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Gummosis of Prunus & Citrus

THESIS

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BY

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WITH PLATES VII-X

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A Study on Gummosis of *Prunus* and *Citrus*, with Observations on Squamosis and Exanthema of the *Citrus*.¹

BY

ORMOND BUTLER.

With Plates VII-X and three Figures in the Text.

PREFACE.

THE studies which form the groundwork of the present memoir were begun during the autumn of 1907, under the direction of Professor R. E. Smith, at the Pathological Laboratory, Whittier, California, being later continued in Professor B. M. Duggar's laboratory, Cornell University.

The histological studies on gummosis were almost exclusively confined to the *Citrus* during my stay in Southern California. Having satisfied myself that the gum diseases of *Prunus* and *Citrus* were histologically similar maladies—in other words, confirmed the opinion held by Savastano in 1884—I attempted to trace out, using Mikosch's recent memoir as a guide, the elusive first stages of gummosis, but only succeeded in reaching the decided opinion that this author's views would not apply in the case of the *Citrus*. When, therefore, I transferred my work to Cornell University, I decided to reinvestigate gummosis in *Prunus*; as the results arrived at by Mikosch would bear confirmation, while, at the same time, continuing my histological studies on *Citrus*. The present paper is the outcome of this combined study.

Gummosis is a fairly common phenomenon in the vegetable kingdom, but this malady, in other plants, has hardly been sufficiently studied to warrant a comparative review in the present state of our knowledge. I have, however, devoted a few remarks to squamosis and exanthema of the *Citrus*, as these maladies have features common to gummosis, and have not, hitherto, been histologically described.

In closing this preface it is my pleasant duty to acknowledge my indebtedness to Professor B. M. Duggar, and I know not how better to do so than by saying that it is far greater than could be told.

O. B.

June, 1910.

¹ Laboratory of Plant Physiology, Cornell University, Contribution No. 1.

I. INTRODUCTION.

REVIEW OF LITERATURE.

The identity of the gum disease of *Prunus* and *Citrus*, an identity implied in the title of this paper, has been, if not definitely proved, at least vouchsafed for by Savastano and Delacroix. According to Savastano the histology of gummosis in the *Citrus* is similar to that described by various authors for species of *Prunus* and *Citrus*. He agrees with the histological characters of these diseases as they are given by Beijerinck, Comes, Briosi, Sorauer, Frank, and especially Trécul and Prillieux. Delacroix, on the other hand, observed some differences: starch, always present in the gumogenetic tissues of *Prunus*, is absent from the affected tissues of *Citrus*, and gummosis usually begins at the periphery of the gum parenchyma in the latter, instead of in the centre as in *Prunus*. Otherwise the histogenesis of the disease is the same in both genera. In other works on gummosis of the *Citrus* that I have consulted, the histology of the malady is not studied at sufficient length to warrant any conclusions regarding the validity of Savastano's and Delacroix's conclusions. For this reason it appeared to me that a reinvestigation of the development of gummosis in the *Citrus* could be profitably undertaken.

Gummosis of *Prunus*, on the other hand, has been frequently studied, and it would appear, at first sight, that little remained to be discovered regarding this disease. A review of the most important memoirs that have appeared shows very clearly, however, that our knowledge is still imperfect, and that further studies on this important malady are necessary to elucidate doubtful and obscure points.

Previously to 1860, when Trécul's observations on gummosis of the stone fruits appeared in the *Comptes Rendus*, no studies of any importance had been published on this malady.

Trécul¹ believed that gummosis was induced by a variety of causes, all of which were instrumental in accumulating in the pathognomonic zone a superabundance of sap. The disease affected either the young wood in formation, or the more differentiated and lignified tissues behind it. In the embryonic wood cells the gum pockets were formed by resorption of the cells, and more especially in the regions where the vessels appeared to be laid down. The lacunae thus formed were filled with fluid containing the dissolved contents of the resorbed cells, the remnants of their cell walls, still in process of dissolution, together with entire cells broken free from the periphery of the gum cavities; these latter cells sometimes enlarged considerably and divided transversely into several cells before being, in turn,

¹ Trécul, A.: *Maladie de la gomme chez les cerisiers, les pruniers, les abricotiers, les amandiers*. *Comptes Rendus*, li, 1860, pp. 621-624.

dissolved away. The gum pockets enlarged by continued destruction of the peripheral cells. When occurring within the young wood, the gum pockets were also produced by resorption of the cells, and were even observed originating in the vessels. When gummosis was not severe, and conditions favourable to normal growth recurred, sound wood was formed again by the unaffected cambium, which *de novo* laid down gum-forming tissue when conditions favourable for its production were brought about.

Trécul mentioned one causal condition of gummosis : rain.

In 1863 Wigand¹ published an important memoir on gummosis. He studied gum formation in *Prunus avium*, and, as a result of his investigations, concluded that : (1) the gum found within the vessels was due to a change in the cell-walls, the evidence in favour of this view being derived from the presence and absence, even within the same vessel, of the internal surface sculpturing ; (2) except when occurring in the vessels, gum formation in the wood was always preceded by the development of an abnormal wood parenchyma. This abnormal parenchyma was laid down between the medullary rays at any time during the year's growth, and within it the gum pockets were produced lysigenously : one pocket only was usually formed, though at times radial rows of three were produced, each one separated from the other by normal xylem ; (3) the gum pockets could be laid down year after year in the successive annual rings ; (4) gum formation in the cortex was also preceded by an abnormal parenchymatous growth, the extension of individual bast rays ; (5) the formation of gum pockets was due to a centripetal dissolution of the cell-walls, the gum filling the cavities thus formed being derived from the dissolved walls and from the starch within the affected cells which had suffered a synchronous gummous degeneration ; (6) gummosis could begin in the wood and proceed over into the bark, and *e converso* ; (7) cherry and plum gums were a mixture of arabin and of a gum resembling bassorin, i. e. cerasin, which, unlike it, however, was soluble in boiling water.

Wigand did not consider gummosis a malady *per se*, but rather a symptom of weakness of the affected tissues.

In his study on vegetable mucilages, Frank² devoted a section to gummosis of the cherry. He found that gum is formed from the secondary membranes in normally formed wood, or through the disorganization of an abnormally formed wood parenchyma and certain cortical tissues. These different areas of gummosis were not necessarily interdependent as regards formation, though whenever the seat of the disease was in the wood all three forms were observed.

¹ Wigand : Ueber die Desorganisation der Pflanzenzelle, insbesondere über die physiol. Bedeutung von Gummi und Harz. Pringsheim's Jahrb., iii, 1863, pp. 115-182.

² Frank, A. B. : Ueber die anatomische Bedeutung und die Entstehung der vegetabilischen Schleime. VI : Kirschgummi. Pringsheim's Jahrb., v, 1866-67, pp. 184-198.

In pathognomonic tissues the intercellular substances and primary membranes first changed over into gum, the secondary membranes then became affected, and finally the starch within the cells dissolved away. In certain cases the starch in apparently healthy tissues degenerated with the formation of gum while the cell-walls still remained normal; when this occurred the gum appeared as irregular yellow drops, or in the form of the original granule. The most marked changes in the starch occurred, however, in the cells bordering the gum pockets; the granules lost their form, became irregular masses, and finally gum droplets, the transformation stages being readily followed by their fainter and fainter blue coloration when treated with iodine.

Frank also believed that the plant sap, besides the cell-walls and starch, was an important constituent of gum.

We gather further from Frank's memoir that: (1) cherry gum was insoluble in cold and hot water, in which it did not even swell; (2) when sections of affected tissues were boiled in a solution of caustic potash, the secondary membranes of healthy cells gave the cellulose reaction with iodine and sulphuric acid, while the gum-forming tissues were coloured yellow.

Prillieux¹ in his study on gum formation in fruit trees confirmed in the main the conclusions arrived at by previous workers, notably Wigand, Frank, and Sorauer, but differed from them as regards the formation of the gum. As a result of his investigations, which were confined largely to the apricot, he concluded that: (1) the gum in the vessels was due to infiltration and not to a change in the cell-walls, as the internal spiral and punctiform thickenings remained unaffected even when the lumen was completely filled with gum; (2) the gum filling the lumen of the wood-cells and bast fibre bundles was also due to infiltration or starch; no change in the cell-walls was observed except sometimes a slight swelling in the outermost fibres of the bast fibre bundle; (3) the first stages of gum formation in the abnormal wood parenchyma were a transient rift between the middle lamellae and the cell-walls, followed by the appearance of gum within the suture. Coincident with the appearance of gum, the middle lamellae began to lose their identity, and soon became indistinguishable from the accumulating gum; (4) the starch grains did not gradually change over into gum. With the first appearance of gum within the cells the starch grains became agglomerated. As the production of gum increased the starch became resorbed, but no transitional stages were ever observed; the starch grains at all stages reacted blue to iodine, the gum always yellow.

Hydrochloric acid and chloriodide of zinc were found useful in studying the younger stages of gum formation. The former coloured the wood-cells of plum, apricot, and peach violet, and the gum yellow—recently formed

¹ Prillieux, Ed.: Étude sur la formation de la gomme dans les arbres fruitiers. Annales des Sciences Naturelles (Botanique), sér. 6, vol. i, 1875, pp. 176-200.

gum, however, sometimes coloured violet; the latter coloured the gum yellow at all times.

Prillieux believed that the starch, which is abundant in and around diseased tissues, plays an important rôle in gum formation, the main bulk of the gum being derived indirectly from it.

In his study on *Clasterosporium carpophilum* Aderhold¹ made a number of inoculation experiments on cherry, apricot, peach, and plum trees with the view of determining the rôle played by this parasite in gummosis. He used both pot-grown plants and standard trees, and his inoculation experiments extended over the greater part of the growing season. Numerous inoculations were always made and alternated, with an equal number of witnesses, up the trunk or along the branches, as the case might be. The wounds were made, with one or two exceptions, small and unimportant, but were not protected, as a rule, from the possibilities of contamination. However, this non-protection of the wounds did not affect the experiments, which proved quite decisive. The inoculated wounds always produced gum, which usually pearly more or less upon the surface, sometimes even appearing in three days; whereas the witnesses invariably healed up normally. In some experiments *C. carpophilum* was inoculated, not into the cambium but just beneath the epidermis, or shallowly within the cortex. In these cases no gum appeared in either the inoculated or witness wounds. Aderhold also made one experiment on cherry in which he used *Cladosporium herbarum* in lieu of *C. carpophilum*. No gum appeared in either the inoculated or witness wounds.

Aderhold recorded one or two experiments in which an increased gum flow distinctly followed watering. He made no comment, however.

In the section of his paper devoted to the histology of gummosis, Aderhold described the characteristic appearance of diseased tissues: the production of an abnormal wood parenchyma and its subsequent breaking down to form gum pockets; the reproduction of healthy tissues once more. He also recorded an extensive formation of gum in the mid-rib of a cherry leaf, and the swelling and final dissolution of the pulp-cells of the fruit invaded by the hyphae of *C. carpophilum*. The swelling of the cell-walls and their final dissolution could be brought about either by the secretion of an enzyme, by the hyphae, or from the cells reacting to the stimulus induced by them. Aderhold mentioned both possibilities and accepted the former.

In 1906 Beijerinck and Rant² published a memoir dealing very largely with the effect of stimuli on gum formation. They found that the peach

¹ Aderhold, R.: Über *Clasterosporium carpophilum* (Lév.) Aderh. und Beziehungen desselben zum Gummißusse des Steinobstes. Arbeiten d. biolog. Abt. f. land- und forstwirthschaftl. Gesundheitsamte, ii, 1902, pp. 515, 559.

² Beijerinck, M. W., and Rant, A. Sur l'excitation par traumatisme, le parasitisme et l'écoulement gommeux chez les Amygdalées. Archives Néerlandaises des Sciences Exactes et Naturelles, sér. 2, vol. xi, 1906, pp. 184-198.

and almond were very sensitive to wound stimuli, but the cherry, plum, and apricot were less responsive and gummed less readily. When, at the height of the growing season, shoots of peach and almond were wounded into the cambium and secondary wood, at intervals, beginning from the apex, droplets of gum were observed pearling at some of the wounds in less than a week, sometimes even in four days. Dividing the affected shoots into zones they found that gum production was closely related to the position occupied by the wounds on the shoots. At the apices gummosis was slight, then rapidly reached a maximum, decreased again, and finally ceased.

The zone of maximum gum formation occurred just beneath the region of maximum growth in length, and was probably correlated, Beijerinck and Rant believed, with the growth in thickness of the cambium, and also of the procambium.

When shoots were wounded during the course of the summer, but after suberization had taken place, they generally failed to produce gum. In this case the production or non-production of gum appeared to be correlated with seasonal peculiarities.

The gum was formed in the young wood in formation and the diseased tissues, while young, always abutted directly on the cambium. The length and importance of the gum pockets depended largely upon the size and position of the wound upon the shoot; their distal ends were always further removed from the centre of propagation than their basal ends. Wood normally formed never produced gum. The gum pockets buried in the wood were formed by cambial activity at some previous time, and a series of gum pockets in a single season's growth were due to conditions being such that the cambium laid down normal and abnormal embryonic xylem alternately.

Beijerinck and Rant obtained some interesting results with corrosive sublimate and burning as the stimuli of gum formation. When mercuric chloride was introduced into wounds made on young growing shoots of peach, abundant gummosis occurred after the lapse of four to seven days. Burning was also effective, but not as much so as corrosive sublimate, probably owing to the fact that the area of tissue stimulated was less extensive than in the case of the latter, the burns being produced by focusing the sun's rays upon the shoots.

Gummous degeneration of the tissues Beijerinck and Rant believed to be due to a cytolytic enzyme becoming active the moment necrobiosis set in. In necrobiotic cells the protoplasm was killed, but the enzyme remained alive and active, and was able to diffuse out into the surrounding tissues, which it transformed into gum. The greater gum flow when mercuric chloride was used than when the tissues were simply burned or only wounded lent support, they believed, to this view. The gumming of tissue infected by *Coryneum Beyerinckii*, the hyphae of this parasite only showing

gummosis degeneration when growing within living tissues, was regarded as further evidence of the activity of a cytolytic enzyme; the ferment, passing out from the cells killed by the activity of the fungus, attacked both the living plant cells and the mycelium, transforming them into gum.

The work of Beijerinck and Rant was more fully developed somewhat later in the same year by the junior author.¹

To the stimuli inducing gummosis mentioned in the joint memoir, Rant made a few additions; as causal agents he mentioned a number of Fungi, Bacteria, insects, chemical substances, and traumatisms. The histology of the disease was also described at greater length.

Rant did not believe that the gum in the vessels was due to a degeneration of the inner lamellae of the walls, and advanced the view, perhaps somewhat hesitatingly, that it originated either from the decomposition of thylloses, which, however, he admitted never having observed, or from degenerating environing cells. He was of the opinion that the gum could originate both intra- and inter-cellularly. The intracellular gum originated in the manner described by Prillieux. This form of gummosis he considered, however, unimportant, as it did not contribute to the formation of the exudate that appeared on the surface of diseased trees. This latter gum originated exclusively from the degeneration of the cambial and embryonic wood-cells.

The gum cavities arose between the medullary rays. The diseased cells separated from one another and a certain and variable number, depending on the virulence of the disease, became resorbed. The cells bordering the gum cavities thus formed became rounded and grew out into the lacunae; these ingrowing cells gave rise by division to the cells floating free in the gum. When gummosis was severe the medullary rays were destroyed, and the cambium also sometimes killed. When, however, conditions favourable for growth recurred and the cambium had not been affected, normal wood was laid down over the gum-forming tissues, which gradually lignified.

In 1906 Mikosch² also published an extensive article on gummosis of the cherry. He found that branches of plum, peach, apricot, and almond, when cut into 10 cm. lengths and placed in water at room temperature, developed gummosis readily, and his studies were confined very largely to pathognomonic material obtained in this manner. In all the species of *Prunus* experimented with, gum readily formed in eight days, in some cases even earlier, and could be seen with the naked eye pearling between the cortex and the wood. The statement that gummosis developed to a visible extent was generally, though not specifically, correct. To be exact, branches removed in October, November, and June gummed promptly; branches cut

¹ Rant, A.: De Gummosis der Amygdalaceae. Thesis, Amsterdam, 1906.

² Mikosch, K.: Untersuchungen über die Entstehung des Kirschgummi. Sitzungsber. d. k. Akad. d. Wiss. Wien (math.-nat. Klasse), cxv, 1906, pp. 911-61.

in December gummed slowly and less abundantly, and, finally, branches removed in May, when the trees were in leaf, did not show any symptoms of gummosis. There thus appeared to be a certain relation between the time of year at which the branches were cut and their gumogenetic power. Mikosch observed and mentioned this apparent correlation, but offered no explanation for it.

Cherry gum was composed of a water-soluble substance (arabin) and a water-insoluble substance (cerasin); cerasin was, however, soluble in lime-water. These gums occurred together in an intimate mixture. Mikosch was able, however, to separate the arabin almost completely from the cerasin by levigation. When cerasin had been as thoroughly as possible freed from arabin it would, on the addition of alcohol, contract into a homogeneous vitreous mass; pure arabin, on the other hand, gave a white floccose precipitate on the addition of alcohol, or better acid alcohol.

Cherry gum was insoluble in 50 % alcohol. In preparing sections of diseased material for study Mikosch cut from fresh branches, and examined the sections in water, or in 33 % alcohol, if they had to be preserved any length of time. This strength of alcohol was used for the reason that the gum only contracted slowly at this strength. Glycerine and water, equal parts, was also used when the sections were not to be kept for any great length of time.

Mikosch had very little success with staining reagents. He found double staining with neutral red and acid green more reliable than ruthenium red. Chloriodide of zinc was fairly satisfactory; the cell-walls stained blue, as in healthy tissue, and the gum yellow.

The gum was produced in the young wood in formation, and the physiological changes leading to its formation were as follows:—

In the central cells of the groups boxed in, as it were, between the cambium, xylem, and adjacent medullary rays, the protoplasm appeared denser than normally, was granular, and contained starch grains. Following very closely the increased protoplasmic content and appearance of starch, the diseased cells separated schizogenously, and further changes rapidly followed in the cytoplasm. The starch grains became transformed into as many gum droplets, and their remnants, when present, stained reddish brown with iodine solutions. The gum droplets were precipitated *in situ* when sections were first placed in alcohol, but appeared as clear spots when the alcohol was replaced by water, owing to the partial solubility of cherry gum in the latter. Soon after their formation the gum droplets collected together between the protoplast and the cell-wall. If we call, for convenience, the apex of the cell that portion of it which is bounded by the intercellular space, then the gum may be said to have accumulated invariably between the protoplasm and the apex of the cell. Starch does not appear, however, to have been the only source of gum formation,

for Mikosch tells us that owing to the activity of the protoplast gum was continually excreted. This excretion continued until the protoplast was gradually pushed to the base of the cell, where a few remnants of it might be found. The cell-wall was scarcely if at all affected and remained distinct. It may be noted that in some cases, evidently when gum secretion was only active periodically, the protoplast would secrete a layer of gum, then a layer of cellulose, then a layer of gum and a layer of cellulose. Mikosch figured a cell with three distinct walls enclosing two gum layers.

Mikosch did not make it very clear how the gum finally became extracellular. The cell-walls persisted for a long time unaltered, and it was only towards the centre of a gum pocket that they became indistinguishable from the gum matrix. These cell-walls furnished the cerasin component of the gum.

Gum pockets were not necessarily confined to the wood in formation: they also occurred in the cortex.

When the attack of gummosis was not severe, and conditions favourable for its development were soon followed by conditions unfavourable, then normal wood would be laid down on the outside of the gum-forming tissues, and the cells lying within the pathognomonic zone gradually lignified.

Mikosch concluded from his study that gummosis was a pathological phenomenon due to the response of the cambium to wound stimuli.

Ruhland¹ in 1907 advanced the opinion that gum formation was not due *per se* to traumatism, but to the fact that they allow, when deep enough, air to penetrate to the cambium, or more properly to the young wood in formation. Atmospheric oxygen, he believed, was the active agent in gum formation. This gas acted upon the pectins and pectinates of the cell-walls and upon the carbohydrates within the cells that should have gone to the building of the new septa following cell-division, oxidizing them into gum.

The hypothesis regarding the intracellular formation of gum was arrived at through a study of diseased tissues. Ruhland found that a number of the large swollen cells bordering the gum pockets contained two well-developed nuclei but no cross-walls, and he concluded that intracellular gum was formed through a process of oxydation homologous to that which was effective in producing intercellular gum.

Ruhland performed a few experiments with the view of determining the effect of oxygen exclusion upon gum formation. He used cuttings of peach, plum, and cherry, the bottom ends of which were placed in water, while the distal cut surfaces were capped with a mantle of hydrogen or nitrogen, or some such impervious substance as cocoa-butter, paraffin, &c. The experiments gave very marked results: no gum formed in the cuttings

¹ Ruhland, W.: Zur Physiologie der Gummibildung bei den Amygdalaceen. Ber. d. Deutsch. Bot. Ges., xxv, 1907, pp. 302-15.

in which the cut surfaces were protected from the air (oxygen), whereas in the witnesses from 14 % to 100 % of the cuttings showed the characteristic lesions. Taking the most extensive experiment recorded the witnesses showed approximately a mean of 67 % diseased cuttings.

Sorauer¹ in his *Handbuch der Pflanzenkrankheiten*, art. 'Gummifluss der Kirschen', described the usual appearance of diseased tissues, and ascribed gum formation to a latent capacity possessed by embryonic and full-grown cells to produce gum. He did not lay any stress at all upon the development of gum pockets in the wood in formation, as may be gathered from the summary of his observations, which may be translated thus: (1) gum formation is preceded by a development of parenchymatous tissue within the xylem which presents a nidulose appearance, and lies usually between two medullary rays, and exceptionally includes one or more; (2) the abnormal parenchyma develops independently of wound stimuli; (3) the tissues finally break down into gum and the medullary rays grow out into the pockets thus formed.

Sorauer believed that gum formation was due *pro parte* to a zymogenetic substance present in embryonic and fully developed cells, which became active when certain life functions connected with the growth of the cell-wall were interfered with. But the retardation in the development of the cell-wall thus brought about, as also the transformation of the part already laid down into gum, could also be readily accounted for, he thought, by assuming an increase in the oxygen supply irrespective of the action of enzymes, an opinion also held by Ruhland, as we have seen. This necessary supply of oxygen was obtained directly from the atmosphere through the intermediation of wounds, or from organic or inorganic stimuli.

In order to show that gummosis followed stimulation by an oxydizing agent, Sorauer performed the following experiment: He made two incisions into the limb of a healthy cherry-tree: into one he poured oxalic acid and into the other, which was to serve as witness, distilled water. The wound treated with oxalic acid gummed freely during the course of the summer, the flow only ceasing when the acid had become, in the course of time, neutralized; whereas the witness remained healthy.

II. GUMMOSIS.

Syn.: *France*: Colle, Gommose, Maladie de la gomme; *Germany*: Gummifluss, Gummikrankheit, Gummosis; *Great Britain*: Gummy, gummosis, gum disease, sore shin, foot-rot (erroneously); *Italy*: Mal di gomma, Male della gomma; *Portugal*: Lagrima; *Spain*: Enfermedad.

¹ Sorauer, P.: *Handbuch der Pflanzenkrankheiten*, i, 1909, pp. 693-701.

I. HISTORICAL.

The phenomenon of gummosis has long been known in the genus *Prunus*. The malady has been more frequently noted in the cherry and peach, though, as modern authors have shown, it affects also the apricot, almond, and the plum, both the American and European species. At no time, however, has the disease attained great economical importance, and, unlike other maladies, it has no history. Gummosis has always affected the *Prunus*, now more, now less, but in no sense has it ever been a scourge. In the case of the *Citrus*, on the other hand, gummosis was hardly known, and then not as a malady, before 1834, when, Fouqué¹ informs us, it suddenly appeared in the groves of San Miguel. Watt,² however, mentions *Citrus medica*, *C. decumana*, and *C. Aurantium* as yielding an unimportant gum, samples of which were exhibited at Madras in 1855. The orange and citron gums were sent from Masulipatam, but the place of collection of the shaddock gum is not given. Gummosis must, therefore, have been a sufficiently common phenomenon to attract attention, though not so intensive a one as to affect the health of the trees, hence the gum was collected and sent to the Madras Exhibition, as being perhaps of some economic value. Gummosis, we may therefore safely conclude, was no unusual occurrence in the eyes of the natives, and had been for years past a common affection of the trees. The disease was known in India in all probability before it was first observed in the Azores; in fact, there is evidence that the malady, in a very benign form, was in the orange groves of Europe in 1818; it appears even to have been described 'briefly and precisely' as long ago as the middle of the seventeenth century.³ However this may be, gummosis does not appear to have been a malady of much economic importance prior to its sudden and virulent outbreak first in the Azores, and then in the different orangeries of Europe, towards the middle of the nineteenth century.

The malady was first observed in San Miguel in 1834, where it became, within a few years, enormously destructive. In 1840 the disease was at its height. 'Entire orangeries were destroyed, others partially ruined, and it is estimated that one-quarter of the orange trees on the island had to be dug out.'⁴ In 1842 the disease had become less virulent and in 1873, though still extant, was no longer feared.⁵ In Portugal gummosis appeared in 1845, affecting first the groves around Lisbon, and was at the height of its destructiveness between 1858 and 1861. In 1851 it was present in the orangeries of Hyères,⁶ and in 1855 appeared in the groves at Limone on

¹ Fouqué, F.: Voyages géologiques aux Açores. III: Les oranges de San Miguel, les cultures et le monde organique aux Açores. Revue des Deux Mondes, civ, 1873, p. 836.

² Watt, G.: Dictionary of Economic Products of India. ii, pp. 344, 349.

³ Ferrare, J. B.: Hesperides sive de Malorum aureorum cultura et usu 1646, cited by Savastana, L. Della cura della gommosi e carie degli agrumi. Comizio Agrario, Napoli, iv, 1887.

⁴ Fouqué: loc. cit., p. 836.

⁵ Ibid., p. 836.

⁶ Rendu, V.: Note sur une maladie des orangers d'Hyères. Comptes Rendus, xxxiii.

the Lago di Garda.¹ In Sicily the disease was first observed in the province of Messina in 1862,² and was also discovered about this time or somewhat later in Spain, Corsica, and Algeria. The groves of New South Wales were devastated between 1860 and 1870.³ According to Curtis⁴ the disease probably appeared about 1876 in Florida, though few people, he remarked, could remember seeing it prior to 1880; in California it was observed in 1875.⁵ At the present day gummosis is known wherever the *Citrus* are extensively grown.

2. DESCRIPTION.

General. Unlike most maladies gummosis appears to be autogenous as well as superinduced by traumatism of very diverse nature. Fungous, insectile, physical and chemical injuries, when they affect directly the cambium layer, all induce gummosis, provided growth is taking place. Furthermore, gum is not produced in quantities unless the tree affected is well supplied with water. The development of the disease depends, then, upon the rapidity with which new tissues are being laid down at the time of its initiation; and this rapidity, as is well known, is a function, broadly speaking, of the amount of water available to the roots of the affected tree. Growth and water are, therefore, essential to the appearance of gummosis, and both are limiting factors. The relation of these two factors, *growth* and *water*, to gum formation, here only postulated, will be more fully dealt with in subsequent pages of this memoir, and is mentioned at this time solely with the view of rendering more comprehensible the development of gummosis in the orchard, and the cultural and environmental conditions affecting the same.

Gummosis develops sporadically, sub-generally, or generally, depending on the nature of the inducing factor. It is usually only sporadic in the case of insect attack, and the same remark is true for many of the fungi. Some fungus-parasites, notably *Coryneum Beyerinckii* and *Clasterosporium carpophilum*, produce, on the other hand, sub-general or general gummosis, the atmospheric conditions favourable to the development of these parasites being also favourable to the formation of gumogenetic tissues in the host. Autogenous gummosis appears sporadically or sub-generally. In most species of *Prunus* and *Citrus*, the disease develops sporadically, only becoming generalized, as a rule, in *P. Cerasus*, *P. avium*, and *Citrus Limonum*.

¹ Rapporto della Commissione per studiare la malattia degli agrumi in Sicilia, &c. *Annali di Agricoltura Siciliana*, n.s., i.

² *Ibid.*

³ Alderton, G. E.: *Treatise and Handbook of Orange Culture in Auckland, New Zealand.*

⁴ Curtis, A. H.: *Sore Shin or Gum Disease.* Florida Agr. Expt. Sta., Bull. ii.

⁵ Mills, J. W.: *Citrus Fruit Culture.* California Agr. Experiment Sta., Bull. cxxvii.

The conditions favourable to the development of gummosis (inducive in the autogenous form, conducive in other cases) are: (1) Growing trees in heavy, retentive, poorly drained soils; (2) Growing trees in an otherwise suitable soil, but underlaid by an impermeable subsoil, the situation not being such as to afford ample drainage; (3) Irrigation methods allowing an excessive accumulation of water around the trees for a period of time; (4) High fertility combined with soil and drainage conditions as mentioned under 1, 2, and irrigation methods as mentioned under 3; (5) High fertility and excessive irrigation; (6) Excessive fertilization, especially with nitrogenous manures; (7) Continuous wet weather in spring.

Too much stress, however, should not be laid on any one of the above factors alone. For instance, if we take soil texture in its relation to autogenous gummosis we find that in the *Citrus*, where this form of the malady has been best studied, there is no *ne varietur* relationship whatsoever.

With these few remarks on the development of gummosis in the orchard, I pass to the study of the symptoms of the disease.

Minute. Gummosis begins and attains even a high state of development within the plant before any symptoms appear externally. Only after the gum has accumulated to such an extent that sufficient pressure has been produced to raise the epidermis, or to drive the flux pearling through the crevasses in the same, when it is no longer intact, does the disease become apparent. This is generally considered a young stage of gummosis, though in reality the disease may have developed very considerably by this time. With the further development of the malady the distended epidermis bursts and the gum flows out in greater or less amount, or, if the first external symptom was a pearling through epidermal crevasses, simply shows as an increase in the outflow.

Gummosis may affect a tree locally or generally; the fruit (Saucer Peach, almond, orange, and citron), the twigs and smaller branches, one or more limbs, the trunk, or the entire tree. In *Prunus* the disease appears usually in the larger limbs and trunk (Pl. VIII), and is generally more frequent in the neighbourhood of the crotches. In the *Citrus* gum flow appears more generally, one might almost say exclusively, upon the trunk towards the base, frequently immediately above the union of stock and scion (Plate VII). Gummosis may, however, when extremely intense, affect the entire tree in both *Prunus* and *Citrus*, gum oozing out from both the trunk and the limbs. Such generalized gummosis, though rare in other species of *Prunus*, is not infrequently observed in young cherries; in the *Citrus*, on the other hand, it is quite exceptional and has only been observed on the lemon.

When gummosis progresses sufficiently for gum to appear on the surface of the bark, the cortex in the neighbourhood of the exudate will

be found more or less permeated with gum. The death of this infiltrated bark is but a matter of time; it becomes extremely hard, cracks, curls more or less, and sloughs off.

Severe cases of gummosis are always accompanied by chlorosis and, when the malady recurs from year to year, by a marked decrease in vigour. As the disease indirectly, by dehiscing the bark, destroys the avenue of food supply to the roots, death from starvation must ultimately result. With the reduced root development consequent on the failure of the proper supply of elaborated material, there follows a decrease in the absorption of mineral nutriment, and it does not appear to me at all doubtful that it is to this reduced absorption that chlorosis of the diseased trees is due. A number of analyses of healthy and diseased *Citrus* have been made at various times with the idea of throwing some light on the cause of gummosis. Diseased and healthy trees show considerable difference in ash content, but this difference is purely a relative one, and the affected trees show not, as has sometimes been thought, selective absorption, either forced or otherwise, but rather reduced normal absorption. The following curves drawn from the data of Ricciardi and Silvestri clearly show that relative absorption is normal in diseased as well as healthy trees (Text-fig. 1).

Gummosis having been detected by means of the pustulated epidermis or the exudate, the extent of the development of the disease within the tissues may be readily followed by a series of cross-sections taken at intervals above and below the place of gum accumulation or outflow.

The internal characters of the disease are best shown on slightly affected stout branches two or more years old. A cross-section cut through the swollen epidermis or the exudate will show that the pathognomonic tissues are situated in the young wood and young wood in formation, and extend to a greater or less extent around the xylem. The diseased area is fusoid in form from the greater development of the diseased tissues near the point of gum accumulation and their lesser and lesser development as one proceeds further away. The gum exudes *en masse* from the centre of the sickle, but as one proceeds towards its extremities it will appear in droplets of decreasing magnitude, and separated with larger intervening spaces of apparently healthy tissue. The gum in the centre of the sickle is more or less tinted yellow, depending on its age, whereas that pearly from the tissues at its extremities is always colourless (Pl. IX, Fig. 3).

If sections are now cut at various distances above and below the centre of disease, it will be found that gum formation proceeds downward to a much less extent than upward. Furthermore, if we imagine a line drawn through the middle of the pathognomonic tissues, it will be found, as one proceeds upwards and downwards, that the gumming sickle diminishes in size.

GRAPHS SHOWING RELATIVE ABSORPTION OF MINERAL NUTRIMENTS
IN PARTS PER 100 BY HEALTHY AND DISEASED LEMONS.

TEXT-FIG. 1.

A.C. from the analyses of Ricciardi.

B.D. from the analyses of Silvestri.

———— CaO, MgO absorbed by healthy lemon.

----- CaO, MgO absorbed by diseased lemon.

———— K₂O + Na₂O absorbed by healthy lemon.

----- K₂O + Na₂O absorbed by diseased lemon.

———— P₂O₅ absorbed by healthy lemon.

----- P₂O₅ absorbed by diseased lemon.

———— P₂O₅ absorbed by healthy lemon.

----- P₂O₅ absorbed by diseased lemon.

The cortex remains healthy except where the gum has accumulated sub-epidermally, or broken out on the surface of the bark. In these places, as I have already mentioned, it becomes more or less infiltrated and discoloured.

Within the wood, if the branch examined is old enough, brown maculations and fusoid areas may be observed amidst the healthy tissue; they are due to attacks of gummosis at some previous period, when the now dead tissues were a part of the meristematic zone.

When the attack of gummosis is very severe the branches or trunk, as the case may be, may become completely ringed with gumming tissue, and the fusoid character accompanying mild infection is lost. The wood is more or less deeply infiltrated and discoloured and the disease gives the false impression of working centripetally.

Gummosis is characterized, as we have seen, macroscopically by a more or less copious gummous exudate on the surface of the bark, and by a zone of pathognomonic tissue in the outermost region of the xylem, which, however, is only recognized by the unaided eye when gum flows or pearls from it. The gum is, therefore, an important symptom of the disease, and, as such, is deserving of separate study.

3. CHEMISTRY OF THE GUMS OF *PRUNUS* AND *CITRUS*.

The gums¹ of *Prunus* are derivatives of the hemicelluloses and represent, to use the illustration of Grüss, the homologous stage in their hydration to that of the dextrans in the hydrolysis of starch. The changes taking place during the hydrolysis may be illustrated thus:—

araban	. . .	arabin	. . .	arabinose
starch	. . .	dextrin	. . .	maltose

The gums of the *Citrus* do not appear to have been closely studied up to the present, but, as will be seen from the following table, they behave in essentially the same manner as the gums of *Prunus* towards the various reagents, and like the latter are hemicellulose derivatives. As type of the *Citrus* gums I have taken lemon gum.

¹ The chemistry of the gums is still imperfectly known and consequently not entirely free from inaccuracies. Vide Wiesner, J.: *Die Rohstoffe des Pflanzenreiches*, vol. i, 2. Bd. Czapek, F.: *Biochemie der Pflanzen*, vol. i, 1905. The literature is cited in these works.

QUALITATIVE REACTIONS OF *PRUNUS* AND *CITRUS* GUMS.

Reagent.	Lemon gum.	Cherry gum.	Plum gum.
Hydrochloric acid and phloroglucin	bright red, then dark pp.	bright red, then dark pp.	bright red, then dark pp.
Alcohol 95 per cent. excess	white pp.	white curdy pp.	white pp.
Acetic acid, dil.	white pp.	white pp.	white pp.
Glacial acetic acid, excess	solution coloured yellow	solution coloured yellow	solution coloured yellow
Nitric acid	colour heightened	colour slightly heightened	colour heightened
" " and ammonia	solution deep red	solution yellowish	solution reddened
Millon's reagent		yellow pp.	
Tannic and acetic acids .	_____	_____	_____
Sodium molybdate	_____	_____	_____
Potassium-mercuric iodide	_____	_____	_____
Phosphotungstic and sulphuric acids	cloudy white pp.	large curdy pp.	liquid cloudy
Sodium phosphomolybdate	_____	solution thickened	_____
Sodium phosphotungstate	_____	white pp.	trace of reaction
Mercuric nitrate and nitric acid	fine pp. remaining in suspension	_____	fine pp. remaining in suspension
Adamkiewicz reaction . .	_____	_____	_____
Biuret test	_____	_____	_____
Lead acetate and sodium hydrate	_____	_____	_____
Hydrochloric acid	bright yellow, then clear amber	bright yellow, then dark brown	bright yellow then dark brown, slight pp.
Ferric chloride	red gelatinous pp.	_____	_____
Fehling solution	_____	_____	reaction doubtful
Lead acetate	milky, slight pp.	_____	milky
Ammoniacal lead acetate	abundant gelatinous pp.	abundant gelatinous pp.	abundant gelatinous pp.
Basic lead acetate,	fine curdy pp.	_____	fine gelatinous pp.
Calcium hydrate	_____	_____	_____
Potassium hydrate	_____	sol. slightly thickened	_____
Sodium hydrate	sol. thickened	sol. thickened	sol. thickened
Barium hydrate	very slight fine pp. remaining in sol. as a faint cloud.	very slight fine pp. remaining in sol. as a faint cloud	very slight fine pp. remaining in sol. as a faint cloud
Aluminium hydrate	sol. thickened, faint cloud	sol. thickened, slight indefinite pp.	sol. thickened, faint cloud
Sodium borate	sol. thickened	sol. thickened	sol. thickened
Copper sulphate	very slight fine pp. remaining in suspension	_____	_____

The gums of *Prunus* and *Citrus* I have found to be soluble in water. A 6 per cent. solution of cherry gum is very thick, but the gum dissolves with shaking within twelve hours at this concentration. The lemon and plum gums dissolve much more readily than the cherry gum, and at the same concentration as the latter form a less viscous solution. All the gums contain nitrogenous matter, probably in variable amount.

4. TECHNIQUE.

In our study of the gums of *Prunus* and *Citrus* we found that they are soluble in water, from which they are precipitated out by strong alcohol, or an excess of glacial acetic acid. In preserving material for examination it was, therefore, quite evident that alcohol and acetic acid mixtures, or

alcohol alone, must be used if the earlier stages of the disease were to be preserved. I early determined that when alcohol and glycerine, equal parts, was used as the fixing and preserving fluid, sections of shoots in which very young gum pockets were to be found always showed a great paucity of gum in the lacunae. I also observed that the glycerine-alcohol solution became more viscous when specimens had stood in it for sufficient time, and this viscosity, upon analysis, proved to be due to dissolved gum. Contrary to Mikosch's observations, then, gum is soluble in 50 % alcohol. As woody tissues are always hardened in alcohol, it became a matter of no small interest to determine what was the minimum strength of alcohol in which the gum remained insoluble. I selected a sample of fresh fluid pellucid cherry gum—in other words, gum recently formed—and placed aliquot parts of it in 50 %, 75 %, 85 % and 95 % alcohol. The gum dissolved in all but the 95 % solution. In alcohol of this strength the gum lost somewhat in volume, and became brittle and perfectly translucent, except at one corner where it was somewhat white opaque.

In acetic acid the gum is quite soluble except in a large excess of the reagent.

Farmer's fixing solution (alcohol absolute 2 pts., glacial acetic acid 1 pt.) may be used in lieu of 95 % alcohol, but the gum, instead of remaining a homogeneous mass, appears as a fine precipitate which is not very suitable for study.

Mikosch found in the course of his study on gummosis of the cherry that staining reagents were of little or no avail, chloriodide of zinc being in all cases the most trustworthy. This opinion is, however, only relatively true. The gum behaves differently towards stains when young than when old, and at its incipency its reactions cannot be made out at all, for the simple reason that it is too soluble in water; the gum passes into solution and is lost. For this stage a differential alcoholic stain would be necessary, and up to the present I have found none. If we cut our sections, on the other hand, from material showing well-formed gum pockets, and in which, even in alcoholic solution, the gum occupies the lacunae as a homogeneous mass, the gum will be found to stain with Böhmer's haematoxylin¹ usually more rapidly than the cellulose walls (*Citrus* in particular) or colour yellow in chloriodide of zinc (*Prunus* and *Citrus*). If now sections are taken through diseased material in which the gum pockets are well developed, and the gum in the lacunae has assumed centrally a yellow tinge, it will be found that Böhmer's haematoxylin stains the gum peripherally (i.e. where colourless), but has little or no effect where it already shows coloration. Chloriodide of zinc in these cases gives no valuable indications at all; it is

¹ For formula vide Zimmermann, A.: Micro-technique, p. 181. Delafield's haematoxylin (cf. Chamberlain, C. J.: Methods in Plant Histology, p. 249) is a more energetic stain, but does not give as good a differentiation.

difficult to say what effect it has upon the already yellowish gum. Lastly, if we cut a section through a gum pocket already old, and in which gum formation has not taken place for some time, and in fact is no longer capable of taking place, it will be found that the gum no longer stains with Böhmer's haematoxylin or chloriodide of zinc, but, on the other hand, is brought out more or less distinctly by lignocellulose stains—orcein and hydrochloric acid, methylene blue, Bismarck brown, fuchsin.

Other stains useful in the study of the early but not nascent stages are Congo red and aniline blue 2 v. Congo red may be used as a counter-stain with haematoxylin as it does not stain the gum; the cell-walls and protoplasm are coloured red, the gum blue. Aniline blue 2 v. stains the protoplasm only and may be used, therefore, more or less as a check upon the other stains.

Suitable material (fruit, non-ligneous stems) may be embedded in paraffin or celloidin—I think the latter preferable as it hardens less—cut and mounted as usual.

Canada balsam or Venetian turpentine may be used as mounting media, but for unstained sections their indices of refraction are somewhat high, and I am inclined to prefer a mixture composed of castor oil 2 pts. and of oil of thyme 1 pt. When, however, the sections to be mounted will bear passage through water, I use Farrant's medium.

5. HISTOLOGY.

The anatomy of gummosis has been studied (I refer to *Prunus* in particular) from time to time during the last fifty years, and our knowledge of the various appearances that pathognomonic tissues may present is now very complete as regards the later stages of the disease. Of the initial and post-initial stages, however, practically nothing is at present known,—hence very largely the lack of agreement between authors regarding the nature and origin of gum. In taking up in turn the histology of gummosis, I have, therefore, devoted considerable time to the study of these important stages with the view of answering definitely, if may be, the question: Where and how does the gum originate?

In my description of the gross internal characters of twigs, branches, limbs, or trunks affected by gummosis, I mentioned that, when the disease was not sufficiently intense to completely encircle the stem, the pathognomonic tissues were fusiform in outline. In these cases, it will be remembered, I described gum formation as abundant at the centre, but decreasing in amount and finally becoming unobservable as the apices were approached. The tissues at the centre of the sickle have been longest affected by the disease and may have reached the ultimate stage of decomposition, while those nearer the apices only show young, if not the initial, stages of gummosis. One might, therefore, readily imagine an ideal

sickle of pathognomonic tissues in which all stages of gummosis, from the initial to the large and extensive gum pockets, are to be found. In such an ideal sickle of pathognomonic tissue one would be able to follow, beginning at an apex, the malady through its various developmental stages.

Premonitory to the first symptoms of gummosis or coincident therewith, the cambium frequently lays down centripetally cells rich in granular protoplasm, though this increased protoplasmic content of the embryonic wood cells is not necessary, as Mikosch believed, to gum formation. A dense protoplasmic content is not an invariable accompaniment of gummosis, and essentially it is only an indicator of active metabolism. The importance of starch has also been greatly overestimated. It is invariably absent from the young pathognomonic tissues, and I am unable, therefore, to agree with the view that it plays an important rôle in gum formation. The cell contents take no part in the initial stages of gummosis: it will be shown as we proceed that they remain passive at all times. The cell-wall, on the other hand, is the seat of the malady *ab initio* and throughout its subsequent development.

Material fixed, preserved, and examined in 95 % alcohol will show perhaps rather exceptionable activity on the part of the cambium, and frequently an increased protoplasmic content of the embryonic wood cells, giving them a pseudo-nidulose appearance. The wood in formation, as in the case of healthy trees, is more or less distinctly enframed between the xylem, the cambium, and the medullary rays. The sections appear normal, and it would be impossible to say that they were not (Pl. X, Fig. 5). If, however, we replace the 95 % alcohol by progressively weaker solutions, finally arriving at water, we will notice that while the protoplasm remains unaffected, a change takes place in the cell-walls. The walls appear to stretch and swell, the swelling taking place in the secondary membrane. This change constitutes the initial stage of gummosis and is quite fugacious, being rapidly followed by further and more marked swelling and disappearance of the primary membrane. By further absorption of water the primary and secondary membranes, henceforth indifferentiable, increase in bulk, become semi-fluid and accumulate, preferably it would appear at the corners of the cells, i. e. at the place where a given pressure would be most likely to force the cells apart from one another (Pl. X, Figs. 6, 7). The gum, however, may also accumulate medianly, pushing the cell-walls apart, forming small elliptical cavities. Gummous degeneration of the cell-walls now continues centripetally, the third lamella rapidly dissolves away, the cell contents become part of the gum matrix, which occupies, as a homogeneous mass, the spot but a short time before filled by the degenerating cells. The cells bordering the gum pockets become markedly convex owing to the release of pressure following the disappearance of the abutting cell or cells, and the real period of the growth of the gum pocket may be

said to begin¹ (Pl. X, Figs. 4, 7). Gummosis now spreads more and more deeply into the circumambient tissues. The cells bordering the pocket are sloughed off from the subjacent cells, which then become, in turn, convex on their free ends, and finally loosened and freed by a process exactly similar to that which brought about the first formation of the gum cavity, and which may continue until all the tissue capable of gummous degeneration has been destroyed. The growth of any given gum pocket is, therefore, only limited by the amount of susceptible tissue (embryonic wood cells) laid down by the cambium, though it may develop to such an extent as to destroy the cambium, the medullary rays, and, in rare instances, the bordering cells of the xylem.

Having described in general terms the method of growth of the gum pocket, I shall now consider at some length the changes that accompany and follow the sloughing off of the cells.

The cells freed during the development of the gum pocket may be either totally destroyed or remain apparently unaffected, depending upon the rapidity with which gummous degeneration is taking place. If gummosis develops slowly or has nearly run its course, the tertiary membrane, now the only envelope of the cell, remains unaffected and life continues; but, if the disease is progressing rapidly, it gelatinizes centripetally and the cell finally vanishes, completely absorbed by the gum surrounding it.

I shall study at some length the cells sloughed off during the growth of the gum pocket.

If one examines, in a suitable medium, sections cut from material fixed and preserved in 95% alcohol, and in which there are young and actively growing gum pockets, he will observe that the gum pocket either contains homogeneous gum or is more or less filled with a mesh of thick strands, anastomosing in such a manner as to present an alveolar appearance (Pl. X, Fig. 3), which vanishes immediately when water is run under the cover-glass, being replaced by gum. I at first supposed that this structural pattern was a stage in gummous degeneration of the cells; but a careful study of a number of sections made it evident that this cellular appearance was due to an alveolation of the gum brought about by the fixative used, i. e. 95% alcohol.

In young gum pockets, cells will sometimes be observed floating in the

¹ According to Delacroix, gummosis begins towards the centre of the pathognomonic embryonic wood cells, enframed between the cambium and the xylem, on the one hand, and two adjacent medullary rays on the other, in *Prunus*, but rather nearer the cambium in the case of the *Citrus*. I have been unable to confirm this observation. Gummosis begins, I have found, now centrally, now more laterally. When, however, it begins near one of the medullary rays the extension of the disease is necessarily more unilateral (the medullary rays only becoming affected in severe cases), and, when more fully formed, the gum pocket may give the false impression of having originated near the centre. Again, if gummosis began somewhat mildly and gradually grew worse as new tissues were laid down, we might obtain the equally false impression that the disease had worked centripetally when, in reality, it had worked centrifugally.

gum. These cells immediately vanish upon the addition of water, but without any perceptible change taking place in their protoplasm (Pl. X, Fig. 6, section through gum pocket observed in alcohol ; Fig. 2, same section after being placed in water : three cells have vanished).

If now we examine a gum pocket which is no longer developing actively, we will observe at its periphery, or scattered promiscuously within it, a certain number of free cells that no longer vanish upon the addition of water (Pl. X, Fig. 8). In studying these cells we find, as hitherto, that the protoplasm plays no rôle in gummosis. The *Citrus* are best suited for the study of this stage as the gum in the pockets does not dissolve very rapidly, and staining may be resorted to. In sections stained with Böhmer's haematoxylin, the gum colours more rapidly than the cell-walls, and the contents of the cells remain unaffected. Sections stained in Böhmer's haematoxylin may be counterstained with Congo red ; the gum then appears blue, as before, but the protoplasm is always coloured red, as likewise the healthy cell-walls. If on the other hand we stain sections in aniline blue 2 v. the protoplasm only will be coloured. With Böhmer's haematoxylin on the one hand, and aniline blue 2 v. on the other, we are able to determine exactly when we are dealing with gum and when with protoplasm. We, therefore, arrive at the conclusion that the cell contents are free from gum and take no part in its formation from the initial stages of gummosis until they finally vanish upon the gelatinization of the tertiary membrane. With the dissolution of this membrane the protoplasm is added to the gum.

We have seen that when a gum pocket is to be formed the following changes take place: (1) a susceptible tissue, in reality embryonic wood cells, is laid down by the cambium ; (2) at no very determinate position the secondary and primary lamellae of certain of these cells gelatinize and form a nucleus, as it were, for the future gum pocket ; (3) the gelatinization of these lamellae detaches the cells one from the other ; (4) the third lamella gelatinizes and the cells vanish in the gum mass ; (5) the gum is composed of gelatinized cell-walls and of the contents of the dissolved cells (i. e. protoplasm), together forming a homogeneous mass. We have still to describe the changes that take place in this susceptible tissue when the growth of the gum pocket is arrested before it has attained its ultimate development, that is to say, before all the cells predisposed to gummous degeneration have become affected.

Following the arrestation of growth of the gum pocket, and this may occur at any time during its development, the cells of the susceptible tissue lignify. The cells growing into the gum, to use figuratively a phrase that has been erroneously employed in the absolute sense, as well as those bordering the gum pocket, together with the remaining cells of the susceptible tissue, gradually thicken their walls. The lignification of the susceptible tissue cells, however, is never as complete as in normal xylem,

and the extent of this tissue formation can always be traced by this character, as well as by the asymmetry of the cells due to the pressure exerted upon them by the gum, which occupies more space than the cells from which it is derived.

Coincidentally with the lignification of the susceptible tissues, starch may appear within the cells ; it will be observed also in the cells within and bordering the gum pocket. I have never observed any quantity of starch in these cells myself, though there is no reason for supposing that it cannot accumulate to a considerable extent in them. The starch is laid down in these tissues when the trees are accumulating food reserves ; its production cannot be considered, therefore, a pathological phenomenon.

Before lignification sets in in the susceptible tissues the gum may undergo changes. I have already pointed out that it becomes yellowish in the more highly developed gum pocket and at the same time less soluble than when colourless ; it will also be remembered that at this stage it no longer stains with Böhmer's haematoxylin. I may now add that this loss of power of fixing haematoxylin is brought about by a gradual increase of affinity for lignocellulose stains, and a further decrease in solubility. Finally, after lignification of the enclosing cells has taken place, the gum becomes apparently quite insoluble, and takes the lignocellulose stains as well as the xylem, or even better.

The changes that take place in the gum parallel very closely those that accompany the lignification of the cell-walls, and we may draw the conclusion that, whether we are dealing with the changes that take place in the cell-walls of the susceptible tissue that do not degenerate into gum, or with the gum that has originated from the cell-walls of certain of them, the ultimate changes are similar in both cases ; the cell-walls lignify and the gum assumes all the characters of lignocellulose.

As soon as the susceptible tissue ceases to be affected by gummosis the conditions favourable for its development no longer exist, and the cambium assumes normal activity. Healthy wood cells are formed once more, and a certain development of xylem may have enclosed the gum pocket and surrounding tissues before lignification has set in in them (Pl. X, Fig. 1).

6. CAUSE OF GUMMOSIS.

Prunus and *Citrus* species, broadly speaking, are susceptible to gummosis whenever conditions are favourable for active growth of the cambium. It will be remembered that the disease may be produced either autogenously or by external agents. The autogenous form of gummosis appears to be confined very largely to the cherry and the lemon, and is induced when vigorous growth is accompanied by an excess of water in the substratum. In the lemon and cherry this form of the disease develops severely under these conditions, and is not induced by injuries of one kind or another, as

one may gather from the fact that the outflow of the gum does not correspond to places of injury, as it always does when the latter are the inciting cause. The autogenous development of gummosis is, however, much less common than that resulting from direct injuries to the cambium.

At one time it was believed that gummosis was due to one or, at most, several forms of injury, but to-day it must be admitted that, provided the plant is in the proper condition when wounded, any traumatism will produce the malady. This will be clearly shown in the following examples.

1. Gummosis is produced by a variety of parasitic Fungi, to wit:—*Coryneum Beyerinckii*, which is capable of producing marked gummosis in the peach; ¹ *Clasterosporium carpophilum*, Lev. Aderh.², a common parasite of the cherry, plum, peach, and almond; *Sclerotinia fructigena*, (Pers.) Shroet.; *Cladosporium epiphyllum*; ³ *Valsa leucostoma*, Pers.; *Tubercularia vulgaris*, Tode; *Botrytis cinerea*, Pers.; ⁴ *Plowrightia morbosa*, (Schw.) Sacc.; *Exoascus deformans*, (Berk.) Fuckel.⁵

2. According to the researches of Aderhold and Ruhland ⁶ on the one hand, and of Brzezinski ⁷ on the other, it would appear that Bacteria also produce gummosis; the former have described a species of *Bacillus* that induces the malady in the cherry, and the latter has isolated a Bacterium from the peach, plum, and apricot which he believes is the cause of the disease in these trees.

3. Certain insects or their larvae also cause more or less copious gumming: the peach-borer, the curculio on the fruit of plums, the larvae of *Tortrix woerberiana*, Shiff.⁸ Gummosis may also be produced by piercing the young shoots near the apex with a needle,⁹ tearing the bark, knife-wounds, bruising the bark with a mallet. This latter method I have tried experimentally on orchard trees, but not with very marked success. I hammered the bark of peach, cherry, and plum limbs, being careful not to cause rifts. Gum appeared on the surface in 10 % of the wounded cherries and in 50 % of the wounded peaches and plums.

4. Gummosis may be produced in young shoots by burning with a hot iron, and freezing; chemical agents are, however, very much more effective. I have successfully used sulphuric (Pl. VIII, upper left-hand figure), phosphoric, nitric, and lactic acids; acetic acid, on the other hand, has given me

¹ Smith, R. E.: Peach Blight. California Agr. Expt. Sta. Bull.

² Aderhold, R.: Über *Clasterosporium carpophilum*, &c., loc. cit., ante.

³ Massee, G.: A Textbook of Plant Diseases, p. 306.

⁴ Rant, A.: De Gummosis der Amygdalaceae.

⁵ Pierce, N. B.: Peach-leaf Curl: its Nature and Treatment. U. S. Dept. Agr., Div. Veg. Path. and Phys., Bull. xx.

⁶ Aderhold, R., and Ruhland, W.: Der Bakterienbrand der Kirschbäume. Arbeiten kaiserl. biolog. Anstalt, v, 1907, pp. 293-340.

⁷ Brzezinski, P. J.: Étologie du chancre et de la gomme des arbres fruitiers. Comptes Rendus, cxxxiv, 1902, pp. 1170-3.

⁸ Rant, A.: loc. cit.

⁹ Beijerinck, M. W., and Rant, A.: loc. cit., ante.

no results, owing probably to the fact that it volatilizes before penetrating the tissues. Oxalic acid appears to have been successfully used by Sorauer.¹ The disease may also be produced by the alkalis, and I have successfully used potassium hydrate. Corrosive sublimate was found very effective by Beijerinck and Rant.² I have tried the hydrocarbon kerosene, but apparently it is not sufficiently penetrating.

7. NATURE OF GUMMOSIS.

We may now inquire, What is the nature of gummosis? In recent years several hypotheses have been advanced to account for the formation of gum within the plant. Beijerinck and Rant³ supposed that traumatism, chemical substances, or fungous parasites acted in an exactly similar manner. The cells of the cambium, according to the view of these authors, contain a cytase, which, while they remain alive, is unable to attack the cell-wall, owing to the semipermeability of the protoplasm. When, however, any cells of the embryonic wood are killed by penetrating hyphae, traumatism, or a toxic agent such as mercuric bichloride, the contained cytase diffuses out and is able to attack the walls of the circumambient healthy cells, which become gummous, and finally dissolve away. More cytase is thus released, attacks other healthy cell-walls, and in this manner the gum pocket is formed. The authors further point out that mercuric bichloride yields more gum than a traumatism; and this they interpret as strengthening their hypothesis, for mercuric bichloride kills more cells than a simple traumatism. As further proof of the existence of a cytase they point out that the hyphae of *Coryneum Beyerinckii* are affected by gummous degeneration, as well as the cells amidst which they are growing.

Certain objections can be raised against this hypothesis of Beijerinck and Rant. If gummosis is due to the action of a cytolytic enzyme diffusing outwardly from necrobiotic cells it seems to me that all sources initiating gummosis should distinctly tend to cause ultimately an equal development of the disease.

Let us imagine, for simplicity's sake, that the embryonic wood cells occupy a plane figure instead of a solid, i. e. a rectangle instead of a parallelepiped. We will call the base of this rectangle B , and its height H . Now if we kill the tissues somewhere within BH so as to have an area of necrobiotic cells equal to BG , then the cytase formed will diffuse in two directions only. But if we kill the tissues at a point P , the diffusion of the cytase will proceed in all directions and gummosis will develop with increasing rapidity until an area BG' is affected. When this stage in the development of the malady is reached its further progress will be neither more nor less rapid than in the first case considered. The ultimate development of all gum pockets should be, therefore, approximately the same.

¹ Loc. cit., ante. ² Beijerinck, M. W., and Rant, A.: loc. cit., ante. ³ Loc. cit., ante.

It may also be pointed out that the death of a cell is not necessary, as we have already seen, for the development of gummosis. Nor could we, under the cytase hypothesis, have live cells protected only by the tertiary lamella lying unaffected in the gum. Nor does it appear possible to explain the greater extension of the gum pockets upwards from the centre of initiation if diffusion of cytase from the dead cells is the cause of gummous degeneration, for a solution tends to diffuse equally in all directions. The lesser development of the gum pockets as one proceeds laterally of the inciting centre is also difficult to explain, for the medullary rays are very resistant to the disease, and, therefore, arrest lateral diffusion; but allowing, for argument's sake, the intercommunication of all the gum pockets, the asymmetrical oval development of the tissues affected by gummosis is not explainable on the hypothesis of a cytase diffusion from necrobiotic cells. For these reasons, and for others that will be developed later, I am unable to accept Beijerinck and Rant's explanation.

Ruhland's¹ hypothesis that gum is formed by oxidation of the carbohydrate substances within the cells that go to the making of the cross-septa after division, and of the pectins and pectinates of the cell-walls, is not tenable, as a chemical study of the gums clearly shows that they are not oxidation products of carbohydrates; and for the same reason Sorauer's² notion that the increased gum flow induced by oxalic acid was due to oxidation is equally erroneous.

It being, therefore, impossible to accept the views heretofore held regarding the nature of gummosis, it will not be out of place to determine whether or not an explanation may be advanced that does not conflict with any of the observed facts. Our task then will be, first, to correlate the observations recorded in the previous pages, and second, to show that the explanation I would substitute for those of Beijerinck and Rant, Ruhland, and Sorauer satisfactorily explains this correlation.

1. For gummosis to develop two conditions must be simultaneously fulfilled. The cambium must be actively laying down new tissue elements, and a superabundance of water must be present in the soil.

Though neither growth nor a superabundance of water can of themselves induce the disease, their rôle in the development of gummosis may be separately discussed if we are careful to continually bear in mind that neither is operative without the other. I shall, therefore, consider under separate captions the relation between growth and gummosis, and the relation between water and gummosis.

Relation between extent of gummous degeneration and growth. When gummosis is artificially produced by means of sulphuric acid, for instance, we find that the extent to which the disease develops is very closely related to the amount of growth taking place at the time of the wounding. Gum-

¹ Loc. cit., ante,

² Loc. cit., ante,

mosis develops freely in actively growing shoots, but less readily and extensively when growth is no longer active.

The relation of growth to gummosis is also brought out by a study of the anatomy of the disease. The gum pockets are formed, it will be remembered, in a sickle of susceptible tissue laid down symmetrically on both sides of the wound. This tissue will be found, upon close examination, to show all the characters, especially noticeable in the medullary rays, of having been rapidly formed. The greatest growth will have taken place in the immediate neighbourhood of the wound, the least at the edge of the sickle. The most extensive gum pockets occur necessarily in the area of greatest growth. If now one studies sections taken from diseased shoots that have shown various degrees of growth activity, he will observe that the size of the sickle of susceptible tissue formed is directly proportional to the rapidity of growth; he will also observe that the gum is the more fluid the more sapid the tissues.

The question will be asked, Why is the growth of the susceptible tissues more marked in the neighbourhood of the wound, and less and less extensive as one proceeds nearer to the apices of the sickle? It is generally supposed that this is due to a response of the plant to traumatic stimuli which are necessarily more marked near the place of origin, but an explanation less vague, I think, can also be offered. It is a matter of common observation that, when a ligneous branch is pruned in the proper position with respect to a bud, a callous tissue forms and the bark becomes slightly raised; again, when the trunk of a vigorously growing tree is slit longitudinally, marked growth occurs, and the two lips of the bark are pushed apart. In these two cases, what, in reality, takes place? It appears to me simply this. The cambium is capable of laying down new tissue elements with extreme rapidity under favourable conditions of growth, but, owing to the pressure exerted by the cortex, the number of cells laid down in a given time are fewer in number than would be the case if the pressure were removed. When, therefore, through any kind of a wound, the pressure exerted by the cortex is more or less greatly reduced the genetic power of the cambium proportionally increases. As the pressure of the ruptured bark from approximately zero will increase gradually as one passes away from the wound, finally reaching the height it had attained before the release was effected, it must necessarily follow that the activity of the cambium will suffer a gradual reduction, and we are prepared to understand the fusoid development of the susceptible tissues. In a similar manner we will be able to explain the greater length attained by the gum pockets upwards from the point of initiation than downwards. The growth of the cambium depends on the amount of elaborated material that the phloem is able to supply, and it must necessarily follow that when it is destroyed the cambium below the lesion must decrease in activity. The amount of

susceptible tissue produced below the wound will be less extensive than that which is laid down above. An elliptical development of xylem tissue round a wound is, therefore, a perfectly normal phenomenon, but whenever the tissues are at the same time gumogenetic, as in *Prunus* and *Citrus*, we must consider this character as an acquired one. Growth and gummosis are nevertheless directly correlated.

Relation between extent of gummous degeneration and water. The relation between gummosis and growth is direct, the relation between gummosis and sapidity of the tissues indirect. Sapidity of the tissues is insufficient in itself for the development of the disease, but as soon as it is combined with the necessary factor, growth, we may say that its rôle changes: it will then govern the extent of gummous degeneration.

The effect of a high water-content of the soil on increasing the susceptibility of *Prunus* and *Citrus* to gummous degeneration has been mentioned by most writers on gummosis, its significance, peculiarly enough, only being overlooked by those who have studied the histology of the disease. Amongst this latter class, however, we find an exception in Trécul,¹ who states that the disease arises from a too abundant accumulation of sap at any point, this accumulation being due to a variety of causes, rain being the only one that he specifically mentions.

In one of Aderhold's² infection experiments with *Clasterosporium carpophilum*, we find it mentioned that the gum produced in infected wounds largely increased after watering. The trees experimented on were peach, apricot, and cherry.

In the case of *Prunus* the horticultural writers agree that excessive soil moisture favours the development of the disease. Gummosis occurs in retentive, poorly drained soils; in non-clay soils underlaid by an impermeable subsoil and unfavourably situated as regards drainage; under abnormal climatic conditions, such as a very wet spring, or long sequence of cloudy weather inducing a rupture of equilibrium between transpiration and absorption.

In the case of the *Citrus* the opinions of the writers who have studied the disease in this genus are no less emphatic. Excessive humidity of the soil, they all agree, is very favourable to the development of gummosis. Whether the trees are grown in poorly drained retentive clays, or upon an impermeable subsoil, or are subject to excessive irrigation, the result is the same—the trees succumb to the disease in a comparatively short time. The climatic conditions favourable to the development of gummosis in *Prunus* are also conducive to its outbreak in *Citrus*.

It is, therefore, clear that growth and a high water-content of the tissues are necessary for gummous degeneration. We have now to determine to what degree our studies on the nature and origin of the resulting

¹ Loc. cit., ante.

² Loc. cit., ante.

gum are satisfactorily explained on the basis of a growth and water relation.

In our study of the gums it was clearly brought out that they were composed of hydrated carbohydrates and nitrogenous matters. From our histological studies we are able to assign the origin of the hydrated carbohydrates to the cell-walls, the nitrogenous matters to the cell contents. The power of diseased cell-walls to swell upon the addition of water, to contract on dehydration, and of the gum to change from a hemicellulose, when first formed, to a xylose-like substance with the lignification and dehydration of the surrounding tissues, all these facts point to the conclusion that gummosis is due to a hydration of the embryonic wood cells. The importance of the water relation is, therefore, capital. If not enough water is present in the tissues to completely hydrate the affected cell-walls, we will find cells floating free in the gum, or only partly detached from one another, in fact any of the changes described in the section devoted to the histology of the disease.

Growth and high sapidity of the tissues are essential for the development of gummosis. Must we assume also the concomitant action of a cytolytic enzyme? Let us see what the consequences of such an assumption would be.

If we suppose that the gum is formed as a result of enzyme action in conjunction with the essential factors, growth and water, we would find serious difficulties in explaining the unilateral excretion of the cytase, the form that must be assumed if we are not to enter into conflict with the conditions described in our histological study of the disease. Again, the cytase excreted from the protoplasm would necessarily permeate the third lamella, and, while hydrolysing the secondary membranes, would also be acting upon it. But microscopic observation shows that gummous degeneration proceeds centripetally in the cell-walls, and yet we know that the cells, under certain conditions, will vanish instantly when sections are placed in water, without the third lamella showing signs of diffusing into the cell lumen. If an enzyme does induce, in the presence of water, hydrolysis of the cell-walls, for it to act in the manner observation shows that it does it would have to attain the power of action only after having diffused out into the secondary and primary lamellae, thus only being able to attack the third membrane, which it permeates, after having hydrolysed the former, and that in a centripetal manner. Such a mode of action, it must be admitted, would be indeed very peculiar. Again, if the gum originated through enzymatic hydrolysis of the cell-walls, we would have to suppose that, with a change in the water or growth relation, it was acted on by another enzyme excreted from the cells, and transformed gradually into an insoluble xylose-like compound. The cells of the susceptible tissue would first have to excrete a hydrolysing enzyme, and then an enzyme capable of bringing

about recondensation. Such phenomena of solution and recondensation are perfectly well known in the plant kingdom, but I think that to ascribe gummosis to such an analogous process is hardly warrantable. It seems to me, therefore, that gummosis degeneration is to be explained in some other way than by assuming the pathological action or excretion of an enzyme acting in the presence of the factors growth and water. But how shall we explain it? I am inclined to believe that were the genesis and development of the cell-wall better known gummosis could be explained on a purely physico-chemical basis, but to venture to do so in the present state of our knowledge would have but a single issue: failure.

8. PREVENTIVE AND REMEDIAL MEASURES.

It will be well, before discussing the preventive and remedial measures employed against gummosis, to recapitulate briefly the conditions under which the malady develops.

Gummosis affects trees planted in moist retentive soils and poorly drained soils; excessive irrigation is inducive in any soil, but particularly so in those that, either from their situation, the nature of their subsoil, or their physical composition, are classed as moist soils. Trees planted in rich soils, or highly fertilized, especially with nitrogenous manures, are more subject to the disease, *ceteris paribus*, than those grown in poorer soils and in orchards less intensively cultivated. Plantations in which intercalary cultures requiring a large amount of water and high fertilization, i. e. vegetables, are grown soon become decimated. Various parasitic Fungi, boring and gnawing insects, wounds due to pruning, hailstones, sun-scald, accidental abrasions during cultural operations due to ploughing, cultivating, or fruit-gathering, inordinate pruning during vegetative activity, unequal growth between stock and scion, planting the trees in such a manner that the union between stock and scion occurs below ground—to all these various agencies gummosis has been justly ascribed. In the following remarks on preventive and remedial measures, however, some of these causes of gummosis will not be further considered. When the cause of the disease is traceable to Fungi or insects, sun-scald, or abrasions incidental to cultural operations, the remedies are obvious, and therefore need no particular consideration.

Preventive measures. As gummosis does not occur unless the affected trees are growing rapidly and their tissues are, at the same time, sapid, there will be four possible methods, and they have all been employed, of preventing the development of the disease.

1. Drainage, and, when irrigation is practised, more rational distribution of the water. In draining an orchard, or land intended for an orchard, care should be taken to make the drainage efficient; but whether this shall be accomplished by tile draining, open ditches, by growing the trees on ridges, or by combining two or more of these methods, is a question

which the grower must solve for himself. I may observe, however, that excessive humidity being harmful as regards gummosis only during the vegetative period, it is quite evident that the distribution of the rainfall throughout the year will have to be considered in planning a drainage system.

As regards methods of irrigation, the grower should aim at maintaining in his soil the proper moisture for growth by frequent irrigations. Infrequent and copious irrigations should be avoided, as they produce recurrent periods of saturation favourable to the development of gummosis, which, under such a cultural regimen, if once induced is particularly difficult to cure. In heavy retentive soils flooding ought never to be used and should be replaced, where employed, by a furrow system of irrigation. In light well-drained soils flooding may, of course, be used without danger, which is fortunate, as frequently it is the only satisfactory method of irrigation for such soils.

2. Resistant stocks. It was early observed that, *ceteris paribus*, the various species of *Citrus* differed widely in their resistant power to gummosis, but in *Prunus* we have absolutely no reliable knowledge regarding the susceptibility of the various species. It has, however, been pointed out by M'Intosh that grafting the cherry on a stock that did not develop equally with the scion was more conducive to gummosis than when they were of equal growth, and there is no reason for assuming that the same observations would not apply to the other species of *Prunus* and *Citrus*.

The relative susceptibility of the different species of *Citrus* to gummosis has only been empirically determined, and, consequently, the exact position certain of them should occupy is a matter of some doubt. One may expect, therefore, that careful study will reveal certain minor inaccuracies in the following resistance scale :—

Citrus trifoliata, maximum resistance.

C. amara

Rough Lemon (*C. Limonum* × *C. decumana*?)

C. Limetta

C. bergamia

C. nobilis

C. decumana

C. Aurantium

C. medica

C. Limonum, resistance zero.

} intermediate resistance.

As resistance stocks *C. amara* and Rough Lemon are the most used at the present day. It would seem that *a priori* the stock most resistant to gummosis should be used exclusively, but it is found in practice that another factor is of immense importance. The cultivated *Citrus* frequently form better trees on one stock than on another, hence the necessity of

sacrificing, even at some hazard, a certain amount of resistance power for better affinity. It should also be pointed out that in soils in which *C. Limonum* on their own roots are only slightly affected by gummosis, there is no object in using the very resistant *Citrus* as stocks; the question of affinity should alone be considered.

3. Moderating growth by root-pruning. This method of preventing gummosis is mentioned by McIntosh and *per se* is unobjectionable. The difficulties of applying it and the complications liable to result from its injudicious use are, however, sufficient to prevent it being ever more than a gardener's palliative.

4. Amendments. The amendment mostly employed in preventing gummosis is lime, and its use is advocated by a number of horticulturists. Its beneficial action is probably due to the increased porosity of the soil following the flocculation of the clay. Salt has also been recommended by Van Hecke, who observed in China a peach orchard which, though covered at flood-tide by salt water, remained quite free from gummosis, whereas an orchard irrigated by river water was badly affected. Van Hecke was particularly struck by the fine appearance of the orchard submerged at flood-tide, and, upon his return to Belgium, tried the following instructive experiment. He selected¹ four peach-trees growing under similar conditions, and treated them as follows immediately before the opening of the buds:—

No. 1. Received 1,500 grm. salt.

No. 2. Received 1,000 grm. salt.

No. 3. Received 500 grm. salt.

No. 4. Received 0 grm. salt (witness).

The result of the experiment was most striking; the witness gummed considerably and even lost several branches, the trees that received one half and one kilo of salt respectively were only very slightly diseased, and the remaining tree was not affected. Van Hecke's observation and subsequent experiment remind one of the remark of Downing's that salt is one of the best fertilizers for the plum.² Salt as a preventive of gummosis could be made, it seems to me, the subject of further inquiry.

Besides the major methods of preventing gummosis just described, there are several minor ones that are deserving of mention.

1. In the district of Carcagente,³ Spain, where the soil is somewhat sandy and very permeable, and the orchards are presumably not over irrigated, as the water has to be drawn from rather deep wells, the oranges, grafted on limes or citrons, are planted sufficiently deeply for the scion to

¹ Hecke, E. van: Le sel de cuisine et les arbres fruitiers à noyau. Journ. des Soc. Agric. Brabant et Hainault, 30 mars 1907.

² Downing, A. J.: Fruits and Fruit Trees of America, p. 266, ed. 1850.

³ Vide Trabut, L.: L'Oranger en Algérie. Direction de l'Agriculture gouv. gén. Algérie, Bull. 44, 68, 1908.

develop adventitious roots. When these roots have become sufficiently stout to support the tree, the soil is removed from around the crown, and the trunk is severed immediately below their insertion. The basin formed by removing the soil from around the crown of the tree is not filled in, and the base of the trunk remains permanently exposed to the air. The irrigation water is, of course, never allowed to flow into this basin, and the rains are never sufficiently abundant to maintain a dangerous humidity round the roots. This method of the Carcagente growers has, horticulturally, its justification; oranges produce better fruit and grow to best advantage on their own roots, or, which is the same thing, when grafted on orange seedlings. From the physiological point of view the increased resistance to gummosis obtained by growing the oranges with their crowns above ground is probably due to the fact that no change in the pressure of the bark upon the cambium, which is diminished when the trunk comes in contact with soil, occurs. When kept moist the bark becomes more sapid and, therefore, more elastic; the cambium develops more freely, susceptible tissue may be laid down, and gummosis follow.

2. It was early observed that the resistance of the *Citrus* to gummosis when grafted on appropriate stocks was markedly increased by high budding, and this method of still further reducing susceptibility to the disease has been advocated by the best writers on *Citrus* culture. High budding is successfully and extensively used at the present day. The fact that high budding in itself is capable of ensuring relative immunity to gummosis, even when the stock used is of low resistance, does not appear, however, to have been sufficiently emphasized. For instance the lemon, badly affected by the disease when budded low on the orange, will be quite resistant, *ceteris paribus*, when budded high on the same stock.

Remedial measures. The important preventive method drainage is also remedial, not an absolute remedy in all cases, but unquestionably the only one, under conditions favourable for the development of gummosis, that is really efficient and can effect *per se* a permanent cure. The conditions necessary for the development of gummosis are growth and a superabundance of water in the soil, or in other words very sapid tissues. Without the concurrence of the two essential factors growth and water, the development of gummosis to any marked extent cannot take place. The importance of drainage as a remedial measure is, therefore, capital. That in certain soils and situations, under particular climatic conditions or cultural methods, drainage does not prove an absolute safeguard is to be expected from the nature of the office it has to perform.

Besides drainage a number of remedies have been proposed for gummosis, but they are in reality alleviations only. I may mention root-pruning, which has already been referred to as a preventive, and slitting the bark of affected trees. This latter method is very generally and

successfully employed in cases where trees are only subjected at one season of the year, or irregularly and accidentally, to conditions favourable to the development of gummosis. The bark of the affected trees may be slit cross-wise, longitudinally, or spirally. For cases of generalized gummosis this latter method is the only efficient one.

It is a common practice in the treatment of gummosis, and one that has been advocated in the literature, to cut the bark away on each side of the exudate until clean healthy tissues are reached, removing at the same time all infiltrated and discoloured wood. The wounds thus formed, and they frequently grow to an immense size, are then covered with wax, iron sulphate solution, a mixture of tar and phenol, Saint-Fiacre ungent, and a variety of other substances. This practice has, however, like most empirical remedies, more objectionable than unobjectionable features. The removal of the discoloured infiltrated wood is absolutely without justification, no matter how severely the trees may be affected by gummosis, and cutting away the bark is only advantageous after it has been killed, for we know that as long as the cambium remains alive it is capable, upon the re-establishment of normal conditions, of laying down xylem again immediately over the susceptible tissues or the gum masses resulting from their degeneration. The removal of the cambial layer while it is still alive is, therefore, a serious error. Dead bark, on the other hand, should be cut away as a matter of hygiene, and also that the healing tissues may grow more easily over the area covered by it.

The tissues exposed by the removal of the dead bark may be covered with shellac, or any other suitable substance provided it contains neither free acid nor free alkali.

SUMMARY.

I. Gummosis of *Prunus* and gummosis of *Citrus* are indistinguishable maladies. They are identical in histological development; they are identical in their causal relationships; no species in either genus is entirely immune to the disease, though in both the malady predominates in one (*Citrus*) or several species (*Prunus*); in both genera we find species in which the malady is predominately one of the fruit (citron and Saucer Peach)—in a word, any manifestation of gummosis observed in one genus will be found in replica in the other.

II. Gummosis is due to hydrolysis of the walls of the embryonic wood cells, which develop into a susceptible tissue. The dissolution of the cell-walls begins in the secondary lamella and almost coincidently 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.

III. The cell contents are at no time actively concerned in gum

formation. Starch, it should be emphasized, contrary to the prevalent view, plays no rôle whatsoever in gum formation.

IV. The secondary lamella of the wood fibres and that of the vessels, as well as parenchymatous tissue, may show gummous degeneration in severe cases of gummosis when they are near the zone of active development of the malady.

V. Gummosis develops autogenously and is induced by all manner of traumatisms, provided they act directly or indirectly as growth stimulants to the cambium. Once incited the simultaneous concurrence of two conditions, one physiological, 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 is inoperative alone.

III. SQUAMOSIS.¹

Syn.: Scaly bark.

I. HISTORICAL.

Squamosis is at present only known in the *Citrus* groves of Southern California and Florida. The malady was probably first observed in California prior to 1880, but it has always remained so inactive that little or no attention has been paid to it. In Florida, on the other hand, squamosis has recently excited some interest, though its general behaviour in that State must be very much the same as in California, since it was first observed there about 1860.

2. DESCRIPTION.

General. Squamosis is primarily a disease of the orange-tree; in fact, the malady does not appear to have been observed on the other *Citrus*. The disease always develops sporadically, and the conditions that favour its development appear only to occur at infrequent and long intervals of time. I have a note regarding an orange grove in which, during a period of twenty years, no new trees became affected.

Besides the infrequency of its appearance in *Citrus* groves, squamosis has another marked peculiarity: the disease develops extremely slowly on the affected trees. The scaling of the bark, which is the striking and characteristic symptom of squamosis, first appears as a single scale or group of scales upon the trunk or limbs, the area of affected tissues gradually growing from this small beginning until the trunk or the limbs are completely girdled. An affected tree may live for fifteen to twenty years, but

¹ BIBLIOGRAPHY.

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this probably only occurs when the disease begins in the trunk, for if it starts in the smaller limbs these latter are soon girdled and chlorosis sets in, followed later by progressive death from the apex downwards (Pl. VII, lower figure). Squamosis on limbs has practically destroyed one half the tree). When the disease begins in the trunk or large limbs chlorosis appears very much later and is much less intense; in these cases the foliar appearance of diseased trees is not very indicative. In fact one might say that squamosis under these conditions, may be present in a grove without the affected trees showing, to the casual observer, any pathognomonic symptoms, a scaling of the bark, though abnormal in the *Citrus*, being sufficiently common in trees in general not to excite any particular fears, especially since the disease is not correlated with definite environmental conditions. Squamosis may be observed in light soils and heavy soils, in dry soils and moist soils, but apparently never when the conditions are favourable for the development of gummosis; it is, however, a form of this latter malady, and from this fact we are necessarily led to the conclusion that its development must also depend on a growth and water relation.

Minute. The characteristic of squamosis, i. e. the scaling of the bark, has already been briefly mentioned. I shall now describe in greater detail the symptoms of the disease.

Squamosis first appears, as has already been mentioned, as a rounded or an irregular exfoliation of the bark, about an inch or somewhat less in diameter, which is pushed out by the growth of the subjacent tissues upon which it stands in slight *alto rilievo*. The detached bark soon dies from the periphery inwards, and curls more or less. The curling of the dying bark reveals the subjacent cortical tissues, which appear rugose, white, or white with a yellow tinge, and somewhat mealy. When the bark finally falls a more or less highly developed pustule will be found occupying the centre of the exfoliated area. The centre of the pustule is navicular, and gum infrequently oozes from it in small amount. Not infrequently the pustular outgrowth, especially after the disease has attained a certain development, becomes less prominent and is replaced by a general swelling of the sub-epidermal cortical layers which causes the bark to flake off in large strips (Pl. VIII, upper right-hand figure). In Florida the shoots are also affected by squamosis, but in California this form of the disease is confined to water sprouts coming from the neighbourhood of diseased tissues. The shoots are discoloured subepidermally into rather well-defined shield-like maculations somewhat raised above the surface. With age these discoloured maculations become indurated, darker in colour, and further raised above the normal surface of the bark. The epidermis ruptures around the diseased areas, which then present, even more markedly than before, a scuteliform appearance. When the shoots are more seriously affected the maculations lose their definiteness of outline, become larger, and the

epidermis apparently less brittle, for it rifts within the diseased areas and not at their boundary. The rifts are parallel to the axis of the shoot, more or less numerous, and inclined to be labiate. In these cases the pathognomonic tissues form marked swellings on the shoots.

When diseased limbs are observed in cross-section they will appear more or less zoned with brown, and rings of growth will be quite prominent. The dark zones indicate the beginning of a new growth annelid. In some cases, however, the zonation may be somewhat masked by a more general discoloration of the wood. The innermost zone of brown indicates when squamosis first affected the tree, but the innermost extent of wood discoloration should be considered as indicating a certain amount of centripetal infiltration. In the youngest wood it will be observed that the pustules are composed of an outer layer of normal xylem enclosing a thin-walled xylem parenchyma ; the whole structure would remind a mycologist of a young sorus. When the shoots are examined in cross-section they may show a similar zonation to the older branches, but instead of many dark circles or partial circles, there will be generally only one, sometimes two, rarely more ; the bark shows infiltrated yellowish brown areas which appear to be cut off by suber, and, when the shoots are old enough, rings of growth are plainly discernible.

3. HISTOLOGY.

I have mentioned that squamosis has characters in common with gummosis ; we have also seen that it has characters particular to itself. In the following histological sketch, I shall consider squamosis in the former rather than in the latter aspect.

Gum pockets are the histological characteristic of squamosis. As in gummosis, they are formed in a susceptible tissue, but do not develop to the same extent except in transitional stages. In squamosis the gum pockets are typically inextensive, and the medullary rays are not, as a rule, affected ; nor do the cells become totally destroyed to the same extent as in gummosis. The secondary and primary membranes are affected by gummous degeneration, but only a few cells suffer complete dissolution. The failure of the gum pockets to develop as in gummosis is undoubtedly due to the fact that conditions favourable to gummous degeneration are transient, as is shown by the gum in young pockets being largely insoluble and soon tinged with yellow. Furthermore, lignification soon begins in the susceptible tissue, and no sharp distinction between it and the xylem can well be drawn ; one might readily be deceived by the appearance into believing that it was xylem parenchyma that suffered gummous degeneration and not embryonic wood cells.

Circular rows of the gum cavities are produced with considerable regularity at the beginning of every new annelid of growth, and it is, therefore, to them that the zonation of the xylem is due.

In the trunk and limbs, besides the gum pockets just described, we also find, corresponding to the pustules, large almond-shaped areas of susceptible tissue being laid down, degenerating somewhat and then lignifying. Over this tissue normal xylem may be formed, or the cells, while regularly laid down, may remain comparatively thin-walled and show rows of gum pockets wherever an increase in the rapidity of growth takes place. It should be noted, *en passant*, that localized areas of rapid growth occurring quite close together may be frequently observed in the xylem without the accompaniment of gummous degeneration, thus proving that the diseased trees function most irregularly.

TEXT-FIG. 2. Cross-section through edges of an almond-shaped area, showing development of ligneous parenchyma and one of a row of gum pockets beyond which lies the cortex (not shown).

When the xylem appears discoloured to macroscopic vision this is due largely to infiltration, though it appears that, in some cases, the middle lamellae of the vessels show gummous degeneration, as likewise those of the wood fibres.

In the bark the tissues die from the periphery inward, the cells being largely occluded by a homogeneous yellowish brown mass, and are cut off by suber or a proliferation of the subjacent cortical cells.

4. CAUSE OF SQUAMOSIS.

Gummosis, as we have seen, may be brought about by various agencies: in the case of squamosis, however, we are still ignorant as to the cause of the malady. It may be an autogenous disease, though it would appear from the manner in which it develops, especially on the trunk and limbs, that it is due rather to bark-binding, the particular symptoms of the malady being induced by the manner in which the pressure on the cambium is released, the release occurring at a time when the affected tree was growing rapidly and the tissues were quite sapid. This explanation may perhaps also apply in the case of the shoots, seeing that the irregularities of growth distinctly shown in the older parts of diseased trees may also be seen in them. Added the death of the bark from fungous or other injury introducing changes of pressure on the cambium and we can conceive how the

pathognomonic structural characters of the xylem arise. Squamosis, however, must be further studied before its cause or causes can be definitely ascertained. We are only certain of one thing: the disease is a form of gummosis, and consequently growth and water are factors in its development.

5. PREVENTIVE AND REMEDIAL MEASURES.

Little is known regarding the remedial and preventive methods for squamosis. The only preventive measure that appears, at present, to offer any guarantee of success is growth regulation. This may be accomplished by cultural methods. As for remedies, there are at present none. Slitting the bark may be considered a palliative, nothing more.

IV. EXANTHEMA.¹

1. HISTORICAL.

Exanthema is at present only known in the United States. In Florida it is found throughout the orange-growing region, and has probably been a malady of the *Citrus* in that State since the introduction of their culture. In California exanthema is only known in the orange groves of Los Angeles, San Bernardino, and San Diego Counties, Southern California, and, as in the case of Florida, the date of the first appearance of the malady does not appear to have been recorded.

2. DESCRIPTION.

General. According to Hume, exanthema affects all varieties of the *Citrus*. Young trees—the malady has even been observed in nursery stock—and old trees are equally susceptible. The malady develops in California either on very permeable granitic soils or in shallow clay soils underlaid by an impermeable subsoil, in other words, in dry soils. In Florida the soils are typically sandy and belong, therefore, to the class of permeable dry soils. In that state the disease is worst when the soils are poorly drained or underlaid by an impermeable ferruginous sandstone, but it is also found in groves growing on the best ‘hammock’ soils.

*Minute.*² Exanthema is characteristically a disease of the small branches and shoots, though the fruit shows symptoms of diagnostic value.

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² Largely after Swingle and Webber, loc. cit., p. 16 et seq.

A luxuriant growth, a deep green colour of the foliage, and thick-skinned fruit, though not in themselves specific, precede the first symptoms of the disease sufficiently frequently to possess the value of an indication. Exanthema, however, cannot be diagnosed until the shoots become more or less stained sub-epidermally by a yellowish brown material and begin to die back; or the fruit becomes similarly stained and the epidermis so indurated that it cracks and splits, due to the pressure of the developing pulp-cells. Either one or the other of these symptoms must be observed before exanthema can be accurately diagnosed.

When conditions are not very favourable for the development of exanthema, an affected tree may live for years without developing any further pathognomonic symptoms; when conditions are favourable for its development, however, further changes take place, especially in the shoots and branches. When young the shoots swell at the nodes (Pl. VIII, Fig. A), infrequently on the internodes—the homologues of these swellings are sometimes observed on the fruit, but as they become more mature linear erumpent pustules break out on the internodes (Pl. VIII, Fig. C); these pustules may even alternate with nodal swellings. On the older branches the nodal swellings are not observed, but the pustules may become exceedingly numerous, and a small amount of gum may be observed in them. Gum may also be observed exuding through the bark in small amount. On shoots and branches that are not covered with swellings and pustules, there frequently occurs a marked proliferation of young buds (Pl. VIII, Fig. D), which may develop into short branches with chlorotic foliage, thus producing a pseudo witch's-broom effect (Pl. VIII, Fig. B).

When diseased shoots are examined in cross-section it will be observed: (1) that the swellings are due to an accumulation of gum and are entirely similar to those sometimes met with in gummosis; (2) that the pustules are produced by a proliferation of the cortical tissues, accompanied by the formation of a sickle of susceptible tissue containing gum pockets. It will also be observed that rings of growth are extremely marked, much more so than in squamosis, and altogether too numerous to be considered variations in growth due to seasonal changes.

3. HISTOLOGY.

A cursory histological study of the swellings and erumpent pustules—in other words, the most striking and typical characters of exanthema—is sufficient to show that this malady is very closely related to gummosis; in fact, were it not for the erumpent pustules, it would be impossible to point out any essential anatomical differences between the two maladies. The swellings are nothing more nor less than well-developed gum pockets in which the gum has accumulated to a considerable extent, it not having been able to break through the epidermis. These gum pockets are not only

histologically identical with those formed in gummosis, but are even found, to my knowledge, on the shoots of young trees affected by this malady. This form of exanthema need not, therefore, be further studied.

In the development of the erumpent pustule we meet with a character not observed in gummosis. Owing to the pressure from beneath, the epidermis ruptures and the cortical cells begin to proliferate, the proliferating cells, at first sub-epidermal, gradually extending deeper into the cortex until they finally make up all the tissue exterior to the last formed row of pericyclic fibre bundles. With the beginning of the proliferation or coincident therewith, a sickle of susceptible tissue is laid down by the cambium and gum pockets are formed, in the manner described in our histological study of gummosis, between the medullary rays, which themselves are rarely involved in gummous degeneration. When large pustules are pro-

TEXT-FIG. 3. Cross-section through sickle of pathognomonic tissue showing development of gum pockets in rows, and separated from one another by more or less perfectly formed ligneous cells.

duced the sickle of susceptible tissue laid down may become relatively thick at its middle point, and contain several rows of gum pockets more or less perfectly separated by a fillet of tissue not affected by gummosis (Pl. IX, Fig. 4; Text-fig. 3). The growth in thickness of the sickle of susceptible tissue is thus clearly due to a period of normal or subnormal growth being rapidly followed by the further laying down of gummo-genetic tissues. Such a peculiar development as this, and one in every respect homologous, may be observed infrequently in gummosis.

When the shoots affected by exanthema show typical gummosis characters (Pl. IX, Fig. 1), normal wood may re-form over the gum pockets, but I have not observed it laid down *de novo* over the pathognomonic sickles. Lignification of the cells of the susceptible tissue (sickle formation) begins with the cessation of the conditions favourable to gummous degeneration, but is less perfect than in squamosis or gummosis.

From the brief sketch of the histology of exanthema that I have just given it appears evident that were it not for the erumpent pustules this malady would be undifferentiable from gummosis. I am inclined to think that the proliferation of the cortex may not be a differentiating character of very great importance. May it not be simply due to the epidermis becoming inelastic and preventing for a time normal cellular division, which is able to proceed with vigour the moment a rupture occurs? At the time the rupture in the epidermis takes place there is every reason to believe that metabolism is unusually active, and the tissues at the same time very sapid—gumous degeneration which only occurs, as we know, when the cambium is active, and when the plant has an abundant supply of water at its command, is an evidence of this—and that owing to this concurrence the cells of the cortex become meristematic the moment pressure is released. With the release of pressure due to the rupture of the epidermis the growth stimulus should be transmitted centripetally and only to a slight extent tangentially if my hypothesis were correct, and we have seen that this is exactly what happens.

4. CAUSE OF EXANTHEMA.

The conditions favourable for the development of exanthema, as well as an histological study of the tissues of diseased trees, indicate that the malady is induced, like gummosis, by the concurrence of active growth and sapid tissues. In the case of the latter malady these conditions prevail for a period of time, but in the case of the former they are extremely transient.

The soils in which exanthema occur are typically dry soils, which, when saturated by irrigation water or rains, promptly become dry once more when the weather clears or irrigation is discontinued. The rings of growth, which, as we have seen, are very marked in diseased shoots and branches of trees affected by exanthema, could not be caused except by a more or less rapid succession of maxima and minima of growth, and such an alteration could only be accounted for by synchronous changes in the amount of available water present in the soil, or a like succession of favourable and unfavourable climatic conditions. Obviously climatic changes cannot be considered as in any way favouring or inhibiting the development of the disease, and we have no alternative but to assign the unusual and marked development of rings of growth to changes in the water relation.

Exanthema has another character in common with gummosis: trees are *ceteris paribus* much more severely affected in rich soils than in poor soils. In Florida it is even considered that heavy fertilization with organic manures, such as cotton-seed meal and dried blood, is alone sufficient to cause the appearance of the disease in healthy trees. This opinion is in a certain sense well founded, for nitrogen is a growth stimulant, and would, therefore, tend to widen the range between the maxima and minima of

growth in the various rings; and it is quite conceivable that the widening of this range could be sufficient to produce exanthema. The inorganic manures are reputed to be less favourable to the development of exanthema than organic manures, and it is altogether probable that this is due to the solubility of the former. Nitrates are soon carried into the drainage waters and lost, whereas the organic nitrogen must first be nitrified, and this only occurs under suitable conditions of soil moisture, conditions which would be realized when the available water in the soil became sufficient to promote vigorous growth in the trees. Hence the bad effects following the use of organic manures.

Swingle and Webber¹ observe that cultivation increases the susceptibility of the *Citrus* to exanthema, and even causes a more virulent outbreak of the disease in affected trees. They explain this effect of cultivation on the ground that the surface roots are destroyed and the trees forced to grow down into an unfavourable subsoil, but this supposition is not well founded: cultivation simply increases nitrification and, when deep enough, prevents excessive evaporation of water from the soil. We must, therefore, consider in humid climates nitrification due to cultivation as a possible source of danger and one not to be neglected if no cultivation is as effective a prophylactic measure as it appears to be.

5. PREVENTIVE AND REMEDIAL MEASURES.

All the preventive measures used in combating exanthema are also remedial; similarly, the remedial measures are all prophylactic. It is unnecessary, therefore, to distinguish between the two.

To successfully combat exanthema one must prevent the irregularity of growth which is such a marked feature of the disease. The methods employed for attaining this end are quite various, and sometimes even apparently conflicting. I shall not attempt to exhaust the list.

When exanthema is traceable to the excessive use of organic manures we know that for these manures to be inductive sufficient available water must be present in the soil, and we have two cases to consider: (1) exanthema does not appear when the groves are not fertilized, and (2) the malady is present in a mild form when manures are not employed. In the first case drainage would be beneficial though not absolutely essential; in the second drainage would be indispensable, and in both the amount of nitrogen furnished the trees should not exceed their immediate requirements. The nitrogen would probably be most advantageously obtained from green manure crops, though the various other sources are not precluded. Heavy fertilization with potassic and phosphatic manures, which salts act somewhat as growth restrainers, is, in many cases, desirable, and has proved effective.

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DESCRIPTION OF PLATES VII-X.

Illustrating Dr. O. Butler's paper on *Gummosis*.

PLATE VII.

Upper figure. *Citrus Limonum*. Cultivated variety affected by gummosis, environs of Whittier, Los Angeles County, California.

Lower figure. Same tree as in upper right-hand figure, Plate VIII, showing marked loss in vigour and development of chlorosis due to squamosis on limbs.

PLATE VIII.

Upper left-hand figure. *Prunus avium*. Cultivated variety. Limb on right washed with sulphuric acid diluted one-half with water over a small oblong area near origin, and showing gummous exudation twenty-two days after treatment.

Upper right-hand figure. *Citrus Aurantium*. Cultivated variety affected by squamosis, environs of Whittier, Los Angeles County, California.

Lower figures. *Citrus Aurantium*. Cultivated variety. Shoots from tree affected by exanthema. A, nodal gum postules; B, pseudo witch's-broom growth following bud proliferation; C, erumpent pustules; D, bud proliferation (enlarged). Fig. B from California Agr. Expt. Sta., Bull. cc.

PLATE IX.

Fig. 1. *Citrus Aurantium*. Cultivated variety. Cross-section of shoot affected by exanthema and showing development of gum pockets as in gummosis.

Fig. 2. *Prunus avium*. Cultivated variety. Cross-section of a shoot showing various stages in the development of gum pockets. Gummosis was induced by *Plowrightia morbosa*.

Fig. 3. *Prunus* sp. Cross-section of shoot of plum showing first stage of disease (fusoid development of pathognomonic tissues). Shaded area shows part of bark killed by burning with sulphuric acid diluted one-third with water.

Fig. 4. *Citrus Aurantium*. Cultivated variety. Cross-section of shoot affected by exanthema showing typical fusoid development of gumogenetic tissues and cortical proliferation.

Fig. 5. *Prunus avium*. Cultivated variety. Cross-section through a shoot burned with a drop of sulphuric acid diluted one-half with water, and showing development of gum pockets and re-formation (in progress) of normal xylem after the lapse of fourteen days. The shoot was sectioned somewhat above the spot burned.

B, bark; C, cambium; E, epidermis; G, gum pockets; M, medulla; P, pericycle bundles; S, suber; X, xylem; X', imperfect xylem.

PLATE X.

Fig. 1. *Prunus avium*. Cultivated variety. Cross-section through part of a gum pocket in branch affected by *Plowrightia morbosa* some little distance above the 'black knot'. Normal xylem is being laid down over the gum pocket.

Fig. 2. *Citrus Aurantium* var. Valencia. Cross-section through gum pocket showing disappearance of cells upon the addition of water. Compare with Fig. 6, which represents the same section in alcohol before the addition of water.

Fig. 3. *Prunus domestica* var. Robe de sargent. Cross-section through gum pocket showing pseudo-cellular structure of gum.

Fig. 4. *Prunus domestica* var. Robe de sargent. Cross-section through young gum pocket.

Fig. 5. *Citrus Limonum*. Cultivated variety. Cross-section through portion of a fibro-vascular bundle in rind of fruit showing in the dense granular protoplasmic area the first stage in gummosis, degeneration of cell-walls. Change in cell-walls only becomes manifest when sections are placed in water.

Fig. 6. *Citrus Aurantium* var. Valencia. Cross-section of a gum pocket. Section in alcohol to compare with same section (Fig. 2) in water. Figs. 2 and 6 illustrate the disappearance of cells upon the addition of water.

Fig. 7. *Citrus Limonum*. Cultivated variety. Cross-section through young gum pockets in various stages of development.

Fig. 8. *Citrus Aurantium*. Cultivated variety. Cross-section through a gum pocket showing a number of cells floating free in the gum that do not vanish when alcohol is replaced by water.

BUTLER — GUMMOSIS.

Huth London.

A.

B.

D

C. BUTLER—GUMMOSIS.