Station Bulletin 222 March, 1926

Oregon Agricultural College

Experiment Station

European Canker of Pomaceous Fruit Trees

By

S. M. Zeller,

Plant Pathologist

CORVALLIS, OREGON

BOARD OF REGENTS OF THE OREGON AGRICULTURAL COLLEGE AND EXPERIMENT STATION

Hon. J. K. Weatherford, President	Albany
Hon. E. E. Wilson, Secretary	Corvallis
Hon. B. F. Irvine, Treasurer	Portland
Hon. Walter M. Pierce, Governor	Salem
Hon. Sam A. Kozer, Secretary of State	Salem
Hon. J. A. Churchill, Superintendent of Public Instruction	Salem
Hon. George A. Palmiter, Master of State Grange	Hood River
Hon. Mrs. W. S. Kinney	Astoria
Hon. Sam H. Brown	Gervais
Hon. Harry Bailey	Lakeview
Hon. Geo. M. Cornwall	Portland
Hon. E. B. Aldrich	Pendleton
Hon. Jefferson Myers	Portland

STATION STAFF

W. J. Kerr, D.Sc., LL.D.	President
I. T. Jardine, B.S.	Director
E. T. Reed, B.S., A.B.	Editor
H. P. Barss, A.B., S.M.	Plant Pathologist
F. D. Bailey, M.S.	Asst. Pathologist, Insecticide and Fungicide Bd., U. S. Dept. of Agri.
B. B. Bayles	Junior Agronomist, Office of Cer. Inves., U. S. Dept. of Agri.
R. S. Besse, M.S.	Associate in Farm Management
P. M. Brandt, B.S., A.M.	Dairy Husbandman
A. G. Bouquet, B.S.	Horticulturist (Vegetable Gardening)
E. N. Bressman, B.S.	Associate Agronomist
G. G. Brown, B.S.	Horticulturist, Hood River Br. Exp. Station, Hood River
W. S. Brown, A.B., M.S.	Horticulturist in Charge
D. E. Bullis, B.S.	Assistant Chemist
A. S. Burrier, M.S.	Assistant in Farm Management
Leroy Childs, A.B.	Supt. Hood River Branch Exp. Station, Hood River
G. V. Copson, M.S.	Bacteriologist
H. K. Dean, B.S.	Supt. Umatilla Branch Exp. Station, Hermiston
C. R. Donham, D.V.M.	Assistant Veterinarian
Theo. P. Dykstra, M.S.	Assistant Plant Pathologist, U. S. Dept. of Agri.
E. M. Edwards, B.S.	Asst. Animal Husbandman, East Ore. Br. Exp. Sta., Union
A. E. Engbretson, B.S.	Supt. John Jacob Astor Br. Exp. Station, Astoria
L. N. Goodding, B.A., B.S.	Jr. Plant Pathologist, U. S. Dept. of Agri.
W. V. Halversen, Ph.D.	Associate Bacteriologist
H. Hartman, M.S.	Associate Horticulturist (Pomology)
E. M. Harvey, Ph.D.	Horticulturist (Physiology)
Bertha C. Hite, B.A.	Scientific Assistant Seed Lab., U. S. Dept. of Agri. (Seed Analyst)
R. E. Hutchinson, B.S.	Asst. to Supt. of Harney Valley Branch Exp. Station, Burns
G. R. Hyslop, B. S.	Agronomist
W. T. Johnson, D.V.M.	Poultry Pathologist
I. R. Jones, Ph.D.	Associate Dairy Husbandman
J. S. Jones, M.S.	Chemist
G. W. Kable, M.S.	Agricultural Engineer
F. L. Knowlton, B.S.	Poultry Husbandman
A. G. Lunn, B.S.	Poultry Husbandman in Charge
M. B. McKay, M.S.	Plant Pathologist
H. G. Miller, Ph.D.	Chemist
G. A. Mitchell, B.S.	Asst. to Supt. of Sherman County Branch Exp. Station, Moro
Don C. Mote, M.S.	Entomologist
O. M. Nelson, B.S.	Animal Husbandman
R. K. Norris, B.S.	Assistant to Supt. of Southern Oregon Branch Exp. Station, Talent
A. W. Oliver, B.S.	Assistant Animal Husbandman
E. L. Potter, M.S.	Animal Husbandman
W. L. Powers, M.S.	Chief, Department of Soils
F. C. Reimer, M.S.	Supt. Southern Oregon Br. Exp. Station, Talent
R. H. Robinson, A.B., M.S.	Chemist
C. C. Ruth, M.S.	Associate Agronomist
C. V. Ruzek, B.S.	Associate in Soils (Fertility)
H. A. Schoth, M.S.	Asst. Agronomist, Forage Crops Investigation, U. S. Dept. of Agri.
C. E. Schuster, M.S.	Associate Horticulturist (Pomology)
H. D. Scudder, B.S.	Chief in Farm Management
H. E. Selby, B.S.	Associate in Farm Management
O. Shattuck, M.S.	Supt. Harney Valley Branch Exp. Station, Burns
J. N. Shaw, D.V.M.	Assistant Veterinarian
J. E. Simmons, M.S.	Assistant Bacteriologist
B. T. Simms, D.V.M.	Veterinarian
D. E. Stephens, B.S.	Supt. Sherman County Br. Exp. Station, Moro
R. E. Stephenson, Ph.D.	Associate Soils Specialist
B. G. Thompson, M.S.	Assistant Entomologist
E. F. Torgerson, B.S.	Assistant in Soils (Soil Survey)
E. H. Wiegand, B.S.	Horticulturist (Horticultural Products)
Joseph Wilcox, B.S.	Assistant in Entomology
Maud Wilson, B.S.	Home Economist
Robt. Withycombe, B.S.	Supt. Eastern Ore. Branch Exp. Station, Union
W. W. Yates, B.S.	Assistant Chemist
S. M. Zeller, Ph.D.	Plant Pathologist

CONTENTS

CONTENTS (Continued)

European Canker of Pomaceous Fruit Trees

By

S. M. Zeller*

The European canker of pomaceous fruit trees was not recorded in America earlier than 1899 when Paddock[33] reported its occurrence in Nova Scotia and New York. Ten years later it was mentioned[8] as a disease of apple in New Hampshire and soon in other states until now it is known to be widely distributed throughout the northern United States and Canada from the Atlantic to the Pacific. In Europe it is said to constitute the most serious of all diseases of fruits and it has become of noticeable importance in the Northwest coast states.

HISTORY OF THE DISEASE

The early literature pertaining to this disease appeared in European publications under the general term "canker." The canker as it was known for several decades prior to 1865, when its causal organism was described as *Nectria ditissima,* was a forest tree disease, although often observed as a canker of orchard trees. The extent of the early writings on this subject indicates the wide distribution of the disease in Europe and the wide range of host trees embodied under the early inclusive conception of the causal organism. Today it is believed that these early workers were dealing with at least two, perhaps three, distinct fungi. In 1866 Willkomm[45] showed that the beech canker was caused by an imperfect fungus known at that time as *Fusidium candidum* Link. Goethe,[18] working from 1877 to 1904, showed that the conidia from the Nectria causing canker of apple would infect the copper beech by cross-inoculation. He also applied conidia and ascospores of *Nectria ditissima* to uninjured twigs which were cut from the trees and left in a moderately moist atmosphere without their cut ends in water. Under such circumstances infections had their incipiency in lenticels. At the same time Hartig (1877-1889), was observing cankers due to *N. ditissima* on many hosts, such as beech, oak, hazel, ash, hornbean, alder, maple, lime, dogwood, wild cherry, and apple. He was able to infect young leaves with conidia and ascospores and also noticed that the usual infections in the bark are preceded by injury due to insects, crotch cracking, the bruises caused by hailstones, or more often frost or freeze injury. It was Aderhold,[1] however, who proved conclusively the parasitism of the organism on apple bark. This work was later verified experimentally by Wollenweber[55] and now has been confirmed by many workers. Other European workers were interested in the taxonomy, morphology, and physiology of the causal organism, the work of Lapine,[27] Münch,[31] Weese,[43] and others being noteworthy. More recently, since Bresadola[7] definitely described the canker on orchard trees as *Nectria galligena* Bres., Wiltshire, Cayley,[10] and Ferdinandsen[16] have made some noteworthy observations.

*The sincere thanks of the writer are extended to Professor H. P. Barss for helpful suggestions and criticisms in the preparation of this manuscript.

Wiltshire[48] has described in detail apple stem infections through leaf scars. Apparently entrance is obtained through small cracks in these scars immediately after leaf-fall or in the spring when the neighboring buds are swelling. Again, he[49] has discovered that the canker-producing organism infects through wounds first produced by the scab organism *(Venturia inequalis)*. Cayley[10] has followed the complicated life-history of *Nectria galligena* through its morphological development in order to learn the strategic spore-producing periods for the application of fungicides. Ferdinandsen[16] described for the first time the rot of pear and apple fruits caused by this fungus.

In America the canker on apple was first reported by Paddock[33] in 1900. Specimens sent by Paddock from Nova Scotia and New York were identified

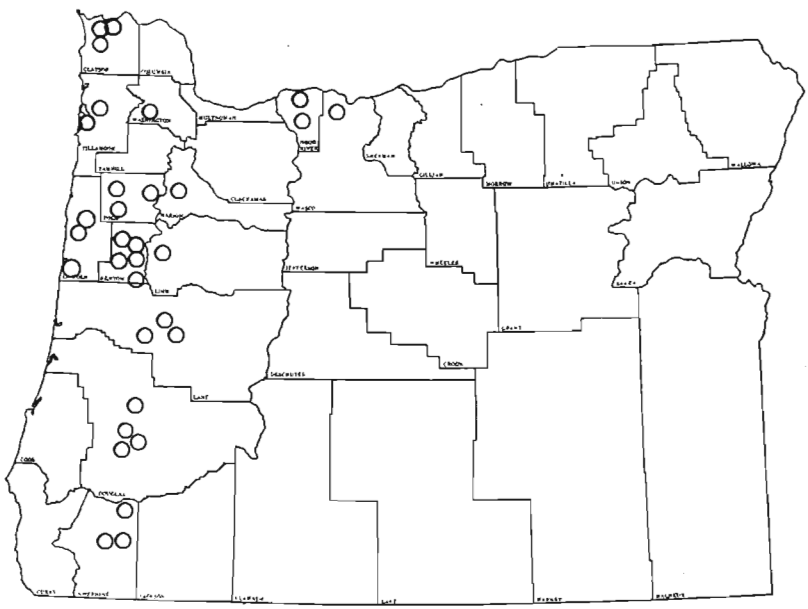

Fig. 1. Map of Oregon showing the stations (O) from which European Canker has been reported.

by Hartig as caused by *Nectria ditissima*. In 1909 Brooks[8] illustrated and described the canker in New Hampshire, and since then reports of the canker on apple have been noted in various parts of the United States and Canada.

In the early part of the year 1918, H. P. Barss identified specimens on apple, sent in from Marion county, Oregon. This was the first report of the canker from the Pacific Coast States, although it is now known to have been observed in Douglas county, Oregon, as an unidentified, but serious, canker of the Howell pear since 1915. Since 1920 when the writer first began to make observations of the disease in Oregon, two short reports of its occurrence have been published.[57, 59] Fig. 1 shows the stations in Oregon from which European canker has been reported.

PREVALENCE OF EUROPEAN CANKER

GEOGRAPHIC DISTRIBUTION

This canker has been reported from all fruit growing districts of northern Europe, New Zealand, and Australia. In America it is most serious in southeastern Canada and in the northeastern and northwestern portions of the United States, being distributed more or less, however, across southern Canada and the northern United States wherever pomaceous fruits are grown to any extent.

ECONOMIC IMPORTANCE

In Europe this canker is the most destructive and most dangerous of all fruit diseases because of its prevalence and because it attacks the very life of the trees. Furthermore, the worst ravages come when the trees are in their prime, when thousands of trees are killed because of trunk or scaffold-branch infections. In many European localities certain varieties of apples can no longer be grown, and there even are districts where apple growing has become a real problem because of this canker.

In America its destructiveness has not become so serious as even to approach that reported for Europe, but here the canker is troublesome and is on the increase. Its prevalence is dependent on so many factors that the economic losses due to canker cannot be estimated definitely; they vary considerably in different localities. The losses in Oregon have been mostly in young pear trees 3 to 15 years of age. For instance, in one orchard of 408 trees of 3-year-old Surprise stock, 135 trees or nearly 34 percent of the trees were trunk-infected; in another case 42 percent of the trees in a 140-acre orchard of the same variety were cankered in the trunks or scaffold branches.

The prevalence of the European canker disease may be considered in its relation to several factors which may be conducive to, or limiting, the extent of infection. These factors, which will be discussed below, are the prevalence of the disease in relation to (1) climatic factors, (2) lay of the land and the kind of soil, (3) age and vigor of the host tree, (4) infection courts, (5) the physiology and morphology of the host, and (6) susceptible varieties of orchard trees and neighboring wild hosts.

RELATION TO CLIMATIC FACTORS

The climatic conditions which favor canker infections may be considered jointly under (1) temperature, (2) rainfall, and (3) atmospheric humidity. In America the nearest to an optimum of these factors for canker infection exists in the rain belt of the northern Pacific Slope west of the Cascade Range. Canker under the conditions which prevail here during the rainy season perhaps reaches more destructive proportions than in the northeastern United States. During the late fall, winter, and early spring months our mild temperatures are conducive to the growth and sporulation of the European canker organism and the intermittent rainfall and high atmospheric humidity keep the surfaces of bark or woody wounds moist enough for spore germination. It is under these moist conditions that the European canker develops best after infection, contrary to the activity of wood-decaying organisms, or other wood-inhabiting fungi which go deeper into the trunks of trees. The attack of wood by these deeper-growing fungi is fa-

vored by a sufficient air (oxygen) supply in proportion to the moisture present in the wood, as suggested by Münch[31] and the writer.[56] An example of this type of canker invasion is the blister-canker of apple which Gloyer[17] points out as epidemic after drought periods.

RELATION TO LAY OF LAND AND KIND OF SOIL

Cotton[11] has observed that the location of an orchard and the type of soil are factors influencing the susceptibility of trees to canker. He says: "Canker is always worse on a low site and a clay subsoil, so much so that it is impossible in such positions to grow certain varieties of apples successfully. The effect of damp, heavy soil is to cause rank growth which is probably more readily injured by the canker fungus and is more liable to frost injury." These observations are substantiated by the conditions under which European canker has been seen in Western Oregon. Orchards grown on hillsides in lighter soils have not been subject to canker as much as those on heavier soils in lower places where air drainage is poor. The only exceptions to this are cases of extremely susceptible varieties of pear trees on slopes. In cases where soils are shallow and underlaid with hard-pan or impervious rock, or where the water-table in winter is high due to poor drainage, die-back results, since with the first indication of dry weather the reduced root system puts the tree under extreme drouth conditions. The following seasons such trees send up vigorously growing shoots which during a few subsequent years are very subject to canker infection.

In an orchard of Howell pears observed in 1920 which is planted partly on a gradual slope and partly on the flat river-bottom land below, the frequency of lowland infection over upland is brought out strikingly. On the slope but 4 percent of the trees showed small cankers, while on the flat lowland below, the cankers were mostly large trunk and crotch cankers affecting 72 percent of the trees. This cannot be laid to any one of the three factors, (1) lay of land, (2) possibility of winter injury in the lower land, or (3) the better river-bottom land producing vigorous growth; but it is an excellent example of the combined effect of the three factors. In the same orchard, in the spring of 1921, one block of Anjou pears high up on a well-drained, open soil had 2 percent of the trees with canker, while in a block of the same variety on much heavier soil, which was low and flat but well-drained, 31 percent of the trees had some canker, many being severely cankered.

Orchards in sod are so few in Oregon that few data have been secured on European canker under these conditions. Some canker has been found in abandoned orchards after severe winters. The organism in these cases was not active as a parasite but had obtained a foothold in the dead bark over large cankers produced by winter injury. So little vigorous wood is found in such orchards that canker in an active state is scarce.

RELATION TO AGE AND VIGOR OF THE HOST TREE

As has been suggested the greatest losses due to European canker are suffered through the destruction of nursery trees and transplants up to about 12 to 15 years of age. This is particularly true of pear stock which is damaged to a much greater degree than apple under Oregon conditions (Fig. 2). Older trees are attacked in their smaller branches, but in such cases the permanent loss of the tree-scaffold need not be sustained. Thus, the vigor of

youthful trees is a handicap in combating European canker, while on the other hand trees in old age or trees which grow slowly from various causes, are as a rule less susceptible.

Fig. 2. An eight-year-old Anjou pear tree with extensive cankers on trunk and scaffold branches. These cankers followed winter injury.

RELATION TO UNPROTECTED INFECTION COURTS

The European canker fungus gains entrance to a tree by means of unprotected wounds and small injuries or through such natural structures of

the host as leaf scars. Apart from such wounds the myriads of spores which alight upon the bark are unable to infect a tree. The three chief means of infection seem to be through leaf scars, winter injury, and pruning cuts.

Leaf-scar infection. Wiltshire[48] has described in detail the infection of apple stems by this canker fungus through the leaf scars. The fungus enters through small cracks which appear in the leaf scar tissues in the autumn immediately following leaf-fall, and in the spring when the buds are swelling. This type of infection is responsible for a large percentage of the canker in England. In that country the infection of the shoots takes place during the year succeeding their growth; for instance, the wood produced in 1920 was infected in the spring of 1921.

In Western Oregon the European canker on pear is caused in large measure by leaf-scar infections. The writer has observed many suckers, or water sprouts, of Anjou, Howell, Surprise, and Bosc pear where all the buds were cankered except a very few at the tip. To illustrate, in the winter of 1920-21, on shoots of Anjou pear less than 1½ inches in diameter at the base and approximately 4 feet long, 18 to 23 buds were found with cankers which had started through the leaf scars. The only healthy buds were near the tips. Many such examples may be found in orchards where canker infections are frequent. Trees which hold their leaves until late in the fall often escape this type of infection, but if early frosts are at all damaging and mild weather follows during the winter such trees may become infected through winter-injured buds, spurs, and bark.

Infection through winter injury. Varieties of trees which are subject to winter injury may also be said to be susceptible to European canker, for trees which show bark devitalized by various forms of winter injury are sure to be infected if exposed to the disease. Some workers have maintained that all varieties are equally susceptible to canker if their chance of infection is the same, and in European countries cankerous varieties have always been associated with their proclivities to winter injury. In Oregon our most severe instances of European canker infection have followed occasions of greater low temperatures than those to which the trees are usually subjected. Thin-barked varieties of pears which are subject to winter injury under Oregon conditions are the most subject to canker. Following severe winter injury, *Nectria galligena*, among other fungi, is a saprophytic on the resulting dead or devitalized bark or twigs. Fig. 3 shows a canker on a winter-injured branch of Anjou pear. Such cankers often bleed, as do pruning cuts after the effects of low temperatures on the wood.

Infection through cankers produced by other fungi. Many instances of European canker infection through current year infections of the apple-tree anthracnose *(Neofabraea malicorticis)* have been observed. These have all been instances where the anthracnose had attacked Anjou or Bosc pears. Fig. 4 shows the appearance of this type of infection. In small anthracnose cankers attacked by European canker the anthracnose organism advances no further but the European canker enlarges, usually entirely surrounding the original canker. The bark of the anthracnose infection finally falls away, exposing a wood area in the central part of the canker.

Wiltshire[60] has shown by detailed study that canker in England enters through lesions in the bark which were produced earlier by infections of apple scab *(Venturia inaequalis* (Cooke) Winter*)*. The scab fungus infects the shoots of susceptible varieties of the apple during the autumn and winter

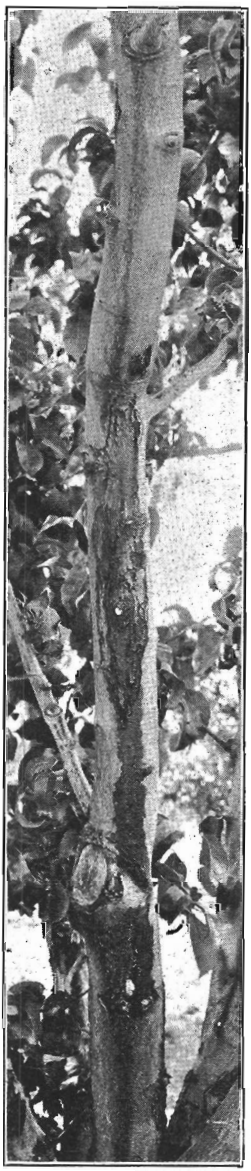

Fig. 3. Cankered branch of Anjou pear showing the "bleeding" which often follows winter injury of the wood beneath the canker.

following their growth, the first infections appearing before the leaves fall, but soon the corky calluses of the healthy bark exclude the scab organism, unless they are subsequently infected by *Nectria galligena,* in which event they are surrounded by a narrow, sunken, darkened border. In vigorous trees where these infections are merely superficial, the formation of callus may completely exclude the canker organism; but in cases where the infection reaches the wood typical cankers are formed. In America scab infections are rarely found on apple twigs, but pear-scab *(Venturia pyrina)* is common on twigs in pear orchards which are abandoned or do not have the best of care.

In Oregon European canker sometimes obtains entrance through lesions produced by a species of *Monilia* and by a strain of *Botrytis cinerea* Pers. on pear twigs. Many instances of this have been found on Winter Nelis twigs in an abandoned orchard where these diseases were plentiful after the severe freeze of December, 1919. During the spring of 1920 many blossom spurs were infected with the *Monilia.* By the next fall and winter many of the bases of Monilia-infected spurs were surrounded with small cankers due to *Nectria galligena,* which had spread into the healthy callus bark and wood beginning to form around the Monilia cankers. The development of these infections is very similar to those which gain entrance through scab infections. Of course, the practical orchardist who is consistent with his spray program, will not suffer from European canker which gains entrance

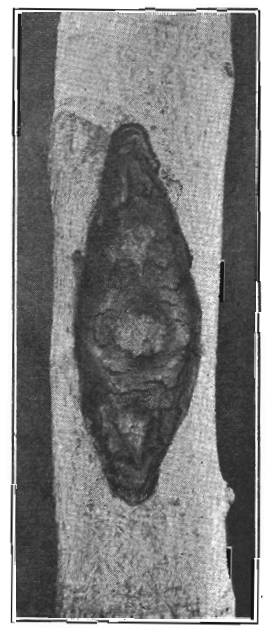

Fig. 4. European canker having its origin in a small anthracnose canker on Anjou pear.

through the wounds produced by anthracnose, scab, or Monilia, for none are found in the well-sprayed orchard.

Infection through crotch, frost, and other cracks in the bark. Hartig,[21] Münch, [31] Lapine,[27] Goethe,[18] Cotton[11] and other European workers all mention the occurrence of cankers arising from infections through cracks at the crotches of branches (Fig. 5). Frost and other cracks in the bark are open to infection until they callus over. Spores lodge and germinate in such courts, and if the way is clear to broken cortical tissues of the bark, infections are very likely to occur.

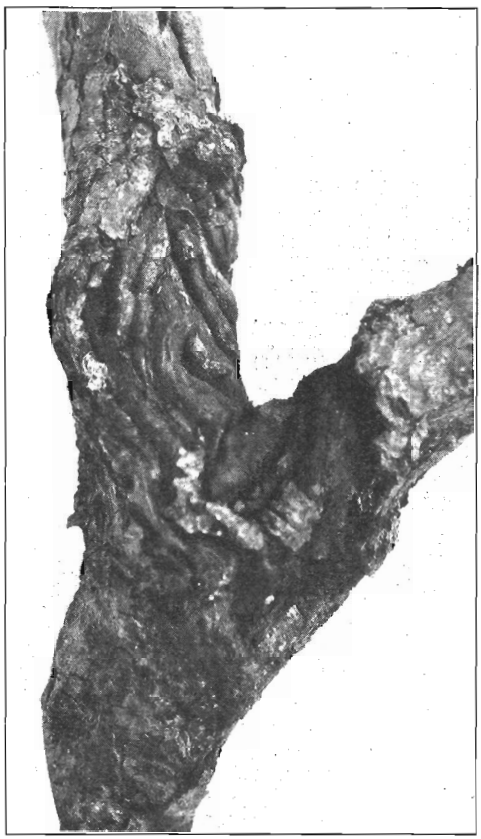

Fig. 5. An open crotch canker of apple.

Infection through insect wounds. In England it is reported by Cotton[11] that, although the canker is due to *Nectria galligena,* the injury caused by woolly aphis is very similar in appearance, and in fact the two pests are not infrequently found together and aid each other in the damage they cause. Further, "probably one of the most frequent methods by which the fungus gains admittance is through injuries caused by woolly aphis. The soft, swollen tissue produced by the aphis is very apt to become cracked during the winter, and when the aphis return the following season to the wounded areas, *Nectria* spores are attached to their bodies and introduced into the cracks with the aphis." Wiltshire[47] says that the European canker fungus grows very vigorously in the gall tissue produced by the aphis, which becomes very soft and disorganized. It appears that infection takes place in the autumn through the wood elements exposed by the splitting of the aphis gall. Eckstein[14] reports that wounds produced by the beech louse are followed by the canker fungus, *Nectria ditissima,* which finally kills the tree.

The writer has often found woolly aphis in the crevices and under the dead bark of cankers produced by *Nectria galligena,* on both apple and pear,

but no particular sequence of the injuries produced by the pests has been determined, although there is much incriminating evidence that these insects disseminate the spores from place to place on an individual tree.

Infection through pruning cuts or other wood exposures. Pruning cuts or other wounds exposing the wood of the tree are a source of infection unless they are treated with a wound dressing or thoroughly drenched with a fungicidal spray or perhaps dust. Such wounds do not account for as high percentage of the infections as do leaf scars and winter injured bark, but many small pruning cuts on pear varieties have been found to be courts of infection (Figs. 6 and 7). In the inoculation tests reported below, the inoculated pruning cuts yielded fully as many cankers as the leaf scars, but under natural conditions this does not seem to be true. This is perhaps due to the fact that the inoculations on prun-

Fig. 6. Infection through a pruning cut on pear.

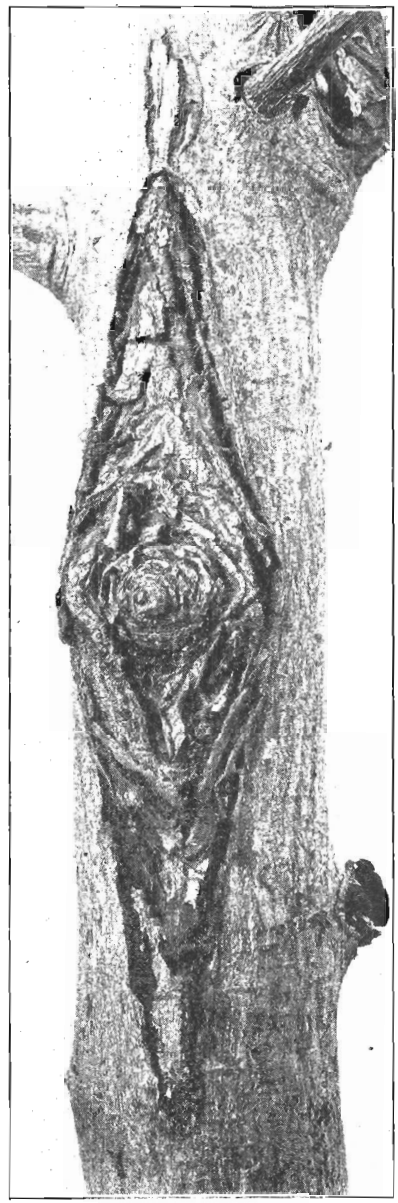

Fig. 7. Infection through a broken pear twig.

ing cuts were made on freshly cut wood which had not oxidized or weathered, where under natural conditions the chance of spore infection under such favorable conditions is greatly reduced.

Bruised bark as the result of the impact of hailstones has been reported by European workers as infection courts for this canker organism.

RELATION TO PHYSIOLOGY AND MORPHOLOGY OF THE HOST TREE

The physiology of the tree is intimately tied up (1) with climatic factors, temperature, rainfall, humidity, and (2) with soil texture, tilth, fertility, etc. For this reason these paragraphs will in some respects repeat ideas stated in preceding paragraphs.

The susceptibility of vigorously growing shoots has been mentioned above. It is very important, on the other hand, to maintain the well balanced vigor of the whole tree, inasmuch as any tree or shrub which is suffering devitalization from the lack of overabundance of any factor is usually more subject to the attacks of parasitic fungi in mild cases and even to the devastation of saprophytic forms in more severe cases of devitalization. The greatest damage done by European canker is exemplified by its activity on trees which lack the general vigor they would possess provided they had been subject to the best of cultivation in order to conserve the moisture during summer drouth and provided they had been planted in well-drained land.

Under Oregon conditions the conservation of moisture during the dry summer by adequate cultivation is extremely essential to proper growth vigor, but without the early cessation of tillage trees may be carried into late fall too active to withstand the early low temperatures. *Early dormancy is the greatest factor in the prevention of winter injury through which European canker becomes very destructive.* Without regard to this fact, and having in mind the possibility of infection through leaf scars, Wiltshire[48] says that "vigorous trees usually hold their leaves longer than weakly ones and any treatment therefore which would tend to increase the vigor of the trees may, by delaying autumnal defoliation, reduce the risk of infection."

Poor drainage, underlying rock, or hard-pans, which keep the water-table too near the surface of the ground, are conducive to another abnormal physiological condition in which trees are rendered susceptible to canker infection. The writer has determined the pH of the soil in contact with the roots of trees whose roots have been flooded with water below the high water-table and found the acidity to range from pH 6.4 up to 4.8 (average pH 5.6), while the soil in contact with the roots of normal trees which were better drained had a pH value ranging from 7.4 up to 5.6 (average pH 6.2). In the roots which have been flooded with water, respiration must proceed without the free oxygen of the air. Carbon dioxide is commonly an end product of this type of respiration, together with alcohols, acetic and lactic acids, free H-ions, and other products. Since no free oxygen is available the decomposition resulting in carbon dioxide and the other products mentioned is obviously brought about by the rearrangement of the atomic groups in the organic molecules, or an anaerobic intramolecular respiration. With a deficiency of oxygen in the soil there is also an increasing amount of acids in the roots and lower stems. As the cells die under these conditions, the tissues darken and the acids are liberated into the surrounding soils.

Harris[20] has shown that under Western Oregon conditions the roots of orchard trees start their growth in the fall after the soil is well moistened by the rains and this growth continues until the temperatures are lowered to near the freezing point, or until they are submersed by the rising water-table. When root growth is stopped by low temperatures the tips will renew their growing processes, but when growth is stopped by submergence for any considerable length of time, root growth is not resumed by the affected roots.

The trees, however, do not die, unless there is complete submergence of the root system for some time, but there is a natural pruning of the tops, in the form of dying spurs, twigs, and even branches to equalize the dying of the roots. This brings about the condition which has been commonly called "die-back." This condition becomes more severe when followed by a dry summer season which causes a struggle for life in the roots which were forced to grow in the surface soils above a high water-table.

Trees suffering with die-back are very susceptible to canker and other weaker fungous parasites because of the low vigor of the spurs, branches, and even the trunks. Such trees frequently throw out new growth to replenish the tops after the root system has become reestablished. The following fall and winter these more vigorous shoots are liable to canker.

Another factor which has been mentioned by growers and general observers of canker in European countries, as a cause of susceptibility to the disease, is unbalanced nutrition whether produced by deficiencies in the soil or by overbearing. The writer has made no observations contributory to this view.

The only morphological characters of trees, other than susceptible leaf scars, which are conducive to infection, bear a rather indirect relation to prevalence of the disease. One of these which is referred to is the thinness of the bark in certain varieties of orchard trees. Such trees certainly are the most susceptible, but whether this is due to this character directly, or to the fact that many of such varieties are subject to winter injury, is an open question.

RELATION TO SUSCEPTIBLE VARIETIES OF ORCHARD TREES AND NEIGHBORING WILD HOSTS

Besides the above-mentioned factors which influence the susceptibility of trees to canker there is evidence of innate varietal characters which supply the resistant or susceptible tendency. Why one variety is resistant, or another liable to the disease is not known. In England among apple varieties, which are most subject to attack, are mentioned Ribston, Pippin, and Wellington, while among those which are less susceptible are Blenheim, Orange, and Bismark. In Germany susceptible varieties include, among American grown varieties, the Hubbardston and Canada Reinette, while the resistant ones include Boiken and Gravestein.

In Oregon the European canker was originally discovered on the Red Cheek Pippin, but it seems to be most commonly found on Northern Spy and Winter Banana. It has also been observed on Pennock, Bismark, Delicious, Bellflower, Spitzenburg, and Yellow Newtown. The greatest damage from European canker in Oregon is among pear varieties. The Anjou, Bosc, Howell, Surprise, and Old Home pear trees are the most susceptible,

while Bartlett, Winter Nelis, and Clairgeau, although sometimes found with some infection, are usually free from canker.

Nectria galligena has not been found on other host trees native to Western Oregon, although the three closely related fungi, *Nectria ditissima, N. coccinea,* and *N. sanguinea,* have been found on some of the deciduous trees of the forests. The difficulty of identifying these fungi in the field often leads one to suspect the true orchard canker disease on some of the native hosts such as oak, dogwood, maple, willow, etc. In 1911 Weese reported that up to that time *N. galligena* had been identified as a canker-producing fungus on apple, pear, ash, hazel, and black alder *(Rhamnus frangula),* but that a wider search will perhaps yield more hosts.

THE CAUSAL FUNGUS

TAXONOMY

The most generally accepted name for the organism causing the European canker of pomaceous fruit trees is *Nectria galligena* Bres. There are several species belonging to the section *Willkommiotes* Wollenw.[34] of the genus *Nectria* which have often been confused in literature. Of this group, *N. galligena* is the most recent segregate [Bresadola[7]]. The probable synonymy and some of the descriptive mycological literature pertaining to this species should be discussed briefly in this connection.

In 1866 Willkomm gave to the conidial stage of the fungus causing canker of deciduous trees the name *Fusidium candidum* Willk. One year before this the Tulasnes[40] had described *Nectria ditissima* Tul., the name which has been applied to the canker organism until more recent years. Hartig[21] and others had so many times observed that the conidial stage of the fungus described by Willkomm was followed by Tulasnes' *N. ditissima* on cankers of forest trees, that both conditions were considered manifestations of the same fungus. Later it was found that the conidiophores arise from a sporodochium, and Saccardo (1906) recombined the name of the conidial stage, as *Fusarium candidum* (Willk.) Sacc. This name was preoccupied (*F. candidum* Link), and hence Lindau (1909) gave the fungus the new name, *Fusarium Willkommii* Lindau. Thus in the literature of this period up to about 1910 these names have been used, according to the fruiting stage which the worker may have observed.

It was not until Weese[42] in 1911 made a critical study of the difference between *Nectria galligena* Bres. and such species as *N. coccinea* (Pers.) Fries and *N. ditissima* Tul. that the former was seriously considered as the true cause of the canker of pomaceous fruit trees. Weese differentiated *N. galligena* by the structure of the perithecia, by characteristics of the spores, and chiefly by its behavior as a canker producer. He was of the opinion that *Fusarium Willkommii* Lindau could not be the conidial stage of *N. galligena* as Appel and Wollenweber[3] had suggested, but the next year Weese[44] agreed with them, for since his publication in 1911 he had found what he considered to be *Fusarium Willkommii* on the apple canker.

The important contributions by Weese called forth a discussion in which Voges and Wollenweber have been the chief participants. Voges[47] is of the opinion that the work of Weese will lead to much confusion among pathologists and mycologists, since there will always be a question as to the species

of Nectria with which earlier workers experimented. He is inclined to believe that the great variability among closely related segregates of this group of Nectria cannot be satisfactorily separated from *N. ditissima*. He was able by inoculation with mycelium and both ascospores and conidiospores to produce canker on apple bark with what is known to him as *N. ditissima*, but not *N. galligena*, a fungus which he has never found. On the other hand, Wollenweber[54, 55] has identified *Nectria galligena* Bres. with the apple tree canker from both European and American collections of the fungus, and at first considered *Fusarium Willkommii* Lindau as its conidial form. In 1913, however, he called attention to the fact that a conidial form almost identical with that of *N. galligena* was isolated from an apple core and he believed it to be identical with *Fusarium Mali* Allescher. This fungus from the core of the apple when used for artificial inoculation also produced the true apple stem canker, but no fruit rot. Thus, the apple fruit becomes an over-wintering place for the canker-producing organism. In the same paper Wollenweber[54] described the form genus, *Cylindrocarpon* Wollenw. to include those species of *Fusarium* with cylindrical macrospores having broadly-ellipsoid, sometimes one-sided terminal cells (never with a narrowed, long-attenuated point), and with rounded to ellipsoid or flattened base. The conidia are 3 to 5 or even more septate, although in attenuated cultures this may be decreased to few-septate, or even one-celled forms. He also described the Section *Willkommiotes* Wollenw. of the genus *Nectria* Fries to include those species which have a conidial stage conforming to the form-genus, *Cylindrocarpon*. After about ten years' additional study of these species by way of pure culture and direct and cross-inoculation, in 1924 Wollenweber[56] has still continued in his belief that *Fusarium mali* All., which now becomes *Cylindrocarpon mali* (All.) Wollenw., is the conidial stage of *Nectria galligena* and that *Fusarium Willkommii* Lindau and *F. fractum* Sacc. and Cav., which now become *Cylindrocarpon Willkommii* (Lindau) Wr. and *Cyl. fractum* (Sacc. and Cav.) Wr., respectively, are the conidial stages of *Nectria ditissima* Tul. and *N. coccinea* (Pers.) Fr., respectively. Besides these three species of *Nectria*, the section *Willkommiotes*, also includes *Nectria curcurbitula* (Tode) Fr. with the conidial stage, *Cylindrocarpon cylindrioides* Wr., and *N. sanguinea* (Sibth.) Fr. and *N. Jungneri* P. Henn. with unnamed *Cylindrocarpon* species as conidial forms. Pure cultures from conidia of all of these forms, except *N. curcurbitula* have produced mature ascospores. Upon these pure culture studies Wollenweber bases his classification of the section *Willkommiotes* and he has given a chart which contains host characters, and measurements of perithecia, ascospores, and conidia of each species. The results of his inoculations will be mentioned in following paragraphs concerning inoculation work. From the evidence in the literature and from personal studies the writer believes that the cause of the canker of pomaceous fruit trees as it occurs in the Pacific Coast states is *Nectria galligena* Bres. The characteristics of its conidia produced in pure culture agree with those given by Wollenweber for *Cylindrocarpon mali* (All.) Wr. Although several other species of *Cylindrocarpon* have been found on pomaceous hosts in Oregon and Washington, *C. mali* is the most prevalent form in Western Oregon.

ISOLATION AND CULTURAL CHARACTERS

Pure cultures of *Nectria galligena* are readily isolated through the use of spores or mycelium from the canker. Very active and virulent cultures

may be obtained from all these sources, although cultures from spores are most readily obtained. Cultures from ascospores may be obtained by crushing a mature perithecium in a loop of sterile water placed in the bottom of a sterile Petri dish and then transferring by loop to a water blank for dilution, when poured plates of potato glucose agar may be made. Loops of conidiospores may be transferred from sporodochia on the bark directly to water blanks and then plated out. Pure cultures from ascospores may be obtained directly by the discharge of the spores onto inverted agar plates. This method was suggested by Wiltshire,[47] but his method has been simplified as follows: A small piece of bark containing perithecia is soaked in water for a short time until the bark and perithecia have imbibed enough moisture to revive the latter so that the asci will begin spore ejection. The piece of bark is then sponged off between filter papers or tissue toweling and then is placed in the bottom of an inverted lid of a Petri dish so that the perithecia have their ostioles directed towards the inverted plate of nutrient agar, which is now placed over the cover. Ascospores are ejected within an hour. No contaminations have been experienced by this method, which is essentially the same as that described by the writer[56] for wood-destroying fungi.

Although cultures can be obtained from the mycelium in bark tissues, the method is impractical if spores can be obtained. If conidia are not to be found on a canker they usually will appear in a short time providing the branch with canker has one end immersed in water in a damp chamber, partly covering the canker. Sporodochia will soon appear on the portion of the canker above water.

Cultural characteristics. The mycelium of *Nectria galligena* was grown on many different culture media, such as Richard's, Czapek's, Reed's and Dunham's nutrient solutions besides potato decoction alone or supplemented with either mannite, phloridzin, or salicin. A modification of Richard's E nutrient solution proved to be the best liquid medium as determined by the weight of the dry mycelial mat after a growth period of 10 days. To bring out certain distinct morphological characters other media, such as sterile apple twigs, potato plugs, rice, oatmeal, and sweet clover stems *(Melilotus alba)*, were used.

CULTURE MEDIA USED

Potato decoction. The potato decoction was made up in the proportion of 200 g. of potato in 1000 c.c. of water. This was autoclaved at 10 to 15 lbs. pressure for 30 minutes and then the decoction was strained through a rather heavy cloth filter and made up to volume. All of the potato decoction used was made up at one time so that portions used in the various modifications mentioned below are comparable. To each of four 500 c.c. portions was added 2.5 g. glucose, 0.25 g. phloridzin, 0.25 g. salicin, and 2 g. mannite, respectively. Some was used without modification.

To sterile 25 c.c. portions of each of these solutions in Ehrlenmeyer flasks plugged with cotton were added inocula of *Nectria galligena* and *N. sanguinea* which had previously been prepared in the following manner. Poured Petri-dish plates of potato hard agar were inoculated with cultures of the fungi. After mycelial growth had well covered the plates inocula

were prepared by cutting the agar into squares about 5 x 5 mm. by use of a sterile scalpel [see Zeller[56]].

After the mycelium of these fungi had grown on the solutions for ten days the mycelium was caught on a filter paper, dried at 70° C. for three days, and weighed. The weights are recorded in Table I, page 22.

Richard's solution. This solution for fungi was slightly modified so as to conform with Richard's[35] solution and Richard's E solution but contained M/8 glucose instead of 5 percent sugar. Their formulae follow:

Richard's A solution (modified)

KH_2PO_4	5.0 g.
$MgSo_4$	2.5 g.
NH_4NO_3	10.0 g.
$FeSO_4$	Trace
Glucose	23.0 g.
Water	1000 c.c.

Richard's E solution (modified) was made up as in (A) above except 0.5 g. KH_2PO_4 and 4 g. KNO_3 were used as the source of potassium. The growth of *Nectria galligena* and *N. sanguinea* was observed under the same conditions as for potato decoction (see Table I, page 22):

Potato plugs. Slanted potato plugs were prepared in the usual way. A wad of absorbent cotton was placed in the bottom of the test tubes for the potato plugs to rest upon. A sufficient quantity of 1 percent glycerine in water completely to cover the potato was then added, and the tubes were autoclaved and stored until needed. Before inoculation the superfluous glycerine solution was poured off. This method suggested by Cayley[10] provides a medium which will keep moist over a long period and upon which *Nectria galligena* will produce perithecia.

Rice and rolled oats. These two media were prepared in the usual manner except that before steaming they were covered with one percent glycerine in water as were the potato cylinders. Upon these two media *N. galligena* produced perithecia.

Czapek's[12] *solution* and *Dunham's solution* were made up as usual. *Reed's*[34] *solution* was supplemented with small bits of filter paper added as a source of carbon.

PHYSIOLOGY OF *NECTRIA GALLIGENA* AS STUDIED IN CULTURE

Observations on the laboratory work with pure cultures of *Nectria galligena* have given some interesting facts concerning the following factors in growth: (1) active acidity of the substratum upon which the fungus grows, (2) temperature, (3) factors affecting fruiting, and vegetating of the fungus, and (4) sources of carbon in the nutrition of the organism.

Active acidity. In order to test out the optimum hydrion concentration for the growth of *N. galligena* Richard's E solution was prepared as mentioned above with such changes as to produce highly buffered solutions

at pH values ranging from pH 3.4 to pH 6.6. To do this the glucose was reduced to 1 percent, the $MgSO_4$ was reduced to 0.25 g. to allow the free solubility of the phosphates, and the KH_2PO_4 was so modified by adding to 25 c.c. quantities of the nutrient solution such amounts of M/3 K_3PO_4, M/3 KH_2PO_4 and M/3 K_2HPO_4 solutions as to give a total of 31.8 c.c. of solution in each culture flask. This resulted in solutions with the pH values, 3.4, 4.2, 4.6, 5.2, 5.6, 6.2, and 6.6. These seven solutions were made up in duplicate and inoculated with inocula of *N. galligena* prepared as mentioned above. After 12 days incubation on these solutions the mycelial mats were collected on tared filter papers, dried at 70° C. for 4 days and weighed.

From the data secured in this experiment the accompanying growth curve (Fig. 8) was plotted. The optimum acidity for the growth of *N. galligena* is between pH 4.2 and 5.2. This is of greater interest when correlated with the acidity of pear and apple bark. Rose[37] found the H-ion concentration of apple bark to range between pH 4.29 and 5.15. The writer pressed out the sap from succulent bark of apple and pear twigs, and after filtering a clear sap was obtained. The same was done with older bark from larger branches. The acidity of these samples of sap ranged as follows: apple bark pH 4.2 to 5.0 and pear bark pH 4.4 to 4.8. According to these results it is evident that *Nectria galligena* finds in the bark of these orchard trees a medium of optimum acidity for its growth.

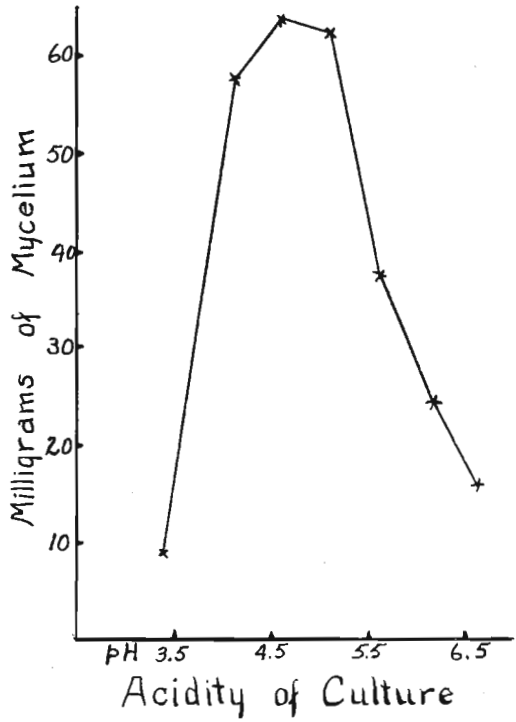

Fig. 8. Curve showing the relation of active acidity to the production of vegetative growth by *Nectria galligena* in culture.

Temperature. The optimum temperature for the growth of the organism has not been definitely determined, but best results with cultures have been obtained when the room or incubators in which the cultures were stored were maintained at a temperature ranging between 18° C. and 24° C.

Growth and fruiting. Tubes of Cayley's glycerinated potato medium described above were drained so as to leave different amounts of the glycerine solution in the test tubes. These were inoculated and growth observations

made. When enough moisture is present to keep the air very humid growth of mycelium is excessive but no fruiting occurs. When the moisture is at a minimum, however, sporodochia are formed in quantity in about 14 to 18 days. With further drying and exposure to light, growth is very much limited, and in the course of 4 to 5 weeks tiny red perithecia are to be found in the sporodochia. When the perithecia are about 40 microns in diameter they are still white, but as they increase in size to 48 to 56 microns they begin to change to a very delicate pink at the ostiole and as they enlarge up to 85 to 90 microns in diameter the distal end has become a dark pink which shades off into a light pink at the base. These perithecia are borne on a hyaline or brownish disk-like entanglement of thick-walled hyphae making up the only stromatic tissue evident in this species. In all cases the macroconidia *(Cylindrocarpon mali)* were produced on the sporodochia preceding the formation of perithecia. There is little indication from pure culture studies that a rest period is necessary to fruiting. Usually, however, when *Nectria galligena* is grown on sweet clover stems or apple twigs as a pure culture medium mycelial growth is limited and fruiting proceeds to the formation of pionnotes and sporodochia, but unless additional sterile water is added no perithecia are formed. This perhaps is due to a lack of moisture rather than the necessity of a rest period before perithecia are formed.

Under natural conditions the sequence of fruiting in *Nectria galligena* is first sporodochial macrospores followed by the ascogenous stage. In pure cultures the ascogenous stage is produced under conditions of comparatively high carbohydrate concentration, while macrospores are produced at concentrations of greater dilution. Since the more solid culture media become more and more dessicated as the fungus grows the food constituents become more concentrated and the sequence of reproductive processes is the same as under natural conditions. Whether this usual sequence could be altered by plantings of the fungus on concentrated media first and by later dilution is questionable. It has not occurred for the writer on relatively dry glycerinated rice, rolled oats, or potato cylinders. In fact, under these conditions no type of sporulation occurred. In the historical work of Falck[15] zygospore formation was induced in *Sporodinia grandis* when the nutrient medium was highly concentrated, while more dilute concentrations favored the development of sporangia. Leonian[28] also observed that the sudden increase of food-concentration is more favorable to pycnidium formation in certain *Sphaeropsidales* than weaker concentrations.

Sources of carbon. As mentioned above in the descriptions of culture media used in the laboratory experiments, representatives of the sugars, starches, alcohols, glucosides, and celluloses were used as sources of carbon in some of the media of more or less definite composition, such as Richard's,[33] Reed's, [34] and Czapek's[12] solutions and potato decoction and Dunham's solution made up following a definite procedure.

The results of the first test of these solutions are given in Table I.

TABLE I. GROWTH OF MYCELIA OF *NECTRIA GALLIGENA* AND *NECTRIA SANGUINEA* ON NUTRIENT SOLUTIONS

Fungus	Weight in milligrams of 10 days' growth of mycelium on following solutions (dried at 70° C. for 3 days)								
	Reed's	Dunham's	Czapek's	Potato decoction	Potato decoction + salicin	Potato decoction + phloridzin	Potato decoction + mannite	Richard's E (modified)	Richard's A (modified)
Nectria galligena	3.7	8.8	22.2	25.1	22.3	30.6	26.7	80.3	7.18
Nectria sanguinea	2.5	7.3	11.0	22.9	28.7	21.0	25.2	76.7	80.1

Since the preliminary test of these various solutions indicated that Richard's E solution was the best for the mycelial growth and that since phloridzin accelerated the growth of *N. galligena* but retarded the growth of *N. sanguinea* and salicin had brought about the reverse influence on growth, further experiments were planned using Richard's E solution and potato decoction.

For this experiment Richard's E solution was made up, as mentioned above, so as to have an H-ion concentration of pH 4.6. To portions of this were added salicin and phloridzin to make one percent of phloridzin and salicin, and one series was prepared with 1 percent phloridzin with the glucose reduced to M/16 instead of M/8. Potato decoction was used in the same way, supplemented by 1 percent mannite, 1 percent phloridzin, and 1 percent salicin. Duplicate cultures of each were prepared, and the mycelial growth of *N. galligena* and *N. sanguinea* was recorded as above after an incubation period of two weeks. The filtrates were saved when the mycelial mats were collected on filter papers. Aliquot parts of the filtrates were tested with Fehling's solutions for reducing sugars, but no accurate titrations were made. The observations on this test will be mentioned below. Table II gives the dry weights of mycelial mats.

TABLE II. GROWTH OF MYCELIUM OF *NECTRIA GALLIGENA* AND *NECTRIA SANGUINEA* ON NUTRIENT SOLUTIONS

Organism	Weight in milligrams of 14-day-old mycelium (dried at 70° C. for 3 days)							
	Potato decoction	Potato decoction + 1% mannite	Potato decoction + 1% phloridzin	Potato decoction + 1% salicin	Richard's E solution	Richard's E solution 1% phloridzin M/8 glucose. ++	Richard's E solution 1% phloridzin M/16 glucose ++	Richard's E solution + 1% salicin
Nectria galligena	26.2	28.2	32.6	23.2	81.1	99.2	87.7	52.9
Nectria sanguinea	23.0	26.4	21.9	29.3	75.2	54.1	62.8	89.9

The essential difference between the available carbon of the potato decoction and the Richard's E solution is the difference between potato starch of the former and glucose of the latter. Both fungi utilize glucose readily,

and greater production is realized where such a monosaccharide is directly available than in culture solutions where polysaccharides must first be broken down. The alcohol, mannite, evidently is not utilized to any great extent as indicated by the relatively small increment in growth where saccharides are present in the medium.

The experiments as thus far conducted relative to the effect of the glucosides, phloridzin and salicin, on growth although extremely meager open a field for further investigation and some speculation as to the possible influence on infection. As culture experiments show, phloridzin increases the growth of *Nectria galligena* and retards the growth of *N. sanguinea*. The reverse seems to be true when salicin is given. Whether phloridzin is essentially utilized by *N. galligena* or whether its presence acts merely as a growth stimulant can be answered only after further experimental work. The rough estimates of reducing sugars remaining in the culture solutions after the fungi had grown in them for two weeks would indicate that the phloridzin was utilized by *N. galligena* and salicin by *N. sanguinea*. Further work is necessary to determine whether a definite enzyme for the splitting of glucose from the phloridzin molecule is possessed by the mycelium of *N. galligena*, and also the role of phloridzin in the parasitism of the European canker organism.

Reed's[34] solution was supplemented with macerated filter paper and the paragalactan from the endosperm of date seeds. The latter was prepared and sliced as previously described by the writer.[35] After *N. galligena* and *N. sanguinea* were grown on these solutions for three weeks it was found that both organisms did very poorly when either source of carbon was given, but both grew better on the medium containing the hemicellulose. In both cases the organisms were able to hydrolyze, to a slight degree, the hemicellulose to simpler sugars as evidenced by the reduction of Fehling's solution. To summarize, then, the few experiments thus far conducted show that among the carbohydrates and alcohols tested, the European canker organism appears to utilize sugars, starches and glucosides more readily than the alcohol, mannite, or cellulose and hemicellulose.

MORPHOLOGY AND LIFE-HISTORY OF *NECTRIA GALLIGENA*

MORPHOLOGICAL CHARACTERS IN CULTURE AND IN NATURE

Every species of fungus which will grow as a pure culture on artificial media usually produces on certain media some morphological character or combination of characters which will more or less readily distinguish it from other species. Thus the morphological characters in culture which identify the European canker organism will be given below. In the study of a species of Nectria in culture the following morphological growths are usually taken into consideration: chlamydospores, sporodochia, stromata, pionnotes, sclerotia, conidia, perithecia, ascospores, and the colors produced on certain media. Of these the writer has never seen the production of chlamydospores in cultures of *Nectria galligena*. The other characters will be taken up as listed

Sporodochia. These are creamy-white pustules of conidiophores arising from the medium which is covered with a rather closely growing white mycelium having tints of yellow and brown. On sweet clover and apple stems the mycelium is mostly in the surface of the bark and there arise from it, usually at the inoculation points, the cream-colored sporodochia. Sometimes the conidiophores break through small openings in the epidermis, in thickly divaricate tufts, which are evenly spreading, usually forming hemispherical sporodochia. These are usually 1 to 2 mm. in diameter but may become 3 mm. in unusual cases. When grown on a bare medium like glucose agar the stroma is composed of loosely interwoven hyphae on a felty mycelial mat. Often there appear globular or hemispherical sclerotium-like bodies about 1 to 2 mm. in diameter. These are sterile, plectenchymatous tissues of a reddish brown color giving the impression of perithecia but they have not been proved to be connected with the formation of the latter. Sporodochia have been observed on potato glucose agar, apple and sweet clover stems, and Cayley's glycerinated potato cylinders. A more diffusely spread cluster of conidiophores is sometimes observed on sweet clover stems and nutrient agar. These have the same color as the sporodochia due to the same type of macroconidia produced. These are the pionnotes. Both types of conidial clusters arise from the thin diffuse stromata. Fig. 9 illustrates the white sporodochia on a cankered Anjou pear branch.

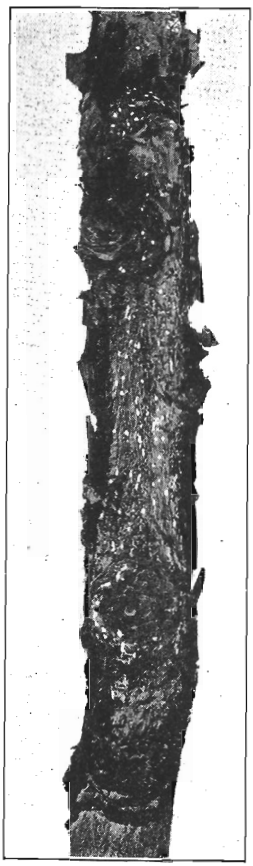

Fig. 9. Sporodochia on a pear canker. Two-thirds natural size.

Conidiophores. The conidiophores are usually divaricate and branched as shown in Fig. 10.

Conidia. The conidiospores are of two sorts: the cylindrical *macroconidia* and the ellipsoid *microconidia*. The macroconidia are produced on the conidiophores of the sporodochia and pionnotes. In masses they are usually a creamy yellow but after aging they often become a dull or light yellowish brown. When dried out the masses of spores may become chalky white. The large, cylindrical, septate spores are typical of the genus *Cylindrocarpon* Wollenweber. The mature spores are hyaline, 5—7 septate, 52—64 x 4.5—5.5 microns, and have the general appearance as illustrated in Fig. 11.

After the conidia are mature they are found in great numbers on the substrate or mycelium immediately surrounding the sporodochia. Under natural conditions the conidia are found on bark of pear and apple. Under these conditions when the spores lie undisturbed in close proximity to each

other they anastomose as illustrated in Fig. 12. Cayley[10] has previously observed this condition and illustrated it. This conjugation takes place when certain cells of the spores throw out connecting hyphae. One cell of a spore may connect with a cell of a neighboring spore or several cells of one spore may connect with several cells of a neighboring spore or spores.

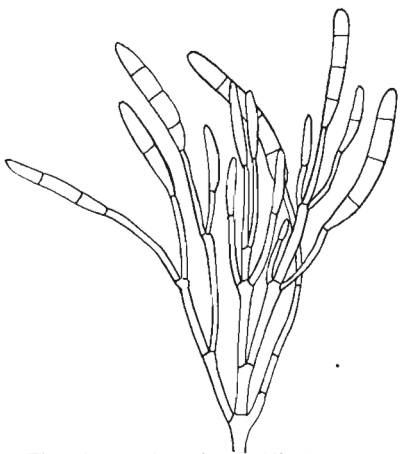

Fig. 10. Portion of a conidiophore from a sporodochium produced on pear bark. X 575.

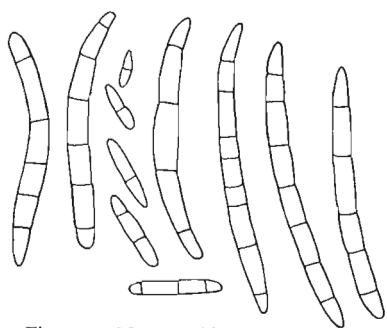

Fig. 11. Macroconidia of *Nectria galligena.* X 575.

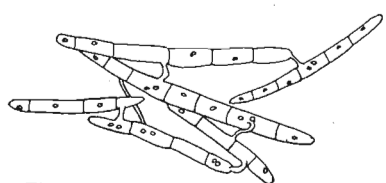

Fig. 12. Conjugation of macroconidia. X 575.

The result of this linking of the conidia of *N. galligena,* according to Cayley, is the formation of a palisade pseudo-tissue which helps to increase the bulk of the sporodochium. "At this stage the behavior of the nuclei of the macrospores is interesting but little understood. The passage of the nucleus from the cell of one spore to that of another has never been observed, but frequently one cell is seen to contain two nuclei and the cell to which it is linked is enucleate." This condition is illustrated in Fig. 12.

Variations in septation, size, and shape of the macroconidia are brought about by variation in nutrition, moisture, temperature, and other factors. Johann[25] has mentioned these variations brought about by temperature on Fusarium spores. Septation, of course, increases with the age of the spore up to a maximum of about seven. Since such factors may influence the morphological characters of spores under conditions obtaining in fungus cultures, our identifications of European canker have rested on the morphology of the fungus under field conditions. However, cultures from typical cankers of pear or apple bark which produce Cylindrocarpon spores conforming to the characters mentioned above, when the temperature ranges between 18° C. and 24° C., have been identified as *Nectria galligena.*

Microspores. These spores are not borne on differentiated conidiophores but are abstricted from the tips of small hyphal branches of the mycelium produced in cultures on various media. These conidia (Fig. 14) are about 4—7 x 1—2 microns and as far as known are functionless in the life-history of the organism.

Perithecia. On suitable media the rudimentary perithecia are to be found in the outer margins of the sporodochia. As to whether these arise directly from the heavy sporodochial mycelium or whether they arise in some way from the entangled pseudo-tissue formed from anastomosed macroconidia is still a question. Cayley could find no differentiated archicarp to which the perithecia could be traced. The early stages of development of the perithecia have been previously mentioned and such stages are found in sporodochia on the host (Fig. 15). The perithecia obtain a size of 350 to 500 microns in height by 250 to 400 microns in diameter, varying somewhat on different hosts and especially according to the position on the bark and such factors as temperature and moisture. Where "die back" branches are thoroughly infected with *Nectria galligena* growing saprophytically the perithecia are often found in great quantities bursting from every opening through the epidermis (Fig. 16). Where such totally-infected branches are horizontal, the perithecia are more numerous and larger on the lower surface of the branches. In the section, *Willkommiotes* Wr., all of the described species have perithecia of nearly the same color and not differing greatly in size. These species are distinguished mostly by the structure of the perithecium and characteristics of the ascospores. Weese[43] and Wollenweber[55] have emphasized these distinguishing characteristics which may be readily followed out.

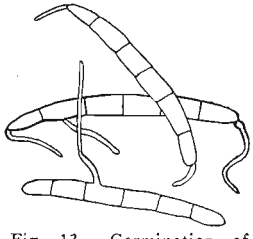

Fig. 13. Germination of macroconidia. X 575.

Fig. 14. Microconidia. X 1000.

The perithecia of *Nectria galligena* are at first cinnabar, then scarlet to blackish red, ovate to pyriform in general vertical section, usually not collapsing at maturity. The mouth is either at the apex of a distinct cone or at the center of a broader more or less sharply defined disc.

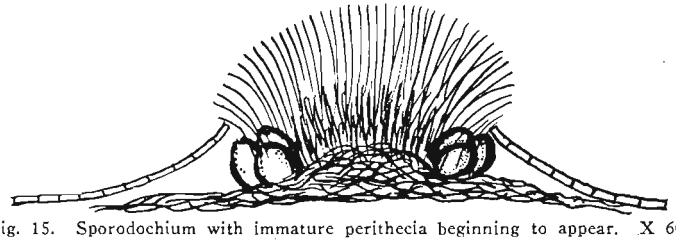

Fig. 15. Sporodochium with immature perithecia beginning to appear. X 60.

The perithecial walls are about 30 to 40 microns thick and are composed in the basal or flask-shaped portion of rather delicate-walled cells, up to 14 microns in size. Only in this lower portion of the perithecial walls is there a distinct parenchymatous structure. In the walls of the upper portion of the perithecia the cells are thick walled, the walls and cells becoming more indistinct from the mouth canal toward the outer surface. The disc of the perithecium near the mouth is composed of converging compact hyphae, or linear cells radiating from the mouth canal to the surface. The exterior cells of the walls take various shapes such as spherical, conical or setaceous represented in the roughness of the perithecia. The canal leading from the main cavity of the perithecium to the opening above is sometimes provided with rather

thickly set, erect, distinct hairs arising from the inner walls. The red perithecial walls stain violet-blue with KOH and discolor to yellowish with acid.

Asci. The asci are thin-walled, cylindric to club-shaped, sessile or distinctly stalked, with rounded thickened apex, 8-spored, 90—125 x 8—15 microns.

Paraphyses. The paraphyses extend above the asci, are thin-walled, septate, 5—8 microns broad, very numerous, gelatinizing at maturity.

Ascospores. These spores are distinctly two-celled; they may or may not be guttulate. They are hyaline, smooth, mostly ellipsoid, sometimes narrowly spindle-shaped, broadly ellipsoid, ovoid and unequal-celled; one-seriate or unequally 2-seriate above, 14—21 x 5—7 microns (Fig. 17).

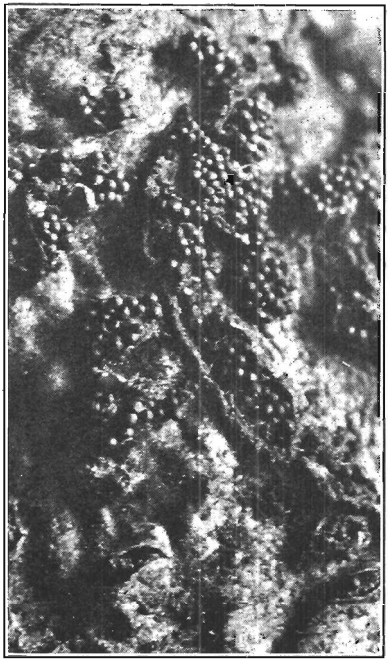

Fig. 16. Perithecia of *Nectria galligena* on bark of winter-killed apple twig where the organism is growing saprophytically. Usually perithecia are more scattered. X 5.

ASCOSPORE DISCHARGE

The perithecia under Oregon conditions start to form shortly after the rainy season begins in the fall. The growth and maturity proceeds rather more rapidly during months of continuous rainfall than during months when the intermittency allows short, drier periods to check growth.

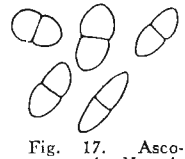

Fig. 17. Ascospores of *Nectria galligena.* X 500.

In the fall of 1920 there were intermittent rains on August 27 and 29, September 9 to 14 and 20 to 26. On October 1 rains began to become continuous with humid atmosphere so that fungus growths such as the sporodochia of *Nectria galligena* kept more or less moistened continuously. Perithecia began to appear large enough to be seen by the aid of the microscope (up to 40 microns in diameter) by October 16. Ascospores were first noticed on November 25, when only a very small percentage of the perithecia were mature. By December 17 no immature perithecia were found and nearly all macroconidia were gone. During the spring of 1921 the discharge of ascospores was observed until June 23. After that time there were no ejected spores found on microscope slides tied over the mouths of perithecia *in situ* on cankers. In the fall of 1921 continuous rains and humid weather beginning October 13 were preceded by rainfall on September 18, 19, and 21 only. But after October 13 wet weather was continuous, perithecia began to develop by October 24 and by December 14 the discharge of ascospores was first noticed. During the following spring, 1922, ejected spores were

found until April 24, but the continuous rains ceased April 16 instead of June 18 as the previous year.

The fall of 1922 was comparatively dry. There was a heavy rain September 26 to 28, but no more until November 1, when continuous humid weather began. The beginnings of perithecial growth were first observed November 20, but spore discharge was not observed until the first week in January (Jan. 5). In this case the dry fall delayed and slowed up perithecial growth so that spore discharge was delayed. In the spring of 1923 more or less continuous precipitation lasted until March 24, and then very light precipitation although with somewhat humid nights continued until June 17. On June 19 the discharge of spores was last observed.

In the three years during which the observations were made it is interesting to note that in 15, 11, and 19 days, respectively, after continuous humid weather set in, the perithecia were first seen in early stages of development. In 55, 63, and 65 days, respectively, after the same dates the ejection of ascospores was first observed. There seems to be very little more distinct correlation between the period of continuous humid weather and spore discharge, than between the latter and the period from the time of the first fall rains; for spore discharge took place in 90, 87, and 101 days, respectively, after the dates of the respective first fall rains.

The above notes concerning the time of discharge of ascospores seem to apply for the great majority of cases. It should be mentioned, however, that as late as March cankers have been observed on which perithecia were found in all stages of development. Undoubtedly many of such perithecia which are just at maturity when humid weather ceases in the spring will revive and discharge spores soon after the first rains in the fall. Some such cases have been found, but most of these holdover perithecia which are not eaten by insects during the summer become livid, collapse, and decay early in the humid period of the fall. The latest that livid, weathered perithecia from the previous season's crop were observed was December 14, 1921. These were empty of spores.

The usual reports of the disease by European workers state that the perithecia discharge the ascospores in the spring and early summer, but the reports are usually rather indefinite as to the exact time of spore liberation. Miss Cayley,[10] however, reports definitely that in England "perithecia begin to form in the late autumn, develop slowly throughout the winter months, and dehisce in spring. Material gathered in February showed perithecia in all stages of development; in April the perithecia are mostly fully grown and about to dehisce." This is later than the perithecia have matured in Oregon during the three winters mentioned above, but perhaps our winters in the Pacific Northwest are milder than is usual for those districts of Europe where this canker is prevalent.

The asci of *Nectria galligena* forcibly eject their spores, as is the case with most Ascomycetes. Variations in humidity, however, may modify conditions within the perithecia to such an extent that the mechanics of spore discharge may be altered. At times conditions are such that the spores of a single ascus may be so ejected from the ostiole that they are thrown for some distance, while at other times whole tendrils of spores slowly pour out from the perithecial mouth. For instance Cayley[10] says that in cultures of *Nectria galligena* "ascospores emerge through the ostiolum in the form of whity-buff tendrils," while Wiltshire[47] mentions the fact that the ascospores were ejected from moistened perithecia so that they were caught on inverted

poured plates of malt extract agar. The writer has observed that the best climatic condition for forcible ejection of the ascospores is that following a rain when the atmosphere remains just humid enough to keep the perithecia pliable. Under these conditions a microscope slide held over perithecia at a distance of 2 to 4 mm. will receive several groups of eight spores in a period of ten minutes. If the perithecia are saturated with water, however, and have reached later maturity, many of the asci discharge at one time. The spores which are more or less glutinous under such moist conditions clog up the canal leading to the ostiole, and further discharge of spores and liberation of epiplasm gradually crowd the mass of spores out. They may be deposited on the mouths of the perithecia in globular whitish masses (Fig. 18) or take the form of spore horns.

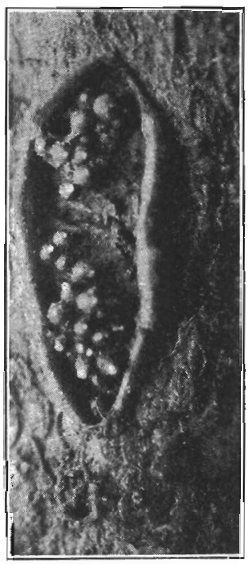

Fig. 18. White masses of ascospores at the mouths of the perithecia. X 12.

DISSEMINATION OF SPORES

Although distance from a source of spore production is undoubtedly to a degree a means of protection from infection, yet many young pear orchards situated at considerable distance from orchards which harbor European canker become infected. This is an indication that there are agencies by which the fungus may be disseminated. Some of the agencies which are usually considered as carriers for fungous diseases are insects, wind, water, and man, but it is difficult to obtain evidence that will fix the blame.

By insects. It has been mentioned above that many instances of European canker are associated with attacks of the apple woolly aphis *(Eriosoma lanigera)*. This fact seems to be well established by numerous European observers. The French worker, Descours Desarces, has shown without doubt that these insects carry the spores about in their woolly covering. The writer has often examined the woolly coverings of the woolly aphis *(Eriosoma pyricola)* inhabiting pear cankers and invariably numerous spores of *Nectria galligena* were immeshed in it. Since these aphids are often seen wandering up and down the branches of cankered trees they must be carriers of the disease, although actual cases have not been traced out by the writer.

Ants carry the ascospores of *Nectria galligena* on their bodies and are doubtless a minor means of transporting the spores up and down the branches of a tree. The bodies of ants, frequenting cankers dotted with clusters of perithecia, were washed in small quantities of water. This water was examined under the microscope and found to contain numerous ascospores. No count was made but from the numbers observed each ant must have carried hundreds of spores.

Although spores may thus be found adhering to the bodies of insects it is the writer's opinion that their role of making lesions or borings in the bark is perhaps of more importance than that of actually transporting spores.

The writer has found that during the summer in many orchards a majority of the perithecia are eaten by insects so that very few fruiting bodies persist until the fall rains. No insects have been found feeding on these fungous tissues, but the frass and partly eaten remains tell of insect activities.

By wind. It is evident that the behavior of the ejected ascospores places them at the disposal of currents of air or wind. The efficiency of the wind as a carrier, as also of the ejection of the spores from the perithecium, is largely dependent upon the moisture conditions. Surely the spores which ooze from the perithecia in masses in the form of droplets or tendrils are not so available for wind dispersal. This condition of the spores as described above may be one of the contributing factors which tends to limit the majority of infections to fall and spring months in most of the regions of the world where *Nectria galligena* is prevalent as a canker producer. Since so many of the infections are confined to leaf scars in the upper part of trees or water sprouts, it seems plausible to suppose that the spores are carried there by means of air currents. No attempts to capture wind blown spores on agar plates exposed in orchards have been made.

By water. That ascospores exuding from cankers are washed away by rain water running down the branches was demonstrated by the use of cotton traps as described by Heald and Gardner.[22] The same is undoubtedly true for macroconidia. Perhaps this wash of spores downward does not account for many infections except those starting in crotches, bark cracks, or open lesions due to other organisms.

PATHOGENICITY

The economic importance of *Nectria galligena* is manifest in its canker-producing or pathogenic activity. The discussion of the pathology of this organism will be taken up here under the general heads of natural infections, descriptions of the lesions on the host, and artificial inoculations and their results.

NATURAL INFECTION

Either the macroconidia or the ascospores of this organism are capable of infecting apple and pear stems through certain types of lesions, and several cases of apple fruit infections and some leaf infections have been mentioned.

Fruit and leaf infections. Allescher[2] was perhaps the first to find the conidial form of this organism inhabiting the core of the fruit of apple. But not until 1923 was the rot produced by *Nectria galligena* described. Ferdinandsen[16] observed the rot of two varieties of apples and two varieties of pears in three different communities of Denmark in 1919 and 1920. He has shown by inoculation that the canker organism isolated either from apple or pear cankers will infect apple and pear fruits to produce rot. On the other hand the organism from rotted pears or apples found in the orchard will cause canker of either apple or pear trees. The rotted spots are brown and sunken with sharp borders. They originated particularly around scab wounds and spread over the greater part of the fruit. In the spots appear the numerous cottony white sporodochia which soon become smooth and

grayish to brownish on drying. The macrospores of the usual type are found on these fruit spots.

In 1924 Kidd and Beaumont[26] listed this organism as one producing a fruit rot rarely found in England, it having been found as a lenticel spot on Bismark and in a small brown rot on Bramley's Seedling.

In 1921 D. F. Fisher sent the writer some apples from the Wenatchee Valley, Washington. These apples were partly rotted. The rot was light brown and on the surface were sporodochium-like fruiting bodies of an organism which was considered at the time to belong to the genus *Ramularia.* The spore masses were light yellowish and the cylindrical spores were usually 1—2 septate and 18—36 x 3—4 microns. After this organism was grown on apple, pear, and sweet clover *(Melilotus alba)* stems in pure culture it developed the *Cylindrocarpon* type of conidia which were not different from those of *Nectria galligena.* Even though this fungus is capable of infecting fruit, the fruit rot produced by it is so rare that it is not of economic importance.

Natural leaf infections by *Nectria galligena* have never been reported although Hartig[21] stated that he was able to infect leaves by inoculation both with the conidia and with ascospores of the organism which he knew as *Nectria ditissima.*

Trunk and branch infections. Earlier in this bulletin the prevalence of the European canker was discussed in a general way in its relation to the various kinds of infection courts, such as leaf scars, winter injury, cankers of other fungi, insect wounds, pruning cuts, and other wood exposures, and such cracks as occur at crotches, bud axils, etc. Since these general discussions have been given above the stages in the development of infection and the development of cankers only will be described here.

Bark infection described. A great majority of the infections in Oregon orchards take place through leaf scars, pruning cuts, and winter injury wounds. Since the early stages of infection and canker development can be traced more readily in the otherwise uninjured bark around a bud and leaf scar most of this discussion concerning bark infection will center about leaf scar infections.

In Oregon most of the leaf-scar infections take place shortly after leaf fall in the autumn. Others occur during the milder periods of the winter months with more frequent infections showing up in the early spring. After defoliation the exposed tissues of the leaf scar become more or less dried out so that tiny cracks occur in the surface cells and infection takes place through these cracks. Wiltshire,[48] working in England, has traced the incipient infection very minutely. He says: "The contraction of the tissue which takes place on drying results in small cracks appearing in the leaf scars, especially in the region of the soft tissue adjacent to the leaf traces. These small cracks allow the canker fungus to enter the host, which it does very readily, before the host has time to form a phellogen. The small depressions between the leaf scars and the main stem hold up small quantities of water which probably aid the germination of the spores of the fungus. The tissue of the leaf base is rather looser than the normal cortex and the canker fungus grows very freely in the intercellular spaces. The spread of the fungus is helped by the slow response of the host to form a limiting phellogen, and the small stem may soon become completely girdled.

"The second period of infection, which takes place when the trees become active in the spring, however, is very similar in its symptoms to the autumn infection. Many tiny cracks in the leaf scar tissue are found especially towards March and April when the buds are bursting. Throughout the winter the buds gradually swell by normal growth, but the swelling is usually sufficiently slow to allow the continuity of the phellogen to be maintained. The growth cracks, however, frequently extend to considerable depths. With the enormous increase in growth in early spring, fissures arise which expose the tissues of the leaf base and readily allow the canker fungus to enter." After the mycelium of the fungus reaches the intercellular spaces just below the cracks the entrance to the host is unimpeded.

DESCRIPTION OF CANKER

The first outward evidences of the canker may not appear immediately after infection. In some of the inoculations of leaf scars of Anjou pear reported below no external discoloration had appeared for several weeks. Within the bark and wood in these cases, however, the infection had spread so that tissues showed a slight discoloration out into some of the inner bark, cambium, and wood which was not immediately under the leaf scar. Infection had apparently followed one or more leaf traces for a short distance then spread laterally, sometimes in one direction, sometimes in another, but not necessarily with the discolored portions centering on the leaf scar. Usually a short time after infection the first external symptom is a tiny circular spot appearing in or along the margin of the leaf scar. This spot at first is slightly darker than the healthy bark but soon takes on a dark purplish brown color. These may first be detected when about 2 to 3 mm. in diameter, and they rapidly grow until 5 to 15 mm. Up to this time in many cases the growth of the fungous mycelium within seems to have had a slight periodicity of radial growth, caused by some unknown factor. This is evidenced by one or two concentric lines which are barely visible at first but which become more distinct as the discolored tissues shrink. At this stage on apple stems these very small cankers shrink slightly, but the epidermis usually does not separate from the cortex. At times, however, this separation does occur and gives to the canker a ragged appearance. In pear cankers, on the other hand, the epidermis and outer layers of the bark have a tendency to separate after the inroads of this organism, and this results in a very rough surface. Since most of these infections take place in the fall the cankers develop slowly all winter over extremely large areas of bark, and the rough surface is due to an edematous condition which is set up in the diseased tissues under the very wet climatic conditions. When this soft sappy tissue dries out during spring months the canker becomes rough and appears to have enlarged very rapidly during the latter part of this period. The truth of the matter, however, is revealed by cutting into the cambium near the visible portion of the canker in the very early spring, or even winter months, and finding that the edematous growth has proceeded far beyond the externally visible limits. Many cankers of this sappy, soft character upon which the macrospores occur have been observed on Anjou pear early in June. For the most part these cankers on larger branches occurred around early spring pruning cuts and unmistakably were due to early spring infections. As reported in 1921 by the writer and Owens,[60] "by the middle of June many of these cankers had already reached 20-22 inches in length and

1.5-2.5 inches in breadth. If the bark around such cankers is removed it will be found that in most cases the fungus has destroyed the cambium far beyond the superficial limits of the cankers." The shaggy appearance of these pear cankers following the edematous condition is illustrated in Fig. 7. When winter injury or sun scald is a forerunner of the bark infection by *Nectria galligena* the edematous condition is extremely rapid in its spread, and such cankers may attain great size.

Types of canker. Thus far the writer has described the development and appearance of European canker during the current season of its infection. The European canker is perennial in its nature, the mycelium hibernating in the diseased tissues of the canker during any inactive period only to start growth again into the surrounding healthy tissues upon the return of growing conditions. Under different circumstances this perennial growth may vary to such an extent that three distinct types of cankers may result. These are designated here as the open, closed and superficial cankers.

Open canker. During the dormant period of the host tree the canker-producing fungus grows vigorously under favorable temperatures, which prevail during the autumn and spring months. At this time the growth of the fungus predominates to such an extent that the dormant tree cannot check the fungus. When the tissues of the host become vigorous in growth, however, a period is reached when the growth of the host plant predominates. At this time the healthy bark around the diseased area begins to grow, forming a new cork cambium and layer of wood which heals over at the margins of the canker, forming a callus. As translocation is shut off by this interruption in the tissues of the callus above and below the canker, these callus tissues become greatly enlarged. The new bark forms over the edge of the callus so that with this increment of bark and wood a swollen appearance results. When this active growth of the host begins a fissure is formed between the healthy and diseased bark. The latter usually adheres to the dead wood beneath and, if it is not roughened because of the previous edematous condition described above, it dries down and fissures as it shrinks. While the callus is forming and the host is predominant in its growth, the mycelium is not entirely inactive. The fungous hyphae in the meantime have advanced in the wood formed the previous year, giving to it a darkened appearance beyond the edges of the callus. During the next growing season for the mycelium of the fungus the mycelium spreads upward and outward into the new year's wood and the bark of the callus. Thus the canker is enlarged and later another callus is formed concentrically about the last. As would be expected the growth in a direction longitudinal with the stem is more rapid and at least on the larger stems elliptically shaped cankers are formed, as has been described for the perennial canker of apple trees caused by *Gloeosporium perennans* Z & C.[58] In this type of canker after several years the central portion becomes deep and open and the old bark which adheres to the wood for the first year or two may fall away in time. This type of canker is illustrated in Figs. 5 and 19. In Oregon this type of canker is seldom found, except occasionally on apple, usually west of the Coast Range. In Europe, however, this open type of canker is the form usually described and illustrated.

Closed canker. On the Pacific Slope, cankers caused by *Nectria galligena* spread very rapidly, often covering large areas of bark in one season.

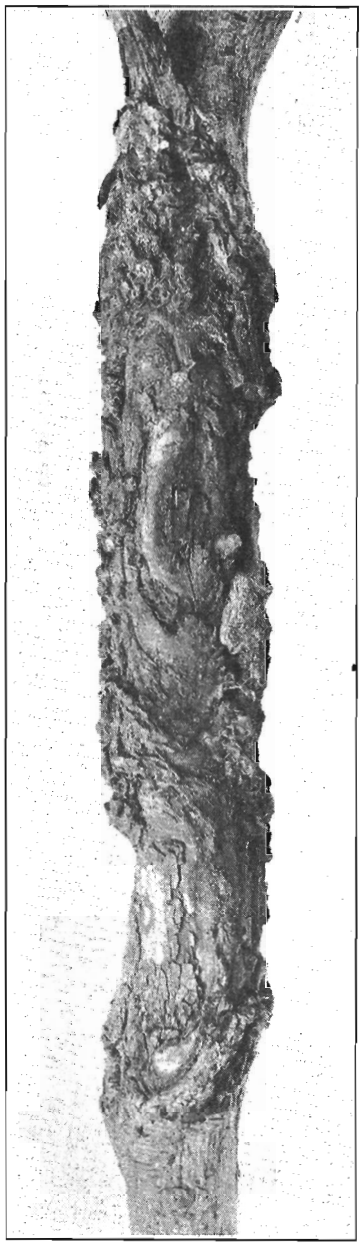

Fig. 19. Open cankers on apple.

In such cankers on pear the infection spreads most rapidly in the neighborhood of the cambium layer so that when bark is cut away from the margins the discoloration of the cambium extends far beyond the external visible limits. Such enormous lateral and longitudinal growth removes the annual calluses so far from each other that the concentric rings of callus are not so apparent as in the open canker. Such cankers both on apple and on pear are covered with a rough fissured gall-like bark and the wood beneath it may become dry and checked. In the apple the cankers arising from crotch infections are particularly of the closed, rough type. Figs. 7 and 20 illustrate this type of canker on pear and apple respectively.

Superficial canker. In contrast to the above mentioned advance of the fungus in the cambium there is a very different type of canker formed at times on pear bark (Figs. 21 and 22). In these cankers there is no damage to the bark deeper than the parenchyma of the pericycle during the first year of the mycelial advance. The cambium is seldom, if ever, invaded during the initial year of infection although the canker may spread through many square inches of bark. During the first season cankers of this type on young Surprise pear trees have been found to spread as much as 30 inches up and down the trunk and out onto the primary branches without any evidence of cambium infection.

This type of canker enlarges during the late autumn, winter, and early spring months. The cankers are edematous. The epidermis breaks during the winter, exposing an oozy, spongy, blackish brown, dissociated tissue. As reported in an earlier publication (Zeller and Owens[60]) the cause of this canker as it occurs in Oregon had not previously been ascribed to *Nectria galligena.* Since the spring of 1922, however, macroconidia have been fre-

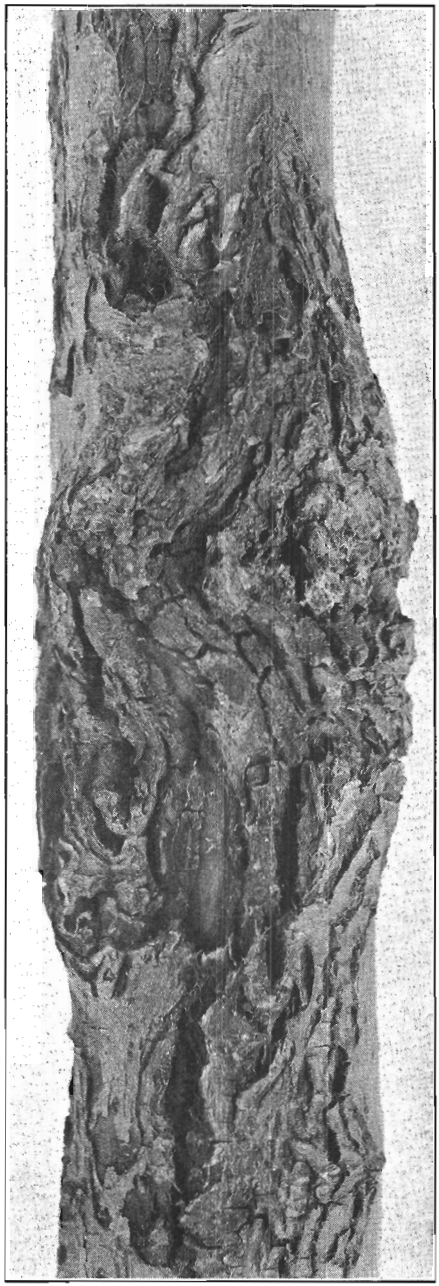

Fig. 20. Closed canker on apple.

quently found on these cankers and the fungus has been isolated from the early stages of this type of development. Sorauer[39] refers to a contribution by Paparozzi in which by inference he refers to this superficial type of closed canker on pear bark. In such cases where frost wounds are super-

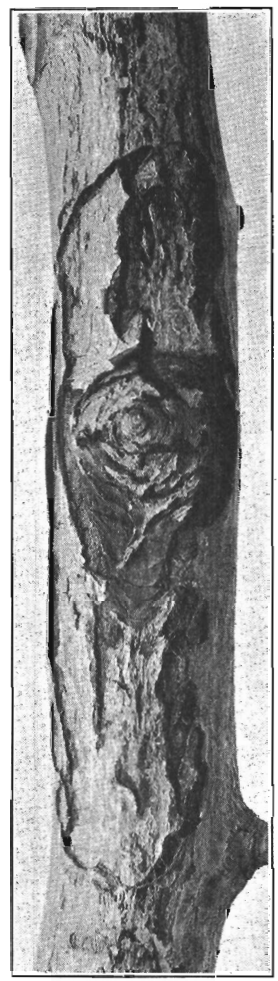

Fig. 21. Early stage of superficial canker on Howell pear.

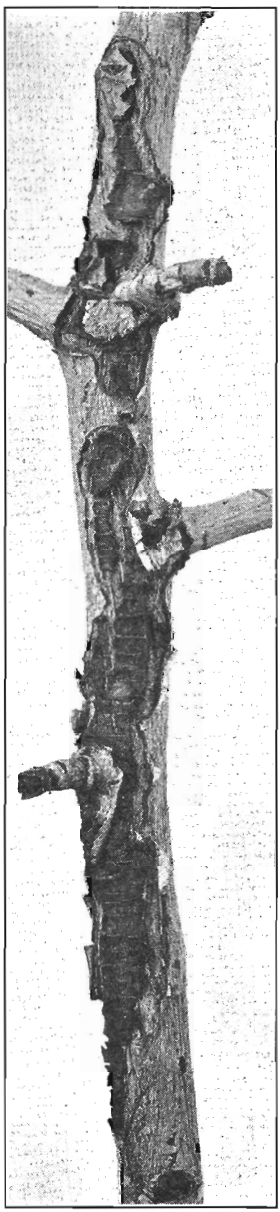

Fig. 22. Superficial canker on Bosc pear.

ficial Paparozzi finds that the infected portions are not bounded by excessive callus formation along the margins, and that these largely depend upon climatic conditions.

Infections leading to this type of canker usually have their start through such superficial infection courts as crotch cracks, frost cracks, leaf scars, and such checkings as are caused by scab *(Venturia pyrina)* infections of the bark. Although obtaining entrance through such courts this type of infection is seldom found on trees whose bark has not sustained at least slight winter injury.

If the apparently healthy bark above or below the canker is gradually pared away with a sharp knife, brownish lines extending parallel with the branch will be found following the parenchyma near the bast bundles. If these tissues are segregated out under sterile conditions the fungus may be obtained in pure culture. Starch tests have shown that the starch disappears in the neighborhood of these brownish lines. The brown discoloration of the bast bundles may be due to winter injury, and not caused by the fungus, but they are surely convenient avenues for the progress of the fungus. From the parenchyma near these bundles the infection spreads to the parenchyma of the outer bark. During winter sporodochia or pionnotes with macrospores are sparingly found in the superficial crevices below the broken epidermis. During the second year the infection may penetrate to the cambium when the canker takes on the appearance and destructiveness of the ordinary closed canker, or, if the weather happens to be relatively dry during the active spring growth of the tree, the pericycle may generate an entire new bark with a sclerotic outer coat that is a great deal more resistant to further infection than the natural, smooth epidermis of the bark. Such superficial cankers which are underlaid with a cork layer are seldom if ever troublesome again.

PATHOLOGICAL ANATOMY

For the most part the abnormal conditions of the host tissues arising from the presence of the canker producing fungus, *Nectria galligena,* have been mentioned above

in the descriptions of the three types of canker. Aside from the described dissociation of bark tissues and discoloration of woody tissues which have been actually penetrated by the fungus there seems to be a perceptible stimulation and abnormal production of wood parenchyma and medullary rays in the healthy tissues of the growing calluses. This adds to the depth and cup-shape of the open cankers. This stimulatory effect of the fungus on parenchyma production has been discussed by Voges.[41]

Bark. As described above in connection with infections causing the superficial type of canker, the mycelium follows the thin-walled parenchyma and the parenchyma close surrounding the sclerenchyma bundles in the bark. In fact the mycelium of *Nectria galligena* is found only in the parenchyma of the bark and in the wood parenchyma, medullary rays and xylem ducts of the woody cylinder. In the parenchyma of the outer cortex or just outside the cylinder of sclerenchyma bundles the mycelium seems to follow no particular course but the growth ramifies this parenchyma both extra- and intracellularly. In the parenchyma cells of the immediate region of the sclerenchymatous bundles, however, the mycelial growth seems to be almost entirely parallel with the bundles, especially in the parenchyma of the pericycle.

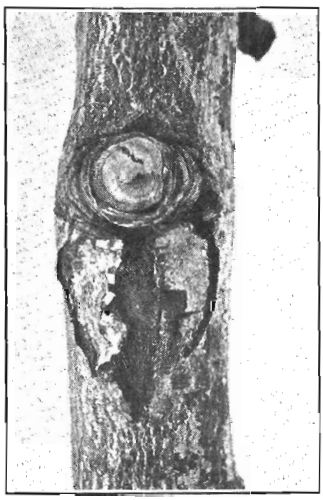

Fig. 23. Late spring infection showing diseased tissues dropping away exposing a well-formed bark underneath.

Wood. In the woody cylinder the mycelium has been traced to a depth of 2.4 mm. from the cambium as single intracellular hyphae passing lengthwise through the medullary ray cells. Longitudinally with the stem the mycelium pervades much of the wood parenchyma both extra- and intracellularly to the same depths as well as in the xylem ducts.

The stimulation of parenchyma production which has been mentioned above in the description of callus formation evidently affects mostly the parenchyma of the pericycle, i. e., that cylinder of parenchyma beneath the sclerenchyma.

In many cases of leaf-scar infection in the late spring the mycelium of *Nectria galligena* does not penetrate beyond the outer layers of the pericyclic parenchyma, provided the tree has started growth activities. In such cases a cork cambium is formed in the inner layers of the parenchyma of the pericycle. This cork cambium connects with that of the outer bark in the surrounding healthy bark. As cork is produced by this newly formed cork cambium the diseased tissues are excluded and as they dry they soon drop away (Fig. 23).

In the case of the superficial type of European canker described above (page 34) practically the same tissues as just described for late spring infections are invaded by the mycelium, and the cork cambium is formed to exclude it in the same manner. There must be, however, some undiscovered penetration below the parenchyma of the pericycle, perhaps into medullary rays, for the cambium region may be infected the second year. When the

diseased outer bark layers are cut away down to the parenchyma of the pericycle, the drying out of the tissues and treatment with a fungicide, together with the formation of the new cork layer, are very effective in excluding the disease.

INOCULATIONS

In December, 1921, and January, March, and April, 1922, inoculations of Anjou pear were made in two ways. First, water suspensions both of macroconidia and ascospores were sprayed from an atomizer on 20 branches each showing (1) no wounds, (2) pruning cuts, and (3) broken spurs. Second, inoculations were made by inserting spores under the bark in T-shaped cuts, 20 inoculations being made each month, 10 with ascospores and 10 with macroconidia. The results of all of these inoculations were taken on June 23, 1922.

TABLE III. RESULTS OF INOCULATIONS BY SPRAYING MACROSPORES AND ASCOSPORES OF *NECTRIA GALLIGENA* ON ANJOU PEAR

Date of inoculation	Condition of host	Number of inoculations	Spores used	Number of cankers formed June 23, 1922
1921				
December	No wounds	20	macroconidia	1
December	No wounds	20	ascospores	0
December	Pruning cuts	20	macrospores	3
December	Pruning cuts	20	ascospores	2
December	Broken spurs	20	macrospores	1
December	Broken spurs	20	ascospores	6
1922				
January	No wounds	20	macroconidia	2
January	No wounds	20	ascospores	
January	Pruning cuts	20	macroconidia	4
January	Pruning cuts	20	ascospores	3
January	Broken spurs	20	macroconidia	3
January	Broken spurs	20	ascospores	2

The results of the inoculations in the first group where the spores were sprayed on the surface are presented in Table III. The same type of inoculations made in March and April resulted in no lasting or deeply infected cankers. The four cankers resulting where no wounds were present at the time of inoculation perhaps resulted from leaf-scar infections as reported by Wiltshire and Spinks.[52]

The results of the inoculations made by introducing spores into the cambial region through T-shaped cuts are recorded in Table IV.

TABLE IV. RESULTS OF INOCULATING THE CAMBIAL REGION OF ANJOU PEAR WITH MACROCONIDIA AND ASCOSPORES OF *NECTRIA GALLIGENA*

Date of inoculation	Number of inoculations	Spores used	Number of cankers formed as observed June 23, 1922
1921			
December	10	macroconidia	4
December	10	ascospores	6
1922			
January	10	macroconidia	5
January	10	ascospores	9
March	10	macroconidia	7
March	10	ascospores	2
April	10	macroconidia	2
April	10	ascospores	4

During the month of October and the early part of November, 1923, rather extensive inoculations of leaf scars and pruning cuts were made. Since our greatest difficulty with European canker in Oregon arises in pear orchards, these inoculations were again made on Anjou pear. Ascospores and macroconidia for these inoculations were obtained from cankers on Anjou pear. These were secured in large quantities and inoculations were made in the following two ways: (1) spores were sprayed on the host in a water suspension and (2) a thick paste of spores in water was smeared on the leaf scars and pruning cuts. It was thought best to try to learn something about the influence of bordeaux spray on the spores of *Nectria galligena* on the host, and home-made bordeaux mixture 4-4-50 was therefore applied at intervals before and after the inoculations were made. The inoculations were made immediately and 7 days after defoliation and immediately and 7 days after pruning. The results of these inoculations which were observed the first week in July, 1924, are recorded in Table V.

DISCUSSION OF INOCULATION RESULTS

General. The following conclusions have been reached after examining the cankers resulting from the inoculations reported in tables III, IV, and V.

(1) Inoculations result in cankers in a greater percentage of cases where spores come directly in contact with freshly wounded cambium than in cases where the spores come in contact with such natural lesions as leaf scars or even in the case of pruning cuts.

(2) In our experiments inoculations resulted in cankers when made between the time of defoliation of the host in the autumn and the awakening of the cambium in the spring. The greater number of inoculations resulted in canker if made in the fall months rather than the spring, although the period of greatest evidence of infection and enlargement of canker extends from early spring until the rapid growth of the host slows up the activity of the fungus. This period usually extends into June under conditions prevailing in Western Oregon.

(3) **Period of incubation.** The period of incubation, or that period from the time of inoculation until the first evidences of canker formation, was usually very short, but the cases are extremely variable. When inoculations are successful in the early spring the indications of infection may show up in four or five days in the case of leaf-scar infections during very damp weather. When the weather is intermittently moist this period is extended, at times there seeming to be a hibernation of the mycelium for a short time until more favorable weather. Damp weather aids the growth to deeper tissues and then canker enlargement is unhindered until growth of the host and warmer weather inhibit growth of the mycelium. In cases of cambium inoculations canker formation may not appear at the surface for two to three weeks in spring inoculations, but intermittent precipitation does not hamper these cases of canker enlargement. In the case of leaf-scar inoculations made late in the spring, infection sometimes took place, but the lesions were mere superficial scales of diseased bark tissue. These are sloughed off early since the host at this period of the year lays down a cork layer which cuts off the inward progress of the fungus. In the case of autumnal inoculations the period of incubation is controlled by the same

TABLE V. NUMBERS AND PERCENTAGE OF INFECTIONS ON LEAF SCARS AND PRUNING CUTS OF ANJOU PEAR INOCULATED WITH *NECTRIA GALLIGENA*

Types of spores used for inoculation	Leaf-scar infections									Pruning-cut infections								
	Inoculated immediately after leaf fall				Inoculated 7 days after leaf fall					Inoculated immediately after pruning				Inoculated 7 days after pruning				
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
	Unsprayed.	Sprayed* just after inoculation.	Sprayed* 7 days after inoculation.	Sprayed* just before inoculation.	Unsprayed.	Sprayed* just after inoculation.	Sprayed* 7 days after inoculation.	Sprayed* 7 days before inoculation.	Sprayed* just before inoculation.	Unsprayed.	Sprayed* with bordeaux 4-4-50 just after inoculation.	Sprayed* with bordeaux 4-4-50 7 days after inoculation.	Sprayed* with bordeaux 4-4-50 just before inoculation.	Unsprayed.	Sprayed* with bordeaux 4-4-50 just after inoculation.	Sprayed* with bordeaux 4-4-50 7 days after inoculation.	Sprayed* with bordeaux 4-4-50 7 days before inoculation.	Sprayed* with bordeaux 4-4-50 just before inoculation.
When spores were sprayed on as a water suspension																		
Ascospores	19 in 25 76%	2 in 25 8%	1 in 25 4%	0 in 20 0%	1 in 25 4%	0 in 25 0%	3 in 25 12%	0 in 20 0%	0 in 15 0%	16 in 25 64%	2 in 25 0%	4 in 25 16%	0 in 20 0%	12 in 25 48%	0 in 25 0%	3 in 25 12%	0 in 20 0%	0 in 20 0%
Macroconidia	7 in 25 28%	1 in 25 4%	0 in 10 0%	0 in 17 0%	0 in 25 0%	0 in 25 0%	1 in 25 4%	0 in 15 0%	0 in 10 0%	9 in 25 36%	0 in 25 0%	1 in 25 4%	0 in 16 0%	7 in 25 28%	0 in 25 0%	1 in 25 4%	0 in 15 0%	0 in 16 0%
When spores were smeared on as a thick paste																		
Ascospores	21 in 27 77.7%	3 in 28 10.7%	0 in 25 0%	0 in 10 0%	0 in 25 0%	1 in 26 3.85%	2 in 20 10%	0 in 20 0%	0 in 18 0%	23 in 25 92%	0 in 25 0%	3 in 20 15%	0 in 20 0%	19 in 25 76%	0 in 25 0%	5 in 25 20%	0 in 20 0%	0 in 20 0%
Macroconidia	18 in 25 72%	0 in 25 0%	0 in 10 0%	0 in 10 0%	2 in 25 8%	1 in 26 3.85%	1 in 15 6.67%	0 in 15 0%	0 in 16 0%	14 in 25 56%	0 in 25 0%	1 in 20 5%	0 in 17 0%	17 in 25 68%	0 in 25 0%	0 in 25 0%	0 in 25 0%	0 in 15 0%

*Sprayed in all cases with home-made bordeaux 4-4-50.

factors which have been mentioned for spring inoculations. In the fall, however, the host is not active and if low temperature or low humidity does slacken the progress of infection and canker enlargement the fungus mycelium hibernates in the diseased tissues, resuming its progress during any favorable periods that may appear in the fall or winter months.

(4) **Results of leaf-scar and pruning-cut inoculations.** As shown by the results tabulated in Table V both leaf scars and pruning cuts inoculated with ascospores yielded a greater number of infections than did either inoculated with macroconidia. There may be at least two reasons for this. First, the ascospores may have been more viable after discharged than were the macroconidia under the prevailing weather conditions. Second, it may be that the extremely large size prevents the macroconidia from obtaining so great depths through capillary suction into the tiny crevices of leaf scars or the exposed xylem ducts of pruning cuts. This last-mentioned ingress of spores has been explained by Brooks and Moore[9] who have observed fungous spores drawn into wood vessels to a maximum distance of 6 mm. by capillarity when a drop of water struck the cut end of the wood.

(5) When inoculation consisted in smearing a heavy paste of spores from a needle onto the surface of pruning cuts or leaf scars a higher percentage of cankers resulted than when the spores were sprayed on as a water suspension from an atomizer. This is doubtless due to the greater chance of infection because of the greater number of spores in the smear.

(6) There was not a great difference of percentage of infections in the leaf-scar and pruning-cut inoculations.

(7) Many more cankers resulted from leaf scars inoculated immediately after defoliation than from those inoculated one week after defoliation. This contrast was striking when either ascospores or macroconidia were used. Although the contrast is not so great, more cankers resulted from pruning cuts inoculated immediately after pruning than from those inoculated a week later.

(8) **Results of spraying just before and just after superficial inoculation.** Home-made bordeaux 4-4-50 sprayed on the inoculation court just before inoculation or 7 days before inoculation gave absolute control in all cases where either ascospores or macroconidia were used. In the case of leaf-scar inoculations these sprays were applied just after defoliation. Sprays applied immediately after inoculation also gave reasonably good control in nearly all cases, but those applied one week after inoculation were not satisfactory. Especially is the latter fact true in the case of the control of ascosporic infection of pruning cuts.

CONTROL

HISTORY OF EUROPEAN CANKER CONTROL

Swales[38] has said that "to go no further than back to the latter half of the 17th century and the first quarter of the 18th will suffice to provoke interesting reflections on the subject of apple canker" control. In 1710 Samuel Gilbert, Editor of The English Gardener, says that "the

canker is as bad a mischief as any that happens to trees, but especially to young trees, which being small, are eaten or tainted round before one is aware of it. Therefore if your fruit be of such a kind as is subject to canker, as of a truth some are more than others * * * in such a case it is hard curing. * * * Sometimes too deep planting causeth trees to canker * * * and in some that are very subject to it a little bruise, and sometimes unseasonable pruning. As I said, if your fruit trees are of such kind as are more than ordinary subject to canker, or the nature of your ground more inclining your trees thereto, your diligence is to be the more in often viewing and searching your nursery or plantation, especially of your youngest and upon the first opportunity to cut out the least speck of canker. * * * This clean cutting of it out, I have found to be sufficient for the cure of the place so cut. * * * But if you find, as indeed it sometimes so falls out, that you cannot rid your trees of the disease, as it happeneth with some sorts of Pippins, Harvie-apples and some others, in some sorts of ground although of a pretty good nature, so that what your trees shoot this year dies the next * * * or before then, it will be your best course to cut off the head of such a tree or trees leaving only some convenient arms or boughs whereon you may graft some other sort of fruit which in a like ground or situation doth bear fruit well and is not so subject to canker." And so in the early part of the 18th century, as suggested by Swales, the ideas of control of cankers were quite advanced. These directions given by Gilbert, Swales calls "extraordinarily efficient and one needs a considerable acquaintance with the vagaries of the canker fungus at present to realize that even today we cannot *greatly* improve upon them in practice. Tim Mourse whose 'Campania Foelix: or discourse of the benefits and improvements of Husbandry' was written in 1697 and published in 1700 * * * says: 'The canker is another disease incident to trees, if it be in the branches, I look upon it as incurable, for the cankered branch being cut off, the after-shoots will likewise be cankered till you pare away all the branches: Esteem therefore such a tree to be fit for nothing.'" The same writer discussed the varieties of apples grown at that time in England which were resistant and susceptible to canker.

Although other European workers from Hartig's time (1880) until well in the twentieth century frequently have discussed control of canker by eradication, they have added little to the knowledge concerning control which had been practiced since the first of the eighteenth century.

Of the more recent writers on the subject of control of the European apple-tree canker Paddock, Brooks, and others have merely recommended sanitary measures in the orchard. In 1918 Owens[32] recommended eradication by cutting out all cankered branches or individual cankers and painting the wounds with bordeaux paste or disinfecting and painting with asphaltum.

Barker, Gimingham, and Wiltshire[5] found that the germination of spores from apple cankers was not inhibited in the least by the presence of sulfur, and that the acid or alkaline reaction brought about by secretions from the spores did not affect the influence of the sulfur on the fungus.

Grubb[19] using lime-sulfur and bordeaux sprays obtained much better control with bordeaux. His spraying showed "that where the trees were

sprayed, the presence of an old spore-producing infection has led to less than one-fifth as many new bud infections as where they were not sprayed." Doubtless in the light of the work of Wiltshire[48] the bud infections referred to were leaf-scar infections. After finding that a majority of the infections have their origin through leaf scars Wiltshire thought there was no valid reason why sprays applied to the leaf scars should not control infection. In his first trials with copper stearate the results were rather gratifying. Later, however, he reports[51] that in trials with bordeaux mixture he had nearly as high a percentage of infection on sprayed trees as on those left unsprayed. Wiltshire expresses the opinion that spraying treatment for autumn infection may not be effective enough to make it economically sound since "first of all the spraying of leaf scars immediately after defoliation is dangerous, because * * * the fruit still remains on the tree * * * and spray fluids may spot the fruit. Further, the spraying of ripe fruit with a copper fungicide is open to obvious objection. * * * Another difficulty in autumn spraying for canker is the rapidity of infection," necessitating, he thinks, at least two sprayings at the time of defoliation.

As an antiseptic for canker wounds which have been cleaned out Macineira[29] recommends the application of a paste made of ferric oxide in crude sardine oil.

MEASURES FOR THE PREVENTION OF INFECTION

Orchard sanitation is one measure which should be practiced to prevent needless infection. It is poor orchard practice to permit branches containing canker to remain near an orchard; such cankers harbor the canker fungus in a saprophytic condition. Such refuse is a constant source of spores during damp weather, and very late in the spring the lower parts of brush piles are damp enough to keep the fungus sufficiently active to discharge spores.

Elimination of diseased tissue by surgery is another preventive means of minimizing the number of new infections, for there is no known spray which will control the advance of the fungus within the tissues of the bark or wood. The thorough elimination of diseased tissues may be brought about in one of two ways: the removal of a cankered branch or the removal of the diseased bark of a canker. The grower must judge these cases from the standpoint of strict economy. It would cost less to remove smaller cankered branches than to clean the cankers upon it, while on the other hand if the producing power of a larger branch will justify it, the cankers should be removed. Even in such cases large cankers sometimes will not justify their treatment. For instance, it would be folly to remove a canker extending more than half way around a branch; the entire branch should be removed.

For the removal of the diseased tissues from cankers certain *tools* have been found useful. A farrier's knife has been extremely useful for trimming the margin of wounds and for working in crotches. Some orchardists prefer the straight blade of a small knife such as is used for budding. In the case of superficial infections which do not reach to the cambium a farrier's knife or draw shave has been found useful for scarifying the bark.

In removing diseased tissues from cankers it is necessary to locate the limits of the infected tissues. In the case of deep cankers this can be found by cutting to the cambium at the visible margin of a canker and gradually cutting back into the healthy bark until a region of the cambium which shows no discoloration is reached on all sides of the canker. The removal of all the diseased bark is important. So far as possible the final wound should be pointed at each end so as to aid in healing. Ragged edges or edges of bark which are not cut perpendicularly to the surface of stem will die back to some extent and become infection courts for fungus spores.

In this connection clean and proper pruning should be emphasized. Pruning cuts should be made as close to the parent branch as possible. This will insure the most rapid possible healing. Any splintering or roughness is highly undesirable. Stubs needlessly delay or wholly prevent healing, and such wounds are almost sure to result in heart rot.

In cases of the superficial type of canker it is only necessary to scrape away the diseased outer bark and paint over the scarified surface with some antiseptic tree paint.

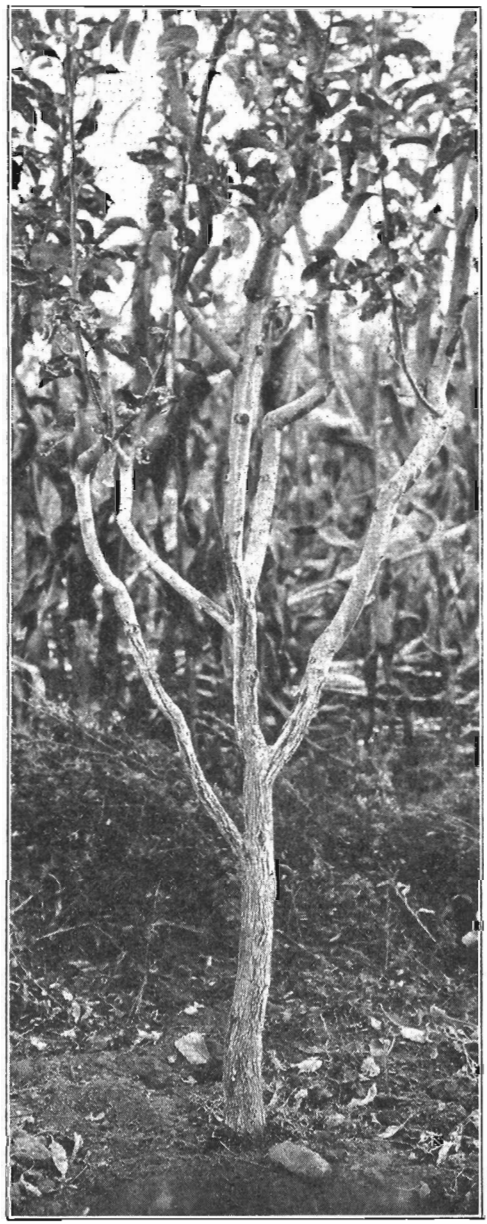

Fig. 24. Trunk of Surprise pear which shows successful scarification control of the superficial type of canker.

In these cases where the wood is not exposed it would be sufficient to leave the wound untreated if thorough cleaning away of diseased tissues has been accomplished. One grower treated these scarifications in three ways and had perfect control in all cases. He used bordeaux paste, as well as concentrated lime-sulfur, and left others exposed to dry out without treating. Many young pear orchards in Oregon have been scarified in the way described above with a very high percentage of success, but it would be inadvisable to allow scarifications to go untreated.

The time to apply wound dressings. Orchardists often ask whether wounds should always be covered with a dressing. In order to answer this question intelligently the writer made a survey of many well-kept, consistently-sprayed apple orchards of Western Oregon.

It was found that the usual spray program in apple and pear orchards seems to be sufficient to wash the ordinary pruning cuts for the prevention of infection by such wound-fungi as European cankers and wood rotting forms. No chances should be taken, however, with any wounds of any considerable size, for the time necessary to bring about complete healing also allows too great a chance for infection. The writer cannot say that smaller cuts will not be infected to some extent in well-sprayed orchards, but the orchardist should treat all apple or pear wounds 2 inches in diameter or more with a fungicidal wound dressing. This should be done soon after canker wounds are cleaned out, or pruning is done to eradicate cankered branches. If the weather is relatively dry when the wounds are fresh two or three days may not be too long to wait, but under damp conditions spores on the surface of wood will germinate in a few hours. Then, too, in an extremely short lapse of time, according to Brooks and Moore,[9] spores may be drawn so far into the wood by capillary water that the fungicide applied to the surface of the wound will not effectively reach it.

WOUND DRESSINGS

Many times the question is asked whether it is necessary to antisepticize a wound before a permanent coating is applied. This precaution is often resorted to, but some materials, such as creosote or carbolineum, may injure the healthy tissues of the tree and should not be used. Nearly all other materials used in this way are water soluble and easily wash away. Another objection is that they may prevent the adhesion of a subsequent paint or such coatings as tar or asphaltum.

Gloyer[37] states that "when tar is used it has in itself the necessary antiseptic properties to sterilize the cut surface," but to make this covering permanent it was applied following a coat of shellac which insures adhesive properties.

A coating of bordeaux paste, which has been used by Volck[38] in the Watsonville district of California and by Owens and others of the Oregon Agricultural College, is effective in its prevention of fungus infection of wood, but it is not durable and the orchardist cannot afford to repeat the wound dressing each season. During the last five years the writer has therefore experimented with some twenty different wound dressings recommended by various workers. Among these the one which combines the necessary properties for a successful tree paint is bordeaux paint.

Bordeaux paint is a wound coating which will remain permanent over a period of four years, at least. According to the writer's experience it does not injure the bark, makes a close union with the wood, and is not hard or thick enough to prevent free and rapid formation of callus. If made up properly it does not crack and yet seems to be sufficiently air-porous and water-porous so that sap pressure does not force it away from the wood causing pockets to form back of the coating. It is fungicidal, is relatively cheap, and can be applied in one operation. Some workers have stated that they have had slight injury to the margins of pruning cuts by using any preparation containing copper. To obviate such injury Volck first treats the cut edge of the bark with a ring of grafting wax, made of asphaltum and paraffin, before applying bordeaux paste to the wood.

Preparation of bordeaux paint. Bordeaux paint is prepared by stirring raw linseed oil *into* one of the commercially prepared powdered bordeaux. The writer has had success with the Sherwin-Williams Fungi-Bordo. A quantity of the dust, sufficient for the treating project at hand or convenient for a day's operations, is placed in a pail. While stirring, raw linseed oil is *slowly* poured in until a very thick paint is formed. It is more desirable that the paint seem too thick for it apparently becomes thinner after standing a short time.

SPRAYING

Under Oregon climatic conditions *Nectria galligena* discharges its ascospores intermittently from very late autumn until the close of the rainy season in the spring, and its macroconidia are discharged over the same period starting with the first rains in autumn. Most of the infections take place through leaf scars. The most logical time for applying sprays to prevent infection, therefore, would be as late as possible in the fall before the first rains and after defoliation of the trees. Some infections take place throughout the whole period of spore discharge, but those that occur later in the spring are usually occluded by the formation of a cork layer by the active bark of the tree and thus a much lower percentage of these are damaging.

Since the canker is not as commonly found in Western Oregon apple orchards which have been well sprayed as in pear orchards, the writer believed that the summer or fall spray of bordeaux mixture 4-4-50 which was applied for the control of apple-tree anthracnose *(Neofabraea malicorticis)* had perhaps been controlling also the European canker. Therefore, some trial sprays were applied to test out this idea.

Trial sprays in 1921. An orchard of Anjou pear trees which was infected with the canker was selected for a test of the bordeaux sprays. Five adjoining rows of 14 trees each were selected for the demonstration.

To Row 1, no spray was applied.

To Row 2, bordeaux 4-4-50 was applied on August 16, 1921 before the fruit was picked.

To Row 3, bordeaux 4-4-50 was applied on August 16 and 6-6-50 was applied September 27, after the fruit was picked.

To Row 4, bordeaux 6-6-50 was applied on September 27.

To Row 5, no spray was applied.

Results of 1921 spray applications. The results from these sprays were taken on June 24, 1922, and although the number of cankers on the unsprayed trees was low there was a very marked control of new infections on the trees receiving spray. The results follow.

28 trees receiving no spray showed 61 new cankers.
14 trees in row 2 showed 4 new cankers.
14 trees in row 3 showed 0 new cankers.
14 trees in row 4 showed 7 new cankers.

These results were sufficiently promising to warrant additional demonstrations of this control.

Trial sprays in 1922. In the fall of 1922 continuous humid weather did not begin until the first of November so that a spray rig could be taken into the orchard in October. Consequently, another spray demonstration was applied on Anjou pear trees which were very near the five rows used in the 1921 demonstration and which were more badly cankered. The rows were numbered the same as in the 1921 trials and sprayed as follows:

To Row 1, no spray was applied.
To Row 2, bordeaux mixture 4-4-50 was applied on October 18, 1922, when the trees were about ¾ defoliated.
To Row 3, bordeaux 4-4-50 was applied on October 18, and 4-4-50 on October 30, when the trees were completely defoliated.
To Row 4, bordeaux 4-4-50 was applied on October 30.
To Row 5, no spray was applied.

Results of 1922 spray applications. The numbers of cankers on these trees were counted on June 26, 1923, and gratifying results were obtained, as shown in Table VI.

TABLE VI. RESULTS OF SPRAYING WITH BORDEAUX MIXTURE 4-4-50 IN THE FALL OF 1922 FOR THE CONTROL OF EUROPEAN CANKER ON ANJOU PEAR

Row number.	Number of trees.	Sprays applied.	Time of spray applications.	Number of cankers resulting from infections of 1921-1922. Counted Oct. 12, 1922.	Number of new cankers resulting from infections of 1922-1923. Counted on June 26, 1923.
1	14	None	.....	68	148
2	14	Bordeaux 4-4-50	Oct. 18, 1922	92	37
3	14	Bordeaux 4-4-50	Oct. 18 and Oct. 30, 1922	103	21
4	14	Bordeaux 4-4-50	Oct. 30, 1922	85	11
5	14	None	.....	37	81

These results support those obtained the previous year in showing that bordeaux mixture brings about marked reduction in the number of

new infections. Apparently, also, there was a little better control where the spray was applied just after all the leaves had fallen than where only three-fourths were off. From the results of both years there is no indication that two sprays are more effective than one.

No tests have been made with an early spring spray although there is a possibility that such a spray might prove a valuable supplement to the fall application. The use of home-made bordeaux mixture as late as is practicable before the first fall rainy period seems from the limited tests conducted to offer the best promise of successful results in preventing infections in the average Oregon orchards.

GENERAL CONSIDERATIONS FOR CONTROL

The value of careful handling of trees in the general orchard practices should be emphasized. Injury to the bark of trees should be avoided whenever possible. Injuries to the bark are liable to result from pickers standing in crotches of the branches or from the careless handling of ladders and implements of cultivation. The importance of guarding against such injuries lies in the fact that European canker may begin in such scars.

Varieties of trees which are very susceptible to winter injury should be shunned in localities where European canker is a serious menace, for such trees are susceptible to a greater extent than those which are resistant to low temperatures. Nonresistant trees, however, to a degree can be protected against winter injury. A good physiological balance of soil moisture throughout the year will do much to keep the trees in vigorous growth and guard against winter injury to root systems. This last mentioned type of winter injury devitalizes the trees so that the branches are more subject to canker. The chief factor in the prevention of winter injury, however, is early dormancy. This may be aided by the cessation of cultivation early in the summer and planting of cover crops in orchards to take up moisture in the fall. As suggested on pages 14 and 15 the normal physiology of the trees must be maintained by good drainage, and good soil fertility, and every effort must be made to induce early dormancy.

RESISTANT VARIETIES

Earlier in this bulletin (page 15) the resistant and susceptible varieties as found in Oregon have been listed. Since several of our best commercial varieties of pears are rather subject to European canker, the question naturally arises as to whether the grower should replace these varieties with more resistant ones. By applying the control methods discussed above to the Surprise pear which the writer regards as one of the most susceptible to European canker, the canker of this variety has been satisfactorily controlled. In the light of the experiences of several growers the writer is of the opinion that the control of canker of susceptible varieties can be accomplished by thorough efforts of the grower and that no grower is warranted to avoid desirable commercial sorts on the grounds of susceptibility to European canker alone. When choice between varieties of equal desirability from a commercial standpoint is to be made, however, those more resistant to the disease would naturally have the preference.

SUMMARY

In this bulletin the European canker of apple and pear trees is fully described with particular references to conditions found in Western Oregon. The history of our knowledge of the taxonomy, physiology and pathogenicity of the causal organism, *Nectria galligena* Bres., is briefly discussed. The factors contributing to the distribution and prevalence of the canker are listed and separately discussed. The morphology of the pathogene is described as it occurs under natural conditions and in culture. The pathological anatomy or reaction of the host to the parasite is described, and the avenues of infection and mycelial inroads of the diseased tissues are followed. The development of cankers is followed from incipiency of infection to the perennial stages of mature cankers. The open, closed, and superficial types of canker are described with reference to the conditions under which they exist. The life-history of *Nectria galligena* Bres. is followed through the seasons of the year with special consideration given to the time of spore liberation.

This study shows that the macroconidia *(Cylindrocarpon)* are liberated from the time the first rains fall in the autumn until December or early in January, according to weather conditions, and then again from early spring until the close of the rainy period in late spring. Ascospores are discharged during a period extending from approximately 90 days after the autumnal rains begin until the rains stop in late spring.

Under natural conditions infection may take place during any part of the rainy months, but the majority of infections occur in the fall and spring, the greater number during the fall months.

A majority of the infections take place through fresh leaf scars, but such wounds as pruning cuts, broken twigs, winter-injury cankers, or cracks and lesions produced by woolly aphis become infection courts.

Control measures are both preventive and eradicatory. Results of inoculations and spray tests made near the time of defoliation of the trees are discussed. These results indicate that one application of bordeaux mixture 4-4-50 as late at practicable will give satisfactory control of new infections. Eradication measures consist of orchard sanitation, and the elimination of diseased tissues from cankers or the complete removal of cankered branches.

It is recommended that the larger wounds be treated with bordeaux paint made up by stirring raw linseed oil into powdered bordeaux.

LITERATURE CITED

[1]Aderhold, R. Impfversuche mit Nectria distissima Tul. Centr. f. Bakt. Par. u. Infektionskrankt. II 10:763—766. 1903.

[2]Allescher. Ber. Bot. Ver. Landshut. 12:130. 1892.

[3]Appel, O., und Wollenweber, H. W. Grundlagen einer Monographie der Gattung Fusarium (Link). Arb. K. biol. Anst. of Land- und Forstw. 8:I—207. Abb. 1—10. Taf. 1—3. 1910. (See pp. 163—174, Taf. 2, 88—93).

[4]———————— Studien uber der Gattung Fusarium (Link.). Zeitsch. f. Pflanzenkrankh. 23:44—45. 1913.

[5]Barker, B. T. P., C. T. Gimingham, and S. P. Wiltshire, Sulphur as a fungicide. Ann. Rept. Agr. and Hort. Res. Sta., Univ. Bristol **1919**:57—75. 1919.

[6]Birmingham, W. A. A. Canker of Apple Tree due to a fungus, Dothiorella Mali. E. n E. Agr. Gaz. of New So. Wales **35**:525—527. 1924.

[7]Bresadola, J. Strasser Pilzfl. Sonntaglb. **4**:413 (in Verhandl. Zoo. Bot. Gesell. Wein). 1901.

[8]Brooks, Chas. Some apple diseases. New Hamp. Agr. Exp. Sta. Bul. 144:128—129. fig. 26. 1909.

[9]Brooks, F. T. and W. C. Moore. On the invasion of woody tissues by wound parasites. Cambridge Phil. Soc. (Biol. Sci.) Proc. **1**:56—58. fig. 1. 1923.

[10]Cayley, Dorothy M. Some observations on the life-history of Nectria galligena Bres. Ann. Bot. **35**:79—92. pl. 4—5 fig. 1—25. 1921.

[11]Cotton, A. D. Apple canker (Nectaria ditissima) Great Brit. Bd. Agr. Jour. **24**:1263—1266. fig. 1—2. (Cir. No. 56). 1918.

[12]Czapek, F. Biochemie der Pflanzen. Jena. 1915. (see 1:294—305).

[13]Duggar, B. M. Fungus diseases of plants. Ginn and Co. 1909. (See pp. 242—243).

[14]Eckstein. Weider die Buchen-Wollschidlaus, Cryptoccoccus fagi. Deutsch. Forstzeitz. **35**:194—195. 1920.

[15]Falck, R. Die Bedingungen und die Bedeutung der Zygotenbildung bei Sporodinia grandis. Bietr. Biol. Pfl. **8**:213. 1901.

[16]Ferdinandsen, C. Uber einen Angriff von Krebs (Fusarium Willkommii Lindau) an Apfel- und Birnfruchten. Angew. Bot. **4**:173—184. Taf. 1—3. 1923.

[17]Gloyer, W. O. Blister canker of apple and its control. N. Y. Agri. Exp. Sta. (Geneva) Bul. 485:1—21. pl. 1—15. 1921.

[18]Goethe, R. Mittheilung uber den Krebs der Appelbaume. Leipsig. 1877.

[19]Grubb, N. H. Tests of fungicides on apple trees. Jour. Pomol. **2**:93—114. fig. 19. 1921.

[20]Harris, G. H. Some observations on the behavior of roots during the winter months. Oregon Agr. College Graduate Thesis. (for M. S. degree). pp. 1—34. Charts 1—8. 1923.

[21]Hartig, R. Der Krebspilz der Laubholzbaume, Nectria ditissima Tul. Unters. a. d. forstbotan. Institut Munchen **1**:109—128. Taf. 6. 1880.

[22]Heald, F. D., and Gardner, M. W. The relative prevalence of pycnospores and ascospores of the chestnut blight fungus during the winter. Phytopathology. **3**:296—305. 1913.

[23]Hesler, Lex R. Apple cankers and their control. N. Y. (Cornell) Exp. Sta. Cir. 28:1—28. fig. 1—16. 1915.

[24]————————————and H. H. Whetzel. Manual of Fruit Diseases. The MacMillan Co., N. Y. 1920. (See pp. 125—130. fig. 33).

[25]Johann, Helen. Influence of temperature on the morphology of Fusarium spores. Phytopathology **13**:51. 1923. [Abstract].

[26]Kidd, M. N. and A. Beaumont. Apple rot fungi in storage. British Myc. Soc. Tras. **10**:98—118. pl. 6—7. 1924.

[27]Lapine, N. Zum Krebs der apfelbaume. Landw. Jahrb. **21**:937. 1892.

[28]Leonian, L. H. A study of factors promoting pycnidia-formation in some Sphaeropsidales. Am. Jour. Bot. **11**:19—50. 1924.

[29]Macineira, F. El chancero canceroso del manzano y un nuevo tratamiento eficaz para combatirlo. El Cultivator Moderno **12**:V:11—12. 1922. [Abstr. New Measure for controlling apple tree canker, Nectria ditissima. Int. Rev. Sci. Practice Ag. N. S. 1:509—511. 1923.]

[30]Morse, W. J. Spraying experiments and studies on certain apple diseases in 1913. Maine Agr. Exp. Sta. Bul. 223-23—24. pl. 3—4. 1914.

[31]Nunch, Ernst. Untersuchungen uber Immunitat und Krankheitsempfng lichkeit der Holzpflanzen. Naturwiss. Zeits. f. Forstund Landwir 7:54—75: 87—114: 129—160. 1909.

[32]Owens, C. E. European apple canker. Ore. Extension Mimeo. Circ. 119: 1—2. 1918.

[33]Paddock, W. European apple tree canker in America. Science N. S. **12**:297—299. fig. a-c. 1900.

[34]Reed, H. S. The enzyme activities involved in certain fruit diseases. Va. Polytec. Inst., Agr. Exp. Sta. Rept. **1911—1912**: 51—77. 1913.

[35]Richards, H. M. Die Beeinflussung des Wachstums einige. Pilze durch chemische Reize. Jahrb. f. wiss. Bot. 30:665—688. 1897.

[36]Roberts, J. W. The sources of apple bitter-rot infections. U. S. Dept. Agr. Bul. 684:1—25. pl. 1—5. 1918.

[37]Rose, Dean H. Blister canker of apple trees: a physiological and chemical study. Bot. Gaz. **67**:105—146. fig. 1—10. 1919.

[38]Sorauer, Paul. Handbuch der Pflanzenkrankheiten I. Die nichtparasitären Krankheiten. Berlin. 1921. See p. 645.

[39]Swales, E. R. Apple canker—two centuries practice in its control. Jour. Pomol., **2**:271—273. 1921.

[40]Tulasne, L .R., et Tulasne, C. Selecta fungorum carpologia, etc. Fol. **3 vol.** 61 plates. 1861—1865. Paris.

[41]Voges, Ernst. Zur Geschichte und Entstehung des Obstbaumkrebses. Centralbl. f. Bakt. Parasit. u. Infekt. II. 39:641—672. fig. 1-4. 1914.

[42]Volck, W. H. Pruning fruit trees, with special reference to the apple. Calif. Com. Hort., Monthly Bul. 6:80—89. fig. 20a—26c. 1916.

[43]Weese, J. Zur Kenntnis der Erregers der Krebskrankheit an den Obst und Laubholzhaumen. Osterreich Landw. Versuchsw. Zeitschur. **14**: 876—885. Taf. 1. 1911.

[44]_____, Studien uber Nectriaceen. Zeitscher. f. Garungsphysiologie. 1:126—155. 1912.

[45]Willkomm, M. Die mikroskopischen Feinde des Waldes. Naturwiss. Beitrag z. Kenntn. d. Baum- und Holzkrankh. fur Forstmanner und Botaniker. Dresden. 1866.

[46]Wiltshire, S. P. A note on the relation between woolly aphis and canker. Univ. Bristol Agr. and Hort. Res. Sta. Ann. Rept. **1914**:94. 1914.

[47]Wiltshire, S. P. The apple canker fungus Ann. Report. Agril. and Hortl. Res. Sta. U. of Bristol. **1919**:23—29. 1919.

[48]_____, Studies on the apple canker fungus. I. Leaf scar infection. Ann. Appl. Biol. **8**:182—192. pl. 3 fig. 1—2. 1921.

[49]———————————, Canker control trials. Univ. Bristol Agr. and Hort. Res. Sta. Ann. Rept. **1921**:70—73. 1921.

[50]———————————, Studies on the apple canker fungus II. Ann. Appl. Biol. 9:275—281. pl. 12. 1922.

[51]———————————, Canker control trials, Univ. Bristol Agr. and Hort. Res. Sta. Ann. Report. **1922**:74. 1922.

[52]———————————, and G. T. Spinks. Apple tree canker. Ann. Rept. Agril. and Hortl. Res. Sta. U. of Bristol: **1920**:82—83. 1920.

[53]Wollenweber, H. W. Studies on the Fusarium problem. Phytopathology **3**:24—50. pl. 5. fig. 1. 1913.

[54]———————————, Ramularia, Mycosphaerella, Nectria, Calonectria—Eine morphologisch pathologische Studie zur Albrenzung von Pilz gruppen mit cylindrischen und sichelformigen Konidienformen. Phytopathology **3**:197—242. pl. 20—22. 1913.

[55]———————————, Pyrenomyceten-Studien. Angew. Bot. **6**:300—313. Tab. 8. 1924.

[56]Zeller, S. M. Studies in the physiology of the fungi. II. Lenzites saepiaria Fries, with special reference to enzyme activity. Ann. Missouri Bot. Gard. **3**:439—512. pl. 8—9. 1916. (See pp. 441—442).

[57]Zeller, S. M. European Canker. Oregon grower **3**:3—7. fig. 1—5 (Sept.) 1921.

[58]———————————, Wood decay in orchard trees in Oregon. Crop pest and Hort Rept. **1915—20**: 132—138. figs. 35—37. 1921.

[59]Zeller, S. M. and Leroy Childs. Perennial canker of apple trees. Oregon Exp. Sta. Bul. 217: 1—18. fig. 1—23. 1925.

[60]———————————, and C. E. Owens. European Canker on the Pacific Slope. Phytopathology **II**:464—468. fig. 1—4. 1921.