mummyberry, fusccoccum canker, alternaria leaf spot and fruit rot, phytophthora root rot, phomopsis twig blight and fruit rot, anthracnose fruit rot, pseudomonas blight, and septoria leaf spot.

The cause of botrytis blight is the common gray mold fungus, Botrytis cinerea. The fungus was first observed on blueberry in New Jersey in 1924. In 1930 botrytis blight appeared on blueberries in Maine. By 1950, Botrytis blight was considered the most important disease of blueberry in the Northwest.[102] In the Southeast the disease can cause severe losses in rabbiteye plantings. Botrytis blight is estimated to infect 95% of Northwestern blueberry fields and 40% of eastern fields.[13][15] Yield losses from uncontrolled Botrytis are estimated at 30-40%.[13][15] The fungus overwinters in
diseased fruit, stems and leaves. Production of spores reaches a peak during the period right after blueberry plants are blossoming. The fungus attacks blossoms, especially when rainy weather persists through bloom. Masses of spores are produced on blighted blossoms. One week after infection, blossoms turn light brown and develop a grayish-brown mold. In most cases infections initiated in blossoms results in ovary mortality, preventing fruit development.[102] Most fungicides applied to control other diseases during the blossom period also control Botrytis blight.[102]

The fungus that causes mummyberry overwinters in shriveled mummified blueberry fruit on the ground. In early spring, cup-shaped structures of the fungus grow on mummified berries. Each cup-shaped structure produces an average of 61,000 spores a day for 9 days.[270] Spores are forcibly ejected into the air and carried by the wind to young, developing twigs and flowers where they blight new blueberry plant growth. All affected tissues eventually fall off the plant.[102] The infections on young plant growth produce masses of spores which are spread by bees to blueberry flowers where they germinate and grow down into the ovary. In this manner, fungal spores travel through pollen tubes, traversing the same path as blueberry pollen. After infecting the ovary, fungal tissue colonizes the developing berry. No visible exterior symptoms appear until near harvest when infected berries turn a salmon or cream color, begin to dry (mummify), and drop to the ground.[269] The mummy retains a shape similar to normal fruit. The berry skin is sloughed off and the mummy is composed primarily of fungal tissue.[271] Yield losses in the field include a reduction in total fruit volume due to the loss of fruiting wood in the spring from twig and flower blight and from infected fruit that mummify and drop to the ground.[272] Uncontrolled mummyberry would lead to average yield losses of 20-50%.[13][15] The state of Maine recently estimated that uncontrolled mummyberry would reduce that state’s yield by 25%.[273] The state of Michigan reported that blueberry losses in the state could be 25-57% if mummyberry were uncontrolled.[275] Greater losses can occur after harvest when a fruit shipment is downgraded due to an excessive incidence of infected fruit. This is particularly important for fruit loads intended for use in processing, for which a tolerance of 0.5% disease incidence is established.[272] Fruit loads exceeding this tolerance are appraised at lower quality grades, resulting in severe economic penalties to producers.[274] Experiments have shown that fungicide applications reduce the incidence of mummyberries at harvest by 98-99% from a disease incidence of 21-24% to 0.4%.[276][278] Experiments showed that blueberry yields were increased by 34% through fungicide use that controlled mummyberry.[277] Experiments with a biological fungicide (Bacillus subtilis) were not successful in reducing mummyberry infections.[279] It was theorized that the biocontrol agent failed to establish sufficiently high populations inside the flowers to control the fungus.[279] Increased fungicide use in Georgia in the 1990s significantly reduced losses to mummyberry (Figure 13).

During the early 1960’s a canker-dieback disease was found widespread in Michigan’s blueberry fields.[280] The disease was identified as fusccocum canker and remains one of the main limiting factors of blueberry production on the upper peninsula of Michigan and causes serious annual disease problems on Michigan’s lower peninsula.[281] Disease severity ranges from wilting and death of one or two stems to wilting and death
of almost all stems and consequent death of the entire plant.[102] Captan applications provide preventive control of stem infections.[102] Research has shown that regular fungicide applications can reduce the number of cankers appearing on infected plants by 82-95%.[281] Uncontrolled fusicoccum canker is estimated to reduce blueberry yields by 30%.[15]

A leaf spot disease of blueberry caused by a species of *Alternaria* was first observed in North Carolina in 1973.[282] The fungus was also identified as the principal cause of fruit decay in harvested blueberries in North Carolina.[102] The fungus has been reported to cause fruit rot in most blueberry growing regions. Symptoms of the fruit infection develop when the fruit ripens. The calyx end is covered with a greenish fungal growth. After storage at room temperature for 10-20 hours, fruit may become leaky.[102] Because of its importance as a post-harvest fruit decay organism, any increase in the fungus on blueberry leaves in May can cause considerable damage to the quality of fruit when harvested in June (North Carolina).[282] Control of alternaria leaf spot and preharvest fruit rot is essential to the delivery of high quality fruit to the market.[102] A fungicidal spray program beginning at early bloom and continuing at two week intervals until harvest is usually effective.[102]

Phytophthora root rot was first described as a disease in blueberry in 1963. In 1967 the fungus was recovered from 40% of the blueberry plantings surveyed in southeastern North Carolina.[102] Current estimates are that the root rot fungus infects 80% of eastern blueberry acreage with the potential to reduce yields of 25%.[15] Symptoms include cessation of growth, defoliation, yellowing of leaves, and conspicuous reddening of leaves.[283] Many of the roots die back from the tips. Spores are attracted to small roots where they germinate and penetrate the root’s surface.

Phomopsis twig blight was one of the first diseases observed in cultivated blueberry fields, first noted in 1924.[102] Twig blight has increased tremendously in prevalence and severity in the southeastern blueberry-growing regions since and is estimated to infect 90% of eastern blueberry fields.[15] The fungus enters young developing fruit and succulent shoots and progresses toward the base, ultimately girdling the old branches and killing the part of the plant above the girdle. This results in the loss of fruit production.[284] Blueberry fruits that escape this initial infection are susceptible to rots caused by the fungus at harvest. Infected fruit are typically soft and often split open and leak juice. The infected fleshy tissue is reddish brown and mushy. Losses from twig blight infections can amount to three pints of berries per plant when susceptible plants are not sprayed with an effective fungicide.[102] Research has shown that fungicides can reduce yield loss due to phomopsis twig blight from 30% to 4%.[285]

The common name, ripe rot, is sometimes used instead of the name anthracnose because it is more descriptive of the disease on blueberry fruit. This fruit rot is a serious problem in the northern and southern blueberry-growing regions of the U.S. and is estimated to infect 30% of eastern blueberry acreage.[15] Anthracnose fruit rot occurs on blueberries as they mature and ripen at harvest.[102] Infected fruit remain symptomless until maturity. Upon maturity the blossom end of the ripe fruit softens and becomes slightly
sunken and masses of orange spores are exuded. Losses during storage sometimes approach 100% because the spores from a single diseased berry may rapidly infect an entire container of fruit.[102] Preharvest control of ripe rot on susceptible cultivars is best achieved with applications of an effective fungicide at intervals of 7 to 10 days.[102]

Septoria leaf spot is prevalent on most of the highbush and rabbiteye blueberry cultivars grown in the southeastern U.S.[102] Leaf lesions are small, circular, white to tan spots with purple borders. Stem lesions are typically sunken, with a gray or tan center and a reddish brown margin. The fungus overwinters in infected leaves on the ground and in stem lesions. Leaf spots appear on blueberry plants by early May and then increase rapidly from June until September. Leaf spots are most apparent after harvest and severe infection during this period can result in premature defoliation.[286] Early loss of leaves can negatively impact initiation of flower buds during the fall due to reduced carbohydrate supply which, in turn, can result in decreased fruit yields the following year.[286] Yield reductions occur even in the absence of defoliation if infected leaves contribute little to photosynthesis.[286] Recent research with fenbuconazole applied at harvest led to significant yield gains the year following application. These gains can be attributed to prevention of septoria leaf spot’s adverse affects on the photosynthetic abilities of the plant: the fungicide treated blueberry bushes averaged 45% greater yield.[287]

Copper is used in the Northwest during the dormant season to reduce populations of bacteria that cause pseudomonas blight. The bacteria overwinter in buds on the canes of raspberry plants. In early spring the bacteria function as an ice nucleator, raising the temperature at which water freezes within plant tissues. The bacteria subsequently invade the tissue damaged by freezing. The bacteria become systemic within the plant tissue and infection continues to spread as long as the weather remains cool and wet. As the temperature rises, the disease usually subsides until cooler weather appears in the fall.[197] Thin strands of glistening ooze are produced on the surface of infected tissues. New growth can be killed to ground level.[197]

The diseases attacking blueberry plants can reduce yield by 25-60% each if left uncontrolled.[15]

6.8 Cabbage

Several applications of fungicides, (chlorothalonil, mancozeb, maneb, azoxystrobin, copper) beginning in midseason, control downy mildew and *Alternaria*. Copper is also used for control of black rot. PCNB is targeted at clubroot. Mefenoxam is used for control of damping off. Fosetyl-Al is used for downy mildew and damping off control.

Downy mildew is a serious disease of cabbage in the U.S. As cabbage approaches maturity under cool weather conditions, downy mildew causes leaf spotting on the lower leaves as well as on the head. Soft rot bacteria may enter head lesions and cause damage in transit and storage.[144] Systemic invasion of the heads by the downy mildew fungus results in loss of grade at harvest and in dry rot in storage.[144] On cabbage heads the
pathogen may cause numerous sunken black spots, varying in size from minute dots to more than an inch in diameter. In Texas, downy mildew is estimated to infest 80% of the cabbage acres with the potential to reduce yields of 55%. Downy mildew occurs in most years on cabbage in Florida, but losses are minimal (2%) because growers effectively control the disease with chemicals. 

Losses to cabbage from Alternaria leaf spot occur from spotting of lower leaves and heads. The discoloration may cover part or all of the head and, although only superficial, can greatly diminish marketability of the crop. Leaf spots that vary in size from pinpoints to 2 to 3 inches in diameter are common on old lower leaves. Severe infections result in large dead areas at lesion sites. They begin as small circular yellow areas, which enlarge in concentric circles and take on a black sooty color. These spots are not economically important unless the spotting is severe enough to require heavy trimming when cabbage is placed in storage. The Alternaria spp. produce many large spores that can be blown, splashed, or carried by tools, humans or animals throughout fields. In Texas it is estimated that 65% of the state cabbage area is affected by Alternaria with a potential to reduce yields by 50% if not controlled.

Black rot is the most serious disease of cabbage worldwide. In the 1890s, black rot devastated the cabbage fields of southeastern Wisconsin. It attacks in temperate and subtropical zones where rainfall or heavy dews are plentiful and where average temperatures are between 60 and 70 degrees. Black rot is caused by a bacterium. The pathogen moves through the leaf into the water-conducting (vascular) system causing blackening and plugging of the veins. Once in the veins the bacteria multiply and spread. Later infections may become systemic, resulting in blackening of veins inside cabbage heads. Black rot is the most serious disease of cabbage in Florida and Georgia, while in Texas it is estimated that in a five-year period 15% of cabbage fields will be affected by black rot. Potential losses from black rot are 100% if fields are not treated when necessary. Losses from black rot are high because of the rapidity of spread when weather conditions favor pathogen growth and dissemination. Under favorable conditions, one plant can produce enough inoculum to cause an epidemic in a field. Soft rot bacteria enter black rot lesions, move into the heads and render the heads worthless. California’s dry summers are not conducive for black rot and it was of minor importance until the increased use of sprinkler irrigation.

The distinctive symptom of clubroot is an abnormal enlargement of roots. Severely distorted roots are unable to absorb minerals and water from the soil; consequently, the tops become stunted, yellowish, and wilted. Such plants usually form small heads. The fungus enters plants through fine hairs on young roots or through wounds in secondary roots or in the stem. After the root enlarges, the fungus slime mold in the plant is transformed into a mass of spores, which are released into the soil. The organism can survive in the soil for at least seven years. PCNB is the only chemical that is effective, non-phytotoxic, and economically feasible for use in large cabbage fields. Cabbage production losses due to clubroot can be as high as 50% in severely infected fields.
Pythium ultimum and Rhizoctonia solani cause damping off and seedling death that can result in up to 50% yield losses in severely infected fields.[581] These fungi are common and persistent in most soils. The fungus penetrates seedlings near the soil line causing water-soaked constriction of the stem which girdle and usually kill the plant. If plants survive the initial attack, the centers of the stems decay while the outer stalks provide enough support to keep them erect. The plants grow very slowly and usually do not develop to maturity.

Without fungicides, cabbage yields are projected to decline by 45-50% in eastern states (Florida, Georgia, New York), while in the dryer western states the decline is projected at 10-15% (California, Arizona).

6.9 Cantaloupes (included in Section 6.17 Cucumbers)

6.10 Carrots

Carrots are a biennial crop grown as an annual for its root. Carrots are sown at a rate of a million seeds per acre for fresh market varieties and at a lower rate for larger processing varieties. Higher densities of up to 1.2 million seeds per acre are often used for the ‘cut and peel’ market. Carrots grown for fresh market generally take 100-140 days to mature, whereas processing carrots require 150 days from seeding to harvest. Most carrots are mechanically harvested. The roots are undercut and then they are lifted out of the soil by grasping the leaves.

Several of the fungicides used on carrots are targeted at one primary disease: mefenoxam for cavity spot and sulfur for powdery mildew. The most-widely used fungicides (chlorothalonil, azoxystrobin, pyraclostrobin, copper, and iprodione) are used to control several foliar diseases of carrots (alternaria leaf blight, cercospora leaf blight, and bacterial leaf blight [copper]).

Leaves need to be protected from leaf blights during the majority of the growing season; therefore, numerous fungicide applications are required for control. Many large-scale carrot growers report that disease problems have increased in severity in recent years.[242] The close spacing needed to produce ‘cut and peel’ carrots has tended to increase disease pressure from foliar diseases such as leaf blights and powdery mildew.

Bacterial blight was first found in California in 1931 and has since been reported in many other states. Symptoms on leaves begin as small yellow spots that expand into brown water-soaked lesions surrounded by a yellow halo. The centers of the lesions become dry and brittle. On stalks, copious bacterial ooze flows downward.[83] The bacterium is commonly found as a contaminant of carrot seeds and is persistent in soil. The bacteria spread plant to plant by splashing rain and irrigation water. Under warm conditions, epidemics occur rapidly.[144] Applications of copper can slow the development of
bacterial leaf blight in the field, particularly if applications are initiated when plants are young.[83] Potential carrot yield loss to uncontrolled bacterial blight has been estimated at 20%.[237]

Alternaria leaf blight is the most common foliar disease of carrot. Under optimal conditions, severe foliar epidemics rapidly develop, leading to loss of foliage and reduced yields. Alternaria also indirectly reduces yields by interfering with mechanical carrot harvests. Leaves weakened by blight break off when gripped by a mechanical harvester and the roots are left behind in the ground.[144] Foliar symptoms appear as dark brown to black water-soaked lesions which expand as the infection develops. When 40% of the leaf area is affected, the entire carrot leaf yellows, collapses, and dies.[83] The pathogen is disseminated on or in seed and overwinters in soil borne debris from diseased carrot tissue and in weed hosts. The fungus sporulates abundantly on dead and dying seedlings. Spores are released and are spread by air turbulence to other plants and nearby fields.[83] In water on leaf surfaces, spores quickly germinate by forming one or more highly-branched germ tubes that grow until they eventually penetrate the leaves. The fungus produces the toxin zinniol, which degrades cell membranes.[630] Copper was the first fungicide used to control Alternaria. Early experiments with synthetic fungicides showed an increase in carrot yield of 5-8 tons per acre through control of Alternaria.[235] Applications of EBDC fungicides reduced defoliation of carrot plants to 3% in comparison to 58% defoliation in unsprayed plots and 20% in copper-sprayed plots.[234] Recent experiments in Wisconsin have shown that use of chlorothalonil or azoxystrobin to control alternaria and cercospora leaf blights increased carrot yield by 4 tons per acre in comparison to the untreated check.[239]

Cercospora leaf blight is present wherever carrots are grown.[83] Cercospora lesions can result in the death and collapse of the entire leaf. Yield losses caused by cercospora leaf blight are primarily due to affected crops left in the field by mechanical harvesters. Blighted foliage snaps off easily when gripped by mechanical harvesters attempting to pull the carrots from the ground during harvest.[83]

Powdery mildew is a relatively new carrot disease. It was first reported in the U.S. in 1975 on carrots in California and Texas. Spores are light and may be carried long distances in the air. In general, powdery mildew tends to be more of a problem in warm, dry climates.[83] The leaves of carrot plants become covered with small-whitish-gray spots on both sides and on their stems. In advanced infections, the whole foliage is covered with fungal growth. Such plants look as if they have been dusted with a white powder.[236] Powdery mildew rarely kills the plant, but significantly reduces its yield.[238] The fungal covering of leaves interferes with photosynthesis, considerably weakening the plant. This weakening of the foliage makes it difficult or impossible to mechanically harvest the carrots. At mature stages, the fungus sends feeding tubes into plant tissues and extracts nutrients, thereby further weakening the plant.[238]

Cavity spot lesions are elliptical, oriented across the breadth of the root, generally smaller than ½ inch in diameter. and no more than 1/8 inch deep.[240] There are no symptoms on the aboveground parts of the carrot plant. In the 1960’s cavity spot was reported to be
a physiological disorder. Recent research in California identified the fungal organism responsible for cavity spot. Little is known about the life cycle of the fungus in the soil. It is assumed that the fungus survives as thick-walled spores, which germinate in response to sugars and other nutrients that exude from carrot roots.[241] Research with metalaxyl demonstrated almost complete control of cavity spot: 40-60% of the untreated carrots developed cavity spot while less than 1% of the treated carrots developed cavity spot.[240] Although the disease does not reduce carrot weights, infected carrots are deemed unsuitable for the fresh market due to the unsightly blemishes.[241] The economic impact of the disease can be substantial. In extreme cases, growers have abandoned fields of carrots with high levels of cavity spot.[241]

A recent USDA Report estimated the likely state-by-state impacts on carrot production if fungicides were not used. Yield loss estimates ranged from 17 to 36%. The loss estimates are based on the lower efficacy of alternatives that would be used if fungicides were not used. The primary alternatives that were included in the analysis were crop rotation, use of resistant varieties, use of clean seed, irrigation management, and field selection for disease management.[243] USDA concluded that the use of these alternatives in the absence of fungicides would result in lower yields.

6.11 Celery

Celery is a biennial plant that produces vegetative growth (edible stalks) during the first year and seed stalks during the second year.

Celery is attacked by several important diseases that affect yield and quality. The most important diseases for which fungicides are applied in the U.S. are Septoria leaf (late) blight, Cercospora leaf (early) blight, northern bacterial blight (bacterial leaf spot), and Sclerotinia (pink rot).

Early and late leaf blight are controlled with foliar applications of fungicides; primarily chlorothalonil, benomyl, and propiconazole. Bacterial blight is controlled primarily with copper sprays. Rhizoctonia is controlled by DCNA.

Bacterial blight begins as small water-soaked spots on leaf blades, which develop a greasy appearance. With age, the lesions become dry and rusty brown.[83] The lesions can coalesce and cause extensive leaf tissue death.[84] Celery stalks are not directly affected by lesions. The development of bacterial blight is promoted by warm, moist conditions. The disease ceases to spread unless frequent rains occur or overhead irrigation is employed.[83] It survives in undecomposed plant residues in the soil. Bacterial blight causes losses mostly due to additional trimming at harvest.[84]

Bacterial blight of celery was first controlled by dusting with a fungicide in 1917, and was the first bacterial plant disease to be controlled in this manner.[87] Research demonstrated that Bordeaux applications would reduce the number of bacterial leafspots on a celery plant from 263-961 to 0-6. The disease infects only celery.[83]
In 1954, control of bacterial blight of celery was greatly improved with sprays of streptomycin, which was subsequently used by celery growers in the Everglades region of Florida.[96] However by late 1959, resistance to streptomycin appeared in a high proportion of the bacterial population, preventing the antibiotic from being of any value as a control for bacterial blight.[96]

Prior to 1989, northern bacterial blight had not been reported in California. In 1989, unfamiliar disease symptoms of celery were observed which were later identified as bacterial blight. In 1989, three commercial fields had mean disease incidences of 70%, 34%, and 81%.[89] By 1991, the disease was found in all celery-growing regions in California.[86] In California, yield losses can reach 100 % if celery is left untreated.[85]

Bacterial blight is currently managed with preventative sprays of copper.

Celery pink rot has been reported since 1920 in most celery-growing areas of the U.S. In the past, it ranked among the most important transit, storage, and market diseases. About 1920, celery transit losses due to pink rot in the U.S. occurred in 57% of 365 rail cars inspected and the amount of rot averaged 50% of the plants affected.[83] Pink rot seriously damaged the Michigan celery crop in 1956. Some fields were heavily infected and were never harvested.[628] Such losses seldom occur now in the U.S. because of new fungicide programs.[83]

In celery production fields, the base of the stalks are affected first and may be pinkish or reddish brown. Entire stalks rot and become brownish, watery, and covered by cottony white fungal growth.[83] The fungus causing pink rot can survive in the soil for up to ten years.[83] Pink rot often is most prevalent following cold weather, and it is most severe during moist, cool conditions.[83]

In the absence of chemical controls, California celery growers report pink rot disease incidence increasing annually in celery fields.[85] Directed sprays of DCNA to the base of celery plants is the current recommended strategy for pink rot control in California.[174] Without the use of DCNA on treated acreage in California, it has been estimated that celery yield would decline by 10 to 15%.[14] In the 1950s, experiments in Michigan with fungicides applied to the soil reduced the incidence of pink rot from 40-59% to 2-5%.[628]

In Florida, pink rot is primarily managed by flooding the soil during the summer months for six weeks, which reduces the number of viable spores available for infection.[90]

Early blight, also known as cercospora blight, is one of the most serious fungal diseases of celery worldwide. First described in Europe in 1863 and in North America in 1881, by 1930 it was widely present throughout the U.S. and Europe.[83] When severe infections develop early in the season, excessive defoliation can result in plant stunting. Although infected leaves and stalks may be trimmed at harvest, this process increases labor costs and reduces yield quality. On numerous occasions, entire fields of celery have been abandoned.[83] During an epidemic in Florida, inadequate fungicide spray coverage
during one severe 3-day infection period resulted in a 20 to 25% loss at harvest from 100
acres.[83] One of the worst epidemics of early blight occurred during the spring of 1957
in the Everglades. Hundreds of acres were abandoned and yield from the remaining
acreage was reduced greatly.[91]

Early blight fungi survive from one season to the next as spores in and on refuse from
diseased celery plants.[83] Additional spores are produced on new blight spots. As the
amount of diseased tissue increases in a field, the number of air-borne spores increases
progressively.

Crop losses occur when blighted stalks or leaves have to be removed at harvest. Under
extreme disease conditions, only the celery hearts may remain after trimming.[84] Early
blight symptoms first appear as small yellow flecks on both the upper and lower leaf
surfaces. These flecks rapidly enlarge into brownish gray spots containing dead tissue.
Spore germination and penetration of celery plant tissue may occur after as few as 5
hours of leaf wetness.[83] The long production season for celery (12-14 weeks) is
conducive to early blight epidemiology since many secondary cycles of spore production
can occur.[83] Early blight lesions eventually develop a dry papery texture. Infected
leaves containing numerous lesions may die. On celery stalks, lesions are more
elongated, with their length running parallel to the long axis of the stalk.[83]

Early blight appears annually in Florida and affects all celery acreage in the state.[90]
Florida’s high relative humidity, long dew periods, and high temperatures present ideal
environmental conditions for development of the disease.[90] Early blight was
occasionally seen in the early 1950’s in California but has been rarely found for the last
20 years.[85]

The early blight fungus is most vulnerable to fungicides only during the relatively short
time between spore formation and spore germination and penetration of the plant. Once
the fungus is inside the plant, most fungicides have no effect. For optimum control,
coverage should be complete and frequent enough to have sufficient fungicide particles
present to kill germinating spores in every drop of foliage surface moisture.

Spraying celery with Bordeaux mixture to control early blight was the accepted practice
in Florida beginning in 1910.[92] However, the Bordeaux mixture left a considerable
surplus of lime on the plants, which injured the celery plants in warmer and drier
months.[92] Many growers abandoned Bordeaux mixtures when low-soluble coppers
became available.[87]

The widespread use of fungicides containing copper on celery fields in Florida led to an
accumulation of copper in the soils, which resulted in the occurrence of copper-induced
iron deficiencies in celery and other vegetables.[93] After 40 years of regular copper use,
Florida celery growers switched to organic chemical fungicides to control early
blight.[93] Ferbam was the first synthetic fungicide used. Not only did ferbam provide
excellent control of early blight, it also corrected the iron deficiency in the soil.[93]
In the 1970s-1980s, mancozeb and chlorothalonil became the fungicides of choice for early blight control due to consistent, efficacious disease control.[94] The use of maneb doubled celery yields in Florida experiments in the 1960s due to early blight control[629]

To avoid crop loss to epidemics of early blight, celery growers use an intense program of preventive sprays of fungicides on a seven day schedule.[84] Regularity is a virtue in spraying for early blight control. The new celery growth must be covered with a well-distributed deposit of fungicide every week.[92]

Research has shown that the weekly sprays increase celery yield by factors of 2 to 4.[84] The marketable yield of celery in a Florida experiment increased from 26,000 pounds/acre to 42,000 pounds per acre with the weekly spray schedule of chlorothalonil or propiconazole.[94]

Late blight can cause even more destruction than early blight in cool, wet seasons and on the later crop in northern states. Both blights cause major problems and occur every season in Michigan. The late blight fungus can attack any part of the plant above ground. As outer leaves and stalks turn dark and wither, the entire field can look scorched.[87] The fungus overwinters on debris from a previous crop. Spores are exuded during wet weather as gelatinous snakelike tendrils and require spattering raindrops for quick spread. Potentially, half a billion spores can be produced on a single celery plant.[87]

Late blight lesions can be distinguished from early blight lesions by the presence of spore containing structures, which appear as black dots and are similar in size and shape to grains of ground pepper. Spores in these structures are surrounded by mucilage, which absorb water and swell. The spores and mucilage are forced out of the lesions, the mucilage dissolves in water on the leaf surface, and spores germinate and infect celery plants.[84] A single lesion can contain 1,500 spores.

Leaf spots expand and eventually cause leaf death.[84] If late blight is not controlled, losses can exceed 70%.[84] Indirect monetary losses also occur because of the increased labor required to remove diseased plant parts during packing.[83]

Late blight, first reported in Italy in 1890, was reported in North America by 1921.[83] During one serious outbreak in Florida, late blight incidence increased 8-fold, from 0.3% to 2.5% defoliation in a 5 day period and 15-fold (36.4% defoliation) in 12 days.[83]

In the 1920s celery growers in Michigan were estimated to lose 25-50% of their crop to late blight.[627] The recommendation was made to spray the crop weekly with Bordeaux mixture.

Experiments in Michigan in the 1970’s demonstrated that fungicides could double the yield of celery by controlling Septoria blight, increasing yield from 14 tons/acre in untreated check plots to 28 tons/acre in treated plots.[97]
Generally 60-100% of California’s coastal celery acreage is infected by late blight each season.[85]

It has been estimated that without fungicide use celery yields would decline by 30% in Florida, 37% in California, and 100% in Michigan.[10][14] The 100% loss in Michigan is based on the assumption that it would not be economical for growers to salvage the remaining yield without fungicide use; the cost of trimming would not be justifiable.

6.12 Cherries (Included in Section 6.31 Peaches)

6.13 Citrus

Arizona, California, and Texas citrus fruit is predominately sold in the fresh market while Florida citrus fruit is predominately processed.

Due to the arid climate of the western citrus states, where most rain falls during the winter, Arizona and California do not have the disease pressure that Florida and Texas do. There are fewer diseases, with almost no foliar or fruit diseases of citrus in the western states.[290] However, western states must rely on irrigation during the hot, dry summer months and, as a result, have a disproportionate amount of root disease associated with irrigation and water recycling.[290] Phytophthora root rot and brown rot are problems in all four states and are controlled with applications of copper, fosetyl-Al, and mefenoxam. Copper is also used to control melanose in Florida and Texas. Copper is used to control greasy spot in Florida and Texas. Other fungicides used for greasy spot control in Florida and Texas include azoxyystrobin and fenbuconazole. In Florida, benomyl has been used to control postbloom fruit drop while ferbam has been used to control scab. Copper is used to control septoria spot in California.

Melanose is present in most citrus growing regions but is important only when rainfall occurs during the period of early fruit development (Florida, Texas).[288] The disease completes its life cycle on dead twigs. Spores ooze from the twigs in a moist, sticky gelatinous mass. Rain is necessary for the dissemination of the spores. Infection of fruit occurs when spore-laden water falls from the infected wood in the top of trees onto fruit below. The fungus never produces spores from the infected fruit. Fruit infections do not affect the overall yield of juice solids per tree. On fruit, melanose lesions begin as light brown circular spots that later become brown to black raised pimples, imparting a sandpaper-like feel to the fruit. On fruit there is a tendency for the disease to be streaked because spores are washed over the surface. When infections are numerous, spots coalesce and scar tissue occurs in masses. Epidermal and subepidermal cells, up to six cells deep, are killed and become impregnated with reddish brown gum.[288] The fungus penetrates the cell walls by means of enzymes which digest the host cells. Unless the fungus is controlled, external rind blemishes of melanose infections reduce the grade of fresh fruit. Severely infected fruit may be unsuitable for the fresh market. Melanose can be responsible for significant financial losses, especially if there is substantial fruit blemishing, resulting in downgrading of unsightly fruit to juice production from fresh market. Prior to the development of fungicides for control of melanose in the 1920s,
60% of Florida’s citrus crop bore melanose blemishes while 4% were so severely scarred that they were packed as third grade or lower or discarded as culls.[289] When copper sprays were applied, 80% of the fruit was graded #1 while 0.4% to 47% of fruit from unsprayed trees was graded #1.[376] Orchards with a history of melanose or those with a large amount of dead wood need yearly copper sprays. Copper fungicides are the mainstay for control of melanose. Research has shown that a carefully timed copper spray can reduce the percent of fruit with conspicuous melanose from 79% to 6%.[293] It is critical that young fruit are covered with copper which kills spores that germinate on the surface of the fruit during rains. In Florida and Texas, one copper spray usually suffices for control of melanose.[288]

Scab was introduced into the U.S. in 1876 on the first Satsuma tree brought in from Japan where it had long existed. Scab-causing fungi overwinter in the tree canopy. The fungus needs a minimum of 2.5 hours of continuous wetness for spore germination and infection. Infection of young fruit promotes the formation of relatively large warty outgrowths on the rind.[288] Fruit remain susceptible to infection for about three months. A recent experiment in Florida resulted in 99% marketable fruit when fungicides were used to control scab in contrast to 50% marketable fruit when scab was uncontrolled.[294] Another experiment indicated that fungicides reduced the incidence of scab from 30% of the fruit affected to 0.4%.[306]

Several species of Phytophthora, a soil-inhabiting fungus, cause foot rot, root rot, and brown rot of citrus. The fungus is present in most citrus soils worldwide. Foot rot is an injury of bark on the trunk or roots of citrus trees near ground level. The fungus survives in the soil as a thick-walled spore capable of withstanding extremes in both moisture and temperature. Given abundant soil moisture, tadpole-like swimming spores that can move short distances are produced. These spores are attracted to root tips and to wounds on roots where they attach, germinate, and directly penetrate the woody tissue. The spores are probably attracted to the new roots by nutrients that are naturally exuded from the root zone.[297] The first indication of foot rot is the appearance of drops of gum on the surface of the bark. If the bark is scraped away, a brown discolored necrotic slippery area will be found. During the dry season, diseased bark may dry, shrink and split. The tree is unable to maintain adequate uptake of water and mineral nutrients, and carbohydrate reserves in the root are depleted by repeated fungal attacks.[297] This results in the reduction of fruit size and yield, loss of leaves, and twig dieback in the canopy. Heavy losses of citrus trees to foot rot in 1900 to 1915 alarmed California citrus growers and led to the establishment of the Citrus Experiment Station where researchers developed Phytophthora control measures with applications of Bordeaux mixtures.[383] Surveys in Arizona revealed that 84% of all groves in Maricopa and Yuma counties contained Phytophthora.[375]

Foot rot can be prevented by painting the trunk with copper fungicides or by spraying systemic fungicides, such as mefenoxam or fosetyl-Al. Systemic fungicides can be used as soil drenches, foliar sprays, or trunk paints to cure existing infections as well as prevent future infections.[288] Movement of mefenoxam, when applied as a soil drench, is mostly upward while fosetyl-Al moves downward systemically from the foliage to the
Research in Arizona demonstrated that the roots of untreated citrus trees grew 50-75% less than trees treated with mefenoxam or fosetyl-Al. Research in California demonstrated that mefenoxam treatments increased citrus yield by 43-46% in comparison to untreated trees. Research in Florida demonstrated that mefenoxam treatment increased the weight of roots by 32% in comparison to untreated trees.

Under wet conditions, *Phytophthora* spores, the causal pathogen of brown rot, are splashed from the soil onto low hanging fruit. When weather is favorable for fungal growth, spores produced on these fruit are then splashed higher into the canopy. Fruit that become infected shortly before harvest may not show symptoms until after they have been held in storage for a few days. Fruit infected with brown rot usually falls to the ground; however some brown rotted fruit remain on the tree. If these are picked and placed in storage, the brown rot develops and may even spread to previously uninfected fruit in cartons after harvest. The decay is first observed as a light brown discoloration of the rind. The affected area is leathery. Delicate white mold forms on the rind surface under humid conditions. Infected fruit have a characteristic pungent, rancid odor. Copper fungicide sprays applied to the soil and the lower part of the tree canopy prior to anticipated wet weather help to prevent brown rot. If the disease has already appeared, the whole canopy should be sprayed. Record-breaking rains in California in 1977 and 1978, led to heavy damage of the citrus crop by brown rot. The drought years preceding 1977 had encouraged many growers to omit a brown rot spray. These growers sustained brown rot losses amounting to 30-50% of their crop. Brown rot is a relatively minor problem in Florida, primarily because environmental conditions seldom favor its development.

The first mention of greasy spot on citrus leaves was from Cuba and Florida in 1915. Greasy spot is known to occur with certainty only in Florida, Texas, parts of Central America and South America, the Caribbean, and some Asian countries. Greasy spot seems to be important only in areas where nearly 100% relative humidity and high temperatures occur simultaneously for prolonged periods. Greasy spot affects leaves of all commercial plantings. The most serious consequence is defoliation. In Florida losses of up to 25% and 45% of fruit yield of orange and grapefruit respectively have been caused by defoliation induced by greasy spot.

Spores of the fungus are produced in decaying leaf litter on the orchard floor. After a wetting period by either rain or irrigation, the fungus swells and ejects numerous microscopic spores. Spores are airborne and are deposited on the underside of leaves where they germinate. The fungus survives for several weeks as a branching growth on the leaf surface. After extensive growth on the surface of leaves the fungus penetrates leaf tissue. It takes several weeks for the fungus to grow deeply into the host. Growth of the fungus inside the leaf tissue eliminates air spaces and causes cellular swelling resulting in blister formation on the leaf surface. The plant cells die and become impregnated with gum. The blisters later become swollen and darkened and resemble dirty blotches of grease. Heavily infected leaves fall off prematurely. Healthy leaves remain on the tree 12-18 months; infected leaves drop in 8-10 months. Excessive defoliation of citrus causes a reduction in the following spring’s growth flush and a
subsequent yield reduction in the following crop year. The fungus completes its life cycle in the dead leaves on the orchard floor.

Greasy spot also causes a spotting of fruit. Pinpoint black specks occur between the oil glands on the fruit rind. On grapefruit larger specks are produced giving rise to a symptom called greasy spot rind blotch. A serious aspect of greasy spot rind blotch is that living cells adjacent to the specks do not color normally due to retention of chlorophyll and retain a green color.

Because of the superficial growth of the fungus for an extended period on the leaf surface and the long period of time for deep penetration, greasy spot can often be adequately controlled with only one fungicide spray per year. Copper fungicides have proven to be the most reliable materials for controlling greasy spot. Spray oil is also used to control greasy spot. Oil is not chemically fungicidal, but it does interfere with the mechanical penetration of the leaf surface by the fungus. Research has demonstrated that a single application of oil plus copper can reduce defoliation from 59% to 2%.

In Florida postbloom fruit drop (PFD) first appeared in 1983 in southwestern production areas. Subsequently the disease spread throughout the state. The lime groves in which PFD was first detected totaled 270 acres. Two hundred and three acres of these groves had trees that were estimated to be 90-100% infected. During severe outbreaks Florida growers have reported yield losses of 50-90%. The fungus infects blossom petals, producing brownish water-soaked patches. Under moist conditions petals become covered with the slimy peach to orange colored spore masses of the fungus. The spores are dispersed by rain splash to nearby healthy blooms. Infection occurs within 12 hours and symptoms appear in 2-3 days. By 5 days after infection, newly infected petals produce large numbers of spores. With the large number of spores produced and its short life cycle, PFD can very quickly become epidemic in a grove. Over 90% of the blossoms may show symptoms after 2-3 days. Under favorable conditions, disease incidence can double every 3-4 days. Petals eventually fall and the fruitlets drop shortly after, resulting in reduced fruit set and lower yields. After the petals and fruitlets fall, the calyces and floral disks which normally fall off if no fruit is set remain attached to the twig. These “buttons” persist for the life of the twig and are diagnostic for PFD: they are not produced by any other disorder. PFD control is achieved by protecting blossoms with fungicide sprays. Research in Florida demonstrated that regular protective fungicide sprays reduced the percent incidence of blossom blight infection from 86% to 2%, which led to a yield increase of 200%.

Septoria spot symptoms occur on twigs, leaves, and fruit of citrus trees. On leaves, lesions 1 to 4 mm in diameter develop as raised blister-like black spots that are surrounded by a black halo. Symptoms on the fruit include circular, dark, and sunken spots 1 to 2 mm in diameter, which may be surrounded by a reddish brown halo as fruit mature, as well as coalescing lesions that may resemble a “tear stain” pattern or blemish. Lesions are generally shallow and remain in the oil-gland layer of
the peel but may turn into larger pitted lesions (4-6mm in diameter) that extend into the white portion of the peel. Management of septoria spot has historically been done in the grove through the use of protective copper sprays that are applied to new growth and fruit prior to favorable environments for disease.[555] Copper materials completely inhibit germination of pathogen spores. In field studies, a single fall-season application of copper fungicides was very effective in controlling the disease on fruit and leaves. New leaves that grow after fungicide application, however, may become infected. Therefore, a second application may be needed to protect these leaves and late-season fruit.[555]

It has been estimated that without fungicide use, citrus yield on currently treated acres would decline by 50% in Florida and Texas and 25% in California and Arizona.[30]

6.14 Collards

Georgia growers produce collards primarily for the processing market while most collards grown in Alabama, California, North Carolina, and South Carolina go to the fresh market. The value of the crop for fresh market ranges between 20 and 39 cents per pound while the value of collards for processing ranges between 4 and 6 cents per pound.[170] Collard leaves must be blemish-free to be sold in the fresh market.

Fungicides are used to control the three primary diseases of collard plants: Alternaria, downy mildew, and black rot.[170]

The initial symptom of Alternaria is the appearance of small, round, brown spots on the leaf surface. These spots enlarge to form irregular shaped lesions in which can be found a concentric ring pattern. As the spots continue to expand, they can lead to defoliation of the plant if the causal Alternaria fungus is not controlled.[171]

Black rot is a bacterial disease and is recognized by symptomatic V-shaped bright yellow to orange areas on collard leaf margins.[172] Leaf veins within and leading from the disease lesions are typically black. Infected plants generally deteriorate structurally and collapse.[171]

Downy mildew is caused by an airborne fungus. The disease produces small specks surrounded by yellow areas on the upper side of diseased leaves. These yellow areas may grow together to cover all or part of the leaf. The underside of the leaves may show a pale, whitish-gray mold. The diseased areas may enlarge rapidly during warm, moist weather, causing the leaves to wither and die.[171]

Disease-infested collard fields are often not harvested resulting in 100% losses. Some growers harvest their crop prematurely, when the crop is relatively disease-free, suffering a 5 to 10% reduction in yield.[170] When harvested at maturity, growers can sort out infected collard leaves and discard them, reducing the crop yield by 5 to 25%. This practice is widely employed by North Carolina producers.[170] Growers can also raise
the height of their cutter bars during harvest to leave the more heavily infected lower portions of collard plants in the field. This method of disease control reduces yield by 10 to 25%. While these alternative disease control methods are relatively cost effective for fresh market collards, sorting out diseased leaves is not an economically feasible practice for processing collards because of their low market value.

The most effective and practical management strategy for suppression of these diseases is the use of fungicides applied on a timely schedule throughout the season. Copper is the most-widely used fungicide and is used to control all three diseases. Fosetyl-Al is used only for control of downy mildew.

Without fungicides, it is estimated that collard production would decline by 20 to 30% in states that produce for the fresh market. Fresh market growers would substitute earlier harvest and sorting to avoid or remove diseased leaves. Without fungicides, Georgia processing collard production would decline by 75% as its heavily infected fields could not be harvested.

6.15 Cotton

Seed treatments and hopper box treatments are not included in this study (almost all cottonseed is treated with a fungicide before planting to help control seedling diseases). This study includes fungicides that are applied in the furrow at planting to control seedling diseases and fungicides that are applied to cotton foliage to control rust.

Seedling diseases are the most important disease problem of cotton. Seedling diseases occur when developing seedlings are unable to overcome damage caused by invading soilborne pathogenic fungi. The most common seedling diseases in U.S. cotton are *Pythium* (damping off and seed rot) and *Rhizoctonia* (sore shin and damping off). These fungi commonly occur in almost all soils that cotton is planted into. *Pythium* thrives in cool soil while *Rhizoctonia* prefers warm soil. Poor stands are usually the first symptom of a seedling disease problem. Seeds may fail to germinate or die prior to or after emergence. Seedling diseases can kill a high percentage of the seed and seedlings and cause low yields because of plant skips within the row and non-uniform stands. Severe stand losses may require replanting, resulting in additional cash inputs. California growers who plant early are advised to use an in-furrow fungicide for control of *Pythium* and *Rhizoctonia*. *Pythium* is a root rot that infects the seed and the first primary root causing seed rot and preemergence damping off, which is defined as death of the cotton seed after it germinates but before it emerges above the soil surface. Within hours after planting, the seeds are attacked by *Pythium* before the seed can germinate, which results in
pregermination decay of the seed. A diseased seed is usually completely deteriorated and when squeezed, the contents ooze from the tip.[158] Germination of *Pythium* spores in the soil is stimulated by exudates from germinating cotton seeds or seedling roots.[175] Even if the seedling is not destroyed immediately, the plant may die soon afterward because of its damaged root system.[184] *Pythium* destroys the tap root and decreases the plant’s ability to draw moisture out of the soil. As a result, the plant is less vigorous and yields are lower. *Pythium* is the number one disease problem for many producers, especially in colder soils on the northern edge of the cotton-growing region. In Tennessee, late April and early May are prime planting dates, but this time usually brings cold, wet weather.[203]

*Rhizoctonia* can attack seedlings from the time of emergence until the plant is six to eight inches tall.[184] *Rhizoctonia* invades the cotton plant at soil level and produces a brown sunken lesion that girdles the stem, cutting off water and nutrients to the plant. This can cause the seedling to collapse and die (postemergence damping off). The sunken lesion is caused by the death and collapse of cells in the plant.[175] Plants surviving *Rhizoctonia* are weakened and they bear the mark of the stem-girdling lesion at the base of the stem (thus the nickname “sore shin”).[157]

In the mid-south, 20-65% of the cotton acreage is treated with an at planting furrow fungicide application. In the drier growing areas of the West (CA/AZ/NM) a smaller percentage is treated (2-5%). Texas cotton growers do not make soil or foliar fungicide applications.[209]

Growers who plant early or in fields where stand losses from seedling diseases have occurred previously are advised to make a soil fungicide application. In-furrow fungicides are designed to extend a protected zone from the soil surface to just below the seed. Most of the fungicides used in-furrow are effective against either *Rhizoctonia* (PCNB, iprodione) or *Pythium* (etridiazole, mefenoxam). They are often prepackaged together in combination products. Recently, azoxystrobin was registered for use on cotton and has demonstrated efficacy in controlling both *Rhizoctonia* and *Pythium*. Tests in Alabama have shown an average increase in lint yield of 10% as a result of in-furrow fungicide application while tests in Tennessee have shown yield increases of 29 to 45%.[203][204] In recent years, Georgia growers have increased their use of in-furrow fungicides and have reduced the losses due to seedling diseases from $22 million/year to $16 million/year at an increased fungicide cost of $200,000/year. (Figure 14)

It is known that PCNB used in-furrow preferentially binds to the outer tissue of seedlings as they grow through the treated soil. This creates a barrier that protects the seedling from *Rhizoctonia* infections.[749] The efficacy of etridiazole appears to be related to its ability to move systemically in young seedlings.

Rust was first described from Baja California, Mexico in 1893 and from Arizona in 1922. The disease occurs only in certain parts of southern Arizona, New Mexico, west Texas, and northern Mexico. The fungus that causes the disease lives on cotton and grama grass.[702] During summer rains, the spore stage on grama grass germinates to produce
airborne spores that are carried up to eight miles and cause initial infections in cotton. All new infections in cotton are dependent upon spore showers from grama grass; the spores produced on cotton can infect only grama grass.[702] The most common symptom in cotton is the appearance of bright yellow to orange spots on the leaves. These pustules may cause leaf curl, defoliation, dwarfed bolls, premature opening of unripe bolls, reduced yield and quality, and/or broken stems that interfere with mechanical harvesting.[175] Typically, cotton rust appears during the so-called “monsoon season” in July and August. Severe outbreaks can cause yield reductions of over 50%.[702] Rust can be controlled with protective applications of mancozeb.

USDA has estimated that if in-furrow fungicides were not used, cotton yield losses would range from 10 to 22%, while if the rust fungicides were not used, the loss in cotton yield would be 20 to 25%.[205]

6.16 Cranberries

The American cranberry industry began in Massachusetts with the hand harvest of native populations of plants. Today, cranberries are grown in beds that are drained, cleared, leveled, and covered with a one to two inch layer of sand, then planted with selected cranberry vines.[103] Most cranberry beds must be irrigated. Water for irrigation is applied through permanent sprinkler systems that provide complete coverage of the vines.[103]

Cultivated cranberries are perennial, low growing vines that form a dense mat over the surface of the cultivated bed. Cranberry vines will live indefinitely if pests are controlled. In New Jersey and Massachusetts, some beds have been in production for over 100 years.[104] Old cranberry varieties are replaced with new ones from time to time.

In Massachusetts, New Jersey, and Wisconsin, where winters are harsh, cranberry beds are flooded to protect the vines from cold temperatures and from desiccation. Desiccation can result when dry winter winds evaporate moisture from the vines that can not be replaced because the soil around vine roots is frozen. Water is drained from flooded beds in the spring. Cranberry beds are not flooded in Washington or Oregon due to their mild winters. Annual vine growth begins with production of new leaves starting in May, followed by flowering beginning in June. Berries reach maturity about 80 to 100 days after full bloom. Cranberries to be sold as fresh fruit are generally bounced over barrier boards to assure quality: sound berries bounce and travel forward. Fresh cranberries can be stored under refrigeration for as long as three months. Berries destined for processing are generally frozen within 12-24 hours of harvest.[102]

U.S. cranberry growers must control at least eight and as many as fifteen fungal pathogens that can cause significant crop loss. These diseases and their prevalence in New Jersey and Massachusetts are listed in Table 4. In Massachusetts and New Jersey, early rot infects all cranberry beds and can cause 100% losses if not controlled.[15]
Other commonly occurring diseases can produce losses ranging from 25 to 50%.

In western states (Oregon and Washington), cranberry fruit rots are estimated to infect 95% of the cranberry acreage, with the potential of causing yield loss of 20% if they are not adequately controlled.\[13\] Twig blight infects 5% of cranberry beds in Oregon and Washington.\[13\]

In Wisconsin field rots are less severe. However, berries grown in Wisconsin and stored for sale at a later date are subject to storage rots. These cranberries must be protected from fungal inoculation during the growing season to minimize losses to storage rots.\[118\] Approximately 15% of Wisconsin’s cranberries are sold to fresh markets rather than for processing.\[120\] Cottonball is an economically important cranberry disease in Wisconsin. The disease reduces both yield and fruit quality and increases the costs associated with postharvest sorting and handling.\[102\] The fungal pathogen overwinters in the mummified remains of the previous season’s infected berries (mummies). In the spring fungal spores are released from the mummies and inoculate the new crop of host berries. Once inside the new host berries, the pathogen grows as the fruit grows. Infected berries become filled with a white, cottony fungal mass. As healthy berries ripen and turn red, cottonball infected berries remain firm and turn brown.\[102\] Cottonball occurs in all cranberry growing regions of Wisconsin and has increased in importance over the past 25 years.\[119\] When left unchecked, cottonball incidences of 27 to 48% berry infection have been observed.\[119\] When disease levels are high, it is not profitable for growers to deliver fruit to the receiving stations because of the increased handling and sorting required to remove infected berries, resulting in 100% loss of the crop.\[121\] Experiments in Wisconsin showed that marketable yields of cranberries were reduced by 60% without fungicide use for control of cottonball and fruit rots.\[173\]

The fungi that cause fruit rots in cranberries overwinter on infected living and dead vine leaves and stems and on rotted fruit left in the field.\[102\] Wind and wind-driven rain disperse fungal spores from their overwintering hosts. These spores land on blossoms or small, developing fruit and will penetrate the plant tissue if there is a suitable layer of moisture present for 6-8 hours. Fruit are infected during the early stages of development; after a certain point in fruit development, no new infections take place. Some of the fruit rots (bitter rot) remain latent in the fruit until the fruit begins to mature. Early rot first appears as a small light-colored watery spot, which enlarges rapidly until the whole berry becomes soft. Cranberries with end rot are often distended by gas produced by the rotting process and may burst from the increased pressure. The end rot infected fruits eventually collapse, shrink and become yellowish or brownish.\[102\] Twig blight infects leaves on new growth vines and the fungi kill one-year-old wood.\[102\]

Soon after the establishment of the first cranberry beds in New Jersey, a severe fruit disease was eliminating most of the crop. The growers of that era found that a bed could produce healthy fruit for as long as 5 to 10 years before succumbing to the ‘rot.’\[104\] Prior to the use of fungicides, cranberry fruit rot was responsible for up to 100% crop loss in worst-case scenarios and routinely reduced the New Jersey crop by 33%.\[104\] With the development of Bordeaux mixture (copper plus lime sulfur), cranberry fruit rot could
be reduced by as much as 10 to 50%. Four or five applications of the Bordeaux combined with one pound/acre of fish oil soap were made.[105] Development of more effective synthetic fungicides permitted further reductions in the incidence of fruit rot.[104] Cranberry growers stopped using Bordeaux in response to the greater effectiveness of the synthetic fungicides and research that showed vine thinning and damage resulting from repeated annual sprays of Bordeaux.[106] Currently, cranberry fruit rot losses range from less than 1% to 15% annually.[104] The spraying of cranberry beds for disease control has been a general practice of New Jersey cranberry growers since about 1900.[105]

Fungicides are typically used on 100% of the cranberry acreage in Massachusetts, New Jersey, Oregon, and Washington. In Wisconsin approximately 24-70% of the acres are treated annually. With milder winters, both field rots and fungicide use have increased in Wisconsin. Fungicides are typically applied 2 to 3 times each season. The first application is made at bloom followed by 1 or 2 applications; 12 to 14 and 28 days later.[13][15] Fungicides are applied through sprinkler irrigation systems in all states except Wisconsin, where they are typically applied with ground sprayers.[13][15] The primary fungicides used in cranberry growing are chlorothalonil, mancozeb, ferbam, and copper.

Most cranberry fungicides are protectants that are effective only on the plant surfaces to which they are applied. A few, however, have limited systemic activity.[103] Chlorothalonil and mancozeb, the most widely used cranberry fungicides have high efficacy against several major fruit rot diseases. Fungicides used for fruit rot control also generally provide control of foliar diseases. Propiconazole is used exclusively in Wisconsin for control of cottonball. Chlorothalonil is most commonly employed to control twig blight in the Northwest.[107] Copper is generally used when disease inoculum is low: usually at late bloom.

An experiment in Washington demonstrated that cranberry yields were reduced by twig blight infection. In comparison to chlorothalonil treated test plots, an untreated check suffered 67% yield reduction.[113] In a New Jersey experiment, 40% and 55% yield losses were suffered in the first and second years, respectively, after fungicide use was discontinued in a test cranberry bed.[104]

Cranberry flooding and draining of cranberry beds is employed, with varying levels of efficacy, to increase yields, suppress infection, and attempt to reduce use of synthetic fungicides. By draining the winter flood early, the cranberry growing season can be effectively extended, thereby increasing yield. Application of late water is a flood management technique employed to suppress infection; reducing fungicide use and increasing yield. The practice has been in use in Massachusetts since the nineteenth century. The application of late water begins when winter floodwaters are drained in early to mid March. Four weeks after draining, the beds are reflooded and the flood is maintained for four weeks, after which it is again drained. The mechanism by which the application late water affects fruit rot is unknown; however, one effect is to delay the onset of bloom by 10 to 14 days, compressing the bloom period from 30+ days to about
20. This compressed bloom period occurs later in the season than typical cranberry bloom, when drier weather generally prevails and conditions are less favorable for fungal infection. Forcing bloom in this manner reduces the amount of time cranberry plants are susceptible to infection.[114] Flooding is recommended for only one year out of three. More frequent flooding leads to elongation of uprights with little growth from lateral buds, which reduces cranberry yield.[107][114]

Growers are advised to not use late water in years of abnormally warm preceding winter due to negative impacts on yield.[116] Research has shown that late water can reduce the incidence of fruit rot and the rate of fungicide applications during the second and third years after late water is used in addition to reductions or eliminations in the year of late water application.[104][116] In one study the combination of late water plus applications of copper produced equivalent fruit rot control to three applications of chlorothalonil at half of the tested sites.[115] While the application of late water has been demonstrated to be effective in some situations, it has failed to control disease in others. In a five-year study, late water had no effect on yield during two of the observed years while yield was reduced by 9%, 25%, and 35% during the other years.[114][116] Late water treatments have not been effective in managing fruit rot in New Jersey.[104]

In New Jersey and Massachusetts, the loss of registered fungicides for cranberry disease control could lead to nearly complete crop loss within 5 years if replacement fungicides were not registered.[104]

A recent assessment from USDA concluded that, without fungicides for fruit rot and vine disease control, overall yield reductions of 20% would be common among U.S. cranberry producers, with losses ranging from 0 to 100% in individual beds.[107] After sustaining this level of disease damage, it is questionable whether cranberry growers in areas with high disease pressure would be able to produce a crop with quality high enough to justify harvesting.[107]

6.17 Cucumbers (also includes 6.9 Cantaloupes and 6.48 Watermelons)

Chlorothalonil and copper are used for controlling powdery mildew, Alternaria leaf blight, anthracnose, gummy stem blight, and downy mildew. Chlorothalonil also controls scab, Cercospora leaf spot, belly rot, and target leaf spot. Copper additionally controls angular leaf spot and bacterial fruit blotch. Sulfur and myclobutanil are used to control powdery mildew. Azoxystrobin is labeled for the same diseases as chlorothalonil except scab and target leaf spot while mancozeb and maneb control the same diseases as chlorothalonil except for target leaf spot, Cercospora, and powdery mildew. Mefenoxam is targeted at Pythium and Phytophthora. Thiophanate-methyl controls anthracnose, powdery mildew, downy mildew and gummy stem blight. Dimethomorph is used to control Phytophthora.

Control of anthracnose, downy mildew and belly rot in 1966 Florida experiments increased cucumber yields by 42%.[526] In Arkansas the control of anthracnose and
Cercospora leaf spot increased watermelon yields by 128%.[496] A Georgia experiment documented that fungicidal control of downy mildew, belly rot, and gummy stem blight increased cucumber yields by 63%.[530]

Florida cucumber yields doubled during the late 1940s and early 1950s.(Figure 7) The use of new synthetic fungicides was identified as a major factor in the increased yields.[528]

Belly rot, which begins on the underside of the fruit, is caused by *Rhizoctonia solani*, a common soil fungus. Belly rot is a serious disease of cucumbers grown for pickling in the southeastern U.S. *Rhizoctonia* invades cucumber fruit in contact with the soil.[489] The warm, moist conditions resulting from the formation of a dense canopy favor infection of the young fruit developing on the soil surface.[490] Lesions begin as water-soaked areas that subsequently collapse and form brown, sunken irregular cankers on the fruit surface. Yield losses in individual fields can amount to 80%.[490] Blemished fruits cannot be mechanically separated from marketable fruits, and must be removed by hand. If more than 5% of the fruit are rotted, the processor may reject the entire load.[491] Hand culling diseased fruit increases harvesting costs. Effective and economical control of belly rot depends on the application of fungicides to the soil before vine elongation and fruit formation. Research demonstrated that soil treatments of fungicides reduced the proportion of fruits rotted by 64% with marketable yield increases of 24 to 60%.[491][492]

Although scab, or gummosis, is rarely a problem for cucumbers because of the availability of many resistant cultivars, it remains a significant problem in cantaloupes and watermelons.[489] Scab produces the greatest damage on fruit. Spots first appear as small sunken areas. A sticky substance may ooze from the infected area. The fruit canker becomes darker with age; collapsed tissue sinks until a pronounced cavity is formed. A dark-green velvety layer of the fruiting fungus grows in the cavity.[144] Secondary soft-rotting bacteria may also invade the cavities and lead to foul-smelling decay. When environmental conditions are favorable for scab development, severe epidemics can occur on susceptible varieties. In one field of Washington cucumbers in 1961, scab was first noticed on August 14; the percentage of fruit infected by scab more than doubled every week following discovery, increasing from 18% on August 21 to 92% on September 4.[144] Research has shown that scab control with fungicides depends on starting sprays before disease appears. Where scab is anticipated, sprays need to be started when plants are small.[144] Research in Delaware with cantaloupes showed that in the unsprayed control 80% of the fruit was cull quality, while the sprayed plots yielded more than 85% marketable fruit.[493]

Target leaf spot, also called *Corynespora* blight, is a disease of many cucurbits and is most common on cucumbers. The disease is common in Florida, where it can be found all year, and in the southern and mid-Atlantic states in the fall when temperatures and humidity are favorable for its development.[489] The disease was first reported in Florida in 1957. Leaf spots are circular; as the lesions age they turn gray and drop out giving leaves a shredded appearance. Rapid defoliation can result in reduced yield and
early termination of harvesting.[144] Research has demonstrated a maximum infection rate of 62%.[494] In one Florida experiment, symptoms appeared 6 weeks after seeding; defoliation increased to 5% in 10 more days and then increased from 5 to 90% in just 6 days.[494] The rate of disease development can be retarded by starting weekly fungicide sprays as soon as symptoms appear.[144]

Symptoms of Cercospora leaf spot usually appear first on older foliage as small circular spots which gradually enlarge spreading over the leaf surface. The spotted leaves turn yellow, die, and fall off the plant.[495] Defoliation may result in reduced fruit size and quality.[489] The disease has been reported from most watermelon growing regions in the U.S.[495] Research in Arkansas demonstrated that the control of Cercospora leaf spot increased watermelon yields by 65 to 71%.[496]

Gummy stem blight was first described on cucumber in France and then in the U.S., both in 1891. Gummy stem blight is widely distributed over the eastern and central U.S. and has been reported as far west as Arizona. The disease can cause considerable losses by killing emerging seedling and through defoliation.[495] On the leaves the disease is characterized by reddish brown spots. On stems the disease appears as elongated water-soaked areas in which are formed great quantities of spores. These cankers crack open and exude a reddish-amber gum that has given the disease its name.[495] On fruit the disease is known as black rot. In watermelon, the rind immediately below the point of infection is dark brown to black. Decomposition of the pulp occurs rapidly.[144] In cucumber, a black decay may appear at the blossom end; this decay may extend into the pulp. After harvest the first symptoms are greasy, soaked spots that may appear anywhere on the fruit. Sometimes a gummy exudate develops in the center of the spot. At later stages of decay a characteristic symptom is a blackish discoloration of infected areas. This is accompanied by a drying out of diseased tissue with consequent shriveling and wrinkling.[144] The fungus can survive in fields in the absence of host plants for at least two years.[144]

Most cucurbit growers are unable or unwilling to employ rotations longer than one year for several reasons.[497] Because cucurbits can be profitable crops, cucurbit growers in the Southeast devote some portion of their acreage to these crops each year. Many of these growers have invested capital in drip or center-pivot irrigation systems and grow watermelon or cantaloupe each year to ensure a return on this investment. Other growers have a limited amount of land available for rotation away from watermelon and other cucurbits and follow one cucurbit crop with another.[497]

In ten years of monitoring, gummy stem blight was found in almost every commercial watermelon field in Florida. In Florida, crop failures have sometimes occurred due to gummy stem blight epidemics.[144] Gummy stem blight has been observed to cause losses in Florida melons of 30 to 90%. Fruit rot has also occurred on Florida cucumbers during transit to New York.[144] Gummy stem blight remains a threat throughout the growing season because of the reservoir of spores in the soil. All that is needed for an epidemic is a prolonged period of favorable weather. An epidemic in South Carolina in 1991 resulted in abandonment of 15% of the watermelon acreage before harvest.[499]
Epidemics of gummy stem blight in Texas in 1997 resulted in cantaloupe yield losses of 50% and 36% losses in watermelon.\[500\] Gummy stem blight infects 65% of Texas cantaloupe acreage every year.\[559\] Satisfactory chemical control can be obtained by regular applications of protectant fungicides.\[489\] Research has shown that the amount of infected tissue must be kept low from the beginning of the season to adequately manage gummy stem blight.\[499\] The weight of marketable watermelons was increased by 61% with full season fungicide programs in South Carolina.\[499\]

Alternaria leaf blight is a foliar disease that can affect most cucurbit crops. In the U.S., cantaloupes and watermelons are most commonly affected, but the disease can also affect cucumber.\[489\] It is often prevalent in production areas with frequent rains and high temperatures. Infected leaves develop a cupped appearance and then die, exposing fruit to sunscald, which reduces both the quality and quantity of marketable fruit.\[489\] Defoliation results in reduced yield and causes fruit to ripen prematurely; cantaloupe and watermelon fruit can be of such poor quality that they are almost worthless. Reported losses include almost complete loss of the melon crop in Arizona in 1921.\[144\] In Indiana, the disease has been responsible for yield reductions of nearly 50% when fungicides were not used.\[501\] 70% of Texas cantaloupe acreage is infected with *Alternaria* every year.\[559\] Growers rely on repeated applications of protectant fungicides for control of alternaria leaf blight.\[501\]

Anthracnose lesions can form on seedlings, leaves, stems, and fruit of cucumbers, watermelons, and cantaloupes. On fruit, anthracnose lesions are circular, sunken, water-soaked areas, which develop as the fruit approaches maturity and then expand to a large size.\[489\] In moist weather the centers of the black lesions are lined with a gelatinous mass of pink spores. The cankers do not penetrate the flesh, but a melon with a large number of lesions usually is insipid and may be bitter.\[144\] Cankered fruit are usually destroyed by secondary soft-rot organisms that enter through the broken rind. Elongated lesions appear on stems; these together with foliage destruction may kill the whole vine.\[144\] In the field, anthracnose of cantaloupes is characterized by rather complete defoliation while watermelon anthracnose is characterized by the scorched appearance of the foliage.\[502\] Anthracnose occurs wherever cucurbits are grown in a humid environment.

Anthracnose was first noticed on gourds in Italy in 1867; it was noted on gourds in Philadelphia and watermelons in Wisconsin by 1885.\[502\] In 1906, 25 to 60% of the cucumber crop in Ohio was lost due to anthracnose, which also was reported to have caused a 35% loss in Indiana in 1908.\[502\] Anthracnose of cucumbers caused a 50% loss in Michigan 1912, while watermelon losses were 30% in South Carolina in 1916. 100% losses have been recorded in individual fields. At Live Oak, Florida, there was a report of total loss of the watermelon crop on one 15 acre field in 1916 due to anthracnose.\[502\] Infected melons were routinely rejected by inspectors at loading time, while latent undetected infections caused considerable rot in transit and storage.

Early experiments showed that Bordeaux sprays provided partial control of anthracnose and 3,400 acres of watermelons were subsequently sprayed in 1919.\[502\][503\] With the
almost universal adoption of nabam and zineb sprays in the late 1940s, watermelon losses to anthracnose were reduced substantially.[495] Research in the 1980s demonstrated that a 50% loss occurred due to anthracnose in unsprayed plots in comparison to yields in plots sprayed four times.[504]

Angular leaf spot caused by *Pseudomonas syringae* is the most widespread bacterial disease of cucurbits. The disease first appears on leaves, later infecting fruits and contaminating seed.[489] Angular leaf spot first appears as small water-soaked lesions on leaves. In moist conditions, bacteria ooze from these spots in tear-like droplets, which dry to a white residue. These spots later turn gray and die. The organism invades the fruit between the cells, advancing directly toward the central portion. As soon as the soft spongy tissue is penetrated, rapid progress is made toward each end. Light brown rotted streaks running the length of the fruit form, which causes the tissue to break down and results in a soft mushy mass.[506][505] The bacteria overwinters in crop residue and can persist for up to 2.5 years in dry leaves.[489] Prior to the use of fungicides, as much as 50% of the cucumbers in north and central Florida were unmarketable because of angular leaf spot.[506] Research demonstrated that uncontrolled angular leaf spot reduced the number of fruit in the field by 26 to 37% and reduced the weight of the remaining fruit by 19 to 38%.[507] Experiments with copper sprays demonstrated total control of the bacteria with leaf spots reduced to zero.[508] Copper applications were found to increase cucumber yield by 49% in a Wisconsin experiment.[509]

*Pythium* species produce brownish, water-soaked lesions that rapidly develop into watery, soft, rotted areas. The rotted areas quickly become covered with a cottony growth. In most cucurbits, the disease usually starts on the portion of the fruit directly in contact with the soil. The most satisfactory chemical control is a systemic fungicide applied in enough water to soak the chemical into the top 0.5 cm of soil.[489]

The pathogen causing *Ulocladium* leaf spot was first recovered from commercial cucumber fields in western New York in 1988.[489] Only cucumber is susceptible. 100% of New York’s cucumber acres are at risk of infection and typically 10 to 50% of the acreage is affected every year.[557] On leaves, lesions become surrounded by a dark brown ring and a circular brown halo. Fruit infections do not normally occur. Fungicide use reduced the average number of leaf lesions by 90%.[510]

Bacterial fruit blotch first appeared in commercial watermelon fields in the United States in 1989, with isolated severe outbreaks in Florida, South Carolina, and Indiana. Up to 50% of marketable fruit were destroyed in some Florida fields while loss of marketable fruit approached 90% in some commercial production fields in Indiana.[511][512] The disease has spread throughout the Southeast.[513] The characteristic symptom of bacterial fruit blotch of watermelon is a dark olive green stain or blotch on the upper surface of the fruit.[489] As the lesions age, the rind ruptures and a white bacterial ooze is exuded.[511] Secondary rotting organisms are responsible for the ultimate decay and collapse of the fruit.[489] Research has shown that a biweekly application of copper is an effective preventive measure.[513]
Downy mildew is incited by a fungus that only infects cucurbit crops. Downy mildew symptoms are almost exclusively confined to leaves. The downy mildew fungus infects only living plants. The tiny spores of the fungus fall on a leaf, germinate, and force food absorbing organs into the leaf. These root-like structures remove nutrients from the plant. Before finally killing the leaf, however, the fungus sends branches back to the surface. These branches produce a new crop of spores which, in turn, may spread the infection to healthy leaves. A new crop of spores can be produced every 4 to 5 days. Downy mildew can defoliate an entire watermelon field in 10 to 14 days. The purple fungal growth expands resulting in the necrosis of progressively larger leaf areas so that, in a few days, the entire leaf is dead. Death of the leaves exposes the fruit to sunscald, which results in reduction in both quality and quantity of marketable yield. In the U.S. downy mildew of cucurbits occurs mainly in production areas near the Atlantic seaboard and the Gulf of Mexico. The overwintering hosts of downy mildew are in southern Florida and southern Mexico. By means of its millions of tiny spores, which are light enough to be carried by air currents, it always returns to the north in the summer as the disease moves up the Atlantic seaboard. Given an ordinarily, dry, 15 mile per hour wind, it is possible for spores to infect plants with dew moistened leaves 150 to 180 miles distant on the evening following their production. In a wet season, the fungus may reach North Carolina by the last week in May. If the season is dry, it may not reach North Carolina until the first week in July. The fungus reaches Massachusetts in early or middle August. In Texas, 50% of the cantaloupe acres are infected every year.

The downy mildew fungus first appeared in the U.S. in 1889 and has appeared every year since. Early research demonstrated that Bordeaux mixture provided control, however, use of Bordeaux was not satisfactory because of damage to the cucurbit crops. Consequently yield losses occurred in 1924: a 40% loss in sections of Delaware and a 55% loss in sections of New York. Cucumber production ceased in Connecticut due to the severity of the disease. An epidemic of downy mildew reduced cantaloupe production in Georgia by 87% in 1923. In the 1920’s downy mildew reduced cucumber production in Florida by 10 to 70%. In North Carolina it was estimated that from 1935 through 1945 downy mildew cut cucumber production by an average of at least 30% each year. Experiments in North Carolina in 1946 demonstrated that the use of copper and fermate or zerlate increased cucumber yields 80 to 100%. In Florida experiments in the 1950s, cucumber yields were four times greater in fungicide-treated plots than in untreated plots. Watermelon yields were increased by 64% by fungicide use in a 2000 experiment in Florida with chlorothalonil for downy mildew control.

Powdery mildews are present when temperatures are relatively high and moisture occurs as heavy dew rather than splashing rain. Powdery mildew occurs every year throughout most cucurbit production areas of the U.S. Powdery mildew has been a serious problem for the cantaloupe industry in practically all production regions in the southwestern U.S. The first sign is a whitish talcum-like powdery growth. Affected leaves can wither and die and finally become dry and brittle. Vines exposed when foliage dies may become withered and whitish from sunburning. The powdery white growth
includes microscopic stalks that produce chains of 2 to 6 spores daily. Over 2 million of these bodies are estimated to occur on a square inch of infected leaf area.[144] When the chains of spores break up, the individual spores can be blown long distances. Such a large number of spores are produced in such a short period that an entire planting can appear white with mildew within a week. Cantaloupe plants infected with powdery mildew are defoliated under severe disease pressure. The loss of a large number of leaves checks the growth of the vine and exposes the maturing melons to the sun.[520]

Mildew reduces yield by decreasing the size or number of fruit or the length of time crops can be harvested.[489] The fungus does not infect the fruit. It is confined to the surfaces of the leaves and stems. Fruits become sunburned, ripen prematurely, are low in soluble solids, and have poor flavor and texture.[518]

In 1925 cantaloupe yield loss due to powdery mildew in the Imperial Valley was estimated at 15% while in 1926 the yield loss was estimated at 25 to 35%.[520] The cantaloupe growers requested aid from government agencies to develop methods to control powdery mildew. Early tests with fungicides were inconclusive due to the use of some materials that were injurious to the plants.[520] In 1936, Powdery Mildew Resistant Cantaloupe No 45 was released cooperatively by USDA and the California Agricultural Experiment Station. In 1938, a new biological race of the powdery mildew organism appeared to which No 45 was highly susceptible.[521] Cantaloupe production was reduced in 1939-1940 by 45%, attributable to infection by powdery mildew.[521] Experiments with fungicides in the 1940s showed a 91 to 94% reduction in mildew and a 35 to 65% increase in yield.[521] In Texas, it has been recently estimated that melon yields in untreated plots were 50% lower than in plots treated with effective fungicides for powdery mildew control.[500] Experiments in Arizona have shown that the percent of the upper side of a leaf infected with powdery mildew was reduced from 43% to 0% with fungicide treatment while infections on the bottom side of leaves was reduced from 78% to 7%.

The fungus *Phytophthora capsici* can strike cucurbit plants at any stage of growth. The pathogen infects seedlings, vines, leaves and fruit. Phytophthora fruit rot was first reported in the U.S. in 1922. It occurred sporadically in most of the U.S., except California, until the 1980s, when the incidence of fruit rot increased notably in Florida, Georgia, Michigan, Illinois and the northeastern states.[489] In 1993, a severe outbreak of *Phytophthora* occurred in Florida leading to plant death and fruit rot in excess of 90% in watermelon fields.[524] In 1998 the disease was widespread in Florida; 41% of cucumber plants surveyed in Manatee county were found to have the disease.[558] In Michigan losses of cucumbers for processing were estimated at 25% on 5,600 acres.[525] Fruit rot can occur from the time of fruit set until harvest. Fruit rot generally starts on the side of the fruit that is in contact with the soil. However, when an infected leaf or vine comes in contact with a fruit, fruit rot will start at the point of contact.[523] Fruit rot can also develop after harvest, during transit, or in storage. There is a delay of at least 48 hours in symptom expression in cucumber following penetration by the fungus.[637] This delay explains why producers in Michigan who harvest seemingly healthy fruit have had entire loads rejected; the fruit becomes infected in the field but symptoms do not become evident until after delivery to the processor or retailer.[637] Fruit rot typically
begins as a water-soaked lesion which expands, eventually covering the fruit with white mold. It is not uncommon for infected fruit to be entirely enveloped in a white fungal growth. The internal flesh of infected cucumbers turns a pale yellow and the fruit rapidly shrinks and rots. Fruit infection progresses rapidly, resulting in complete collapse of the fruit. Root rot symptoms include a sudden, permanent wilt of infected plants without a change in color. Often plants die within a few days of the first symptom. Infected stems collapse quickly and ultimately the roots are completely destroyed. The fungus produces an enzyme that plays a significant role in breaking down the host plant. The number of spores on a single fruit was estimated to be 44 million with the potential to release 840 million spores. Currently, nonchemical alternative practices that effectively manage Phytophthora are not available. Genetic resistance is not available. Crop rotation is recommended but the fungus can survive 5 to 10 years in the soil. On infected acres, cucurbit losses up to 100% are common without an effective fungicide. It is estimated that the combination of mancozeb and dimethomorph preserves 75% of the potential yield. Experiments in Michigan with dimethomorph documented reductions of infected fruit from 20-30% to 5-10%. USDA has estimated that without fungicides the production of cantaloupes, cucumbers and watermelons would decline by 7-100% on a state-by-state basis.

6.18 Garlic

Approximately 84% of the commercial garlic grown in the United States is produced in California. About 65% of California’s garlic production is grown for dehydrated products while the rest is sold fresh.

In 1998, a devastating outbreak of rust disease affected garlic throughout California. During this year, El Nino weather conditions with record-high rainfall and long periods of cool temperatures favored the development of rust. 100% of the plants were infected in some fields. An estimated 54% of California garlic acreage was affected. Statewide garlic yield declined by 50% in 1998.

Garlic rust attacks the foliage of garlic which results in reduced garlic bulb size and yield. Harvested bulb weight can be reduced by 25 to 60%. Diseased bulbs lack the protective dry outer skins. This causes the garlic to shatter when it is harvested mechanically, resulting in loose cloves that are left in the field. Because the disease stops the plants ability to photosynthesize, the crop is undersized. These small bulbs are difficult to harvest as they pass through standard lifting and sorting equipment leaving a high percentage of bulbs in the field. In 1998, California growers were forced to use hand labor to retrieve the bulbs and harvest costs were three to five times higher than usual.

Early symptoms of the disease consist of small yellow to white flecks, streaks, and spots on leaves. As these small lesions expand, the leaf tissue covering them breaks and orange fungal spores become visible as pustules. In severe infestations, the pustules can almost entirely cover the leaves resulting in complete yellowing, wilting, and drying of
the leaf. As the disease progresses, a second darker spore type may also occur on the same leaves resulting in black pustules. The severity of rust was so extreme in 1998 that entire fields turned prematurely yellow and then brown.[218] The spores are windborne and can be spread long distances. Spores survive for long periods of time and are sources of inoculum for infection of successive crops.

While rust on garlic was reported in California as early as 1934, the disease was never considered to be economically important.[218][219] The disease has occurred regularly since 1998, indicating that rust has developed into an annual problem. Untreated plots in 1999 and 2000 yielded 50% lower than did fungicide treated plots.[221]

A variety trial using 34 garlic cultivars was planted in Monterey County, inoculated with rust spores, and evaluated for resistance to rust. Acceptable resistance was not observed in any cultivar. Therefore, genetic resistance is not an option for garlic rust management.[218]

Fungicides that were registered in 1998 for garlic were not effective in controlling garlic rust.[222] As a result, the yield losses described above occurred.

In research trials, tebuconazole and azoxystrobin consistently provided the best control of rust and the highest garlic yields (50% higher than untreated) when applied as protectants before significant rust development.[218] Following several years of emergency registrations of tebuconazole and azoxystrobin (1999-2001), the latter was granted a full registration for use against garlic rust.

The biofungicide, Actigard, has been shown to be completely ineffective against garlic rust with treated plots having no yield improvement over untreated check plots.[218]

Most California acreage has been treated with two applications of a fungicide for rust control since 1999. As a result, garlic yields have remained high.( Figure 4)

Purple blotch is an occasional problem on garlic and chlorothalonil is the preferred treatment.

The State of California estimated that without fungicide use for garlic rust control, garlic yields would be reduced by 50%. [220]

6.19 Grapes

Powdery mildew was first described in eastern North America, but it gained notoriety when it was introduced into European vineyards in 1845 and spread rapidly throughout the continent. At that time there were at least 15 million acres of grapevines in Europe.[426] In the 1840’s, the annual per capita consumption of wine in France was 76 liters.[701] By 1851, powdery mildew had reached every grape-growing country of Europe, causing its maximum damage in France in 1854. In that year it reduced the
French crop of grapes by 80%. Prior to powdery mildew infection, the annual production of wine in France averaged about one billion gallons. By 1854 output had dropped to 200 million gallons. The wine shortage produced by powdery mildew led to a doubling of wine prices in France. Moreover, the quality of the wine from the mildewed grapes was poor. To satisfy the French appetite for alcohol, import duties on colonial spirits (such as rum) were abolished and imports of alcoholic beverages increased. The granting of subsidies for powdery mildew research led to the discovery that sulfur treatments controlled the disease. The use of sulfur became generally widespread in vineyards and by 1858 French wine grape production returned to its 1847 level.

California was largely spared the destructive impact of the mid-19th century powdery mildew epidemics because the state’s grape industry had not yet developed. Grape cultivation became popular in California after sulfur treatments were in widespread use. Agricultural bulletins in California dating to the 1890s describe the powdery mildew treatment schedule still in use today: regular applications of sulfur at 7 to 14 day intervals during the period of vine susceptibility. A 1907 University of California Bulletin reported that powdery mildew was capable of destroying the entire crop in most vineyards during bad infection seasons if it was not controlled.

The fungus that causes grape powdery mildew is an obligate parasite, which means it must grow on grape tissue and will not parasitize any other species of plants. The powdery mildew fungus overwinters inside dormant buds of the grapevine and on dead leaves and bark. An individual infected leaf can release five million spores. Wind borne spores are released in the spring with rainfall, sprinkler irrigation or fog, which spread the disease to neighboring vines. Infected buds give rise to young shoots completely covered by the fungus. As adjacent leaves become infected, new spores are produced on them and the disease spreads rapidly throughout the vineyard. During favorable temperature periods, the time between spore germination and production of spores by the new colony takes only five days. In a trial on infected grape clusters, a mildew infection of 2 to 6% was observed to expand to 20% in seven days, proceed to 70 to 80% in 14 days, and approach complete infection after 20 days. The powdery mildew fungus can infect all green tissues of grapevines. The fungus penetrates only the epidermal cells sending tubular suckers into them to absorb nutrients.

Uncontrolled powdery mildew reduces vine growth and yield and affects grape quality. If berries are infected before they attain full size, epidermal cells are killed and epidermis growth is prevented. As the interior pulp continues to expand, berries split from internal pressure. Split berries either dry up or rot. A net-like pattern of scar tissue may be observed on the surface of infected berries. Such fruit is unmarketable as fresh fruit and wines made from them may have off flavors. Berries infected with the powdery mildew fungus have higher acid levels than uninfected fruits, indicating they did not develop normally. Wines made from mildewed grapes are of a lower quality both as a result of increased acid concentration and as a direct result of the fungus itself producing off-flavors. Taste testers comments regarding wines made from mildewed grapes ranged from “more bitter” to “distinct off tastes.” Wine quality can be affected when as
few as 3% of the berries are diseased. Chains of spores borne on short stalks make up the fungal growth, giving infected tissues a dusty or powdery appearance. The mass of fungal growth on grape skin gives the impression that the grapes are sprinkled with flour. This impression is enhanced by the smell of moldy flour released by the diseased grapes. Many of the attacked grapes dry up and fall. Mildew infections reduce storage life of table grapes. Powdery mildew can also affect the rate of photosynthesis, thus reducing berry sugar content.

A three year experiment in New York resulted in powdery mildew leaf infection at harvest of 68-92% on unsprayed vines. There was a 40% reduction in the size of unsprayed vines in comparison with sprayed vines. This was associated with a 65% reduction in crop on the unsprayed vines. Generally, wineries in New York specify that the fruit they buy not contain more than 3% by weight powdery mildew infected berries.

The mode of action for sulfur in controlling powdery mildew is not clear, but it is believed it acts as a multi-site inhibitor, either through contact or vapors that are toxic to the fungal respiration process. Sulfur is a protectant: it must be applied to vines and fruit before the arrival of mildew spores. Because sulfur washes off with very little rain, it needs to be reapplied immediately after rain or irrigation. Sulfur also needs to be reapplied in order to cover new growth that occurs following an application. Sulfur has been used for 170 years without any signs of resistance. Newer synthetic fungicides are locally systemic, are not washed off by water, and do not need to be applied as often as sulfur. Because it is such an explosive disease, most California growers base their disease control program on prevention and maintaining grapevine coverage from early in the season until berry softening. All fungicides have standard treatment intervals based largely on the residual activity of the material.

It is estimated that powdery mildew is present in virtually all California vineyards every year, and that all grape bearing acres are treated every year. The USDA has estimated that 100% of U.S. grape acreage is infected with the powdery mildew fungus and that yield loss without control would be 45% in eastern states and 80% in western states. Experiments with the industry standard fungicide in California indicates a reduction in powdery mildew incidence from 99% to 0.4% with its use. Sulfur is permitted for use by organic grape growers and it is estimated that organic growers make seven applications annually, which totals 66 pounds of sulfur per acre.

Black rot of grape is an important fungal disease of American origin. It can cause complete crop loss in warm, humid climates. Because of climatic requirements, this disease is especially important in the grape-growing regions of the Midwestern and eastern U.S. Black rot does not appear in California. The black rot fungus is estimated to infect 95% of eastern vineyards with the potential to reduce yields of 85%. An infected berry first appears light brown, and soon the entire berry turns dark brown. Infected berries shrivel, turn hard and black and are called mummies. Although some of the rotted berries drop to the ground, many remain firmly attached in the fruit clusters. The mummy fruits are covered with very small pimple like structures that contain spores. The black rot fungus lives through the winter in a dormant condition in
mummified fruit on the vines and on the ground. With each succeeding rain during the summer, mummies are moistened, the fungus absorbs water, swells, and discharges spores into the air.[612] Once spores have been discharged into the air, they may be picked up by air currents, deposited on vines, germinate and initiate new infections. Vast numbers of spores are liberated from fungal infections in leaves and fruit during the growing season and cause secondary infections to develop.[600] Rain lasting 1 to 3 hours is optimal for dispersal of spores. On berries of Muscadine grapes, symptoms appear as small black scabby lesions. These lesions do not spread or cause decay of maturing berries as on bunch grapes, although infected young berries may drop and mummify. The skin of infected berries often splits at the edge of larger lesions.[600]

Prior to the development of protectant fungicides, a loss of 25% of the grape crop was tolerated. Fruit losses of 70 to 100% were not uncommon in years favorable to the disease.[612] Black rot was primarily the cause of the abandonment of large acreages of grapes in the East in the early 1900s.[613] Grape growers used Bordeaux mixture for black rot control until the development of synthetic chemical fungicides, which were found to be less harmful to the grape plant than Bordeaux. Research showed that ferbam use increased vine growth and berry size.[614] In 1952, Pennsylvania experiments demonstrated that the substitution of ferbam for Bordeaux yielded 1.6 tons more fruit per acre.[615]

Control of black rot is based on properly timed applications of fungicides, which can give almost 100% control. Most fungicides are protectants and must be applied to the vine before rainfall triggers spore release. Research has shown that fungicide applications can reduce the percent berry infection from 95% to less than 1%.[616]

It is generally accepted that grape downy mildew has always been present in the United States. The parasite would generally annihilate a certain portion of the grape harvest in years favorable for disease development. For example, losses of 70-80% of the grape harvest were recorded in North Carolina in 1869.[617] It was the import of American plant material (the intention being to discover or breed vines that were resistant to powdery mildew) that led to the introduction of the downy mildew fungus into Europe. The downy mildew fungus was first noted in southwestern France about 1878, and by 1882 it had spread to all of France.[600] In 1885 a mixture of copper, lime and water was first used to control downy mildew in a vineyard near Bordeaux, France. Until methods were developed to determine treatment dates, French growers suffered enormous losses in numerous “mildew” years, when prolonged periods of rain encouraged development of the parasitic fungus. Such was the case in 1910 and 1915 when the grape harvest in France was reduced by 50%.[617]

Grape downy mildew occurs in regions where it is warm and wet during the vegetative growth period of the vine. It is estimated that the downy mildew fungus infects 75% of eastern U.S. grape acres, with the potential to reduce yield of 40%.[15] Downy mildew was unknown in California until 1995. It became problematic in several San Joaquin vineyards in the wet springs of 1995 and 1998.[608]
The downy mildew causal fungus attacks all green parts of the vine, particularly the leaves. A delicate dense white cottony growth appears. Severely infected leaves generally drop. Such defoliation reduces photosynthesis and subsequent sugar accumulation in fruit. Young berries are highly susceptible, appearing grayish and covered with a downy felt of fungus sporulation when infected.[600] Infected berries drop easily. The fungus overwinters in fallen leaves. Under optimal conditions the time from germination to penetration is less than 90 minutes.[600]

Temperate or cold damp climates favor Botrytis bunch rot, which is especially severe in California’s cooler coastal areas in years with late season rains. Botrytis bunch rot or gray mold is the major rot of ripening grapes in New York state.[618] In Eastern states, Botrytis bunch rot is estimated to infect 30% of the grape acres with potential to reduce yield of 60%.[15] In California, the disease has been found to cause up to 50% of the clusters to be rotted in some vineyards.[621] In California wine grapes, botrytis bunch rot is estimated to have the potential to reduce harvestable yields by 40%.[624] The reduction in yield is associated with the premature drop of bunches from the stalk, or with the loss of juice and the desiccation of berries, which leads to weight loss. In wine grape production the most serious damage is qualitative, resulting from the modified chemical composition of diseased berries. The fungus converts simple sugars to glycerol and gluconic acid and produces enzymes that catalyze the oxidation of phenolic compounds.[600] Wines produced from rotted grapes have off-flavors and are fragile and sensitive to oxidation and bacterial contamination making them unsuitable for aging. Wineries generally will accept up to 2% rot. More than that may result in significant cullage, increased harvesting costs, and lower quality or yield.[622]

Berries infected with Botrytis left in the vineyard at harvest dry and become mummified. The fungus survives in the mummified berry tissue or in hard dark structures called sclerotia. In the spring sclerotia may germinate to produce masses of spores that can be carried long distances by the wind.[619] The spores germinate by extruding a short germ tube. The end of the germ tube presses against the surface of the grape and enlarges into a suction cup. In the center of the suction cup, a small peg-like structure is formed and forces its way through the grape cuticle.[620] Inside the grape the fungus flattens out and forms side growths, which exude enzymes that degrade and soften the intercellular pectic materials that cement grape tissue cell walls together. The fungus spreads between the skin and pulp before entering and degrading the pulp.[620] Severity of disease increases in years when late-season rains occur, but serious yield losses may occur without rain. In these instances, Botrytis infection of grape berries commonly occurs in cultivars with dense canopies or tight berry clusters, including Zinfandel, Chardonnay, and Chenin Blanc varieties.[623] Bunch rot begins when individually infected berries within the cluster turn brown. Often this stage is known as slipskin because the enzymes break down the cutin in the epidermis and it easily slips off the berry.[606] Cracks form in which spores are produced that spread gray mold to other berries in the cluster and to other clusters of grapes. Under optimal conditions, Botrytis can infect a berry, destroy it with chemical enzymes, and begin to produce spores in only three days.[606] Rotting berries attract fruit flies that carry vinegar producing bacteria. These bacteria attack grapes causing a sour-smelling rot. After the grapes crack, other rot producing
microorganisms may infect the berries.

*Botrytis* severely affects stored table grapes because it can infect the grapes in the field and then continue to grow in the berries in storage, producing nests of gray-white fungus. In later stages of decay, the berries begin to lose their juice, which may collect in the bottom of plastic-wrapped grapes.[606]

In certain cultivars and under certain climatic conditions, *Botrytis* infections are beneficial. The grape varieties grown in the French sauterne district (Sauvignon Blanc, Sauternes, Semillion), the Northeastern corner of Hungary (Tokay), and the German Rhine and Mosel Valleys (White Reisling) lend themselves to a favorable concentration of fruit sugar and flavor by the botrytis mold.[619] Though juice yield is drastically reduced by dehydration of the grapes, the low acid, high sugar, high glycerin content of the juice produces very distinctive sweet white wines that sell for a high price. This form of *Botrytis* is known as “noble rot.” However, a particular sequence of climatic events and a specific timing of infection are needed for the development of noble rot. Secondary pathogens frequently spoil its full development.[625]

Many California grape growers practice leaf removal, which aids in controlling *Botrytis* due to reduced canopy density and increased air circulation through the canopy, enhancing the drying of grapes. Leaf removal also improves spray coverage within the canopy.[622] In years of low rainfall, leaf removal alone is sufficient to control *Botrytis*. However, in wetter more humid years, leaf removal is not sufficient and yield gains are achieved with fungicide applications.[626] Research has shown that fungicide applications can reduce *Botrytis* infections from 46% of the clusters infected to 1 to 2%.[618] Leaf removal allows sulfur applications to penetrate into the canopy for superior coverage.

Eutypa dieback, known formerly as “dead arm,” is one of the most destructive diseases of the woody tissues of grapevines. Eutypa survives in diseased wood and produces spores under conditions of high moisture. Spores are carried with winter storms and infection on grapes occurs through pruning wounds. Studies in the Central Valley of California suggest that viable spores may travel up to 100 km. Cankers and foliage symptoms are not evident until two to four years after infection; then vine deterioration continues until the trunk or arm is finally killed. Losses due to this disease result from loss of fruiting wood as vines die back further each year. Symptoms in the wood are characterized by wedge-shaped darkened cankers. Chemical treatments are most effective if applied directly to the pruning wounds immediately after pruning. The fungicide provides a barrier against the invasion of pruning wounds by the disease spores. Eutypa dieback is estimated to infect 65 and 75% of western and eastern grape vineyards, respectively, with the potential of reducing yields of 10-30%.[13][15]

Phomopsis cane and leaf spot is especially destructive in regions where the climate following budbreak keeps grapevines wet with rain. The fungus is estimated to be a problem in 3 to 5% of western grapevine acres and 75% of eastern acres.[13][15] Uncontrolled *Phomopsis* is estimated to reduce grape yields by 15%. The disease results
in spots or lesions on grape leaves, shoots, stems, and canes. Severely infected leaves fall off the vines. Lesions become inactive in the summer, but early fall rains combined with cool weather may reactivate the fungus, resulting in berry and bunch rotting. The fungus overwinters in bark. In spring the fungus erupts through the epidermis of vines and wet spores ooze out and are spread by rain to shoot tips. Fungicides that are applied during the early shoot growth stages protect young shoots adequately if applied protectively before prolonged cool wet weather. Generally, early season fungicide sprays to control black rot and downy mildew also control *Phomopsis*.

The AFBF recently estimated that without fungicides, grape production in California would decline by 97% due to powdery mildew infections, while in New York the reduction would be 37% on Concord grapes largely due to uncontrolled black rot.[30] Grape losses without fungicides in other eastern states are estimated at 85% due to the prevalence of black rot while in other western states the losses are estimated at 80% due to the prevalence of powdery mildew.

### 6.20 Green Beans

Before 1954, green beans were hand harvested from many small, isolated fields. The mechanical bean harvester introduced in the mid 1950s eliminated this system of production.[245] Mechanical harvesters are not able to differentiate between moldy and good beans as the workers had been doing. A switch was also made from growing pole beans to growing bush beans. Air movement around pole beans helped to prevent mold growth. The dense canopy and resulting shaded soil of bush beans provides an excellent environment for fungi. The later trend of planting beans in narrow rows has increased disease problems. Narrow rows provide an environment with longer leaf-wetness periods which enhances disease development.[248]

On green bean acreage in the United States, eleven fungicides are used to control nine major diseases: PCNB and mefenoxam for *Pythium* and rhizoctonia, copper for bacterial blight, chlorothalonil for rust, *Alternaria*, and anthracnose, myclobutanil for rust, sulfur for rust and powdery mildew, and DCNA, benomyl, thiophanate methyl, iprodione, and vinclozolin for control of white mold and gray mold.

Common bacterial blight affects the foliage and pods of green beans. The disease occurs in all states east of the Rocky Mountains.[144] During extended periods of warm humid weather, the disease can be highly destructive. Leaf symptoms initially appear as water-soaked spots. As lesions enlarge and coalesce, the plants appear to be burned.[232] Pod symptoms consist of lesions that are generally circular, slightly sunken, and dark red-brown in color.[232] Under highly humid conditions, pod lesions are frequently covered with bacterial ooze. Losses range from a trace up to 60%.[231] In Florida, in some fields and in some years, close to 100% of green bean plants may be infected.[244] Green bean growers use copper primarily in the management of bacterial blight.[244] Research has shown that copper sprays can reduce the average number of blight lesions by 90%.[246]
Small flecks or tiny water-soaked spots occur on green leaves and pods infected with Alternaria. Lesions often coalesce to form large areas of dead tissue. As plants approach maturity, growth of the fungus on the surface gives infected leaves, pods, and stems a dark, moldy appearance. Beans discolored by Alternaria are unacceptable for processing. The first report of Alternaria causing leaf spots on green beans came from Florida in 1951 when 100 acres were infected or destroyed by the disease. The disease first occurred in New York in the 1970s when 12% of the pods had flecks which rendered them unfit for processing. Research showed that fungicide applications (chlorothalonil) reduced the alternaria pod flecks by 85%.

From 1912 to 1920, anthracnose was considered the most serious bean disease in the U.S. east of the Mississippi River. Many infected seeds rotted in the ground, or if they germinated, the seedlings were destroyed before they emerged. Thousands of baskets of beans in Florida were discarded as worthless after they had been shipped. Today, losses are greatly reduced and outbreaks are infrequent due to the distribution of disease-free seed. For infections occurring during the growing season, regular applications of protectant fungicides are necessary once the disease becomes present in a field. The most characteristic anthracnose symptoms are the black sunken cankers on the pods. Spot centers contain a salmon-colored ooze consisting of millions of spores. The spores are borne in such immense numbers that they are shed in mass and are held together by a gelatinous coating, which prolongs their life and provides nutrients during germination. Water is necessary to dissolve the gelatinous mass and free spores.

The Rhizoctonia pathogen is very common in the soil. An excess of moisture is recognized as the most important condition favoring infection and disease development. Rhizoctonia root rot is most severe on 2-3 week old bean plants. Infected seedlings wilt and collapse from a water-soaked rot of the stem near the soil line. The disease is common in Florida and stand losses of up to 75% have been reported. Research in Florida in the 1950s showed that soil applications of PCNB reduced the incidence of Rhizoctonia-infected plants from 50% to 3%. In the 1960s Rhizoctonia became a problem in mid-Atlantic states with reductions in bean stands of 25 to 50%. Research demonstrated that PCNB reduced the incidence of Rhizoctonia from 75% infected to 6%.

The Pythium fungus survives in the soil for long periods of time. The spores remain quiescent until stimulated to germinate by an exogenous source of nutrients, such as seed or root exudates. Spores swim in the water films in soils permitting the fungus to reach surface water, in which it may move a greater distance. Spores formed in infected bean tissue are released to the soil when the tissue is further decayed. Infected seedlings rot in the soil (damping off). Young plants can become infected also. The fungus spreads up and down the stem. On the eastern shore of Maryland pythium blight has been reported to destroy up to 30% of the bean plants in a field. In furrow applications of mefenoxam are very effective in controlling Pythium.

Powdery mildew is caused by a fungus that first appears as a white powdery mold on
small areas of upper leaf surfaces. The entire leaf and plant may be covered by cottony fungal growth.[232] Yield losses caused by powdery mildew result from leaf, stem, and pod injury; affected pods are not salable.[144] Powdery mildew occurs every year on beans in the southeastern U.S.[254] In the 1920s growers in the Southeast incurred heavy losses from the disease. Research in the 1930s demonstrated that sulfur applications reduced the incidence of powdery mildew infected plants from 87% to 2%. [254] During a severe powdery mildew epidemic in Florida in 1988, sulfur applications reduced bean infections from 80% to 5% or less[255]

Rust of beans is caused by a fungus that grows only on bean plants.[257] Bean rust most frequently affects leaves but can also affect pods, stems, and all other above ground green portions of the plant.[232] The common sign of bean rust is the reddish brown circular pustule on leaves or pods which ruptures to release thousands of spores. The spores give a rusty color to anything that touches them; even covering the ground with a faint brownish dust.[144] Each spore that invades the leaf can produce a new pustule in about 6 or 7 days. As many as 2,000 pustules can form on one leaf.[144] When leaves become thoroughly rusted, they fall from the plant; during an epidemic, most vines are defoliated and severe yield reductions occur.[144] Rust reached epidemic proportions in Tennessee in 1972 and has since been an annual problem there.[258] Research in Tennessee demonstrated that bean yield following chlorothalonil treatments applied during rust epidemics was four times greater than that in untreated, infected check plots. In the 1930s rust sometimes completely destroyed the green bean crop in Virginia.[266] In 1936, rust was so destructive in Florida that hundreds of acres were plowed under without being harvested. The 1936 loss in Florida was estimated at 40-80% of the crop in Palm Beach and Broward counties.[266] Losses would have been as great the following year had growers not sprayed the bean fields with sulfur to control rust.[266] Experiments conducted in Florida found that uncontrolled rust reduced bean yield by 60% in comparison to the fungicide-treated plots.[256]

White mold is caused by a fungus: Sclerotinia sclerotiorum. White mold can affect all parts of the green bean plant in the field and can continue to develop and cause disease among green beans in transit and in storage.[232] Infection of bean plants is most common during the blossoming period when the surfaces of fully mature flowers are covered with exuded nutrients that are needed by fungal spores to germinate and infect the plant. Stems, leaves and pods in contact with colonized blossoms are then invaded by the pathogen. A mucilaginous, sticky material is discharged that serves to cement accompanying fungal spores to the bean plant. Spores deposited on bean tissue need not infect immediately, but can survive for a considerable period of time. White mold lesions on pods and leaves rapidly increase in size and become slimy. Under moist conditions the lesions develop a white, cottony fungal growth.[232] The white mold fungus produces hard black structures called sclerotia that survive in the soil during the winter. Sclerotia can survive in the soil for five or more years. When suitable environmental conditions return, the sclerotia develop small trumpet-shaped mushroom–like structures in the soil. Each of these structures can discharge more than two million spores during its five to ten day life.[261]
During white mold epidemics in New York, infections in some fields were as high as 68%.[233] Detection of more than 2% diseased pods in a truckload may result in rejection of the whole load at a processing plant.[259]

Before the development of effective fungicides, white mold caused significant economic losses. Bean production was discontinued in some highly desirable locations because of repeated heavy losses from white mold.[260] Green bean growers typically harvested their crops about four days earlier than the scheduled harvest date to hedge against the risk of total crop failure.[260] This early harvest resulted in about a 30% reduction in yield. Tests with fungicides in the early 1970s showed that white mold incidence could be reduced to zero with fungicide treatments in comparison to 100% incidence in untreated areas.[260] Fungicides can provide effective control for 20 days after the last spray application.[262]

Gray mold is caused by the fungus *Botrytis cinerea*. The fungus first colonizes blossoms. Once established in blossoms, the fungus quickly invades the pod or any plant organ the flower falls onto.[232] On pods the lesions are at first water-soaked, later turning grayish brown and sunken. As diseased tissues dry out, spores are formed in a gray-brown powdery mass. In marketed green beans, latent and unnoticed infections established in the field give rise to mats of profuse dirty white fungal growth, a condition known as nesting.[232] Gray mold is a perennial problem in Oregon’s Willamette Valley.[264] The disease results in a watery soft rot of the bean pods, affecting the quality of the crop and results in direct losses to growers and processors.[264] Economic loss is due primarily to pod rot which reduces quality and increases processing costs.[263] Growers are docked for damaged pods that are graded out at the processing plant. In the 1940s bean losses in the Northwest to gray mold were as high as 50% in many fields.[265]

A recent report from the University of Wisconsin concluded that if fungicides were not used for white and gray mold and growers substituted nonchemical management practices (crop rotation, irrigation management) an average yield loss of 40% and reduction in quality of 30% would result in most years.[231]

### 6.21 Hazelnuts

Hazelnuts have been commercially produced in Oregon since the early 1900’s, where they were called “filberts” rather than hazelnuts. In 1981, Oregon “filbert” growers began referring to their crop as hazelnuts to be consistent with the common name for the commodity throughout the world.[211] Oregon’s hazelnut orchards, concentrated in the Willamette Valley, account for 99% of United States hazelnut production and 5% of world production. Hazelnuts reach bearing age in four years and have a life expectancy of over 50 years.

Eastern filbert blight (EFB) is a destructive disease of European hazelnut trees that is present only in North America.[39] The disease is known as eastern filbert blight because a closely related shrub, called American hazelnut, harbors the fungus in the
In the 1920’s, growers tried to start a hazelnut industry in New York by bringing in cultivated European hazelnut trees. Filbert blight destroyed the trees.

Eastern filbert blight is different from most orchard diseases in that the infections are systemic and eventually lethal to the tree.

The potential threat of EFB to Oregon orchards was first recognized in 1922 when the Oregon Department of Agriculture established a quarantine to prevent importation of hazelnut trees from east of the Rocky Mountains.[213]

In 1970, eastern filbert blight was found in a hazelnut orchard in Southwest Washington. This was the first report of the disease west of the Rocky Mountains. In 1974, the disease was first detected in Oregon, infecting an orchard in Columbia county. By 1979, EFB had spread to 49 Washington orchards, most of which were removed because of the disease. In 1986, EFB was found Oregon’s Willamette Valley, infecting a major commercial production area in Clackamas County.[213] The disease has been spreading south in the Valley since that time.

Nearly all orchards within a 10-mile radius of this site of disease detection in Oregon were destroyed because of the disease by 1993.[214]

In hazelnuts, eastern filbert blight can cause cankers that expand perennially at rates up to 1 meter per year. Expanding cankers girdle branches and limbs resulting in canopy dieback and death of trees in 5 to 12 years if diseased limbs are not removed.[216]

The fungus penetrates young hazelnut epidermal cells directly. Once infections are established within a shoot, the fungus colonizes phloem and xylem layers without obvious symptoms of disease.[216] The first distinguishing symptoms of disease occur 12 to 14 months after initial infection. Early symptoms of the blight are black, pimple like pustules on the tree’s twigs. Tiny football-shaped bumps appear about 14 months after the spores infect the tree.[212] These bumps (called stromata) turn black and occur in straight rows along the branch.

The pustules must be wet to expel spores. When wet, the spores are pushed out of the pustule and shot into the air. One tree can produce billions of spores, which are 1/100 the size of pollen grains. Each stromata contains approximately 3 million spores. Spores are released in sticky white ooze. Wind-driven rain and splashing droplets spread the spores to young developing shoots. Spores are ejected into the air all winter long but cannot infect hazelnuts until the spring. The fungus can gain access to immature shoots only when the bud scales begin to separate and leaf tips become visible. As vegetative stems mature, they become resistant to infection. New stromata develop each year as the canker expands. Dieback occurs when expanding cankers girdle branches and limbs.[216] The fungus eventually spreads to the trunk, killing the tree.

Rain traps have been used to capture spores and measure the volume of their release. Spore captures average between 100,000 and 10,000,000 spores per square meter of trap
Farmers can achieve limited control of EFB with pruning techniques, but all infected trees eventually die. The best way to rid an orchard of the disease is to remove and burn the infected branches and then spray healthy trees with fungicides in the spring. The infected wood must be destroyed to prevent future infection of living trees as the fungus can continue to produce spores from cankers sitting in brush piles.

Research demonstrated that hazelnut trees are susceptible to infection by EFB spores after vegetative buds break dormancy in March through leaf emergence and shoot elongation, the latter of which extends into May. Consequently, an effective chemical control program for EFB must protect healthy trees from new infections over a period of susceptibility that may extend up to two months. Research demonstrated that complete control of the disease could be attained with 4-5 fungicide applications on a 2 week schedule lasting into early May.

Numerous experiments have been conducted over the past 30 years to evaluate the effectiveness of fungicides against EFB. Currently used fungicides include both protectants (chlorothalonil) and eradicants (tebuconazole). Protectants must be applied to young hazelnut growth before spores of the EFB fungus are deposited on new tissue. These products work by inhibiting spores of the fungus from germinating or kill the developing germ tubes before they have a chance to get inside young shoots. New plant tissue that develops after application is generally unprotected. This means multiple protectant fungicide applications are needed for effective control. Eradicants have the ability to kill infecting EFB growth even after the fungus makes its way into the leaf tissue. Eradicants can move into young green shoot tissue and cure an already established infection. This property is time limited, usually only effective within a few days after spore germination and the beginning of the infection cycle.

Because EFB kills trees slowly, the yield benefits obtained from fungicidal sprays are not realized until at least three or four years after initial applications.

The State of Oregon has estimated that without the use of fungicides Eastern Filbert Blight would destroy 50% of state’s hazelnut production within 10 years and 75% of production within twenty years.

6.22 Hops

All beer may contain two types of hops. Bittering hops are used to make beer more bitter. Aroma hops are used for flavoring. Hop plants produce annual climbing stems from a perennial crown and rootstock (rhizomes). The stem may grow to 25 feet during a single growing season. Hop plants are remarkably vigorous, sometimes growing 6 to 12 inches in 24 hours. Once established the hop rootstock will produce indefinitely although they are typically managed under a trellis system for a 10-year period. The commercial hop is a female plant; hop cones are its flowers. Mature hop cones contain numerous
lupulin glands, which contain the important brewing constituents of alpha-acids, beta-acids, and essential oils.\[642\]

The United States is the second largest producer of hops in the world after Germany. Over half of the U.S. crop is exported. Seventy-one percent of the hops used by U.S. breweries are domestic while the remaining 29% are imported. At the U.S. hopping rate of 0.21 pound per 31 gallon barrel, the U.S. produces enough hops annually to flavor approximately 9 billion gallons of beer.\[641\]

It is estimated that nearly 100% of U.S. hop acreage is treated with fungicides. Mefenoxam, fosetyl-Al, copper and cymoxanil were targeted at downy mildew while myclobutanil, sulfur, trifloxystrobin and tebuconazole were targeted at powdery mildew in 2002.

The center of domestic hop production in the mid 1800s was New York. More than 21 million pounds of hops were produced in New York in 1879.\[644\] Problems with powdery mildew practically wiped out the production of hops in New York in about 1909. The region revived around 1920 with the discovery of sulfur-based fungicides only to be devastated again in the late 1920s by downy mildew.\[641\] Without management of downy mildew with fungicides, New York growers lost about one-third of their production in an average year.\[643\] Because downy mildew is more severe in areas of high rainfall, hop production in the U.S. shifted to the more arid regions in the Pacific Northwest. In spite of the less favorable environment, the downy mildew fungus has become established in hop yards in the northwest. Downy mildew first appeared in western Washington and Oregon in 1929 and quickly spread. By 1933, annual mildew damage was estimated at $1 million and Bordeaux mixture was used for its control. Copper lime dusts supplanted Bordeaux.

In 1997, despite years of quarantine efforts, hop powdery mildew was reported for the first time in the Pacific Northwest.

The downy mildew fungus overwinters in infected hop crowns and first appears in the spring as an infected shoot, commonly known as a primary “spike” produced by infected crowns. The under surface of the leaves becomes blackened with millions of spores which spread the disease to other shoots. During one outbreak, in which 2% of the crowns were infected, the disease spread to infect 75% of the plants in the yard.\[647\] If an outbreak of downy mildew occurs in late June or July when hop plants are in full bloom, many of the flowers will become infected and die, causing no cones to develop.\[642\] Infected blooms become brown, shrivel and subsequently fall to the ground. If infection occurs later in cone development, a portion of the cone becomes blackened and is unacceptable to brewers.\[642\] Hop vines often die late in the season due to downy mildew infection. The fungal infection may eventually cause the crown to die.

The incidence of downy mildew ranged from 0 to 3% of hills with spikes in replicated field plots treated with metalaxyl and from 25 to 80% in adjacent plots, and yards not
The weight of fresh hops was 38% higher in the treated plots.

Powdery mildew diseases affect a large number of plant species; however, each strain of powdery mildew fungus attacks only one or a few plant species. The fungus causing hop powdery mildew is known as *Podosphaera macularis* and only attacks hops. The fungus overwinters in infected buds on rhizomes. In the spring, the infected buds produce mildew covered shoots. Spores from the infected shoots disperse causing secondary infections, which can move up the plant, infecting hop flowers and cones. The fungus appears as small circular, powdery colonies. More colonies appear and may eventually turn the entire vine white. Infections on flowers result in the death of the infected flower so that no cone is produced. Infection of young cones inhibits cone growth and results in cones developing into small, hard knobs. Infected cones are stunted, malformed, and mature rapidly, leading to cone shatter.

Yield and quality losses from powdery mildew occur in several ways. Powdery mildew infections on cones cause the crop to prematurely ripen, which substantially reduces yield because the last two weeks are a critical time for cone weight and alpha acid to increase in the hop cones. Powdery mildew infections lead to browning of hop cones, which changes the aroma of the cones making them unusable for brewers. The discolored cones are rejected for processing by brewing companies. Unlike many crops where growers can send poor quality raw product to processing or livestock feeding channels, there are no optional markets for hops rejected by brewers because of low quality. These hops are simply destroyed.

In 1997 a newly-introduced hop variety, Symphony, was seriously impacted by powdery mildew in Washington. Over 2,500 acres of Symphony were removed following the 1997 crop year and replaced with other cultivars thought to be resistant to the fungus. However, the new cultivars showed moderate infection levels in 1998 and by 1999 these varieties were considered highly susceptible. No fungicides were available to control powdery mildew when it was first reported in Washington in 1997, or Oregon and Idaho in 1998. A series of section 18 emergency registrations have been approved for the use of preventive fungicide applications to protect hops from the fungal infection. Growers make an average of ten sprays due to the rapid growth of the hop plant which results in new tissue requiring a preventive treatment. The hop industry estimates that without fungicide application 69% yield and quality losses would occur, with losses reaching 100% in many varieties.

The usefulness of biological control agents such as AQ10 and Serenade is limited due to costs, which are 50% higher than the cost of fungicides, adverse impacts on spider mite predator populations, marginal efficacy, and processing concerns with Serenade-treated hops. The product is applied at such a high rate that considerable white residue remains on the plants. The white residue is not acceptable to brewers. No biological control agents have been developed for hop downy mildew.

Although mechanical and chemical pruning provide some disease suppression, they are insufficient without the assistance of synthetic fungicides. While some varietal resistance
has been developed by hop breeders, no resistant varieties include the commercially necessary agronomic and brewing characteristics.

Without fungicide use, it is estimated that marketable hop yields would decline on average by 69%. Marketable yield losses would be 100% for certain varieties, such as ‘Willamette’, which are the primary aroma hop of several beers.

6.23 Hot Peppers

New Mexico is the number one hot pepper producing state, accounting for two-thirds of United States production New Mexico hot pepper growers use copper to control leaf diseases (bacterial spot & cercospora leaf spot) and myclobutanil to control powdery mildew

Epidemics of bacterial spot may occur if overhead irrigation or heavy rainfall make environmental conditions favorable for the disease. All above-ground plant parts can be infected with bacteria. Infected crop debris and weeds are sources of infection. The disease is spread by splashing water, wind or plant-to-plant contact. Infected leaves develop water soaked lesions and eventually fall off.[593] Crop losses result from both yield reduction due to defoliation and severe spotting of fruit, which renders the peppers unfit for market. Sunscald due to defoliation and fruit decay resulting from secondary invasion by opportunistic organisms through bacterial spot lesions are additional fruit problems associated with bacterial spot. After establishment, bacterial spot fruit lesions turn brown. As the spots enlarge, the epidermis ruptures and curls back exposing a raised brown rough wart-like surface. These pod lesions reduce the market value of peppers.[311] Research has demonstrated that copper sprays increased the number of marketable fruit from infected fields by 50%.[312]

Cercospora leaf spot, a fungal disease, causes relatively circular spots on pepper plant leaves. A clear to yellowish halo may surround the spot. The diseased spots dry and drop out, leaving holes in the leaf. Severely infected leaves turn yellow and drop from the plant.[593] Fruit are not infected by Cercospora leaf spot. The disease is also known as frogeye leaf spot because the spots resemble frog eyes. Defoliation often results in stunted and irregularly shaped fruit.[144] Even one lesion on a leaf is enough to cause the leaf to drop. In 7 to 10 days, infected tissue dies, new spores are formed and are disseminated by wind and rain.

Powdery mildew is a relatively new fungal disease of hot peppers in New Mexico, although the disease now occurs every year.[593] Severe infections early in the season cause heavy yield losses. Uncontrolled powdery mildew has caused yield losses of up to 50 to 60% on peppers.[316] As the infection develops, white powdery growth covers the lower surfaces of leaves. The edges of infected leaves may roll upward and infected leaves may drop prematurely, exposing fruit to the sun so that they may be susceptible to sunscald.[310] No resistance to powdery mildew has been seen in the hot pepper varieties grown in New Mexico.[633]
6.24 Kiwi

Kiwifruit plants are woody twining vines that are trellised on a single wire. Kiwifruit originated in China. California grown kiwifruit was first harvested in 1970. Due to California’s late fall kiwifruit harvest, fresh fruit is available in the winter months. With proper storage practices, kiwifruit may be stored for up to 8 months, from October to May.[57]

Kiwifruit are kept in storage for long periods, particularly since controlled-atmosphere (CA) storage has extended the post-harvest life of kiwifruit from a few weeks to several (usually 5 or 6) months. Extended storage helps orderly marketing of fruit but results in losses due to gray mold decay.[51]

Botrytis gray mold storage decay, caused by the fungus *Botrytis cinera*, is the most important disease of kiwifruit. Even though the disease does not manifest itself in California vineyards, postharvest decay is a direct result of infections that occur in the vineyard but remain latent. *B. cinera* is a ubiquitous fungus, widely distributed in kiwifruit orchards growing and sporulating on dead flowers and dead leaf tissue. The spores are readily liberated and are available to contaminate fruit. The pathogen requires moisture for spore germination and infection after which the fungus remains quiescent. Spores are redistributed, through fruit rubbing together, during the picking and packing process.[53] During long-term cold storage, kiwifruit become physiologically susceptible to the pathogen, which then invades the fruit tissues.[51]

Because transit times are frequently as long as 30 days and conditions during transport to distant export markets can be poor, there is a potential for considerable loss of fruit quality and for gray mold decay during shipment. Incidences of gray mold decay as high as 20% have been reported from California. Additional costs result from resorting and re-packing, which considerably increase the price of the final marketable product.[51]

In addition to losses due to decay of fruit, losses are incurred when the fruit infected by Botrytis gray mold produces ethylene in cold storage; the presence of only a few fruit with gray mold accelerates softening of nearby healthy fruit and increases sorting costs.[52]

The fungus grows slowly at low temperatures. Botrytis rots are first obvious 4-6 weeks after fruit has entered cold storage.[53] Diseased flesh is glassy and watersoaked. When a kiwifruit is harvested it is broken from its stalk, leaving a picking wound on the fruit. Botrytis stem-end rot results from contamination of the picking wound by fungal spores.

The first indication of *B. cinerea* in kiwifruit storage is extreme softness localized at the fruit’s stem end. Generally, the fungus is first visible on the fruit surface as small white tufts of the fungus mycelium (threadlike structures). In time, the enlarging diseased area becomes covered with white mycelium of the fungus. In the high humidity of storage rooms, mycelia from rotted fruit develop abundantly and may extend out an inch or more from disease lesions. Mycelia touching a nearby sound fruit readily penetrate the skin to
establish infection. Newly diseased fruit develop aerial mycelia that, in turn, infect other fruit. The result is a constantly enlarging “nest” of diseased fruit.[54]

Fruit quality/condition checks are done on orders of kiwifruit before shipping to assure the absence of gray mold. In an average Botrytis rot year, repacking of product starts around January first.[167] In certain grower lots and during bad Botrytis rot years, repacking will start in early December. If repacking is started too early, and fruit is not shipped out within a timely period after repack, a second round of repacking will be required. The initial repack effort may remove the primary Botrytis rots, but secondary rots or “nesting” will continue to develop.[167] In high infection years, repacking in early December has yielded losses of over 25%. Individual grower losses due to Botrytis rot have been observed and reported as high as 50%.[167] Although 50% loss is an extreme, during high-disease years it is very common for crop loss due to gray mold to run 10-25%.[167] Repacking involves opening up each container, going through all of the fruit, culling the diseased fruit, and then refilling the acceptable boxes for shipment.[167]

Vinclozolin was registered for control of Botrytis rot of kiwifruit in 1983. Fungicide trials in several locations in California showed a significant decrease in the amount of gray mold decay of kiwifruits in storage following four applications of vinclozolin, two of which were made at or near bloom and two near harvest.[51]

Although excellent control has been achieved with 4 sprays, the cost of the fungicide sprays generally exceeded the value of the loss from fruit rot.[51][54] In the early 1990’s research demonstrated that it is possible to predict the levels of postharvest decay of kiwifruit in commercial storage by measuring the incidence of colonization of the fruit by the fungal pathogen in the orchard. Growers can collect samples of kiwifruits 4 months after fruit set, send them to a laboratory to determine colonization by *B. cinera*. Based on infection levels of the samples (low, medium, or high) growers can predict the expected levels (low, moderate, of high) of gray mold decay and decide on the cost-effectiveness of preharvest fungicide sprays. Research demonstrated that preharvest sprays are not warranted when predicted gray mold infection levels are below 6%. With this method, 1-2 sprays of vinclozolin significantly reduced postharvest gray mold incidence.[51]

In 1999, BASF, the registrant of vinclozolin, informed the kiwi industry of its intention to cancel its use on kiwi. The last day for legal use of vinclozolin on kiwi was January 30, 2004.

During crop years 1999/2000 and 2000/2001, research was conducted on a number of reduced risk fungicides and several biological controls. The performance of the biological control agents (Serenade and Aspire) was demonstrated to be non-efficacious.[167] Furthermore, both Aspire and Serenade left a whitish residue on the fruit that was not acceptable to growers or packers.[167] The fungicide fenhexamid significantly reduced the incidence of gray mold decay in comparison to the untreated control.[167]
The State of California requested an emergency exemption for the use of fenhexamid on kiwi which was granted by the USEPA.

Gray mold is a real problem for organic kiwi growers. There are no fungicidal materials for control of gray mold approved for use in organic production systems. To compensate for this, organic growers often have to make sure their fruit is shipped early and not stored long periods. Losses from repacking can be very high in years with heavy Botrytis infections.

USDA has estimated that without fungicide use, 25-40% of California kiwi fruit would be lost to fruit rot.[13] The State of California has recently estimated that once the disease develops, an average of 18% of the crop is lost. [167]

Without fungicides, it is estimated that kiwis lost to rots in storage would amount to 25% of total production on the 33% of the crop acreage treated with fungicides. The increased cost of repacking kiwi due to increased rots is assumed to be offset by the elimination in the cost of fungicides and their application.

6.25 Lettuce

Copper, manebl, fosetyl-al and azoxystrobin are used for downy mildew control. Manebl also controls anthracnose and septoria leaf spot while azoxystrobin controls powdery mildew and anthracnose. Vinclozolin, DCNA, and iprodione are targeted at lettuce drop and bottom rot. Mefenoxam provides control of damping off of seedlings while sulfur is used for powdery mildew control.

Downy mildew is a common fungus in most lettuce growing regions, especially during cool, moist weather. Spores can be blown long distances. Under favorable conditions, downy mildew is a very explosive disease, capable of appearing at high incidence in a field overnight. When spores land on lettuce foliage, they germinate and can penetrate the lettuce leaf within three hours. Lettuce is susceptible at all growth stages to the downy mildew pathogen. Following penetration and establishment in the leaf, fruiting stalks grow through the leaves and branch repeatedly producing several spores on each tip, resulting a whitish mat of millions of spores on each plant.[144] Affected tissues turn brown. The fungus can penetrate to leaves internal to the wrapper leaves. Florida experiments indicated that when downy mildew is not controlled, downy mildew symptoms appeared on the innermost wrapper leaf at the time of harvest.[569] Relatively low levels of infection can downgrade a crop, cause significant trimming losses at harvest and promote decay by bacterial organisms during postharvest transport and storage. During transit, lesions become soft and slimy as secondary decay organisms gain entrance through the tissues infected with the downy mildew fungus.[567] High levels of disease can render a crop unmarketable.[560] At least 13 different genes within the downy mildew genome have been found to govern lettuce plants’ abilities to resist the pathogen, making the task of successfully breeding for long-term resistance extremely difficult. The pathogen can combine these 13 genes into 8,000 separate races.[568]
Prior to 1960, downy mildew was a significant disease on head lettuce in California. It was brought under control with resistance from wild lettuce species in the cultivar Calmar. New races of the fungus appeared in 1976 and overcame the resistance. In 1989 another set of new resistant lettuce cultivars were introduced, but by 1992 control slipped again.[568] Downy mildew problems prevented the widespread growing of lettuce in South Florida until the development of the EBDC fungicides in the 1950s.[570] Research in the 1950s demonstrated that, for an investment of $25/acre in fungicides, a return of $486/acre was achieved. Research in California with maneb has consistently shown large reductions in the incidence of downy mildew. The average number of lesions per 10 plants is reduced from 187 in the untreated rows to 1 with the maneb treatment.[571] Research in Florida indicated that 98% of lettuce heads were marketable after fosetyl-al and an EBDC treatments, whereas only 25% of the heads from untreated plots were marketable.[572]

In the U.S., lettuce drop (or Leaf Drop in Arizona) occurs in all major lettuce-producing states. The fungus causing lettuce drop is present in all parts of the Salinas Valley.[562] Disease incidence can be as high as 70% in some fields.[560] The rot usually begins on the stem near the soil surface and a water-soaked area appears; it can spread downward until roots decay and can spread upward until leaf bases are affected.[144] The pathogen rapidly ascends the stalk, killing the leaves in succession until it reaches the heart of the lettuce plant. Layers of collapsed leaves lie flat on the soil surface after infection. Inner leaves are invaded completely by the fungus, which reduces the head to a wet, slimy mass.[144] The entire plant may collapse in less than two days.[560] Lettuce drop is caused by two species of Sclerotinia, S. minor and S. sclerotiorum. Both species survive in the soil for up to 8-10 years.[560] The fungus shows itself as a white cottony mass on the underside of each infected leaf, in which small black hard growths are formed. As many as 110 of these black growths have been picked out of a single diseased lettuce plant.[561] After detaching from the lettuce plant and embedding in the soil, the black bodies put out small mushroom-like fruiting bodies that release millions of spores, which are dispersed by wind throughout the field and into adjacent fields.[560] A single mushroom-like structure can eject 310 million spores.[561] When these spores land on lettuce plants, infection occurs within two days and symptoms appear in four days. The fungus produces oxalic acid that reduces the viability of lettuce cells.[635]

Application of fungicides after thinning and/or 30-40 days before harvest significantly reduces the incidence of lettuce drop.[560] The fungicides form a chemical barrier between the soil and the developing leaf canopy of the lettuce plant. With this chemical barrier in place, the bottom leaves and stem of each lettuce plant are protected from colonization by the germinating spores of the pathogen.[563] The available fungicides for lettuce drop control (DCNA, iprodione, vinclozolin) provide about 50% control of the incidence of the disease.[564][565] Current losses in California following treatment with fungicides are estimated at 5-20%.[566] In an experiment in New Jersey, iprodione reduced the percent incidence of drop from 47% to 6%.[576]

California lettuce growers used deep plowing as a management tool for lettuce drop.
throughout the 1980s and into the 1990s. The principle is that deep plowing moves the fungus from the top four inches of soil to lower depths, where it is unable to cause lettuce drop infections. However, growers noted a general lack of control in the deep plowed fields. Research demonstrated that deep plowing uniformly redistributed the fungus in the soil and ultimately resulted in higher disease incidence.

Powdery mildew is primarily a problem in the desert production areas of California and Arizona during the winter months and most often infects mature plants. The fungi infect the plant tissue by sending hollow tubes from a spore on the leaf surface into the plant to suck out nutrients. Talcum-like growth of the fungus usually appears first on the upper surfaces of older leaves. These whitened areas enlarge, often until they cover the entire surface of the leaf. The affected foliage gradually loses its luster and tends to curl, so that finally some of the leaves turn yellow, then brown, and die. In severely-infected fields a definite mushroom-like odor may be noted. Powdery mildew spores are carried long distances by wind. Application of sulfur at the first appearance of the disease and at subsequent intervals controls powdery mildew.

Bottom rot is caused by the soilborne fungus *Rhizoctonia solani*. The disease is found wherever lettuce is grown. Bottom rot symptoms usually appear when the heads are nearly mature. Penetration occurs when lower leaves touch the soil and when plants are large enough to result in moisture retention between the leaves and the damp earth. Rust-colored slightly sunken lesions occur. While the lesions are forming, drops of amber liquid oozes from the lettuce tissue. If the disease is checked before the head is destroyed, lesions dry out and become sunken brown spots. The fungus works upward and inward passing from leaf to leaf until the entire lettuce head is decomposed. Such a head becomes a slimy brown mass that soon dries out and turns black as it collapses. During warm wet periods losses may approach 70% of the maturing crop. Research with vinclozolin demonstrated a 36% increase in the weight of marketable lettuce in plots treated for bottom rot.

Also referred to as shot-hole, ring spot and rust, lettuce anthracnose is worldwide in distribution. Losses to anthracnose are considerable, especially during cool and moist periods. Bottom rot led to the abandonment of some lettuce fields in the Santa Maria area of California in the spring of 1998 and 2001 due to heavy rains. Anthracnose symptoms first appear as small, tan, water-soaked spots on the outermost foliage. As the lesions mature, the centers fall out, giving the disease its characteristic shot-hole appearance. Tissues damaged by the pathogen are invaded by secondary organisms, particularly soft-rot bacteria, which can rapidly decay entire heads. Azoxystrobin applications provide effective control of the disease.

The pathogen causing septoria leaf spot lives from season to season in seed, in debris from diseased plants and in weed hosts. Seed production in California and Arizona, where septoria is absent, has almost eliminated it from the United States. The disease generally occurs sporadically and becomes severe only under conditions of prolonged high humidity or rainy weather. On severely infected leaves, lesions may coalesce resulting in extensive wilting and drying.
Pythium and Rhizoctonia spp. cause damping off of lettuce seedlings. Damping off occurs wherever lettuce is grown. The disease is expressed as seed decay, death of seedlings, and infection of the roots or stems of young plants. Pythium spp. survive in soil as thick-walled spores, which are stimulated to germinate by nutrients such as those in lettuce seeds and root exudates.\cite{560} Soil treatments with mefenoxam prevent infections and protect seedlings when they are most susceptible to damping off.

Without fungicides, California lettuce production would decline by an estimated 47%, while the reduction in Arizona is projected to be 45%.\cite{170}\cite{30} Estimated losses without fungicides are projected to be higher in the east: 70% in Florida and 50% in New Jersey.\cite{10} Losses without fungicides in Colorado are projected at 20%.\cite{14}

### 6.26 Mint

Mint was introduced into the United States in colonial times and was first grown in Massachusetts. Mint growing gradually moved westward with the early settlers. The soils and climate of southern Michigan and northern Indiana were so well suited to mint culture that these areas became the major mint-producing areas of the U.S. by about 1920. In 1924, Verticillium wilt was diagnosed in a Michigan field. This disease gradually spread and became more severe eventually causing many Midwestern growers to abandon mint production. After 1950, peppermint acreage increased greatly in Oregon and Washington until the Northwest became the largest mint-producing area of the U.S. and the worlds leading producers of peppermint and spearmint oil.\cite{117} Wilt has become a problem in the Northwest as well and it is estimated that more than 50% of all Pacific Northwest mint fields are infected with wilt.\cite{111} There is no chemical control for Verticillium wilt.

Mint is a perennial crop that produces for six to seven years. The mint plants are grown for the essential oils that they produce in specialized glands on the leaves and stems. The oil is readily recovered by steam distillation of the harvested hay. Peppermint and spearmint oils are widely used to flavor chewing gum, pharmaceuticals, and toothpaste.

Fungicides are used to control two diseases that affect mint: rust (northwest and Midwest) and powdery mildew (northwest). Nine percent of Midwestern mint acres are treated with fungicides while 4 to 39% of mint acres in northwestern states are treated.

Rusted mint leaves turn brown and drop off the plants. Defoliation can be severe late in the season, with accompanying loss in oil yields.\cite{109} Moderate to severe rust infections weaken the plants, reducing winter survival. In 1949, rust in peppermint was found in quantities significant to reduce yields as much as 25% in Columbia County, north of the Willamette Valley. In 1950, Columbia county experienced a 35% yield reduction because of rust.\cite{110} Infected mint stands can suffer as much as a 70% yield loss due to rust.\cite{111} The pathogen overwinters on mint stubble and wild, escaped mint plants and
can be systemic in the plant. It is easily spread over long distances by wind. For many years Willamette Valley growers controlled rust with propane flaming, however, flaming damages mint plants and is often not completely effective because of furrows. Flaming also reduces populations of beneficial pest predators.

Powdery mildew is seldom a problem on peppermint but can be very destructive on Scotch spearmint. Mildew develops rapidly and extensively during prolonged periods of warm, humid, and cloudy weather and with dense mint foliage. Damage by mildew is most serious during the month prior to harvest because it is the leaves that develop at this time that are harvested for oil. High levels of mildew infection earlier in the season can affect mint growth and development. In fungicide trial test plots, the amount of defoliation due to powdery mildew has reached 50%. Fungicide use to control powdery mildew has increased mint yield by 27%.

Fungicides are estimated to increase mint yield by 25% on treated acres.

6.27 Nectarines (included in Section 6.31 Peaches)

6.28 Onions

Onions are affected by four primary foliage diseases that warrant the use of fungicides: purple blotch, botrytis leaf blight, stemphylium blight, and downy mildew. Fungicides are also used to control a bulb rot (botrytis neck rot) and two seedling diseases (damping off and smut).

Purple blotch symptoms first appear on leaves or stalks as small water-soaked lesions that quickly develop white centers. As the lesions enlarge they become brown to purple. After a few large lesions form on a leaf, they may coalesce and girdle the leaf, causing all tissues beyond the lesion to die. Thousands of spores are produced from each lesion, each capable of germinating and causing additional lesion spots. Bulbs may also be attacked. Typically, the fungus invades through the neck. The decay is at first semi-watery and is conspicuous because of the yellow to red color associated with it. The fungus secretes an abundance of a pigment that diffuses through the scale tissues in advance of the fungus. The affected tissue gradually turns to a wine red color. The decayed tissue eventually becomes dark brown to black. The fungus requires the presence of rain or persistent dew for infection. Purple blotch occurrence can be predicted by counting the number of hours that free moisture is present on the leaf surface. When 10 to 12 continuous leaf wetness hours occur, purple blotch will develop.

In the early 1950s, purple blotch became a major problem in Colorado onion fields with estimated losses generally ranging from 30 to 50%, although sometimes reaching as high as 100%. Yield increase of 20-35% or more have been recorded in fields sprayed with fungicides
used to control purple blotch.[23]

Individual *Stemphylium* leaf blight lesions are small, light yellow to brown colored, and water-soaked. These soon develop into elongated spindle-shaped spots.[16] Spots often coalesce into extended patches, blighting the leaves. The pathogen normally invades dead and dying onion tissue including purple blotch and downy mildew lesions. Rainy weather increases populations dramatically.[16] *Stemphylium* leaf blight was first identified in Georgia in 1998 and has since become the most widespread and destructive disease of Vidalia onions.[126]

*Botrytis* spores land on onion leaves and, in the presence of moisture, germinate and produce enzymes that kill leaf tissue. *Botrytis* leaf blight causes early death of the leaves and undersized mature bulbs. Masses of gray *Botrytis* spores develop during periods of high humidity. Lesions may merge or become so numerous that they kill the leaf. The leaf blight lesion begins with a white necrotic center surrounded by a light green halo. The fungus develops rapidly and causes blighting of leaves. Severely affected onion fields may take on a blighted appearance with most leaves dead and dried out. Losses in yield because of smaller bulb sizes occur when leaves are killed.[16]

Botrytis leaf blight kills foliage and spreads so rapidly that growers gave it the name “blast.” Extended periods of leaf wetness are necessary for infection. The fungus survives in the soil and on plant refuse. During hot humid weather, spores are released and land on wet onion foliage where they germinate and enter the plant. The first shower of *Botrytis* spores lodge on wet onion foliage and as they germinate a few cells are killed beneath each spore. These areas are very susceptible to the next shower of spores. The second shower of spores germinate and penetrate the tissue rapidly. The tops of an entire field may be killed within seven days.[28]

Bulb size is reduced 50% or more by botrytis leaf blight depending on the duration of the disease and the degree of bulb development before the onset of the disease.

In 1958-59 in the Lower Rio Grande Valley, botrytis leaf blight was unusually severe; losses from the disease were estimated at 50 to 75% through reduction of expected yields from approximately 10,000 acres.[19]

Botrytis leaf blight is estimated to infest 100% of Texas and New York onion fields.[25][27]

Botrytis neck rot occurs primarily on bulbs in storage although infection originates in the field. Onions develop a semi-watery decay beginning in the neck area, which gradually moves downward through the entire bulb. Onion tissue softens and becomes water-soaked and translucent. The fungus overwinters on rotting bulbs or in the soil. Plants are infected during the growing season but remain without symptoms.[16]

The causal fungus of botrytis neck rot is common in most onion growing soils and spreads via wind-blown spores.[131] A compact gray mold develops on the surface of
the onion. Later, round hard areas containing spores develop in a solid crust around the neck. Several months often elapse before the entire bulb is destroyed.[27] During some seasons, growers have lost 50% or more of their crop because of this disease.[23]

During extended periods of cool humid weather, downy mildew can be highly destructive to onions, causing losses in both yield and quality. The disease commonly starts in spots in a field and spreads to surrounding areas. The spores can be blown long distances. The first evidence of disease is a fine, furry grayish white to purple growth on the surface of older leaves. Affected leaves gradually become pale green and later yellow, and diseased parts of the plant fold over and collapse. Lesions on the stem weaken the stalk so that it breaks. The fungus may destroy the onion foliage in a field almost completely.[16] If humidity remains high, the furry fungus growth becomes widespread and an epidemic occurs. The pathogen overwinters in diseased foliage. Downy mildew is identified by its fluffy gray growth on the underside of leaves. The downy mildew fungus can survive in the soil for 4-5 years. Downy mildew causes progressive leaf death, which in turn reduces bulb size. All of the foliage in an onion field may be destroyed by downy mildew during a growing season.[29]

The injury to and killing of onion leaves caused by downy mildew infections reduces the yield and quality of onion bulbs. Bulbs from a mildew-infected crop are spongier in character and of poorer keeping quality than those from an uninfected crop.[21] Downy mildew infections slowly kill individual leaves, stunt plant growth, and occasionally kill the entire plant.[20]

Onion fields with no symptoms of downy mildew on a Friday have been observed to incur 40% infection over a weekend.[22] An uncontrolled downy mildew infection is estimated to reduce onion yield by 65%.[25]

Most early attempts to control onion downy mildew were unsuccessful.[20] Experiments in 1917 with bordeaux mixture did not produce any significant increase in yield.[20] In the 1920s after three years of spraying onions with bordeaux mixture for leaf blight and downy mildew, the conclusion was reached that although the incidence of leaf blight could be delayed somewhat, yield increase did not justify recommending the practice.[24] The current IPM recommendation is to apply fungicides at the first sign of downy mildew.[131]

The smut fungus survives in the soil for many years in its spore form. In the presence of a new onion crop the spores germinate.[27] The smut fungus penetrates developing seedlings. As the disease progresses, pustules develop which later rupture, releasing spores that re-contaminate the soil. A single lesion may cover an entire leaf causing it to curve downward. Most infected seedlings die within 3-5 weeks after germination.[27] If bulbs form, they become covered with blackish lesions. Smut infections appear as grayish dark streaks and blisters on new leaves and bulbs become filled with dusty black fungal spores. Cold, damp spring weather delays plant emergence and prolongs the period of susceptibility to smut infection. Onions are susceptible to infections by the smut fungus shortly after germination and remain susceptible through the development of
one true leaf. If the weather is warm and dry, the onions germinate rapidly and can escape infection.[23] The smut fungus is estimated to be present in 100% of New York’s onion fields and can cause up to 40% yield loss in severely affected fields.[27] For many years, onion growers drenched the soil with formaldehyde to control smut.[21]

*Pythium* fungi are common in agricultural soils. Spores swim through water films in soil, germinate, infect, and colonize the roots of onion plants causing them to decay.[16] Onion seeds that are infected with *Pythium* fungi become water-soaked and mushy and quickly decompose. Roots of infected onion seedlings initially exhibit a grayish, water-soaked appearance. Infected seedlings quickly collapse and die.[16] Yield losses can be as high as 30% in severely affected fields.[27]

The first experiments with synthetic fungicides for leaf blight and mildew were conducted in the early 1950s. The average yield from 24 onion blocks treated with nabam or zineb was 26% higher than that from untreated check blocks.[26]

Onions are a long season crop, requiring 5-6 months from planting to harvest; therefore fungicides can be applied 10-15 times to insure adequate disease protection.[17] Protective fungicides are applied to onion leaves to inhibit the growth of spore germ tubes.

Foliar applications of azoxystrobin, chlorothalonil, copper, iprodione, mancozeb, manebl, and mefenoxam are used to manage the foliar diseases (downy mildew, botrytis leaf blight, purple blotch, and stemphylium blight) and botrytis neck rot. Seedling diseases are controlled by soil applications of mancozeb (smut) and mefenoxam (damping off).

A recent three year experiment in Georgia resulted in a $38 return for every $1 spent on fungicides [126]

### 6.29 Papaya

Papaya is a short-lived perennial that grows to 30 feet high. Papaya plants are grown from seed. Most production in Hawaii is on porous lava ground. On lava lands, additional virgin soil is brought in and placed in the planting hole. Papaya trees bear fruit throughout the year. The plants will continue to bear for many years, but yields usually decline and picking becomes difficult as the trees age. In commercial production, fields are usually replanted or abandoned after three years.

In March 1940, a fruit-rotting disease of papaya was observed for the first time in Hawaii. At one location, the disease caused the death of 25% of the papaya planting.[149] The disease was identified as *Phytophthora palmivora*.

Infected young fruit on trees usually show water-soaked lesions that exude milky latex.[150] The disease continues to develop and causes the infected fruit to shrivel and become mummified before falling to the ground where further shriveling takes place.
Mummified fruits ultimately become brownish-black, light in weight and stone-like in texture.[155] These mummified fruits are a reservoir for the fungus and constitute a source of inoculum for further infection. The infection on mature fruits is first characterized by the oozing of latex. Later a whitish fungal mass develops containing large quantities of spores.[154] The lesion quickly expands to engulf the entire fruit. As the fungus grows through the tissue, it causes death of the host cells and water-soaking rot.[155]

Older portions of stems also become infected and develop horizontal water-soaked lesions. The infected area enlarges and weakens the stems, causing the plant to break off in strong wind.[150] When the stem is completely girdled, the top of the plant wilts and eventually dies.[155] Rain and wind are the two major factors in the epidemiology of phytophthora fruit rot of papaya. Rain splash is needed for the liberation of spores into the atmosphere from the surface of infected fruit and for the projection of soil inoculum into the air. Wind is required for the dispersal of airborne inoculum. The pathogen produces abundant spores on the surface of infected fruit, causing disease outbreaks in the orchard and in orchards nearby.[150]

Epidemics occur during periods of extremely wet weather. In Hawaii, rainfall during such periods may average several inches per week for several weeks.[151]

In January-April of 1979, Hawaii papaya growers experienced probably the most destructive epidemic of phytophthora fruit rot of papaya since the beginning of production. Nearly 200,000 trees or the equivalent of 325 acres of planted papaya, were destroyed by the disease during this period.[152] Further, trees that became infected were killed during the following months, ultimately resulting in a loss of about 20% of the industry’s producing trees.[152]

Phytophthora fruit rot of papaya can be controlled with preventive applications of the fungicide mancozeb.[150] Nearly complete destruction of orchards not receiving fungicide sprays can occur within 2-3 months.[151]

Research in Hawaii demonstrated that regular applications of an EBDC fungicide (maneb) reduced the incidence of fruit rots by 94%.[153] Although copper controlled the disease, it resulted in fruit russetting.[153]

It has been estimated that 100% of Hawaii’s papaya acreage is sprayed with mancozeb to control Phytophthora.[13] Mancozeb is used at the rate of 2.25 Lbs. AI/A. Papaya growers usually spray from 16-24 times at intervals of 7-14 days when weather conditions are conducive for disease increase.[13] Without the use of a fungicide, it is estimated that there would be a 100% yield loss of Hawaiian papaya.[13]
6.30 Parsley

Parsley is planted by seed and remains in the field eight months or more. Several cuttings are harvested from each crop. Parsley is grown for both the fresh and processed markets.

Parsley septoria blight or leaf spot is a disease that affects the foliage of parsley causing angular, brown leaf spots that can develop on both new and old foliage and on parsley stems. Plant tissue left in the field after each cutting provides spores that can infect new growth. Under severe disease pressure, these spots coalesce and cause a blighting effect. Although this pathogen does not affect yield, the increased costs to harvest and remove diseased plants can often render the crop or cutting unharvestable.[168]

The pathogen does not kill plants. The disease decreases the quality and marketability of the crop due to the blighting effect on the foliage. The blighting effect is magnified with each successive parsley cutting. Second and third cuts are often abandoned due to disease damage and resulting poor quality parsley.[168]

The planting of infested seeds introduces the fungus to new production areas.[83] The disease does not survive in the soil for long periods. Since parsley is a long season crop and can be in the field eight months or more, the likelihood of maintaining inoculum in the field year round is high. Splashing water from winter rains and sprinkler irrigation will infect plants and spread the disease.

Septoria leaf spot had been observed in California fields for several years in the early 1990s. In the early years the disease pressure was not severe. However, beginning in the mid 1990s the disease was especially problematic rendering some fields unharvestable. Reports from fresh market and dehydrated parsley processors indicate that the 1998 crop experienced heavy disease pressure. In Monterey County, the disease infected 60 to 80% of fields, and an average of 16% of the fields were discarded after the first or second cuttings.[168]

There are no effective fungicides currently registered for control of septoria leaf spot of parsley. Copper is registered for use on parsley but shows no activity against septoria.[168]

Research with azoxystrobin demonstrated 95% control of septoria in moderate infections and 75% control of severe infections.[168]

The State of California has been granted emergency exemptions for the use of azoxystrobin on parsley annually since 1999. The state’s petition estimated that parsley yields would be lowered 33% without the use of azoxystrobin.[168]
6.31 Peaches (also includes 6.12 Cherries, 6.27 Nectarines, and 6.36 Plums & Prunes)

California accounts for 99% of the plum/prune and nectarine production while cherry and peach production are much more dispersed, with 99% of production being accounted for by seven and twenty-seven states respectively.

Fungicides are targeted at eleven major diseases: brown rot, rust, russet scab, peach scab, shot hole, leaf curl, powdery mildew, bacterial spot, bacterial canker, green fruit rot, and cherry leaf spot.

Two fungi cause brown rot of stone fruit in the U.S.: Monilinia fructicola is the most common while Monilinia laxa is less common but still important in some western states. It has been estimated that the brown rot fungi infect 100% of U.S. acres of cherries, peaches, and nectarines with the potential to reduce yield of 75%.[13][15] The fungi usually cause blossom blight in the spring which develops into twig blight and provides inoculum for fruit infections. Spores are produced on the infected blossoms and twigs for months after the initial infection. If the weather is damp, these spores germinate on the surface of the fruit, sending out threads that penetrate the skin and start the rot. The enzymes produced by the fungi break down cell walls of the fruit making the fruit soft and brown. Brown rot develops on fruit at the end of the season and after harvest during storage and transport to market.[354] The first symptom of the disease on the fruit is the appearance of a tiny brown speck that rapidly develops into a large spot beneath which the flesh is deeply invaded.[352] The invasion of the fruit by the fungus is quite rapid and the entire fruit may become completely rotten and soft within a few days as a result of a rapidly spreading firm brown decay.[322] Ash-gray masses of millions of spores appear on the fruit surface which serves in turn to spread the disease further.[355] The invaded fruit that remains attached to the tree slowly becomes dried and shriveled, forming a mummy which overwinters and becomes a source of spores the following year. Research has shown that 40,000 spores could be washed from each mummy.[353] Another source of spores in an orchard are mummies that fall to the orchard floor. The fungus develops a mass of tissue within the rotten fruit and, about bloom time, sends up urn like growths containing spores which are released. When the blossoms are attacked they quickly turn brown and die. The fungus may spread by contact to adjacent fruit.

Brown rot caused substantial fruit losses before the development of fungicides. In 1852, it was estimated that in Georgia among most peach varieties growers expected to lose from 50 to 75% of the crop.[356] Many early peach cultivars were supplanted by less-flavored varieties because the older varieties were so susceptible to brown rot.[352] In 1904, it was reported that in Maryland early season peaches were a complete loss due to the disease.[357] In addition to the field losses, losses in transit frequently amounted to 25% of shipments.[352] Beginning in 1908, the losses in the orchard and in transit were greatly reduced by the application of fungicides to the developing fruit.[352] With the development of a finely powdered sulfur about 1912, southeastern peach growers began widespread spraying to control brown rot.[352] The average yield on the sprayed trees
was two and one half times higher than on the unsprayed trees.[362] While unsprayed trees showed 95% brown rot incidence, sprayed orchards demonstrated 25% incidence.[362] Peach losses due to brown rot averaged 13% in Georgia in the 1920s.[308] In a six-year study (1922-1928), it was determined that one-third of all rail shipments of peaches had 5% or more of the fruit affected by brown rot.[372] The development of brown rot in transit was directly linked to spray programs in the orchard: sound fruit from unsprayed trees developed approximately four times more brown rot in storage than sound fruit in storage from sprayed trees. Research in the early 1920s determined that orchard spraying reduced the incidence of brown rot on cherries in storage from 24% to 6% while the rot incidence of stored prunes was reduced from 28% to 7%.[483] In the dryer growing areas of the Pacific Northwest and California, brown rot is a serious problem only once in four or five years.[358] In these areas losses were severe when unexpected rains occurred during harvest time. In California, there were instances of complete destruction of ripe peach fruit following a single rain.[322] In 1965 brown rot destroyed 200,000 tons of California peaches as a result of a single unexpected rain.[478] In 1923, 25% of Oregon’s prune crop was destroyed.[358] In 1936, in Washington, the disease caused a reduction in yield in prunes of 60%, 40% in tart cherries, and 90% in sweet cherries.[359] Standard practice in the West became spraying during the critical periods for infection.[363] The use of sulfur decreased fruit losses substantially, but unacceptable crop losses continued.[361] Approximately 24 million pounds of sulfur per year were used in the U.S. during the 1940s for brown rot control.[307] More effective brown rot control became possible after introduction of captan in the 1950s and benomyl in the 1970s.[361] Recent experiments have shown reductions in the incidence of brown rot due to fungicide use; nectarines (75% to 1%), peaches (69% to 1%, and cherries (65% to 0.3%).[479][480][481] Losses to brown rot have been virtually eliminated.[360] Protection of fruit from infection can be achieved only if fungicides are applied before moisture occurs on the fruit.[323]

On peach and nectarine the rust fungus infects leaves and fruit. On plum and prune, the fungus infects only the leaves. In California, rust occurs on prune every year.[322] In some years, early infection results in heavy defoliation which may reduce tree vigor or productivity in subsequent years. In the coastal areas of California, rust commonly causes heavy defoliation of plum and prune.[323] On peach fruit, circular spots 2 to 3 mm in diameter develop and become water-soaked, green, and sunken as the fruit ripens.[322] Leaf symptoms begin to develop as pale yellowish green spots. The spots become bright yellow. Advanced stages of the disease are expressed by leaves, which turn brown and roll upward. On such leaves the underside is covered with rust pustules.[324] The disease is important before harvest because defoliation hinders the mechanical harvest operation.[324] In California, in most years, most of the leaves on prune trees have rust pustules by the time of natural leaf fall.[324] In recent years rust has been most severe on plums in Sutter and Yuba counties of the Sacramento Valley.[325] The fungus overwinters on twigs. In spring the fungus forms twig cankers filled with rusty-brown spores. In prune orchards with a history of severe rust infections, sulfur is applied in early and mid summer to control leaf infections and delay defoliation until after harvest.[326] In recent years rust has been most severe on nectarines in Sutter and Yuba counties of the Sacramento Valley.[328] Direct crop loss can occur from fruit
infections that develop as sunken greenish lesions as nectarines ripen. The tissue beneath the spots is tough and leathery and clings tightly to adjacent healthy tissue. This characteristic makes the fruit difficult to peel. The rust fungus caused immense losses of peaches in 1926 due to rust lesions which rendered the fruit unfit for canning. Many growers lost their entire crop of certain varieties. Research in the 1930s demonstrated that sulfur sprays killed 90% of the rust spores and reduced the percent of germinating spores from 28% to 1%. Several major outbreaks have occurred on peaches in California since the 1920s. The last major epidemics in 1995 and 1998 resulted in considerable economic losses to growers in northern California. Canneries rejected deformed fruit with more than three lesions and this fruit was diverted to less profitable uses (e.g. juice or animal feed).

Russet scab of prune was first observed in the early 1930s in California. Since then it has caused severe losses in years with heavy rains during the blossom period. The cause of russet scab is unknown. Symptoms are first apparent on green fruit. Wax on the affected surface of the fruit is lacking and the area, first shiny, later develops a brown russet with a netted pattern. Localized drying of the fruit which leads to the characteristic scabby areas could be attributed to an excessive loss of water due to the lack of the wax layer. Severe russetting leads to downgrading of the fruit by causing minute cracks and the development of whitish corky areas on the dehydrated fruit. Such fruit are subsequently used for prune juice. High levels of dehydrated fruit with russet scab result in increased costs for manually sorting these fruit. There have been instances where 50% of the fruit showed severe russet scab after drying. The only detrimental effect on the quality of fruit is in appearance. Treatment with chlorothalonil or captan at full bloom reduces or prevents the development of russet scab, apparently because they stimulate wax formation on the fruit surface. Research has shown that fungicide applications can reduce the incidence of russet scab on dehydrated fruit from 23-25% to 1%.

Shot hole disease can be severe on peach and nectarine. An effective spray program with Bordeaux mixture to control shot hole was developed in 1906 in California. This program prevented the abandonment of peach growing in some parts of California. Severe outbreaks of shot hole were experienced on peaches in California following the years of economic depression in the 1930s when dormant sprays were not applied and the winter phase of the disease was allowed to progress. Symptoms on peach and nectarine are similar. During the wet winter months, the fungus infects and kills dormant buds and twigs. Numerous buds are either attacked directly or invaded by the fungus spreading from a lesion that develops on a twig adjacent to the base of the bud. In the latter case, the diseased buds are noticeable because of the gum they exude. Leaf lesions appear as small spots; as the leaf grows the spots become separated from healthy tissue and the infected area drops away, producing the shot hole effect. On fruit, lesions develop mostly on the upper side and eventually become corky and rough. In the early 1900s shot hole became destructive to peaches in California and a yearly spraying to control the disease became standard orchard practice. Experiments in the 1930s showed that applications of Bordeaux mixture would reduce shot hole on peaches from 80% to 9%. Control of shot hole disease on peach and nectarine
requires protection of dormant buds, shoots, and fruit. A single dormant spray of copper applied before autumn rains provides protection throughout the winter.[322] Ziram, ferbam, and captan have proven as effective as copper.[323] Research in the 1950s showed that ziram reduced the incidence of shot hole lesions from 216/tree to 2/tree.[477] Shot hole is estimated to infest 25% of California’s peach and nectarine acres.[13]

Peach leaf curl, caused by *taphrina deformans*, has been reported worldwide from most production areas of peach and nectarine.[322] Yellow to reddish areas appear on young developing leaves in the spring. These areas progressively thicken and pucker causing the leaf to curl. Chlorophyll soon completely disappears.[336] Puckered areas are crisp and may develop a white coating of spores which are forcibly discharged. Infected leaves fall prematurely resulting in reduced tree vigor, fruit quality, and yield.[334] In California, it is estimated that uncontrolled leaf curl would reduce peach and nectarine production by 25-30%.[13] Fruit infections are characterized by irregular, raised, wrinkled, reddish lesions. In the early 1900s it was estimated that peach yields were reduced by 15% in areas of the U.S. that did not practice spraying for the disease.[336] In California, experiments at the University of California in 1890 showed that winter applications of copper sprays were effective in controlling leaf curl.[336] Copper applications were shown to reduce the incidence of leaf infection from 42% to 0.1%.[333] In the eastern U.S. fungicides for leaf curl are usually applied in early spring before the leaf buds begin to open. In California leaf curl has been controlled by a single spray applied after leaf fall in autumn. Copper fungicides have been commonly used for control of leaf curl because of their long lasting residual quality.[323] In California it is estimated that 90% of peach and nectarine acres are treated for leaf curl.[335][327] Experiments in the 1950s demonstrated that ferbam and ziram reduced leaf curl incidence 99%.[476] A recent experiment demonstrated that fungicide applications reduced the incidence of infected shoots from 35% to 3%.[482] Leaf curl is estimated to infest 50% of eastern peach acres with the potential of reducing yield by 25% if left uncontrolled.[15]

Peach scab is of minor economic importance on peaches in the upper Midwest and in the West, but of major economic importance in the southeastern U.S.[334] In the eastern U.S., it is estimated that peach scab is present in 100% of peach acreage and would lower peach yield by 50% if not controlled.[15] Losses of greater than 80% have been recorded routinely in field trials in South Carolina. Peach scab reduces the appearance, quality, and market value of the fruit and, when severe, it creates cracks which serve as entry points for the brown rot fungus. Peach scab develops as circular black velvety spots on fruit. Fruit lesions tend to be concentrated at the stem end. Lesions on fruit may be followed by cracking of fruit. The fungus overwinters in twig lesions.[334] On fruit, penetration occurs directly at the base of hairs on peach.[322] Protective cork layers form under the infected areas and compartmentalize the fungus after the superficial host cells die.[322] The direct injury is superficial involving at most only a few layers of cells which typically become separated from the adjacent normal tissues by the formation of cork.[337] As the peaches grow rapidly just prior to maturity, the corked areas can not expand as readily as normal tissues and the growth stresses causes cracking of the cork layers. In 1910, it was reported that scabby peaches were bringing in 25% less in the
market than the same fruit free of scab.[338] Research in the early 1900s demonstrated that fungicide sprays would reduce the incidence of scabbed peaches from 87% to 8%.[337] Research in the 1950s demonstrated that fungicide sprays could reduce the incidence of peach scab from 98% to 2%.[475] Scab on peaches is controlled entirely by fungicide sprays.[322] Usually 4-5 sprays are needed.

In the U.S., bacterial spot has been reported in all peach-growing areas east of the Rocky Mountains; it has not been reported in the Pacific Coast area. Bacterial spot is more common and most severe in areas where peaches are grown in light, sandy soils and where the environment is humid or moist and warm.[322] Bacterial spot is estimated to be present in 50% of the peach acres in Eastern states with the potential to reduce yields by 20% on average if not controlled.[15] Plums are frequently more severely damaged than peaches. In the early 1900s, plums were grown very extensively in central Georgia only to be removed due to the problem of bacterial spot.[340] Bacterial spot occurs on leaves, twigs, and fruit. Leaf symptoms are first visible as angular grayish water-soaked lesions. The leaf tissues killed by the organism dry out and tear away from the healthy tissue and a very ragged appearance results which leads to premature leaf drop.[340] In a 1923 experiment the rate of leaf drop was ascertained from trees infected with bacterial spot over a 9 day period.[340] Single trees lost as many as 1,848 leaves during the 9 day period. Fruit infected early in the growing season develop unsightly blemishes on the skin. Pits or cracks extend into the flesh resulting in depressed brown to black lesions.[334] When the bacteria invade the tissues and fill the large intercellular cavities, the supply of oxygen is cut off, and the cells are killed by asphyxiation.[340] As the cell walls collapse the bacteria enter the cells. When the enlarged spots have begun to sink in, the bacteria reach the surface as numerous, tiny, rounded pale yellow gum-like masses.[339] Eventually the epidermis is ruptured by the upward pressure of the mass of bacteria which then oozes through the rupture. Secondary spread of bacteria oozing from the fruit lesions can occur in repeated cycles during warm wet weather.[334] The pathogen overwinters in twig lesions and buds. In spring, the bacteria are spread by wind-blown rain to leaves, twigs and fruit. On highly susceptible cultivars, the entire fruit crop can be lost in years when bacterial spot is severe.[322] The injury to peach fruit ranges from 33 to 76% on average trees.[340] Early season applications of copper are beneficial in preventing leaf infections, reducing overwintering inoculum, and inhibiting bacterial movement from overwintering cankers to newly emerging leaves and fruit.[322] In one experiment the combination of copper plus ziram in an early season spray reduced the incidence of bacterial spot at harvest from 31% to 9%.[341] Oxytetracycline is also used to control bacterial spot in peaches.

Cherry, nectarine, peach, and plum are susceptible to powdery mildew. In orchards, the largest economic losses usually result from fruit infections. Powdery mildew is particularly troublesome in the semiarid areas of California and the Pacific Northwest.[322] Mildew is favored by dry summers with intermittent periods of high humidity and moisture. It has been estimated that powdery mildew infects 100% of western cherry acreage with the potential of reducing yield an average of 25%.[13] Fifteen percent of western peach/nectarine acres are estimated to be infected with a potential reduction in yield of 5-10%.[13] In eastern cherry and peach orchards the
potential yield reduction from powdery mildew is estimated at 10% with 2% and 5% of the acres infested respectively.[15] Powdery mildew of tart cherry in Michigan is of sporadic importance. However, in years favorable for disease development, the problem can be quite severe. In 2003, in unsprayed orchards, the incidence of powdery mildew was 46%.[474] Powdery mildew is common on foliage, which serve as sources of inoculum for fruit infections. Many Washington state cherry producers have reported the rejection of entire crops destined for fresh-market sale due to fruit infection by powdery mildew.[342] The principal economic loss from powdery mildew of peach in California results from fruit infection which, if severe, renders the fruit unacceptable to canneries.[343] Early symptoms on foliage include a fine netlike growth. As infections progress, large numbers of spores produced on the leaf surface result in a white, mealy appearance.[322] Infected fruit are deformed with slightly depressed or raised areas. A scabby condition remains visible on nectarine, peach, and plum even at maturity.[323] On sweet cherries, infected areas on fruit are most noticeable near harvest as the fungus on fruit surfaces develops a web-like growth.[323] Mildew on green cherries appears as shiny, red blotches often with white spores in the center.[334] In Washington where the disease is particularly severe, rain showers during fruit development trigger outbreaks of the disease on fruit.[322] Mildew symptoms typically appear on ripe fruit within several days of a rain shower.[344] The recommendation is to protect immature fruit with fungicides whenever the fungus has been detected in the orchard air.[345] Research in Washington state showed that fungicide treatments reduced the number of powdery mildew colonies on cherry trees by 96%.[346] In California experiments, it was shown that, at harvest time, the amount of marketable peaches in sprayed trees was about 20% greater than on unsprayed trees.[347] The severity of damage from bacterial canker caused by *pseudomonas syringae* varies from subtle, almost undetectable effects to rapid death of trees in orchards.[322] Bacterial canker is estimated to infect 50% of eastern peach acreage (mostly in Southeastern states) with the potential to reduce yield 5-10% on the average.[15][334] An average of 2-5% of California’s plum, nectarine, and peach acres are treated for bacterial cankers.[325][327][335] Bacterial canker is particularly severe in Northwest cherry orchards. In Oregon, when conditions have been favorable for the disease, tree losses of 75% have been observed in young orchards.[350] Under normal conditions, losses between 10 and 20% are not uncommon.[350] Bacterial canker was first identified as a problem in Michigan in 1968 when the disease reached epidemic proportions on sweet cherry followed by a severe epidemic on tart cherries in 1976.[348][349] Cankers develop on twigs, branches, and trunks of peaches, nectarines, plums, and cherries. These cankers may exude large amounts of gum.[323] If girdled by a canker, the diseased branch or trunk dies within weeks.[322] Leaf and fruit infections sometimes appear, especially on cherry. The bacteria ooze out of the branch and leaf infections when they are moist and then are spread around the tree by rain. When the leaves dry the bacteria are left on the leaf surfaces. It has been estimated that there as many as 500,000 bacteria per leaf on an unsprayed cherry tree leaf by October.[350] Lesions on green cherry fruit are brown with a margin of wet or water-soaked tissue. The affected tissues collapse leaving deep black depressions in the fruit.[334] Chemical control of bacterial canker is based primarily on protective copper sprays in autumn and spring. These
Green fruit rot, also known as blossom rot or jacket rot, is a disease of stone fruit blossoms and immature fruit. Both *Botrytis cinera* and *Sclerotinia sclerotiorum* cause green fruit rot. The disease has been found on cherries, peaches, nectarines, and plums in California. In eastern orchards green fruit rot is estimated to infect 20% of the cherry acres and 5% of the peach acres with the potential to reduce yields of 5-10%. The first recorded epidemic of green fruit rot in California cherries was in 1983, when a survey of orchards indicated that 17 to 92% of the blossoms had symptoms. The fungus attacks all floral parts and entire clusters of blossoms may be blighted. Infection of developing fruit by the fungus begins at points where the fruit contacts the diseased floral parts. Blossom rot is most common in coastal areas of California where protracted rains occur during the tree blossoming period. The infection of fruit is often walled off resulting in scarring or deformity in mature fruits. In cherries, green fruit rot results in a smooth round lesion. Mummified fruit from previous seasons are present in the soil and spore-releasing structures grow on the mummies. Spores are released from these structures by a change in relative humidity. Research has shown that fruit and blossom infection can be prevented by spraying protective fungicides during the full-bloom period of the tree.

Leaf spot is the most important fungal disease of cherry in the eastern U.S. where it is estimated to infect 80% of the orchards with the potential to reduce yields by 100% if not controlled. The disease is caused by a fungus known as *Coccomyces hiemalis* which lives over the winter in the old leaves on the ground. The first infection of new foliage in the early summer is caused by spores which are discharged from these old leaves. After the fungus develops on the new leaves, more spores are produced and they may cause further spread of the disease. On leaves, infections appear as small reddish to purple spots. The individual spots never become large, but they may be so numerous that they coalesce and thus kill large areas of the leaf. The appearance of numerous spots on the leaf is usually followed by rapid yellowing and dropping. Infection of fruit is rare. However, fruit on severely defoliated trees fail to mature normally, and they are light colored, low in soluble solids, soft, and watery. Defoliation from leaf spot reduces the number of flower buds and subsequent fruit set for the following year. Defoliated trees are less cold hardy and may be killed by low temperatures in winter. In southern Pennsylvania early defoliation in 1945 was followed by the death of more than 25,000 trees, which represented 10% of the total cherry acreage. No sprayed trees died. The yield in 1946 averaged 36 pounds per tree on unsprayed trees and 107 pounds per tree on sprayed trees. In experiments in the 1940s it was demonstrated that poor control of leaf spot resulted in the death of 72% of the tree branches during the winter months. Growers who sprayed regularly and thoroughly every year seldom suffer any serious loss from leaf spot. Research has shown that fungicide applications reduced defoliation of cherry trees from 80% to 0.3% and from 98% to 3%.

The potential impact of eliminating fungicide use on peaches in Georgia and South Carolina has been estimated at 100%, while for California a 45% reduction in yield is projected without fungicides.
projected to be similar to peach losses (-45%). Because of the pervasiveness of brown rot (100% acres infested) peach and cherry losses in all remaining states are estimated at 75% without fungicides.

6.32 Peanuts

Peanut production in the United States is concentrated in three major geographic areas; the Southeast, including Alabama, Georgia, and Florida, the Southwest including New Mexico, Oklahoma and Texas and Virginia-Carolina which includes North Carolina, South Carolina and Virginia

Fungicides are used to control ten major diseases of peanuts in the U.S. These diseases and their incidence in the three peanut-producing regions are listed in Table 5. Without control of these diseases it has been estimated that peanut yields would decline by 78% in the Southeast, 72% in Virginia/North Carolina, and 59% in the Southwest.[536][537][538] The practice of rotating peanuts with another crop provides 30-40% control of most disease organisms that will not permit sustained peanut production in the U.S.[536] The use of resistant varieties provides 20-40% control of the leaf spot diseases, rust and stem rot, but no control of the other diseases. In addition it is estimated that use of resistant varieties results in a yield reduction of 5%. Chlorothalonil, the most-widely applied fungicide, provides 90% control of leaf spot diseases and leaf scorch, while also providing 70% control of rust and 80% control of web blotch.[536] Azoxyrstobin and tebuconazole are effective on leaf spot, rust, Rhizoctonia, white mold and Cylindrocladium Black Rot (CBR). Propiconazole, sulfur and copper applications are targeted at leaf spot diseases, while trifloxystrobin targets leaf spot and rust. Flutolanil is used for white mold and Rhizoctonia while fluazinam targets sclerotinia blight. Mancozeb mixed with sulfur is effective on rust and provides partial control of leaf spots.

Organic peanut growers report that their yields are one-half to two-thirds that of conventional yields and that sulfur and copper sprays are used for disease control.[598][599]

Two related fungi Cercospora arachidicola and Cercospora personatum cause early and late leaf spot of peanuts, respectively. These diseases are also known as Cercospora leaf spot. Both diseases can be found at any time during the growing season. In some areas early leaf spot is the predominant disease, while in others late leaf spot is predominant.[531] Both leaf spots first appear as tiny yellow spots that enlarge and become tan (early) or black (late). The fungi penetrate leaf cells and withdraw their contents causing the cells to collapse and die, forming the spots. The coloration of the spots is imparted as a result of an oxidation of the cell contents.[551] If conditions are favorable, the fungus may produce a new crop of spores every three weeks. When fungicide sprays are not used, pod losses of up to 50% are common. Losses to late leaf spot as high as 70% have been recorded in research plots where the disease was not controlled.[531] These losses are attributed to a reduction in leaf area caused by leaf
death and defoliation. Reduction in light interception and canopy photosynthesis due to defoliation leads to decreased pod growth and increased pod loss.[552] Before 1971, dust formulations of copper and sulfur were routinely used to suppress peanut leaf spots in the U.S.[553] Fifteen to twenty-five pounds of sulfur and copper combinations were used per acre. For decades, peanut harvesting started when the peanuts in a field were stripped of their leaves by one of these diseases.[595] After the introduction of benomyl and chlorothalonil, there was a rapid change from dusting to spraying, which provided improved control and sizable yield gains. Peanut yields increased dramatically between 1969 and 1984. Improved leafspot control is cited as one of the major factors accounting for the yield increase along with the development of higher yielding cultivars and improved harvest methods.[597] (Figure 8) Research demonstrated that chlorothalonil applications improved peanut yields by 34% in comparison to a 23% improvement with copper/sulfur dust.[554] In the Southeast, peanut yield losses to leaf spot average 5% with the use of fungicides but would likely approach 50 to 70% without fungicides.[596]

Web blotch of peanut was first observed in the United States in Texas in 1972. Soon thereafter the disease was found in most peanut-producing states in the Southeast and Southwest. Web blotch was first observed in Virginia in 1979.[533] Losses in New Mexico reach 50% in some years, and the disease can have a heavy impact on the quality of Valencia peanuts marketed in-shell.[531] Web blotch causes defoliation and a subsequent reduction in pod yield.[532] This disease is often present as part of a foliar disease complex that may include web blotch, early leafspot and rust. The first signs of web blotch are small dark brown or tan blotches on peanut leaves. As these lesions mature, they may completely cover the upper side of the leaf. The leaf becomes brittle and is liable to disintegrate and detach from the plant.[531] Networks of the advancing fungus grow between the cuticle and epidermis and kill adjacent cells, resulting in the web symptom. Web blotch development is generally more severe under cool moist conditions and is more common on irrigated than on rain-fed crops. Under conditions that favor web blotch infection, foliar fungicides are very effective at reducing yield loss.[531]

In the U.S., peanut rust has been reported from each of the principal-producing states, but serious epidemics of rust have been limited to the peanut-producing areas of southern Texas. Peanut rust does not survive from season to season in the U.S.; airborne spores are annually introduced from other peanut-producing countries.[532] Establishment of the disease early in the growing season causes reduced pod fill and necessitates early harvesting.[531] Peanut yield losses due to rust measured at two locations in Texas were 50 and 70%.[534] Orange pustules first appear on the lower surface of leaves and rupture to release reddish brown spores.[534] Rust infected leaves become necrotic and dry. Given favorable conditions, disease spread continues throughout the season and may cause total desiccation of the foliage.[534] The dust formulations of copper and sulfur that were used in the U.S. up to the 1960’s controlled leaf spots well but only partially controlled rust.[534] Chlorothalonil and tebuconazole are effective against both rust and leafspots, diseases that frequently occur together.[531] Research in Texas demonstrated that peanut yields were tripled in comparison to control with eight chlorothalonil applications for rust control.[535]
Leptosphaerulina crassiasca is the causal organism of a disease of peanuts with two distinct symptoms. Pepper spot symptoms consist of dark brown to black lesions and leaf scorch symptoms that develop from the tips of leaves. Scorch symptoms consist of wedge-shaped lesions with bright yellow zones along the periphery of the advancing margins of the lesions.[532] Pepper spot and leaf scorch are controlled with fungicides currently used for control of leafspots.

Cylindrocladium crotalariae is a soilborne pathogen that causes cylindrocladium black rot (CBR) of peanut. The pathogen can infect and rot any below ground portion of the plant including roots, pegs and pods. Infected roots die and turn black. Frequently, the entire root system is destroyed. Entire pods may turn black and rot.[532] The pathogenic fungus may survive several years in the soil without a host crop. Infected plants appear stunted with yellow foliage that wilts on hot days. These plants may collapse and die. Small reddish-orange fruiting bodies of the pathogen develop in dense clusters on stems, pegs and pods. At first, spores are forcibly discharged, but later the remaining spores are exuded in a viscous ooze. Disease incidences in excess of 80% and yield losses of 50% or more have occurred. CBR of peanut was first observed in Georgia in 1965. Soon thereafter it was recovered in other peanut producing areas of the United States, Japan, India and Australia.[531] The disease has been a cause of major concern, particularly in North Carolina and Virginia, because of its widespread occurrence and chronic threat to peanut production in these states. CBR was found in all peanut-producing counties of Virginia and North Carolina by 1976.[532] CBR has become increasingly prevalent in Alabama, Florida and Georgia in recent years. Until recently, the only control option for CBR was fumigating the soil with metam sodium. However, research with tebuconazole shows suppression of CBR either with foliar or in furrow applications. In a Georgia experiment with an abundant CBR infestation, tebuconazole significantly reduced the severity of CBR and increased yields by 1,500 pounds per acre.[539]

The peanut plant is susceptible to diseases caused by Rhizoctonia solani from planting until harvest. The fungus is responsible for rotting pods and any above ground portions of the plant in contact with the soil. The fungus can cause enough damage to roots or branches to cause plant death.[531] As the disease progresses branches and pegs are partially or completely girdled and killed, resulting in the loss of pods or immature pods when the crop is harvested.[540] Researchers have noted up to 70% defoliation of the lower part of the peanut plant by a foliar blight caused by Rhizoctonia solani.[532] The pathogenicity of Rhizoctonia solani on peanuts may be due to its ability to produce large amounts of phenolic acids.[532] Rhizoctonia limb rot of peanuts in Georgia was first reported after an epidemic in 1981. Rhizoctonia limb rot has become a major problem particularly in irrigated fields.[531] 40-70% of peanut plants showed signs of disease in Texas.[532] Research with foliar fungicide sprays (tebuconazole) reduced the percent of vines with symptoms of Rhizoctonia limb rot from 30% to 3%.[541]

White mold of peanuts, also known as stem rot, southern stem rot, southern blight, and Sclerotium rot, is found in virtually all major peanut-producing areas of the world.[531] The most distinctive sign of the disease is the white mat of fungal growth found on
diseased stems, crop debris, and soil surfaces around the base of infected plants. Sometimes the disease may kill only one or two branches of a plant, but usually the entire plant dies. Up to 10 to 12 adjacent plants may die. The fungus girdles the main stem and lateral branches, killing the plant. The fungus can infect plant parts below ground, severely damaging pegs and pods. Infected pods are usually rotted with a wet spongy texture and may occur on plants without any visible above ground symptoms.[531] The causal fungus, *Sclerotium rolfsii* produces large amounts of oxalic acid, a phytotoxin which kills host epidermal cells well before penetration by the fungus.[532] A large array of enzymes produced by the fungus increase permeability of host cells to the fullest advantage of the invading fungus.[532] White mold does not usually appear until midseason when the foliage has covered the row middles. Yield losses typically do not exceed 25% but may be as great as 80% under heavy infection.[531] Every year many growers concerned about the presence of white mold dig their peanuts early which results in a yield loss of 10-15%.[542] Although the search for peanut cultivars resistant to *S. rolfsii* originated in 1917, a high degree of resistance has not yet been found.[543] Industry has been slow to adopt peanut cultivars with partial resistance due to undesirable characteristics such as slow emergence, late maturity, small-seeded, and undesirable blanching characteristics.[544] Attempts at chemical control of *S. rolfsii* were not successful until the introduction of PCNB in the 1950’s. PCNB alone or in combination with ethoprop, fensulfothion, and chlorpyrifos reduced white mold incidence and increased peanut yields by 15%.[545] Tebuconazole was registered for white mold control in peanuts in 1994. Research demonstrated that tebuconazole applications reduced the incidence of white mold by 80-90% (from 25 white mold hits per 100 feet of row to 3) and, as a result, increased peanut yields by 58% over an untreated check.[546] Peanut production losses due to white mold declined significantly in the late 1990s as growers began to increase the use of the new fungicides (Figure 15).

Several species of *Pythium* cause pod breakdown (pod rot), damping off, vascular wilt, and root rot diseases of peanut. Such losses from *Pythium* can be as high as 80%.[531] Symptoms include a black root rot, in which the roots are rapidly decayed, and the collapse of the top of the plant, generally without the invasion of the stem above the ground.[531] Seedlings that emerge are stunted. Root systems are water soaked and clumped. Seed infected with *Pythium* become water-soaked and mushy.[532] The junction between pegs and pods can be weakened by the disease resulting in substantial loss of pods at harvest. In Oklahoma, 43% of 37 peanut fields sampled in 1983 had pod rot and disease incidence in these fields ranged from 5-37%.[547]

Sclerotinia blight was first observed in the United States in Virginia in 1971, thereafter spreading to North Carolina, Oklahoma and Texas. By 1982, sclerotinia blight was considered the most important disease of peanuts in Virginia and Oklahoma.[531] A survey of peanut growers in Oklahoma in 1997/98 revealed that 45% of the acreage is infested with the fungus causing sclerotinia blight.[549] 70% of the land suitable for peanut production in Virginia is estimated to be infested with the fungus. Sclerotinia blight has not been found in Georgia, Florida or Alabama.[532] The fungus survives in the soil as small seed-like structures called sclerotia. Even 3 to 4 year crop rotations with non-hosts (corn, cotton, small grains) do not reduce numbers of sclerotia in soil to levels...
that result in effective control of the disease. Sclerotia near the soil surface germinate and colonize adjacent stems, pegs and pods. *Sclerotinia* produces a fluffy white mold in the lower canopy which resembles cotton balls. Once stems are girdled, branches die. Eventually entire plants are killed as fungus progress through the plant canopy. After the fungus has consumed all available food from a colonized stem, it produces survival structures (sclerotia) that resemble mouse dung in size and appearance.

Pods become detached from stems before or during digging as a result of peg decay due to sclerotinia. In areas showing severe symptoms of disease 1,300-1,800 lbs. of pods per acre often remain in the soil after harvest. Pod losses often exceed 50% of expected yield.[531] Trials in Virginia over a four year period indicate that the disease claims an average of more than 33% of potential yield in naturally-infested fields.[548] A five year experiment in Virginia with fluazinam resulted in an average yield increase of 35% or 1,500 lbs./ac as a result of sclerotinia blight control.[550]

### 6.33 Pears

In eastern states broad spectrum fungicides are used to control pear scab, fabraea leaf spot, mycosphaerella leaf spot, sooty blotch, and fly speck. In western states, fungicides are used to control scab and powdery mildew of pears. In both western and eastern pear orchards, antibiotics are used for control of fire blight.

Pear scab occurs wherever pears are grown.[703] Pear scab is caused by the fungus *Venturia pirina*. A closely related fungus (*V. inaequalis*) causes apple scab, but it cannot infect pears and the pear scab fungus cannot infect apples.[95] The fungus overwinters in infected twigs and in leaves on the orchard floor. Overwintering pustules rupture during moist weather and liberate spores. The spores can be carried long distances by air currents. Scab spores are disseminated only during moist periods. If the fruit stems are attacked, the young fruits are usually weakened and drop. If fruit growth continues, badly misshapen pears result. Malformation of the fruit is caused by cessation of growth in the region attacked by the fungus while healthy tissue continues growth. These deformities render the pear useless for the fresh market, canning or baby food.[65][267] Gerber baby food company will not accept scab infested fruit.[267] The scab spots usually enlarge as the fruit grows. Early infections eventually may involve the whole side of a fruit. The central or older part of an early scab infection may die and leave a corky or russeted skin area.[95] The scab fungus is restricted to the zone between the cuticle and the epidermis.[703] The fungus pushes or breaks through the surface of infected fruit or leaves and produces masses of summer spores.[95] These processes may occur many times during the season and numerous secondary infections may result. Old fruit infections often crack open. Cracks are surrounded by russeted, corky tissue and then an olive-color ring of active fungus growth.[291] If fruit is infected late in the season, pinpoint scab spots often show up a month or two later in storage. As the scab spots mature and dry out, they may crack or tear, leaving a jagged, black-rimmed hole in the leaf. Scabby leaves photosynthesize less. Numerous infections may cause the leaves
to weaken, dry up, and drop prematurely. Continued attacks will devitalize the trees. If control measures are neglected, practically all of the fruit may be unmarketable. [95]

Infected leaves decrease their rate of food manufacture and contribute little to the buildup of reserves. Consequently, bud formation is decreased and the next year's crop may be adversely affected. [62] USDA estimates that pear scab infects 80% of eastern pear acreage with a potential for 85% yield loss if not controlled. In western states, scab is estimated to infect 25 to 50% of the pear acres with potential yield losses of 10 to 30%. [13][15]

Fabrea leaf spot is a pest of pears in the East. The leaves, shoots and fruit of pear trees can be infected by this disease. When the disease is severe, it can cause defoliation of the trees late in the summer. [631] Severe defoliation reduces tree vigor and yield. Severely infected Bosc trees can lose most of their leaves by late August and may fail to form fruit buds the following spring. Spores formed in leaves on the orchard floor and spores formed in cankers on shoots are the sources of primary infection. The infection first appears as small black dots. Spores ooze from the center of small black pimples. The spores have a distinctive insect-like appearance. Each of the initial infections can produce millions of slimy spores that are disseminated by rain or by insects. [291] If fungicide protection is lacking, fruit can become severely infected. Lesions on fruit become slightly sunken as the fruit expand; severely infected fruit may crack. [703] Fruits with spots cannot be sold. [291]

Mycosphaerella leaf spot has been a significant problem in a few Michigan orchards. [631] The fungus can build up and cause early defoliation in unsprayed orchards. The disease can infect the leaves and fruit of pear trees. When leaf lesions are numerous, defoliation occurs. The pears have small brown to black lesions on their skin. The primary source of this disease is spores from leaves on the orchard floor.

Sooty blotch and fly speck cause a lowering of the quality and market value of pears. The diseases are recognized by their distinctive patterns occurring on fruit simultaneously with mutually exclusive colonies. Sooty blotch is caused by a fungus that produces clusters of colonies, which range from sooty and smudge like to much darker blotches. Flyspeck appears on fruit as sharply defined black shiny dots in groups of a few to nearly 100. [631] Both pathogens overwinter on twigs of woody plants. Sooty blotch fungi are spread by waterborne spores that are broken off by rainfall. Flyspeck fungi are spread by airborne spores discharged during rainy periods. [631] USDA estimates that 40 to 50% of eastern pear acres are infected by sooty blotch and fly speck with a potential yield loss of 40 to 60%. [15]

In the U.S., powdery mildew on pears is particularly important in the Pacific Northwest. [703] Powdery mildew of pears is caused by 

Podospora leucotricha.

Although this fungus is more common on apples, it is readily spread from apples to pears. [62] The fungus commonly overwinters on infected apple buds and is disseminated to pears in the spring. Infected pear tissue becomes covered with a white fluffy fungal growth on which spores are borne. These spores can infect tender young fruits, leaves and shoots. The disease cycle may be repeated every 5 to 7 days. Pear buds weakened
by powdery mildew are more susceptible to winter damage and are often severely injured during cold weather. Early infection of the young fruit may cause serious russetting and render it unsalable. Control of powdery mildew is achieved by several fungicide applications beginning at the pre-pink stage.[62]

Fire blight is estimated to be present in 100% of Eastern and 90% of Western pear orchards, with a potential to reduce yield by 100%.[13][15] Fire blight is a major limiting factor in pear production since most cultivars are susceptible to moderate or severe damage when infection occurs. Fire Blight favors warm humid conditions and, for this reason, pears are not grown commercially in eastern states south of Pennsylvania.[13] Fire blight once eliminated commercial pear production in the eastern U.S. where the industry was once centered.

Fire blight is the most devastating bacterial disease affecting pears in the U.S. and around the world.[703] Fire blight was first described in New York in the late 1700s, and moved west with settlers, becoming established throughout North American pear production areas by the early 1900s. While fire blight has always been a concern in Eastern pear production, severe outbreaks in the West in recent years have caused growers there to adopt more consistent and vigorous monitoring and management programs as well.[444]

Fire blight is caused by the bacterial organism *Erwinia amylovora*, which grows readily and utilizes sugars and acids as food sources. Fire blight bacteria are microscopic: 25,000 laid side by side would not measure more than an inch. The disease can affect all parts of the tree, including blossoms and fruit, twigs and leaves, trunk and rootstock.[703] An infected tree may die within a few months of infection, or may survive with reduced yield for a few years, or until the grower pulls it out.

Fire blight was so named because the plant parts it infects look as though they have been burned, with infected tissue becoming progressively darker until it turns black. Blighted blossoms and young fruit remain attached to branches but look scorched and wilted, and blighted young shoots wilt into a characteristic shepherd’s crook shape. Within a few days, infections can move 6 to 12 inches or more into the shoot. The bacteria may live for long periods as a resident in or on apparently healthy pear tissue.

Each bacterial cell is completely independent and multiplies by dividing at a phenomenal rate, reaching 10 billion in 72 hours. As the bacterial cells multiply, they advance en masse through the tissue, giving rise to typical symptoms.[703] In addition to the scorched appearance, tissue infected with fire blight will exude droplets of sticky ooze, particularly under humid conditions, that contain fresh inoculum.[334] Favorable conditions lead to rapid multiplication of the bacteria, so rapid that the bacteria are forced through the diseased plant tissue, forming drops on its surface. The ooze may be clear, milky or reddish brown. Infected fruits may exude copious amounts of ooze.

Fire blight produces lesions on infected wood. When the bacteria enter an inactive, overwintering phase and lesion expansion slows, lesion margins become sunken and
cracked, forming a canker. The bacteria overwinter in the canker edges, and ooze out again in the spring. Primary infection from this fresh, inoculum-filled ooze is spread to other plant tissue and other hosts by insects, wind and rain. During a fire blight epidemic, many insect species are attracted to the bacterial ooze on infected trees and help spread the pathogen. Insects are very effective inoculating agents, carrying up to 100,000 cells of the bacteria per insect. The bacteria also produce an aerosol that floats through the air like a fog and infects blossoms of nearby trees. New fire blight infections begin through existing openings in plant tissue. In the early spring, primary infections start in blossoms. In the blossoms the bacteria multiply rapidly and produce toxins, which penetrate cells and kill them. Sugars in invaded plant cells are used by the bacteria for growth and the process is continued until the whole blossom is killed and the bacteria move on.

Copper sprays have been used in commercial orchards for fire blight control since the 1930’s.[522] Copper applied pre-bloom when green shoots are emerging may reduce inoculum on leaves, although insufficient efficacy against fire blight and potential to russet fruit currently limits its usefulness.[527] Little progress was made in the control of fire blight until the 1950’s when antibiotics first developed for human treatment were used as a means of control. Use of streptomycin and oxytetracycline showed very high efficacy as compared to copper products. Streptomycin has been the effective treatment of choice since, applied two or three times during bloom or following rain and hail storms. Organic pear growers are allowed the use of streptomycin and copper for fire blight control.

6.34 Pecans

Although named varieties of the pecan were first planted in orchards in Louisiana and Mississippi around 1880, the tree was not generally planted for commercial production east of the Mississippi River until after 1890. By 1900, several large orchards had been planted in Georgia and Florida.[42]

Pecans are affected by 7-8 fungal pathogens that can cause significant losses if not controlled. Pecan scab is the most significant disease, which is found in most southeastern plantings and can cause up to 100% reduction in the crop if not controlled. Other diseases affecting pecan nuts and foliage include downy spot, brown leaf spot, fungal leaf scorch, powdery mildew, liver spot, and zonate leaf spot.

The scab fungus was first found on the leaves of a hickory tree in Illinois in 1882. Six years later it was found on the leaves of a pecan tree in Louisiana. Through the 1920s topworking trees to scab-resistant varieties was considered the best means of long-range scab control. Topworking is a technique that allows old cultivars to be converted to new varieties by cutting back limbs to stumps and replacing them with shoots of a new variety through grafting. By the early 1920s several cultivars previously believed to be scab resistant became highly susceptible. This trend continued until all the old popular cultivars had lost scab resistance.[35]
In 1956, the cultivar Stuart was reportedly heavily damaged by scab. Stuart was the last of the popular old cultivars to become scab susceptible.[35] Stuart was rated as very resistant to scab for over 40 years. Beginning in the 1950s and continuing through the 1960s orchard plowing was gradually phased out in favor of a permanent sod orchard floor that facilitated mechanized harvest. This shift in practices also ended the destruction of primary inoculum as a major scab control measure.[35]

The scab fungus survives the winter months in masses of thick walled cells formed in lesions on twigs, nut shucks, and leaflets. During the spring spores are abundantly produced. Infection by the fungal spores occurs during periods when rains, high humidity, and dew periods are frequent. Scab pressure is particularly high in the Southeast where high humidity and rainfall are prevalent. Scab presents few problems in West Texas, New Mexico, Arizona, and California where conditions are less favorable for disease development.

Under proper conditions, scab spores can germinate and infect pecan tissue in 2 to 12 hours. Within 7 to 10 days after infection, the fungus colonizes host tissue sufficiently to erupt back through the plant surface, forming lesions that develop spores. This cycle repeats itself many times during a growing season.[41]

The primary scab infections are first observed as elongated olive-brown lesions, which at first appearance are of pinpoint size but soon enlarge. Leaves can appear to be almost entirely black due to the coalescing of these lesions.[37] On the nuts, the spots of infection are small, black, circular, and slightly raised at first, but later may become sunken. The nuts of highly susceptible varieties may have so many infections on them that practically the entire surface of the nut appears black. Severely infected nuts may drop prematurely or they may almost entirely stop growing and remain attached to the shoots indefinitely.[37] Nuts remaining on the tree are usually of very poor quality because the kernels do not develop.[43]

When epidemics occur, all leaves may fall from the tree during the summer.[43] This defoliation prevents flower production the next year, which results in a total crop failure.[43]

Fruit remains susceptible to infection throughout the nearly six month growing season. In the absence of control programs widespread losses approaching 100% can occur in wet years while localized losses of 50-70% can occur in years considered somewhat dry.[39]

Tests in the early 1900s showed that spray applications of Bordeaux mixture (a combination of copper and lime) gave the best control of scab.[34] However, Bordeaux was also found to be phytotoxic to young unfolding leaves.[35] Bordeaux mixture remained the only treatment for pecan scab control through the early 1930s. Bordeaux was used despite periodic losses due to phytotoxicity and occasional failure of the program in years with very wet springs.[35] A major breakthrough came in 1940 when it was reported that low lime Bordeaux mixture would give effective and safe control of
pecan scab.[35] The low lime program was adopted and used for many years. Ziram was introduced in the 1950s as the first synthetic fungicide for scab control. Growers switched to the synthetic fungicides from use of Bordeaux due to their effectiveness in controlling scab, reductions in damage to the foliage, and reduced infestations of black aphids.[34]

Several highly effective scab control fungicides were introduced in the 1960s: dodine, benomyl, and TPTH. Research in the 1960s indicated that applications of dodine or TPTH increased the yield of pecan trees by almost 100% in comparison to Bordeaux applications (11 lbs/tree vs. 5 lbs/tree vs. 1 lb/tree with no treatment).[36] A recent three-year experiment showed that pecan yields were on average 55% greater under a regular fungicide treatment program than when left untreated.[38]

Sprays for scab control are also effective against the other major diseases of pecan.[32]

Two of the main reasons that led to a quadrupling of pecan yields since the early 1900s are the introduction of fungicides for scab control and the development of airblast spray technology for dispersing the fungicides throughout an orchard.[40] (Figure 9)

The current trend in scab control programs is to combine fungicides with newer and older chemistries in reduced-rate tank mixes. These mixtures provide a broader range of disease control than any single product.[39]

Small plantings and isolated trees that are not regularly treated for scab have irregular crops due to sporadic severe scab years, which often causes alternate bearing.[15] Copper is available as a fungicide for organic pecan growers. Copper is not recommended for conventional growers due to its low efficacy.

Because the fungus overwinters in diseased nut shucks and twigs, it is necessary to follow a spray program each season for commercial production of nuts. A protective fungicide must be on foliage and fruit throughout the growing season.[35]

Fungicides are applied to 85-90% of pecan acreage in southeastern states and in Texas annually. Due to dry growing conditions and subsequent low disease pressure, fungicides are not applied in pecan orchards in New Mexico, Arizona, and California. The primary fungicides used in pecan orchards in the U.S. are TPTH, fenbuconazole, propiconazole, and azoxystrobin.

There is some use of sulfur and thiophanate methyl in pecan orchards. Sulfur is used for powdery mildew control while thiophanate methyl is used for zonate leaf spot and for scab where resistant populations have not developed.

Without the use of fungicides it is estimated that pecan yields would decline an average of 55% on treated acres.
6.35 Pistachios

The pistachio is a native plant of Asia Minor and thrives in climates offering cool wet winters and long hot, dry summers. In 1929 an American plant scientist spent six months in Persia (now Iran) and returned with twenty pounds of pistachio seed. The next year, experimental plantings were established in California, the perfect location with its desert-like climate. With pistachio trees requiring six or more years to reach maturity, it was 1950 before one stand-out tree emerged. Additional years were spent in budding this variety (called ‘Kerman’) to heartier rootstocks. Planting of pistachio trees expanded throughout California in the 1960s.[224]

California entered the world pistachio market in 1976 with its first commercial crop of 1.5 million pounds, harvested on 4,350 acres. This placed the U.S. fifth among pistachio producers, behind Iran, Syria, Turkey, and Greece. Within a decade, the California industry grew to become the second largest producer in the world, second only to Iran.

A small bushy tree, pistachio rarely grows more than 20 feet in height. The fruit are borne in grape-like clusters. A red-blushed fleshy hull surrounds the nut. Normally, as the nut matures, the hull separates from the shell and then the shell splits open. California pistachios are mechanically shaken from the tree onto a catching frame. Each tree takes less than a minute to harvest.

Panicle and shoot blight (also known as *Botryosphaeria*) was first discovered in a commercial orchard in Butte county in the Northern Sacramento Valley in 1984. In the orchard where the disease was discovered, as well as a few other orchards in Butte and Tehama counties, yield losses from 40% to 100% were not uncommon.[225] Since its initial discovery in 1984, panicle and shoot blight has become a disease of major importance for pistachios grown in California. In 1998, warm wet spring weather triggered by El Nino provided optimum conditions for spore dispersal and infection. The disease became a problem in pistachio plantings throughout the state except for orchards in the southern part of Kern County and on the west side of the San Joaquin Valley.[225] Total production lost in 1998 was estimated at 20 million pounds.

The effects of the 1998 epidemic lingered into 1999, as blight-induced death of shoots and fruiting buds caused an estimated loss of 12 million pounds. From 1999 to 2002 the disease severity was relatively light due to unfavorable weather conditions, pruning out of blighted wood, removal of inoculum sources, and application of effective fungicides.[225]

Pistachio trees break dormancy in early April. When buds are infected by the fungus, they either will not emerge (total blight) or emerge but the resulting flower or shoot eventually dies. Symptoms appear as dark lesions. Shoots originating from heavily infested buds grow to a short length, become black, and die. In mid-May, leaves on infected shoots wither in 3 to 5 days, and later turn brown. Blighted leaves become distinct among the healthy green foliage. These infections can lead to the collapse of the fruit clusters.[225] Infected leaves begin to drop in July and severe defoliation can occur
by late summer. Infections on the surface of the hull appear in mid-summer as pin-sized round black spots. Most of the blighted fruit are light tan, the consequence of girdling. Some of the infected fruit dehydrate and the fungus produces abundant spores under the epidermis causing a gray color.[225] It takes about 2 to 3 weeks for an infection to kill an entire fruit cluster.[225]

Infections on current season shoots develop into cankers. The infection is limited to the bark. These cankers are the major source of spores that are released in the winter and spring when rains occur. Cankers produce viable spores for at least six years.[225]

Surveys demonstrated that over the five-year period, 1994 through 1999, orchards with panicle and shoot blight produced an average of 23% less than orchards without the disease.[227] The disease was particularly damaging in 1998 when the average yield was reduced by 54% in orchards with the disease.

Panicle and shoot blight is estimated to commonly occur in 37% of California’s acreage.[229] Uncontrolled, the disease is estimated to reduce pistachio yield from 17% to 70%.[229]

Alternaria late blight infects both leaves and fruit, causing early defoliation, severe brown-black stains on the shells, and mold contamination of the shells and kernels.[230] In addition, fewer shells split open among Alternaria infected fruit. The closed shells combined with staining can result in losses of as much as $1,000 per acre. Other damage from Alternaria includes poor flavor and possible mycotoxin contamination. Copper hydroxide applications give partial control of Alternaria.[226]

Alternaria is a common occurrence in approximately 50% of the states acreage.[229] Uncontrolled Alternaria is estimated to reduce pistachio yields by 30-52%.[229]

Following several years of emergency registrations of fungicides, azoxystrobin was granted a full registration on pistachios for control of panicle and shoot blight. Fungicides are used at bloom and again in spring and summer. In 2002, the following fungicides were used for panicle and shoot blight of pistachio; benomyl, chlorothalonil, and azoxystrobin. Research has shown that multiple fungicide applications are necessary to achieve control.[226] The fungicides must be applied as protectants. Growers have reported that azoxystrobin use reduced panicle and shoot blight incidence from 75-100% among untreated trees to less than 1% among treated trees.[228]

6.36 Plums & Prunes (included in Section 6.31 Peaches)

6.37 Potatoes

Potato growers used sixteen active ingredients to control seven major diseases in 2002: late blight, early blight, powdery mildew, pythium leak, pink rot, Rhizoctonia and white
mold. Many of the same fungicides are used for both early and late blight management: chlorothalonil, mancozeb, maneb, metiram, TPTH, and azoxystrobin. Copper provides fair control of early blight and good control of late blight. Several fungicides are targeted exclusively at late blight with no activity on early blight: dimethomorph, cymoxanil, fluazinam, and propamocarb. Mefenoxam is primarily used for pink rot and pythium leak. PCNB and iprodione are used for white mold. Sulfur is used for powdery mildew control. In furrow applications of PCNB, azoxystrobin, and flutolanil are used for Rhizoctonia control.

Mexico has been identified as the source of the pathogen Phytophthora infestans, which causes a disease known as late blight. Late blight is the most important disease of potatoes worldwide.

The disease is known as late blight because in most sections of the country it attacks the plants at or after the blossoming stage. The blight starts as small yellow lesions on the plant; then a cottony growth of spores appears. Spores from infected plants can be carried hundreds of miles by the wind to land on healthy potato plants. Following infection, the fungus grows within plant tissues, breaking down cell walls so that it can use the nutrients found within them. This breakdown of cells causes the blight symptoms. In the field, severely affected plants have an acrid odor which is the result of dying potato tissue. Spores are produced from lesions under conditions of high moisture and moderate temperatures. A single lesion can produce up to 300,000 spores over night. The disease spreads rapidly, with the result that all the plants in a field may be killed in a few days.

Spores produced in the foliage can be washed from leaves to infect the tubers. On a tuber, the fungus spreads irregularly from the surface through the flesh like the diffusion of a brown stain. The flesh may remain dry and mummify or become infected with other organisms. An effect of the disease in storage is the wet rot phase, which is due to the invasion of secondary bacteria following late blight development. This phase develops rapidly under warm humid conditions and causes a very wet, soft, or slimy and foul smelling rot. Potato tubers infected by the late blight pathogen secrete a liquid that favors the growth of soft rot bacteria and thereby causes the wet rot phase. Under severe infection conditions, entire storages must be discarded sometimes producing huge piles of culls (unusable potatoes).

Late blight was first reported in the United States in Philadelphia in 1843, and subsequently spread throughout the country. Late blight was reported in Europe in 1845 where it had spread to Belgium, England, and Ireland. Potatoes were introduced into Ireland in the sixteenth century. The climate and soils were ideal for potato cultivation and allowed abundant production. Because of plentiful potato crops, the population of Ireland had increased to about 8 million by 1845. Irish peasants subsisted almost entirely on potatoes. 40% of the Irish potato crop was destroyed by late blight in 1845 and almost 100% destruction occurred in 1846. An estimated 1.5 million Irish died of famine and disease during the late blight epidemic, and a similar number of people emigrated, mainly to North America.
Late blight epidemics in Europe stimulated intense investigations into the nature of plant diseases and are generally regarded as initiating the development of plant pathology as a discrete discipline.[423] Late blight continued to be a devastating disease until the 1880s when the first fungicide was discovered. A mixture of copper sulphate and slaked lime (Bordeaux mixture) was found to prevent late blight infections if applied to the potato plant before fungal spores arrived. Bordeaux mixture became widely-used in Europe for control of late blight in the early 1900s. During World War I all the copper that Germany had was used for shell casings and electric wire. There was none to spare for making copper sulphate to spray potatoes.[426] A severe late blight outbreak in Germany’s potatoes in 1916 went untreated and the potatoes rotted in the fields. The resulting scarcity of potatoes led to the deaths of 700,000 German civilians from starvation during the winter of 1916-17.[426]

In the U.S. the losses of the potato crop to late blight in 1844 were estimated by state: New Hampshire (25%), Vermont (25%), Massachusetts (25-30%), Rhode Island (10%), Connecticut (25-30%), New York (50%), New Jersey (15%), Pennsylvania (20-25%), Maine (90%) and Delaware (25-30%).[33][427] Periodic epidemics occurred throughout the late 1800s. Experiments with Bordeaux mixture for control of late blight began in the U.S. in the late 1880s. A summary of twenty years of experimental data in Vermont (1890-1910) showed an average increase in potato yield of 64% with the use of Bordeaux.[428] Increased use of Bordeaux mixture is credited with reducing potato losses due to late blight to an average of 2.8% during the 1930s.[307] Growers who sprayed 10-12 times with Bordeaux had minimal late blight in their fields or in storage.[431][432] Growers who did not spray late in the season and harvested potatoes from fields in which late blight was still active experienced storage losses. For example, in 1927, several carloads of potatoes from Maine were dumped into Boston Harbor because such a large proportion had developed tuber rot from late blight infections.[433]

In the U.S. the work of breeding blight resistant potato varieties began in the mid 1850s.[434] Despite the breeding of thousands of new varieties, disease suppression associated with specific host plant resistance has proven unstable and has not contributed effectively to efforts to control late blight because the pathogen rapidly overcomes the resistance.[430] The fungus continues to mutate giving rise to new physiological races. No potato variety has been produced combining resistance genes to all late blight races with the many genetic traits needed to produce a commercial variety. Plants with individual resistance genes have succumbed to new races of the fungus.

Research with zineb and nabam in the 1940s showed potato yield increases of 23 and 35% in comparison to Bordeaux treatments in Ohio and Michigan respectively.[435][436] Many growers switched immediately to Dithane not only because of its greater effectiveness in controlling late blight but also because of its lack of plant injury in comparison to Bordeaux treatments.[437] Potato yields increased dramatically following the widespread adoption of the synthetic fungicides (Figure 6). Mancozeb and chlorothalonil became widely-used as protectant fungicides for control of late blight in the 1960s and 1970s. The introduction of metalaxyl in the 1970s provided growers with a fungicide that could eradicate an established infection with systemic
action. The use of metalaxyl in combination with protectant fungicides provided almost complete protection against late blight. As a result of the use of effective fungicides, late blight infections were not a major concern in the U.S. until the early 1990s.

The cause of the reemergence of late blight was the immigration of exotic strains of the fungus.[440] Up until the 1980s, the fungus in the U.S. consisted of only one kind—the A1 mating type, such that only asexual reproduction was possible. The A1 type is short-lived and can only exist on a living potato family host issues such as vines, foliage, stems, and tubers. In the 1980s, a second mating type (A2) immigrated to the United States from Mexico. Sexual mating between A1 and A2 types produce a tougher spore with a thicker protective cell wall more able to survive freezing temperatures and exist outside a living host. Thus, it became possible for the fungus to survive in soil and plant debris for long periods: months and perhaps even years. The pathogen population became increasingly fit and more aggressive. In the early 1990s the A2 fungus and new forms of the fungus resulting from A1/A2 mating spread throughout all potato growing areas of the U.S., including areas such as Idaho and Oregon where late blight had not previously been a problem.[441][423] The new exotic strains were nearly all resistant to metalaxyl. The exotic strains are more aggressive than the traditional strains and produce more spores. They have a faster lesion expansion rate and complete their disease cycle in fewer days resulting in more cycles of infection. As a result, the exotic strains require more fungicide applications (25% more) for adequate suppression of late blight symptoms than was previously required.[440] Growers need to spray every 2-3 days instead of every 4-5 days.

Epidemics of late blight occurred in parts of the U.S. during the early 1990s as the new strains established themselves. These epidemics were locally devastating, sometimes resulting in total crop losses and severe economic hardship for many potato growers.[423] For example, one New York grower suffered an 80 to 85% crop loss on 200 hectares of potatoes in 1994 and was subsequently forced out of business. Another New York grower discarded 300 metric tons of potatoes only 5 days after harvest.[440] The disease destroyed huge acreages of potatoes. By the time it was clear that the disease was resistant to metalaxyl, the disease was so well established that there was insufficient time to use the protectant fungicides.[423] Farmers were not prepared for the rate at which the new strains spread. In one Pennsylvania county (Potter) the fungus destroyed 90% of the 1,600 acre crop causing a $2.2 million loss.[443] Due to the speed of the epidemics, there was often only a few days between disease detection and total foliage destruction.[423] In 1995, in Oregon it was estimated that tuber losses in storage due to late blight amounted to $3 million.[442] In North Dakota and Minnesota, 20 storage bins of potatoes were dumped in 1993-94, which totaled a loss of 60 million pounds of potatoes.

After these most recent epidemics, EPA registered several fungicides with some curative activity for late blight: propamocarb, cymoxanil, and dimethomorph. Severe losses have not been repeated in the U.S. due to an increase in the application of fungicides by potato growers. In the early 1990s potato growers in Washington typically treated 70% of their acres with fungicides while in the late 1990s, 90-100% of the acres were regularly
treated. In Idaho, the percentage of potato acres treated with fungicides rose from 40% in the early 1990s to 70-90% in the late 1990s. The total amount of fungicide active ingredient applied per treated acre rose significantly in the 1990s as growers made more applications and used more active ingredients. (Figure 12)

Field experiments with the new late blight strains have shown significant yield reductions in the untreated plots in comparison to plots receiving fungicide treatments: Pennsylvania (-50%), Oregon (-55%), Washington (-50%), Michigan (-48%), New York (-48%), Wisconsin (-26%), North Carolina (-51%), Idaho (-41%), and Virginia (-66%). [447]-[455]

Early blight occurs earlier in the season and in drier weather than late blight; it is also rather common late in the season when weather conditions are favorable for its development. [439] Early blight, which is also prevalent in the areas where late blight is found, is the major fungal problem in arid areas typified by western states. Foliar lesions associated with early blight decrease photosynthetic leaf surface area, which in turn reduces tuber yield potential. [415] The premature death of the foliage results in decreased yields. Foliar lesions are dark spots which may be surrounded by a yellow border and usually contain concentric circles of alternately raised and depressed necrotic tissue. The yellow colored area is caused by alternaric acid, a toxic substance produced by the early blight organism. An individual early blight spot on a leaf may produce 1,500 to 3,000 spores in two or three eruptions during a season. [420] Spores produced on foliar lesions also contaminate soil and infect tubers through wounds made during harvest. Tuber lesions are dark, circular to irregular in shape, and frequently sunken. The decayed tissue appears leathery or corky. [415] Spores can move long distances on air currents, so that all but the most isolated fields will be exposed. Spores deposited on leaf surfaces can remain viable for at least 8 weeks and can infect leaves when conditions become favorable. [415] Early blight is a problem in most potato production regions in the United States. [416]

In the early 1900s, it was estimated that uncontrolled early blight reduced potato yields 25% in Michigan, 10-25% in Wisconsin, and 5-15% in Minnesota. [417][418][419] Recent experiments in Minnesota showed reductions in tuber yield caused by early blight of 18 and 21% in two crops with uncontrolled epidemics. [416] Early experiments in Wisconsin with Bordeaux mixture led to potato yield increase of 18-27% as a result of early blight control. [420] Experiments with chlorothalonil showed a reduction in the number of plants damaged by early blight from 25% to 3%. [421] Weekly spraying of chlorothalonil in a Pennsylvania led to a 20% increase in potato yield due to early blight control. [422] The optimum time for the first fungicide application is when airborne spores first appear. [415] Generally, 2 to 4 fungicide applications are sufficient to control early blight.

Powdery mildew can be an important foliar disease of potatoes in arid and semiarid climates. [415] A heavy rain stops the disease. Powdery mildew is seldom a problem on potato plants grown under sprinkler irrigation. [415] In the U.S. it is of economic importance in Washington and Oregon. A white powder appears over plant surfaces, the
plants become twisted and brittle and then die.[438] Powdery mildew on rill irrigated potatoes in the Columbia basin develops in early July, becomes severe in late August, and may kill plants by October.[438] It is recommended that growers apply sulfur at the first sign of powdery mildew and continue treatment biweekly.[415] Research in Washington showed that sulfur applications led to potato yield increases of 6-13%.[438]

Leak, also called shell rot, occurs wherever potatoes are grown, but it is of most importance in the north central states.[440] Leak only affects tubers. Leak is caused by *Pythium ultimum*, a soilborne fungus that exists in many, if not most, agricultural soils.[415] The fungus enters tubers only through wounds. Internally, the rotted tissue is spongy and wet and may contain cavities. The most characteristic symptom of leak is the extremely watery nature of affected tissues. The water is usually held by the disintegrating tissues, but when pressure is applied, a yellowish to brown liquid is given off readily.[439] After infection, a tuber may become so completely rotted within one week that even the slightest pressure causes the skin to rupture and large quantities of liquid to exude.[415] Sometime, the entire inside of the potato may rot and leave an outer shell about one-fourth inch thick with only small external lesions are visible.[440] Potato yield losses of up to 75% can occur in severely affected fields.[445] A recent experiment with mefenoxam demonstrated a yield increase of 21% due to leak control.[446]

Pink rot develops in soils approaching saturation from poor drainage, excessive precipitation, or irrigation. The disease is most frequently observed in mature plants approaching harvest. During harvest and bin filling, diseased tubers can infect healthy tubers.[415] Infected potato skin is easily rubbed off. Rotted tubers remain intact but have a rubbery texture. When cut and exposed to air, recently infected surfaces change from cream-colored to salmon pink in 20 minutes and then to black in about an hour.[415] Infection imparts a vinegary flavor to the entire tuber. When an infected tuber is squeezed, droplets of liquid exude from the eyes. Sometimes gases are given off which form small bubbles in the droplets.[457] Infected tubers subject to pressure by squeezing do not regain their shape. This is noticeable as infected potatoes become flattened when they are in storage piles.[457] Rotted tubers give off a pungent odor like that of formaldehyde. In the 1930s and 1940s in Maine, losses of 20% occurred in low lying fields in wet years with 10% of the tubers from such fields becoming affected with pink rot in storage.[458] In some infected fields in Idaho as much as 50% of the tubers showed symptoms of pink rot as they were dug.[459] In western Nebraska, up to 10-15% infection was found in some storage bins.[457] A recent experiment with mefenoxam demonstrated an increase in yield of 97% as a result of pink rot control.[456]

White mold, also called Sclerotinia stalk rot, has gained importance as a disease of potatoes with the increased use of sprinkler irrigation and maintenance of high fertility levels. These two practices promote lush, dense canopies, long periods of high humidity in the canopy, free moisture on the foliage, and relatively stable temperatures. All of these environmental factors favor white mold growth.[415] White fungal growth commonly develops on lesions or in the pith of stems. Tubers near the soil surface may be infected but this phase of the disease is seldom seen.[415] Saucer-shaped spore
release structures develop in the soil where the fungus can survive for at least 3 years. Each saucer may release in excess of 8 million spores.[415]

There are two phases of *Rhizoctonia*: the stem phase and the tuber phase. The stem phase occurs early in the growing season in the form of lesions or nipping off of the growing tips of sprouts. Damage results in skips or delayed emergence and is expressed in uneven stands. Potato plants affected at this stage are characterized by a lack of vigor because much of the energy has to be used to produce secondary sprouts before a plant emerges.[639] The second and most noticeable phase of the disease is the formation of sclerotia on tubers. Sclerotia are survival structures of the pathogen. These sclerotia are often referred to as black scurf and gives rise to the name “the dirt that won’t wash off.” As the potato plant starts to die, the fungus begins forming the sclerotia on tubers. Rhizoctonia black scurf was suppressed by 90% in trials where azoxystrobin was applied at planting.[640]

In organic potato production, “The frequency of copper applications may be quite high and exceed the 9 to 15 sprays reported with conventional fungicides.”[249]

**6.38 Raspberries**

There are two major types of raspberries, red and black. The following discussion addresses red raspberries. Raspberry production in the U.S. is largely accounted for by three states: California, Oregon, and Washington. Most of the raspberries harvested in Oregon and Washington (>90%) are machine harvested and used for processing. Most of the raspberries grown in California are used in the fresh market. Fresh market raspberries are hand harvested.

Raspberry plants have perennial roots and crowns and biennial shoots. A plant may remain productive for 15 years. The shoots (canes) grow vegetatively during the first growing season and then become dormant during the winter. The following spring, they produce lateral branches that flower and produce fruit. The entire cane dies after fruiting. Canes in their first growing season are referred to as primocanes and those in their second growing season are floricanes. Primocanes are trained to a single trellis wire about five feet from the ground. New primocanes emerge while the older floricanes fruit, so mature plants have both active primocanes and floricanes in evidence each season.

The major diseases in raspberries are botrytis fruit rot (gray mold), yellow rust, and phytophthora fruit rot.[198]

Gray mold, or botrytis fruit rot, is the most common disease of raspberries worldwide. In the western United States, it has been estimated that the gray mold fungus infects 100% of the raspberry acres. It has the potential to reduce yields by 35% if left uncontrolled.[13] Spores from dead leaves and mummified berries are the main sources of primary *Botrytis* inoculum. As soon as flowers open, they become susceptible to infection. The initial infections of flower parts are latent.[197] The fungus lies dormant
until the fruit is nearly ripe (or after harvest) at which time symptoms develop rapidly at high humidity.

Infected berries are covered by a grayish brown, dusty mass of spores, which gives the disease its name “gray mold.”[197] Infected berries begin to leak inoculum and adjacent berries become colonized. The mold releases spores that cause additional fruit and cane infections.[200]

Due to the microscopic nature of latent blossom infections, monitoring for the disease is impractical.[199] Preventive fungicide sprays during the bloom period are used to suppress gray mold. Recent research showed an 88% incidence of gray mold in untreated harvests while harvests after fungicide treatments had a disease incidence of 5-6%.[206]

Recent research with the biofungicides, Messenger and Serenade, showed a total lack of effectiveness in reducing gray mold infections among raspberries.[207]

The yellow rust fungus overwinters in the bark of floricanes, where up to 400 spores per square centimeter have been recorded.[197] Yellow rust is widespread in most raspberry fields, particularly in years when spring rains continue late.[200] The first symptom of yellow rust is the development of orange-yellow pustules on the upper surfaces of leaves. Severely infected leaves turn yellow and drop. Fruit often dies on the canes before maturing if leaves on fruiting laterals are infected early in the summer.[200] The pustules contain numerous spores which are carried about by wind currents or splashing raindrops, which can propagate new infections all summer. In favorable weather, all the plants in a field can become infected.[208]

Due to a shift towards wet and mild conditions in the Pacific Northwest weather cycle, yellow rust has been appearing earlier in the season and has been spreading more rapidly than before.[201] The shift in weather patterns has also enabled spore stages that did not normally overwinter to do so in the last few mild winters.

In the Spring of 1998, a sudden and widespread outbreak of yellow rust infected Willamette Valley raspberries. The rust infected cultivars had previously been resistant to the disease.[198] “Meeker,” the most widely-planted cultivar in the Pacific Northwest is normally slow to develop rust symptoms. However, yellow rust is appearing in fields of “Meeker” and the disease is developing quickly.[201] The State of Oregon estimated that without fungicide use yellow rust would lower raspberry yields 25% statewide.[201]

Research has shown that fungicide applications provide 98-100% control of yellow rust.[201]

Phytophthora root rot is regarded as a major cause of declining raspberry plantings in many commercial production areas worldwide.[197] The fungus can directly invade and kill root and crown tissue. Plant density in diseased areas is usually reduced and the number of emerging primocanes is reduced by the infection. Infected primocanes may rapidly wilt and collapse shortly after emergence, often after developing a dark, water-
soaked lesion at their base.[197] Floricanes of severely infected plants often wilt and die before harvest. Spores can remain viable for a number of years in soil. Optimum discharge of spores occurs when soils become saturated with water. Significant movement of spores can occur through flagellar propulsion or passively through flowing or splashing water.[197]

Under favorable conditions, if *Phytophthora* is left uncontrolled, yield losses can reach 75%.[199] Metalaxyl and fosetyl-al have provided significant levels of control.[197] Mefenoxam is usually applied once in the fall or early spring as a band treatment in the row.

Organic growers use old style trellises that keep the canopy more open with two extensions instead of keeping the canopy in a single line. While somewhat expensive, this trellising technique increases air circulation and light penetration in the plant canopy, making conditions less favorable for fungal infection and reducing the need for fungicide sprays to manage diseases.[202] The more open canopy also results in less yield. Organic growers usually budget for about three tons of production per acre which is about 25% lower than average yields. In organic production, replanting tends to be more frequent than in conventional plantings due to root rot problems.

### 6.39 Rice

Trifloxystrobin and azoxystrobin are targeted at both sheath blight and rice blast. Flutolanil is used for sheath blight control while propiconazole is used to control sheath blight, false smut and kernel smut.

Recent experiments with azoxystrobin show that rice yield is increased by 35% when sheath blight is controlled and by 45% when blast is controlled.[751] Fungicide treatment prevents considerable lodging. In experiments, nearly half of the untreated check plants were down at harvest while azoxystrobin treatment reduced lodging to 3%.[760] About 20-30% of the yield increase comes from control of minor diseases.[760] Azoxystrobin reduces stem rot and sheath rot by 50% and reduces leaf scald by 30%.[760]

Rice is grown in an aquatic system, resulting in a humid microclimate that favors disease development. Disease damage to rice can greatly impair productivity and sometimes totally destroy a crop. Direct losses to disease include reduction in plant stands, lodging, spotted kernels, fewer and smaller grains per plant, and a general reduction in plant efficiency.[744] The State of Arkansas has estimated that during an epidemic year, the combination of sheath blight and rice blast would reduce statewide yields by 45%.[757]

Diseases have become more important in U.S. rice for several reasons. Sheath blight was once considered a curiosity until farmers adopted high-yielding semi-dwarf cultivars, denser stands, and increased nitrogen rates.[754] These practices create a microclimate with high humidity and cooler temperatures, which increase the severity of many rice
diseases including blast, sheath blight and kernel smut. All of these diseases are now epidemic in Arkansas every year.[754] High nitrogen fertility not only increases yield but also increases susceptibility of rice to diseases. Over the past few years, rice growers have continued to adopt shorter rotations and reduced tillage, primarily for economic and soil conservation reasons. There is limited availability of new land for long rotations. In many areas the most common crop rotation is rice-soybean-rice with the only seedbed preparation done in the fall. This “stale seedbed” is left undisturbed until the following spring when the weed cover is killed with a herbicide, and rice (or soybeans) is seeded into the previous crop’s residue with minimum or no tillage.[754] Since survival between crops of the sheath blight and rice blast fungi have been linked to infected rice residue, the incidence of these two diseases has increased since the residue is no longer destroyed on no-till land.

California’s arid climate provides an unfavorable climate for foliar diseases when compared to the Mid-South rice regions. California has historically achieved the highest rice yields per acre due, in part, to favorable, disease-free growing conditions. Sheath blight is not present in California. Historically, California’s two worst rice diseases have been stem rot and sheath spot. Fall burning of rice straw provided the most efficient means of controlling these diseases. The practice of burning is being phased out due to air quality concerns stemming from the smoke of field burning. There are no chemical methods for control of these diseases registered in California.[761] Blast first appeared in California rice fields in 1996.

Sheath blight is the most important rice disease in the Mid-South and in Texas, affecting more than 50% of planted acreage each year.[756] Sheath blight is caused by the fungus *Rhizoctonia solani*. The sheath blight pathogen is soilborne. The fungus does not produce spores and must grow from plant to plant. The fungus can spread across a surface of water to adjacent plants. The fungus also grows across touching plant parts, for example from leaf to leaf, causing infections on nearby plants.[744] As lesions coalesce on the sheath, the flow of water and nutrients is interrupted and the leaf may die. As lesions age, the fungus forms white balls, which turn dark brown. They are the survival structures of the sheath blight fungus called sclerotia. More than fifty may form on a single plant.[745] Sclerotia are easily dislodged from plants during harvest and fall to the soil surface. When a succeeding crop of rice is flooded, the sclerotia float to the top of the water and contact rice plants; the fungus grows out, and moves into the rice leaf. Sclerotia are able to germinate up to eight times and infect plants each time. They can survive in the soil for several years in absence of a rice crop. A survey in Arkansas found sheath blight in 90% of the fields surveyed.[752]

Sheath blight mainly kills foliage that contributes carbohydrates for grain-filling. The primary cause of yield loss results from a reduction in effective leaf area. Incomplete grain fill reduces total yield and results in lower head rice yields because the poorly filled grain breaks during the milling process. Loss in grain yield may reach 42% and additional economic losses may be incurred by reductions in milling quality of up to 20%.[747]
The sheath blight pathogen kills most of the stubble and thereby severely reduces retilling. This is particularly important in Texas where about half of the rice acreage is ratoon-cropped, i.e., allowed to retiller and then harvested a second time. **[743]** Ratoon yield usually runs half or less of main crop yield because too few tillers come back to form a thick canopy. **[760]** Much of this failure is due to sheath blight, which kills tillers. Two years of research data show that one application of fungicide on the first crop produce about twice as many resprouted tillers. **[760]**

Development of resistant cultivars has been slow, because resistance is linked to undesirable traits such as tall plant stature, late maturity and poor milling quality. **[747]** Taller varieties have less dense leaf canopy and have the major grain filling leaves and sheaths higher above the soil, making sheath blight infection less likely. **[757]** Unfortunately, these types often lodge, resulting in yield losses of up to 50% because harvesters cannot recover the grain lying on the ground. Rotation with a non host crop such as pasture results in less disease incidence compared to soybean. However, pasture is not an economically viable crop in the Mississippi Delta. **[747]** In soybeans *Rhizoctonia solani* causes aerial blight, which adds inoculum to the field in rotation years.

Untreated rice tillers in one experiment showed 67% disease while azoxystrobin reduced the infection to 10%. **[760]** One application of azoxystrobin at panicle differentiation gave almost season long control of sheath blight. **[751]** Loss from sheath blight is about 100 pounds per acre in yield for every day after heading that the rice is left untreated. **[746]**

The blast fungus overwinters in rice straw and stubble. The disease spreads rapidly in the field by means of airborne spores. Severe infestations can lead to large areas of dead plants. A disease cycle begins when a blast spore infects and produces a lesion on the rice plant and ends when the fungus sporulates repeatedly for about twenty days and disperses many new airborne spores. Thousands of airborne spores are produced on lesions and are carried considerable distances by air currents. In one experiment, the maximum number of spores produced in one night was 20,000 on one leaf lesion and 60,000 on one spikelet. **[753]** The spores germinate on rice plants and develop an appressorium at the tip of the germ tube, which attaches to the surface of plant tissues. An infection-peg from the appressorium then penetrates into plant tissue. Germinating spores, germ tubes and appressoria excrete adhesive materials, called mucilage, which enable the fungal body to tightly adhere to the rice epidermis. When a blast spore penetrates the surface of a rice leaf, it does so with a force equal to 80 times atmospheric pressure or 1,600 pounds per square inch. **[742]**

Although the biochemical basis of the pathogenicity of the blast fungus is not clear, toxins are a likely component, since a number of toxins from the pathogen have been identified. **[755]** Depending on the portion of the plant affected, the disease is also called leaf blast, node blast, panicle blast, collar blast, and rotten neck blast. Spores land on leaves, germinate, penetrate the leaf, and cause a lesion four days later; more spores are produced in as little as six days following infection. When it hits the head or neck, blast
stops nutrients and water from getting to the kernels, stopping kernel development. Portions of the grain head (with panicle blast) or the entire grain head (with rotten neck blast) will be white in contrast to green or tan color of healthy grain. This “blasted” appearance is caused by sterile or blank grain. Leaves and whole plants are often killed.[744] On stem nodes, the host tissue turns black and becomes shriveled. Plants lodge or break off at the infection point. Rotten neck symptoms include plant tissue turning brown and shriveling, causing the stem to snap and lodge. In Mississippi in 1987, blast was estimated to have resulted in a 15 to 20 bushel per acre yield loss on over 45% of the state’s rice acres. Blast has the potential to reduce yields by 80%.[760]

Complete resistance to blast, where the fungus is unable to cause sporulating lesions on the plant, is well known. However it has also been associated with spectacular breakdowns in cultivar resistance.[755] This type of resistance is controlled by one or two genes and is generally effective for two or three years.[755] A variety with this type of resistance, if planted on large acreage, can lead to development of a new race of the blast fungus that can attack the variety in only a few years. A famous example of this phenomenon occurred in the mid 1980s to the variety, Newbonnet, which was resistant to the then-predominant blast races. Released in 1983, this variety rapidly became the most popular in Arkansas, planted on 70% of the acres by 1986. During that year, intense and widespread epidemics of neck blast wiped out entire fields and heavily damaged many thousands of acres.[757] Two minor blast races had quickly become predominant and caused the epidemics. Two new blast resistant varieties, Katy and Kaybonnet, were released, but they do not yield as well and their kernel size was somewhat small.[758] Kaybonnet was released seven years after the successful initial cross breeding. Currently Arkansas growers have planted a majority of acres with high yielding rice blast susceptible varieties such as Wells and use fungicides to control blast.[759] In rice blast epidemics in Texas in 1991 and 1992, fungicides were credited with raising yield by 19% on 53% of the planted acres, each year saving 263 million pounds of rice valued at $15 million.[741]

Rice blast was first found in California in 1996. Scientists can only speculate on how the fungus was introduced into California. Some possible sources were contaminated equipment from infected areas in the South or infected seed.[762] In California, average percent yield reductions from infected fields attributable to blast range from 15 to 30%.[764] Blast resistant cultivars from the Mid-South cannot tolerate California’s relatively cold night time temperatures. Since California breeders traditionally were not concerned with a disease that did not exist in their state, virtually all of the California cultivars are susceptible to blast.[763] Azoxystrobin is used in California for blast control.

Kernel smut infects developing rice kernels and replaces the starches inside with its own black spores. The disease is most noticeable in the early morning when dew causes smutted grain to swell and erupt, releasing spores.[755] Smutted kernels often break during harvest. As the spores dry, they are blown around the field. Spores are released from smutted grains before or during harvest and contaminate healthy kernels, crop debris and soil.[755] The pathogen is ubiquitous in Mid-South soils, where the spores
can survive for more than two years. When the next rice crop is planted, the pathogen floats to the surface of the water and germinates, forcibly releasing spores in great numbers. During harvest, kernel smut spores blow out the back of the combine and coat the machine. When parboiled, smutted rice has a dark gray tint throughout the grain. Kernel smut is most important in rice that is sold for parboiling because partially filled, discolored grains break frequently during milling. Rice mills routinely discount for “smutty” rice. Fields have been observed in Louisiana with 20-40% of the florets affected on 10% or more of the panicles in a field. A survey in Arkansas found kernel smut in 73% of the fields. During the last decade, Arkansas researchers have measured yield losses of 10% due to kernel smut. Large scale testing of fungicides in commercial rice fields found that propiconazole averaged 85 to 95% reduction in smutted kernels.

The false smut fungus invades the ovary of rice kernels at the early flowering stage. The spores can infect a developing ovary if its florets are open. The ovary is destroyed and the rice kernels are replaced by spore balls, which burst out from between the glumes producing a silvery white gall that later grows into a large, orange ball (1/2 inch diameter). This ball eventually turns black at maturity. The galls are covered with spores that can rapidly spread the disease to nearby plants. The galls are harvested along with rice grain, resulting in unsightly lots of rice that have to be cleaned prior to use. The fungus survives in soil as hardened spore balls for up to four months and infects other plants between rice plantings. Propiconazole applications have reduced the number of smut balls in harvested grain by 50 to 75%.

A recent USDA assessment estimated that without fungicide use, rice yields would decline by 30% in Arkansas, 15% in Louisiana, and 9% in Texas.

6.40 Soybeans

Foliar fungicides are used on soybeans in eight southeastern and delta states. The eight states represent 11% of United States acreage and 9% of domestic production of soybeans.

Azoxystrobin was the most commonly used fungicide active ingredient. Experiments with azoxystrobin have resulted in soybean yield increases of approximately 25% as a result of control of foliar diseases.

Foliar diseases of soybean plants are a greater problem in southern states because the hot humid climate of the summer is conducive to disease outbreaks. Several of the diseases that attack soybean plants directly damage pods and seeds as well as the leaves of the plants. The rapid killing of leaves prevent movement of nutrients to the pods, resulting in reduced seed size and yields.

Anthracnose occurs wherever soybeans are grown, however it causes damage only in warm, humid areas. The disease reduces stand and seed quality and yields by 16-26% or more in the southern U.S. Anthracnose is generally a late-season disease. Infections
occur during bloom or early pod development when conditions are wet and humid for a prolonged period. Fungal spore germ tubes develop large dark appressoria when in contact with the surface of the soybean plant. A narrow infection peg extends out and directly penetrates the cuticle and cell wall of soybean tissues. After penetration, the fungus spreads both between and within host cells.[48] The fungus produces an abundance of spores that infect and kill lower branches, leaves, and young pods.[55] Later, these areas are covered with black fruiting bodies that resemble tiny pin cushions. Premature defoliation may occur throughout the canopy. When soybean pods are infected early, either no seeds are formed or seed size and quantity is reduced.[48] The fungus can completely fill pod cavities and seeds may become moldy and shriveled.[48] Anthracnose is controlled by applying foliar fungicides between bloom and pod fill.[55]

Soybean losses from cercospora leaf blight were estimated at 130 million bushels in 15 southern states in 1978.[48] Cercospora leaf blight causes small reddish purple lesions on the upper leaves of soybean plants. The lesions become leathery and dark purplish-red. Black lesions occur on pods.[48] Heavily infected leaves become yellow and fall.[55] The most obvious symptom is the blighting of young upper leaves over large areas, even entire fields.[48] Most soybean fields surveyed in Arkansas during the full-pod growth stage have some damage from the disease.[48] Although some cultivars possess resistance, control is primarily achieved by application of foliar fungicides starting at early pod set.[55]

Frogeye leaf spot disease occurs worldwide, but is most common in warmer regions during warm and humid weather. Yields from susceptible cultivars may be reduced by 15% in the U.S.[48] Frogeye leaf spot is primarily a disease of foliage, but stems, pods and seeds may also be infected. The lesions are circular to angular spots. They begin as dark, water-soaked spots. As lesions age, the center becomes tan to nearly white and the margin darkens. When lesions are numerous, leaves wither and fall prematurely.[48] Often all the leaves on a plant are infected. Lesions on pods are sunken. The fungus frequently grows through pod walls into maturing seeds. Five physiologic races of the fungus have been reported in the United States.[48] Fungicides applied at the late flowering and beginning seed growth stages protect against frogeye infection.[55]

Soybean yield losses of 30% have been attributed to rhizoctonia aerial blight.[82] Besides soybeans, the fungus attacks rice, which is commonly rotated with soybeans in Arkansas, Louisiana, and Mississippi. The fungus also survives in the soil and on plant debris, as well as on certain weed hosts.[55] Infection begins at flowering during prolonged periods of high humidity and warm temperatures. Diseased tissues in old lesions generally fall out during dry weather, giving the plant a ragged appearance. Severely infected plants may be totally defoliated.[48] Control strategies include applying foliar fungicides.[55]

Pod and stem blight fungi initially cause a latent infection throughout the soybean plant.[48] In wet seasons latent infections produce fungal growth over the entire plant. The most characteristic symptom of this disease is the arrangement of black fruiting structures in linear rows on soybean stems.[55] The main losses are from the pod blight
phase, resulting in moldy seeds, which may not be harvested, weigh less, or lead to grade and price reductions. Heavily infected seeds are badly cracked and shriveled and are frequently covered with a white fungal growth. Infected seeds produce low-quality oil and meal. When harvest is delayed under wet conditions, seeds may be infected throughout the plant.[48] Pod and stem blight is managed by foliar application of fungicides from mid-flowering to late pod.[55]

Without fungicide use, soybean yields are projected to decline by 25% on treated acres.

### 6.41 Spinach

Spinach is principally grown in six states: Arizona, California, Colorado, Maryland, New Jersey, and Texas. Growers in all principal states produce spinach for fresh market consumption; growers in Texas and California also produce for the processing market.

The most prevalent diseases which require fungicide sprays are downy mildew and white rust. The presence of either of these diseases can render an entire field unmarketable under favorable disease conditions.[170] Most U.S. spinach acreage receives a preplant fungicide treatment and a foliar spray for control of these pathogens.[170]

Downy mildew, also know as blue mold, is probably the most widespread and potentially destructive disease of spinach worldwide.[194] The disease first appears on leaves as small pale or yellow spots that enlarge rapidly. The spots, yellow on top and gray beneath, later die producing light brown areas on the leaves.[186] A fuzzy bluish gray growth appears on the underside of the leaves (hence the common name ’blue mold’). These are the fungal structures in which spores are found. Spores are transported by wind to other sites.[185] When environmental conditions are favorable, epidemics can progress very rapidly and an entire crop may be lost in a short period.[189]

Until the late 1950’s the U.S. spinach industry was not highly viable. Cultivars were susceptible to downy mildew, which caused severe reductions in quality and in some cases complete loss of the crop.[185] Control by fungicide spray was not practical. Because there were no effective controls, a search for resistance to downy mildew was begun in 1946.[188] Over 40,000 plants from 19 commercial varieties were inoculated without finding any healthy plants. In 1947, nine lots of wild spinach from Iran were tested. Two lots were found to have a few plants that remained completely free of downy mildew.[186] As a result of cross breeding with commercial varieties, new commercial hybrids with immunity to downy mildew were released in the mid 1950s. At this time, there were only two races of the organism causing downy mildew on spinach worldwide and the hybrid plants had immunity to both races.[185]

For twenty years, downy mildew was unknown on spinach in the U.S. In the late 1970s a new race of the downy mildew fungus appeared and spread rapidly in U.S. spinach fields causing heavy losses.[185][187] A gene for resistance to race three was used in spinach cultivars as early as 1982 and provided resistance to downy mildew on spinach in the U.S. until 1989 when race 4 became established.[189] As a result, spinach cultivars with
resistance to races 1, 2 and 3 became infected with downy mildew. Many spinach fields near Ventura and Santa Maria, California were lost to the new races.

Little or no work had been done with fungicides on spinach because the resistance had been so effective. Research revealed that metalaxyl was highly effective in controlling downy mildew; whereas untreated plots incurred a 43% reduction in yield, the metalaxyl treatments reduced the yield loss to 1%. Fosetyl-Al was granted Section 18 registrations to control downy mildew following the discovery of race 4.

A mysterious downy mildew that first hit spinach growers in 1997 was identified as a fifth distinct race. That epidemic was followed the next year by the appearance of downy mildew race 6. Race 7 was then discovered in Europe. In 2003, race 8 was discovered in Europe and in 2004 races 9 and 10 were found in California spinach fields.

Downy mildew is relatively rare in spinach fields because of the use of fungicides. Blue mold is best controlled when treatment is used as a preventative measure, rather than waiting for the onset of disease symptoms. If there is heavy rain, one can anticipate blue mold.

White rust is a very important disease in all U.S. spinach production areas east of the Rocky Mountains. White rust does not occur on spinach in western production areas and has not been reported on spinach outside the U.S. White rust first caused great damage to spinach in Texas in 1937 when it destroyed 25% of the crop.

Initial symptoms of white rust are small lesions on leaves. Lesions coalesce and glassy white pustules which release spores can cover entire leaves causing them to die.

Some cultivars have some tolerance to white rust (rust pustules are fewer in number and stay lower on the plant). Growers can raise the cutting height of harvesters above diseased leaves and harvest early to escape disease heavy losses. Both of these practices reduce yield.

Metalaxyl proved effective in controlling white rust: reducing yield losses from 50% without treatment to 1% with treatment.

6.42 Strawberries

United States strawberry growers use fungicides to control nine primary diseases which include four leaf diseases (powdery mildew, leaf scorch, leaf spot, angular leaf spot), two root diseases (red stele, black root rot), and three fruit rots (gray mold, anthracnose, leather rot). The distribution of these diseases by state is shown in Table 6.

Angular leaf spot of strawberry is caused by a bacterium and was first reported in 1960. Typical symptoms initially appear as small, water-soaked lesions on the
lower leaf surface. These lesions enlarge to form angular spots. A yellow bacterial ooze often appears on the under side of older leaves. When it dries, the exudate forms a white, scaly film. Heavily infected leaves may die, especially if major veins are infected.[129] Research in Florida showed that angular leaf spot reduces strawberry yields by 8%.[176]

Red stele (red core) is a major disease in areas with cool, moist climates. In recent years it has caused particularly heavy losses to strawberry growers in northeastern states.[129] Losses from red stele tend to be more serious where strawberries are grown as a perennial crop because the spores can remain viable in the soil for several years. Secondary metabolites exuded from the roots of strawberry plants attract spores of the red stele fungus which penetrate the roots and then grow into the stele (root core) which begins to turn red shortly after infection.[129] Within a few days after infection the roots begin to rot from the tip. Plants with severe root rot are stunted. Later, the plants produce little or no fruit.[129] Historically, many resistant cultivars have lost their usefulness after being attacked by highly virulent races of red stele that have overcome the genetic resistance of these varieties.[129] Early research with fungicides demonstrated that treated plants had zero infection while untreated plants exhibited 90% infection with red stele.[134]

Leaf scorch is a widespread and damaging foliar disease of strawberry in the U.S.[177] Leaf scorch is recognized by its symptoms on strawberry leaves. Numerous irregular purplish or brownish blotches develop on the leaf. The blotches grow together and the leaf tissue turns purple to bright red.[129] Scorch can seriously weaken plants, so that the growth of leaves and roots declines sharply. Research demonstrated reductions of marketable yield due to leaf scorch of 23-57%.[129]

Because of black root rot, growers in the early 1900s avoided planting strawberries twice in succession on the same land, thinking that somehow the cultivation of strawberries affected the soil adversely.[129] Extensive blackening and death of roots and declining plant vigor and productivity are the most typical symptoms of black root rot.[129]

Leaf spot is one of the most common diseases of strawberry. Before the development of control programs, the economic impact of leaf spot was so great that it was considered the most important strawberry disease.[129] In a 1957 outbreak in Arkansas due to high rainfall, some growers experienced a total crop loss.[133] During this outbreak there were no reports of severe infection in fields where captan had been applied directly.[133] Without fungicide control leaf spot would lower strawberry yield by 20%.[15]

Leather rot was first identified in the U.S. in Arkansas in 1922, where it reduced strawberry production by 20%.[178] In 1964 heavy rainfall during harvesting provided optimum conditions for the development of leather rot in Louisiana. Losses of 15% to 100% due to leather rot occurred in shipments of Louisiana strawberries arriving on the Chicago market.[179] Leather rot was not reported in Ohio until 1981 when fruit losses of 20-30% were common in many fields and some growers experienced a 50% crop loss.[180] As the rot spreads, the entire fruit becomes brown, maintains a rough texture, becomes tough, and appears leathery.[180] Infected fruit dry to form hard shriveled mummies which fall to the ground. As the mummies disintegrate, spores are released in
the soil where they can survive for long periods of time.[129] Fruit affected by leather rot have an unpleasant odor and taste. The smell of leather rot was once detected a quarter of a mile away. Even healthy tissue on a slightly rotted strawberry has a very bad flavor. Growers have experienced complaints from customers about off-flavored jams and jellies after processing fruit from fields where leather rot was present.[180] The level of tolerance for leather rot in a strawberry field is very low.

Ripening strawberries are very susceptible to anthracnose. The pathogen spreads rapidly through fruiting fields during rainy, warm harvest seasons and can quickly destroy a crop.[129] Light-brown water-soaked spots form on ripening fruit and rapidly develop into firm round lesions followed by the eruption of pink spore masses in a slimy sticky matrix which are dispersed by splashing or wind-driven rain.[129] Anthracnose has caused the loss of all fruit in some instances and early abandonment of many fields.[181]

Severe foliar powdery mildew infection damages leaves and reduces photosynthesis through the development of white powdery fungal colonies on the underside of leaves. These colonies grow together and cover the entire lower surface. The leaf edges curl upward revealing the white powdery fungal growth.[129] Powdery mildew-induced yield losses of up to 60% have been reported in the U.S.[127] In California, the marketable yield of a popular variety was reduced by 35% when the disease was left uncontrolled for the entire fruit production season.[127]

Gray mold, also called Botrytis fruit rot, sometimes appears in the field before harvest, especially when there is persistent wetness in the crop, but it chiefly develops in picked fruit. *Botrytis* spores are produced in tremendous quantities and are transported by wind.[130] Infected fruit usually remain symptomless and colonization by the pathogen does not become progressive until the fruit ripens.[129] The fungus produces a velvety gray growth on the surface of the fruit, but in high humidity, the surface growth may be cottony and white.[122] Postharvest fruit rot is often rapid and devastating, quickly rendering fruit unsalable and unusable.[129] Gray mold spreads in shipping containers when the fungus grows from a rotted berry to an adjacent healthy fruit.[132] Early experiments with EBDC fungicides showed that treatment reduced gray mold rot by 63%.[124] Later experiments with captan and benomyl showed that treatments increased strawberry yields by 78-81%.[125][128] Weekly applications of captan or thiram in conjunction with bloom applications of iprodione or fenhexamid are effective against both preharvest and postharvest infections.[135]

The gray mold fungus is estimated to infest 100% of U.S. strawberry acreage with a likely reduction in yield of 45-50% if not controlled.[13][15]

A recent University of California production budget for organic strawberries identifies the typical practice for powdery mildew control to be nine applications of sulfur at five pounds per application per acre.[98] Since no organically approved fungicide has proven effective for gray mold control, this fruit rot is managed by culling diseased fruit. The budget identifies the typical practice to be 225 hours of labor per acre to removed diseased fruit in the field prior to harvest.
Myclobutanil and sulfur are used for powdery mildew control while copper is used for angular leaf spot control. Fosetyl-Al and mefenoxam control red stele and leather rot. Mefenoxam is also used for control of black root rot. The remaining fungicides (benomyl, captan, iprodione, thiophanate-methyl, thiram, fenhexamid, azoxystrobin, cyprodinil, and fludioxonil) are targeted at the fruit rot fungi.

A recent USDA Report estimated the likely state-by-state impacts on strawberry production if fungicides were not used. Yield loss estimates by state ranged from 15% to 90%. The loss estimates are based on the lower efficacy of alternatives that would be employed if fungicides were not used. The alternatives examined in the analysis included planting resistant cultivars, solarization, straw mulch, clean transplants, maximum air circulation, avoidance of excessive nitrogen, raised beds, crop rotation, improved soil drainage, improved weed control, and use of plastic tunnels. USDA concluded that the use of these alternatives in the absence of fungicides would result in severe losses.[137]

### 6.43 Sugarbeets

The sugar recovery rate from sugarbeets is approximately 15%. Sugar produced from sugarbeets totals approximately eight billion pounds annually in the U.S. The majority of sugar produced from the U.S. sugarbeet crop is sold within the country for domestic use. More than half of the sugar produced in the U.S. comes from sugarbeets.

Sugarbeets are biennial and have a two-year life cycle. In the U.S., sugarbeets are grown for only the first year of their life cycle. During this time, the crop is in a non-reproductive stage and produces large storage roots that are harvested for sugar extraction. Sugarbeets are not edible in their natural state. The sugar is extracted in processing plants located near producing areas. Sugarbeets are known for their abundant foliage; sugar content is proportional to the size and development of leaf surface.

Fungicides are used to control two primary diseases of sugarbeets: powdery mildew and cercospora leaf spot.

Powdery mildew of sugarbeets became widespread in California in 1974. The only prior report of this disease in the state was its occurrence on a single plant in 1937.[668] Except for sporadic occurrences in Washington and Oregon, the disease was rare in the United States until 1974 when it broke out in epidemic proportions in California and fourteen other western states. Estimated losses in California in 1974 were as high as three tons/acre in sugarbeet root yield.[670] The sudden outbreak of powdery mildew in 1974 was probably due to the introduction of a more virulent strain of the disease from some other part of the world or the spontaneous development of a new strain in California.[670] The disease now occurs to some extent annually wherever sugarbeets are grown in the U.S.[669] Sugarbeet powdery mildew occurs annually in March or April in the warm, dry inland valleys of southern California. Since the spores cannot overwinter in colder northern climates, it is believed that those formed in the southwest
are blown northward and eastward by prevailing winds to areas where the planting season begins later. The pattern in which the disease spread in 1974 is typical of the annual pattern.[670] (Figure 12)

Powdery mildew spores that land on sugarbeet foliage germinate and start growing on the surface, forming a visible white film that can be seen in 4 to 5 days after infection. The fungus grows through the cell wall of the plant and absorbs nutrients from the leaves. It can spread to all leaves, turning older leaves yellow and killing them. The powdery mildew fungi are unique in their response to humidity. Most fungal spores require 100% relative humidity or free water to germinate. Powdery mildew spores are capable of germination at any humidity. This enables the pathogen to spread during the entire growing season. Spores are produced continuously, day and night, throughout the summer as the disease spreads throughout the crop. If allowed to go unchecked, the disease will cover all the leaves in a field within a month.[671] The disease can cause a significant reduction in yield and percent sugar. No commercial cultivar with significant resistance to the pathogen has been developed. Thus, the only available method of control is the use of fungicides.[669]

Research into powdery mildew control began in California in 1974 and showed that in untreated plots, sugarbeet sugar yields were reduced by 27% or 6 tons per acre.[668] The disease reduced leaf area of the untreated plants by 70%. Research in Idaho demonstrated a yield reduction in beet root tonnage in untreated plots of 22% with a 13% reduction in sucrose content of the root.[672] Nearly complete control of powdery mildew was obtained in tests with sulfur. Control of the disease with sulfur increased sugar yields by 38%.[673]

The fungus causing cercospora leaf spot of sugarbeets overwinters on infected beet residue in the soil. The disease can survive for at least two years in the soil. (Figure 1) Crop rotation is normally practiced by sugarbeet growers, with a three year rotation considered a minimum. Thus, disease pressure is likely to occur from nearby fields that had sugarbeets the previous year. During humid weather, spores are formed and are spread by wind, water and insects. An individual spot on a beet leaf results from the invasion of a germ tube from a spore and the subsequent fungal growth in the tissues of the plant. Successful infection by the fungus usually requires seven days or more. During this time the fungus grows from its initial penetration site to invade and kill cells in the surrounding leaf tissue. The fungus produces a toxin called cercosporin, which produces molecules that attack the fatty acids that make up plant membranes. Eventually, the membranes rupture and the plant cells die. The leaf spot is actually a colony of fungi feeding on degraded plant material.[678] A typical mature leaf spot produces 50 to 100 spores that can travel up to a quarter mile on wind currents.[677] As the disease progresses, numerous individual spots coalesce to form large areas of dead tissue. Severely infected leaves wither and die. The entire plant can be defoliated. If leaf spots cover at least 3% of the foliage by harvest, economic losses occur through reduced root tonnage and lowered sucrose content. Under conditions of severe leaf spot attack, every green leaf may be dried up, causing the field to appear as if it had been scorched.[676] The destruction of the leaf is met by the beet with prompt replacement of
the destroyed area. This replacement is made at the expense of the stored food reserves in the root, resulting in depressed sugar content in the root. A second effect is the checking of growth as the plant turns from growth to repair.[674] Losses due to cercospora leaf spot have gone as high as a 42% reduction in gross sugar and a 32% reduction in root weight.[674]

In the early 1900s, *Cercospora* was the limiting factor in sugarbeet production in many regions of the United States.[676] Experiments in Colorado in the 1920s demonstrated that three applications of a copper-lime mixture at 35 pounds/acre/application controlled leaf spot and resulted in a yield increase of 20%.[676]

*Cercospora* leaf spot was not economically important to the sugarbeet crop produced in the Red River Valley and southern Minnesota before 1980. In the late 1970s growers began switching the varieties they planted from cultivars that were highly resistant to *Cercospora* to ones that were highly susceptible but with higher yield potentials.[675] Favorable weather conditions in 1980 resulted in an epidemic of *Cercospora* affecting 80% of the crop with loss estimated at 4,000 to 6,000 lbs./acre.[675] Economic losses ranged from $250 to $285 per hectare. Starting with the 1981 crop, sugar cooperatives instituted an aggressive calendar spray schedule policy to control *Cercospora*. [680]

Most currently approved sugarbeet varieties in North Dakota/Minnesota are considered moderately resistant to moderately susceptible to *Cercospora*, but yield well. Varieties that are more resistant have lower sugar yield.[677] There are four to five genes responsible for *Cercospora* resistance in sugarbeets. It is difficult to incorporate *Cercospora* resistance genes into sugarbeet varieties with higher agronomic traits because in-breeding may result in poor plant vigor.

Research has shown that there is a strong correlation between susceptibility to *Cercospora* and yield. Greater resistance to *Cercospora* results in less yield. Research has shown that the best economic returns are produced by planting high yielding cultivars, which are susceptible to *Cercospora*, and treating them with fungicides.[680] Research demonstrated a 1.0 ton/acre increase in sugar yield for each increment in increased susceptibility on the *Cercospora* damage scale.[695] (Figure 16)

During the years 1985 through 1995, less than 1% of the sugarbeet acres in Michigan were sprayed for *Cercospora*. Due to the initiation of sugarbeet root aphid in the mid 1990s, varieties were changed towards greater resistance to this insect. These varieties were less resistant to *Cercospora* leaf spot than previously-used varieties, therefore, spraying for leaf spot increased.[693]

*Cercospora* leaf spot control relies on the use of fungicides. Recent research demonstrated that tetraconazole treatments increased the amount of extractable sucrose by 30% in comparison to plots untreated for *Cercospora*. [680] *Cercospora* now infects about half of all U.S. sugarbeet acreage.[679]
6.44 Sweet Corn

Florida and Georgia produce sweet corn exclusively for the fresh market while Minnesota production is exclusively for the processing market. Approximately two-thirds of United States sweet corn production is for the processing market with one-third of the production for the fresh market.

Sweet corn growers use fungicides to control three primary diseases: northern corn leaf blight, southern corn leaf blight, and rust. In Florida, where all three diseases appear every year, fungicides targeted against the leaf blights control rust as well.[592] Fungicide use is lower in mid-Atlantic states because the majority of the crop in these states is produced prior to favorable environmental conditions for disease (late summer, early fall).[10]

Although they contain desirable flavor and horticultural characteristics, many sweet corn hybrids lack multiple-disease resistance.[590]

By 1950, northern corn leaf blight (NCLB) was possibly the factor that most limited sweet corn production in Florida and is still considered the state’s most serious disease.[590] Four races of the disease have been reported in Florida, which complicates attempts to control NCLB using host plant resistance.[590] In 1957, lack of spraying for NCLB resulted in several thousand acres being abandoned and in reduced yield and quality from much of the remaining acreage.[591] In 1955 when the disease was present but never building up to severe levels, the yield reduction was approximately 32%.[591] In 1957, yield reduction in the untreated plots was 90%.[591] The characteristic symptoms are large spindle-shaped dead spots on the leaf blade, up to one half on an inch wide and four inches long. When individual NCLB lesions coalesce, extensive leaf areas can be killed, resulting in plants that resemble those damaged by frost or drought. The lesion area that produces spores may be grayish green. The final disease severity depends on the number of life cycles completed by the fungus on the plant. The number of life cycles depends on favorable weather to promote spore production, penetration and symptom development. Favorable weather includes heavy and frequent rains, high relative humidity, heavy dews occurring nightly, and relatively low temperatures; disease is checked by warm dry periods.[144] Absolute disease control is unnecessary because yield is not affected appreciably unless defoliation exceeds 20% at tassel stage.[144] A very high infection rate of about 40% is possible for short periods during the early stages of epidemic development, when fungicides are not used.[144] Northern leaf blight can be extremely severe in Illinois, Wisconsin, and Minnesota in August and September on susceptible, sugary sweet corn hybrids grown for processing.[585] Fungicide spraying with chlorothalonil, manebe and mancozeb is the major control practice where disease occurs early enough to cause yield loss.[144]

Southern corn leaf blight development is favored by warm climates and is more prevalent in the southern U.S. than in northern areas. Sweet corn losses occur in the field, during transit and in storage. Most documented sweet corn losses involve corn grown in Florida. During the 1950s, SCLB was important in fall plantings. During favorable weather, a
crop could be ruined in a few days if fungicides were not used. In some fields up to 25% of sweet corn ears were affected in one planting. A 24% loss was reported in a shipment of Florida sweet corn during transit to Indiana. Similar losses occurred during transit to Chicago in other shipments of sweet corn from Florida. In 1970, a historic SCLB epidemic occurred in the U.S. on both sweet and field corn. In spite of a regular fungicide program followed for NCLB control in Florida, over 25% of the ears were not marketable in 300 acres of a variety susceptible to SCLB. Loss resulted from fungus penetration through husks into kernels. One 40 acre block had such severe damage that it was abandoned. The financial loss due to this one disease was estimated to be 50% in one 300 acre field in Florida in 1970. Lesions may merge and result in blighting of entire leaves. A black mold characteristic of the SCLB fungus may develop on ears during transit. In North America the fungus survives in corn debris over a wide area that extends at least from Florida to North Dakota and to Ontario. The fungal spores can be carried by wind currents. Mass migration of spores by winds from southern to northern areas was suspected during the 1970 epidemic. One factor that allowed the 1970 epidemic to develop was the presence of such an extensive planting of susceptible varieties; over 80% of all corn planted in the U.S. was susceptible. Rate of disease increase can be reduced by use of resistant varieties. Good control of SCLB is possible with fungicides, including maneb, mancozeb and chlorothalonil.

Early rust infections can weaken plants and result in smaller ears with dehydrated kernels. Later infections typically do not affect yield, but the brown pustules on the husks render ears unsalable for fresh market. Fresh market producers have not widely adopted mechanical harvesting in part because in rust-infected fields, machine-picked ears become covered by rust and are rendered unmarketable. Yield losses due to rust can be up to 20% in processing sweet corn fields with early infections; and up to 50% in fresh market sweet corn (due to cosmetic damage). For many years, rust was considered a disease of minor importance. However, in the 1920s, rust was severe on some hybrids. In the U.S. an epidemic was reported in 1950 on both field and sweet corn. Rust damage was most significant on sweet corn resulting in up to 10% loss of canning corn in Illinois. Common rust is characterized by presence of circular to elongate cinnamon-brown pustules scattered on both surfaces of leaves and on husks, necks, and tassels. As a pustule develops, the epidermis becomes raised and splits open, exposing the cinnamon-brown powder-like spores. Each pustule produces about 5000 spores. A large proportion of inbred sweet corn lines in the U.S. carries general resistance and is considered adequately resistant to rust in the Midwestern U.S., however most are not sufficiently resistant under severe epidemic conditions. Many growers use resistant varieties, but, in some cases, growers plant more susceptible varieties with higher sugar content and taste appeal. Rust has become a greater problem in processing sweet corn in Minnesota and Wisconsin due to the need to plant and harvest very late in the season to maximize the use of processing facilities. Also increased production of corn in the southern Mississippi River Valley provides a closer overwintering site for the fungus, resulting in earlier arrival of spores in the Upper Midwest than in the past. A new race of the rust fungus appeared in the U.S. in the late 1990s. This new race is a strain of the fungus that infects corn with single gene resistance. For twenty years this single resistance gene controlled common rust. It
is likely that this race was introduced from South America, Hawaii, or Southern Africa. Research has shown that rust has its greatest impact on the fill of ear tips resulting in fewer prime ears (-23%), while reducing yield in comparison to fungicide treatment by 10%. Research has also shown that azoxystrobin controls the new race resulting in a percent leaf area diseased of 1.5% in comparison to a 24.3% infection level in untreated plots.[586]

6.45 Sweet Peppers

Sweet pepper growers target five major diseases: bacterial spot, cercospora leaf spot, anthracnose, powdery mildew, and phytophthora blight.

Bacterial spot is most significant in the Southeast. Crop losses result from the actual yield reduction caused by defoliation and from severe spotting of fruit which renders it unfit for market.[310] The bacterial spot pathogen infects all above ground parts of the plant. Spots form on leaves and fruit. Leaves turn yellow and drop prematurely. Rapid defoliation may continue until the pepper plant is devoid of foliage.[311] As new leaves develop, they become infected and are shed before they attain one-third of their normal size. Bacterial spot fruit lesions turn brown. As the spots enlarge, the epidermis ruptures and curls back exposing a raised brown rough wart-like surface. These pod lesions reduce the market value of the peppers.[311] Fruit decay resulting from invasion by secondary organisms through bacterial spot lesions and sunscald resulting from defoliation are secondary fruit problems associated with bacterial spot infections. Up to 70% of fruit in fields have been affected by bacterial spot outbreaks.[144] Research has demonstrated that copper sprays increased the number of marketable fruit by 50%.[312] Further research demonstrated that copper mixed with mancozeb or maneb improved control of bacterial spot in comparison to copper applications alone.[313]

Cercospora leaf spot is also known as frogeye leaf spot because the spots resemble frog eyes. In the U.S. the disease is most common in the southeastern states, stretching from Florida to North Carolina and into Texas.[310] Lesions form on leaves, which turn yellow and drop. Defoliation often results in stunted and irregularly shaped fruit.[144] Even one lesion on a leaf can be enough to cause the leaf to drop. As the lesions expand, an outer water-soaked area and dark ring may form beyond the original border so that the lesion center is surrounded with concentric rings.[310] In 7 to 10 days, the invaded tissue dies, and new spores are formed and disseminated by wind and rain. In Georgia, nearly complete defoliation occurred during rainy summers of 1919 and 1920.[144]

Anthracnose fruit lesions usually develop during ripening. Severe losses can occur when the disease is left untreated and the weather is favorable for disease development.[310] Infected fruit develop dark, circular, sunken spots that are often an inch or more in diameter. These spots become covered with dark, raised specks that are the spore containing bodies of the fungus.[314] The fungus grows through the flesh and then spreads throughout the seed cavity. The fungus secretes a toxic substance which kills host cells before they are actually penetrated by the pathogen.[315] In later stages,
elements of the cell wall are usually pushed out of place indicating that considerable force is exerted by the advancing fungi.[315]

Powdery mildew was not a problem on peppers in California until 1992, when it infected numerous fields in coastal production districts from San Benito County to Ventura County, causing losses of up to 50 to 60%.[316] After 1992, pepper powdery mildew spread to the San Joaquin Valley and the desert production districts and it now can be found in all major pepper production areas of the state. The most noticeable sign of the disease is a white powdery growth on the underside of leaves. Eventually, the entire leaf turns pale yellow. Infected leaves drop prematurely from a plant, exposing fruit to the sun so that they may be susceptible to sunscald.[310] Severe losses occurred in the early years of the epidemic when growers and consultants were unprepared.[316] Since that time, losses have been minimized with the application of fungicides. Research has shown that two applications of myclobutanil at 0.1 lbs. ai/acre provided complete control of the disease.[316] Another effective treatment was 6 sulfur applications at 5 lbs./acre/application.[316] Sulfur is the principal method that organic growers use to control pepper powdery mildew.[316] AQ-10 is a fungus that parasitizes and kills powdery mildew fungi. Research demonstrated that applications of AQ-10 provided limited control early in the season, but efficacy quickly declined and final yield following six applications was no different than the untreated check.[316]

Phytophthora blight is a widespread and devastating disease of pepper and can occur almost anywhere peppers are grown.[310] The disease appears very suddenly and infected plants die promptly. The fungus causes root and crown rot of peppers and distinctive black lesions on stems. Root infection typically leads to wilting and death of the entire plant. The disease can also infect fruit causing lesions that are typically covered with a white fungal growth. The fruit are infected at the stem; then the fungus grows into the fruit and the infected tissue becomes dark green and water-soaked. Within a few days a fruit can be completely infected. Shortly after infection the fruit rapidly dries out, shrinks, and wrinkles.[144] Nearly complete loss of plants can occur in heavily infected fields.[310] In 1958 and 1959 approximately 90% of 1,900 acres of peppers in Broward County, Florida were destroyed by phytophthora root rot.[318] In southern New Jersey, phytophthora blight losses to peppers in 1978 averaged about 20%, however some locations suffered losses as high as 100%.[319] In a few very severely infected locations the growers plowed under the pepper crop before harvest. Phytophthora blight can be controlled chemically with mefenoxam. Because the fungicide is transported systemically through the plant from the roots to leaves, soil applications provide root protection as well as protection of aboveground portions of the plant from splash-dispersed spores. Research demonstrated that applications of mefenoxam in combination with copper increased pepper yield by 63% in comparison to the untreated check, yielding 65,000 peppers per acre in comparison to 40,000 peppers per acre.[320]

Numerous attempts have been made to find sources of resistance to Phytophthora in peppers; yet few resistant cultivars have been commercially deployed.[321] Two resistant cultivars, Adra and Emerald Isle, do not possess sufficient horticultural characteristics to be accepted by the majority of bell pepper growers in the U.S.[321]
The cultivar Paladin possess excellent resistance to the crown rot phase of Phytophthora and has excellent horticultural characteristics; however, it does not possess resistance to the foliar phase of phytophthora blight, the control of which requires regular applications of copper fungicide.[321]

The potential impact of fungicides on sweet pepper yield has been estimated by state in a report from USDA.[10] Florida and Georgia would lose 100% of sweet pepper yield without fungicides while the remaining states would lose 35-80%.

6.46 Tomatoes

U.S. tomato growers used eleven fungicide active ingredients to control eighteen diseases in 2002. Chlorothalonil, mancozeb, maneb, and azoxystrobin control many foliar diseases and fruit rots: black mold, late blight, early blight, gray leaf spot, target spot, leaf mold, buckeye rot, phoma rot, anthracnose, gray mold, and septoria leaf spot. Azoxystrobin is also used to control powdery mildew, as are myclobutanil and sulfur. Copper is used to control three bacterial diseases: canker, speck, and spot. Mefenoxam is targeted at Pythium, Phytophthora, late blight, and buckeye rot. Fosetyl-Al is also targeted at Phytophthora. PCNB controls southern blight, while benomyl provides control of leaf mold, gray mold, and septoria leaf spot.

Target spot, caused by the pathogen Corynespora cassiicola, is a serious disease of tomatoes in Florida. In the past, target spot had not been considered an important pathogen. However, in 1977 the disease defoliated 20 acres of tomatoes and affected 200 acres before being brought under control with fungicides.[365] Since then the disease has increased in both severity and occurrence and has become one of the most damaging wet-weather diseases in the state.[364] Small pinpoint water-soaked lesions first appear on the leaves. These gradually increase in size developing conspicuous yellow halos. Lesions of fruit first appear as dark, sunken, brown spots which enlarge and develop into craters.[364] Ripe fruit develops large circular lesions with pale brown centers which crack open. Research in Florida has shown that chlorothalonil sprays held foliage to disease incidence of 2-3%, reduced from over 90% defoliation that resulted from uncontrolled target spot infections.[366] The disease caused severe fruit loss (up to 91%) on the nonsprayed plots because of the development of fruit lesions.[366]

Gray leaf spot was first noticed on tomatoes in Florida in 1924 when a 30 acre field was totally lost due to the disease.[367] By 1928, the disease had spread throughout the state of Florida, causing widespread defoliation. In the early to mid 1940s, some fields in the mid-Atlantic states were completely defoliated.[144] Currently, it remains one of the most destructive diseases of tomato plants throughout the southeastern states.[364] Gray leaf spot lesions are limited almost entirely to leaf blades. The affected leaves die rapidly, become brown, and are shed.[367] The fungi fruit abundantly on dead leaves lying in moist soil; the fungi overwinter on crop refuse. Spores are spread over extensive areas by wind and germinate quickly in the presence of water producing extensive growth during a single night.[364] Regular chlorothalonil sprays must be used to control the
Leaf mold is most severe where tomatoes are grown in high humidity. The pathogen was first identified in South Carolina, in 1883. By 1896, it had spread to New Jersey, New York, and Ohio. Foliage is generally the only tissue affected. When infections are severe, the foliage is killed. The invaded tissue becomes yellowish brown; leaves curl, wither, and drop prematurely; and defoliation progresses up the plant. An olive green mold is associated with the discolored areas. The loss of foliage retards the growth of fruit and reduces the volume and quality of the crop. Resistance to leaf mold is available in some cultivars, but the value of the resistance is limited because there are at least 12 races of the pathogen. A variety with resistance to one race may be susceptible to other races. Fungicides effectively control the disease. Research in Florida demonstrated that fungicide applications increased tomato yields by 27% by controlling leaf mold.

Septoria leaf spot is one of the most destructive diseases of tomato foliage. In the U.S. the disease occurs in most areas where tomatoes are grown. It is particularly severe in areas where wet humid weather conditions persist for extended periods. In seasons of moderate temperatures and abundant rainfall, the disease often destroys so much of the foliage that either the fruit fails to mature properly or sunscald becomes a major problem. Septoria leaf spot caused near failure of the tomato crop in Michigan in 1915; 80% defoliation in epidemic years before 1924 resulted in an increase in sunscald and yield reduction estimated at 250,000 tons per year for the country. In 1928, a survey of 2,000 tomato acres in Indiana showed that moderate infections (26% of total acres) reduced yields by 19% while severe attacks (14% of the acres) reduced the yield by 34%. With the adoption of routine applications of organic fungicides, septoria leaf spot became a minor problem in the U.S. Currently grown cultivars are susceptible to septoria leaf spot and must be treated with fungicides at regular intervals during the growing season. Several fungicides are effective against Septoria including mancozeb and chlorothalonil, each applied at 7 to 10 day intervals.

Gray mold probably occurs wherever tomatoes are grown. The causal fungus, Botrytis cinera is ubiquitous. Its greatest effect is fruit rot which can occur in the field as well as in postharvest shipments. Diseased plant tissue has a fuzzy, gray-brown appearance which may resemble felt. Clouds of spores can be shaken from the diseased plants after periods of high humidity. The infected areas become covered by the brown fungus, and the leaf collapses and withers. As this occurs, the fungus progresses into the stem and produces cankers which extend for some distance up the stem. Rotted areas may develop on fruit that touches diseased foliage. Infected fruit becomes water-soaked and soft at the point of infection. Lesions on fruit are typical of soft rot with decayed areas being whitish. Usually the skin ruptures in the center of the decayed area. Eventually the whole fruit becomes affected and mummified. The production of halos, called ghost spots, on the fruit is an unusual symptom of this disease. Gray mold ghost spots occur after spores germinate on the surface of the fruit under cool humid conditions and penetrate the surface with germ tubes but are subsequently killed by warm, sunny weather. Although fruit with ghost spots are not rotted, the many halos on the fruit make...
Gray mold is a major fungal disease of tomato in Florida and has caused 100% loss of specific plantings. Gray mold is the most important preharvest and postharvest fruit-decay problem for growers and packers of fresh market tomatoes in California. Losses in fields can reach as high as 35%, a level at which apparently healthy fruits cannot be packed because the risk that they will rot during shipment and storage is too high.

One of the striking features of tomato anthracnose is that lesions develop only on ripe or ripening fruit. Anthracnose lesions on mature fruit arise from infections which occur while the fruits are still green. Following penetration of fruit tissue, the fungus may remain dormant under the cuticle for as long as 3 or 4 weeks. As fruit approach maturity, growth of the pathogen resumes and surface lesions appear. The fruit cell walls in contact with the fungus are pushed aside and slowly dissolved. Cell contents are pushed into a small mass at the base of the cell which stains. Growth of the fungus is rapid and the fungus grows in all directions. The fungus penetrates deeply into the flesh and thousands of spores may be present beneath a single lesion. Cream-colored or pink spore masses ooze from the center of the fruit spot during periods of high humidity. The ultimate size of the sunken fruit spots may vary from one fourth of an inch to one inch in diameter. Red ripe tomatoes which have been picked and allowed to stand overnight frequently are so badly spotted with anthracnose the following morning that they must be resorted to be acceptable at the grading station. A high mold count in processed tomatoes may result from blending a very small percentage of anthracnose-infected fruit with those free from the disease. A single anthracnose lesion causes infected fruit to be considered as culls. Thus, even a trace of anthracnose may prove costly to both the grower and processor because of the additional time required for picking, sorting, and trimming. Tomato processors restrict the proportion of fruit with anthracnose to 3-5%. It was not until 1941 when a new fungicidal material (ferbam) gave such outstanding control of anthracnose that specific experimentation was begun for the purpose of controlling the disease. The average increase in usable fruits due to spraying was approximately 50% or 3 tons per acre. In the absence of sprays the incidence of anthracnose was 22-70% in 1944-1947 in Ohio tests. Chlorothalonil and mancozeb are effective in controlling anthracnose. In a recent experiment in New Jersey, regular applications of chlorothalonil to control anthracnose increased tomato yield 2.3 times over the untreated check and resulted in a financial benefit of $710/acre. Since the fungus is capable of remaining latent in green tomato fruits, effective control with fungicides is dependent upon good coverage of each fruit throughout the entire period of its development on the vine. Ohio researchers have concluded that due to the need for 99% control of anthracnose production of processing tomatoes in the Midwest would be impossible without fungicides.

Early blight is damaging to tomatoes grown in New England, the Southeast, Middle Atlantic, and central states but is of minor importance in California. Early blight is the most important defoliation disease of tomato in the northeast. In Pennsylvania, when controls are not used, potential yield in processing tomatoes is reduced an average of 30% and fruit size is reduced 10%. In Ohio, severe fruit infections in 1943 resulted in up to 35% fruit reduction in a field experiment. Fruit infection in processing tomatoes
can result in high mold counts.

Circular leaf spots containing dark concentric rings are symptomatic of early blight infections. Yellowish areas develop on infected leaves which turn brown and drop from the plant. It is possible for entire plants to be defoliated and killed. The exposed fruit is then subject to sunscald. About 100 spores can be found on a single leaf spot; the same spot can produce up to 4 crops of new spores every 5-7 days. Typical early blight fruit spots occur at the stem end as a rot which is brown to black, up to 1 inch in diameter, firm, leathery, depressed, with distinct concentric rings, and covered by a velvety mass of black spores. Fungicides effectively reduce the rate of infection. Chlorothalonil residues persist and provide protection for 11-13 days. Applications of chlorothalonil to control early blight increased marketable yields of vine-ripe fruit by 22% in Pennsylvania and 87% in Tennessee. In a South Carolina experiment, tomato yield was increased 60% with fungicide sprays to control early blight. The cost of the fungicide treatment ($276/acre) represents less than 1% of the total crop value but can lean to an increase in return of $6,000/acre.

Late blight of tomatoes is caused by *Phytophthora infestans*, a fungus which also causes late blight of potatoes. The late blight pathogen has been responsible for numerous epidemics on tomatoes. In 1946 and 1947, 80% of the early tomato seedbeds in Florida were a complete loss; over 50% of the tomato crop was lost in eastern states from New York to Florida and 25% of the crop was lost in Midwestern states. After an absence of over thirty years, the late blight fungus became an established annual threat to tomatoes in coastal regions of Southern California in the 1980s. The fungus attacks all aboveground parts of the tomato plant. Infected foliage becomes brown, shrivels and soon dies. Lesions can expand rapidly and result in complete defoliation in 14 days. When severe, all plants in a field may be killed in a week or two. On tomato fruit, greenish brown, greasy appearing spots may enlarge to cover the entire fruit. Decaying vines may be identified by a foul odor. Heavy losses can occur in transit; symptoms can occur within 5 days of harvest on fruit that was infected but appeared symptomless at picking. The spores can be disseminated up to 30 or 40 miles by wind or over short distances in dew and splashing rain. Each spore may swim in a film of water on plant surfaces to initiate new a infection. When the weather is favorable, infection moves so rapidly that affected plants appear as though they have been damaged by frost. The use of effective fungicides on a regular spray schedule has contributed to reduced losses to late blight in the U.S. since the late 1940s. Prior to 1947 less than 10% of New York’s tomato acres were sprayed with fungicides. Since the early 1950s, over 90% of New York tomatoes have been sprayed annually. Fungicides are recommended when the weather favors disease development. Fungicide treatments reduce the incidence of late blight to zero in comparison to 97% infection of untreated plants. Gross yields of tomato were increased by 33% as a result of fungicide applications for late blight control. New strains of the late blight fungus entered the U.S. in the 1990s and resulted in tomato production losses of 75-90% in New Jersey and Maryland and 30% in California when left untreated.

Alternaria stem canker (black mold) first appeared in California in the early 1960s.
Currently the disease is limited to California where it occurs every year. [364] Dark brown sunken lesions often with concentric rings may occur on green fruit. The lesions may not always be apparent when the fruit is picked, but may develop rapidly while it is in transit. In the fresh market tomato, up to 75% rot has occurred in transit. [144] The lesions may enlarge to affect up to one third of the fruit surface, usually extend into the fruit wall, and sometimes reach into the seed cavity. [144] The fungus survives for at least 13 months on infected tomato debris in the soil. Infection occurs when spores are blown onto plants or when plants contact infested soil. [364] In humid weather, the fungus produces a black, velvety layer of spores on the surface of the sunken lesions. [392] Black mold reduces yield when heavily infected fruit decay and fall from the vine or truck loads with an 8% or greater incidence of mold are rejected at grading stations. [392] In Solano County, 12% of the fruit was lost to black mold during a three year study. [392] A grower who made a single application of chlorothalonil to control black mold realized a net gain of $395/a. [392] Fungicides are often used since entire shipments can be rejected if black mold infections take hole. Moreover, growers are threatened with the potential loss of entire fields as unfavorable cool, wet weather promotes infection late in the season. [392] An application of a protectant fungicide made several weeks before harvest is relatively inexpensive insurance against the potential of an unharvestable or unmarketable crop. [392]

Phoma rot has been a major problem in the southern United States where it appears sporadically. [364] Major losses associated with phoma rot result from fruit damage that occurs in transit. From 1925 to 1928 about 28% of tomato car-lots shipped from Florida had some phoma rot; the average loss per car due to phoma rot was about 9%. [144] The fungus enters the fruit through growth cracks, stem scars, and mechanical injuries. When it invades the fruit, dark brown to black sunken spots develop. The lesions enlarge and affect large sections of the fruit. The lesions are leathery and have dark areas that contain black speckled eruptions of spores which are exuded in flesh-colored gelatinous coils. [144] During high-moisture periods masses of spores are released. In transit, lesions may expand causing the fruit to be rejected at market. Experiments in Florida in the early 1930s demonstrated that the use of Bordeaux mixture reduced the percent of fruit with phoma rot from 35% to 6%. [393]

Buckeye rot is most common in the southeast and south central states. Buckeye rot is caused by a soil-borne fungus. Numerous swimming spores are discharged under wet conditions. [144] Buckeye rot lesions may cover half or more of the fruit and exhibit pale-brown concentric rings that resemble the markings of a buckeye nut. The skin is not decomposed but the discoloration may extend to the fruit center. Under moist conditions, a white cottony fungal growth appears on the lesion. The pathogen does not affect the foliage. Fungicides applied for late blight aid in the control of buckeye fruit rot. [364]

Phytophthora root rot is a major disease of tomatoes in California. [364] Symptoms of root infections on plants include water-soaked lesions, which gradually dry and turn dark brown. Severely infected roots may be girdled by lesions and the decay may be extensive. [364] Research in California demonstrated that the disease could be controlled by maintaining high concentrations of fosetyl-al in tomato plants over a long period of
Southern blight is also known as Sclerotium stem rot. It is most important in the southern and southeastern states during the warm rainy season.[364] The most common symptom is a brown to black rot of the stem near the soil line. The lesion develops rapidly, completely girdling the stem and resulting in a sudden and permanent wilt of all aboveground parts. On seedlings all tissue is invaded so that the plant dies quickly. Older plants that contain woody tissue are not invaded throughout but are girdled and will finally die. The fungus moves downward from the stem and destroys the root system.[144] The fungus usually forms appreciable growth on the surface of the plant before it is able to enter. It must first develop enzymes that dissolve the outer cell layer before it can penetrate a host.[144] Once fruit become infected, they collapses within 3-4 days. White spore masses quickly fill the lesion cavity.[364] PCNB is used for control of southern blight of tomatoes.[400]

Several *Pythium* species may attack tomato plants during their early stages of growth causing seed rot, damping off, or stem rot. When the dark-colored and soft *Pythium* lesion develops around a major portion or the entirety of the stem, the seedling falls over, withers, and dies.[364] *Pythium* species also cause a fruit rot called cottony leak. The fruit rot usually starts as a small water-soaked lesion on fruit that is in contact with the soil. Within 72 hours the rot engulfs the entire fruit, rupturing the epidermis, collapsing the fruit, and releasing its watery contents.[364] Following an unseasonal rain in early September 1969, heavy losses from cottony leak of ripe tomatoes were sustained in the central valley of California.[399]

Powdery mildew was first reported on tomato in the U.S. in 1978 in the Imperial Valley in California. Since then it has been found in all of the major tomato-growing regions of California.[401] The disease is restricted to warm, arid and semiarid climatic regions.[364] The most common symptoms are yellow spots on leaves. Under conditions favorable for disease development a fine talcum-like powder growth develops on the leaves.[144] Powdery mildew infections have resulted in the loss of 30-40% of the leaf canopy in tomato fields.[401] Defoliation predisposes fruit to sunscald and reduced quality. The tomatoes become soft or are burned before they reach maturity.[403] Some defoliated fields have been harvested early to reduce chances of damage.[402] In a 1994 test, plants affected by powdery mildew had a pack out of marketable fruit of less than one third of what plants treated for the mildew control produced.[403] Other researchers have reported 40% yield losses from sunburn damage associated with severe defoliation by mildew.[402]

Bacterial canker was first observed in 1909 in Michigan and was originally referred to as the Grand Rapids disease.[364] Until 1927, it was believed that the disease was confined to the northeastern U.S. After that time numerous reports of its presence were made throughout tomato-growing regions of the U.S., including southern and western states. The principal symptom of canker is a systemic wilt of the plant. Yellowish white streaks appear on the stems; these streaks often crack open forming cankers.[404] A yellow ooze may be produced from a stem if pressure is applied. The fruit symptoms have been
referred to as bird’s eye spot; lesions with raised brown centers that are surrounded by a white halo. Spot centers become pustular and break open. Internally, fruit contain yellow tissue leading to the seed. The bacteria can reach the seed and penetrate the seed coat. Infected fruit when cut open show extensive yellow discoloration swarming with bacteria.[404] Losses are associated with stunting, wilting, cankering, and sometimes death of infected plants and with fruit spotting.[144] On the most seriously infected plants no fruit reaches normal size.[404] The organism does not produce a rot of the fruit. Losses from culling spotted fruit may be considerable however.[405] Bacterial canker is one of the most dreaded and potentially devastating diseases of tomatoes. Canker was first recorded in North Carolina in 1958 and by 1962 threatened to destroy the industry. Losses to individual growers ran as high as 70 to 80% of the crop.[405] In Georgia, losses ranged from 10 to 50% and in some parts of fields as high as 70%.[404] The bacterial canker pathogen can survive in the soil up to 2 to 5 years.[144] Experiments with copper sprays showed substantial reductions in the spread of the disease. In North Carolina experiments, with no sprays the incidence of bacterial canker was 63% while with copper sprays infection incidence was reduced to 4%.[406]

Bacterial speck was demonstrated as a new disease in Florida in 1931. Since then it has been reported in most tomato-producing areas of the United States.[144] In the 1970s tomato speck became a serious problem on processing tomatoes in many of California’s production areas.[408] Speck is accurately described by its name with individual lesions on fruit ranging from a barely visible fleck of necrotic tissue to lesions 1 mm in diameter.[409] Specks are primarily surface developments and tend to protrude just enough to be detected by touch. Lesions may coalesce in scabby areas covering one-fourth or more of the fruit surface.[409] The epidermis is raised and separated from the underlying tissue by a dense mat of bacteria.[407] The pathogen penetrates deeper into fruit tissue and the plant responds by attempting to black inward process of the disease.[407] Masses of bacteria are extruded from cracks in the fruit-lesion surface.[144] In production fields yield is not frequently reduced, but fruit spotting reduces quality and usable yield of fresh market tomatoes and can increase the difficulty in removing skins from canned tomatoes.[144] Speck lesions may be deep enough to reduce quality after mechanical removal of the skin.[409] In field production tests, up to 70% of fruit have been affected when plants were infected early and controls were not used.[144] Experiments in Florida with copper resulted in excellent control of bacterial speck with the number of lesions per leaf reduced from 107 to 1.[366]

Bacterial spot was observed in Texas in 1912 and in Florida in 1917. Since then bacterial spot has become widespread in the U.S. It is one of the most serious diseases of tomatoes in Florida. Crop losses result from both from actual yield reduction occurring from defoliation and from severely spotted fruit which is unsalable.[364] A general yellowing may occur on leaves with many bacterial spot lesions. Often the dead foliage remains on the plant giving it a scorched appearance. Fruit lesions begin as minute, slightly raised blisters. As a spot increases in size it becomes brown, scab-like, and raised. The center of the spot disintegrates, sinks, and becomes fibrous and rough. Centers may be sharply sunken and form a crater with a ruptured epidermis. Fruit spots are superficial and do not penetrate to the seed cavity. The upper layer of cells are killed while deeper-lying cells
may be stimulated to enlarge and divide so that the center of the lesion is pushed upward to form an elevated scab.[410] Each lesion contains millions of the bacteria. The bacterial mass may ooze out to the surface and produce sticky exudates from which infections may be spread.[410] All plants in a field can become infected from an initial source present at only one edge of the field.[144] Studies in Florida have shown a reduction in marketable tomato yield of 50% from uncontrolled bacterial spot.[411][412] Fruit losses were as high as 5% at canneries and 50% in some Indiana fields in 1918 and 1919. Studies in Indiana in the 1920s revealed that from the canners point of view bacterial spot is very objectionable.[410] Spotted fruit are not suitable for first grade canning stock and can be used only for ketchup. The older lesions are not readily removable with the skin. Research demonstrated that the most effective control of bacterial spot was achieved with combinations of copper with maneb or mancozeb.[413]

Four reports have estimated state-by-state impacts on tomato production if fungicides were not used.[30][10][14][400] The state loss estimates range from 8% in California to 100% in Florida.

### 6.47 Walnuts

California produces 99% of the walnuts grown in the United States and 38% of those grown worldwide. California’s walnut production has increased significantly since 1930. One of the factors that facilitated increased production of walnuts in California was the development of an effective spray program for control of walnut blight. Other factors included increased irrigation, higher yielding varieties, and closer tree plantings.

Walnut blight is the most destructive disease of walnuts. Walnut blight is caused by a bacterium that infects only walnuts. All new growth is susceptible to walnut blight. Symptoms on leaves begin as small dark brown specks surrounded by a yellowish green halo. As the infection spreads, larger areas of dark dead tissue appear. When nuts become infected before the shells harden, the kernels may shrivel, making them unmarketable. Later infections will darken the kernel, lowering the quality of the harvest.[61]

Walnut blight causes little, if any, defoliation of the tree even in seasons especially favorable to its development. If the nut is infected, the infected area enlarges and turns black as the bacteria invade the surrounding tissues. Drops of black slimy exudates containing myriads of bacteria and decomposed cellular materials may ooze out of the lesions during periods of high humidity. The interior of the nut may be infected without the disease being outwardly plainly perceptible. If such a nut is cut open, the central tissue will be found badly diseased. Nuts that are infected early in their development commonly fail to mature as the bacteria generally gain access to the interior and parasitize the tissues within, resulting in premature nut drops lowering yield. If infection occurs after the nuts are about one-half grown, the lesions are usually confined to the outer portion of the fleshy hull. However, in some cases the bacteria may succeed in reaching the shell. In such a case the infected parts usually stick tightly to the shell at
maturity. A brown stain that is not removable by ordinary bleaching agents is left on the shell when the lesions are scraped off, thereby reducing the marketability of the nut.[58]

At first, the bacterial organisms apparently migrate through the walnut tissues in a free-swimming condition. Later, as the organisms increase in number they consolidate and appear to operate collectively, apparently migrating in mass. When the bacteria invade the tissues, the intercellular spaces soon become filled with bacteria and the metabolic products of bacterial activity. Large cavities are subsequently formed, which are filled with bacteria imbedded in a slimy matrix. Enzymic action, osmosis, internal pressures, and perhaps asphyxiation appear to be responsible for the death and disintegration of walnut cellular structure.

The causal organism apparently lives in the dead tissues of blighted buds for at least two and possibly as many as five years after infection occurs. The pathogen overwinters in a relatively large percentage of the lesions on twigs of current growth. During spring blight season and early tree growth, walnut blight bacteria establish themselves inside the following year’s buds. There, the bacteria oversummer and overwinter, causing the host no apparent damage. As buds begin to grow, the bacteria regenerate and infect the surrounding healthy tissues. Lesions develop. When dampened, the epidermis over the lesions erupts and bacteria are released in an ooze, ready for dissemination to new infection sites. This process of bacterial release is repeated every time free moisture is present.

Rainfall is the major contributor to disease spread and infection; thus, walnut blight is worse in wet years.[60] There may be an abundance of inoculum present in the trees; yet, epidemic outbreaks of the disease will not occur unless frequent rains of sufficient duration occur during the infection period. The greater the extent of rainfall during the critical period for infection the greater will be the incidence of the disease.[58]

The organism causing walnut blight is estimated to infect 50% of California’s walnut acreage.[13] Walnut yield loss without control is estimated at 10-90%.[13] Walnut blight is a greater problem in Northern California where spring rainfall increases disease pressure.

Walnut blight was first observed in Los Angeles County California about 1891. The disease spread rapidly to neighboring regions and became increasingly destructive. It was not long until walnut blight became so destructive that the California Walnut Growers Association offered a reward of $20,000 to anyone who could find a practical method of control. In 1905 the California State Legislature, in response to urgent requests from walnut growers of the state for help, granted an annual appropriation of $4,000 for a study of this disease and its control.[58]

In years of severe disease outbreaks as much as 75% of the crop was lost from walnut blight. In 1903, for example walnut blight caused an estimated loss of approximately 50% of the California walnut crop.[58] In 1912, the comment was made that: “It is probably conservative to state that in the seedling groves of southern California the
average loss of at least 50% of the crop which would otherwise have been harvested has been caused by the blight during the past 10 years.”[63] Another early commenter declared that: “Frequently, the average annual losses vary from 75-80% in the San Francisco Bay region as well as in the important walnut growing communities of the San Joaquin and Sacramento valleys, particularly when climatic conditions are favorable to the disease.”[64]

It was not until 1928 that any advancement was made in the development of a satisfactory control program. An extensive series of spraying tests were carried out during the growing season instead of in the dormant period, as had been the case in most of the earlier work. The conclusion from these and later studies was that the disease could be controlled by timely spraying with Bordeaux mixture or with red cuprous oxide during the growing season. Attempts made in 1930 and 1931 to control walnut blight through the use of this spray program met with variable results. Good control of the disease followed its use under certain conditions while under others it failed to control the disease satisfactorily. Moreover, the use of Bordeaux mixture at the recommended concentration resulted in injury to young walnut leaves.[58] (Bordeaux mixture is a 4-1-100 concentration of copper, lime, and water [4 pounds of copper sulphate, 1 pound of caustic lime or 2 pounds of hydrated lime, and 100 gallons of water].)

In 1929, the federal government appropriated funds for further studies of the control of walnut blight. Control studies under this grant were initiated in 1930 and were actively carried out by the United States Department of Agriculture for 16 seasons, 1930 to 1945.

The research demonstrated that the only practical method of controlling walnut blight was by spraying or dusting with protectant bactericides. Of 30 different spray materials tested, homemade Bordeaux mixture consistently provided the most effective control. Copper sprays approximated Bordeaux mixture in effectiveness under normal conditions, but they were not quite so efficacious when conditions for control were difficult.[58]

Control practices for walnut blight have not changed substantially for decades. They are still based upon use of copper-containing materials.

In dry years, nut blight losses can usually be held below 10% with two to three copper sprays. In rainy years, it is difficult, if not impossible, to keep nut blight below 30% regardless of the spray schedule or copper formulation used. Copper materials do not effectively penetrate walnut buds and, therefore, cannot eradicate blight bacteria from overwintering sites.[59]

The number of copper applications varies from 1 to 10, depending upon the length of the rainy season and the blight history of the orchard. Usually, one to three treatments are sufficient in the central San Joaquin Valley’s dryer areas; three to six or more are needed in areas of greater rainfall.[60]

Copper protects leaves and nuts by killing the bacteria before they infect. Copper applications are sprayed on as suspensions. Free copper ions, slowly released from the
suspension, penetrate and kill the bacterial cells by deactivating proteins and enzymes.[73] Sprays must be frequent and provide good coverage to replenish the copper coating and to protect new growth.[61] The University of California IPM guidelines for walnuts state that in orchards with histories of heavy infections and high overwintering bacterial populations, protective copper treatments must be made at 7-10 day intervals during prolonged wet springs for adequate protection.[75]

More than 40 years of copper usage has caused selection for copper-resistant strains of the walnut blight pathogen. In 1990, strains of copper resistant walnut blight bacterium were first found.[69] Research demonstrated that the addition of the EBDC fungicide maneb to copper significantly improved control of resistant bacterium in comparison to standard copper treatments alone.[69] The addition of maneb to copper sprays reduces blight infections 50% greater than copper alone.[76] The copper plots had 6% blight while the plots that were treated with copper plus maneb had 3%. Recent results demonstrate the reductions in walnut blight with copper (-55%) and copper/maneb combinations (-80%).[69]

EPA has granted an emergency registration for maneb usage in California walnuts for ten consecutive years (1994-2003).[74] EPA is currently not considering any permanent additions to the maneb label until a full risk review of the EBDC fungicides has been completed.

Cost of production budgets for walnuts, which have been developed to reflect typical production practices in 2001/2002 in the Sacramento Valley and Northern San Joaquin Valley, are based on 3 sprays for blight control and include both copper and maneb.[70][71]

The use of copper is approved for organic walnut production. A cost of production budget, which includes common practices used by organic walnut growers in the Sacramento Valley, references a single application of 8 pounds of copper hydroxide per acre.[72] The organic budget document notes that control of blight is difficult because of copper resistant strains of the walnut blight bacteria.

One organic walnut operation with 12,000 trees on 400 acres is located 100 miles northwest of Sacramento in the foothills of the Mendocino Mountains. The organic production is possible without the use of fungicides due to the remote location far from central valley problems of blight.

One prominent organic walnut farm in California’s San Joaquin Valley is the Ferrari Farm, which was profiled in the 1989 NAS report Alternative Agriculture.[67] In more recent years the Ferrari Farm has experienced significant losses due to walnut blight because of copper resistant strains of the blight pathogen.[68]

Without the use of fungicides it is estimated that California walnut yields would decline by 50% (midpoint of 10-90% range) on the 54% of the acres treated with fungicide
6.48 Watermelons (included in Section 6.17 Cucumbers)

6.49 Wheat

Wheat is a cultivated grass similar to maize and rice. The history of wheat cultivation dates back to 10,000 years ago and is considered to be as old as civilization itself. In a way, wheat marked the beginnings of agriculture, which transformed human lifestyles from nomadic living to settlement around areas of wheat cultivation due to its dependable harvest. Wheat was milled into flour for bread in ancient Egypt and was the grain of choice during the Roman Empire.

Wheat rust was particularly troublesome around the Mediterranean basin and Roman wheat farmers were plagued by rusts. The Romans created a god, Robigus, the rust god who was honored in the Robigalia, a religious ceremony held on April 25 and practiced for over 1,700 years. There was a temple to Robigus, three miles from Rome; the priest at this temple offered prayers and annually sacrificed a red animal (usually a dog) to appease the rust god in an attempt to spare their grain and avoid rust infections. Climate change in the first century A.D. produced wetter and cooler conditions that led to frequent, severe outbreaks of wheat rust. Crop failure followed leading to famine and social disruption that contributed to the decline of the Roman Empire.

Approximately one half of U.S. production occurs in thirty states where foliar fungicides are normally used.

Foliar fungicides have not historically been used on wheat in the United States because of the low value of the crop. Most years, roughly 20% of U.S. wheat is lost to disease either in the field or in storage.[691] When wheat sells for $3.50 per bushel, savings gained from preventing a 10% loss among a potential yield of 45 bushels/acre is about equal the cost of applying a fungicide. In recent years, farmers using intensive crop management practices have found it profitable to use fungicides.[12] Fungicide use on wheat acres is greatest in Southeastern states where foliar diseases are a more consistent problem because weather conditions are almost always conducive to infections by powdery mildew, septoria leaf blotch, glume blotch and leaf, stripe and stem rust.[681]

Experiments in Tennessee with leaf rust, glume blotch and powdery mildew demonstrated that fungicide applications raised wheat yields an average of 28%. [665] Research in Arkansas with leaf rust and septoria leaf blotch demonstrated wheat yield increases of 20-30% with fungicide application.[666] In Kansas under conditions of severe leaf rust and septoria leaf blotch disease epidemics, a single, well-timed fungicide application increased wheat yields 46-68%.[667]

In general, powdery mildew is a problem in the humid eastern wheat regions, particularly along the Atlantic Coast, the Southeast, the Great Lake states, and in Pennsylvania and Ohio.[12] Powdery mildew occurs throughout the Great Plains and the West, but
infections in these drier areas usually occur too late to damage yields.[682]

The powdery mildew fungus utilizes the nutrients in wheat plants for growth, which reduces the wheat plants’ photosynthetic capabilities. Infected plants lose vigor, and growth, heading and seed filling, is impaired. Heavily infected leaves and even entire plants can be killed prematurely. Yield losses result from reduced head numbers and kernel weight.[688] The pathogen is entirely superficial except for tubes that penetrate epidermal cells and withdraw nutrients from the wheat plant. The fungus appears as cottony white patches, which often merge and cover large areas of the leaf surface. New spores are produced on infected plants and serve to spread the disease during the growing season. New crops of spores are produced every 10 days to two weeks.

The powdery mildew fungus survives between grain crops on volunteer grains and weed hosts or as spore-forming structures on crop debris. When conditions are favorable, spores produced on these sources are transported by wind to infect new wheat crops.[683] Infection and disease development are favored by high humidity, cool temperatures and dense stands. Powdery mildew is controlled to a large extent by planting resistant cultivars. However, in areas where the disease is a consistent problem (Ohio, Pennsylvania and the Southeast) only some cultivars have resistance. These resistant cultivars are not the highest yielding ones available, however.[12] New races of the fungus can develop within a few years and cause disease on varieties that had been resistant.[685] Some varieties last only 1-2 years before a new race of the pathogen develops and breeches variety resistance.[682] When powdery mildew becomes a serious problem in a field, growers have no choice except to apply a foliar fungicide.[686] Yield losses ranging up to 34% have been reported when resistance is lost.[687]

Septoria leaf blotch is caused by two fungi, *Septoria tritici* and *Septoria nodorum*. Leaf lesions caused by *S. tritici* are irregular in shape with more or less parallel sides. Lesions caused by *S. nodorum* are ovular in comparison. Septoria leaf blotch and glume blotch are diseases that occur when there are very long wet periods. Septoria leaf and glume blotch are consistent disease problems along the Gulf of Mexico, the Mississippi Delta, the Red River Valley area of Minnesota and North Dakota, and in western Washington and Oregon. Both types begin as water soaked spots, then become dry and yellow, and finally turn brown. Glume blotch is caused by *S. nodorum*, one of the fungi that causes Septoria leaf blotch. Unlike *S. tritici*, *S. nodorum* also attacks plant stems and heads, causing a much greater effect on yield than *S. tritici*.[684] Under severe disease conditions, yield can be reduced by as much as 50% although losses of 10-15% are more typical.[12] Small spots occur on the lower part of the glumes (husk or outer covering of the flower or grain[chaff]). These spots grow until most or all of the glume becomes discolored. Grain is extremely shriveled in heavily diseased heads. The fungus may infect the surface layers of developing grain. When leaves are wet, spores are produced in the infected areas within a gelatinous matrix that oozes out like toothpaste from a tube. The spores are splashed by rain to other leaves and neighboring plants. The pathogens may produce airborne spores that are spread over long distances. The leaf and glume blotch pathogens survive within infested straw and on volunteer wheat, serving as
sources of inoculum to start off the disease cycle in a new crop of wheat. Most wheat varieties are susceptible to both Septoria species. A few varieties are moderately resistant to one or the other of the pathogens.[684] As cultivars with resistance to S. tritica are more widely grown, S. nodorum is becoming a greater problem.[685] Although all cultivars become infected, disease development is delayed on resistant cultivars. Fungicides have been most effective when applied at the onset of epidemics to slow disease development on leaves and reduce glume infection.[689] Some fungicide-protected fields yield 10-20% more grain than fields in which Septoria leaf and glume blotch are allowed to develop.[688]

In 1916, stem rust destroyed about 38% of the U.S. wheat crop.[646] A stem rust epidemic in the spring wheat regions of Minnesota, North Dakota and South Dakota during 1953 and 1954 caused nearly complete destruction of the durum wheat crop on approximately 2 million acres, and 30 to 35% loss of spring wheat on 10 million acres. This epidemic was caused by a new race of the stem rust pathogen. Another epidemic broke out in 1974 when losses of 5 to 20% occurred in the Southeast. No single race dominated in this epidemic, but a mild winter allowed the pathogen to overwinter throughout a large area.[594] In 1985 Texas lost 28% of its wheat crop to leaf rust.

Three different rust diseases affect wheat in the U.S.: stripe rust, leaf rust, and stem rust. Each is caused by a different fungal species but all have similar life cycles. Each of these species is composed of numerous physiologic races. There are more than 100 known races of stripe rust.[735] Stripe rust pustules tend to grow together into thin stripes on leaves; stem rust occurs primarily on stems; leaf rust occurs primarily on leaves. Stripe rust is prevalent in the western and south central states, leaf rust occurs in all wheat producing regions, and stem rust occurs primarily in the central states and the Pacific Northwest. Rust fungi survive from one season to the next on volunteer grains and some weed hosts. Large spore populations build up in the southern wheat areas in the winter. The spores produced on these hosts can be carried for hundreds of miles on air currents.[683] Spores of the stem rust fungus have been found several thousand feet above the earth.[682] Spores that land on wheat plants germinate and infect the plant through stomata, the natural breathing pores on the surface of leaves and stems. The rust fungus grows between host cells just under the surface or epidermis. Tiny tubes penetrate host cells to withdraw nutrients. Fungus tissue proliferates beneath the epidermis and, as masses of spores are formed, the epidermis bursts and pustules erupt through the plant surface. Each pustule contains tens of thousands of spores that can be carried by the wind to infect other plants. The leaf rust fungus can produce a new crop of spores every 7 to 14 days. Studies of the epidemiology of stripe rust determined that only one infected leaf/acre can cause an epidemic to develop.[653] Photosynthetic output is reduced by loss of leaf area and water loss is increased due to the cracks in the epidermis. Grain yield is reduced by interference with grain filling, which results in a reduction in the number and size of kernels and the formation of shriveled kernels.[683] Infected leaves die prematurely. Under severe disease pressure, yield can be reduced as much as 50% although losses of 10-15% are probably more typical.[12] The first commercial use of foliar sprays for cereal rust control in the U.S. occurred in 1981 in the Pacific Northwest. That year, the use of fungicides prevented the loss of more than 52 million
pounds of wheat in Washington. Similar losses were prevented in Oregon and Idaho.[692] Since then foliar fungicides have become a part of rust control programs.

Many rust races that were prevalent in the past are insignificant today because of breeding efforts that incorporated specific resistances into modern wheat varieties. However, new rust races continually surface to threaten wheat production because of the pathogens’ capacity for mutation and sexual reproduction.[688] An infrequent race can become predominant in a few weeks. Wheat cultivars with leaf rust resistance genes have been available since the mid 1940s in the United States. Within a few years of release, virulent leaf rust strains appear that render the varietal resistance ineffective.[652] Over 45 leaf rust resistance genes have been characterized in wheat. Almost all of these genes condition specific resistance to leaf rust. The near continuous use of wheat cultivars with leaf rust resistance genes in the U.S. has selected fungal populations that have overcome many of the resistance genes. Many wheat cultivars show good leaf rust resistance when initially released, yet within a few years are generally susceptible because of a virulent leaf rust strain.[652]

In 2003 a major stripe rust epidemic caught wheat growers by surprise in California. Yield losses ranged from 5% to 75% causing 18 counties to file for federal disaster assistance [650] Researchers identified 12 new stripe rust races in California in 2003 which led to infections in previously-resistant cultivars.[317] Growers who treated their wheat with fungicides saved a thousand pounds of wheat per acre, even though most of them came in late with their applications.[698]

In Oregon, a popular wheat cultivar showed susceptibility to rust for the first time in 2004 and wheat yields were lowered by 10 to 20%.[697] The State of Idaho estimated that wheat yield loss to rust would be 20% on 500,000 acres without the use of a fungicide.[694] Experiments have shown that the application of tebuconazole reduced wheat foliage loss from 80% to 2%.[681] In field trials in California, fungicides increased wheat yields by up to 50%.[654] In Texas in 2004, it was estimated that fungicide use to control rust would increase yield by 15 to 25%.[651]

In South Dakota, it has been reported that some of the recently released cultivars with better tolerance of Fusarium Head Blight are more susceptible to the currently prevalent races of leaf rust.[690]

In addition to wheat, the stem rust fungus completes part of its life cycle on alternate hosts, especially barberry plants. Europeans noted that the disease was worse when barberry plants were around, and, in the 1600s in France, the first efforts to control wheat rust by eliminating barberries were made. Barberry laws were enacted in several New England colonies in the mid-1700s.

In 1918, after the devastating 1916 U.S. epidemic, a federal barberry eradication program was established. The focus of the eradication program was the Northern Plains states, since the rust fungus could only survive the winters there by overwintering on a barberry plant. This program employed a great number of people during the Great Depression and
over a half billion barberry plants were eliminated. Barberry eradication did not totally control stem rust on the Northern Plains since rust spores blow northward from wheat areas in Mexico and Texas to infect younger wheat plants germinating farther north later in the year. The effect of barberry eradication was to delay potential rust epidemics by several weeks.

Fusarium head blight (FHB), also called scab, is a fungal disease that affects wheat and other small grains. Outbreaks tend to occur when high moisture conditions from rain, dew or humidity coincide with the flowering and grain-fill stages of wheat plants. The fungus overwinters in crop residue left over from corn and small grain harvests. In spring and summer, spores are carried by wind or splashing water to newly developing wheat plants. Infected kernels are shrunken and discolored and their interior develops a floury texture. During prolonged wet weather, the fungus produces pink to salmon-colored spore masses. Under favorable conditions of high moisture and warm temperatures, head blight symptoms can develop within three days of infection. Lush green fields become blighted seemingly overnight.[696] Because disease development is influenced by temperature and moisture during and after flowering, the severity of scab varies greatly from year to year.

Scab caused an estimated loss of 80 million bushels of wheat in the U.S. in 1919. In the 1980s cool wet weather in May and June led to epidemics of scab in the Great Plains. In 1982, scab caused an estimated 4% reduction in total U.S. wheat production: 100 million bushels.[696] In 1993, scab struck the spring wheat areas of Minnesota, North Dakota and South Dakota. In parts of North Dakota wheat yields dropped 45%.[696] Yield and quality losses caused producers in the region to suffer an estimated $1 billion loss. Approximately 18% of the wheat acreage in northwestern Minnesota was not harvested. The scab epidemics in 1993 were associated with seasons of above average rainfall and the widespread adoption of conservation tillage practices, which leaves abundant crop residue on the soil surface.

Scab has recurred in parts of the spring wheat areas annually since 1993. Scab is damaging in areas that receive moisture during flowering and grain fill. In 1996 a scab epidemic hit Illinois ($38 million in losses), Indiana ($38 million in losses), Ohio ($100 million in losses), and Michigan ($56 million in losses, 50% total crop loss).[696] 2003 was generally considered to have been one of the most severe FHB epidemic years ever in Southeastern states. Wheat yields in Maryland were reduced by nearly 60% while in Virginia statewide wheat yield was cut by 40%. In the coastal plain of North Carolina, yields were decreased by 30 to 100%.[700] Minimum losses in 40 counties were estimated at $17 million.

Economic losses to scab occur in several ways. Production is decreased because of kernel shriveling, some kernels being so light that they are blown from the combine. In addition to losses from shriveled kernels, damage from head blight includes reduced yields, discoloration of kernels, reduction in seed weight, and contamination from a mycotoxin produced by the fungus. If the mycotoxin is consumed at high levels, it induces vomiting. Consequently, it is commonly known as vomitoxin. The chemical
name for vomitoxin is deoxynivalenol, or DON. In the 1993 FHB epidemic, if any vomitoxin was found in a wheat shipment, the buying price was reduced by 18%. Wheat producers lost an estimated $86 million due to vomitoxin discounts in 1993. The milling industry seeks scab free wheat to blend with the vomitoxin-contaminated wheat in order to meet FDA levels of concern for vomitoxin. DON is a stable toxin and may remain in stored grain and foods derived from wheat indefinitely.

Improvement in FHB control by fungicide applications is one of the priorities of the “United States National Wheat and Barley Scab Initiative,” which is a $3 million annual federal program. In three trials in North Dakota and one in Illinois, scab severity was reduced by up to 80% by some fungicide treatments. Yield responses with the best treatments ranged from 18 to 23%. Levels of vomitoxin (DON) were also reduced from 28 to 56%. The State of North Dakota has estimated that wheat yield would be increased by 20% on fungicide-treated acres in comparison to untreated acres and that net return would be increased by $23/acre. Disease may be reduced by as much as 60% while yield is increased by up to 20% over untreated fields with the application of fungicides. USDA has estimated that, in 1998, wheat farmers in North Dakota gained 11 million bushels of wheat as a result of using fungicides to control scab on one million acres. The net economic gain for farmers who used fungicides was estimated at $20/acre, for a total net gain of $20 million/year in 1998.

6.50 Wild Rice

Wild rice originated in Minnesota and the surrounding Great Lakes. It is the only cereal native to North America that was domesticated from a wild plant. Before commercial wild rice production began, Native Americans hand harvested it from wild stands. The first commercial field of wild rice was planted in 1950 in Minnesota using seed from natural stands. After one successful season, fungal brown spot destroyed the second crop in this field. In the next decade, fungal brown spot destroyed many other wild rice crops. Epidemics in 1973 and 1974 resulted in complete crop loss in many Minnesota paddies and contributed to the demise of several large wild rice farms.

Wild rice production began in California in 1978, and by the mid-1980’s California production equaled that of Minnesota. Fungal brown spot is absent, or economically unimportant, in the less humid wild rice paddies of California.

In 2000, Minnesota accounted for 29% of U.S. wild rice production while California accounted for the remaining 71%.

Fungal brown spot disease inoculum comes mainly from infected crop debris. When paddies are flooded in the spring, infected crop debris floats to the surface and disease organisms infect the leaves and stems of wild rice as it emerges from the water.
Brown spot forms brown lesions on the leaves and stems, interrupting photosynthesis and preventing seed formation. Infected panicles can become covered by a dense mat of fungus that generates massive amounts of fungal spores that disperse above the wild rice canopy.[45]

After infection, leaves may shrink and die and stems may become girdled and break, causing the panicle to fall and seed to be lost. If flowers and seeds are infected, they become gray.[47] Growers report that the brown spot pathogen infects up to 100% of wild rice acreage in most, if not all, years.[47]

Growers and experts estimate that wild rice losses from fungal brown spot can reach 75-100% without fungicide use but are typically reduced 5-30% with the use of fungicides.[47]

Mancozeb, a protective fungicide, was used on Minnesota wild rice from 1974 to 1988. As a protective spray, mancozeb protected wild rice plants from fungal brown spot for 7 to 10 days after treatment and significantly increased yield.[45] The use of mancozeb was one of the key factors accounting for significant increases in Minnesota wild rice production beginning during the mid-1970s.(Figure 10)

On September 6, 1989, the four technical product registrants of EBDC fungicides applied to EPA to remove 42 of the 55 registered EBDC food uses.[108] The label for the use of mancozeb, an EBDC fungicide, on wild rice was among the uses that was dropped voluntarily as part of this action. Mancozeb’s registration for use on wild rice was revoked on January 2, 1990.

Propiconazole (Tilt), a systemic fungicide, was evaluated for fungal brown spot control potential at the University of Minnesota’s Agricultural Experiment Station in 1985, 1986, and 1987.[49] The research demonstrated that a single application of propiconazole is as effective as two mancozeb applications at controlling fungal diseases late in the growing season. Propiconazole has residual systemic activity, whereas mancozeb is strictly a protectant that can wash off the plant. In addition to its systemic activity, propiconazole provides disease control even when applied after infection has occurred.

In 1985 and 1986, two applications of propiconazole increased wild rice yield by 68% and 40%, respectively, and increased yield by 113% in 1987.[49]

In 1990 through 1992 the State of Minnesota requested, and was granted, emergency exemption registrations to apply propiconazole on wild rice for the control of fungal brown spot. EPA issued a full registration for the use of propiconazole on wild rice in 1993.

USDA recently concluded that without propiconazole, a severe outbreak of fungal brown spot could destroy the entire Minnesota wild rice crop.[47]
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