

T H E S I S

ON

AN INVESTIGATION OF CHERRY GUMMOSIS.

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AN INVESTIGATION OF CHERRY GUMMOSIS.*

PREFACE.

This thesis is the result of an investigation begun during the winter of 1907, with the object of seeking all possible causes of the diseased condition of cherry trees very prevalent in Oregon, commonly called "gummosis", and of applying the appropriate preventives or remedies.

All of the writer's time has not been devoted to this investigation, as other college and station work has required much attention. The first year's work was largely devoted to the accumulation and study of the literature bearing upon the subject. Active work began with a partial 'gummosis survey' of the principal cherry growing districts of the State during the summer of 1908, and the field and laboratory phases of the investigation have been continued up to the present time.

The writer realizes the absence of many desirable features that would have rendered the investigation more complete. While the subject is somewhat difficult, he feels that a much better thesis would have resulted had his capabilities been better organized at the start. He hopes, however, that the work here pre-

sented shows enough promise of ability to conduct independent investigations in the field of Plant Pathology, to be worthy of acceptance and to merit the honor of degree of Master of Science in Agriculture.

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F.L.G.

Introduction.

The formation of gummy or mucilagenous substances is not confined to any one species of plant but is rather widely distributed among several different genera. It is necessary therefore that the literature dealing with the phenomena of gum flow in general should receive attention so that by a comparison and study of the factors associated with the production of gum in other plants, we may arrive at a better understanding of the significance of gummosis in *Prunus avium* and *P. cerasus*.

In the study of the literature on the subject, the most careful attention has been given to the descriptions and investigations of gum-flow of the stone fruits.

A Review of the Literature.

While the phenomenon of gum flow from certain plants has undoubtedly been known and appreciated since very early times, the first accounts of studies on the formation and general significance of these gum exudations date from the middle of the last century.

The first horticultural reference found is that of Cole's (21), 1849, in which he states that the

rapid growth of the cherry renders it liable to injury, extremes of heat and cold bursting the vessels in the tender young wood and causing the gum to ooze out, which is very injurious. He advocated propagating the cherry upon Mazzard stock.

M~~W~~atosh (57), in 1855, pointed out that grafting the cherry upon stocks that did not develop equally with the scion was more conducive to gummosis than when stock and scion developed equally.

Mohl(60) in 1857 was the first botanist to publish the results of studies on the origin and nature of gummosis. His article dealt with tragacanth gum, but was not reviewed.

Karsten(44), also in 1857, published an article on the origin of resin, wax, gum and slime through the activity of the cell membranes. He mentioned that in the stone fruits, especially in the cherry, the gum originated through the transformation of the cell membranes of the wood and the starch contained in the cells.

Trécul(90) in 1860 gave out the results of the first important study of gummosis in the stone fruits. He had observed that the Acacias and certain of the stone fruits gummed most abundantly when sickly and in an abnormal condition caused by a fullness of sap in the young growing tissues. The disease, as he con-

sidered it, affected both the embryonic woody tissues and the more differentiated and lignified tissue adjoining it. Gum pockets were formed in the young woody tissue by the liquidation of the cell walls, the cavity thus formed being filled with the disorganized cell walls and the cell contents. Gum pockets were enlarged by the continual destruction of the peripheral cells. Trécul also stated that the cambium could form abnormal and normal woody tissue alternately, depending upon the prevalence of unfavorable conditions, rain being the chief factor in causing gummosis.

Wigand(98), in 1863, published the results of his studies on the gummosis of *Prunus avium*, which were mainly from the histological standpoint. He found that gum formation in the wood was always preceded by the formation of an abnormal wood parenchyma, which could occur at any time during the season of growth.

Frank(31) in 1866 in his study of vegetable slimes, mentions cherry gum. This he found to be formed from the secondary membranes of normal woody tissue, or through the disorganization of abnormally formed woody parenchyma as well as certain cortical tissues. He thought that in the tissues affected

with gummosis, the intracellular substances and primary membranes first changed into gum, the secondary membranes then following in the disorganization. The starch contents of the cells as well as the plant sap, also entered into the gum formation.

Sorauer (83) in 1872, agreed in the main with ^{the} anatomical findings of Frank. He regarded gummosis as a disease symptom, the true disease being an abnormal accumulation of plastic substances in the cambial regions, but which could not be normally utilized because the development of these embryonic tissues were retarded by the presence of too much water or by wounds.

Prillieux (68) in his study of the gum formation in stone fruit trees, while agreeing with Wigand and Frank that the gum formation began in the liquidation of the cell walls of abnormally formed woody parenchyma, and due to unfavorable external stimuli, differed in the explanation of the gum in the vessels and normally formed cells of the wood and bast. The gum in these latter tissues was due to infiltration and not to a change of the cell walls. Prillieux believed that the starch contents of the cells adjoining the affected tissues also took part in gum formation, the main bulk of the gum being derived from it. He was unable, however, by staining, to detect any intermediate stages of starch transformation.

Wiesner (97) in 1885, described an enzyme which he considered as active in the transformation of cellulose into gum.

Beijerinck (8), in 1886, advanced the hypothesis that the gum following attacks of *Coryneum* was due to the activities of a cytolytic enzyme which became active in the dying protoplasm of the cells, and diffusing out into the adjoining tissues, transformed them into gum.

Sorauer (83) in his article on Flux diseases gives a general account of cherry gummosis. As in his earlier work, he states that gummosis is simply the symptom of a disease. He believed the true disease to be an accumulation of plastic substances in the young growing tissues which could not be normally utilized either because they were not present in sufficient quantities, or because the growth of these tissues was retarded by the presence of too much water, or other unfavorable factors. Gummosis originated in the embryonic woody parenchyma and spread to the cambium or bark.

Sorauer made experiments in which the buds were removed from cherry trees in spring. Gummosis appeared in most instances. Peeling the bark, or any injury to the roots or crown caused gumming. He found that cherries on clay soil were more subject to

gummosis than those growing on more porous warm ground.

Arthur (5) in 1884 believed that the abnormal large amount of gum produced in the peach and related trees is due to the inciting influence of bacteria or fungi.

Garros (33) in his study of gums, isolated a ferment from cherry gum which he believed to be the cause of the gum formation. Through experiments he demonstrated that the arabin in gum arabic is not identical with the soluble part of the cherry gum.

Frank(31) compared the resin flow of the Conifers with the gum flow of the *Amygdalaceae* and found complete analogy between the two phenomena. Any sort of wound exerting an irritating influence upon the cambium, or more obscure factors such as unfavorable soil conditions, frost injury, or anything leading to weakened activity would result in gum formation. Regarding the physiological significance of gummosis, Frank believed the infiltration of the diseased areas with gum tended to protect the rest of the living tissues.

Lutz (5) believed the formation of gum in certain species of *Acacias* to be brought about by the activity of a diastatic ~~gum~~ ferment.

Woodworth (99) in 1895, mentioned that the profuse gumming of various stone fruits often followed attacks of the root by various members of the toad-

stool fungi. The disease seemd to be uncontrollable and was most prevalent in oak clearings.

Solla (?) attributed the gumming of the peach to an excess of water in the soil. He retarded the flow of gum by slitting the bark of the trunk and main branches.

Taft(86) reported a gum disease of the peach as being prevalent in many Michigan orchards, it being most common on neglected trees and those growing in poor peach soil. The gumming was most often associated with blighted buds, which formed part of a localized cankered area on the twigs. The cankers were attributed to some unknown fungus.

Powell(66)in his bulletin on the cherry, mentions, under diseases, sunscald and bursting of the bark. He stated that during late winter and early spring, the trunks of sweet cherries often crack open and exude quantities of gum. The trouble was most prevalent on the South and Southwest portions of the trunk and occurred after freezing and thawing weather had prevailed.

Too fertile a soil, abundant fall rains following a summer drouth, over-stimulation of wood growth, or other factors tending to prevent the maturity of the wood seemed to favor the trouble.

As sunscald and bursting of the bark were most

common on high headed trees or on exposed trunks, Powell favored the formation of low spreading trees to protect the bodies from the sun. Cultural methods favoring the early maturity of the wood were also recommended.

Cardwell(30) in discussing the apparent relation of climatic factors to gummosis, mentioned certain seasons in which warm rains, followed by occasional sunshine, during late winter and early spring, were followed by freezing weather of several days duration, such extremes of temperature produced sunscald and bursting of the bark on the Southwest sides of fruit trees. The stone fruits in such cases gummed badly. The remedy was to split the bark to give freer vent to the exuding gum and in bad cases, to cut back the tops. He also stated that among the cherry trees, the Royal Ann seemed most affected.

Hedrick (41) in his bulletin on the Prune, stated that gummosis was prevalent upon the prune, peach and cherry in Oregon, and that the same injury occurred in parts of California, Colorado, Texas, the South Atlantic States and Pennsylvania. Gummosis made its appearance at any time of the year, being most abundant in spring, especially if the trees has been injured by frost. Trees would frequently gum in mid-summer after a hot wave or a shower.

All attempts to establish the relationship of possible causal organisms to the disease were negative. Hedrick stated that injury to the plant cells through the agency of frost or heat seemed to be the chief cause of the trouble and he quoted prominent horticulturists as being of the same opinion.

Craig(22) reported gummosis of peach twigs as accompanying and following an attack of the brown rot. He marked certain twigs and branches on trees affected with *Monilia* in the Autumn and the next year, examination showed that these marked branches were gumming profusely. From these and other observations he believed *Monilia* to be a common cause of gummosis of the peach.

Selby (79) also reported a twig disease of the peach accompanied by gumming. The symptoms were exudations of gum upon the twigs, branches and sometimes upon the body of the tree. The specific nature of the disease was indicated by its spread through an orchard, but no causal organism was mentioned.

Masse (52) in 1899 reported what is probably the first instance where a fungus has been directly proved to be the cause of gummosis. The branches of the flowering shrub, *Prunus japonica* were frequently attacked by a disease characterized by the exudation of drops of gum. The fungus *Cladosporium*

epiphyllum was found constantly associated with the disease and by experiment was proved to be the cause.

Frank (31) in 1899, described a disease of sweet cherries which was very prevalent along the Rhine. Branches, limbs or the entire tree would die suddenly in various stages of vegetation and at different time of the year. Some of the branches or trees would die over winter, others after leafing out in the spring, others succumbed in summer during or after ripening of the fruit, while still others would not die until autumn. The line between the diseased and normal tissue was generally sharply drawn. Water sprouts would frequently grow from the normal wood just below the diseased area.

Profuse gumming was one of the characteristic indications of the disease. The gum, however, did not always exude, remaining in the affected wood in gaps or fissures, or infiltrating the tissues, causing them to feel soft and spongy.

Frank considered the disease to be of a parasitic nature and due to the fungus *Cytospora rubescens*. The fruiting bodies of the fungus were very constant on the dead cherry branches, and where stroma was absent, the mycelium could generally be demonstrated in the dead bark. The lenticels were considered as the most probable source of infection.

Frank did not consider unfavorable soil, climatic or cultural conditions to have much influence on the disease. The trouble occurred upon all kinds of soil, in varying locations, and as frost did no damage to the easily affected nectarines and apricots growing under the same conditions, as the cherries, he considered frost of secondary importance.

Goethe(34) who also investigated the Rhenish cherry disease, opposed Frank's views. He held the late frost to be the real cause of the injury and regarded the fungus as being of secondary importance. Goethe explained the prevalence of the trouble on the cherry and absence on other members of the stone fruits to the fact that at blossoming time the stone fruits go through various stages of sensibility to frost. The cherries happened to be caught at the most sensitive period.

Sorauer (83) also supported Goethe in his explanation of the Rhenish disease. In explaining the origin of the gum through the agency of the frost, Sorauer believed that by the buds being checked, the plastic substances were prevented from being normally utilized and were eventually transformed into gum. The gumification of the tissues progressed gradually and it was not until large areas of the branches or trunk were affected that it became noticeable.

Labonte (47) in describing the disease of the sweet cherries along the Rhine, ascribed it to the late spring frost of the year. Unfavorable soil, lack of cultural methods, planting the trees too deep and injuries of various kinds all tended to bring about the disease.

Stewart(82) in 1900 was the first American to call attention to the parasitic nature of *Cytospora* on the stone fruits. He found the fungus intimately associated with dead twigs of the peach without any other apparent cause of the trouble being present.

Young peach trees found dead in spring and commonly ascribed to 'winter kill' would often have the affected wood thickly studded with the pimples or fruiting bodies of a fungus which ultimately proved to be the stroma of *Cytospora*. The same fungus was found on the dead trunks of plums and apricots. Several cherry orchards were also observed in which the trees were unhealthy and bore dead limbs and branches which were dotted with *Cytospora* pustules.

Maiden(50 $\frac{1}{2}$) in discussing the gumming of *Acacia penninervis* in Australia mentioned that the gum was obtained in abundance from the trees growing on low wet ground, that the trees growing on higher elevations produced very little or no gum. This fact was found true in other species.

Aderhold (1) in studying the relationship of *Clasterosporium carpophilum* to gummosis of certain members of the stone fruits, made a series of inoculations on the cherry, peach, apricot and plum. These experiments were extended over the growing season and numerous inoculations with an equal number of checks were made on the body and branches. As a result of these inoculations where the fungus was introduced deeply into the wood, gumming was produced in every instance, while the checks healed up. The more shallow inoculations were less productive of results.

Aderhold believed that the gummification of the affected tissues was brought about through the agency of enzymes produced by the hyphae of the fungus. As a result of his investigation, he concluded that the fungus was of the nature of a wound parasite and as such could induce gum flow from the stone fruit trees.

Brezezinski (15) in 1902 published a summary of his investigations concerning the etiology of cankers produced on certain fruit trees. He first isolated an organism from the apple and ^{by} inoculations with pure cultures, proved its pathogenicity. A bacterium was isolated from the peach, plum and apricot where it was found associated with gummosis. This organism was pathogenic in pure cultures but differed in its cultural characteristics ^{with} ~~from~~ the organism isolated from

the apple.

Brezezinski also mentioned finding an organism similar to the one obtained from the peach, associated with gummosis of the cherry, but while cultural characteristics were stated as being different, the difference was not stated.

McAlpine(54) in his diseases of the stone fruits in Australia mentions gummosis as being common. He considered gummosis to be a symptom of a diseased condition and relief could only be sought in the removal of the factors inducing it. He made the general recommendation of: (1) attending to the tillage and fertilizing, using lime especially which reduced gummosis, (2) to avoid deep planting and extremes of wet and drouth when an artificial supply of water is available.

McAlpine stated that gummosis among the cherries was especially abundant after a cold wet spring. He found various fungi associated with gummosis but did not consider them ^{to be} the cause. He states that one grower had apricots on peach stocks that withstood gummosis while those on their own roots died. Another grower reported that cherries did not gum much unless worked on the Mahaleb stock.

McAlpine mentions *Cytospora leucostoma* as occurring on the almond, cherry, peach and plum in Australia.

Paddock(63) described a disease condition of a young^{cherry} orchard of ~~some cherries~~ where large bark wounds were found on the trunks. The white mycelia of a fungus were found beneath the dead bark but the relation of the fungus to the injury was not determined.

Smith (80) in his study of the gums exuded from Australian plants, reached the conclusion that they were pathological products. In making cultures of the tissues, from which gum was exuding ~~of~~ *Acacia binervata*, Smith found two organisms which developed a gum under laboratory conditions and on certain media that behaved to re-agents, produced the same oxidation products and contained the same constituents, arabinan and galactan, as the natural gums.

Smith concluded that the natural gum was a bacterial product, the organisms being of a parasitic nature^{and} obtaining their food from the plant juices, which they elaborated into the gum. Having thus proved the bacterial origin of this variety of gum *acacia*, Smith assumed that all other gums of the arabin group are bacterial products. The two organisms considered the cause of the gummosis of *Acacia binervate* were named *Bacterium acaciae* and *Bacterium metarrbinum*, both being new species. Smith did not mention^{of} experimentally inoculating the host with

with pure cultures of the organism.

Aderhold (1) in 1903 published the results of his re-investigation of the Rhenish cherry disease, his object being to settle the controversy existing among prominent investigators of the disease over the causal organisms or factors associated with the trouble. Aderhold accordingly undertook to prove whether the *Cytospora*, so constantly associated with the diseased trees, was parasitic or not, and also to find out to what extent the late frost and other conditions could aid in causing the disease. Aderhold gave the same general characteristics of the disease as Frank had described in his earlier work. He found the stroma of a fungus almost constantly associated with the cankers and diseased area on the trunks and branches. Often, when the fruiting bodies of the fungus were not apparent, the mycelia could be found in the diseased wood.

Aderhold found that at Camp, where the disease was most prevalent, two varieties of cherries were grown in particular. The Geisepeter, an early variety, was much more affected with the disease than the Kesterter, a late sort.

Aderhold also mentioned that in the Rhenish cherry districts it is customary to plant seedlings, gathered in the nearby forests, which are improved

by grafting the desired varieties on the branches after they have developed into young trees, often being five or more years old before top worked.

In his experiments to establish the positive or negative parasitic nature of the fungus, which proved to be *Valsa leucostoma*, and which Frank in his earlier investigation had found to be the probable cause of the Rhenish disease, Aderhold set out to determine three points: (1) could uninjured trees become infected with the spores of the fungus, (2) would wounds of various kind provide entrance for the fungus, (3) are not certain varieties of cherries more susceptible to fungus attack than others?

From the results of numerous and evidently carefully planned experiments, Aderhold concluded that the fungus could not infect healthy uninjured trees, but if the spores found lodgment in wounds or on diseased parts where it could succeed in getting established sprophytically, it would encroach parasitically upon the bordering normal wood, penetrating both the wood and bark and in time killing the branches or even the entire tree.

In considering the second point of the investigation, Aderhold after careful consideration, excluded all possible sources of injury excepting frost.

That "a cherry tiredness" of the soil was not responsi-

ble was evident from the disease occurring in both old and young orchards on a variety of soils. Root troubles were not considered because of the nature of the disease and its distribution. Although Brezezinski had reported bacteria as causing gummosis in the cherry, Aderhold obtained only negative results from his experiments of this nature. The possibility of other fungi being a cause of the trouble was eliminated because of the constant appearance of Valsa, and only the sporadic association of the other fungi of a more saprophytic nature with the disease.

Frost injury and other climatic conditions that would result in more or less injury to the bark of the cherry, Aderhold considered as being primarily responsible for the Rhenish cherry disease, in that it provided entrance for the Valsa spores which, developing as wound parasites, were responsible for the disease.

Concerning the third point on the investigation, Aderhold found that while the early Geisepeter variety was more susceptible to the injury than other later cherries, the others were also susceptible to attacks of the fungus if wounded through any cause.

Aderhold considered frost injury as being mainly responsible for the primary bark injury, through which the Valsa gained entrance. As a means of com-

batting the disease, he advised the growing of more resistant varieties. The sanitary measure of cutting out and burning all diseased wood as soon as detected was emphasized as of great importance. Ordinary spraying operations appeared to have no effect on the control of the disease.

Trabut (89) described a disease of stone fruits in Algeria caused by *Coryneum beijerincki*. There were two distinct forms of the disease, one attacking the leaves only, the other affecting the young shoots and branches, causing a copious production of gum. The disease was especially noticable on the apricot.

Smith(80) made a study of the gum exuding from the fruit of *Macrozamia spiralis*. Finding bacterial cells in microscopic slides of the gum, Smith made cultures of the gum and obtained a growth of bacteria. The organism produced a gummy slime upon certain culturing media that agreed in all of its physical and chemical properties with the natural gum. Smith concluded that the organism was the cause of the gumming, naming it *Bacterium macropamiae*(n. sp.)and giving the principal cultural characteristics.

Beijerinck and Rant(8) in 1896, working upon the hypothesis that gum flow in the *Amygdalaceae* was caused by external irritation of the cambium, published the results of their experiments with wound stimuli.

In experiments made upon the peach, almond, apricot, plum and cherry, they found that all would respond to wounds by gumming, but that peaches and almonds were generally more responsive than the others.

From their wounding experiments on young green twigs as well as older branches made at intervals during the growing season, they concluded that in the case of the young twigs, when wounded during their development, it was the embryonic woody tissue associated with the regions of greatest cambial activity that gummed the most easily and extensively. The same was true of the newly formed wood in older branches, but so soon as ~~subirrigation~~ ^{suberization} took place, the gum flow ceased. The formation of gum was apparently correlated to the seasonal activities of the cambium.

While not understanding the nature of the stimulus exerted on the cambium by wound irritation, Beijerinck and Rant assumed that it was induced through the dying necrobiotic cells. Necrobiosis is understood as the enzymatic activity within a cell continuing after the death of the protoplasts. Hence they believed that a strong poisonous substance placed in contact with the living cells would provoke abundant gum flow, because of the greater area of cells affected by the poison.

Acting on this hypothesis, they introduced a so-

lution of corrosive sublimate into young growing peach shoots and in older wood. Gumming followed in both cases, being much more abundant than that from the simple wounds made at the same time.

From the result of their experiments Beijerinck and Rand were lead to the view that the degeneration of the embryonic wood cells into gum were due to a cytolytic enzyme that became active the moment necrobiosis set in, and which diffused out into the surrounding tissue and transformed the cell walls into gum. In the case of the gumming following the introduction of poison, the more profuse gumming which followed was due to the comparatively greater number of cells affected, consequently making a larger amount of enzymes available.

Gummosis following an attack of *Coryneum beijerincki*, in which the mycelium of the fungus was also affected with gummy degeneration, was cited as further proof of the enzyme. In this case it was assumed that the enzyme passed out of the cells killed by the fungus and attacked the cells of both the host and parasite and transformed them into gum.

Heald(39) reported an undoubted case of parasitism of the cherry by *Schizophyllum commune*, ordinarily looked upon as a saprophyte. A young cherry orchard five to six years old presented an unthrifty

appearance early in the season, but it was not until June that the external symptoms of the disease appeared, when the sporophores began to develop as minute white specks upon the trunks. By November the fruiting bodies were abundant and well developed upon the trunk and branches. Not a tree in the orchard remained unaffected. It was thought the fungus gained entrance through the roots which were injured by close cultivation.

Heald mentioned later a root rot of the cherry due to some basidiomycete. The trees grafted on Mahalib appeared much more susceptible to the disease than those on their own roots.

Rant(70) treated more in detail the subject which he had developed with Beijerinck. Among other stimuli capable of inducing gummosis, Rant mentioned certain fungi, bacteria, insects, various chemicals and wounds. The fungi mentioned were *Clasterosporium carpophilum*, *Coryneum beijerinckii*, *Cytospora* and *Valsa leucostoma*. The latter were mentioned as the causal organisms inducing the gumming of the cherry.

Rant made a distinction between the gum formed within the cells and that found in the intra-cellular spaces. The latter gum which originated exclusively from the degeneration of the cambial and embryonic woody tissue, formed the gum that exuded from diseased

trees.

Mikosch(56) in 1906, published the results of his investigations concerning the origin of cherry gum. He considered the gum to be a pathological product. The diseased condition of the tree which produced the gum could be induced by any traumatism or irritating stimulus that affected the cambium.

He found cherry gum to be a mixture of two substances, one being soluble in water(arabin) and the other insoluble(cerasin). By the application of alcohol the arabin is formed into a solid, vitreous mass, while the cerasin is thrown down as a whitish flocculent precipitate.

When irritated by any wound stimulus, the cambium produces abnormal thin walled parenchyma cells (gum parenchyma), instead of the normal tissue. An influx of the plastic substances towards the gum parenchyma occurs but is not utilized in the normal thickening of the cell walls and enters into the gum formation.

The gum originates in the living substance of the gum parenchyma cells and is excreted(so to speak) by the plasm as a solution between the epidermal layers and the primary membrane. The process goes on centripetally in the cell, the primary membrane remaining intact until the last stages of gummifica-

tion.

The gum formed from the metamorphosis of the cell membranes corresponds to the cerasin or part insoluble in water while the gum originating from the starch and other cell contents forms the arabin or water soluble part of the cherry gum.

While no enzyme could be proved to be associated with the gum formation, Mikosch considered the presence of such a ferment very probable. He also explained the comparatively large amounts of gum sometimes exuded by the stone fruits, as being due to the abnormally large amount of gum parenchyma formed partly from the cambium and partly from the medullary rays. Through the activity of these tissues new quantities of gum were constantly produced. Under some conditions the cell walls of normal tissue might be affected, but there as in the gum parenchyme, always begins in the secondary membrane and proceeds to the primary membrane.

Aderhold and Ruhland(1 e) described a disease of cherry trees accompanied by gumming, that was caused by bacteria. The disease first became noticeably destructive in 1905. Young nursery trees were chiefly affected, although the trouble also occurred upon the body and branches of older trees.

The disease appeared in mild and severe forms.

In the first case wounds closely resembling the lesions produced by a frost and sunscald were formed, but they generally healed without spreading. In the severe form, the blighted areas were larger and more generalized and the affected limbs and bodies usually succumb. Both forms of the malady were generally accompanied with gumming.

The blight developed most abundantly during March and April, spread during the next two months, but during the summer and fall became more quiescent. Beginning with late winter and early spring, the organisms became very active.

The bacteria isolated from the lesions on the blighted cherry trees were demonstrated by inoculation experiments to be the cause of the disease. Aderhold and Ruhland named the organism *Bacillus spongiosus*. It was described as motile, rod-shaped organism 1.6 to 4 in length and from 0.6 to 0.8 in diameter, bearing polar flagella.

As a remedy and preventive measure, careful cutting out was advised, with the prompt destruction of the diseased tissue. The sterilizing of pruning instruments and wounds was also recommended.

Voglino(93) in 1907 described a disease of the cherry which affected the leaves, twigs and fruit, *Clasterosporium carpophilum* being the causal agent.

A copious flow of gum accompanied the disease but the author did not establish the relationship of the fungus to the gumming.

Ruhland(75) in his discussion of the various theories dealing with the physiology of gum formation in the *Amygdalaceae*, advanced the opinion that gumming was not due directly to the irritative stimuli induced by wounding, but was caused through the agency of the atmospheric oxygen that penetrated to the cambial regions through such wounds.

Ruhland held the oxygen to be the active agent in gum formation as it acted upon the carbohydrates on the cell walls and in the cell contents of the newly formed woody tissues and oxidized them into gum.

Rolfs (76) in 1907, reported the parastism of *Valsa leucostoma* on the twigs, limbs, and trunks of the peach, plum, apricot and cherry.

Through the inoculation experiments, Rolfs proved that *Cytospora rubescens* which appeared first on the affected trees, was the pycnidial stage of *Valsa leucostoma*, and that the fungus was ~~se~~mi-parasitic upon the peach.

Cadoret (18) in his article on the cherry in the valley of the Rhone, classes gummosis along with frost under the head of accidents. He stated that

the gum seemed to result from the transformation and disorganization of the cellulose membranes. Gummosis occurred when accidental wounds irritated the bark. Also every time there was a loss of equilibrium between the absorption of the roots and the evaporation of the leaves. Again, defective grafting might also result in gumming.

Cadoret says the growers there look upon gummosis as being due to an excess of sap. They have noticed that gum is most abundant when heavy rainfall succeeds drouth.

Under remedies, Cadoret advises treatment varied to fit the original cause of the trouble. The gum cankers should be cut out and the wound disinfected. In case of poor soil, fertilizers should be applied. If no treatment at all is given the wounds often extend and result in general necrosis of the tree. From his observations in the Rhone Valley Cadoret believes that the splitting of the bark is the best preventive measure. Although he mentions frost injury, gumming is not mentioned as accompanying shrub traumatism. Of the cryptogamic diseases, *Coryneum* is mentioned as being the worst fungus enemy of the cherry.

Ewert(29) in 1900, from his investigations of the Rhenish cherry disease, believed that the chief

trouble was caused by planting cherry trees in unfavorable environmental conditions.

Boucher (13) in 1908 after commenting upon the gum ferment discovered by Weisner in 1885 and the fact that the presence of such ferments were demonstrated at later times by Reinitzer, Tschirch and Lutz, stated his belief that it is because of the influence of one or more hydrating enzymes that gum is found in living plants.

In his own investigation, Boucher found a soluble splitting ferment in gum that he called amygdaline.

Smith and Butler(81) in their study of the gummosis of the lemon, found a definite relationship of the disease to certain soil conditions. Gum was almost invariably found upon trees growing in poorly drained soil. Allowing the soil to become packed around the base of the trunk, especially when it covered the bud, also favored gummosis.

The effect of gummosis upon the tissues of the lemon was apparently the same as in the stone fruits. The secondary thickening of the walls of the newly formed wood cell is halted and the plastic substances that should be utilized for this purpose are transformed into gum.

For the prevention of gummosis of the lemon, the selection of a well drained soil is emphasized as of

first importance. The use of certain stocks more resistant to the gumming is commended. High budding is favored, although this need not be practiced if care be taken at all times to avoid burying the scion.

For the treatment of affected trees, they recommend first of all the improvement of soil conditions, stating that if this is done in time the tree will recover of itself.

Cutting, slitting and stripping the bark are advised, to facilitate healing.

Scaly bark, foot rot, exanthema and the gumming of citrus nursery stock are other forms of gummosis affecting the citrus trees of California, and their cause and prevention, in the main, is the same as gummosis in the lemon.

Smith and Butler conclude that gumming is the almost constant accompaniment of any injury to or interference with the normal functioning of citrus trees.

Heald(39) in his symptoms of disease in plants states that the cherry and peach suffer more from gummosis than any other of the stone fruits. Gumming may be induced by wounds or it may be the symptom attending the presence of a fungus parasite. It is often due to a deep seated disturbance of the nutritive processes which result in the decomposition of the

tissue. Some of the external factors predisposing to this trouble are heavy soils, high water levels, etc.

Blin (10) in 1909, writing on the gummosis of fruit trees, stated that it had been proved that trees growing on wet soil are more prone to attack.

He mentioned that a solution of acetic acid applied to the diseased parts by soaking a cloth in the solution and wrapping the wound from which the diseased tissues had been removed, appeared to stimulate callus formation and gave good results.

Blin quoted the pomological section of the Nat. Hort. Soc. of France as advising, as a curative measure up to a certain point, the making of longitudinal incisions of the gum blisters. If the wound does not heal, cut out with sharp instruments, wash with Bordeaux and cover with wax. Blin stated that if the trouble is not prevented when it first appears it will spread to other trees in an orchard, causing serious destruction. He concluded by saying that it was necessary to treat the symptoms of disease, the cause of which is unknown.

Sorauer(83) published the results of a general investigation of factors producing gummosis in cherries. He believed that both the young growing and fully matured cells retained a latent ability to form gum. While Sorauer held that gum formation was pre-

ceded by the development of certain parenchymetous tissues within the Xylem and lying between the medullary rays, he did not think that the development of such abnormal tissue was dependent upon wound stimuli.

As an explanation of the gum forming ability of certain tissue, Sorauer advanced the theory that such cells contained substances of the nature of enzymes which became active when certain functions connected with the formation of the cell wall were distributed. The oxidation of the cell membranes under conditions unfavorable for their normal development was also thought to be possible, without the co-operation of enzymes.

Savastano and Majmone(77) in 1909 after discussing the formation of gum in citrus trees, concluded that the gum formation may be due to three distinct factors: (1) A bacteriological agent, probably *Bacterium gummi*, (2) A breaking down of cellular tissue due to the excessive formation of cell sap, (3) to external causes such as wounds.

Stormer(84) in describing the general symptoms, causes and factors associated with the diseases of certain trees in different parts of Europe, stated that the Rhenish cherry disease was caused primarily by unfavorable soil and climatic conditions that

weakened the vitality of the plants, permitting the entrance of parasites like *Valsa leucostoma*.

Muller and Stormer (59) in 1910 treated upon the premature dying of cherry trees which was preceded by a yellowing of the foliage, blighting of the flowers and fruit and a breaking down of the bark of the trunk and limbs accompanied with gumming.

More than one parasite is associated with the disease, Bacteria found in the diseased wood are the primary cause of the disease, while the fungus *Cytospora* was thought to be purely secondary.

Muller and Stormer described the gum flow as originating in abnormal wood tissue produced under the stimulus of wound irritation. As to the actual cause of the gum formation, they believed that normal plants produced cytolytic substances which engage in the vascular and tracheiden formation. The physiological gum thus produced is usually completely reabsorbed, but may sometimes remain in the cells unabsorbed.

Gummosis depends upon an abnormal increase of the effect of those cytolytic substances brought about by the necrobiosis of certain cells. The irritation of parasites may also abnormally increase the amount of cytolytic enzymes.

The authors hold that while *Cytospora*(*Valsa*)

is almost constantly present in every cherry orchard, the fungus cannot attack healthy trees and it is only after the trees are weakened through some other cause that this parasite gets a foot-hold. Unfavorable soil and climatic conditions are the chief cause.

Rolfs(76) in 1910 published the results of his investigations concerning the association of *Valaia leucostoma* with the "winter killing", "cankering" and "sunscald" of the peach. The injury to the larger limbs is often accompanied by a copious flow of gum.

Rolfs found the fungus to be of a semi-parasitic nature and only able to get a foothold on weakened trees. Infection usually occurred at buds, but wound infection could also take place. A spell of warm weather in late winter or early spring followed by a freeze, apparently caused injuries to the peach favoring the development of the fungus. Twigs and branches were often killed back and large limbs severely injured. Infections on the branches may form large cankers, while on the body of the tree the "sunscald" effect is often produced.

The age and physical condition of the tree have a great deal to do with the extent of injury, old neglected trees often being permanently injured while young, vigorously growing trees are only slightly injured. The greatest source of infection is through

frost which injures or weakens the tree.

The semi-parasitic nature of the fungus enables it to live saprophytically for an indefinite time upon the dead tissue of the trunk or branches, where it may become a center for the infection of weakened tissue.

Frost, unfavorable soil, or any other factor that weakens the tree, facilitates the growth of the fungus.

For controlling the disease, fall applications of Lime Sulphur or Bordeaux will reduce the amount of infection, but because of the nature of the fungus, it cannot be controlled by such applications of spray. It is necessary to supplement spraying with the careful cutting out and removal of all infested or diseased tissue in the tree. The prompt burning of such diseased wood is recommended.

The most recent and important contributions to our knowledge concerning the formation of gum in the stone and citrus fruits is given in Butler's thesis (17). From his investigation, in which he made extensive histological studies of gummosis of the lemon Butler reached the conclusion that gummosis in *Prunus* and *Citrus* are ⁱⁿ⁻ distinguishable maladies, being identical in histological development and in their causal

relationship. No species in either genus is entirely immune from the disease and any manifestation of gummosis in one species will be found in replica in the other.

Gummosis is due to a hydrolysis of the cell walls of the embryonic wood cells, which develop into a susceptible tissue. The dissolution of the cell wall begins in the secondary lamella and almost coincidentally, in the primary membrane, the dissolution of the third lamella proceeds centripetally and with its final destruction, the cell contents become a part of the gum mass. The secondary lamella of the wood fibers and that of the vessels, as well as parenchymatous tissue, may show gummosis degeneration in severe cases of gummosis when they are near the zone of active development of the disease.

The cell contents are at no time actively engaged in gum formation and starch plays no part whatsoever in gum formation .

Gummosis may develop autogenously or be induced by all manner of traumatisms, provided they act directly as growth stimulants upon the cambium. Once incited, the simultaneous concurrence of two conditions, one physiological and the other environmental, is necessary for the development of the disease. The cambium must be actively growing and an abundant

supply of water must be available to the roots, either factor being inoperative alone.

Gruss and Sorauer(35), in a review and discussion of gummosis of the Amygdalaceae, claim that the trouble results from an abnormal metabolic assimilation process in consequence of wounds, nutrition disturbances, bacteria, etc., which cause irregularities in the assimilation and nutrition processes.

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THE HOST.

The Cherry. It is necessary to understand something of the botanical relationship of the cherry as well as its principal characteristics and normal behavior under domestication, before taking up the study of the abnormal or diseased condition of certain of the cultivated varieties.

Botanical Relationship. According to de Candolle (19) and later to Bailey(6), the cultivated cherries are descended from two ancestral types, native to Southern Europe and Western Asia. Linnaeus was the first to separate the cultivated cherries into the two groups and he distinguished and named the species. *Prunus avium* is the type from which all sweet cherries have originated while *P. cerasus* embraces all of the sour types of cultivated cherries.

There are other species of more or less horticultural importance because of their value as stocks. *P. Mahaleb*, a native of Southern Europe, where it is found growing wild in the more rocky to sandy soils, is much used by American nurserymen for propagating both the sweet and sour varieties. *P. beasseyi* and *P. pumila* are native to the middle West and have been experimented as stocks to some extent, but apparently without good results. *P. demissa* is native to the Western States and has been successfully utilized as a stock.

Horticultural Grouping. According to Bailey(6), the correct grouping of our cultivated cherries would be as follows:

The sour cherries, or *P. cerasus* group, which are characterized by a diffuse and mostly round-headed growth with a habit of suckering from the roots, bear the flower in clusters from lateral buds which generally precede the leaves. The fruit is always red in color and the flesh soft and sour. *P. cerasus* includes two general types, Amarelles and the Morellos.

P. avium, the sweet cherry group, is characterized by tall growing trees, erect in habit, and having bark that peels off in birch-like rings. The

flowers appear with the leaves on lateral spurs. The fruit is variously colored and shaped, being either hard or soft and generally sweet. The group includes the Mazzards, the Hearts, Bigarreaus, and Dukes. The Mazzards are sweet cherry seedlings, originally native to Eastern Europe and Western Asia, but now naturalized and found growing wild in the Eastern United States. The Mazzards are extensively used by nurserymen for propagating the sweet cherries.

History and Distribution. Horticultural writers agree that the original home of the cultivated cherries was in Asia Minor and its history as such dates back to the beginning of the Christian Era. The Roman general Lucullus is credited with introducing it into Italy from the village of Cerasus in the province of Pontus, in Asia Minor. Pliny wrote that a century later the Romans had at least eight varieties in cultivation and that from Italy they spread all over Europe. de Candolle(19), however, takes exception to this assertion of Pliny's, believing that the cherry had become naturalized at the dawn of Grecian civilization and was growing in Italy before the time of Lucullus.

Whatever may have been the development of the cherry in Europe, it is certain that it was introduced into Eastern American from Western Europe soon after

the first colonization.

The severe winters of the East confined the culture of the cherry to the hardier sour varieties, although the tender sweet sorts were cultivated in a small way in certain protected localities.

As migration moved westward, the favorite fruits of the pioneers followed. Cherry culture in the Northwest began with the arrival of the wagon load of nursery stock brought across the plains by Henderson Luelling in 1847. Since that date there has been a steady development of cherry growing in Oregon, and ^{the} at present time it has attained the rank of an industry.

Distribution. Cherry culture in Oregon is mainly confined to the sweet cherries. The sour types do well, but the perfection the sweet sort attain have eliminated the sour cherry as a commercial fruit and its culture is relegated to the home garden.

Practically all of the sweet cherries thrive in the cherry districts of the State, but the commercial demands have centered upon three or four varieties and the larger orchards already planted, and being planted, contain one or more of these varieties.

The cherry industry in Oregon at the present time is centered in certain localities. The Dalles and Cove districts East of the Cascade Mountains are

noted for their fine cherries, while in the Willamette Valley the cherry industry is centered around Eugene, Salem, Newberg and Portland. There are other districts in the State where the cherry is doing, or will do as well as in the districts named.

Characteristics of the Species and its Varieties.

Since this investigation happens to be concerned with the principal cultivated varieties of *P. avium*, the sour cherries will be mentioned only generally. As the first commercial demand for the cherry was for canning purposes, the industry was mainly devoted to the raising of the Royal Ann and closely related light colored cherries which this trade demanded. Along with the improvement of shipping facilities, however, several native seedlings developed such superior shipping and eating qualities that a demand was created through the middle West and Eastern cities for these superior fruits. Hence the Bing and Lambert are now being planted very extensively to meet the demands of this new trade.

The Royal Ann is an Oregon ~~born~~ for the Napoleon Bigarreau and was thus named by Luelling(42) in 1848. The Bing is a seedling of the Black Republican and originated in the nursery of Seth Luelling in 1875. The Black Republican in turn originated as a seedling of the Black Eagle at Milwaukie, Oregon in 1860. The

Lambert was originated also at Milwaukie, Oregon, but its ancestor is not definitely known.

Culture and Economic Importance. The cherry, like all other plants attains its optimum development under certain climatic, soil and cultural conditions. The districts in Oregon mentioned above, in which cherry growing is centered, are evidently favored by the climatic factors, if this can be judged by the general excellence of their fruit. Hoskins(42) however, believed that the climate of the lower Columbia and Willamette Valley was not exactly adapted to the requirements of the Bigarreau type of cherry, as there was not enough contrast between winter and spring to prevent frost injury in certain seasons.

The cherry trees in these districts are found growing on a variety of soil, ranging from the sandy, gravelly or clayey loams of the valley to the lighter clays of the hills. Vigorous orchards are found in both the valleys and on the slopes, the one important factor in either case is that of adequate drainage. A soil fairly deep, underlaid with a porous subsoil, produces the most vigorous trees.

Cherry trees are planted at distances apart varying from twenty-five to forty feet, depending upon the kind of soil. The rich moist soils of Western Oregon will demand fewer trees to the acre.

One year old nursery budded trees are used by most growers in starting their orchards. Nurserymen have been utilizing both the Mahaleb and Mazzard stocks for propagating but experience has shown that while Mahaleb is the better stock on which to work the sour cherries and has the apparent advantage of thriving on the drier and more unsatisfactory soils, the Mazzard is the best stock on which to propagate the sweet cherries.

In endeavoring to protect the trees from the so called sunscald and gummosis trouble, many growers are planting seedling trees and then top working to the desired varieties. This practice is eliminating the disease from the body and crotch of the tree and is to be commended. Various hardy and resistant stocks may be used for this purpose. The Mazzard is probably utilized the most, but the Morello, May and Late Duke, and one of the native western cherries, P. demissa, can also be used for this purpose. The question of stocks will be treated in more detail elsewhere in this work.

The cultural treatment of the cherry, in the main is, the same as with the other tree fruits. Clean cultivation for the first five or six years is usually employed, stopping the cultivation or irrigation early enough to favor proper maturity of

of the wood. As the trees reach bearing age cover crops should be sown after the last summer cultivation. Winter cover cropping as both the advantage of checking too late fall growth of the wood as well as adding fertility and humus to the soil.

Systematic pruning should be employed until the trees are five or six years old. Low heading as well as severe heading back will form the best foundation skeleton for the future tree. The severe pruning of the younger trees should be done in late winter and early spring. Pinching back is practiced by some during the summer with the object of stopping growth in length or starting laterals. While bearing trees require but little pruning, the removal of unnecessary or injured limbs and branches can be best accomplished just after the fruit is harvested.

Pests and Diseases. In Oregon as elsewhere, the cherry is subject to the attack of various insect and fungus pests. The cherry fruit worm infests the late cherries in the Eastern part of the State. Borers also infest the trunks under some conditions. The larvae of some moths also infest the flower buds of the cherry and may cause misshapen fruit. The San Jose scale infests neglected trees.

Among the cryptogamic pests, hairy-root and crown gall must be looked for in nursery stock. The

cherry is subject to attacks of the mushroom root rot but this trouble is rather limited. The shot hole fungus is common, but only attains the rank of a serious pest during favorable seasons, when it may infest the fruit pedicels, causing small, unsalable fruit.

All of the organisms enumerated above are easily controlled, and none may be ranked as a major pest in Oregon.

The most dreaded disease or diseased condition to which the cherry tree is subject, especially in Western Oregon, is that commonly known as "gummosis". While no section of the State is entirely free from the trouble, it is in the more humid region West of the Cascade Mountains that the malady has reached the nature of a scourge.

The term gummosis originated because of the characteristic exudations of gum that are an accompaniment of certain diseased conditions of the plant. Cherry Gummosis is not a disease, in the strict meaning of that term, but is rather the external sign or symptom of a pathological condition of the tree.

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THE DISEASE.

Gummosis. (Syn. English: gumming, gummosis, gum disease; German: gummifluss, gummikrankheit, gummosis; French: gommose, maladie de la gomme.) A pathological condition of certain plants, characterized by the more or less copious exudation of gummy or mucilagenous substances from fissures in the epidermis of the trunk, branches, fruit spurs, and through the ends of blighted buds.

History and Distribution. A study of the literature dealing with the gummosis of the cherry and related plants indicates that the phenomena of gum flow had probably been noted in the genus *Prunus* since very early times although reference to it as a malady is of comparatively recent origin.

Among the various species of *Prunus* affected with gummosis, it is in the cherry that the exudation is most seriously associated with a diseased condition of the plant. Although the pathological condition, of which the gum flow is a symptom, has been recognized as a malady, either because the varieties grown have been comparative immune or because its culture was scattering, the disease has not been considered important enough to have a history.

Gummosis may occur in both the sweet and sour

varieties of cherries, but the latter as a rule, are much more immune to the trouble. The early history of the disease in Europe is meagre, Trecul, 1860, Weisner 1863, and Frank 1866, being the first careful investigators of the nature of gum flow of the cherry. It is evident from later references that gummosis is associated with the culture of the sweet cherry wherever it is grown. Cadoret(18) in France, McAlpine(54) in Australia, Powell(66) in the Eastern United States, and Wickson(96) and Thornber(88) on the Pacific Coast mentioned gummosis as being a common ailment of the cherry under certain conditions.

The first reference found concerning the malady in the United States was that of Cole(21), 1949, in which he states that the rapid growth of the cherry renders it liable to injury. Extremes of heat and cold may injure the young wood and through bursting of the vessels, cause gum to ooze out, which is very injurious.

Other early horticultural works mention the difficulty experienced in raising cherries in the West and South on account of the sun bursting the bark on the trunk.

The malady appeared in the Pacific Northwest (Oregon) soon after the importation of the first nursery stock(1847). Cardwell(10) reports that as early

as 1853, under certain climatic conditions the cherry would gum profusely.

The distribution of the malady has kept pace with the increase in the cherry industry of this State, the disease being more prevalent some seasons than others, and affecting certain varieties to such an extent that its appearance in an orchard is dreaded.

At the present time, this diseased condition of cherries is the greatest drawback to the industry and the orchard is rare, especially in the humid portion of the State, that has not had from one to fifty percent. of the trees affected.

Gummosis, as a malady, is most severe on such sweet cherries as the Royal Ann, Bing, Lambert, Centennial, Gov. Wood, Waterhouse, and seedlings of the Bigarreau type.

Field Characteristics. Gummosis of the cherry may be general or local, with gradations between the two, sometimes merging one into the other, to an extent that makes it impossible to place under either head.

Generalized gummosis, without any exudation is not uncommon. The cortex of branches, limbs, or trunk, becomes discolored and more or less infiltrated

with gum without any exudation occurring. Under such conditions, part or the entire soft wood of the affected trunk or branches is killed. It is first detected, without cutting through the bark, when the buds fail to open. About the time of leafing out, however, close inspection will sometimes disclose light colored areas beneath the epidermis, upon the removal of which, the diseased tissue is disclosed. It often happens that the leaves on the terminal portions of certain branches will suddenly wilt, the branches being apparently normal. Inspection, however, will disclose that such limbs are girdled by diseased wood.

Generalized gummosis, accompanied by a pustulated epidermis and a more or less copious exudation is perhaps the most common form of the disease. Examinations of such gum masses will show their associations with irregular shaped areas of discolored tissue upon the body or branches of the tree. The gum blisters and exudate may form either in connection with the fruit spurs or anywhere in between.

In both the above mentioned generalized forms it is difficult to determine any point of origin of the disease. The malady either progresses through the tissue during the dormant period of the plant or the entire mass of tissue becomes affected at the same

time, as the diseased condition is often not noted until the active state is resumed, when the exudate, lack of bud development, or wilting of the leaves indicates the trouble.

Localized gummosis accompanied by a pustulated epidermis or an exudation is perhaps the most common form of this malady. Limited areas of tissue, either on the body, at the crotch or on the limbs become diseased and become noticeable because of the blisters or the exudate. The localized areas on the body or branches can generally be traced to a blighted bud or fruit spur. So common is this relation that it suggests to one the possibility of such buds or spurs being the possible source of infection by pathogenic organisms.

Another localized condition apparently associated with gummosis but with which ^{an} ~~no~~ exudation ^{does not always} appears is characterized by a blighting of the buds and fruit spurs. A distinction must be made between this form and the ordinary 'self-pruning' action of the plant in which through competition, or other causes, certain lateral buds and spurs upon the limbs and branches fail to develop and eventually die. Examination shows that the majority of such buds and spurs are cut off, so to speak, by a layer of corky tissue at their basal end.

In the apparently diseased condition of the buds and fruit spurs, gradations of the trouble may be found ranging from spurs in which the terminal buds or ends are discolored and dead to those where the diseased area extends for various distances down the spur, or into the tissue of the branch or limb adjoining the basal end.

What adds significance to this blighting of the buds and fruit spurs is the fact that the buds apparently form normally in fall, but sometimes during the winter or early spring they become affected and are conspicuous by their non-development when the neighboring buds or spurs are unfolding. Such spurs and buds may be scattered indiscriminately throughout a tree or be confined to one or more branches.

Economic Importance. A study of the literature relating to gummosis indicates that while gummosis in some form has apparently always affected the cherry, wherever grown, its destructive propensities were most appreciated in those districts where the industry has become intensified. In the Pacific Northwest, especially in the humid regions of Western Oregon and Washington, gummosis is the one serious drawback to the industry. From the fact that two of the three principal commercial varieties are very subject to the disease, an investigation of the causal factors,

to the end that the proper control measures may be intelligently applied, is certainly pertinent.

The last report of the President of the State Board of Horticulture conservatively estimates the cherry crop of Oregon as being valued at \$200,000. No estimate regarding the cherry acreage in the State is available but it is certain that it is large and is constantly increasing.

Pathological Morphology. As mentioned under field characteristics, in both the generalized and localized forms, accompanied with, or without the exudation, the affected tissues are discolored, dead and usually infiltrated with the gum. They may feel spongy, but not necessarily so. The point of egress of the exudate is often through the tips of blighted buds or through lesions in the fruit spurs or cracks in the epidermis, of the bark on the branches or trunk. The exudate from the buds and fruit spurs may assume the form of filaments and be more or less curled. Such filaments appear to have been forced out under considerable pressure. Again, the exudate assumes the form of drops or small irregular shaped masses.

When the exudation is associated with larger areas of affected tissue, it may swell up from fissures in the epidermis of the trunk or limbs and

form great masses of gum. When exuded during rainy weather such gum masses are usually washed off.

Gummosis may have affected a large area of tissue without any external symptoms appearing. Upon cutting or stripping the birch-like epidermis, the bark tissue, as far as the wood, is discolored and more or less infiltrated with the gum. Upon such diseased wood various fungi may appear. Some are undoubtedly saprophytes, while others, of which we shall speak later, appear to have a more parasitic nature.

Pathological Histology. The histology of tissues affected with gummosis has been studied, as we have seen, at intervals for fifty years. The writer has not conducted any investigation of this nature and under this head he will give the results of the most recent investigation concerning the histology of gummosis. Butler after a comparative study of gummosis in *Prunus* and *Citrus* reached the conclusion that within both genera, the origin and development of the gum were identical.

Butler found that when gummosis was not sufficiently intense to encircle the stem, the pathognomonic tissues were fusiform in outline. Gum formation was most abundant at the centers of such spaces, but decreased as the apices were approached. The tissues at the center of the sickle have been longest affected

by the disease and may have reached the ultimate stage of decomposition, while those nearer the apices show only the initial stages of degeneration.

Butler observed that at the beginning of gummosis, the cambium formed cells rich in protoplasmic contents, although this increased cell content was not an invariable accompaniment of gummosis, its appearance in the embryonic wood tissues being essentially an indication of active metabolism. The cell contents do not play any part in gum formation except to swell the total volume of the gum to the last stages of the disease. The transformation of the gum originates through the cell walls of the abnormally formed young wood, which are the seats of gum formation. In his study of the cambial tissue, Butler found it impossible to separate the normal young wood from those susceptible to gummosis, without the aid of reagents, both being enframed between the cambium, xylem, and medullary rays. With the aid of progressive weak solutions of alcohol, a change could be detected in the cell walls, although the cell contents remained the same. As the alcohol became more watery a stretching of the cell walls was observed and the secondary membranes began to swell. This, Butler considered as the initial stage of gummosis. This was followed by a marked swelling of the primary membrane,

which became indiffentiable, both membranes increasing in bulk, and becoming semi-fluid. Gummy degeneration proceeds centripetally and upon the dissolving of the third lamella, the cell contents become a part of the general mass, which occupies the space formerly filled with the affected cells, and a gum pocket results.

The gum pockets enlarge by the sloughing off of the cells bordering the cavity through the degeneration of their cell walls and the process may continue until all of the embryonic wood cells are destroyed. The size of the pockets are thus limited to the amount of susceptible tissue developed by the cambium, although in rare instances the cambium, medullary rays and xylem may be affected.

The development of the gum pocket may be halted before all the cells predisposed to gummosis are affected. Such an arrestation may take place at any time during their development and is followed by a lignification of the cells bordering the cavities as well as the balance of the susceptible tissue. But such thickening of the cell walls is never as complete as in normal xylem.

A brief summary of Butler's study of the histology of gummosis indicates the following: A susceptible tissue, in the form of embryonic wood cells, is

formed by the cambium. The secondary and primary lamellae of certain of these cells gelatinize and the cells become detached from each other. Upon the gelatinization of the third lamellae, the cells and their contents become part of the gum mass, filling the cavities thus formed. If the process of gummification is halted before all of the susceptible tissue is affected the remaining cells become more or less lignified. The cambium may form normal woody tissue again and the gum pockets become enclosed by normal xylem.

Pathological Physiology. Accepting Butler's explanation of the histological origin of the cherry gum, we come to the more difficult question of why and under what conditions is such susceptible xylem formed by the cambium.

That the gum is a pathological product is admitted by all. That all cherry trees, even of the same varieties and growing under the same conditions do not gum is a common observation. The gum is not formed through normal functioning of the plant and since it is the result of a diseased condition, we must scrutinize every feature of environment that might irritate or predispose the plant to disease.

Frank(31)thought the gum to be of physiological significance in that it protected the normal tis-

sues adjacent to the diseased cells by infiltrating the boundry cells, thus establishing a limit to the disease. But from the fact that the pressure sometimes generated in forcing the gum to the exterior may cause large areas of normal tissue to become permeated with the gum, such tissue eventually dying, it is improbable that any protective influence is rendered by the gum.

~~Physiological~~ Etiology. Autogenous Development.

Gummosis often appears to be self induced as no traumatic or active external stimulus can be found associated with it. But since it is known that the gum is a pathological product, having its origin in abnormally formed embryonic xylem, the cause of the apparent autogenous formation of gum must be sought for in those non-traumatic factors of environment that may effect the growth of the plant.

Relation of Growth to Gummosis. From the study of the literature concerning gummosis, we found the investigators of the histological origin of the same agreed that the gum has its origin in certain embryonic growing tissues of the plant. Without conditions being such as to favor the activity of the cambial tissue, no gummosis could occur. Growth having started, there must be the co-operative action of some stimulus to induce the formation of the abnormal

xylem.

From our present state of knowledge concerning the relation of the tree to the soil, we may assume that a non-traumatic stimulus capable of inducing the autogenous development of gummosis, if such there be, can only come through the influence of the food or water content of the soil.

Too rich a soil may induce rank growth, and the consequent formation of large amounts of susceptible tissue. The water relation may also influence the formation of defective wood, but the exact nature of the stimulus necessary to induce the formation of susceptible tissue, is not clear.

Butler (17) maintains that, whatever may be the inducing factor, before gummosis can develop, two conditions must occur simultaneously. First, the cambium must be actively forming new tissue, and secondly, there must be a superabundance of water in the soil. Cambial activity is necessary to the formation of the susceptible tissue and the importance of the water relation is capital when it is considered that the gum is formed by the hydrolysis of the cell walls and unless there is sufficient water the liquidation is incomplete.

The Relation of Non-Traumatic Stimuli to Gummosis.

Whatever may be the relation of the soil-water con-

tent to gummosis, most horticultural writers are agreed that excessive soil moisture favors the development of the disease. Many growers state their experience to the effect that trees on heavy, retentive soils are much more susceptible to gummosis than those situated so as to have proper drainage.

It has been the writer's observation that trees standing in hollows or receiving the seepage of nearby hills are often more badly diseased than the others in the same orchard on higher soil. But the fact that when the disease is prevalent, it can be found scattered throughout an orchard, the trees on the ridges and slopes being affected as well as those on the lower levels, and growing in sandy and gravelly loams, underlaid with a gravelly or otherwise porous subsoil, would tend to make the excessive soil moisture theory untenable in explaining the appearance of the disease on trees in such apparently favored locations.

Butler, however, in his study of the gummosis in Citrus, found that gumming of the lemon was always accompanied by an excess of soil moisture, either in the form of imperfect drainage or over irrigation or both.

Enzymatic Development. That the hydrolysis of the cell walls in the initial stages of gum formation was brought about by enzyme action has been believed

by many investigators. Wiesner(97) in 1885, described a ferment which he considered active in the transformation of cellulose into gum. Beijerinck(8) the following year advanced the theory that the gum flow following attacks of *Coryneum* was the result of cytolytic enzymes set free by necrobiotic cells. Garros(33), Lutz(50), and Boucher(13) and others have reported the presence of ferments in gums and have ascribed the formation of gum in plants to enzyme action.

Beijerinck and Rant(8) were perhaps the most prominent supporters of the enzyme theory. From their investigation regarding the effect of various wound stimuli upon the formation of gum in the stone fruits, they reached the conclusion that any stimulus, whether caused by direct wounding, chemical substances, or the attacks of parasitic organisms, capable of inducing necrobiosis, would result in the liberation of cytolytic enzymes from the affected cells that would attack the cell walls of the surrounding tissue and transform them into gum. It was only after the cells had been injured to the extent of inducing necrobiosis that the gum formation could begin.

Ruhland(75) and Soreauer(83) believed that the formation of the gum could be explained by the ac-

tivity of atmospheric oxygen which, gaining entrance to the tissues through wounds, would attack certain carbohydrates in the cell membranes and oxidize them into gum.

Butler(17) from his histological study of the origin of the gum, holds both the enzymatic and oxidation theories untenable. The enzyme theory was not consistent because there could be only a lateral or one-sided excretion of the cytase to bring about the fusiform outline of the gum pockets. Again, if an enzyme were excreted from the protoplasm, it would have to permeate the third lamella and would be acting upon it while hydrolyzing the secondary membranes, while microscopic observation indicates that gummosis degeneration proceeds centripetally in the cell walls. The oxidation theory was opposed with the fact that chemical analysis shows the gums to not be oxidation products of the carbohydrates.

Varietal Immunity and Susceptibility. Gummosis appears much more abundantly in certain varieties of cherries than in others, even when growing under the same conditions. The Napoleon Bigarreau in the humid regions of Western Oregon is without doubt the most susceptible variety of sweet cherry grown. The Bing is nearly as much affected, and the Lambert appears to be the most resistant of the three.

The fact that gummosis may appear only on certain trees in an orchard, the unaffected trees growing under the same soil and climatic conditions, makes it difficult to hold soil and climatic stimuli as being the sole factors inducing gumming unless there is some individual resistance to account for it.

Regarding the varietal susceptibility to gummosis, Hoskins(42)believed the climatic conditions prevailing in the lower Columbia and Willamette River Valleys to be disadvantageous to the Bigarreau type of cherry on account of there not being enough contrast between winter and spring.

Resistant Stocks. For propagating the sweet cherry, two stocks have chiefly used. The Mahaleb, or St. Lucie, is used principally as a stock for budding the sour cherries, but some nurserymen prefer it for the sweet varieties also, finding it hardier where the winters are sever. The budding season being longer and the nursery stock less subject to leaf blight also favor its use by many nurserymen.

In California and the Northwest, the majority of the nurserymen use Mazzard stock for the sweet varieties, and the experience of cherry growers in Oregon all tend to indicate that the Mazzard worked trees are uniformly more vigorous and longer lived.

Wickson(96) states that the cherry growers of California, after fifty years' experience, are satisfied that the Mazzard is the better stock for the sweet cherries. Wickson also advises the double grafting of certain resistant stocks, when the variety desired does not do well when grafted direct. He mentions the Morello seedling as a case in point, as it proves unhospitable to the buds of varieties like the Early Purple Cragne and the Royal Ann. As the Black Tarterian takes well on the Morello root and the other varieties show affinity for the Tartarian, such double grafting may prove serviceable. Another case of double working apparently giving good results is the top working of the Governor Wood, which is budded into Mazzard roots, the other sweet cherries taking well on the Gov. Wood. One prominent California nurseryman, though, states that when the sweet cherry is to be planted on soil undergoing the extremes of wet and dry conditions those on the Mahaleb do the best.

Thornber(88)states that the Mahaleb makes the best stock for the sour cherries, but, while commonly used for the sweet varieties, is not satisfactory.

In Western Oregon and Washington, where the trunks of the young trees suffer most severely from "sunscald" and "gummosis", the growers are finding it

advantageous to top work those varieties most susceptible to the malady to more hardy and resistant stocks. Since the wood of the Royal Ann, the Bing, and a few other sweet cherries is so susceptible to the disease it is to be expected that when gummosis appears on the bodies of root grafted or budded trees it is very apt to result in the partial or complete girdling of the trunks. The diseased condition of the bodies of young trees is dreaded and it results in the loss or weakened condition of many trees before they reach bearing age.

In a young cherry orchard planted on the Station Experimental grounds it was found this season that of the _____ two-year old root budded trees, 17% were diseased. The exudate and cankered tissue were confined mainly to the South and Southwest sides of the trees. In older orchards, in various portions of the State many of the trees that have survived, bear large wounds on the Southwest portions of the trunk. The May and Late Duke, Morella, and the native seedling P. demisa have all been utilized for this purpose but at the present time by far the greater number are top worked on the Mazzard, which is proving very satisfactory.

A few growers in using resistant stocks, top graft the body of the seedling. This method is

objectionable from the fact that gummosis often appears in the crotch of the tree, partially or wholly girdling one or more of the limbs. A significant fact also associated with many body grafted trees is that the disease area often extends only to or slightly below the graft union.

The advantage in top working the seedlings is obvious. The Mazzard has proved to be very resistant to gummosis and planting the seedlings, which are comparatively cheap, and then top working (preferably by budding) when they are three to five years old removes the gummosis factor from the trunk and crotch.

An added advantage in top working the Mazzard or other resistant seedlings is the opportunity it gives the grower to select his buds or scions from vigorous, heavy producing trees. One instance was reported to the writer by a nurseryman who in propagating his choicest sweet cherries, selected the buds from a certain old and very vigorous tree. Running short of budding sticks one season he had to obtain some from a tree affected with gummosis. Of the trees resulting from the scions from the two trees, those from the healthy tree remained very free from gummosis while those from the diseased trees soon became affected and many died.

While, of course other conditions may have influenced the results in this instance, we cannot but believe that the present indiscriminate method of selecting buds and scions is partly responsible for many unsatisfactory trees.

Specific Causes. Traumatisms. The association of frost injury with cherry gummosis has been noted by ^{many} investigators. An early American horticultural writer, Cole(21) ascribed gumming of the cherry to bursting of the vessels, following extremes of heat and cold.

Goethe(34), Sorauer(83) and Labonte(47) considered frost injury as being the cause of the Rhenish cherry disease, which, from their description, resembles the disease condition in Oregon very closely. Aderhold(1 d), however, who made the most complete investigation of the Rhenish disease, held that frost injury was responsible only to the extent of weakening the trees and making them susceptible to the attacks of certain fungi, which were the active cause of the disease.

Among the horticultural authorities in the Northwest, we may quote Cardwell(30), Hedrick(41), Hoskins(42), and Thornber(88) as ascribing cherry gummosis to the injury produced by frost. While none of those mentioned investigated the histological

origin of the gum through frost injury, all had noticed that gummosis was much more prevalent in those seasons in which freezing weather followed warm spells during later winter and early spring/

The appearance of "sun scald" wounds upon the South and West portions of the trunks of cherries has also suggested the relationship of solar heat to certain phases of the disease. As before mentioned, this sun scald effect is most prevalent upon the bodies of young high headed, root budded or grafted trees.

Wounds induced by pruning, cultivating, boring insects, etc. may cause gum to exude. In such cases the injury is purely localized, and the exudate is closely associated with the lesion.

The introduction of various chemicals experimentally as strong acids, corrosive sublimate, etc. has been followed by gum flow.

Causal Organisms. Insects. While the cherry is subject to attack by various insects, in Oregon only two have been found closely associated with gummosis. The cherry, especially in Eastern sections of the State is subject to attacks by the borer similar to that infesting the peach and prune. The gumming is localized in such instances, being associated with the burrows of the insect.

In the case of the other insect, the "San Jose Scale", the trouble assumes a more generalized form and the other conditions of neglect usually co-existent in orchards infested with scale probably contribute as much to the diseased condition of the trees, as the insect.

Fungi. Several different fungi are commonly found on cherry trees that have been affected with gummosis. Two or three, especially, although commonly looked upon as saprophytes or at the most as wound parasites, are so abundantly and constantly associated with gummosis stricken trees as to warrant special mention.

The first is the common shelf fungus, *Schizorhynchium commune* which is often found growing out of gummosis cankers. The fact that it usually does not make its appearance until the second season after the disease appeared has caused it to be regarded purely as a saprophytic. An undoubted case of parasitism of the cherry by this fungus, though, reported by Heald (39) who found the fungus infesting young cherry trees in Nebraska, demands that a special investigation be made concerning its relation to the diseased cherry trees in Oregon.

The mushroom root rot, caused by species of *Armillaria* is also found on some of the cherry trees.

The trees infested with the root rot usually succumb without exuding much gum, although it is possible that in the first stages of the disease the gum appears and is ascribed to other causes. Woodworth(99) reported gummosis as being prevalent upon trees affected with root rot in California but the fungus in question was not specified.

A fungus of greater significance, because of its proven parasitism on the cherry in Germany, is *Valsa leucostoma*. What appears to be the pycnidial ~~or~~ Cytospora stage, is often associated with diseased cherry trees in the Willamette Valley. An investigation of the relationship of this fungus to gummosis affected trees is now being made.

Valsa is of wide distribution, being reported as occurring on the cherry and other stone fruits in many sections of the United States, Germany, Italy, and Australia. It was long looked upon as a saprophyte and regarded as being able to develop only on the dead or dying wood of trees injured through other causes.

Frank(3 c) was the first to call attention to the probable parasitic nature of the fungus. In his investigation of the Rhenish cherry disease he found the fungus almost constantly associated with the disease. The Rhenish disease was popularly ascribed to frost

injury. The fungus was apparently overlooked by the other investigators. Frank considered its constant appearance as being of more significance than an ordinary saprophyte, and by producing the disease through direct inoculation experiments, he believed to have confirmed his suspicions regarding its at least semi-parasitic nature.

Aderhold(1 d) later made a more searching investigation regarding the Rhenish cherry disease in order to settle the conflicting theories regarding the cause. He found the stroma of fungus almost constantly associated with the disease and in many of the diseased trees not showing the fruiting bodies, the mycelia could be demonstrated in the diseased wood. The pyrenidial form of the fungus which was most common and appeared first, was found to be the *Cytospora* stage of *Valsa leucostoma*. As a result of his investigations, Aderhold concluded that the spores of *Valsa leucostoma* while not able to infest the wood of normal, uninjured trees could, through the action of frost or any other stimuli capable of inducing injuries or lesions in the bark, get established saprophytically, after which the fungus would encroach parasitically upon the normal wood, penetrating both the wood and bark and in time killing the branches or even the entire tree.

Stewart(82)was the first American to call attention to the probable parasitic nature of *Cytospora* on the stone fruits. He found the fungus so constantly associated with dead and dying peach trees that he believed it to be semi-parasitic, at least. He also found what he thought to be the same fungus on apricot and plum.

Rolfs(76)recently proved conclusively the semi-parasitic nature of *Cytospora* and its perfect stage *Valsa leucostoma*, by his investigation of the relation of the fungus to the "winter killing", "cankering", and "sun scald" of the peach tree. The trees weakened from any cause, especially those which have been winter injured, suffer severely from the disease. Infection of the twigs and branches, usually occurring in connection with buds, form rough cankerous wounds, while the infections of the larger limbs and trunks form larger areas of dead tissue known as "sun scald".

Rolfs found that the physical condition of the tree had a marked influence upon the amount of injury done by the fungus. Young and vigorously growing trees were not much injured, but on trees weakened by neglect, frost or unfavorable soil and cultural conditions, were most readily attacked, and succumbed quickest.

Since the semi-parasitic nature of *Valsa leucostoma* and its conidial form, *Cytospora* has been established on both the cherry and peach, elsewhere its rather abundant appearance upon diseased cherry trees in this state will merit special investigation. This the writer has been unable to do since his time has been largely devoted to a bacteriological aspect of the disease.

Bacteria. Bacteria have been found associated with the gum exuding from some plants and the investigators looked upon them as the causal agent. Smith (80) in studying the gums exuding from certain Australian plants, reached the conclusion that they were pathological products. In making cultures of the tissues of *Acacia binervata* from which gum was exuding, he isolated to organisms. Both developed a gum on certain laboratory media which contained the same constituents and gave the same chemical reactions as the natural gums. Because of the resemblance of the laboratory product to the natural gums, Smith assumed that the natural gums were a product of the organisms which lived parasitically in the tree. He did not mention establishing the pathogenicity of the organisms by inoculating with pure cultures.

Smith (80 b) later conducted similar experiments with an organism found associated with the exudation

or *Macrozamia spiralis* and obtained similar results.

Brezezinski(15) was the first to report bacteria as causing gummosis in the stone fruits. He isolated a bacterium from gum cankers on the peach, plum, and apricot, which proved to be pathogenic in pure cultures. He also reported finding an organism associated with the gummosis of the cherry, but while the cultural characteristics were stated as being different, a description of neither organism was given.

Aderhold and Ruhland(16) in 1905) described briefly, and later treated more fully upon a disease of cherry trees, accompanied by gumming, caused by bacteria. Young nursery trees were chiefly affected although the disease appeared on older trees.

The disease appeared in both mild and severe forms, the first mentioned closely resembling the wounds produced by frost. The other form was more generalized, the blighted areas being larger and the affected trunks and limbs usually succumb.

The organism was described as motile, rod-shaped in form and bore polar flagellae. It was named *Bacillus spongiosus*(n. sp.) by the authors.

The writer found an organism associated with a blighting of the buds and fruit spurs of the cherry in March, 1909. Although thinking they were

of a purely saprophytic nature, the blighted spurs from a number of different trees were examined and smear and broth tissue cultures indicated the organism was fairly constant. After calling the attention of the head of the department and several of the assistants to the presence of the bacteria in the blighted spurs, the writer, who was at that time a student in Bacteriology under Professor E.F. Pernot, called the latter's attention to the germs. At his suggestion, and with his help, more material was brought into the laboratory and fresh tissue cultures were made. Some of the 'blind' cultures thus made were injected into a few apparently normal fruit spurs by Professor Pernot, with the writer assisting. No checks were made. In the course of four or five weeks the spurs blighted and were taken to the bacteriology laboratory. Stained smears from discolored areas in the spurs showed the presence of the germs.

On account of the pressure of other work at that time, further work on this bacteriological phase of gummosis was dropped. Professor Pernot did some independent work on the problem, but to the writer's knowledge, the results have not been published.

The study of the relation of the bacteria to

the fruit spurs was renewed in the spring of 1910, as soon as the blighting appeared. A large number of tissue cultures were made from material obtained at different places. Upon plating out, the organisms appeared almost constantly in pure cultures. A series of inoculations were made as follows:

Feb. 24, 1910. Branch from Royal Ann cherry tree containing blighted fruit spurs taken to the laboratory.

Tissue cultures were taken from the blighted spurs by first making a horizontal cut, and searing the surface thus exposed with a hot knife. With a flamed spear-pointed needle a deep incision was made and tissue beneath the seared surface transferred to a tube of broth.

After inoculating 24 hours, at 37°, agar plate dilution cultures were made from certain of the tubes, the original tissue cultures being used for inoculating normal spurs.

Feb. 26. One of the broth tissue cultures made 2/24/10, was used for inoculating apparently normal fruit spurs on a Bing tree in the college orchard. Fourteen spurs on a certain limb were punctured with a flame needle. Each alternate spur thus punctured was inoculated with a needle transfer of the fluid culture known as D-1.

March 1, 1911. The agar plate sub-culture from D-1 was an apparently pure culture of scattering colonies.

April 5, 1910. Of the 14 fruit spurs in the experiment the seven inoculated spurs were blighted with some gumming. The seven check spurs were apparently normal and were leafing out.

April 7, 1910. Broth tissue cultures from the blighted spurs inoculated with D-1 were made. Incubated 24 hours.

April 8, 1910. Agar Plate sub-cultures made from the broth culture.

April 11. The Agar plates contained scattering colonies all alike macroscopically.

April 12. Broth sub-culture made from typical colony on agar plate. Incubated 24 hours at 37°C. marked as colony A.

April 13. Broth culture used for inoculation, first making an agar plate dilution culture as a check.

16 spurs on a branch of Bing cherry tree punctured with a flamed needle. Each alternate spur thus punctured was inoculated with a platinum needle carrying fluid culture of culture A. 8 check spurs and 8 inoculated.

April 16. Agar plate dilution culture (check on

culture A) contains a few scattering colonies similar to those of April 11. Pure culture.

April 26. No signs of fruit spurs blighting.

May 7. The eight inoculated spurs blighted. Some gumming. The checks apparently normal.

Owing to stress of other station work and the confusion incident to moving the laboratory, the cultural characteristics of the organisms were not obtained.

Season of 1911.

As soon as the buds began to swell so that a difference might be noted between the normal and blighted buds and spurs, tissue cultures were made from the diseased wood.

March 17, 1911. Numerous blighted buds and spurs found on Bing tree near Campus were brought to the laboratory and placed in water.

March 18, 1911. After sponging the blighted spurs with a solution of corrosive sublimate (1-1000), the spurs were cut back to where normal colored wood adjoined the discolored area. After searing the surface with a flamed knife small sections of the diseased tissue bordering on the normal wood were transferred on a sterile needle to tubes of broth. After allowing to stand in the broth about 30 minutes to

allow all organisms present to diffuse out, occasionally shaking the tubes, a loop transfer from broth to melted agar was made. After shaking the tubed agar the contents were poured into petri dishes. The usual methods and care to insure non-contamination were used. The cultures from these specimens were called the B series. All cultures were incubated at 30°C.

- Tube B-1. Tissue culture of discolored wood of branch at the base of a blighted spur.
- Tube B-2. Tissue culture of diseased wood at base of blighted spur.
- Tube B-3. Tissue culture of diseased wood at base of blighted spur.
- Tube B-4. Tissue culture from the interior of a spur, all the wood of which was discolored.
- Tube B-5. Tissue culture from heart of blighted spur.

March 20. Agar plates developing colonies and broth cultures cloudy with exception of B-5, which produced no growth.

- B-1. Agar Plate colonies abundant, apparently a pure culture. Broth cloudy.
- B-2. Agar plate colonies abundant, apparently a pure culture. Broth cloudy.
- B-3. Agar plate, colonies scattering, pure culture. Broth cloudy.
- B-4. Agar plate, colonies scattering, pure culture. Broth cloudy.
- B-5. Agar plate, no growth. Broth not cloudy.

March 23. A typical colony in Plate B-3 marked. A broth sub-culture of this colony was made. Labeled as B-3, and incubated at 30° for 48 hours.

March 25. After making a broth sub-culture, and an agar plate dilution culture of B-3, this fluid culture was used for inoculating part of the fruit spurs on a certain branch of a healthy Bing cherry in the college orchard.

All the fruit spurs on a certain branch reserved for the experiment. Entire branch sponged with a solution(1-1000)of corrosive sublimate and allowed to dry. Every spur on this branch was then punctured with a sterile needle, care being taken to avoid making too large a wound. The larger spurs were punctured about the middle in length and at right angles to their axis. Each alternate spur, beginning with the second from the basal end of branch was inoculated with a needle transfer of the fluid culture. Sixteen spurs, eight inoculated; eight checks.

March 28. Plate subculture from B-3 developing scattering colonies. Apparently a pure culture.

April 17, 1911. All of the inoculated spurs were apparently blighting. Checks spurs apparently normal.

April 24. The eight inoculated spurs blighted, seven of which gummed. Two of the check spurs were apparently "frost nipped", the leaves being brown but not wilting and the spurs were not gumming. The

other checks were apparently normal, with one exception.

- Spur 1. Check. No gumming. Leaves not wilted.
- Spur 2. Inoculated. Gumming. Leaves dead. Tissue discolored above and below line of puncture.
- Spur 3. Check. Gumming. Leaves wilted.
- Spur 4. Inoculated. No gum. Spur blighted and diseased area running into branch.
- Spur 5. Check. No gum. Spur apparently normal.
- Spur 6. Inoculated. Gumming. Wood discolored above and below puncture.
- Spur 7. Check. Apparently normal.
- Spur 8. Inoculated. Gumming. Entire spur affected.
- Spur 9. Check. Apparently normal.
- Spur 10. Inoculated. Spur blighted with gum welling out of puncture.
- Spur 11. Check. Apparently normal.
- Spur 12. Inoculated. Gumming. Discolored area extending above and below puncture.
- Spur 13. Check. Apparently normal. Leaves browned. Frost.
- Spur 14. Inoculated. Gumming. Entire spur discolored, extending into branch.
- Spur 15. Check. Apparently normal. Leaves browned. Frost?
- Spur 16. Inoculated. Gumming. Discolored area extending above and below puncture.

April 24. Broth tissue cultures and Agar Plate dilution cultures made from the diseased spurs.

The spurs from which all leaves and debris were removed were sponged with a corrosive sublimate solution and allowed to dry. The spurs were sectioned so as to reach discolored tissue as far above and below the line of puncture as possible. The surface thus exposed was seared with a hot knife. With a flamed needle, sections of diseased tissue adjoining

normal colored wood were transferred to tubes of broth. The sections in broth were allowed to stand about 30 minutes, shaking occasionally, to allow all possible organisms to diffuse out.

A loop of broth was then transferred to tubed liquid agar and the plates poured. Each plate and tube number corresponds to a blighted spur. Blighted spurs 14 and 16 were not used, being removed for histological examination. Cultures incubated 48 hours at 30°C.

- B3-1 broth tissue culture agar plate dilution culture from spur 2.
- B3-1 broth tissue culture agar plate dilution culture from spur 2.
- B3-2 broth tissue culture and agar plate dilution culture of spur 3.
- B3-3 broth tissue culture and agar plate dilution culture of spur 4.
- B3-4 broth tissue culture and agar plate dilution culture of spur 6.
- B3-5 broth tissue culture and agar plate dilution culture of spur 8.
- B3-6 broth tissue culture and agar plate dilution culture of spur 10.
- B3-7 broth tissue culture and agar plate dilution culture of spur 12.

March 27. Broth cultures all cloudy. Agar Plate cultures all developing colonies.

- B3-1 Pure culture. Typical B 3 colonies.
- B3-2 Mixed culture. Rapid surface growing organism taking plate. Scattering colonies of B 3.
- B 3-3 Pure culture. Typical B 3 colonies.
- B 3-4 Pure culture. Typical B 3 colonies.
- B 3-5 Mixed culture. Surface spreading organisms and B 3. B 3 predominating.

B 3-6 Mixed culture, B 3 predominating.
B 3-7 Plate dense with B 3.

April 27. Broth sub-cultures made from agar plates 1, 2 and 7. A typical D 3 colony in plates 1 and 2 was marked and needle transfers to broth made. It being impossible to distinguish between the punctiform colonies in plate 7, a composite culture by transferring a section of the agar to broth was made. On account of other work which was pressing, these broth sub-cultures were placed in the cold incubator (20°C) and allowed to remain until May 10.

May 10. Broth subcultures 1, 2, and 7 and parent culture B 3, rejuvenated by transferring to broth and incubating for 24 hours at 30°C on each of three successive days.

May 14. Broth subculture and agar plate dilution cultures of B 3-1, B 3-2, B 3-7 and B 3 made from the 5/12 cultures, the former being checks on the 5/12 cultures which are to be used for re-inoculation.

May 15. Certain healthy branches on a Royal Ann tree in college orchard tagged for experiment. All the spurs and intervening wood on these branches sponged with a solution of corrosive sublimate and allowed to dry. Every other spur on the branch was then punctured with a needle flamed between each puncture. Every alternate spur not thus punctured

was then pricked with a needle carrying a fluid culture of the organism, the needle being flamed between each transfer. The larger spurs were punctured at their center, the smaller receiving the wound at the basal end adjoining the branch.

B 3-1	10 inoculations,	10 checks.
B 3-2	10 inoculations,	10 checks.
B 3-7	6 inoculations,	6 checks.
B 3	10 inoculations,	10 checks.

In experiments made to establish the relationship of the daughter cultures obtained from the inoculated spurs, with the original B strain used for the inoculations, all four cultures were rejuvenated in broth and plated out in agar from which the agar slant stock cultures were made.

The morphological, cultural and certain of the biochemical features obtained by growing the organisms in nutrient broth; agar; gelatine; potato; milk, both plain and litmus; dextrose, lactose and saccharose broth; as well as the action on starch and nitrates were practically the same in every instance, establishing beyond a doubt that the organisms regained from the inoculated spurs were the same as those used for the inoculations.

The morphological, cultural, physical and biochemical features of the organism will be found on the Descriptive Chart appended at the end.

The fruit spurs inoculated May 15 with the B'

strain were examined July 10 and the following results noted:

B 3-1 7 of the 10 inoculated spurs were gumming. The checks were normal (healing without gumming.)
B 3-2 8 of the 10 inoculated spurs were gumming. Checks were normal.
B 3-7 2 of the 6 inoculated spurs were gumming. Checks normal.
B 3 3 of the 10 inoculated spurs were gumming. Checks normal.

On account of the fact that only about 55% of the inoculations produced positive results, a new series of inoculations were made, (July 10) with the B' strain after rejuvenating the stock cultures in broth and plating in agar. As the organisms appeared in pure cultures in the agar plates, which were thinly sown, the inoculations were made directly from the colonies in the plates, a portion of a new colony being used for each inoculation.

The bodies of healthy two year old cherry trees were used for these inoculations, the method being the same as mentioned before.

July 15.	B'3	Tree 8.	10 inoculations.	10 checks.
	B'3-1	" 9.	10 inoculations.	10 checks.
	B'3-2	" 11.	10 inoculations.	10 checks.
	B'3-7	" 12.	8 inoculations.	7 checks.

Aug. 1. The inoculations were examined and the following results noted:

B'3	10 inoculations	gumming.	1 check	gumming.
B'3-1	10 inoculations	gumming.	0 checks	gumming.
B'3-2	7 inoculations	gumming.	0 checks	gumming.
B'3-7	10 inoculations	gumming.	0 checks	gumming.

The writer regrets that he has not the results of

several hundred inoculations and re-inoculations upon which to base his conclusions, but inasmuch as he believes he had complied with all of Koch's Rules of Proof he feels safe in assuming that he had isolated a schizomycete that is pathogenic to certain varieties of sweet cherries and which is capable of inducing at least a localized form of "Gummosis".

The D strain series of Cultures and Inoculations.

On April 8, 1911, diseased branches bearing many blighted fruit spurs were brought in from an orchard near Salem. After removing the leaves and debris, the entire surface of the branches and fruit spurs were sponged with corrosive sublimate and allowed to dry. Tissue cultures into broth were made from the diseased spurs and from the affected tissues of the branches, plating into agar after giving time for a maximum number of organisms to diffuse out of the tissues but not allowing time for incubation.

Of the nine plates thus made, two were accidentally broken, but the remaining seven plates developed typical colonies of the "gummosis" organism in 48 hours at 30°C.

On April 12, agar slant sub cultures were made from typical colonies on three of the plates. Borth sub-cultures were made at the same time, the slants

being used for inoculation while the others were retained as checks. These are hereafter known as the D' strain.

On April 14, a series of inoculations were made at Salem, Oregon in the orchard from which the original cultures were obtained. Normal branches were selected free from gummosis and blighted spurs. After sponging with corrosive sublimate and allowing to dry, every alternate spur, beginning at the base of the limb, was pricked with a needle carrying a culture of the organism. The remaining spurs were then punctured with a sterile needle and left as checks.

Branch D1-A	10 inoculations.	10 checks.
" D2-1	7	" 7 "
" D2-2	6	" 6 "
" D4-1	11	" 11 "
" D4-2	9	" 9 "
" D4-3	11	" 11 "

On the same day, a few more inoculations were made with the D' strain in an orchard at Eola, Polk Co., near Salem.

Branch D1	27 inoculations.	27 checks
" D2	6	" 6 "
" D4	9	" 9 "

On May 24, the inoculations made April 14 were inspected and the following results noted:

D1-A. The 10 inoculated spurs were either blighted or exuding gum from the mouth of the puncture. The check spurs were apparently normal, giving no indications of gum.

D2-1. Six of the inoculated spurs were blighted or gumming. None of the checks were affected.

D2-2. Five of the 6 inoculated spurs were more or less blighted and all were gumming profusely. The checks were apparently normal.

D4-1. Nine of the 11 inoculated spurs were blighted or gumming. One of the checks had blighted without gumming, while the rest were normal.

D4-2. Nine inoculated spurs were gumming, while all of the checks were apparently normal.

D4-3. Three of the inoculated and 1 check spur were blighted or gumming. The rest of the checks were normal.

D1 As only seven of the inoculated spurs were gumming, the branch was not disturbed. None of the checks were affected.

D2 Six inoculated spurs were either blighted or gumming. Checks normal.

D4 Eight of the 9 inoculated spurs were blighted or gumming. One check was gumming while the rest were normal.

Broth tissue cultures were made from the blighted spurs inoculated with each of the above mentioned cultures, plating directly into agar before incubation could occur but allowing time for the organisms to diffuse out into the liquid.

Pure cultures thus obtained were compared with rejuvenated sub-cultures of the original D and D' strain. The morphological, cultural and certain of the biochemical features, obtained from broth, agar, gelatine, potato, milk, both plain and litmus, and saccharose, lactose

and dextrose broth, indicated that the several strains were the one and same species. These features were the same as for the B strain.

On May 27, the bodies of healthy two year old cherry trees were inoculated with a 48 hour agar slant cultures isolated from the spurs blighted with the D' strain. These sub-cultures are known as the D" strain. The bodies of the selected trees were sponged with a corrosive sublimate solution and after drying were punctured with a needle carrying agar culture of the organisms, one check being left for every inoculation. 17 inoculations were made, 9 trees being used in the experiment.

July 21 the trees were examined. The 17 inoculations were gumming more or less profusely while only one of the checks showed any indication of gum.

Koch's Rules of Proof were thus applied to the D strain of the "gummosis" organism with the results mentioned. The cultural features of the D strain are the same as those of the B strain.

The E strain series of Cultures and Inoculations.

Blighted buds and fruit spurs were obtained from a Royal Ann orchard at Eola, Polk County, Oregon, April 14, 1911. The branches bearing the affected tissue were removed and taken to the laboratory at Corvallis.

April 15. Broth tissue cultures were made from the diseased wood, transferring a loop of broth to liquid agar and plating directly in the manner before mentioned. Eight plates were poured each supposedly containing bacteria from as many different affected spurs.

April 17. The characteristic colonies of the 'gummosis' organism were appearing in each plate and in pure cultures. Agar slant sub-cultures were made from an isolated colony in each plate and were marked as the E' strain.

April 19. Agar slant sub-cultures of the E' strain made and used for inoculation purposes.

April 22. A series of inoculations were made at Salem, Oregon in Kimball's Royal Ann orchard. Healthy branches bearing 1,2, and 3 year old wood were selected. After sterilizing the branches by sponging with corrosive sublimate and allowing to dry, alternate spurs on each branch were inoculated in the usual manner, the intermediate spurs being punctured with a sterile needle and left as checks.

Branch E'2	13 inoculations.	13 checks.
Branch E'3	18 inoculations.	18 checks.

June 6. The inoculations of April 22 were examined and the following results noted:

Branch E'2. The eight inoculated spurs on two and three year wood were blighted or gumming, while the 5 buds on one year wood were not apparently affected. The checks were all free from gum.

Branch E'3. Sixteen of the eighteen inoculated spurs were blighted or gumming. The checks were all free from gum.

The branches bearing the above mentioned spurs were taken to the laboratory and tissue cultures were made from each inoculated spur, plating directly into agar in the manner afore mentioned (June 10).

June 12. The typical colonies of the 'gummosis' organism appeared in more or less abundance in each plate. Agar slant sub-cultures were made from selected colonies in these plates and marked as the E" strain.

June 22. The trunks of two year old Bing and Royal Ann cherry trees, free from gummosis were inoculated from a 2 day/^{agar} slant culture of the E" strain. The bodies of five trees were inoculated in the usual manner, three inoculations and three checks being made in each tree.

July 31. The trees inoculated June 22 were examined and the following results noted: Every one of the fifteen inoculations were gumming more or less profusely, large gum blisters being formed near six of the inoculations. The checks remained free from gum and the puncture wounds were healing.

The writer regrets that he has not the results of several hundred inoculations upon which to base his conclusions and he would like to do at least another year's work before committing himself regarding the

method of infection. But as this Thesis will contain the results of all of his work in this investigation, he will make the following suggestions:

It is possible that the organism is introduced into the spurs and buds by insect punctures, but inasmuch as the most of the blighting of the buds and fruit spurs seems to occur while the tree is still in a dormant condition, it is more likely that the bacteria are transferred by the wind or animals to lesions produced by climatic injuries.

The simplest method of control that suggests itself is the cutting out of the affected parts and the sterilizing of the resulting wounds. Ordinarily the localized form of the spur blighting is not abundant enough to warrant the expense of cutting out, but if it should prove that the more generalized form of gummosis is also due to the organism, systematic cutting out, sterilizing of the wounds and burning of the diseased cuttings will be necessary.

In our endeavor to determine whether our organism is the same as those mentioned by Brezizinski(15) and Aderhold(1 d) we had to forego all comparisons with the first named as in the only reference we could find regarding Brezizinski's organism, no morphological or cultural features were given.

In the description of *Bacillus spongiosus* by

Aderhold and Ruhland(1 d)however, we were able to make a more detailed^{comparison} study of the salient features of the respective organisms.

In comparing the morphological features of B. spongiosus with our 'gummosis' organism, we found several points in common. Both are non-sporegenic, motile, rod-shaped organisms bearing polar flagella. The rods are often found in pairs. B. spongiosus is mentioned as sometimes forming long chains, especially in certain media(Old Potato Juice Broth). This feature we have not noted. Both organisms liquify gelatin. We have not been able to obtain, however, certain cultural features on agar and gelatin plates containing 5% beet sugar, features which Aderhold and Ruhold regarded important and upon which they based their specific name. These features were the 'spongy' or 'vacuolated' appearance of the colonies in plates of agar and gelatin containing 5% beet sugar.

We have^{also} found^{that} what we regard as an important cultural feature of our organism is not mentioned by Aderhold and Ruhland(1d) at all. This feature which is constant and characteristic of our organism on certain media is its chromogenic property in agar, gelatin, broth and Uschinsky's solution^(very slight in the latter). In or on all of this media a decided greenish color is noticable after five to seven days. On agar slants and in agar plates and

stabs this greening of the media becomes noticable often after five days. The bacterial masses themselves do not partake of this color, remaining opaque and dirty white in color. In liquified gelatin as well as in broth and Uschinsky's solution, the liquid in the upper portion of the tubes becomes decidedly green while that in the lower half remains unchanged. This coloring of the liquid media is most pronounced in the liquified gelatin.

As this chromogenic property of the organism is so constant and characteristic on the media named, it certainly would have been mentioned had it been seen by Aderhold or Ruhland.

Under the present system for distinguishing between and describing bacterial species, we regard the differences in the cultural features mentioned above as being sufficient to place *B. spongiosus* and our "gummosis" organism in different species. We would prefer^{to do} at least another year's work with our organism before describing and naming it, but as this Thesis will contain all of the results of our work with the gummosis organism, we tentatively describe the organism which we have isolated from localized gummosis lesions and which we have proved, according to Koch's Rules of Proof, of being the specific cause of that form of Cherry Gummosis, as a new species. Following Migula's system of classification, we name the organism

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Pseudomonas cerasus (n. sp.) Griffin and give it the Group Number Ps. 211.2322433.

The salient morphological, cultural and biochemical features are recorded on the Descriptive Chart issued by the Society of American Bacteriologists, and which is appended.

CONTROL.

We have seen that the investigators of the histology of gummosis, both in Citrus and Prunus are agreed that the disease begins in a susceptible tissue laid down by the cambium in the form of embryonic young wood. The gum originates through the hydrolysis of the cell walls of this predisposed tissue, but the forces governing the initial stages of the process of gummification are not understood.

The formation of the abnormal woody tissue by the cambium appears to result when any factor of environment, capable of exerting an irritating influence upon the cambium disturbs the metabolic processes during cambial activity. The apparent autogenous development of gummosis in both the lemon and cherry, will probably

be explained when all the stimuli acting upon the host are better understood.

The relationship of the high water content of the soil to gummosis has been established by a recent investigator. While inoperative alone, when this environmental factor is acting simultaneously with the physiological factor of growth, any stimulus to the cambium may incite gummification.

Gummosis of the cherry, under the conditions prevailing in Oregon, especially in the Willamette and lower Columbia River valleys, is found to have the following environmental factors associated with or preceding its appearance: Frost injury, unfavorable soil conditions, fungi, and bacteria. The first two factors are undoubtedly the primary cause of the disease, at least in the generalized form, but they may be inoperative without the co-operation of the two last named. This appears to be true so far as fungi are concerned, but the results of Aderhold and Ruhland in Germany and those obtained by the writer indicate the more actively parasitic nature of bacteria, at least in certain phases of gummosis.

CONTROL.

Preventive. Resistant Varieties. The susceptibility of the cherry wood to gummosis is apparently an

inherent quality, but the fact that *P. avium* is much more affected than the sour type, and that certain of the sweet varieties are more resistant than others, leads to the hope of finding or developing disease-resistant strains. The varieties of commercial importance in this State are the Lambert, Bing, and Royal Ann. The first named is more resistant to gummosis than the others, but has been observed to gum badly under certain conditions.

Among all of these varieties, growing in the same orchard and apparently under the same soil conditions, certain trees are vigorous and free from gum while adjoining trees are badly diseased. It is possible and probable that propagating from older trees that have withstood the disease may induce a greater resistance. It is also possible that by crossing the susceptible varieties with the hardier but inferior cherries, to produce or gradually establish a strain less susceptible to the disease.

Resistant Stocks. The one important preventive measure that is overcoming much of the gummosis trouble is the use of resistant stocks. As mentioned elsewhere, nurserymen use principally the Mahaleb and Mazzard stocks for propagating the sweet cherries. While the Mahaleb has been favored by nurserymen because of the long budding season and freedom

from leaf disease, the experience of orchardists is that the sweet cherry will give uniformly better results upon the Mazzard.

The measure, par excellence, however, is the top working of Mazzard seedlings to the desired sweet varieties. This practice appears to have been developed most extensively by growers in the vicinity of Salem in this State. The seedlings are headed at the desired height and the proper crotch formed. At from three to five years of age, the main limbs are budded or grafted at least one foot above the crotch. Budding appears to form better unions and produce a better appearing tree unless the grafting is carefully done.

A few growers have practiced body grafting, but this method is questionable on account of forming a crotch and part of the body of gummosis susceptible wood. Many of such trees have been observed where the limbs were girdled at the crotch or where the diseased area extended down the trunk to or slightly below the graft union.

The Mazzard is not absolutely immune to gummosis, but can be rated as of maximum resistance. In tracing the source of a number of seedlings, the body of which were affected by gummosis, ^{it was} disclosed that the seed came from Royal Ann or other improved sweet

cherry trees and hence were not Black Mazzards.

It is a common practice for growers to obtain their nursery stock by taking the "suckers" that often appear around neglected trees. The seedling trees that spring up in uncultivated orchards are also used. Both practices are to be condemned, if gummosis resistant bodies are desired.

The natural seedling, *P. demissa*, has been utilized as a stock for the sweet cherry with apparent good results. Various systems of double grafting are also recommended. But until the superiority of other stocks over the Mazzard have been unquestionably demonstrated, their use should be tested cautiously.

Soil and Cultural Treatment. Next to the factor of climate, unfavorable soil conditions have been ascribed as the principal cause of gummosis. The most badly diseased trees are often found in those parts of the orchard where the soils are heaviest, shallow or most needing drainage, but the fact that gummosis appears on trees in all sorts of soils, in well cultivated orchards as well as those in sod, and is not worse in heavily fertilized orchards than in those receiving no amendments whatever, makes it difficult to attribute the disease to soil conditions.

The fact that gummosis is most prevalent in the humid portions of the state I believe to be explained better by the factor of climate than that of soil. Before planting a cherry orchard, though, it is well to investigate the adaptability of the soil to the fruit, rolling or sloping hill soil, when fairly deep, giving better results in Western Oregon than the more flat valley soil.

The cultural treatment should be such as to prevent depletion of the humus and nitrogen content of the soil, to conserve the moisture and to insure early maturity of wood.

The system of clean cultivation during late spring and summer, striving to maintain a dust mulch, and the sowing of a winter cover crop early enough in fall to germinate with the first rains, will produce as good results for the cherry as for the pome fruits. The nature of the cover crop will be varied according to the requirements of the soil, the legumes, grasses, and other plants all adding to the humus content. The early maturity of the wood, especially in the case of young trees, which tend to make too rank a growth, is desirable and undoubtedly will be hastened by an early sown cover crop.

Cherry trees, headed back rather severely during the first four or five years will form a more satis-

factory framework for the future fruit bearing wood than if left to develop long branches. Judicious summer pruning or pinching back will lessen the amount of more severe pruning that otherwise would be necessary.

Sanitation and Spraying. Several growers have expressed surprise that the scale insects will infest cherry trees. That so little systematic spraying of cherries is done may account for many diseased orchards. The shot hole fungus becomes a pest in certain seasons, when it may attack the fruit pedicels, and warrant control.

An application of Lime-Sulphur(winter strength) in the fall, as the leaves are turning color, or in spring, as the buds are swelling, will be insurance against the scale and possible fungi.

An application of either Bordeaux or Lime-Sulphur in late summer or early fall before the rains begin will check the development of the saprophytic fungi that so often infest the tissues weakened or destroyed through gummosis. The wound fungi of a more parasitic nature would also be better controlled by a spraying at this time. If Lime-Sulphur be used for this spray, a later application for scale is unnecessary.

REMEDIAL.

Soil and Cultural Treatment. If the diseased trees are in a soil, manifestly too wet, drainage can consistently be advised, but when gummosis is making its appearance upon trees in a soil that could not be improved by drainage such advise is superfluous.

The cultural treatment, advised above, should be adopted or continued as it encourages the best development of the tree.

Spraying, Surgery and Sanitation. Spraying trees affected with gummosis will not, of course, cure them, but one or more applications used as mentioned under prevention, will at least partially control the fungi of suspected parasitic nature so often associated with diseased trees.

Slitting the bark of body or larger branches of affected trees is practiced by many growers who believe that it relieves a bark binding or internal pressure that induces gumming. Slitting the bark after gummosis appears will probably help to the extent of providing egress for the gum and preventing a more wide-spread infiltration of the still normal tissues.

In generalized gummosis, or when the more localized form results in girdling, the badly diseased branches and limbs should be removed. Judgment should

be utilized, however, as trunks and branches only partially girdled will often recover. If over half of the tissue of a branch is affected it will be better perhaps to remove it and start a new one.

Tree surgery to the extent of removing the dead or infiltrated wood of localized cankers and when possible, the more generalized form, while not recommended for gummosis in Citrus, should be practiced in the case of cherries. In the cherry, the cambium of such diseased areas is nearly always affected and not able to develop new tissue. Since various wound fungi and those of possible parasitic nature appear in such diseased areas, supplementing spraying with cutting out, will best conserve the weakened trees and protect the surrounding ones.

If the apparent parasitic nature of the bacteria which are found to be associated with the more localized form of gummosis shall require remedial treatment, cutting out the affected parts will be the only practical method of control, and will have to be supplemented by the disinfection of all wounds thus made, using either a solution of Bordeaux or corrosive sublimate.

The dead trunks and branches of the trees affected with gummosis are often left lying in, or near the orchard. The fruiting bodies of shelf fungi are

common on trees in many cherry orchards, although the tree is only partially or apparently not all diseased. As a sanitary measure, all dead and dying wood, as well as the fruiting bodies and related affected tissue, on otherwise normal trees, should be removed from the trees and orchard and burned.

All the wounds made in the removal of dead or diseased branches on localized areas should be washed with some good fungicide (Bordeaux or corrosive sublimate) and then painted with white lead, coal tar or grafting wax.

CONCLUSION.

Gummosis is the name given the most destructive diseased condition of the cherry in Oregon. Its effect on certain varieties of the sweet cherry is the nature of a scourge. Gummosis is characterized by the exudation of gum from fissures in the bark of the trunk and limbs and fruit spurs or through the ends of blighted buds.

The gum originates through the hydrolysis of the cell walls of an abnormal embryonic wood tissue formed by the cambium in response to certain stimuli.

In the so-called autogenous form of gummosis, it is difficult to connect any environmental factor with the disease. In some cases, gummosis is associated

with unfavorable soil conditions, but most often it ^{to} is/the more active stimuli produced by frost injury and the attacks of fungi and bacteria that gummosis can be chiefly ascribed.

According to the most recent investigation, the simultaneous occurrence of both growth and a high sapidity of the tissues is essential before irritative stimuli can produce gummosis.

For preventing cherry gummosis, the top working of resistant stocks is doing more to eliminate the disease from young orchards and preventing its occurrence in the body or crotch of the tree, than any other one measure. The true Mazzard seedlings are giving the best results as stocks for such top worked trees.

Planting cherry trees in a congenial environment as regards both climatic and soil factors is also of primary importance in preventing the disease.

Remedial measures recommended are the correction of unfavorable soil or cultural conditions if they exist, and judicious spraying and surgery to prevent the possible attack or spread of parasitic fungi and bacteria.

Investigations regarding the parasitism of both fungi and bacteria constantly associated with certain forms of gummosis are being continued and the results

will be announced later.

EXPLANATION OF PLATES.

- Plate I. Localized gummosis. Blighting of buds and fruit spurs. Fig. 1 and 2 show affected branches at blossoming time. Fig. 3 A gummy ~~ex~~ exudate is almost constantly associated with the blighted spurs.
- Plate II. Fig. 1,2,3 and 4 show different views of blighted spurs. The exudate is usually found near the base of affected spurs, but it apparently follows the line of least resistance and may "well out" from a fissure in the epidermis of the branch, to one side, or above, or below the diseased spur.
- Plate III. Fig. 1. The blighted buds and spurs first become noticeable as the buds begin to expand. In the affected spurs the buds remain quiescent. Fig. 2 and 3. The exudate from blighted spurs often has the appearance of being forced out under pressure.
- Plate IV. Fruit spurs with one to all of the buds affected. From the diseased tissue associated with such blighted spurs, as shown in Plates I, II, III and IV an organism has been isolated, which appears nearly constant in pure cultures from tissue cultures of the affected wood and which has caused the characteristic blighting and gumming of the buds and spurs when inoculated in pure cultures, conforming to Koch's other rules of proof as well.
- Plate V. Fig. 1. 2 year old root budded tree. It is common for young trees to be weakened or killed by the disease partially or wholly girdling the body. Top working resistant stocks will eliminate this condition. Fig. 2, the affected tissue removed to show extent of injury.

- Plate VI. Diseased body and crotch of 11 year old Royal Ann. Branch to the left completely girdled. Over half of the remaining trees in this orchard, which were body grafted, are more or less diseased at the crotch.
- Plate VII. Same tree as on Plate VI. Diseased limb and affected tissue removed to show extent of injury. Camera moved a little to the left.
- Plate VIII. The gumming exudation from the body and branches characterize the generalized form of the disease. Diseased bodies and crotches may be avoided by the use of resistant stocks, top worked.
- Plate IX. Trying to overcome^a diseased crotch by bridge grafting. Several trees in this orchard were similarly affected. They were body grafted, probably on Morello and the stock usually sprouts just below the graft union.
- Plate X. Fig. 1. An unsymmetrical Bing tree, caused by the diseased branches being removed. Fig. 2. The body of this Royal Ann tree was girdled at the crotch after leafing out.
- Plate XI. Another body-grafted cherry girdled at the crotch. Diseased wood removed to show extent of injury which extends slightly below the graft union.
- Plate XII. Affected tissue on large limb removed to indicate the extent of localized gummosis. This form is almost always associated with a blighted bud or spur.
- Plate XIII. The small blisters or pimple-like structures on the bark contain the fruiting bodies of the fungus so common on diseased trees.
- Plate XIV. Trees weakened thru gummosis are often infested with the fungus *Schizophyllum commune*. In fig. 1 the burrow-openings of a wood boring beetle that infests diseased trees are seen. Fig. 2. The characteristic spore-bearing bodies of the fungus.

- Plate XV. *Polystictus* sp. (Fig. 1) and *Polyporus* sp. (Fig. 2) are other saprophytic fungi that infest diseased cherries.
- Plate XVI. Cross sections of a sun scald wound on a cherry trunk. Sections of body made thru above and below wound, showing the work of a wood rotting fungus.
- Plate XVII. Fig. 1,2 and 3 show to-worked seedlings of different ages. The trees in Fig. 1 and 2 were budded and Fig. 3 grafted. This method of starting cherry trees tends to eliminate gummosis. Budding produces smoother and better appearing unions than grafting.
- Plate XVIII. A seedling body-grafted. A method not advisable because of producing a crotch of gummosis susceptible wood.
- Plate XIX. A top-worked seedling, headed back for two seasons and forming a good frame work for the future fruit producing wood.
- Plate XX. Fig. 1. The result of one series of inoculations in 1910. The blighted spurs were inoculated with a pure culture of the Bacteria (D 11) while the alternate spurs were punctured with a sterile needle and left as checks. See history of experiment.
Fig. 2. Result of an inoculation with a pure culture of the organism in 1911 (D 5-2). See history of experiment.
- Plate XXI. Fig. 1. Results of inoculation with D2-5 1911(see history). Leaves on normal check spurs cut away to show blighting of the inoculated spurs.
Fig. 2. Five weeks after inoculating with D4-2(1911). Leaves cut away to show blighting. The even numbered spurs, beginning at base were inoculated. The alternate spurs(odd numbered)were punctured with sterile needle and left as checks. See history.



Fig. 1.



Fig. 2



Fig. 3.



Fig.1.



Fig.2



Fig.3



Fig.4.



Fig.1



Fig.2



Fig.3.





Fig. 1.



Fig. 2.











Fig. 1.



Fig. 2









Fig.1



Fig.2.



Fig. 1.

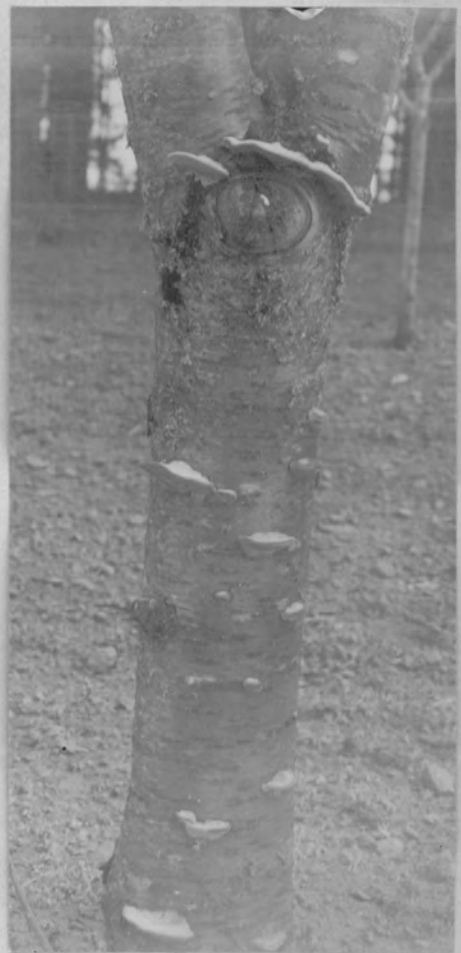


Fig. 2.





Fig. 1.



Fig. 2.



Fig. 3.







Fig. 1.



Fig. 2.



Fig. 1.



Fig. 2.

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