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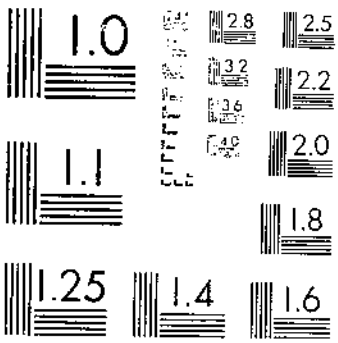
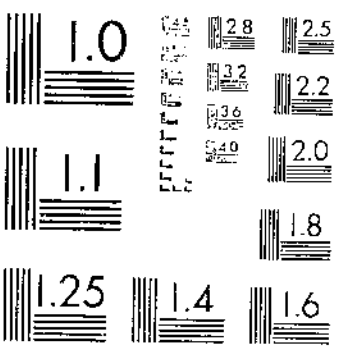


MICROCOPY RESOLUTION TEST CHART



MICROCOPY RESOLUTION TEST CHART

TECHNICAL BULLETIN No. 328 NOVEMBER, 1932

UNITED STATES DEPARTMENT OF AGRICULTURE
WASHINGTON, D. C.

PEACH BROWN ROT

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INTRODUCTION

Brown rot is a destructive disease of the peach, *Prunus persica* Batsch,[2] and is caused by the fungus *Sclerotinia fructicola* (Wint.) Rehm. It is often called "peach rot" or "the rot" because, in the United States, with the occasional exception of Rhizopus rot, it is the only rot of the peach that is of great importance to producer, shipper, carrier, commission merchant, retailer, and consumer. All these at times suffer heavy financial losses because of brown rot. The disease is also the common rot of the cherry, plum, and other stone fruits. The apple, pear, and other pome fruits are sometimes attacked, but in the United States brown rot of these fruits is of slight importance. Peach brown rot is an important disease in all the peach-growing sections of the eastern part and particularly the southeastern part of the United States. It is of less importance in the Middle West but is often destructive in all peach orchards except those in arid or semiarid regions.

[1] The junior writer was transferred from Georgia to Arkansas in the fall of 1928, and the experiments in 1929 were made possible through the cooperation of W. F. Turner, at that time horticulturist for the Central of Georgia Railway. The writers wish to express their gratitude to Mr. Turner for his kindness in collecting and dispatching the cankers.

[2] Scientific names of host plants follow the usage of Rehder (73).[3] Common names follow Standardized Plant Names (2).

[3] Italic numbers in parentheses refer to Literature Cited, p. 54.

The first symptom of the disease on the fruit is the appearance of a tiny brown spot that rapidly develops into a large spot beneath which the flesh is deeply invaded. Soon the whole fruit may be involved if conditions of heat and moisture are favorable to rot development. The rotting of the tissue produced by the fungus is not of the soft watery type commonly caused by species of Penicillium and Rhizopus but is of the solid type similar to the black rot of apple caused by *Physalospora malorum* (Pk.) Shear.

Spore cushions or coremia appear on the surface of the rotted area, frequently within 24 hours after the initial appearance of the disease on the fruit. In a short time most of the surface of the spot is covered with ashen-gray masses of conidia, which are often, but not always, grouped in irregular concentric rings. The invaded fruit that remains attached to the tree slowly becomes dried and shriveled, forming what is usually called a mummy.

When the blossoms are attacked, they quickly turn brown and die. The blighted blossoms are not easily dislodged from the twig and frequently adhere throughout the season. They are covered with masses of conidia, which soon disappear if dry weather prevails; however, new crops of conidia may be produced throughout the season after each period of rainy weather.

The formation of twig cankers as the result of the fungus passing from the floral parts through the peduncles and into the tissues of the twigs is a frequent sequel to blossom blight. Twig cankers may also result from the passage of the fungus from infected fruits through the fruit stems and into the tissues of the twigs, but more often the twigs are killed. Twig cankers are easily recognized as brown, depressed spots at the bases of blighted blossoms or stems of infected fruits.

The leaves adjacent to, or in contact with, twig cankers, blighted blossoms, and rotted fruits are sometimes invaded by the fungus and become brown, but as a disease of peach foliage brown rot is of itself not important. The infected leaves have a water-soaked appearance and are frequently covered with conidial masses. The fungus may occur also on leaves that have been injured by other agencies, particularly the leaf-curl fungus, *Exoascus deformans* (Berk.) Fckl.

HISTORY OF THE DISEASE IN THE UNITED STATES

Most writers on brown rot credit Peck (*64*) with being the first to describe the disease as it occurs in the United States and the first to call attention to its economic importance. Humphrey (*46, p. 85*) states: "* * * the chief handbooks of plant diseases pass it with very brief mention or with none. * * * Here it was first described by Peck, in 1881, and by Arthur, in 1884." Quaintance (*70, p. 246*) makes a similar statement: "In the United States, the disease was first described in 1881 by Dr. C. H. Peck, and in 1884 by Dr. J. C. Arthur." Substantially the same statements are made by Scott and Ayres (*87*) and by Ezekiel (*34*).

Peck (*64*) was probably the first to give a good description of the rot and to demonstrate the pathogenicity of the fungus by means of inoculations. He also discussed the nomenclature of the fungus and included in his paper illustrations of the diseased fruit. Peck un-

doubtedly deserves all the credit that writers on the subject of brown rot have given him. It would be erroneous, however, to infer that brown rot had not been serious and had not been noted by various writers previous to 1880.

Tilton (*96, p. 194*), of Bellevue, near Wilmington, Del., in a letter to Richard Peters, of Philadelphia, dated November 6, 1807, and published in 1815, stated that "a little beetle, called curculio, about the size of a pea bug, is the insect which punctures the fruit, and occasions it to fall off or rot, before it comes to maturity." Evidently brown rot followed curculio in 1807, just as it does at the present time.

In 1817 Coxe (*23*) wrote the first American book on fruit growing. Although peach yellows and fire blight of pear are mentioned, nothing is said of the rot of peaches. However, in the unpublished manuscript for the second edition, which is dated May 30, 1829, and is in the library of the United States Department of Agriculture, the comment "not subject to rotting" follows a description of the peach variety, Morris's Large White Rareripe, on page 736.

In 1852 White (*99, p. 402*), of Athens, Ga., wrote:

> But our greatest obstacle in the culture of the plum is its tendency, in common with several varieties of the peach, nectarine, and grape, to rot before maturing. In a dry season, no matter how hot it may be, the fruit is not in much danger. But in a year like this, of warm, abundant, and continual rain, the cultivator may expect to lose, in the case of most varieties, from half to three-fourths of his crop, and of some it may be the whole will decay. He can guard against this only by selecting the varieties least affected.

Late in the same year he (*100, p. 556*) states:

> But in seasons like the present, the loss of peaches by decay while approaching maturity is more annoying than anything else in peach culture. When the season is warm and wet, very few kinds of peaches will ripen well, especially on moist or very rich soils.

Barry (*7, p. 355*), in his book on fruit growing, published in 1854, states:

> Peaches and other soft fruits should be pressed as lightly as possible, for anything like a squeeze is certainly followed by decay in the form of a brown spot, and this is the reason why it is exceedingly difficult to find a perfectly *sound* and at the same time *ripe* peach in our markets.

Kirtland (*49*) published an excellent description of plum brown rot in 1855 and stated he had known the disease for 30 years. He realized clearly that brown rot is a disease, as the following quotation shows:

> The plum crop, of late years, has generally failed in northern Ohio. The result has been charged to the curculio, and in many instances correctly; but a fatal disease has been insidiously progressing among our fruit orchards which has done more injury than that insect. The effect of the two evils has not usually been discriminated one from the other. Indeed few cultivators seem to be aware of the prevalence of any such disease.

Not only did Kirtland realize that brown rot was a definite disease, but he recognized its fungus origin and made an effort to identify the fungus. In Warder's book on American Pomology (*98, p. 182–183*), published in 1867, the following is quoted from an address by Doctor Kirtland before the Ohio Pomological Society:

> I have watched carefully the sudden and premature decay of our plum crop, at the period of its ripening, for the last fifteen years. From hints offered by the work of Prof. Mitchell, and several microscopic observations of my

own, I was induced to publish an article in "The Florist," of Philadelphia, in the year 1855, in which I imputed the origin of the disease to the Torula or some analogous species of parasite fungi. The disease still prevails among us, and it is sure to destroy all the plums which escape puncture by the curculio. It is, however, generally overlooked by pomologists, and its effects are charged to the depredations of that insect. Similar disease occasionally impairs our peach and apple crops, to a less extent. Whenever it occurs on either of these varieties of fruit, the spurs and young wood blight or canker, and cease to be fruitful for several years.

The blight of peach here mentioned was probably that caused by the brown-rot fungus, but the blight of the apple was more likely that caused by *Bacillus amylovorus* (Burr.) Trev.

Riley (*75, p. 52–53*), in 1869 in a discussion of the curculio, states:

* * * By its punctures it causes the dreaded peach-rot to spread, whenever that disease is prevalent, though it cannot possibly be the first cause of the disease. The peach-rot is now pretty generally acknowledged to be a contagious disease, of a fungoid nature, and I believe that the spores of this fungus, "a million of which might be put upon the point of a stick whittled down to nothing," attach themselves more readily to fruit which has the skin abraded, and from which the gum issues, than to whole or unpunctured fruit. With this belief I made some effort to procure, for the benefit of my readers, a synopsis of the growth of this fungus, but, alas! I find that nothing but confusion exists with regard to it. Upon applying to my friend, Dr. T. C. Hilgard, of St. Louis, a recognized authority on such subjects, he furnished me with the article which may be found in the *Journal of Agriculture* of January 16th, 1869. I most respectfully declined publishing it in these pages knowing that the reader would not be likely to understand what was either too *profound* or too befogged for my own comprehension, and those who require a *synopsis* of this fungus, are referred to that article. Verily we must conclude that Peach-rot is not yet much understood, if a more clear exposition of it cannot be given.[4]

Until quite recent times brown rot was accepted by most people as a thing inherent in the peach and bound to appear in moist weather as the peach reaches maturity, or as caused by the curculio. The fungus, when noticed, was apparently accepted as a natural and normal growth following the rot rather than causing it. A careful examination of Downing's (*30*) book (edition of 1876) on fruit growing fails to disclose a reference to peach brown rot, although the curculio, borer, and yellows are given much attention. Even so late as 1889 Smith (*91, p. 124*) stated:

The peach is well known to be a delicate and perishable fruit, but it is not so generally known through just what agencies this decay occurs. * * * Fruit growers, as a rule, are entirely ignorant of the presence of any fungus. They do not know the cause of the rot, but are painfully conscious of the result, since the latter can be expressed in pecuniary terms. The rot is frequently known as "scald" and is usually ascribed to hot and wet weather * * *.

In 1898 Selby (*89, p. 218*) wrote:

The belief that peaches rot solely because of the weather is often expressed; but while, to be sure, the weather influences the amount of rot, it is only a condition and not the cause of peach rot.

Brown rot of the peach, therefore, appears to have been an important disease for at least a hundred years; but although its true nature was suspected by a few, it was not generally regarded as a definite disease until after 1880.

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[4] In the Journal of Agriculture of Jan. 30, 1869, there is a letter from Hilgard protesting that his paper had been "so utterly and illegibly defaced * * * by grievous errata." Despite ridiculous typographical errors, the paper shows that Hilgard had considerable knowledge of the fungus.

LOSSES CAUSED BY THE DISEASE

In the humid sections of the Eastern and especially the Southeastern States, brown rot is a menace to the peach crop nearly every year. Often it is the principal topic of conversation as peaches begin to ripen, for the orchardist well knows that at this time moist weather may favor the development of brown rot to such a degree that the crop which he has nurtured so carefully for many months may be destroyed as it reaches maturity.

Were it not for the control methods in common use, many sections in moist years would suffer an almost total loss. In seasons of high rainfall the writers frequently have seen the entire crop of individual orchards destroyed by the disease and the crop of whole sections reduced by as much as 75 per cent. In such years a large percentage of the remaining fruits, which are soft and watery and covered with conidia of the brown-rot fungus washed down on them from the infected fruits, may rot soon after picking. In regions of little rainfall peach brown rot is not present or, if present, is not important.

Smith (*91*) estimated the loss to peach growers of the Delaware-Chesapeake Peninsula in 1888 as $400,000. Quaintance (*70*) considered the loss to Georgia growers in 1900 as somewhere between $500,000 and $700,000. For the whole of the United States, Scott and Ayres (*87*) in 1910 estimated the annual losses caused by brown rot to be at least $5,000,000. They estimated that with a fair crop and average brown-rot conditions the loss to Georgia growers alone was $1,000,000 annually.

Orton (*62, p. 583*) in 1904 reported:

> Brown rot (*Sclerotinia fructigena*) varied in severity according to the weather in different sections. The main peach crop of middle Georgia was nearly free from rot. There was much greater loss in north Georgia, amounting to 15 per cent of the crop. In Maryland plums suffered most, the loss on varieties like Wickson and Abundance being 30 to 100 per cent, while early peaches were a complete loss and midseason varieties rotted badly. In the Northern States the disease occurred about as usual where the crop had not been destroyed by winter injury. There was little in Michigan.

In 1905 Orton (*63, p. 604*) further reported:

> Brown rot * * * was on the whole more destructive than usual. In Georgia the loss was greater in the southern than in the middle and northern peach sections of that State, and amounted to one-third of the crop, or 800 carloads. Maryland, New Jersey, and Connecticut suffered severely. In one instance in Pennsylvania 20 carloads were lost. The estimated damage in Ohio was $250,000. There was serious injury to the crop in West Virginia.

Two decades later the plant disease survey of the Bureau of Plant Industry, United States Department of Agriculture,[5] reported that in 1924 brown rot reduced the yield of peaches in the United States by 2,392,000 bushels, and in 1925 by 692,000 bushels. The average annual reduction for the years 1917 to 1925, inclusive, approximated 3,200,000 bushels. In 1926 the reduction in yield was estimated at 3 per cent, or 1,868,000 bushels; in 1927, it was 5 per cent, or 2,379,000 bushels.

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[5] UNITED STATES DEPARTMENT OF AGRICULTURE, BUREAU OF PLANT INDUSTRY. CROP LOSSES FROM PLANT DISEASES * * * Plant Disease Bul. Sup. 6 (1919), 12 (1920), 18 (1921), 24 (1922). Plant Disease Rptr., Sup. 30 (1923), 36 (1924), 43 (1925), 49 (1926), 56 (1927), 64 (1928). [Mimeographed.]

These estimates, however, do not include the losses due to rot after the peaches have left the hands of the grower. Each year losses in transit and on the market are large, and frequently enormous. Peaches from sections where brown rot is prevalent, when shipped long distances, even in refrigerator cars, are sure to suffer from rot to some extent, especially in years of heavy rainfall, when peaches contain a high percentage of water and conidia of the fungus are invariably present. Frequently the loss is 25 per cent or more. Then in commission houses, stores, on fruit stands, and finally in the hands of the consumer, brown rot takes further toll. Beginning about 1908, the losses both in the orchard and in the market have been greatly reduced by the application of sprays to the developing fruits.

It is probable that most estimates do not include losses from the blossom-blight phase of the disease, which occasionally may prevent the setting of a full crop. The writers have seen cases in which 95 per cent of the blossoms of individual orchards have been killed by this phase of the disease.

VARIETAL SUSCEPTIBILITY

The modern commercial varieties of the peach show considerable resistance to brown rot in normal years. One of the reasons why they have supplanted the older and often better-flavored varieties is that they are less susceptible to brown rot in the orchard, in transit, in the market, and in the home.

Because of differences in time of ripening it is impossible to classify varieties according to their relative susceptibility to the rot phase of the disease, since in the same season the conditions at the time of ripening of one variety may be favorable to the development of rot, and at that of another variety unfavorable. Varieties that are normally resistant may in seasons of excessive rainfall produce fruit which, in place of having the usual firmness, is so soft and watery that it is very susceptible. Frequently the percentage of fruits punctured by the curculio determines the percentage of fruits affected with brown rot, regardless of the variety.

Practically all modern commercial varieties are more resistant to the blossom-blight phase of the disease than are the older ones, but all may be quite susceptible under optimum conditions for infection.

THE FUNGUS CAUSING THE DISEASE

TAXONOMIC POSITION

A discussion of the taxonomic position of the fungus causing the brown-rot disease of peaches in North America should start with the classification of similar fungi in Europe, because early American workers were in close contact with European mycologists, used the European systems of classification, and in a number of cases submitted their American specimens to European workers for identification. Reliance on European mycologists was logical and would have eliminated the confusion that now exists if, as was thought until recently, the American organism had been identical with a previously known European organism.

In 1796 Persoon (*65*) described a fungus causing a rot of fruit in Europe under the name of *Torula fructigena*. In 1801 he (*66*) decided that it belonged to the genus Monilia and renamed it *Monilia fructigena*. Kunze and Schmidt (*50*) considered that it should be called *Oidium fructigenum*. After considerable discussion by various writers, *M. fructigena* became the generally accepted name, although *O. fructigenum* was also used.

American mycologists early in the nineteenth century knew that a fungus was associated with the rot of peaches but apparently were not interested in considering whether it caused the disease or not. In fact, one infers that they assumed it to be a saprophyte developing in the rotted fruit.

De Schweinitz (*35, p. 128*), in his Synopsis Fungorum Carolinae Superioris, supposed to have been published in 1822, reports as item No. 130 "Torula fructigena. In Persicis frequens."

In Michener's collections,[6] volume 23, sheet 32, there is a specimen labeled "*Oidium fructigenum* Per. in fruct. Pruni. S. Car. dedet Ravenel." Also on sheet 34, "*Oidium fructigenum* Per. in fruct. putrescent. Pruni. S. Car. H. W. R."

The influence of the European workers is clearly shown in the names ascribed to the fungi collected by these early American investigators, and even as late as 1913 the American fungus was considered to be identical with *Monilia fructigena*.

In 1851 Bonorden (*13*) described another species occurring on fruits in Europe and named it *Monilia cinerea*. Although it had been accepted by Saccardo (*83, v. 4, p. 34*) and by Schröter (*84*), little attention was paid to this name until 1900, when Woronin (*111*) showed that the *M. fructigena* of Persoon and the *M. cinerea* of Bonorden were distinct species.

Eugene Rau, of Bethlehem, Pa., found in May, 1883, in a garden a decayed peach on which was growing the apothecia of a fungus. He kept part of this material with his own collection and sent a part to Winter (*104*), who described the fungus as a new species which he named *Ciboria fructicola*. Saccardo (*83, v. 18, p. 41*) accepted Winter's new species and copied Winter's description in the Sylloge Fungorum but placed it in the genus Sclerotinia on the authority of Rehm. The name appears in the Sylloge Fungorum as *Sclerotinia fructicola* (Wint.) Rehm.

In 1888, five years after Rau's discovery, Woronin (*110*) demonstrated the connection between a Monilia and a Sclerotinia growing on *Vaccinium vitis-idaea*. This discovery led Schröter (*84*) in 1893 to propose that *Monilia fructigena* and *M. cinerea* be placed in the genus Sclerotinia with the names *Sclerotinia fructigena* and *S. cinerea*, respectively. This is apparently the first time that these two fruit-rotting fungi were placed in the genus Sclerotinia, and it must be pointed out that Schröter had no grounds for his assumption other than analogy with the fungus on Vaccinium. He either overlooked Winter's description of *Ciboria fructicola* or failed to attach any significance to it.

The next development came in 1902 with the finding by Norton of apothecia of a species of Sclerotinia developing from peach mum-

[6] The Ezra Michener collection of fungi, in the herbarium of the Division of Mycology and Disease Survey, Bureau of Plant Industry, U. S. Department of Agriculture.

mies. By cultural work he demonstrated beyond any doubt their connection with the common Monilia causing a rot of fruits. Since this Monilia was generally believed to be identical with *M. fructigena* of Europe, Norton (*59*) named the fungus *Sclerotinia fructigena* (Pers.) Norton.

In 1905 Aderhold and Ruhland (*1*) reported the discovery of a Sclerotinia which they demonstrated to be the ascogenous stage of *Monilia fructigena*. They also declared the American fungus to be *Sclerotinia cinerea*, since the Monilia form of the American fungus and *M. cinerea* of Europe are so nearly alike. Their description of *S. cinerea* is based on preserved apothecia from the United States and on conidial material collected in Europe.

In the United States the work of Aderhold and Ruhland was not generally accepted, and *Sclerotinia fructigena* was commonly used as the name of the peach-rot fungus until about 1913, when Matheny (*56*) after studying fresh material from Europe and North America showed conclusively that the fungus was quite different from *S. fructigena* but resembled *S. cinerea* very closely. Matheny's conclusions have been verified by Conel (*18*), Bartram (*9*), Jehle (*48*) and others who have examined material or grown the fungi in culture. *S. cinerea* was generally accepted as the correct name for the common American fungus, and its identity with the European form was not questioned until about 1920.

In 1919 and 1920 Wormald (*105*) distinguished a number of different forms of *Sclerotinia cinerea*. Cultures of the American form fruited more readily on artificial media than did the strains occurring in England. Other differences between the Monilia forms were noted, including differences in enzyme production, in manner of growth on artificial media, and in length of germ tubes before branching. Wormald regarded the American form as distinct and referred to it as *Monilia cinerea* forma *americana*.

In 1921 Wormald (*106*) announced the discovery of a species of Sclerotinia which he showed to be the perfect stage of *Monilia cinerea*. He accepted the name *Sclerotinia cinerea* (Bon.) Schröter for the fungus, the ascogenous stage of which Schröter had never seen.

In 1923, at the December meeting of the American Phytopathological Society, Norton and Ezekiel (*61*) proposed the name *Sclerotinia americana* (Wormald) comb. nov. for the American form. They stated:

> The species mentioned can be differentiated macroscopically on potato-glucose agar plates by variation in the rate of growth, production of conidia and aerial hyphae, and shape and elevation of the colony.

At the time Rau made his discovery of apothecia arising from decayed peaches no connection between Monilia and Sclerotinia had been recognized or even postulated, so European and American investigators gave no particular attention to his discovery. It was not until 1909, 21 years after Woronin's discovery of the connection between Monilia and Sclerotinia, 16 years after Schröter's assumption that sclerotinial stages of *Monilia fructigena* and *M. cinerea* existed, and 7 years after Norton's demonstration of the sclerotinial stage of the common American fungus, that attention was called to the fungus collected by Rau and described by Winter. In that year

Pollock (*67, p. 53*) pointed out that since Winter's measurements of asci and ascospores agreed very closely with those given by Reade for the ascogenous stage of the American fungus, Winter must have had that species. He stated:

> If the rule of priority is to apply to the species name first associated with the perfect stage, the correct name of this fungus is Sclerotinia fructicola (Winter) Rehm instead of Sclerotinia fructigena (Pers.) Norton.

In 1920 Pollock[7] again called attention to *Sclerotinia fructicola*, which seemed to him to be the correct name of the common American brown-rot fungus, particularly since he now considered the American fungus distinct from European forms.

The writers (*77*) in 1924 reported that they had examined and made measurements of cotype specimens of *Sclerotinia fructicola* found in Rau's collections. They showed that the fungus collected by Rau and described by Winter was morphologically identical with the species of Sclerotinia commonly found on decayed peaches at the present time. They stated that if the American form is considered as a distinct species it should be called *Sclerotinia fructicola*, since that name had priority over *S. americana*. At that time, however, since the writers were not convinced that the American form was sufficiently distinct from the European to warrant specific rank, they preferred the name *S. cinerea*.

Ezekiel (*34*) in 1924 confirmed and enlarged upon Wormald's work. He showed that the American form and *Sclerotinia cinerea* forma *pruni* differ as to germ tubes, oxidase production, length of hyphal cells, rates of growth on artificial media and in inoculated fruits, and in the manner of growth on artificial media. Many of these differences had been noted by the writers but were not considered sufficient to constitute a distinct species, since the American form itself showed considerable physiologic variation.

Jackson (*47*) and Posey (*69*) as early as 1915 and Barss (*8*) in 1923 called attention to a Monilia attacking fruits on the Pacific coast which they reported as different from the common one found there. Barss (*8*) proposed for it the name *Monilia oregonensis* Barss and Posey. Ezekiel (*35*) in 1925 considered this fungus to be identical with *M. cinerea* forma *pruni* of Europe. He grew the different forms in culture and showed the close resemblance in physiologic behavior between *M. cinerea* of Europe and the Pacific coast form. The writers in 1927 (*79*) confirmed Ezekiel's results. Wormald (*107*) also confirmed them, affirming the Pacific coast fungus to be identical in culture with *M. cinerea* forma *pruni*. Since the typical American form and the fungus occurring on the Pacific coast can be distinguished in nature and in herbarium specimens as well as in artificial culture, the writers have receded from their former position and consider the common American form a distinct species, but they maintain that, since they have shown its identity with a fungus first described as *Ciboria fructicola*, the name *Sclerotinia fructicola* should be accepted as correct. Ezekiel (*35*) still prefers the name *S. americana* because there is, at present, no way of dis-

[7] POLLOCK, J. B. IDENTITY AND THE NAME OF THE SCLEROTINIA CAUSING THE BROWN ROT OF STONE FRUITS IN THE UNITED STATES. Paper read before the mycological section of the Botanical Society of America, Chicago, Dec. 30, 1920. (Unpublished.) Information also given in a letter to the senior writer.

tinguishing the apothecial stage of the common American fungus from that of *S. cinerea*. Wormald (*108*) has accepted *S. americana*, believing that the earlier name may have applied equally well to apothecia of *S. cinerea*. Wormald (*109*) takes this stand while pointing out that *S. cinerea* has been found only in the Pacific coast section of the United States, which is more than 2,000 miles from Pennsylvania and is about two-thirds as far from that State as the latter is from England. It should also be pointed out that the apothecial (Sclerotinia) stage of *S. cinerea* has never been found in the United States and that failure to find it has thrown some slight doubt on the accuracy of the determination of the Pacific coast fungus as *S. cinerea*. If these statements are correct, then it seems inconsistent to accept *S. cinerea* for the Pacific coast fungus, and to reject *S. fructicola* in favor of *S. americana* for the common brown-rot fungus.

Furthermore, it seems illogical to reject the name *S. fructicola* as Ezekiel and Wormald have done on the ground that the technical description does not distinguish between *S. fructicola* and *S. cinerea*. When *S. fructicola* was described in 1883, there was no description of the apothecial characters of *S. cinerea*. The apothecia of the latter were not found until 1921 (38 years later).[8] It seems to the writers that unless the apothecial characters of *S. cinerea* can be shown to be different from those of *S. fructicola* the question is really centered about the validity of the name *S. cinerea* and not about *S. fructicola*.

Many American investigators accept *Sclerotinia fructicola* as the correct name for the fungus, and Harrison (*40, p. 113–114*), an Australian investigator, states:

> The first valid name applied to the apothecial stage of the American Brown Rot fungus should be universally adopted when describing that fungus. Throughout this paper, therefore, the name *S. fructicola* (Wint.) Rehm will be used in preference to *S. americana* (Worm.) Norton and Ezekiel.

Honey (*44*) has recently chosen *Sclerotinia fructicola* as the type of a new genus, Monilinia, which is to include those forms formerly included in Sclerotinia that have a conidial stage belonging to the imperfect genus Monilia. Acceptance of this classification would give the brown-rot fungus the name *Monilinia fructicola* (Wint.) Honey. Hino (*43*), on the other hand, because of the existence of intermediate forms, thinks that the genus Sclerotinia should not be divided.

Granting that the common American fungus belongs to the genus Sclerotinia, then there are four names—*S. fructigena* (Pers.) Schröt., *S. cinerea* (Bon.) Schröt., *S. fructicola* (Wint.) Rehm, and *S. americana* (Wormald) Norton and Ezekiel—that must be considered before deciding on a name. Of these, *S. fructigena* has been shown by Woronin (*110*), Aderhold and Ruhland (*1*), Wormald (*105*), and others to be a name applied to a European fungus which Pollock (*67*), Matheny (*56*), Conel (*18*), Bartram (*9*), Jehle (*48*), and Wormald (*105*) have demonstrated is quite different from any known

[8] If *Sclerotinia cinerea* is considered identical with *S. laxa*, the year would be 1904 and the interval 21 years (Aderhold and Ruhland, *1*). T. H. Harrison, of the Hawkesbury Agricultural College, Richmond, New South Wales, Australia, in March, 1932, showed the writers an unpublished manuscript in which the identity of *S. laxa* and *S. cinerea* and the priority of the name *S. laxa* are demonstrated.

American species. The principal differences noted by them are as follows:

The conidial tufts do not agree in size, shape, or color. Those of *Sclerotinia fructigena* are yellow or buff in color and often grow together to form a smooth upper surface. Those of the common American form are ashen gray in color, much smaller, and do not coalesce.

The conidia of *S. fructigena* are regularly and consistently larger than those of the common American fungus. The former are short lived, the latter relatively long lived.

Although measurements of the asci and ascospores of both forms correspond very closely, the ascospores of *S. fructigena* are sharply pointed at each end and are free from oil droplets, whereas those of the American form are rounded at the end and possess oil droplets.

The American fungus usually rots fruits much more rapidly than does *S. fructigena*.

In artificial culture *S. fructigena* and the common American fungus behave quite differently.

The writers have grown *Sclerotinia fructigena* in artificial culture and have performed inoculation experiments with it. They also have examined fruits naturally infected by this fungus. They are in complete agreement with the foregoing statements. It should be added that the conidial tufts of *S. fructigena* are mainly composed of mycelium rather than conidia, whereas those of the American form are mainly composed of conidia. The conidial chains of the latter are much more easily broken up than are those of the former. All who have investigated the subject are agreed that *S. fructigena* is a species quite distinct from any form known to occur in North America.

After it had been shown so conclusively that *S. fructigena* of Europe is not identical with the common American form, *S. cinerea* became the commonly accepted name for the latter, because the conidial measurements were approximately the same and because *S. cinerea* was supposed to be serious as a disease of stone fruits only. Comparative studies of the Monilia stages of *S. cinerea* from Europe and of the American fungus, made by Pollock (*67*), Wormald (*105*), Norton and Ezekiel (*61*), and Roberts and Dunegan (*77*), revealed certain differences between the two organisms; and when Jackson (*47*), Posey (*69*), Barss (*8*), and Rudolph (*82*) concluded that another brown-rot fungus exists in the United States, the Monilia stage of which Ezekiel (*35*) has shown to be similar to, and probably identical with, that of the true *S. cinerea* of Europe, it became evident that the common form could no longer correctly be called *S. cinerea*.

The chief reasons for considering the common American form distinct from *S. cinerea* are as follows:

S. cinerea causes principally a destructive blossom, spur, and twig blight of both pome and stone fruits rather than a fruit rot primarily of stone fruits.

It forms on the blighted parts persistent conidia-producing mycelial cushions which are quite different from the evanescent tufts of conidial chains produced on diseased parts by the common form. From these cushions conidia are produced mainly in late winter or early spring of the year following infection. From twig cankers caused by the common form, tufts of conidia are produced soon after the blight begins to appear, and may continue to be produced on the cankers at intervals throughout the season, but are produced rarely in the late winter or early spring of the year following infection.

No apothecial stage of *S. cinerea* has been found in the United States, whereas the apothecial stage of the common form is abundant.

The common American form usually rots fruits much more rapidly than does *S. cinerea*.

The lengths of the conidial germ tubes before branching and the manner of branching are different in the two forms.

The common American form gives a much more powerful test for the presence of oxidases than does *S. cinerea*.

On culture media the common American form usually produces conidia more readily and grows more rapidly. The margin of its growth is usually even, whereas that of *S. cinerea* is usually scalloped or lobed.

Sclerotinia americana can not be accepted as a valid name because it is antedated by *S. fructicola*.

S. fructicola should be accepted as the correct name for the common American fungus because it was the first name to be applied to it. The proposal of the name was accompanied by an adequate description and complied with all the customary rules of nomenclature.

The reasons for considering the apothecia, collected in 1883, and described as *Ciboria fructicola* by Winter in the same year, identical with the apothecial stage of the common American fungus are as follows:

The apothecia collected in 1883 developed from sclerotia investing a decayed peach.

They were found in Pennsylvania, where brown rot is a serious disease, and in May, which in the latitude of Pennsylvania is within the season for the production of apothecia by the fungus.

The original collection has been examined and found to consist of dried apothecia and sclerotia. Measurements of apothecia, ascospores, asci, and paraphyses from this collection correspond closely with those of the common brown-rot fungus collected at the present time.

Apothecia of no other species of Sclerotinia which could be confused with those of the common brown-rot fungus are known to occur on fallen-peach mummies in Pennsylvania or elsewhere in the United States. The apothecial stage of the only other species of Monilia known to occur on peaches in the United States has never been collected in this country, even on the Pacific coast, which is the only section in which the fungus is known to occur.

GEOGRAPHIC DISTRIBUTION

The fungus *Sclerotinia fructicola*, causing the common brown rot of fruit in the United States, has also been reported from Canada, Australia, and New Zealand. Wormald (*107*, *108*) states that he received a culture from Holland which was indistinguishable from the common American form. It is doubtful, he thinks, whether the common American brown-rot fungus occurs naturally in Europe.

A fungus considered to be identical with *S. cinerea*, common in Europe, occurs on the Pacific coast of the United States and Canada along with the common form, but so far as known does not cause a serious fruit rot of the peach.

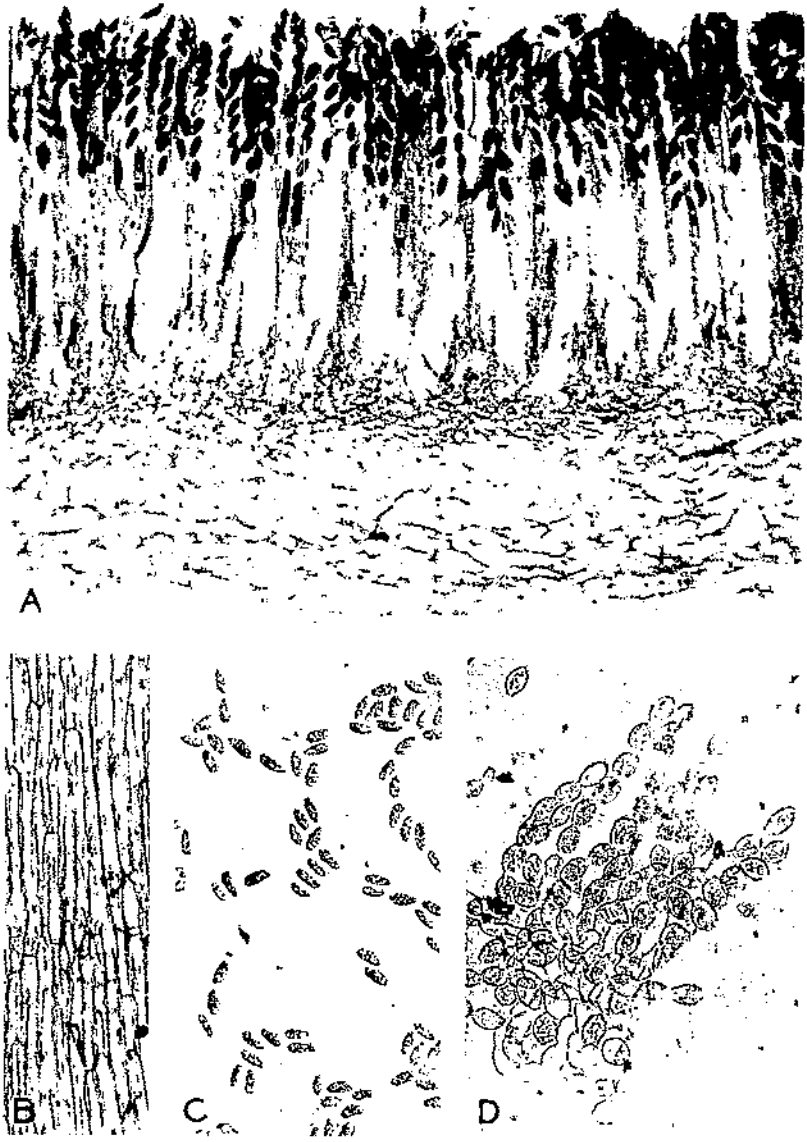
MORPHOLOGY

APOTHECIA

In 1902 Norton (*59*, *p. 93*) published an excellent account of the apothecia of the fungus. The following description is quoted from his paper:

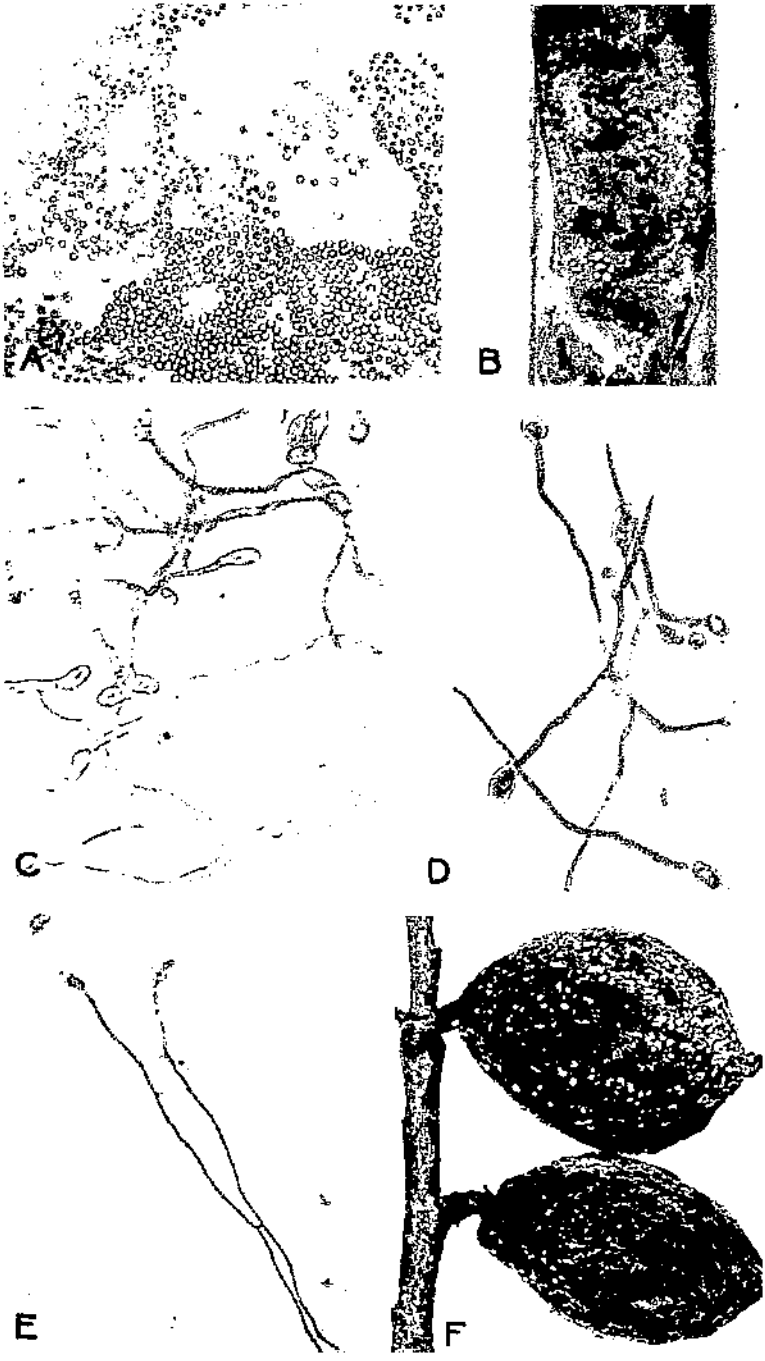
The apothecia arise from the familiar sclerotia in the tissues of the so-called mummy fruits beneath the soil or occasionally on the surface in moist places. Usually several arise from the under side of each fruit and appear in a ring around it at the surface of the ground, from 1 to 20 appearing above one fruit.

The sinuous stipe is from .5–3 cm. long, depending on the length it must grow to bring the spore-bearing surface above the ground. It is from .3–1.5



SCLEROTINIA FRUCTICOLA

A, Portion of the hymenium of an apothecium, showing mature asci and spores; B, portion of the stipe of an apothecium, showing the structure; C, ascospores; D, conidia. All ×310.



SCLEROTINIA FRUCTICOLA AND PEACH BROWN-ROT MUMMIES

A, Microconidia. × 340. B, Culture 77 days old on oatmeal agar, showing masses of microconidia collected on the surfaces as viscid droplets. × 1½. C, Germinating ascospores. × 340. D, Germinating conidia. × 340. E, Germinating conidia, showing anastomoses between parallel germ tubes. × 340. F, Brown-rot mummies, Uneeda variety, covered with fresh pustules of conidia. Collected December 21, 1923. Natural size.

mm. thick. The lower part is covered with closely adherent particles of soil entangled in a mass of slender dark-colored septate rhizoids 1 mm. or less in length. These gradually disappear upward, the upper part of the stipe being smooth. The color is dark brown below running into the lighter brown of the disk above. The body of the stipe is made up of somewhat elongated cells in the center with short dark-colored cells on the outside, composing the cortex which continues around the outside of the disk and projects at the edges somewhat beyond the hymenium. The subhymenium is composed of elongated intertwined cells much like those in the center of the stipe.

The stipe enlarges into the at first campanulate disk slightly broader below the top. The disk widens out until cup-shaped and finally flat. Older ones often have the edges torn and recurved. The disk becomes again campanulate in drying up and is then darker colored. The expanded disk is from 2–15 mm. wide, usually about 5–8 mm. In its later stages it is often whitish from a deposit of spores.

The structure of the hymenium and the stipe is illustrated in Plate 1, A and B.

ASCI

The asci (pl. 1, A) are cylindrical to club-shaped with rounded apices. They taper gradually in the lower half to the point of attachment. The pore, according to Reade (*72*) and Roberts and Dunegan (*77*), is stained blue with iodine, while Aderhold and Ruhland (*1*) state that it was not so stained in the material (apothecia preserved in spirits received from Norton) that they studied.

ASCOSPORES

The eight monostichous hyaline ascospores found in each ascus are ellipsoid to ovoid in shape with rounded ends. The monostichous arrangement becomes irregular just before the spores are discharged. The spores contain oil droplets and are nonseptate while in the ascus but may become 2-celled prior to germination. (Pl. 1, C, and Pl. 2, C.) Measurements of asci and ascospores by various investigators are summarized in Table 1.

TABLE 1.—*Summary of measurements of asci and ascospores of Sclerotinia fructicola*

Investigator	Date	Host	Asci	Ascospores	Remarks
			Microns	*Microns*	
Winter (*104*)	1883	Peach	130–160 by 8–8.5	10–12.5 by 4–5.5	Dried material from Rau.
Norton (*59*) [1]	1902	do	45–60 by 3–4	6–7 by 3–3.5	Fresh material.
Aderhold and Ruhland (*1*).	1905	do	89.3–107.6 by 5.9–6.8	6.2–9.3 by 3.1–4.6	Preserved material from Norton.
Reade (*72*)	1908	Stone fruits	125–215 by 7–10	10–15 by 5–8	Fresh material.
Pollock (*67*)	1909	Plum	130–179 by 9.2–11.5	11.4–14.4 by 5–7	Do.
Matheny (*56*)	1913	Peach	135–190 by 6.9–10.5	10.5–14.5 by 5.2–7.5	
Do	1913	Plum	137–173 by 6.8–10.8	9.3–14.2 by 5–7.4	
Valleau (*97*)	1914	do	102–166 by 3.5–5.7	5.6–8.9 by 2.9–3.8	Do.
Bertram (*9*)	1916	Stone fruits	150.4–8.8	10.1 by 7.1	
Jehle (*60*)	1912–1920	do	136–185 by 7.8–10	10–16 by 5.8	
Roberts (*77*)	1920	Peach	152–176 by 8–10	6–15 by 4–8	Do.
Norton (*60*)	1910–1923	Stone fruits	89–125–150	9.2–16.7 by 4.1–8.1	
Harrison (*40*)	1922	Plum	116–190 by 10	10.5–16.3 by 5.75–8.2	Fresh Australian material.
Do	1922	Peach	135.0 by 7.8		Preserved material from New Zealand.
Dunegan (*77*)	1922–1923.	do	130–186 by 5.7–13.3	9.5–14 by 5.7–7.5	Fresh material.
Do	1924	do	117–161 by 5.7–9.5	6.8–13.1 by 3.4–6.8	Rau's dried material.

[1] Norton, Ezekiel, and Jehle (*60, p. 12*) state: "The measurements by Norton (1902) are about one-half the usual sizes, apparently due to an error in computing the magnification."

PARAPHYSES

The paraphyses are filiform, hyaline, septate, with rounded, slightly swollen tips. In general they are unbranched, although branched ones are occasionally observed. They are approximately the same length as the asci and from 2 to 4 μ wide.

CONIDIA

Reade (*72*) appears to have been the first to give a complete description of the conidial stage. He apparently agreed with Humphrey (*46*) that the conidia should be considered as chlamydospores, following Brefeld's usage of the term for spores not produced in fructificative fashion on specialized spore-bearing threads. Reade's description (*72, p. 115*) is as follows:

> Chlamydospores [conidia] cespitulose, pulvinate, scattered or in concentric circles, minute to 2 mm usually 0.5 to 1 mm in diameter, at first cinereous, later on cherries and plums becoming ochraceous-buff to Isabella color (R.), on peaches ecru drab to Isabella color (R.), on pears and apples acquiring a blackish tinge, lemon-shaped, 10–28≍7–17 μ, mostly 17≍11 μ continuous, hyaline, in articulate, di- or trichotomously branched chains without disjunctors.

Matheny (*56*), Conel (*18*), and Bartram (*9*) describe the conidial tufts as "ashy" or "ashen-gray."

The writers have observed the conidial tufts on a large number of different hosts. They find the conidial tufts to be ashen gray when young, becoming darker to almost black with age. The tufts are easily rubbed off and are not formed from definite mycelial cushions as are those of *Sclerotinia cinerea* and *S. fructigena*. Measurements of conidia by various investigators reveal considerable variation in size. These measurements are summarized in Table 2. The formation of conidia in chains is illustrated in Plate 1, D.

TABLE 2.—*Summary of measurements of conidia of Sclerotinia fructicola*

Authority	Date	Host	Measurements of conidia
			Microns
Reade (*72*)	1908	Stone fruits	10–28 by 7–17.
Pollock (*67*)	1909	Plum	14.4–24 by 9.6–14.4.
Matheny (*56*)	1913	Peach and plum	14.4 by 10.8.
Conel (*18*)	1914	(?)	13.3 by 7.6.
Valleau (*97*)	1915	Plum	15.95–17.38 by 10.08–12.1.
Do (*97*)	1915	Apple	15.3–15.8 by 10.76–10.81.
Bartram (*9*)	1916	Plum	14.3–18 by 9–14.3.
Dunegan (*77*)	1922–1923	Peach	7.6–19 by 5.7–15.2.
Wormald (*107*)	1927	Peach, 3 sources	9–22 by 7.5–16. 10–18.5 by 8–11.5. 8–18 by 6.5–14.
Do (*107*)	1927	Plum	11–24.5 by 8.5–19.
Harrison (*40*)	1928	Peach	10.9–21.7 by 6.7–14.9.
Do	1928	Quince	11.5–19.2 by 9.6–13.4.

MICROCONIDIA

The microconidial phase was described by Humphrey (*46, p. 88*) as follows:

> * * * the mycelium of the fungus has given rise to immense numbers of closely-set flask-shaped sterigmata, reminding one of those of *Aspergillus*. Each of these produces at its outer or neck end small globular spores of about 3 μ in diameter, every one of which contains a conspicuous oil globule. One rarely finds more than one of these attached to the sterigma, but their vast number and the occasional observation of several still united shows that they must be produced in chains, like *Aspergillus* spores.

Reade (*72, p. 115*) states: "Microconidia formed in cultures on germinating spores, chlamydospores [conidia] and mycelium, spherical, hyaline, 2 to 4 μ, with a central refractive spot."

Pollock (*67, p. 52*) observes:

* * * mycelium from the ascospores also produces flask-shaped conidiophores with microconidia, globular bodies 2.5–3 micromillimeters in diameter. In some cases these flask-shaped conidiophores grow almost directly upon the ascospores or crowded on a very short germ tube. Dense tufts of these conidiophores bearing conidia are also found on the well-developed mycelium.

Valleau (*97, p. 374*) states:

The production of the microconidia was first seen by the writer in a potato-plug culture of the local fungus nearly a year old. The spores ranged from 2.2 to 2.6 μ in diameter, were spherical, and contained a large refractive globule. They were later found on agar cultures in great abundance, in hanging drops of distilled water, and also in hanging drops of 1 per cent malic, 0.062 gallic, 0.062 and 0.25 per cent tannic acids. In the latter cases the flask-shaped sterigmata could be seen. Chains of from 15 to 20 spores were not uncommon. They were also produced in great abundance on the surface of a very young Surprise plum picked and inoculated June 3. These spores ranged in size from 2.55 μ to 3.22 μ, averaging for 25 measurements 2.72 μ. The microconidia produced in the 1 per cent malic-acid solution were larger, ranging from 2.60 to 3.79 μ, measurements of 25 spores averaging 3.14 μ.

The writers have observed the production of microconidia on flask-shaped conidiophores that had developed from vegetative hyphae, on germ tubes produced by the germination of ascospores and conidia, and occasionally directly on ascospores, which apparently had been discharged and had fallen back on the surface of the apothecia. The microconidiophores were produced directly from the ascospore without the intervention of a germ tube.

The flask-shaped microconidiophores have an average length of about 6 μ and an average width of about 2 μ. They may occur singly or in compact masses which seem to be arranged in whorls. The microconidia, which are produced in chains at the tops of the conidiophores, are globose, uniformly 2 to 3 μ in diameter, with an oil drop. (Pl. 2, A.) In cultures microconidia are frequently produced in great abundance, and their masses are visible as viscid, cream-colored droplets on the surface of the medium. (Pl. 2, B.) They may be present in 3-day-old cultures, but they begin to appear in abundance after two weeks. They are usually much more abundant in dextrose-potato agar cultures than in potato agar cultures without dextrose. In old cultures the droplets may run together to form a whitish slime over the surface of the culture medium.

Hino (*43*) considers the microconidia of species of Sclerotinia to be degenerated abortive forms of normal macroconidia that have lost the power of germination and further growth. He claims that microconidia are a symbol of the genus Sclerotinia because they are to be found in all species under all conditions.

MYCELIUM

The mycelium has received slight attention from most investigators, with the exception of Ezekiel (*34*), who recognized hyphal characters as an aid in distinguishing *Sclerotinia fructicola* from *S. cinerea*. He described the hyphae as long, straight, and simply branched in artificial cultures. The mean length of hyphal cells as determined by many measurements was 66.2 μ.

SCLEROTIUM

The sclerotium is a dark-colored, tough, horny, more or less wrinkled structure which invests the mummied peach and from which apothecia, conidia, and microconidia may be produced. Norton, Ezekiel, and Jehle (*60, p. 6*) describe it as

> a dense layer of dark, thick, hyphae of the fungus mingled with or surrounding the partly preserved cells of the fruit. On the outside of the sclerotium the fungus threads are darker and thicker-walled, the inner layers lighter.

Honey (*44*) gives excellent reasons why this mixture of fungus and host tissues should be called a pseudosclerotium.

GERM TUBES

The characters of the conidial germ tubes are of value in distinguishing *Sclerotinia fructicola* from *S. cinerea*, as has been pointed out by Wormald (*105*) and by Ezekiel (*34*). *S. fructicola* has long straight germ tubes which do not branch until they have attained a length of at least 100 μ. (Pl. 2, D.) The germ tubes of *S. cinerea* are much shorter and are twisted and bent. Branching begins soon after the germ tubes are protruded.

OTHER STRUCTURES

Other structures have been noted by investigators from time to time, especially when the fungus is grown in artificial cultures. The thick-walled resting spores mentioned by Humphrey (*46*) and Quaintance (*70*) are often seen and are believed by the writers to be sclerotial in nature. The oblong bodies observed by Humphrey (*46*) budding from many points along certain hyphal threads have also been observed by the writers. They seem to be aborted conidial chains.

STRAINS OR PHYSIOLOGIC FORMS

Differences in the behavior of strains of the fungus obtained from different sources have been noted particularly by Ezekiel (*34*) and by Seal (*88*). The former has shown that strains of *Sclerotinia fructicola* vary in cultural characters, production of oxidase, and effects on inoculated fruits. He also believes that they show constant variations in the size of conidia, but to the writers the differences seem too slight to be significant. He also noticed variations in the shape of apothecia, to which, however, he did not give much consideration, since he did not know whether or not they were constant.

On the basis of differences in growth on potato-dextrose agar, Ezekiel (*34, p. 138*) distinguished 6 varieties into which the more than 30 strains that he studied could be grouped. The varieties are distinguished by their habit of growth on potato-dextrose agar at 25° C. in tube cultures, and at 15° in plate cultures, as follows:

Var. I. Tube culture: conidia and microconidia abundant, hyphae only trace, no hyphal masses. Cultures flat.
Plate culture: conidia very abundant in concentric circles, hyphae inconspicuous, no hyphal masses.

Var. II. Tube culture: both hyphae and conidia medium to abundant, hyphal masses rarely if ever present.
Plate culture: conidia abundant in concentric circles. Hyphae few but visible macroscopically.

Var. III. Tube culture: hyphae and conidia medium to abundant, hyphal masses present.
Plate culture: conidia abundant in concentric circles. Hyphal masses may be present.
Var. IV. Tube culture: conidia trace to few, only at the top of slant. Hyphal masses very abundant.
Plate culture: conidia abundant in concentric circles. Hyphal masses may be present.
Var. V. Tube culture: conidia very few, if present visible only microscopically and not as pustules. Hyphae abundant, not in hyphal masses.
Plate culture: conidia present in concentric circles in center of colony, hyphae more abundant toward the periphery.
Var. VI. Tube culture: conidia very few, if present visible only microscopically and not as pustules. Hyphae abundant, never in definite hyphal masses.
Plate culture: conidia as in tube cultures. Hyphae abundant, forming concentric circles. No hyphal masses.

Seal (*88, Table 1, p. 282*) distinguished at least two physiologic forms, which showed constant differences when grown on various substrata and under various environmental conditions. The two forms showed the following physiologic characters:

FORM 1

On fruits: Slow rot; necrotic area more or less confined to the surface area, not extending deeply into the fruit; fruiting pustules small, compact, and olive colored; * * * at first scanty, but after 10 to 15 days dense, compact * * * . The fruit blackens on decay.
On artificial media: Sparse production of aerial mycelium. Spores produced abundantly in small, ashy-gray tufts. On prune agar and in liquid cultures only little mycelium produced. Darkens the substrate to a dark brown or black. On prune agar spore production very scant.

FORM 2

On fruits: More rapid rot; necrotic area more or less confined to the surface, but extending deeper into the flesh of the fruit; fruiting pustules larger, less compact, scattered, and ash gray to olive in color. Fruiting pustules after 10 to 15 days not so compact, but mycelium more abundantly produced. The fruit turns brown on decay, not black.
On artificial media: Moderately heavy production of aerial mycelium. Spore production less abundant and in medium-sized ashy-gray tufts. On prune agar and in liquid cultures mycelial development more abundant. Darkens the substrate only slightly or not at all. On prune agar spore production more abundant.

The writers have noted that some strains when grown on 4 per cent potato agar grow much more rapidly than others. Some produce conidia abundantly, others very sparsely. Occasionally a form will be isolated which at first produces conidia sparsely and microconidia in great abundance; but after it is grown in culture for several years, the production of microconidia is greatly decreased. The writers isolated a strain of this type from an apple in 1924. Some strains when grown on beef agar darken the medium very rapidly; others require two weeks or more. On the other hand, one strain has been grown for 10 years, and two strains for 7 years continuously in tube cultures of potato hard agar, with and without dextrose, all of which have at different times shown the various combinations of characters that distinguish Ezekiel's varieties.

HOMOTHALLISM

According to Ezekiel (*34*), *Sclerotinia fructicola* is homothallic. He came to this conclusion because apothecia were developed from

peach fruits inoculated with strains arising from single ascospores, and because there was no apparent reaction, other than anastomoses, between strains grown near each other in the same Petri dish. Whether the fungus is homothallic or heterothallic would be extremely difficult to demonstrate conclusively, because no sexual organs are produced on artificial media and because one can not be sure that fruit inoculated with single ascospore cultures may not also become naturally inoculated with vagrant conidia commonly present on the surface of peaches and plums.

GROWTH ON ARTIFICIAL MEDIA

In general the fungus may be said to grow readily on a variety of artificial media. A much-favored medium is 4 per cent potato agar or potato-dextrose agar. On potato-dextrose agar conidia are frequently produced in two days, and the entire slant may be covered with conidial masses in three days. When the dextrose is omitted growth is slower, but conidial production is much more profuse and without the production of aerial hyphae.

Another medium, which the authors have used extensively, is potato plugs soaked in 7 per cent malic acid for 48 hours as recommended by Wiltshire (*103*). The fungus grows readily on the plugs, with profuse production of conidia and microconidia.

Most cultures of the fungus grow very rapidly and produce conidia and microconidia profusely on the potato agars and to a lesser extent on all the common media; some, however, grow more slowly with a comparatively scant production of conidia on the potato agars and with little or no conidial production on many of the common media. Some media, such as beef agar, are turned black by the growth of the fungus. On other media, such as corn-meal agar, the growth is almost invisible to the unaided eye except at points where conidia are produced.

As pointed out by Wormald (*105*) and Ezekiel (*34*), growth characters on artificial media are an aid in differentiating *S. fructicola* and *S. cinerea* forma *pruni*. In Petri dishes of potato agar the former grows very rapidly, producing conidia profusely, usually in concentric zones, whereas *S. cinerea* forma *pruni* grows much more slowly and produces few or no conidia. The margins of the colonies of *S. fructicola* are smooth, but those of *S. cinerea* forma *pruni* are deeply scalloped.

So far as known, the Sclerotinia stage has not been produced in artificial cultures except that Reade (*72, p. 115*) states that the fungus has been grown on artificial media from ascospores to ascospores again.

ENZYME PRODUCTION

By use of the guaiacum emulsion and pyrogallic acid tests Wormald (*106*) demonstrated the production of a much greater quantity of oxidase by *Sclerotinia fructicola* than by *S. cinerea*. By use of the guaiacum emulsion test Ezekiel (*34*) confirmed Wormald's results, but showed that the various strains of *S. fructicola* vary greatly in the readiness with which they produce oxidase.

Cooley (*20*) found that there was a very slight cytolytic action with respect to cellulose from the plum, but that cellulose from filter

paper was readily hydrolyzed when it was the only carbohydrate available.

In the discussion of enzymes involved in the changes of pectic substances the classification used by Atkins (*6*) and by Willaman (*101*) will be followed, i. e.:

Pectase.—Coagulates soluble pectin to a gel in the presence of traces of salts.
Pectinase.—Hydrolyzes soluble pectin and also the gel formed by pectase to sugars.
Pectosinase.—Dissolves the middle lamella to form soluble pectin.

Cooley (*20*) demonstrated the production of pectase by growing the organism in a solution of pectin obtained from plums. He was unable, however, to demonstrate that the fungus dissolved the middle lamella in tissues of infected fruits and was unable to observe any action by the fungus on calcium pectinate prepared from pectin obtained from plums. Therefore, there appeared to be no production of pectosinase.

Valleau (*97*) noted that the middle lamella is dissolved slightly in advance of the penetration of the hyphae but was unable to demonstrate the presence of pectosinase in rotting fruits or to extract it from a culture of the fungus on apple cider.

Willaman (*101*) demonstrated the production of pectase and showed that the middle lamella was probably changed to soluble pectin by the action of pectosinase. The production of pectinase was postulated but not demonstrated.

Muhleman (*58*) prepared an active pectosinase[9] solution by drying and grinding in silica the feltlike growth of mycelium produced on prune juice. He found that a growth of 3 to 5 days gave the most active pectosinase but that the color of the felts and of the ground material and not the age of the crop in days or hours should govern one's conclusions as to its activities. In the more active preparations the color of the ground mass was light chocolate. With active pectosinase preparations disks of green plums, green apples, green peaches, and ripe tomatoes 0.5 mm thick were macerated in 1.5 to 4 hours.

Hawkins (*41*), as a result of studies on the effects of the brown-rot fungus on the composition of the peach, found that the pentosan content remains the same, the acid content is increased, total sugar is decreased, and sucrose is changed to invert sugar. He also found that the amount of alcohol-insoluble substances that reduce Fehling's solution when hydrolyzed with dilute hydrochloric acid decreases.

Davidson and Willaman (*28*) prepared pectase and pectinase, but not protopectinase, from mycelium of *Sclerotinia fructicola*.

FUNCTION OF VITAMINS IN THE METABOLISM OF THE FUNGUS

Willaman (*102*) found that the fungus would not grow in a medium composed of sucrose, various salts, and asparagine. When small amounts of plant decoctions, especially those from peaches and plums, were added to the medium, growth was induced. His experiments indicated that the factor supplied by the plant decoction was of the nature of a vitamin.

[9] In Muhleman's paper the preparation is termed an "active pectinase solution." Since the preparation attacked the middle lamella in fruit disks, the writers are calling it pectosinase in accord with the classification of Atkins and Willaman.

There was some evidence from his experiments that two separate vitamin factors, one for vegetative growth and one for reproduction, are involved in the life history of the fungus. However, most of the experimental evidence he obtained indicated that the existence of but a single vitamin for the fungus is the more plausible hypothesis. No definite conclusions were reached concerning the identity of the vitamin involved, but he thought it might possibly be identical with water-soluble vitamin B.

TEMPERATURE RELATIONS

Many peach growers think that the higher temperatures of summer are more favorable to the development of brown rot than are the average or lower temperatures. They do not consider that when the maximum for the day is in the 90's the temperature may be between 75° and 80° F. during a large part of the 24 hours. This is the optimum temperature range for the growth of the fungus, whereas temperatures over 90° are distinctly unfavorable to its growth. The writers have had cultures on potato agar die in summer under the high temperatures (90° to 100°) prevailing in their laboratories. Sometimes for several days the temperatures would not fall below 90° even at night, when the closed laboratories prevented the entrance of the cooler night air.

Ames (*3*) showed that the fungus would germinate at 0° C., but that growth was very slow. The optimum temperature for growth was 25°, and the thermal death point was about 53°. Conidia (plum strain) did not germinate at 36°, and after 36 hours at 37° did not germinate when removed to an optimum temperature. Conidia (peach strain) did not germinate above 30°.

In experiments by Brooks and Cooley (*14*) the fungus produced measurable apple rots at 5° C. in one week and at 0° C. in two weeks. It also grew on corn-meal agar at 0°, making a measurable growth at the end of the second day. Both on fruit and on artificial media the optimum for growth was 25°, with some growth at 30°. In further experiments the same authors (*15*) found that the fungus produced measurable rots on peaches at 10° in 3 days, at 5° in 6 days, and at 2.5° in 12 days. "Low temperatures," they stated (*15, p. 465*), "have resulted in relatively less inhibition of the growth with Monilia when grown on peaches than when grown on potato-dextrose agar." Ezekiel (*34*) detected no consistent differences in the cardinal temperatures of various strains, the minimum, optimum, and maximum falling near 3°, 25°, and 33°, respectively, for all.

RELATION OF HYDROGEN-ION CONCENTRATION TO GROWTH

Cooley (*20*) grew the fungus in a series of flasks each of which contained sterilized cherry juice of different but known acidity. He found that although the fungus would grow on a medium as acid as the natural juice of sour cherries, it grew more luxuriantly on a somewhat less acid medium. On nearly neutral media there was at first no perceptible growth, but at the end of two weeks there was nearly as much growth as on the acid media.

Ezekiel (*33*) and Norton, Ezekiel, and Jehle (*60*) made a special study of the effect of hydrogen-ion concentration on the production of apothecia. By partly immersing sclerotia in fluids of various pH

values and at the same time maintaining other conditions favorable for apothecial production, they found that the optimum hydrogen-ion concentration for the growth of apothecia appeared to be near pH 2.5. Apothecia at this concentration developed more rapidly and more abundantly than at any of the others. In one series, for example, apothecia at pH 2.5 were mature and expelled their ascospores two days earlier than those at pH 3.8, and these in turn matured much more rapidly than the checks, the sclerotia of which were partly immersed in distilled water. With one exception the limits of growth were pH 1.4 and 6.8, and even at these points the apothecia did not mature normally.

Dunn (*31*) grew the fungus in sterilized peach solutions adjusted to different pH values by the use of a number of acids. With sulphuric acid the greatest growth was obtained at pH 2.84, with phosphoric acid at pH 3.90, and with formic acid at pH 4.37. In one series with acetic acid growth was 109 per cent of that of the control at pH 4.66, while in another series there was no increase over the control. With butyric and salicylic acids, an increase in active acidity caused no increase in growth over the control for the series. With increasing hydrogen-ion concentration the pH values of the first cultures that showed no growth depended on the acids used. Thus, for sulphuric acid the pH value was 1.85, for phosphoric acid 2.20, for formic acid 3.87, for acetic acid 4.45, for butyric acid 4.5, and for salicylic acid slightly above 4.64. The values for the fatty acids were dependent on the initial acidity of the culture solution. As the acidity increased, the amount of mycelium decreased, and usually there was a marked increase in conidial production until the higher limits of acidity were reached.

VIABILITY OF SPORES

ASCOSPORES

On March 28, 1921, mature apothecia were placed hymenium downward in clean dry shell vials, one apothecium to a vial, so that the ascospores were discharged against the bottom and walls of the vials. Two days later the dried apothecia were removed. At intervals a small amount of sterile water was poured into one of the vials, the walls and bottom scraped, and the water stirred with a glass rod. The entire amount of water with suspended ascospores was used in pouring five plates of potato agar. The results are presented in Table 3. These results indicate that, if kept dry, ascospores may remain viable for five weeks, but that the percentage of viability decreases rapidly from the time of maturity.

TABLE 3.—*Longevity of ascospores of Sclerotinia fructicola*

Date	Time after collection of apothecia	Colonies of S. fructicola present in poured plates	Date	Time after collection of apothecia	Colonies of S. fructicola present in poured plates
	Days			*Days*	
Mar. 30	3	191	Apr. 26	29	0
Apr. 5	8	60	May 2	35	12
Apr. 11	14	6	June 3	67	0
Apr. 18	21	1	June 8	72	0

CONIDIA

Conidia produced naturally in the orchard are often long lived and able to survive subzero temperatures. Arthur (*5*) in 1886 germinated conidia from cherry mummies that had hung on a tree all winter. Galloway (*39*) in 1889 reported that in May, 1888, he had germinated conidia from mummies collected in July, 1886. Smith (*92*) in 1891 was able to germinate conidia from dry material 1 year old. Chester (*17*) in 1893 noted that conidia on mummies collected in Delaware in December were larger and thicker walled than those found in summer. Later in the winter he tested the viability of conidia collected from the peach, plum, cherry, and quince. Many failed to germinate, but others produced vigorous germ tubes.

Cordley (*21*) found that conidia kept in a dry place for nearly two years failed to germinate. Conel (*18*) was able to germinate conidia from mummies collected as late as March 11, 1913, in northern Illinois, but not so late as April 12. Bartram (*9*) in 1916 reported on the effects of low temperature on the viability of the conidia. He tested the viability of conidia from plums hanging on the tree and in cultures placed in an open barn. Despite long periods of subzero temperatures, at times as low as −32° C., some conidia were viable as late as April 17. Cunningham (*24*) in New Zealand noted that conidia produced from mummies hanging on the tree during the winter months are slightly thicker walled than those produced during the growing period and are capable of remaining viable for several months, thus differing from summer conidia, which remain viable for only a short time—six weeks or even less.

The writers can confirm the statement that conidia are shorter lived during the summer than during the winter. The viability of conidia is greatly decreased by the unfavorable action of high temperatures. Conidia produced on culture media and kept at room temperature or somewhat higher are about as short lived as those produced naturally out of doors in summer. In germination trials made by the senior writer, cultures about 2 weeks old usually produced the highest percentage of viable conidia; but in summer, after a period when the laboratory temperatures were not lower than 90° F. for several successive days (much of the time about 98° and sometimes over 100°), it was difficult to find a viable conidium of any age. Transfers of masses of conidia to fresh media very commonly resulted in no growth.

MICROCONIDIA

No information is available concerning the viability of the microconidia. In fact, Humphrey (*46*) is the only investigator who claims to have germinated them, and he did not make any tests with microconidia of different known ages.

GERMINATION OF SPORES

ASCOSPORES

The ascospores when newly formed germinate readily in four to six hours, producing usually a single germ tube (pl. 2, C) which later branches. Many of the ascospores that fall back on the hymenial surface of the apothecium germinate in situ, producing the usual

germ tube, which, however, may soon give rise to the flask-shaped microconidiophores and microconidia. Sometimes the germ tube gives rise to these organs directly upon its emergence, and sometimes the microconidiophores seem to be developed directly from the ascospore without the production of a germ tube.

CONIDIA

The conidia germinate readily in distilled water and in most culture media. In sterile prune juice germination is very rapid, the germ tubes sometimes appearing within an hour when temperatures are at or near the optimum. In 24 hours growth is so profuse that if many conidia are present and germinating it is difficult to follow the development of individual germ tubes. The germ tube is comparatively straight and usually does not branch until it has become quite long. (Pl. 2, D.) Ezekiel (*34*) gives as typical a length of 113 μ before branching. Wormald (*105, v. 34, p. 166*) states: "* * * It is usually at least 200 μ in length before it begins to branch, and unbranched germ tubes 650 μ and 750 μ in length have been observed."

Especially in drops of distilled water, and apparently when conditions for germination are not near the optimum, separate germ tubes have been seen to fuse with each other and with conidia. (Pl. 2, E.) Often a number of conidia lying close together will be joined apparently by the end of a germ tube of one fusing with the end of another, or by the end of a germ tube fusing with a conidium.

Doran (*29*) reported that conidia of *Sclerotinia fructicola* germinate quite as well in sunlight (whether it be direct, diffuse, glass filtered, or not glass filtered) as in darkness. He concluded that conidia require precipitated moisture for germination, since none germinated on a dry slide in a moist chamber, but all those in a drop of distilled water germinated.

MICROCONIDIA

Humphrey (*46, p. 88–89*) states with regard to microconidia:

> When some of these spores are sown on nutrient gelatine they germinate readily, first swelling to double their former diameter, and produce abundant mycelia (fig. 8). * * * These spores were also found fallen from their attachments and beginning to germinate (fig. 6, *a*). * * * While these spores can germinate without nourishment, they suffer no preliminary increase in size. On prune-gelatine they swell and germinate as above described.

Humphrey's figures bear out his statements to some extent, but it is possible that the "swollen" spores may have been conidia rather than microconidia. Those in his Figure 8 greatly resemble conidia, but those in his Figure 6, *a*, appear to be true microconidia.

The writers have repeatedly tried to germinate microconidia in distilled water and in various nutrient fluids, but without success. The junior writer has placed masses of microconidia on the stigmas of fresh peach blossoms, thinking that they might need some special stimulus for germination. If they germinated, they did not produce any symptoms of infection on the blossom.

Hino (*43*), working with microconidia produced by several species of Sclerotinia, was unsuccessful in his germination tests except in a few rare cases. In the one case mentioned in the English résumé of

his paper, microconidia of *S. trifoliorum* Erikss., produced germ tubes 4 by 1 μ in size but developed no further.

Ramsey (*71*) states that approximately 10 per cent of his attempts to germinate microconidia of species of Sclerotinia attacking vegetables were successful. From the germinated microconidia he succeeded in obtaining only two cultures that developed a vegetative growth which could be recovered.

DISSEMINATION OF THE FUNGUS

In 1868, at a meeting of the Illinois Horticultural Society, Freeman (*38*) stated that Doctor Hilgard, of St. Louis, at a meeting of the American Association for the Advancement of Science at Chicago, had declared that the spores of peach rot float in the air, live in the ground, and thrive under conditions of warmth and moisture. Hilgard (*42*), in a publication of his own in 1869, stated:

> No doubt the subtle germ is widely diffused and most abundantly present on every healthy peach, since every drop of trickling rain may carry thousands of moist, rapidly infectious germs from the resoaked gum.

Smith (*91*) stated: "Rains, winds, birds, insects, etc., all help to disseminate the spores." Scott and Ayres (*87, p. 13*) stated: "The spores are undoubtedly distributed broadcast by the wind, so that they are in most cases ever present on the fruit * * *." Brooks and Fisher (*16*) also consider wind the important agent, with insects also playing a part. Cunningham (*24, p. 88*), in New Zealand, after stating that the conidia are carried to the fruits by winds, insects, or even birds, gives a specific instance of a bird [10] acting as a distributing agent:

> With their beaks, the birds commonly pierce infected fruits, and turn from these to healthy fruits, especially those showing color, such as nectarines, which in turn they puncture, probably with a view to ascertaining whether they are edible.

That wind, rain, insects, and birds are factors in the dissemination of the fungus in the orchard there can be little doubt. Of these, wind is undoubtedly the most important. One has only to disturb slightly a half-rotted fruit or a conidia-covered mummy to convince himself that wind is an effective agent of distribution. The myriads of conidia that rise as a dust or smoke are seen to float away in the air to be dispersed by the wind and doubtless carried long distances. The ascospores also are discharged as a dust which is with apparently equal ease dispersed by the wind.

Conidia collect about the periphery of drops of water and are easily splashed away by the falling of successive drops or are washed down by currents or trickles. Insects and birds distribute the disease to some extent by alternate contact with diseased and sound fruits. The plum curculio may even insert spores when puncturing sound fruits. The dreaded epiphytotics of brown rot are undoubtedly due in the main to the fact that the conidia are so easily and effectively distributed by air currents. After such a distribution,

[10] A small bird known locally at Wenaroa, New Zealand, as silver eye or white eye (*Zosterops lateralis* Latham).

optimum conditions of temperature and moisture as the fruit nears maturity are certain to bring about an epiphytotic of brown rot.

As pointed out by Quaintance (*70*), in the gathering of fruit for shipment there is a general scattering of conidia. Mummied and rotten fruits are disturbed, and the pickers' hands become covered with conidia, which are rubbed over sound fruits. In this way the fruit becomes covered with conidia, which may initiate the disease in the packing shed, in transit, or on the market.

HOST PLANTS

A list of host plants can not be considered complete, since there has been no attempt to make a survey of these plants, and can not be considered accurate, because of the confusion that has existed regarding the classification of this and closely related species of Sclerotinia. Under favorable conditions the fungus is probably able to infect all drupaceous and pomaceous species as well as many other members of the Rosaceae. The following list has been assembled from the literature and from the writers' observations.

Chaenomeles japonica Lindl	Flowering quince.
Cydonia oblonga Mill	Common quince.
Malus pumila Mill	Apple.
Prunus americana Marsh	American plum.
P. americana × *P. hortulana*	Plum.
P. americana × *P. salicina*	Do.
P. angustifolia Marsh	Chickasaw plum.
P. avium L	Mazzard.
P. avium × *P. cerasus*	Cherry.
P. armeniaca L	Apricot.
P. besseyi Bailey	Bessey cherry.
P. besseyi × *P. hortulana*	Compass plum.
P. besseyi × *P. pennsylvanica*	Plum.
P. besseyi × *P. salicina*	Do.
P. cerasus L	Sour cherry.
P. communis Arcang	Almond.
P. domestica L	Common plum.
P. fenzliana Fritsch	Almond.
P. glandulosa Thunb	Flowering almond.
P. hortulana Bailey	Hortulan plum.
P. instititia L	Damson plum.
P. japonica Thunb	Chinese bush cherry.
P. mume Sieb. and Zucc	Japanese apricot.
P. munsoniana Wight and Hedr	Wildgoose plum.
P. nigra Ait	Canada plum.
P. pennsylvanica L	Pin cherry.
P. persica Batsch	Peach.
P. persica var. *nucipersica* Schneid	Nectarine.
P. pumila L	Sand cherry.
P. pumila × *P. pennsylvanica*	Do.
P. reverchonii Sarg	Hog plum.
P. salicina Lindl	Japanese plum.
Prunus sp	Rocky Mountain dwarf cherry.
P. tomentosa Thunb	Nanking cherry.
P. triloba Lindl	Flowering plum.
P. umbellata Ell	Southern sloe.
P. virginiana L	Common chokecherry.
Pyrus communis L	Pear.
Rosa sp	Rose.
Rubus occidentalis L	Common blackcap.
Rubus sp	Blackberries.

SEASONAL LIFE HISTORY AND PATHOGENICITY OF SCLEROTINIA FRUCTICOLA

Investigations of the seasonal life history and pathogenicity of the brown-rot fungus have engaged the attention of many botanists and plant pathologists since 1880. Previous to that time Hilgard (*42*), Kirtland (*49*), and probably others, as pointed out earlier in this bulletin, had some knowledge of the pathogene and its behavior.

For the sake of convenience the discussion of the seasonal life history and pathogenicity will be grouped under several headings and subdivisions. It should be clearly realized that such a division is purely artificial and that the various activities of the fungus proceed concurrently or overlap.

SURVIVAL IN WINTER

After it became clearly recognized that the common rot of the peach was caused by a fungus, plant pathologists began a search for the means whereby the fungus could survive the unfavorable conditions of winter. As a result it has been found that the fungus can pass the winter in a resting stage (1) on mummies hanging on the tree, which produce conidia in the early spring and on which also many conidia produced the previous season may survive, (2) on mummies lying on or in the ground, many of which may produce the apothecial stage at blossoming time, and (3) in cankers on twigs and branches, from which conidia may or may not be produced the following season.

SURVIVAL THROUGH FORMATION OF CONIDIA ON MUMMIES

Arthur (*5*) appears to have been the first to show that conidia from mummied fruits of the previous year could act as infection sources. He germinated conidia from such sources (mummied cherries) and showed that they could cause disease. He states (*5*, *p. 281*):

> When the fruit is attacked before it is ripe, it usually remains hanging to the tree through the winter, even till fruit is ripe again, and spores of the fungus are to be found on it during the whole time.

Similar results with cherry were obtained by Galloway (*39*).

Smith (*91*) seems to have been the first to show that peach mummies hanging on the tree produce new crops of conidia in the early spring that are capable of germinating and causing infection. He also showed that conidia can survive the winter. These results were soon confirmed by Humphrey (*46*) and by Chester (*17*). Bartram (*9*) demonstrated the viability of a fair percentage of conidia that had passed through winter temperatures as low as −32° C.

Most brown-rot mummies become loosened and drop to the ground of their own weight or are blown loose by the wind, but there are always some that remain hanging on the trees through the winter and early spring.

Since the brown-rot fungus completely invests the outer tissues of the peach during the formation of the mummy and forms a sclerotial membrane, it can withstand long periods of adverse environmental conditions and still produce fruiting bodies. Mummies hanging on the trees produce conidia only, whereas those on the ground produce

apothecia and rarely, if ever, produce conidia; but Valleau (*97*) found that mummied plums that have hung on the tree for one year still have the power of producing apothecia when buried in the ground. Mummies on the ground are frequently the more reliable source of infection at blossoming time, because many of them retain sufficient moisture to produce apothecia even in a dry spring.

It is possible to demonstrate the survival of the fungus in mummies by removing the inner tissue of the mummies under sterile conditions and obtaining cultures of the fungus from it; but the demonstration that the fungus is alive is not the essential point. It not only must remain alive but also must produce spores for it to be considered an effective agent in the spread of brown rot.

In Georgia the writers observed periods of conidial production from mummies hanging on the trees in every month from September to February in the years 1921 to 1925. February 17 was the latest date on which conidial production was observed. The production of conidia is closely correlated with periods of rainy weather and, as the above dates indicate, takes place at relatively low temperatures provided sufficient moisture is present. The percentage of mummies that produce conidia is at times very high. On November 11, 1921, 95 to 98 per cent of the peach mummies (variety Early Wheeler) hanging on the trees bore fresh tufts of conidia. (Pl. 2, F.) On December 31, 117 of a total of 132, or 91 per cent, of the same variety were producing conidia. On September 18, 1924, 75 per cent of the Elberta and 69 per cent of the Yellow Hiley mummies out of a total of 100 collected from each variety were producing conidia. On the other hand, in some seasons conidial production by mummies on the trees is rare. Peach and plum mummies collected by the senior writer in the winter of 1920 from trees at the Arlington Experiment Farm, Rosslyn, Va., rarely produced conidia when placed in moist chambers, although in other years they have usually done so.

Although the writers have examined mummies from most sections in which brown rot occurs, they have never observed production of conidia by mummies on the ground, nor have they been able to induce conidial production by placing these mummies in moist chambers. Just why these mummies should not produce conidia as well as ascospores is not known.

It is evident from the observations of many investigators that the fungus may remain alive in mummied fruits on the tree and on the ground and that many of those on the tree are capable of producing conidia for many months. By late spring, mummies on the tree, becoming dried, cease to produce conidia and, falling to the ground, soon disintegrate.

SURVIVAL THROUGH FORMATION OF APOTHECIA FROM MUMMIES

An important phase in the development of the fungus from overwintering sources is the formation of apothecia from the sclerotia enveloping the mummied fruits that have fallen to the ground. Norton (*59*) in 1902 made the most important single contribution to the life history of this fungus when he showed that these apothecia represent the ascigerous stage of the brown-rot fungus. For nearly 20 years after Norton's discovery it was generally accepted that mummies had to lie on the ground through two winters before

apothecia were produced; but, as has been shown by Roberts (*76*), Ezekiel (*32*), and Cunningham (*25*, *26*), apothecia may develop in the spring from fruit that had rotted and fallen to the ground during the previous summer or autumn. The observations and experiments of the writers reported later in this bulletin indicate that in most seasons apothecia are formed in much greater abundance from rotted fruits that have lain on the ground through only one winter than from those that have been exposed two or more winters. However, the importance of the production of apothecia from the older mummies should not be overlooked. The apothecia from the older mummies aid in reestablishing the fungus in the orchards following seasons in which there are no newly formed mummies because of the failure of the trees to bear or because conditions for infection are unfavorable.

SURVIVAL IN CANKERS ON TWIGS AND BRANCHES FROM WHICH CONIDIA MAY OR MAY NOT BE PRODUCED THE FOLLOWING SEASON

The status of cankers as carriers of the fungus over winter and producers of conidia the following season is somewhat uncertain. That the fungus survives the winter in some of the cankers has been shown by many investigators, but few have observed the production of conidia on overwintering cankers. Smith (*91*, *p. 131*) in 1889 stated:

> As a rule the fungus produces its conidial tufts much less frequently on stems than on fruit. Occasionally I have seen them on branches of the previous season's growth, but generally they are more abundant on tissues only recently out of the meristematic condition * * *.

Quaintance (*70*) states that the fungus is capable of developing the spore tufts from blighted fruit spurs and twigs of the previous year but does not state that he observed such development in orchards. Cook (*19*) in 1919 observed the production of conidia from cankers of the previous year and considered them as important in the production of blossom blight. Cunningham (*24*) also considered cankers of the previous year important infection centers. Berkeley (*10*) reports that at St. Catharines, Ontario, in the spring of 1926, many cankers were active and conidia were present on them. He found active 2-year-old cankers and concluded that possibly cankers are sources of infection for the blossoms of the following year.

Only once have the writers observed the formation of conidia on overwintering cankers, although they have made observations as opportunity presented over a period of about 10 years. It would appear that, as compared with mummied fruits, cankers are not of great importance as overwintering sources of infection, except perhaps in certain sections where weather conditions favor the continued development of cankers and conidial production from them. The formation and persistence of cankers will be discussed subsequently.

OCCURRENCE OF APOTHECIA

As pointed out by Norton (*59*), the duration of apothecial production is about that of the peach flowers. It is interesting to observe how closely these two periods coincide over a term of years. In eight years' (1920–1927) observations at the Arlington Experi-

ment Farm near Washington, D. C., the time of full bloom of Elberta peaches varied from March 20 to April 21, with apothecial production showing practically the same variation. Norton, Ezekiel, and Jehle (*60*) have also noted that the production of apothecia coincides with the germination of peach and plum seeds lying under the trees.

Apothecia may be produced in considerable numbers. McCubbin (*54*) found 111 in one cluster and a total of 1,163 clusters under 225 peach trees, an average of 5.1 to the tree. In one peach orchard there were 26.3 clusters to the tree. Leslie Pierce, of the Bureau of Plant Industry, has informed the writers that in Indiana in 1927 he found 87 clusters under one peach tree.

The sclerotia investing mummied fruits are quite persistent and may produce apothecia for many years, although the crop usually becomes less with each succeeding year. Pollock (*68*) found apothecia produced from plum mummies that had lain on the ground for 10 years. Ezekiel (*30*), however, has shown that mummies when buried in the ground, a condition that would obtain in most orchards, disintegrate very rapidly.

In the fall of 1921 the writers started a series of experiments at Fort Valley, Ga., to determine how long apothecial production would continue. Peach mummies of the 1921 crop were picked from the trees and buried in sandy clay soil at depths varying from one-half inch to 3 inches. There were no peach trees in the vicinity, but as a safeguard against the accidental introduction of other mummies wire cages were placed over the different lots of mummies. Apothecia were produced in abundance in the spring of 1922 and in slowly diminishing numbers each successive year until the spring of 1928, when none developed. In these experiments mummies of known ages (i. e., of the 1921 crop) produced apothecia for six successive years.

Brooks and Fisher (*16*) have observed that with prunes and cherries apothecia occur chiefly on mummies near the surface of the soil and are rare or lacking in orchards in which mummies are regularly plowed under. Ezekiel (*33*) showed that cold is probably a factor in the production of apothecia, since chilled mummies produce apothecia 25 weeks after inoculation. He also showed that burying mummies beneath the surface of the ground inhibits the production of apothecia, even if production had started when the mummies were buried. He (*33*) found that apothecia developed best at pH 2.5 and made good growth from pH 1.4 to 5.8, but that even slight alkalinity inhibited growth.

Cunningham (*26, p. 228*) in New Zealand considered the following as favoring apothecial production:

(1) Showery weather accompanied by warm days and cool nights.
(2) Depth at which mummies are buried. The number of apothecia produced decreased with the depth of the soil.
(3) Age of mummies. Those of the past season produced apothecia most abundantly.
(4) Soil conditions. Apothecia were more abundant in compact than in loose soil.

In general the observations of the writers are in agreement with those reviewed above. The largest crop of apothecia is, under favorable conditions, produced by mummies 1 year old and it decreases with age. At the Arlington Experiment Farm, a growth of chick-

weed (*Cerastium vulgatum* L.), which begins very early in the season, has always favored apothecial production, probably because it prevents the mummies from becoming dry by shading them.

The experiments of the writers in which yearly observations were made on peach mummies buried at various depths have indicated that, as others have found, the best position for apothecial production is that in which the mummy is only half buried, the apothecia arising from the under surface. Mummies entirely exposed on the surface of the soil uniformly failed to produce apothecia. (Pl. 3, A.) Completely buried mummies may produce apothecia which appear in abundance at the surface of the soil, but they are frequently destroyed by earthworms, and those that escape destruction often produce distorted apothecia. Stipes 6 cm long have been observed arising from deeply buried mummies.

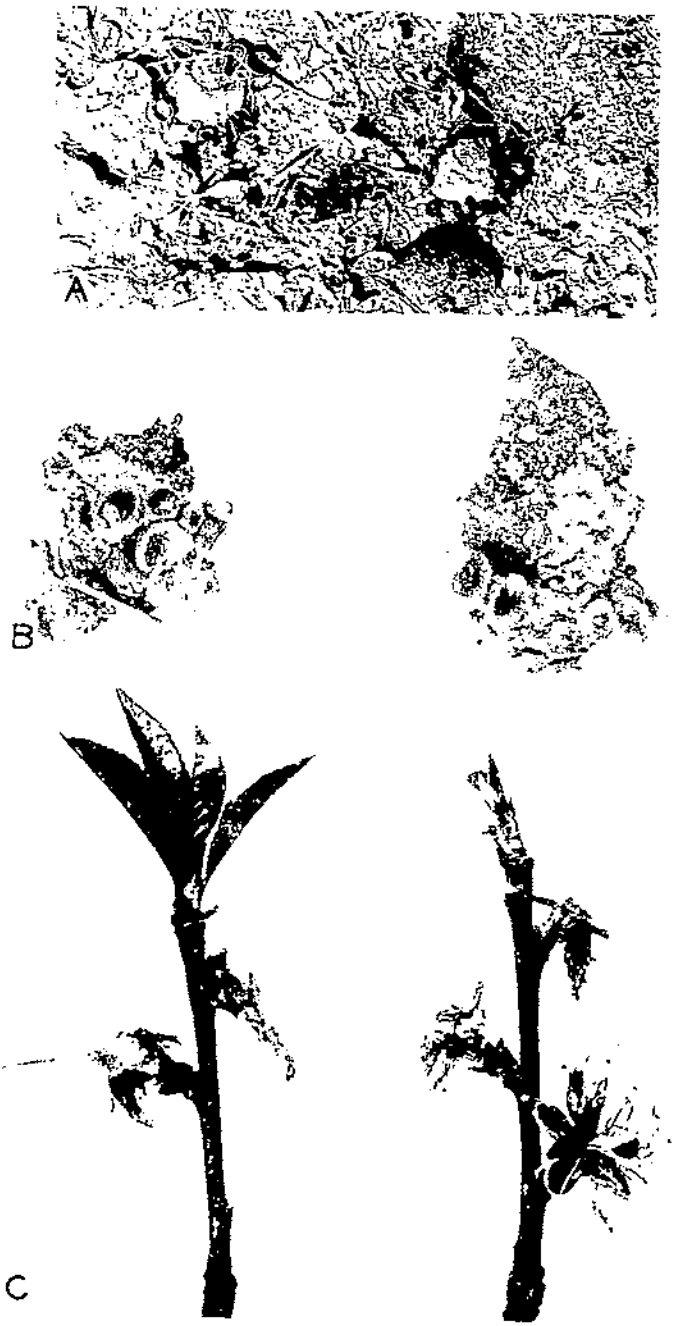
Buried mummies tend to break up as they increase in age, and only fragments of the sclerotia remain. These fragments still possess the ability to produce apothecia. (Pl. 3, B.) In one instance a fragment measuring 3.8 by 1.4 cm was found on which 15 small apothecia were produced. In another instance three apothecia of about average size were produced from a bit of sclerotium 1 cm long and 2 mm wide. Apparently there is a fairly definite period during which mummies must be in the soil before apothecia are produced. Mummies collected from trees in November and buried have produced apothecia within 70 days, but mummies collected in August and kept dry until the following February and then buried failed to produce apothecia in March. The sclerotia had not been killed by the drying process, as apothecia developed from them the following spring. In the vicinity of Washington, D. C., the larvae of the oriental fruit moth (*Grapholitha molesta* Busck) in the decayed fruits appear to aid in the disintegration of mummies.

Apothecia have been reported as occurring on mummied fruits of most of the drupaceous hosts but appear rarely to be produced from mummied fruits of pomaceous hosts. The only authentic record of the production of apothecia from mummied apples appears to be that of Harrison (*40*) in Australia. He obtained apothecia from partly buried apple mummies, and single ascospore cultures from them were pronounced by Wormald to be identical with cultures of *Sclerotinia fructicola* (called by Wormald *S. americana*).

DISCHARGE OF ASCOSPORES

The ascospores are discharged by pressure within the asci, and the simultaneous discharge of many asci from a single apothecium produces a dust or cloud easily discernible to the naked eye. This discharge may be artificially induced by sudden exposure to light or by intensifying the light. Petri dishes of potato agar held over apothecia puffing out clouds of spores are quickly seeded. In a closed room the writers have caught spores in Petri dishes held as high as 20 inches above the discharging apothecia. At this height, however, it is probable that ascending air currents played a considerable part.

The writers have also measured the distance above the surface reached by spores discharged from an apothecium inclosed in a small box when a beam of light played over the surface of the



SCLEROTINIA FRUCTICOLA AND BLOSSOM BLIGHT

A, Apothecia developing from two partially buried mummies. No apothecia have developed from the third mummy, which is resting on the surface of the soil, slightly reduced. B, Apothecia produced in the spring of 1921 from small fragments of a mummy of the 1920 crop. Natural size. C, Blossom blight of the peach, Elberta variety. Note the stiff matted appearance of the floral parts of the infected uppermost blossoms. Natural size.

BROWN ROT OF PEACH AND WILD HOST OF SCLEROTINIA FRUCTICOLA

A, Brown rot of a mature Oneola peach. The source of infection was the blighted blossom located just above the rotted peach. ⅔ natural size. B, Extensive thicket of wild plum (*Prunus angustifolia*) observed near a peach orchard in central Georgia. Apothecia were very abundant under the trees in this thicket in the spring of 1928. Plum thickets such as this constitute a distinct menace to near-by orchards.

apothecium to reveal the spores. The spore discharge was observed through a slit in the box, and the height of the discharge was measured by placing a metric rule behind the apothecium. The placing of the apothecium in the box counteracted, to a certain extent at least, the effect of convection currents, and the measurements may be considered as rough approximations of the actual distances the spores are shot above the apothecium under the sole influence of forces in the ascus. The average height of spore discharge in the 31 measurements was 1.7 cm. The highest discharge observed was 4.3 cm and the lowest 0.5 cm.

Individual apothecia have been collected which produced clouds of spores at intervals for as long as seven days.

In order to determine whether ascospores were prevalent in the air, Petri dishes containing potato agar were exposed in a field which had been an orchard until two years before and in which there were scattered clumps of half-buried mummies producing apothecia. Two dishes placed on the ground and exposed for three minutes developed eight and two colonies, respectively, of the brown-rot fungus. Two dishes exposed for three minutes at a height of 4 feet above the ground each developed one colony of the fungus. So far as could be ascertained, there was no production of conidia from any source, and the colonies were assumed to result from ascospores.

THE BLOSSOM-BLIGHT PHASE OF THE DISEASE

Arthur (*5*) observed blossom blight of cherries in 1886 and produced it artificially by inoculating cherry blossoms with conidia. In 1889 Galloway (*39*) published a detailed description of the disease and called attention to the importance of the blighted blossoms as sources of infection. Smith (*91*, *92*) in 1889 and again in 1891 called attention to the serious nature of blossom blight in the peach orchards of the Delaware-Maryland-Virginia Peninsula. Although Arthur in 1886 had shown that conidia of the brown-rot fungus could infect cherry blossoms, Chester (*17*) in 1893 seems to be the first to announce the production of peach-blossom blight by artificial inoculation. Since that time Quaintance (*70*), Scott and Ayres (*87*), Jehle (*48*), McCubbin (*54*), Cook (*19*), McClintock (*52*, *53*), and others have discussed blossom blight of the peach. Roberts and Dunegan (*78*) in 1926 reported on inoculation experiments of peach blossoms with conidia and ascospores. Both spore forms proved capable of causing blossom blight. All parts of the open flower could be attacked, and infection of the stigma, anthers, petals, and sepals was observed.

With the infection of blossoms in the spring by conidia or ascospores from diseased material that has survived the winter, the fungus makes its first attack of the growing season. The first symptom of infection is a faint discoloration of the part affected, whether it be the petals, stamens, or stigma. The fungus grows rapidly, and the entire floral structure is soon brown and shriveled. Masses of spores are produced on the diseased blossoms. The petals, styles, and filaments of the blighted blossoms become matted together in a dry, brittle mass generally bending downward. (Pl. 3, C.)

The sepals may be and generally are involved in the blighting of the other floral parts, and may be covered with spore tufts when the

young peach is the size of a pea. Usually, but not always, the infected sepals drop to the ground without the fungus gaining entrance to the young fruit.

Galloway (*39*) reported that blighted cherry blossoms remained on the trees three or four weeks; then, if the weather was wet, they dropped from the tree; and, being a mass of soft rotted tissue, they adhered to any part of the tree they touched. In Georgia many of the blighted blossoms are dislodged by various agencies, but the writers have not seen cases in which these dislodged blighted blossoms per se produced infections on either fruit or leaves, nor have they seen them attached to any portion of the tree. On the contrary, they have found blossoms remaining attached to the twigs as late as January of the year following their production.

In humid sections blossom blight is usually present to some extent every year and under favorable conditions may become very prevalent. Its development is favored by the moist, moderately warm weather often encountered at the time peaches are in bloom. Usually blossom blight is not noticed until the blooming season is nearly over, when the browned flowers begin to attract attention.

While blossom blight may cause severe losses by reducing the number of flowers capable of developing into fruits, it more frequently causes severe damage, not so much by reducing the set of fruit as by establishing in the orchards numerous sources of infection which later make it more difficult to prevent the fruit from rotting.

THE FRUIT-ROT AND MUMMY PHASE OF THE DISEASE

The fruit rot or so-called brown rot of the peach is essentially a ripe rot, or rot of the maturing fruit, although the fungus may enter young green peaches through punctures or bruises and cause rot. There are exceptions to this statement. In May, 1924, about three weeks after petal fall and following two weeks of wet weather, the senior writer observed young fruits, about the size of peas, which had been killed by brown rot. Examination of these fruits showed that infection had come about through contact with the closely appressed calyx which had become infected.

Conidia of the fungus are carried over to the maturing fruits from the last year's mummies, green fruits, blighted blossoms (pl. 4, A, and pl. 5, C), and twig cankers of the current year mainly by wind, but to some extent by rain. Leaves affected by leaf curl (*Exoascus deformans*) are also frequently important as sources of infection, as has been observed by McCubbin (*54*) and by Mix (*57*). Early in June, 1920, the senior writer examined an orchard heavily infested with leaf curl. Conidial tufts of the brown-rot fungus were found on nearly every affected leaf examined. It could hardly be doubted that the fungus would be carried over to the ripening fruits from these leaves.

Many investigators, including Smith (*91*) and Chester (*17*), have demonstrated that infection can take place through the uninjured epidermis of fruits but that infection is easier and more common when the surface is bruised or the epidermis punctured. Soft overripe and watery fruits are more easily infected than are the more solid ones. Most commercial varieties of the peach are not so easily infected through the unbroken epidermis as are many of the more

delicate noncommercial varieties. With commercial varieties most of the infections in the orchard take place through punctures made by the plum curculio (*Conotrachelus nenuphar* Hbst.), the oriental fruit moth (*Grapholitha molesta* Busck), and other insect pests, or through cuts or bruises made by hail or other mechanical agencies. As shown by Smith (*91*), the fungus may enter also through lesions made by the scab fungus (*Cladosporium carpophilum* Thüm.).

According to Curtis (*27*), the fungus is able to penetrate the cuticle of plums, cherries, nectarines, peaches, and apricots, but the usual method of effective entry is, in the case of peaches, through the hair sockets. She also observed a considerable number of infections through the stomata. Valleau (*97*), experimenting with plums, found that infection may take place through the uninjured skin at any time during the development of the plum fruit. The hyphae enter through the stomata and lenticels. Varieties show great differences in resistance to infection, owing to the production of parenchymatous plugs which may gill the stomatal cavity, and of lenticels with layers of corky cells through which the hyphae are unable to penetrate. Corky cells lining the stomatal cavity merely delay infection. Valleau also states that the rot is a firm rot due to the mechanical support of the hyphae which completely fill the intercellular spaces left by the collapse of the host cell walls.

Under favorable conditions the incubation period is very short. Chester (*17*) places it at 18 hours. Quaintance (*70, p. 256*) states:

> Spores inserted under the skin of a ripening peach with a needle point developed the usual brown rotten spot, an inch or more in diameter, in 20 hours, and the production of spore tufts followed 3 hours later, thus making the period of reproduction from the spore, under very favorable conditions, at 23 hours.

Conidia produced on infected fruits may be carried by the wind or other agencies to other fruits on the same or neighboring trees and by infecting them cause the disease to spread rapidly.

The fruits when thoroughly invested by the ramifications of the fungus hyphae become dried and may continue to hang on the tree or may fall to the ground. Many of the peaches that fall to the ground do so before they are completely rotted, while those left on the tree frequently remain attached because, since the fruit stem has been killed by the fungus, no abscission layer is formed. The completely rotted and shriveled fruits on the tree and on the ground are the so-called mummies, which are so important in initiating the disease the following spring through the agency of conidia or ascospores produced by them.

THE CANKER AND TWIG-BLIGHT PHASE OF THE DISEASE

The formation of twig cankers as the result of the fungus passing from the floral parts through the peduncle and into the tissues of the twigs is frequently a sequel to blossom blight. Occasionally twig cankers result from growth of the fungus through the stem of decayed fruit and into the twig, but this more often results in a blight of the twig rather than in the formation of cankers. Although cankers and blighted twigs do not necessarily occur every year, they are usually present, because the climatic conditions that are favorable

to blossom blight and fruit rot also favor the growth of the fungus into the twig tissues.

Twig cankers resulting from the growth of the fungus through the blossom peduncle into the tissues of the twig appear first as slightly browned, collapsed areas about the base of the peduncle. (Pl. 5, A.) They are roughly elliptical in shape and at first involve only the portion of the twig adjacent to the blighted blossoms, but by subsequent growth the fungus extends up and down the twig from the original point of entrance. The development of a number of these cankers was followed in the spring of 1923, and it was found that they may increase in length as much as 4.1 cm in a period of 30 days during the early spring. (Table 4.) This rate of growth, however, is not maintained throughout the growing season.

TABLE 4.—*Increase in length of brown-rot twig cankers in 30 days*

Canker No.	Size of canker		Increase	Canker No.	Size of canker		Increase
	Mar. 14, 1923	Apr. 14, 1923			Mar. 14, 1923	Apr. 14, 1923	
	Cm	*Cm*	*Cm*		*Cm*	*Cm*	*Cm*
1	3.9	5.8	1.9	6	2.5	3.0	0.5
2	1.9	4.9	3.0	7	2.4	6.5	4.1
3	2.2	2.5	.3	8	2.6	3.1	.5
4	1.5	5.9	4.4	9	.7	1.0	.3
5	2.5	5.6	3.1	10	.9	1.0	.1

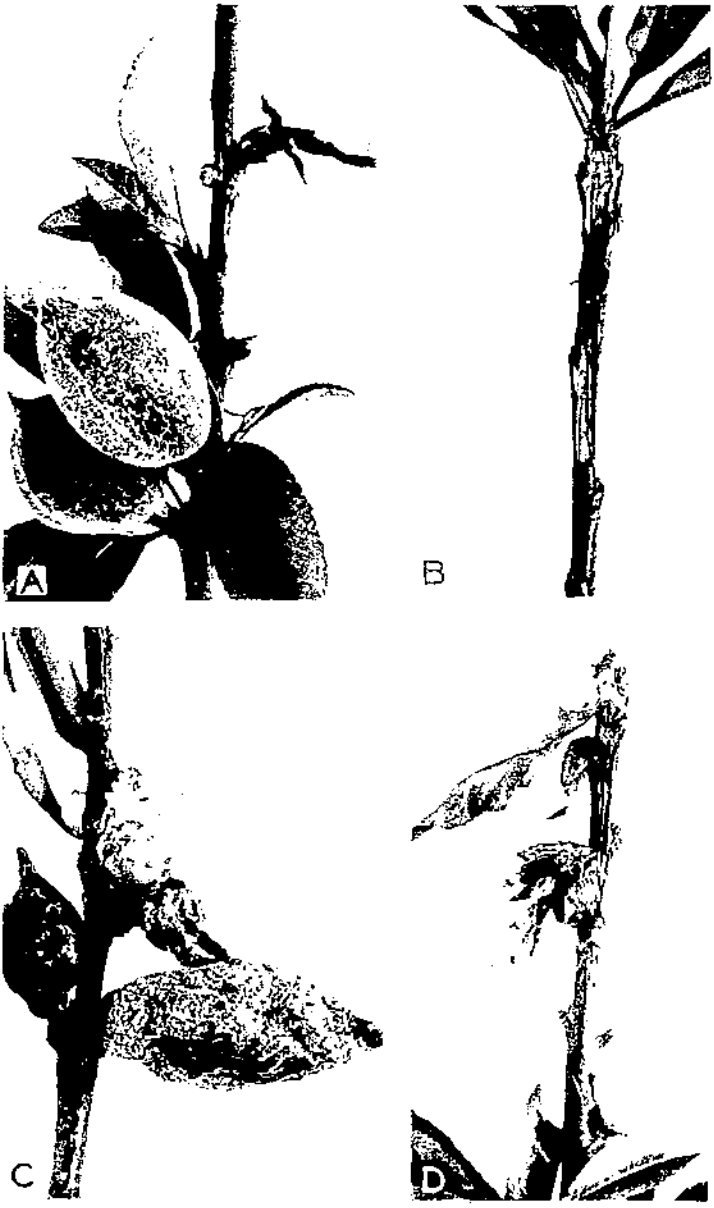
Gum pockets (pl. 5, B, and pl. 8, C) are formed in the tissue by the fungus, and in rainy periods the gum collects on the surface in the form of small droplets which may expand into large masses through the absorption of water by the gum. (Pl. 5, A and C.)

In rainy seasons the fungus frequently encircles and kills the twig above the canker by girdling. (Pl. 5, D.)

As the season advances, the central portion of the canker becomes bleached in color and the sunken bark is ruptured by the growth of callus tissue from the sides, so that, at the end of the growing season, the cankers are represented by distorted regions on the twigs. (Pl. 6, A.)

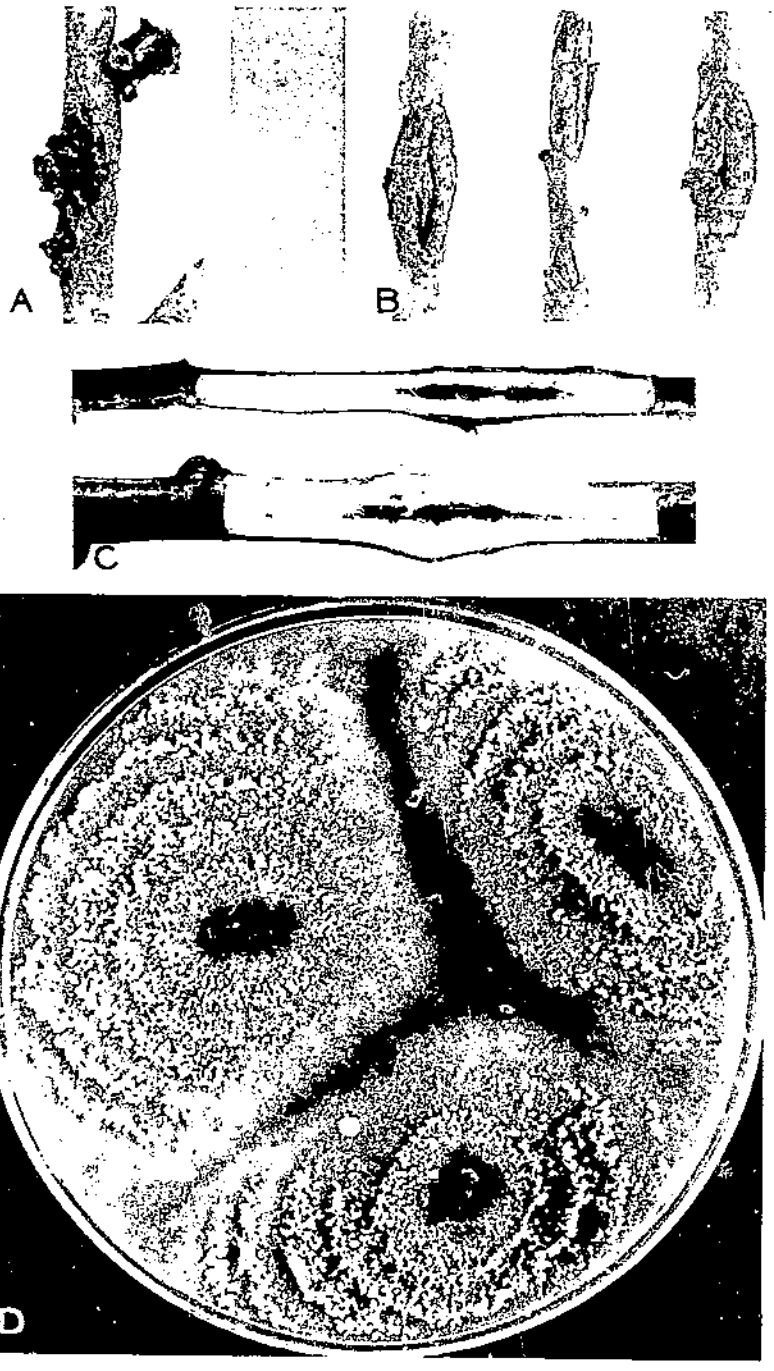
The callus tissue may cover the cankered area by the end of the season in which the canker originated, or the union may not be completed until the second growing season. (Pl. 6, B.) The final result is the covering of the necrotic area by new tissue and the formation of normal tissues following the union of the callus. The necrotic region is merely buried under a layer of new tissue and can be demonstrated in after years by cutting through the twigs. (Pl. 6, C.)

Almost all investigators are agreed that the cankers about the blossoms are the result of the fungus passing from the floral parts into the tissues of the twigs. Cook (*19*), in New Jersey, however, considered that the cankers were formed at the bases of the buds of the young shoots and therefore are at the bases of the flower buds that open the following spring. He found conidia produced on these cankers in the spring, but he did not give any data as to when and how these cankers were formed originally. Cook's description of



TWIG BLIGHT OF THE PEACH CAUSED BY SCLEROTINIA FRUCTICOLA ON UNEEDA VARIETY

A, Small twig. A drop of gum is oozing from the infected tissues. B, Twig canker with the outer tissues cut away to show necrotic region. The dark vertical line at the left represents a gum pocket. C, Large mass of gum adhering to canker on twig. D, Killing of apical portion of twig by the fungus girdling the twig below the blighted blossom. All natural size.



TWIG CANKERS OF THE PEACH CAUSED BY SCLEROTINIA FRUCTICOLA

A, Appearance of twig canker on Uvneda variety at the end of the growing season on which it was initiated. Photographed October 17, 1923, natural size. B, Twig cankers showing varying amounts of callus development. On the left the callus has not united; in the middle twig it has almost met; while in the twig at the right the callus tissue from both sides has united, completely covering the necrotic region. Hiley variety, photographed February 20, 1928, natural size. C, Twig cankers formed in 1927 cut open in February, 1928, showing the original necrotic area embedded in new tissue generated by the callus layer. Natural size. D, Petri-dish culture 15 days old with colonies of *S. fructicola* which have developed from fragments of the inner tissues of twig cankers. Slightly enlarged.

these cankers, his photographs of them and their position, i. e., just below the buds, suggest that they were cankers caused by arsenical sprays. These sprays commonly cause necrotic areas to form at the base of the petioles which appear the following spring as cankers at the base of the buds and often involve the buds. It is possible that the brown-rot fungus might gain entrance to the tissues after they have been ruptured and produce conidia the following spring. The writers doubt that the cankers described by Cook were caused by the brown-rot fungus, although they do not wish to imply that the organism was not present in them and that such cankers, caused presumably by arsenical injury and subsequently invaded by the brown-rot fungus, might not play an important rôle in the dissemination of the fungus.

Frequently the twig to which an infected peach is attached is invaded and killed. These dead twigs and accompanying cankers have received considerable attention from investigators of peach diseases. As early as 1868 the following statement by E. S. Hull, a prominent fruit grower of Alton, Ill., appeared in the Transactions of the Illinois Horticultural Society for that year (*45, p. 265*):

> These [fruits], under certain conditions, rot to such an extent, especially some of the early peaches, as to defoliate and kill all the interior branches, and so impair the vitality of the trees as to render them worthless, and, in some instances, to kill them.

On the plum the killing of spurs and adjoining bark was noted by Kirtland (*49*), who wrote in 1855: "This malignant and cankery action will, likewise, extend to the adjoining bark wood and fruit spurs; and often either entirely destroys their vitality or induces a sickly condition." Smith (*92*), Chester (*17*), and others also observed that the fungus by growth through the fruit stem and bark caused cankers and girdling of the twigs.

Because the brown-rot fungus attacks only the tissues that are not far beyond the meristematic stage, it seldom if ever causes cankers by direct infection of twigs or branches of the commonly planted commercial varieties. Even infection through wounds seems to be a rare occurrence. Many of the older varieties were abandoned because of their susceptibility to brown rot, and doubtless the shoots of some of them could become infected as Chester (*17, p. 61*) suggested in 1893:

> That infection through the bloom and the fruit is the usual method can not be denied, but the fact that trees have been found which have not blossomed and yet which show genuinely blighted twigs indicates that in some cases the fungus can find entrance other than already indicated. My own observations on this point lead me to believe that the tender opening buds of young shoots offer a favorable harbor for the developing fungus, which subsequently appears as a wilting of the young leaves and a blighting of the generally short terminal growths.

Smith (*91, p. 131*) first stated: "In the early spring the young and tender shoots must be infected by spores. Many such shoots were attacked and killed in 1889." Later he (*92, p. 37*) changed his mind and stated: "Heretofore I had supposed it capable of penetrating through the unbroken cuticle of young shoots, but such cases must be exceptional." Jehle (*48*) found it easy to inoculate twigs and branches of all ages through wounds, but concluded that natural infection of limbs occurs either through diseased blossoms

or through diseased fruits. Fant (*37*) concluded that infections take place through wounds as well as through infected blossoms and infected fruit.

The writers have not seen in commercial peach orchards a twig or branch lesion in which the brown-rot fungus was growing that could not be traced to a blighted blossom or to a mummied fruit. They have seen blighted twigs of noncommercial varieties that appeared to have been directly infected through spores.

Jehle (*48*) reported that cankers live over and increase in size from year to year. Although a callus layer forms about these cankers each year, it also may become infected. Smith (*90*) found lesions on stems producing conidial tufts occasionally during their second year. McClintock (*52*) in Georgia and Cunningham (*24*) in New Zealand stated that these stem lesions may produce conidia during the following spring and consider them important as infection centers. In Delaware, Manns and Adams (*55*) reported that the fungus survived the winter of 1921–22 in 75 per cent of the cankers on the Carman variety examined, but that there was no evidence of survival in blossom-blight cankers on the Belle and Elberta varieties. Brooks and Fisher (*16*) were unable to find conidia on cankered limbs of prunes, although a careful search was made. Berkeley (*11*) in Canada reported that in the spring of 1926 many cankers were active and spores were present in them. He also found active 2-year-old cankers.

It has been the writers' experience that the fungus frequently does not live over in blighted twigs and cankers. In March, 1925, at a time when in northern Virginia mummies were producing conidia in large numbers, 19 cankers that had formed about these mummies were collected, and in no case was there a development of the brown-rot fungus from the cankers, although it developed readily from the fruit stems. McClintock (*53*) in the season of 1929 was unable to find conidia produced in such cankers, although he examined hundreds of them. McCubbin (*54*) in Canada was unable to obtain cultures of the brown-rot fungus from dead twigs and cankers on which mummies were found, and he concluded after experimentation that the killing was caused by juice from the rotted peaches passing into the twigs.

Beginning in 1923 the junior writer investigated the longevity of twig cankers in Georgia, particularly those following blossom infections. When the inner tissues of young cankers, collected in April, were removed under aseptic conditions and placed on potato-dextrose agar in Petri dishes, pure cultures of the organism were readily secured. (Pl. 6, D.) Similar results were obtained during May. In August many of the cankers had ruptured, and numerous contaminations developed in cultures from the tissue fragments, but the brown-rot fungus was also obtained in each trial. The experiments were continued during September, October, and December. In these months the number of tests in which the brown-rot organism was not secured increased each month, and in December the organism was secured from only one canker in a series of eight tested. No experiments were performed in 1924, and blossom blight was not severe in 1925 and 1926, so that the twig cankers were not abundant. Blossom blight was severe again in 1927, and work on a larger scale

was started with the cankers that developed that year. Since the earlier work had shown that the organism was alive in a certain number of cankers until December at least, cankers were not examined until January, 1928. One hundred and five cankers were collected at intervals during January and February before the trees started to blossom. Fragments of the interior portion of each canker were removed under aseptic conditions and placed on plates of potato-dextrose agar. The brown-rot organism was secured from 41 cankers, or 39 per cent of the total number. In each case a record was kept as to whether the canker was still open or whether the callus tissue had closed over the original necrotic area. In addition a record was kept of the location of the tissue used in each test, whether from the apical, central, or basal portion of the canker. These data are summarized in Table 5. A higher percentage of successful isolations of the brown-rot fungus was obtained from tissues taken from the center of the cankers than from the apical and basal portions, and also a higher percentage of isolations was obtained from those cankers that were not covered by callus.

TABLE 5.—*Results of attempts to isolate Sclerotinia fructicola from different parts of twig cankers—January and February, 1928*

Part of canker	Cankers used	Cankers from which fungus was isolated	
	Number	*Number*	*Per cent*
Apex	28	11	39
Center	49	22	45
Base	28	8	28
Necrotic region covered by callus tissue	38	5	13
Necrotic region not covered by callus tissue	67	36	54

Blossom blight and twig cankers were prevalent in Georgia again in 1928, and the experiments of the previous year were repeated during February and March, 1929. Fifty-four cankers were studied during this period, and the fungus was isolated from seven of them.

In addition to the isolation studies, field observations were made in 1923, 1924, and 1925 for evidence of the production of conidia on overwintered cankers. No conidia were observed on overwintered cankers, although production of conidia is not particularly uncommon during the season in which the cankers are initiated. In March, 1928, 100 cankers on the trees of the Hiley variety in an orchard near Fort Valley, Ga., were selected at random and tagged. These cankers were the result of blossom infection in 1927. Blossom blight appeared in the orchard on March 28, signifying that the fungus was active, but a careful examination of each canker with a hand lens failed to reveal any signs of spore production on the surface of the cankers.

On April 12, after a rainfall of 2½ inches, two spore tufts were found on one canker. Germination tests showed that the spores were viable. Additional examinations were made during the rest of the season, but spore tufts were not observed on any of the cankers. The canker which produced conidia on April 12 was of the open type, i. e., one in which the callus layer had not united.

From these isolation studies, extending over a period of six years, it seems evident that the fungus remains alive over winter in a certain percentage of the twig cankers each year. This fact would be more important if the field observations during the same period had not failed to reveal the production of spore tufts (with one exception) on the cankers. The survival of the fungus in the tissues of the twig without the production of the spores is of no importance so far as the spread of the disease is concerned. These conclusions, based on studies in Georgia and Virginia, may not be applicable to all peach-growing sections. Under different environmental conditions it is possible that the cankers may assume a more important rôle in the spread of the disease.

LEAF INFECTION

Leaves that have been killed by the brown-rot fungus are common enough, but the number of leaves killed is so slight that the reduction in total foliar surface is inconsequential. Under optimum conditions for infection, leaves may be attacked and the fungus may be found fruiting in brown spots which later may be excised. More frequently the entire leaf is involved, as when the fungus enters from a blossom-blight or a mummied-fruit canker through the petiole, or when a leaf, coming into such close contact with a rotting or mummied fruit that the exudate from the rot causes it to adhere closely, becomes infected throughout.

In moist weather the fungus may enter leaves injured by other agencies, especially by the leaf-curl fungus, *Exoascus deformans*. Like the blighted blossoms, twig cankers, and early-infected fruits, leaves infected early in the season aid in the dissemination of the fungus and help to provide an abundance of conidia that may infect the ripening fruit.

RELATION OF INSECT PESTS TO INFECTION

Although insect pests are probably of little importance in the dissemination of the disease, they are of great importance as agents that produce wounds in the fruit, through which infection readily takes place. The plum curculio (*Conotrachelus nenuphar* Hbst.) is the most important of these pests, although locally the oriental fruit moth (*Grapholitha molesta* Busck) is sometimes of greater importance. So closely is the curculio associated with peach rot that in former times growers apparently considered the curculio the cause of the rot.

In 1869 Riley (*75*, *p. 52*) in a report on the curculio stated: "* * * By its punctures it causes the dreaded peach-rot to spread whenever that disease is prevalent * * *." In the process of feeding and laying eggs the curculio makes numerous punctures over the surface of the peach, and these open wounds furnish ideal entry for brown-rot spores. When the beetles are numerous, the infection of these punctures by the brown-rot fungus causes severe damage to the fruit crop if the weather conditions are favorable. This rôle of the curculio in the spread of brown rot is well known and widely recognized. It is not known whether or not this insect inserts spores when it punctures the fruit, but it is certain that a coating of fungicide gives little protection to fruits punctured by the plum curculio.

When a specimen of the plum curculio is examined under suitable magnification, it is found to be ideally equipped as a carrier of brown-rot spores. The segments of the abdomen, the wing elytra, the femora, tibiae, and tarsi are all covered with closely set bristles or bristlelike hairs, and it is very obvious that if one of these insects brushed against spore tufts on the surface of the fruit many spores would adhere to it.

The first step in testing the truth of these deductions consisted in allowing plum-curculio beetles to walk over vigorously sporulating Petri-dish cultures of the brown-rot fungus. The beetles were then examined under a binocular microscope, and numerous brown-rot spores were found adhering to the under surface of the abdomen and to the various segments of the legs. As a further verification, additional beetles were transferred from the brown-rot cultures to sterile agar plates. In due time colonies of the brown-rot fungus developed along the paths in the agar made by the beetles.

In order to make a test under more nearly natural conditions, beetles confined in large battery jars were allowed to feed on rotting peaches for 24-hour periods, after which they were transferred to other jars containing sound peaches. Numerous brown-rot infections developed on these peaches within 24 hours after the transfer of the beetles, and the infections developed in practically all cases at punctures made by them. Jars containing sound peaches to which no beetles were added were maintained as checks, and these peaches remained sound long after the fruit in the jars to which the beetles were added was completely rotted.

A modification of this test consisted in allowing the beetles to feed for 24 hours on rotting fruit and then transferring them for 24 hours to a jar containing nonsprayed peach foliage. The beetles were then transferred to a jar of ripe peaches. Brown rot developed on these peaches within 48 hours, showing that spores still adhered to the beetles after 24 hours feeding on foliage. The checks behaved as in the previous experiment. These experiments were carried out in 1922 and repeated with similar results in 1924.

Although these experiments definitely demonstrate that the plum curculio can serve as a mechanical agent in the dissemination of brown-rot spores, this insect, according to O. I. Snapp, entomologist in the Bureau of Entomology, United States Department of Agriculture, feeds in the field exclusively on sound peaches. Accordingly it seems very probable that the plum curculio is an effective agent in the mechanical dissemination of brown-rot spores only in years that are favorable to the development of the disease when the chances of brushing against spore masses on rotting fruit are numerous. The habit of feeding exclusively on sound tissues largely precludes the possibility of the plum curculio being of much importance as a biological agent in the spread of the disease by eating brown-rot spores and passing them intact through its digestive system.

In experiments conducted jointly by the Bureaus of Entomology and Plant Industry it has been found that in most years about 90 per cent of the brown-rot infections occurring in peach orchards take place through curculio punctures. In those favored sections in which the fruit is free from insect punctures, particularly those of the plum curculio and the oriental fruit moth, brown rot is comparatively unimportant as an orchard disease.

PATHOLOGICAL ANATOMY

FLORAL PARTS

The mycelium of the brown-rot fungus is intercellular in the petals and sepals of the peach blossom. In both these organs the cells are so loosely arranged and the intercellular spaces so numerous that the mycelium spreads rapidly through the entire organs. Sections prepared from the style and stigma of artificially inoculated peach blossoms show that the conidia germinate (pl. 7, A) on the surface of the stigma and that the germ tubes grow down intercellularly among the loosely arranged cells of the style in much the same manner as the pollen tubes. All the parenchymatous tissues of the ovary may be invaded. (Pl. 7, B and C.) The mycelium was observed in the spaces between the pollen grains in mature anthers. Conidia may be produced on the surface of all floral parts.

FRUITS

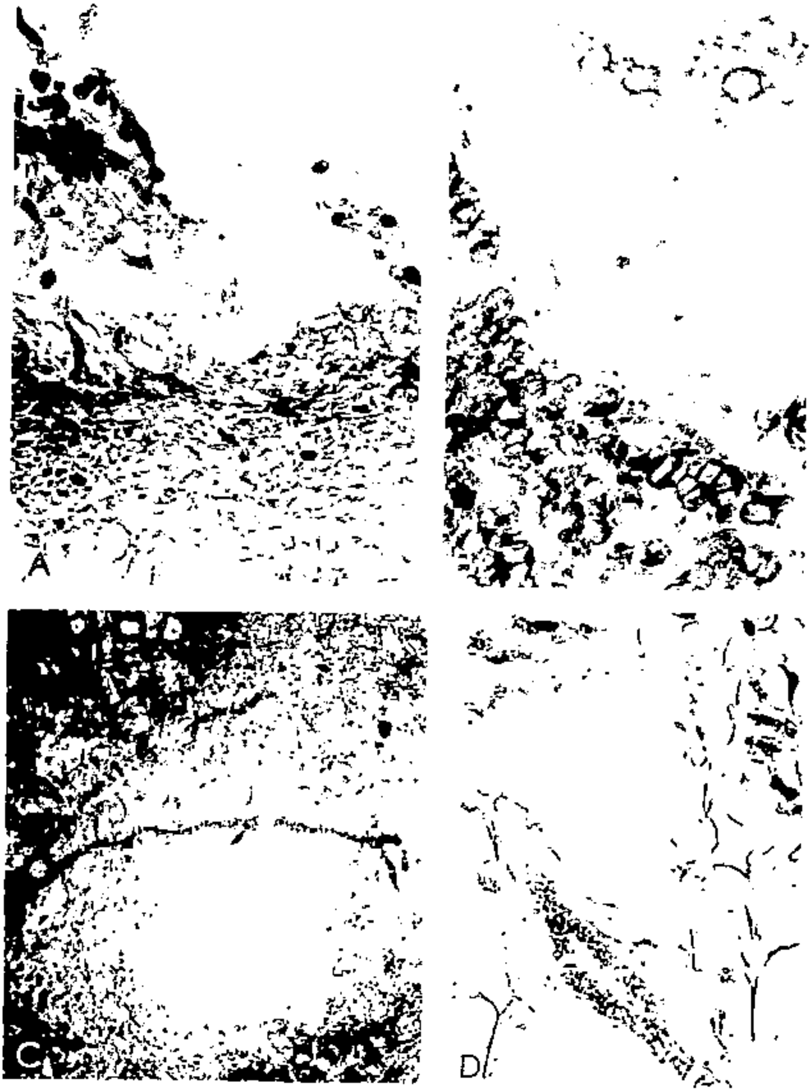
In peach fruits the fungus is intercellular. (Pl. 7, D.) It dissolves the middle lamella and by occupying much of the space thus formed separates the cells of the invaded part of the peach from one another. Galloway (*39*) found the mycelium intercellular but states that in some cases it seemed to have penetrated the cell walls. Cooley (*20*) stated that hyphae penetrate the cells of the plum at any point of contact, but Valleau (*97*) observed that in the tissues of plum and apple the hyphae are entirely intercellular. Valleau also observed that the middle lamella is dissolved slightly in advance of the penetration of the hyphae. He considers that the absence of the middle lamella in the tissues of ripe fruits explains the rapidity with which rot develops in them.

TWIGS

In 1891 Smith (*92*) presented a brief account of the histology of peach-twig cankers caused by the brown-rot fungus, but since that time, so far as the writers are aware, no detailed histological work has been done. It is true that Jehle (*48*), Fant (*37*), and Cook (*19*) have discussed the cankers at some length, but their interest in them was mainly from an etiologic viewpoint.

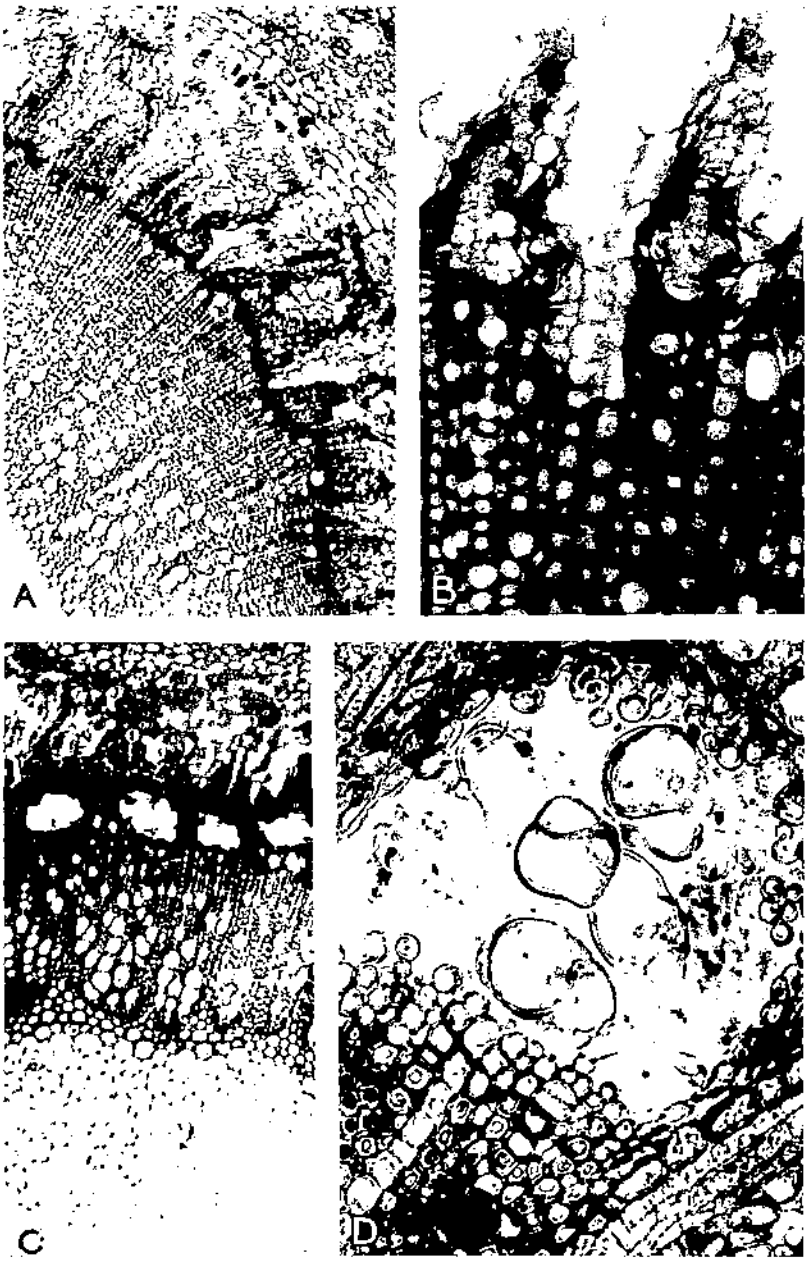
Twigs with blighted blossoms and cankers resulting from blossom infections were collected from trees of the varieties Hiley and Uneeda at various times during the years 1927 and 1928. Sections were prepared from these twigs by means of a sliding microtome. Some of the sections were stained with safranin and light green, and others were mounted unstained. In the first sections examined the fungus was growing through the peduncle but had not reached the tissues of the twig, and the only symptom present was a crescent-shaped discolored region on one side of the peduncle.

The first symptom of canker formation in the tissues of the twig is the presence of a discontinuous narrow brown zone in the region of the cambium. (Pl. 8, A.) The cells in this region are dark colored, and the walls have collapsed. The discolored zone is discontinuous because the cells of the larger pith rays are not affected. This is clearly illustrated in Plate 8, B. The mycelium of the fungus has not been observed in the tissues of very young cankers,



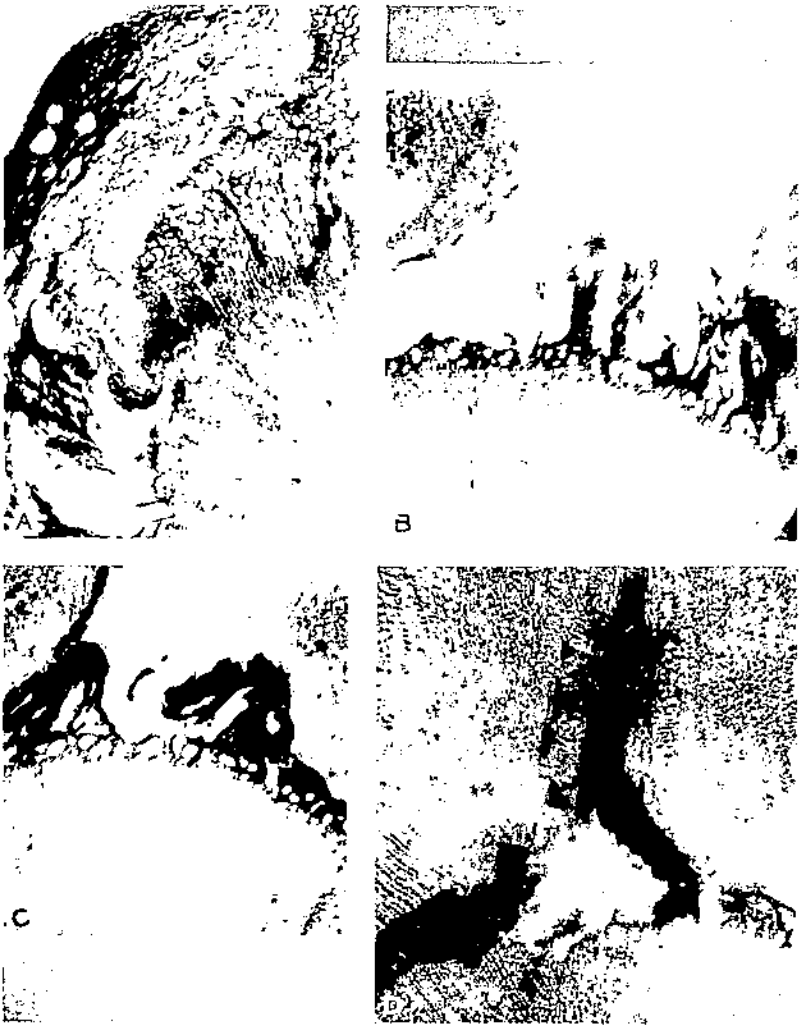
BLOSSOM BLIGHT OF THE PEACH CAUSED BY SCLEROTINIA FRUCTICOLA

A, Conidia germinating on the [illegible] of [illegible] peach blossom; B, mycelium in space between ovary wall and young ovule of infected peach [illegible]; C, [illegible] penetrating tissues of the young ovule; D, [illegible] Yellow Hales peach. All × [illegible].



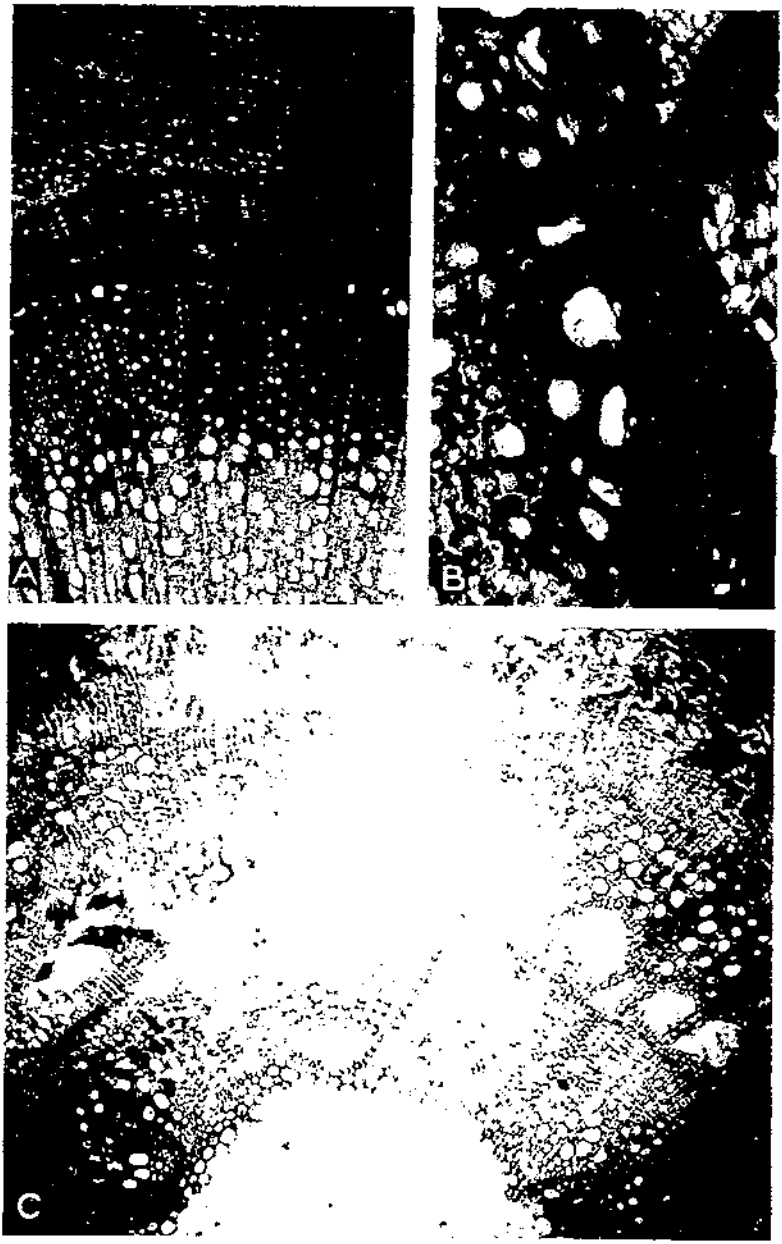
PEACH TWIG CANKERS CAUSED BY SCLEROTINIA FRUCTICOLA

A, Early stage in formation of twig cankers. The discontinuous dark line in the region of the cambium is one of the first symptoms. × 80. B, similar to A but at higher magnification to show more clearly the collapse of the tissue in the region of the cambium. × 340. C, The gum-pocket stage in the formation of the twig canker. × 80. D, Gum pocket at higher magnification. × 340



PEACH TWIG CANKERS CAUSED BY SCLEROTINIA FRUCTICOLA

A, Formation of a wound periderm extending from the epidermis to the cambium at some distance from the necrotic tissue; B, callus tissue rolling in over necrotic region; C, callus tissue from the two sides almost united; D, callus tissue united, leaving a small triangular-shaped gap at the center of the necrotic region. All ×10.



PEACH TWIG CANKERS CAUSED BY SCLEROTINIA FRUCTICOLA

A, Callus and necrotic tissue. The callus tissue does not differ with respect of the necrotic region but is merely superimposed on it. ×80. B, Portion of A at higher magnification. The dark zone is the crushed fragments of the necrotic tissue. ×240. C, Twig collected and sectioned May 19, 1927, showing new tissue formed external to gum pockets which developed as the result of blossom blight in March, 1927. ×80.

and it is not known whether the discoloration and collapse of the cells in the region of the cambium is caused by actual penetration of the cells by the hyphae of the fungus or by enzymes liberated by the fungus. These early symptoms pass very soon into a condition in which there is a circle of gum pockets (pl. 8, C) in the region formerly occupied by the discolored zone. It is very evident that these gum pockets are merely the aftermath of the killing of the cells. While the gum pockets may at times form a band encircling the xylem, they remain as distinct entities because the cells of the larger pith rays are not affected. (Pl. 8, C.) Smith (*92, p. 38*) has described the conditions of the tissue at this period in the development of the cankers as follows:

> The cambium and soft bast cylinders had disappeared almost completely with the formation of extensive gum pockets. These pockets were full of the active mycelium of *Monilia*. This also penetrated into the cortical parenchyma to some extent, and to a lesser degree into the xylem. Practically speaking, the wood and the pith and all the cylinders external to the soft bast were intact. On unmagnified cross sections a zone of discoloration was visible below the wood and the bark. On magnification this was found to consist * * * of a gum cavity containing mycelium and fragments of tissue and bordered by irregular dark zones, the one within composed of young wood and vessels laid down this spring, and the one without composed of remnants of soft bast and phloem rays. The bundles of fibers were also changed from a glistening white to a dirty yellowish brown.

Smith apparently found the mycelium of the fungus in the cortical parenchyma. The writers have not been so fortunate, but there can be no doubt that the cells in this region are seriously affected. There is no pronounced dissolution of the cell walls with formation of gum pockets, but instead the cells lose their turgidity, and the walls, instead of being practically hyaline, are brown and are swollen to almost twice their normal thickness.

The formation of the extensive gum pockets in the region of the cambium and the destruction of the cortical parenchyma are the final episode in the histological changes produced directly by the fungus. The subsequent changes are associated with the development of callus tissue to cover the necrotic region of the twig. A wound periderm is initiated at each side of the necrotic region and extends as a sloping, irregular band from the region of the cambium to the epidermis. (Pl. 9, A.) This periderm is initiated not at the apparent boundary between the mass of necrotic, disrupted cells and the healthy tissues, but at some distance from this boundary in apparently healthy tissues. Microchemical tests to detect a suberization of cells preceding the initiation of a wound periderm gave negative results. The tests, however, were too few to warrant drawing a definite conclusion on this point.

The regeneration of the tissues of the twig presents no striking novelties and is well described by the following generalized description of the process taken from Strasburger (*94, p. 164*):

> In stems of Gymnosperms and Dicotyledons, wounds which extend into the wood become surrounded and finally overcapped by an outgrowth of tissue arising from the exposed cambium. While the callus tissue is still in process of gradually growing over the wounded surface, an outer protective covering of cork is developed; at the same time a new cambium is formed within the callus by the differentiation of an inner layer of cells, continuous with the cambium of the stem. When the margins of the over-growing callus tissue ultimately meet and close together over the wound, the edges of its cambium unite and

form a complete cambial layer, continuing the cambium of the stem over the surface of the wound. The wood formed by this new cambium never coalesces with the old wood which is brown and dead.

The various stages in the overcapping of the necrotic region are illustrated in Plate 9, B, C, and D, and Plate 10, A and B.

The histological changes come to an end when the cambium of the two layers of the callus tissues, which have been gradually approaching each other, unite. This fusion of the cambium is followed by the production of normal rings of wood, and usually by the end of the second season after the initiation of the canker it is impossible to tell from the appearance of the twig surface that a canker had been present.

Earlier in the discussion it was stated that a ring of gum pockets may encircle the xylem. These gum pockets persist in the tissues but cease to enlarge, and new tissues are laid down exterior to them. (Pl. 10, C.) The cambium that initiates the new tissues probably originates by a lateral division of the uninjured cambial cells capping the pith rays.

CONTROL OF PEACH BROWN ROT

SANITARY MEASURES

The removal of mummied fruits, blighted twigs, and all other parts infected by the fungus was recommended by early investigators as the sole means of control of peach brown rot. Later, with the development of spraying, orchard sanitation was recommended only as a supplementary control measure. Smith (*91*) advocated the removal of rotting fruits as soon as they appear in the orchards and before spores are formed on them. He insisted, however, that to be successful it must be a community affair, since one neglected orchard may furnish spores enough to reinfect all the surrounding orchards. Quaintance (*70*) suggested, in addition to removal of mummies and rotted fruits, the pruning out of blighted spurs and twigs.

Jehle (*48*) recommended the removal of mummies early enough to prevent the growth of the fungus through the stem of the mummy and into the twig. He also recommended the removal of cankers. McClintock (*52*) suggested that cankers be pruned out just after harvest so that the cankered twigs may be removed from the orchard along with the rotten fruit collected from the trees and from the ground. Pollock (*68*) considered the plowing under of mummies a doubtful help in control, because he found that the sclerotia from which apothecia arise could live in the soil for at least 10 years. On the other hand, McCubbin (*54*), Archibald (*4*), and Brooks and Fisher (*16*) found that plowing greatly reduced apothecial production, and they considered it as helpful in control. Ezekiel (*33*), as the result of laboratory experiments, suggested that liming the soil might be helpful in the prevention of apothecial production, but he did not make orchard tests with lime. Later (*36*) he found that mummies disintegrate very rapidly when buried in the soil, and accordingly he recommended fall plowing. He also recommended a loosening of the soil by cultivation during the pink-bud stage of the peach in early spring, since mummies disturbed at that time rarely produce apothecia.

It is certain that sanitary measures alone will not control any phase of brown rot, and it is difficult to estimate the benefits of orchard sanitation. Removal of mummies, however, undoubtedly has some value in the control of brown rot in its various phases. As stated by Smith (*90*), to be effective, removal of mummies should be a community affair, because the conidia may be easily borne by the wind from a neglected orchard to one in which all sources of infection have been removed in so far as practicable. It is the dissemination of conidia by the wind that prevents experimentation on the effectiveness of this method of control. One might consider that the removal, in so far as practicable, of all infection sources in one half of an orchard, leaving the other half as a check, should result in less brown rot in the one half than in the other, but it would be found that sufficient conidia had been blown into the cleaned half to start numerous initial infections which would soon become new infection centers.

The removal of newly infected blossoms, leaves, and twigs is quite impracticable, as is also the removal before conidia are formed of newly infected fruits except in small plantings. Frequently growers instruct pickers to collect all rotted and mummied fruits along with the sound, thus enormously increasing the chance of infection by increasing the number of conidia on the surface of the fruit. Collection of the rotted fruits is made for the purpose of removing from the orchard possible sources of infection for the following year, but it should not be done at the time when the sound fruits are gathered. It is, however, both practicable and helpful to remove rotten fruits directly after picking the crop, to prevent twig infection and canker formation, besides reducing the number of prospective mummies. Removal of rotten fruits from trees of early varieties also helps somewhat in the protection of later varieties by reducing the number of infection sources.

The brown-rot fungus attacks many species of wild plum, and all the different phases of the disease described for the peach, viz, blossom blight, twig cankers, fruit rot, and mummy formation, may be observed on these wild hosts. The writers have observed apothecia produced in abundance under trees in thickets such as that illustrated in Plate 4, B. When blossom blight develops in these thickets, with the production of myriads of conidia on the blighted blossoms, it is only too evident that wild plum trees serve as a fertile source of infectious material, both ascospores and conidia, that may be blown to near-by commercial orchards. Since the disease runs its course unchecked on these wild hosts, it is also evident that dangerous centers of infection may exist year after year. Spores blown from these wild plums may initiate brown rot in peach orchards previously free from the disease.

The writers realize the impossibility of completely eliminating wild plums and wild-peach seedlings from any given district, but in sections devoted largely to peach production it is unwise to allow plum and wild-peach seedling thickets like that shown in Plate 4, B, to develop near commercial orchards. Here again the problem is one of concerted community action; and if all the growers cut down the trees on their own property at regular intervals, much of the danger of brown rot from wild plum and peach seedlings is eliminated.

Failing in community action, the individual growers should at least cut down all such trees along fence rows and at the edges of fields bordering their orchards.

Information on orchard sanitation as a supplementary control of brown rot may be summarized as follows:

To be most effective it should be a community affair.

Mummies hanging on the trees should be removed from the orchard as soon as possible after the crop has been harvested.

Mummies on the ground should be plowed under or otherwise disposed of in the fall or in the spring before the blossoms open.

Cankers and dead twigs should be removed at pruning time for the good of the trees, but their removal probably is of little help in controlling the disease.

Wild-plum trees and peach seedlings near peach orchards should be cut down at regular intervals, and if possible thickets of wild-plum trees and peach seedlings should not be allowed to develop in sections devoted to peach culture.

SPRAYING AND DUSTING

Certain sprays or dusts applied during the growing season control the brown-rot disease to a greater or lesser extent, depending on the season. All other methods of control are supplementary to spraying or dusting and are useful only in that they make control by spraying or dusting more nearly complete or more readily accomplished. During at least a part of the season the fungicidal sprays or dusts should be combined with an insecticide for the control of the plum curculio. As previously stated, the control of the curculio is important in the control of brown rot, because, except in years particularly favorable to the development of the rot, about 90 per cent of the brown-rot infections in the orchard take place through curculio punctures.

Sprays applied during the dormant season, even those many times stronger than the lethal dosage for brown-rot spores, have no noticeable effect in controlling the disease. Manns and Adams (*55*) found that from cankers on dormant peach twigs that had been dipped in strong Bordeaux mixture and dried for 24 hours the fungus pushed out through the fungicide and produced abundant conidia.

Fungicides such as Bordeaux mixture, potassium sulphide, and flowers of sulphur had been recommended at times for use on the peach, but it was not until 1907, when Scott (*86*) introduced a mixture of sulphur, freshly slaked quicklime, and water, which he called self-boiled lime-sulphur, that spraying for the control of brown rot became practical and effective. The fungicides previously recommended either injured the trees severely or did not control the disease. Bordeaux mixture, which at one time had been generally recommended, was abandoned because even with the minimum content of copper necessary for the control of brown rot it frequently injured the foliage of the peach severely and caused defoliation in the early summer. Scott's mixture controlled the disease without causing serious injury to foliage or other parts of the tree. Scott, after considerable experimentation in orchards, finally decided that the self-boiled lime-sulphur gave best results when it was composed of 8 pounds of sulphur and 8 pounds of stone lime to 50 gallons of water. At this strength scab and brown rot were controlled, and arsenate of lead could be added to the mixture without danger of

serious injury to the peach. The heat from the slaking of this quantity of lime was effective in forming an intimate mixture of sulphur and milk of lime, which remained in suspension for some time. The sprays now most commonly used are essentially the same as Scott's mixture of sulphur and lime except that these ingredients are brought into suspension by the action of a colloid such as casein or glue instead of heat.[11]

With the development of a finely powdered sulphur, about 1912, and efficient machinery for its application, dusting for the control of brown rot has yielded good results and is widely used. Sulphur in this form is mixed with hydrated lime and powdered arsenate of lead to form a mixture which is effective against brown rot, scab, and curculio, and causes little injury to the peach.

Discussion of the effectiveness of spraying and dusting with fungicides in the control of brown rot will be divided into two parts: (1) Effectiveness in the prevention of blossom blight and resulting twig cankers; (2) effectiveness in the prevention of fruit rot and incidentally of twig and limb infections resulting from fruit rot.

CONTROL OF BLOSSOM BLIGHT

An application of a fungicide just before the blossoms open has long been recommended for the control of the brown-rot blossom blight of stone fruits. Galloway (*39*) recommended it for the control of cherry-blossom blight and Chester (*17*) for the control of peach-blossom blight. Cory (*22*) in Maryland, Berkeley (*11*) in Canada, and Jehle (*48*) in New York have reported good results from applications made before the peach blossoms open, i. e., when the pink color of the petals is visible in the still unopen buds. Cunningham (*24*) in New Zealand recommends two preblossom applications, the first when the buds begin to swell and the second when the pink of the petals is first visible. Brooks and Fisher (*16*) in Washington and Oregon reduced the number of blossom infections of prunes and cherries by a preblossom application, but usually the application did not result in an increase in the crop of fruit.

Rudolph (*82*) in California found that in severe cases of apricot-blossom blight caused by *Sclerotinia cinerea*[12] it was necessary to spray the open blossoms as well as the blossom buds with Bordeaux mixture to obtain control. Tesche (*95*) reports reduction of the apricot-blossom blight by two preblossom applications of Bordeaux mixture to which oil had been added as a spreader. Robertson (*81*) in British Columbia did not control cherry-blossom blight, caused presumably by *S. cinerea* (*Monilia oregonensis*), with an application of Bordeaux mixture in April following a pruning out of useless wood and dead-fruit spurs.

Rice (*74*) in New Zealand reports no effective control of peach-blossom blight by spraying, and McClintock (*52*) in Georgia found that an application of strong lime-sulphur solution applied when the buds were swelling was of little value in the control of blossom blight of Early Wheeler (*Red Bird*) peaches.

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[11] For specific directions for making self-boiled lime-sulphur and its substitutes see Farmers' Bul. 1527 (*80*).

[12] It is commonly accepted that this fungus is present on the Pacific coast, although the apothecial stage has never been observed.

In the years 1922 to 1924, inclusive, the writers performed experiments designed to test the effectiveness of preblossom spraying in the control of brown-rot blossom blight in central Georgia. In that section blossom blight is normally prevalent only on early varieties, although in seasons particularly favorable to its development it may be found to a slight extent on all varieties. Even with the more susceptible early varieties it is only in certain seasons that control measures are needed. Spray experiments were conducted over a period of three years, but no satisfactory results were obtained. In the orchards used there were abundant mummies producing conidia and ascospores, so that there was no lack of infectious material.

The selection of the proper time for applying the spray is an important problem in spraying for the control of blossom blight. Since, as previously shown, all parts of the blossoms are susceptible, it was thought that a spray applied to open blossoms would be effective. It was found, however, that it was not possible to find all the blossoms on all the trees open at anything like the same time. In 1922 only one-half the blossoms were open when the sprays were applied. In 1923 the time of application was delayed; but even when 20 per cent of the blossoms had dropped their petals, 13 per cent were not yet showing pink. In 1924, with an even greater delay, 8 per cent were in a stage too small to be protected. The only solution of the timing problem would be two or three applications during the blossoming period, which would be so expensive as to be prohibitive. Various materials have been used, but lime-sulphur solution (33° Baumé) diluted at the rate of 2 gallons to 50 gallons of water has given the best results. In 1924 when the sprays were applied late in the blossoming period the dilute lime-sulphur solution caused slight injury to the blossoms, and it is quite possible that two or three applications would cause severe injury. Some of the colloidal sulphur sprays could probably be used with safety, but they would not adhere so well as lime-sulphur solution. Self-boiled lime sulphur with and without casein lime was less effective than lime-sulphur solution, and a dust composed of finely ground sulphur, 85 per cent, and hydrated lime, 15 per cent, was wholly ineffective.

CONTROL OF FRUIT ROT

Scott (*86*) not only worked out a safe fungicide for use on the peach in the growing season but also, in cooperation with Quaintance, of the Bureau of Entomology, worked out a combined spraying schedule for the control of the curculio, scab, and brown rot. For varieties approximating the season of the Elberta variety this schedule was as follows:

First application.—When the calyces or "shucks" are shed, which is usually about 10 days after the petals have fallen, spray with a suspension of arsenate of lead and milk of lime in water for the control of the curculio.

Second application.—Two weeks after the first application, or about four weeks after the petals have fallen, spray with self-boiled lime-sulphur to which arsenate of lead is added. This application is primarily for the control of curculio and scab.

Third application.—About one month before the fruit ripens, spray with self-boiled lime-sulphur for the control of brown rot.

Scott found after long experimentation that earlier treatments with self-boiled lime-sulphur were unnecessary. He and Ayres (*87*)

also found that the sprayed fruit was less liable to rot in transit and on the market than the unsprayed. At the present time many investigators advise earlier applications for the control of brown rot, but such applications are of doubtful usefulness.

After the introduction of finely ground dusting sulphur about 1912 the use of this substance for the control of brown rot began, and it is now extensively used for that purpose. It has the advantage of being easily and quickly applied; and applications can be made shortly before harvest, when the fruit is reaching the stage of greatest susceptibility to the disease, without the staining of fruit which often results from applications of spray made at that time.

The spray or dust schedule recommended for general use at the present time follows closely the original schedule devised by Scott and is as follows:

First application.—When calyces or "shucks" are shedding, which is usually about 10 days after the falling of the petals—

Spray: Powdered arsenate of lead,[13] 1 pound (or 2 pounds of the paste), and the milk of lime from 3 pounds of stone lime or 4 pounds of hydrated lime, with water sufficient to make 50 gallons; or

Dust: (1) Hydrated lime, 95 per cent; arsenate of lead, 5 per cent; or (2) sulphur, 80 per cent; arsenate of lead, 5 per cent; hydrated lime, 15 per cent.

Second application.—Two weeks after the first application, or about four weeks after the petals have fallen—

Spray: Self-boiled lime-sulphur 8–8–50 (or substitute), to each 50 gallons of which 1 pound of powdered arsenate of lead (or 2 pounds of the paste) is added: or

Dust: Sulphur, 80 per cent; arsenate of lead, 5 per cent; hydrated lime, 15 per cent.

Third application.—One month before each variety is expected to ripen—

Spray: Self-boiled lime-sulphur 8–8–50 (or substitute) without the addition of arsenate of lead; or

Dust: (1) Sulphur, 80 per cent; hydrated lime, 20 per cent; or (2) sulphur, 80 per cent; arsenate of lead, 5 per cent; hydrated lime, 15 per cent.

For the southeastern part of the United States, including Georgia and the Gulf States, the following schedule is recommended:

First application.—Immediately after 75 per cent of the petals have fallen—

Spray: Powdered arsenate of lead, 1 pound (or 2 pounds of the paste), and the milk of lime from 3 pounds of stone lime or 4 pounds of hydrated lime with water sufficient to make 50 gallons; or

Dust: (1) Hydrated lime, 95 per cent; arsenate of lead, 5 per cent; or (2) sulphur, 80 per cent; arsenate of lead, 5 per cent; hydrated lime, 15 per cent.

Second application.—When calyces or shucks are shedding, which is usually about 10 days after the falling of the petals—

Spray: Same as for first application; or

Dust: Same as for first application.

Third application.—Two weeks after the second application, or about four weeks after the first application—

Spray: Self-boiled lime-sulphur 8–8–50 (or substitute); or

Dust: (1) Sulphur, 80 per cent; hydrated lime, 20 per cent; or (2) sulphur, 80 per cent; arsenate of lead, 5 per cent; hydrated lime, 15 per cent.

Fourth application.—One month before each variety is expected to ripen—

Spray: Self-boiled lime-sulphur 8–8–50 (or substitute), to each 50 gallons of which 1 pound of powdered arsenate of lead (or 2 pounds of the paste) is added; or

Dust: Sulphur, 80 per cent; arsenate of lead, 5 per cent; hydrated lime, 15 per cent.[14]

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[13] Directions for the use of arsenate of lead in the control of the curculio were furnished by the Bureau of Entomology, U. S. Department of Agriculture.

[14] For a more complete discussion of spray schedules, specific directions for making self-boiled lime-sulphur and substitutes for it, and dust formulas, the reader is referred to Farmers' Bulletin 1527 (*80*) and Farmers' Bulletin 1557 (*93*).

FUMIGATION

The lethal effects of the vapors of certain substances on bacteria, fungi, insects, and other forms of life are well known. Their employment as antiseptics, disinfectants, germicides, and insecticides has been a common practice for many years. It is not surprising, therefore, to find a large volume of literature dealing with the subject. A review of the literature dealing only with the more important contributions to the subject would far exceed the limits of this bulletin. It is sufficient to say that the experiments herein reported represent an attempt to determine the possibility of a successful specific application of the well-known principles of fumigation.

As a result of the accidental discovery that *Sclerotinia fructicola* is particularly susceptible to vapors given off by ethyl alcohol, these experiments were begun with the hope that a substance might be found whose vapors would be of practical use in the killing of brown-rot spores and hyphae on peaches and other stone fruits either in closed compartments at packing houses or in refrigerator cars during transit. For many years it has been the custom of the senior writer after transferring fungus cultures from one tube to another to moisten the lower portion of the cotton plug above the new culture with a mixture of chemicals[15] used for the killing of mites. In the writer's experience this mixture had never affected in any noticeable way the growth of the new culture, but in the case of the brown-rot organism culture transfers from 18 different sources all failed to grow. By testing the ingredients of the mixture separately it was found that alcohol was the only one that prevented growth; and since only the vapor of this substance could reach the fungus, it was assumed that it was responsible for the failure of the transferred conidia and hyphae to develop. Of the other ingredients mercuric chloride is well known to be toxic to fungi when in contact with them, but it does not vaporize sufficiently, at least, to be toxic to the brown-rot fungus when a solution of it is applied to the culture-tube plug. The two remaining ingredients, arsenate of lead and glycerin, when applied to culture-tube plugs had no effect on growth.

The choice of substances for use in the experiments followed no general rules. Some of the essential oils, such as oil of thyme, oil of eucalyptus, and oil of peppermint, were chosen because of their well-known antiseptic and germicidal properties. Others were used at the suggestion of G. A. Russell, then of the office of Drug Plant Investigations, Bureau of Plant Industry. Benzaldehyde was used on account of the toxicity of aldehydes for plant growth and because of its cheapness. Copper sulphate was used to ascertain whether or not it had fungicidal powers when not in contact with conidia or hyphae. Mercury and aniline are too toxic to man to be very promising. The former received a trial because Larson (*51*) had found it to be toxic to the larvae and eggs of the bean weevil when in the same jar but not in contact with them. Aniline, acetic acid, carbon tetrachloride, furfural, and several other chemicals were tried because it was considered desirable to try readily obtainable substances

[15] Formula: Alcohol, 95 ml; glycerin, 50 ml; bichloride of mercury, 2 g[16]; arsenate of lead, 1 g.

[16] g (instead of gm.) is the abbreviation recently adopted by the Government Printing Office for gram or grams.

of different chemical composition. Tricresol and toluene were tried only because they are well-known germicides. Chloral hydrate was chosen because of its narcotic properties. Paradichlorobenzene was introduced by Blakeslee (*12*) for the control of the peach borer and has come into general use for that purpose. The toxicity of acetic acid suggested the use of certain acetates even though they are relatively nonvolatile. Certain commonly used fumigants, such as sulphur dioxide and formaldehyde, were not used, for the reason that they were known to be injurious to peach fruits.

Preliminary experiments to eliminate from further tests those liquid substances that did not prevent growth were made by moistening with 1 to 2 ml of each substance the lower ends of the cotton plugs of culture tubes to which the fungus had been newly transferred. Checks were held in all cases. The following substances entirely prevented growth:

Acetic acid, glacial; alcohol, 95 per cent; aniline; benzaldehyde; carbon tetrachloride; cassia oil; chloral hydrate, saturated aqueous solution; *Eucalyptus globulus* oil; eugenol; horsemint (*Monarda punctata*) oil; lemon oil, pressed; peppermint (*Mentha piperita*) oil; safrol; sassafras oil, artificial; sassafras oil, natural; thyme oil, light; toluene; tricresol; wormseed oil.

The following substances did not prevent growth:

Cedar oil, light; clove oil; copper sulphate, saturated aqueous solution; lead arsenate, 1 g in water 100 ml; mercuric chloride, saturated aqueous solution; sodium salicylate, M/1 aqueous solution.

A crystal of copper sulphate ($CuSO_4 \cdot 5H_2O$) placed in a culture tube at the foot of the agar slant did not hinder growth until, slowly dissolving, it permeated the culture medium and finally came into actual contact with the fungus. Mercury, 0.3 ml, at the foot of agar slants, entirely prevented growth.

To determine whether or not growth in cultures could be stopped after a vigorous beginning, 1 to 2 ml of the following substances were dropped on the lower ends of plugs of culture tubes containing 2-day-old growths of the fungus measuring approximately 1 cm across: Benzaldehyde; lemon oil, distilled; horsemint oil; thyme oil, light. Five days later the slants of the untreated tubes that were held as checks were entirely covered by the fungus, while there was no further growth in the treated tubes. Cultures from the latter tubes showed the fungus to be dead.

In some of the experiments on the effects of exposing conidia to vapors the substance under trial was placed in the bottom of the cavity of a hang-drop slide and the spores were exposed in a hanging drop on the under surface of a cover glass placed over the cavity. After the time allowed for exposure had elapsed the cover glass with its drop containing the conidia was removed and placed on a similar slide containing only water, and later examined for germination. This method was abandoned, for the reason that the exposure of conidia in water was not as they would be exposed in practical usage, and because some of the vapors would probably be carried over in the hanging drop when the change to a different slide was made.

The results obtained by this method, however, agreed very well with those obtained by the following method, which was finally adopted as a standard. The fungus was grown on 4 per cent potato

agar; and when conidia production became profuse, bits of the medium covered with conidia were removed and placed on ordinary microscopic slides. These slides were then placed under inverted battery jars the sides of which had been sprayed with the substance (liquid) to be tested. Solid or dry substances were scattered about under the battery jar one and one-half hours before the beginning of the tests. After the time chosen for exposure had elapsed the conidia were exposed to the air of the room for a few minutes and then tested for germination in hanging drops of either distilled water or sterile prune juice, the latter giving the more uniform results. Checks were held in all cases; and unless the percentage of germination of the nontreated conidia was at least 50, the results were not considered. Usually the percentage was above 90. Since trials showed that conidia not germinating in 24 hours did not germinate at all, results were regularly taken after 24 hours. The results of these experiments are recorded in Table 6.

TABLE 6.—*Results of exposure of conidia of Sclerotinia fructicola to vapors of various substances*

Material	Conidia germinating in 24 hours after exposure for [1]—						
	¼ hour	½ hour	1 hour	2 hours	3 hours	4 hours	5 hours
	Per cent	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*
Acetal		0–55	0–05	0–95			
Acetic acid, glacial	0–50	0–05		0–95			
Acetophenone		0–55	0–95	0–95			
Alcohol, ethyl		100–98		25–05	0–50		
m–Aminobenzoic acid			80–05	95–98		90–98	
Aniline		1–90	1–90	0–90	0–90		
Barium acetate			85–95	98–98		75–98	
Benzaldehyde	0–99	0–99	0–98	0–80			
Benzaldehyde, 0.3 ml; in water, 100 ml			1–80	5–80			
Benzaldehyde, 3 ml; in water, 100 ml	10–50	0–80					
Benzyl acetate			0–98	0–98	0–98		
Benzyl alcohol			0. 01–98	0–98	0–98		
Calcium acetate			75–95	95–05		98–98	
Carbon tetrachloride			90–98	95–80			
p–Dichlorobenzene			0–95	0–95			
Ethyl salicylate			0. 05–100	0–100	0–100	0–100	
Ethylene chlorohydrin	100–98	50–98	30–98	0–95		0–90	
Ethylene dichloride	100–98	50–98	50–100	0–95		0–90	
Eugenol		90–99	65–99				
Formamide			0–85	0–85	0–85	0–85	
Furfural		0. 05–85	0–85	0–85	0–85		
Isopropanol	90–98	0–98	0. 003–98	0–95		0–90	
Mercury				8–90	25–90		2–50
Methyl acetate		5–100	0–100	0–100	0–100	0–100	
Methyl salicylate			3–100	0–100	0–100	0–100	
o–Nitrophenol			1–95	0–95			
p–Nitrophenol			80–95	98–98		100–98	
Oil of apricot kernels			50–65	65–65	50–65		
Oil of cassia			0. 5–50	0–99			
Oil of cloves		92–99	95–99				
Oil of cedar, light			75–80		65–85		0. 2–75
Oil of *Eucalyptus globulus*		4–85	0–80				
Oil of lemon		25–90	10–95	5–95	0. 1–90		
Oil of lemon, distilled				6–75	0. 5–75	2–60	0–75
Oil of lemon, pressed				2–75	0. 1–75	0–68	
Oil of *Monarda punctata*	3–90	0. 4–90	0–90	0–99			
Oil of peppermint			7–95	0. 3–94	0–50		
Oil of sassafras, natural			1–80	0. 5–85	0–75		
Oil of sassafras, artificial			0–80	0–75			
Oil of thyme	0–90	0. 5–90	0–90	0–90			
Oil of American wormseed			0. 4–80	0–75			
Phloroglucinol				100–98		100–98	
Resorcinol				100–98		98–98	
Safrol			2–80	4–85	0–75		
Salicylaldehyde		0–55	0–95	0–95			

[1] The first number in each column denotes the percentage of treated and the second number the percentage of control (nontreated) conidia germinating.

Trials with fruits were disappointing because all substances that were toxic to the conidia of the fungus imparted to the fruit a flavor and an odor that lessened or destroyed its market value. Many substances such as acetic acid blackened the fruit in a few minutes, and most of them injured it to some extent. These injuries masked the results in rot control, but apparently the rot was checked.

The problem of finding a successful fumigant is a difficult one. It is necessary that the fumigant be toxic to the fungus, noninjurious to peaches, odorless, tasteless, noninflammable, nontoxic to man, and relatively cheap.

SUMMARY

The history of the brown-rot disease in the United States reveals that while the disease was not recognized generally as such until after 1880, it had been reported at least a century ago. Its fungous origin was clearly understood by a number of early investigators.

In the more humid sections of the United States the disease takes a heavy toll, amounting, in years favorable to its development, to as much as $5,000,000. Some fruit is lost every year, and losses occur while the fruit is in the orchard, in transit, in the markets, and in the possession of the consumer.

Practically all the commercial varieties of the peach show some resistance to brown rot, but it is impossible to evaluate this resistance definitely on account of differences in periods of ripening under varying environmental conditions. The early maturing varieties seem to be more susceptible to blossom blight than those ripening later, but here again it is difficult to eliminate the environmental factor and make an adequate comparison of varietal resistance. It is true, however, that the present-day varieties are more resistant than the older and often better-flavored varieties and have supplanted them largely for this reason.

The taxonomic position of the common brown-rot fungus is reviewed in detail, and the writers reaffirm their previous position that the name *Sclerotinia fructicola* (Wint.) Rehm is the name that should be applied to the fungus.

Sclerotinia fructicola occurs in the United States, Canada, Australia, and New Zealand.

The morphology of the fungus is described, and details of the structure and characters of the apothecia, asci, paraphyses, ascospores, conidia, microconidia, and germ tubes are given. Statements by previous investigators concerning the morphology of the fungus are reviewed.

The work of previous investigators on the occurrence of strains or physiologic forms, homothallism, growth on artificial media, enzyme production, and the function of vitamins in the metabolism of the brown-rot fungus are reviewed, and results of experiments and studies made by the writers are reported.

The optimum temperature for the growth of the fungus lies between 75° and 80° F. Temperatures over 90° are distinctly unfavorable to its growth. Although the conidia will germinate at 32°, the fungus grows slowly at low temperatures.

The fungus grows best on an acid medium, but the point at which increased acidity stopped growth was found to depend not only on the hydrogen-ion concentration but also on the acid used. Thus,

one investigator found the limit for growth was pH 1.85 with sulphuric acid, pH 2.20 with phosphoric acid, pH 3.87 with formic acid, pH 4.45 with acetic acid, pH 4.5 with butyric acid, and slightly above pH 4.64 with salicylic acid. Apothecial production is also influenced by the hydrogen-ion concentration, with a maximum production of apothecia at pH 2.5.

A few ascospores were found to be viable five weeks after their discharge. The conidia produced naturally in the orchard are able to survive winter temperatures. Conidia produced during the summer are shorter lived than those produced in the late fall. High temperatures lessen the viability of conidia. No information is available concerning the longevity of the microconidia, since they have failed to germinate.

Rain, wind, birds, insects, and man are factors in the dissemination of the fungus, and of these the writers consider wind as the most important. Man can be an important agency in the spread of the disease on the harvested fruit through improper methods of picking and handling the fruit.

A list of host plants known to be susceptible is given in the text, but the list is not considered complete, since there has been no attempt to make a survey of these plants and because of the confusion that has existed regarding the classification of the fungus.

The fungus survives the winter in mummied fruits and in cankers. From these the fungus is propagated in early spring (1) through the production of conidia on mummies left on the trees, (2) through the production of apothecia and ascospores from those mummies that fall to the ground and become partly buried, and (3) in some sections through the production of conidia on twig cankers caused by the fungus. These three infection sources are discussed in detail. The overwintering on twig cankers is considered to be of less importance, except in certain limited sections, than the production of apothecia from partly buried mummies or the formation of conidia on mummies hanging on the trees.

The period of apothecial production from mummied fruits and the blossoming of peach trees have been found to coincide very closely over a period of years. Apothecia may be produced from mummies or fragments of mummies for many years, but the number of mummies that produce apothecia and the number of apothecia produced slowly diminish annually. Completely buried mummies tend to disintegrate more rapidly than those only partly buried.

Ascospores are discharged by pressure developed within the ascus and are shot for varying distances above the surface of the apothecium. Air currents undoubtedly play a large part in carrying away the discharged ascospores.

The blossom-blight, fruit-rot, mummy, canker, and twig-blight phases of the disease are considered in detail. Infection of the floral parts either by ascospores or by conidia may cause a reduction in the size of the crop, but the principal danger to the crop is that the infected blossoms may act as centers for the propagation of conidia which later infect the fruit as it matures.

Infection of the fruit may take place through the uninjured epidermis (i. e., by way of the stomata and hair sockets), but in the orchard the great majority of infections follow punctures of

the fruit made by the plum curculio, *Conotrachelus nenuphar* Hbst. Infection through wounds made by the oriental fruit moth, *Grapholitha molesta* Busck, is also quite common.

Twig canker formation may result from the infection of either the blossoms or the fruits. The fungus may remain alive over the winter in twig cankers, but in central Georgia and northern Virginia the writers have observed conidial production from cankers the following spring in only one instance. More profuse production of conidia on overwintered cankers has been observed by investigators in certain other sections.

Peach leaves, particularly those injured by the leaf-curl fungus, *Exoascus deformans* (Berk.) Fckl., occasionally may be infected by the brown-rot fungus and become infection sources.

Field experiments for the control of brown rot have demonstrated that the control of the plum curculio and the control of brown rot are closely correlated because control of the former prevents punctures of the epidermis through which brown-rot infections may take place. The curculio is well fitted to disseminate the brown-rot fungus, since its body is clothed with short bristles to which brown-rot spores readily adhere when the insect comes in contact with a rotted peach. Observations of entomologists indicate that the plum curculio feeds exclusively on sound fruit, so that direct dissemination of the fungus would largely be the result of accidental contact with a rotted peach. Therefore, in spite of the fact that the plum curculio is equipped by nature to disseminate brown-rot spores, its main importance in connection with brown rot is that it makes holes in the epidermis of the fruit through which the germ tubes of spores gain entrance to the inner tissues.

The fungus is intercellular in the tissues of the floral parts and of the fruit. The writers have been unable to demonstrate the mycelium of the fungus between the cells in the tissues of the twigs, but other investigators have observed mycelium in cavities caused by the destruction of the cells of the cortical parenchyma. The changes produced by the entrance of the fungus into the twig tissues are discussed in detail. The regeneration of tissues to cover the necrotic region presents no striking novelties and is illustrated by a series of photomicrographs.

The control of the brown-rot disease on the fruit depends largely upon an adequate and timely use of fungicides, coupled with proper measures for the control of the plum curculio. Spraying in central Georgia just as the blossoms were opening has checked blossom blight, but not enough to warrant recommendation for that section. Sanitary measures, such as the removal of diseased fruits, the plowing under of mummies, the pruning out of twig cankers, and the elimination of extra sources of infectious material, such as wild plum and seedling peach trees, which harbor the disease, are all of importance in a general control program. Because of the physical impossibility of completely eliminating all infectious material, these measures alone can not be relied upon to give adequate control of the disease. Sanitary measures when practiced as a community project and supplemented by a proper spray program are of assistance in controlling the disease, but the importance of a proper spray program can not be overemphasized.

Experiments with certain materials that give off vapors toxic to the brown-rot fungus are reported. Unfortunately, all the materials experimented with that were toxic to the fungus also imparted to the fruit a flavor or an odor that lessened or destroyed its market value. A successful fumigant must be toxic to the fungus, noninjurious to the fruit, odorless, tasteless, noninflammable, nontoxic to man, and of relatively low cost. A material embodying all these characteristics has not been found.

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U. S. GOVERNMENT PRINTING OFFICE: 1932

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