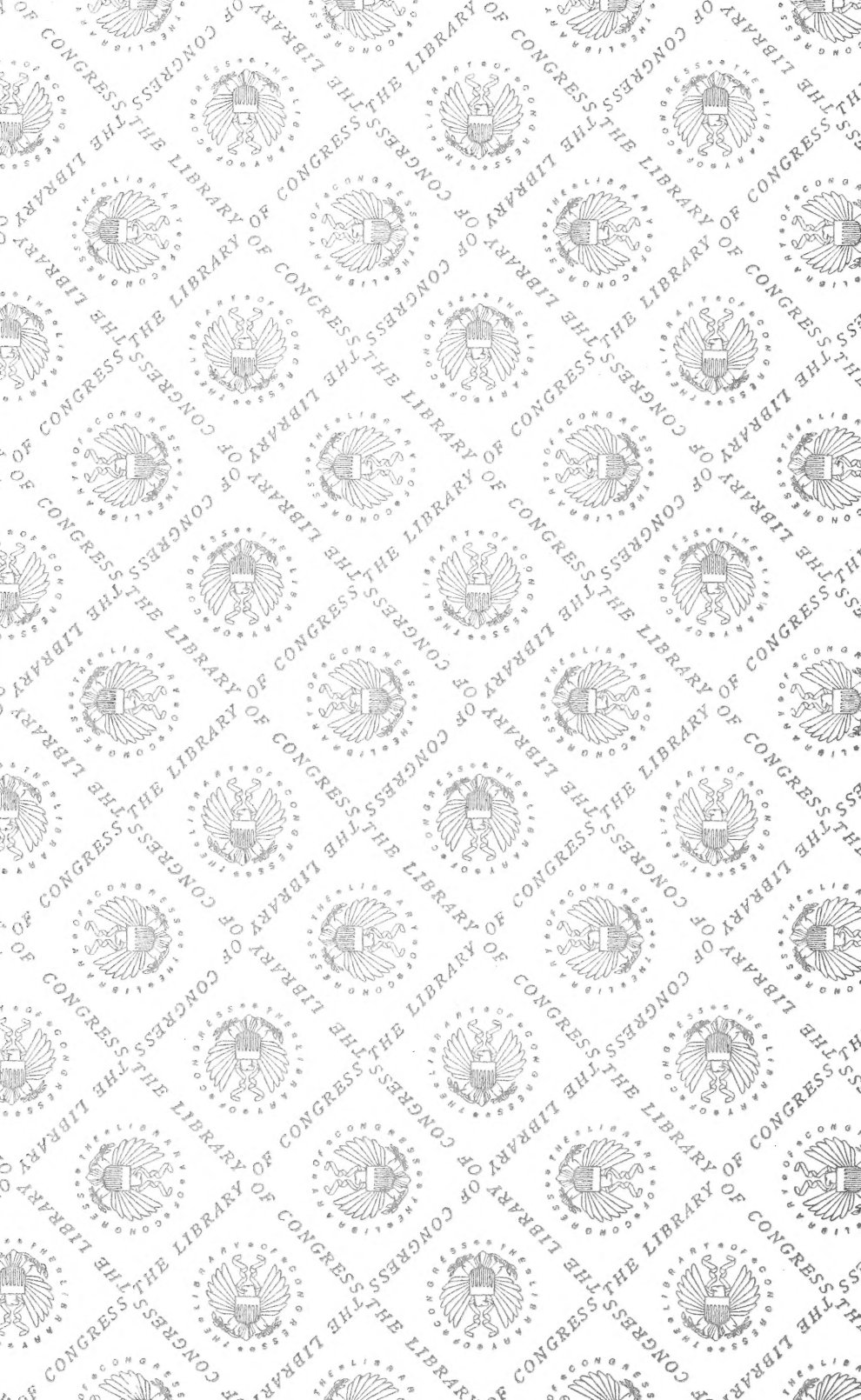
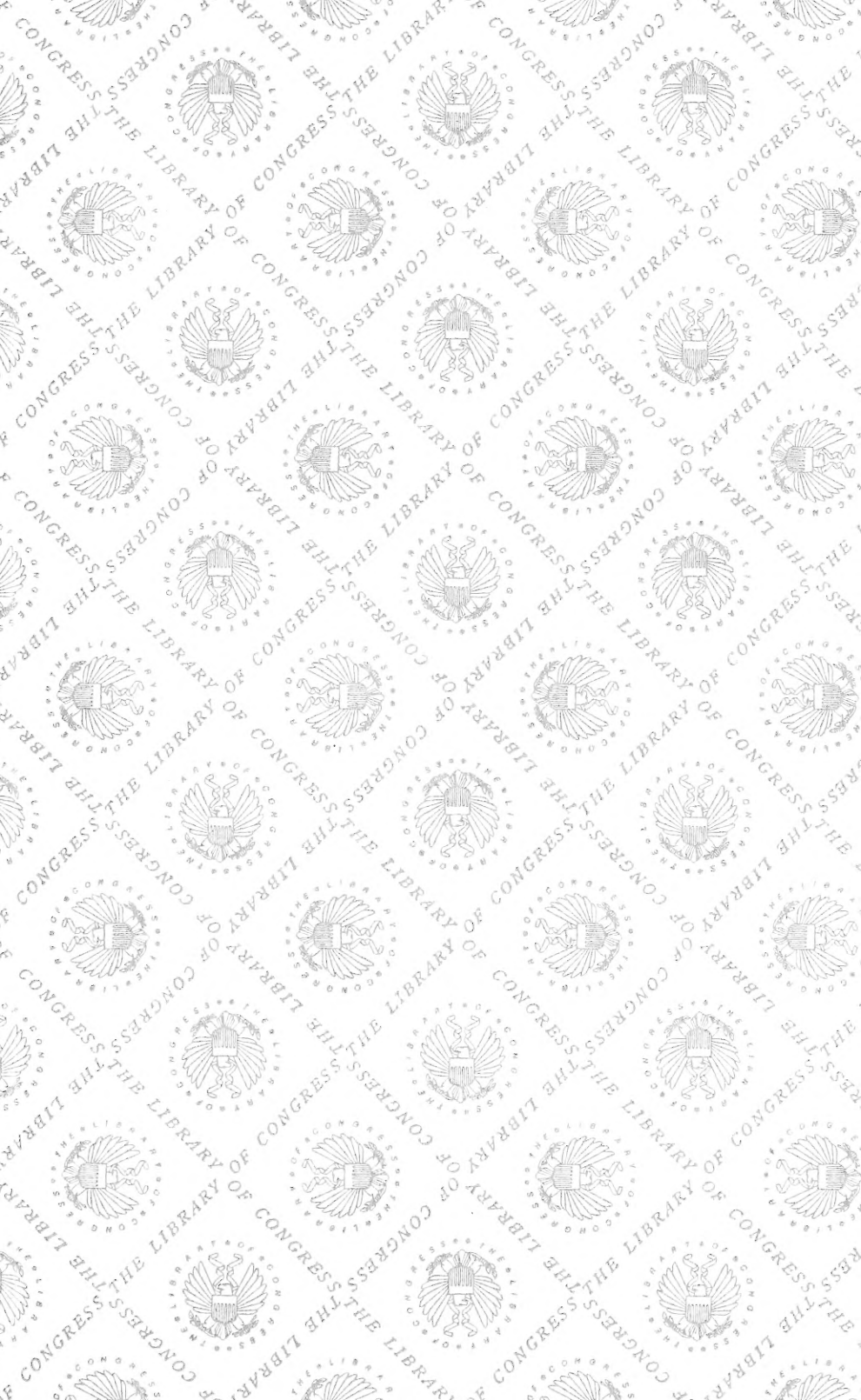


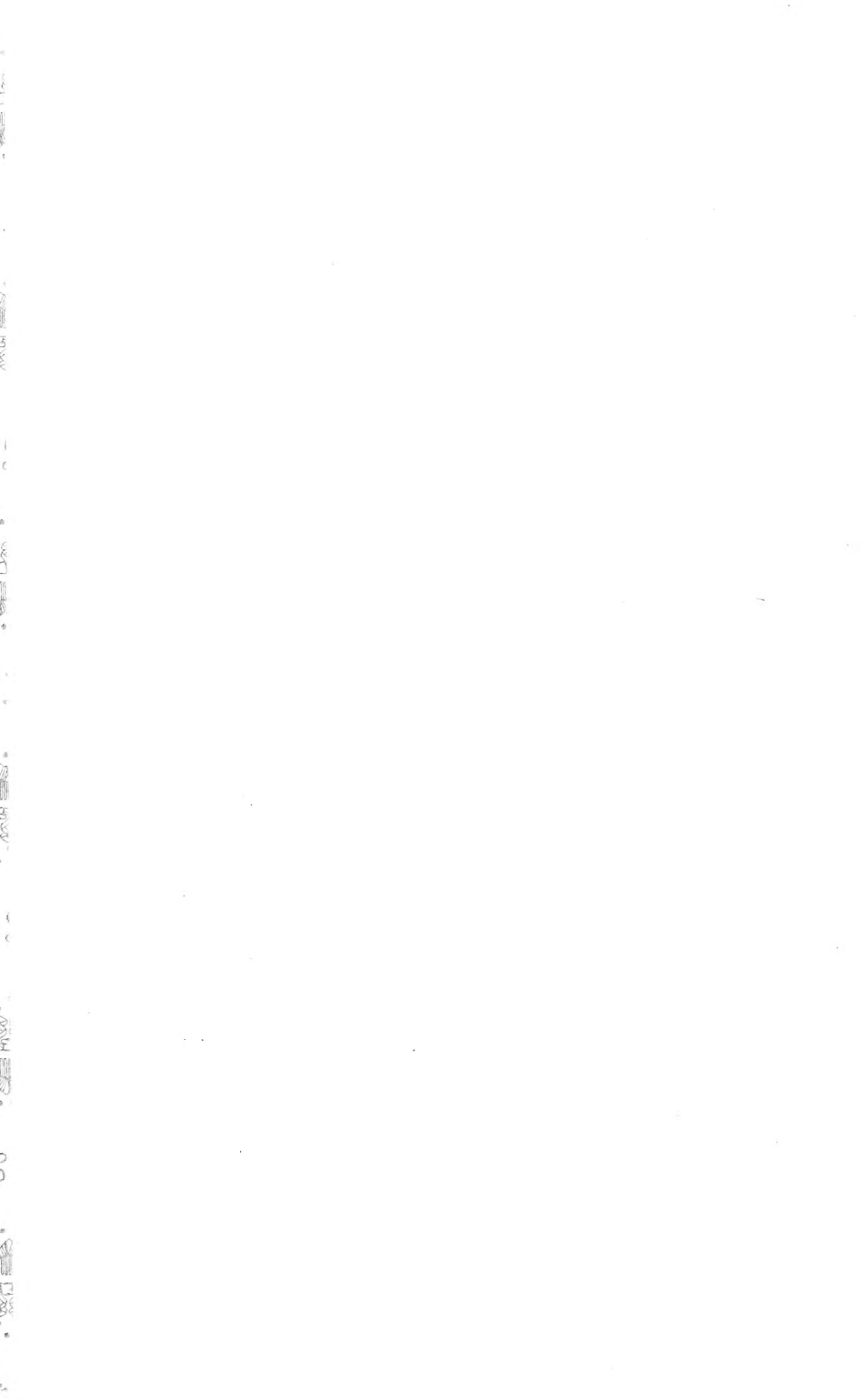
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DEPARTMENT OF FORESTRY

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The *Strumella* Disease of Oak  
and Chestnut Trees

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# THE STRUMELLA DISEASE OF OAK AND CHESTNUT TREES.<sup>1</sup>

BY

F. D. HEALD and R. A. STUDHALTER, *Agents*,

Investigations in Forest Pathology, Bureau of Plant Industry.

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## INTRODUCTION.

The study of chestnut tree blight in Pennsylvania has brought to light a serious disease of the chestnut due to an entirely different organism. The trouble is not confined to the chestnut but attacks various species of oaks. The disease is obscure in its early stages and in some of its more advanced phases, which probably accounts for the fact that it has not been generally observed by foresters. Our recent studies reported by the senior author<sup>2</sup> lead to the belief that it is a rather widespread disease and responsible for the death of many trees, both young and old.

What was probably the same trouble was briefly described in 1899 by Dr. W. A. Buckhout<sup>3</sup>, as causing the death of various species of oak trees, but no mention was made of the occurrence of the disease on chestnut trees. While he regarded the disease as of fungous origin, no definite determination of the causal organism was made. The correctness of our inference that he was dealing with the same trouble is based largely on the similar symptomology as may be noted by comparison of our description with the following quotation: "The black oak is particularly subject to a disease apparently of the same nature if not identical with *Nectria ditissima*, the canker of the apple tree. The early stages of the disease are obscure and inconspicuous, and one's attention is attracted only after serious injury has been done. Apparently the starting point is at the base of a small branch, causing the death and slow decay of surrounding bark

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1. Published by permission of the Secretary of Agriculture.

2. Heald, F. D. A little-known disease of chestnut and oak trees. *Phytopathology* 4: 49. 1914.

3. Buckhout, W. A. The undesirability of red and black oak because of fungus disease. Report Penna. Station, 1899: 250-252.

and wood. Both continuation of decay and the effort of the trunk to repair the injury go on together, but it is generally a losing battle. The dead region spreads in spite of the efforts made to produce a callus, covering an enlarging elliptical area on which the little branch shoot appears in the middle as a sort of centre about which circle the lines representing the different stages in the process. As time proceeds, the rest of the trunk receives the normal additions of wood and bark, while the dead parts become more and more an element of weakness."

Unfortunately no specimens of oak showing this disease are preserved in the State College collections, but the occurrence of the *Strumella* disease in the vicinity of State College at the present time lends added support to the descriptive evidence that Buckhout was certainly dealing with the trouble described in the following pages. It seems strange however, that a disease recognized fifteen years ago should continue its ravages and escape further study until the present time.

Although our knowledge concerning this disease is still very imperfect, sufficient data have been accumulated to justify us in presenting a short account. The main object at the present time is to call attention to the importance of the disease and describe its characters in sufficient detail to enable foresters and pathologists to recognize it.

### SYMPTOMS AND EFFECTS.

There is a rather remarkable parallelism between this new disease and the well-known chestnut tree blight as far as general symptoms and effects are concerned.

Young infections of the *Strumella* disease on smooth-barked trunks can be easily recognized by the presence of yellowish or yellowish-brown patches, slightly raised, and standing out in marked contrast to the normal bark which is darker in color. The area invaded by the fungus may be fairly regular in outline, but it is very frequently irregular in outline, the latter showing what may be designated as the amoeboid type of infection (fig. 1). The most noticeable external difference between young lesions of the chestnut blight (*Endothia*), and those of



the Strumella disease, is the presence in the latter of small black nodules, scattered over the surface of the yellowish area which marks the extent of the lesion. In young infections of the type described the dark nodules are either aborted or rudimentary fruiting pustules. No fertile pustules have ever been found in these young infections, but spore production occurs in the later stages of the disease. The yellowish-brown color is caused by the whitish, or very pale buff-colored vegetative body, or mycelium, of the causal fungus, which is covered only by the thin external layer of cork cells. If some of this cork layer is removed from the advancing edge of the lesion, the mycelium exposed will show its pure white color, but in more central and older parts the mycelium has changed to a pale buff color. In general it is lighter in color than the mycelium of the chestnut blight fungus, and definite "fans" characteristic of the latter are not noticeable. Lesions of this type have been found on shoots up to 4 or 5 inches in diameter, and varying from minute incipient infections to those completely encircling the trunk. The young infections are much more obscure on older or rough-barked trunks, but there is generally sufficient difference in color to indicate the extent of infection.

Two forms of the disease may be recognized with further development of the trouble: first, the *canker type*, in which the progress of the fungus is slow with a more or less pronounced formation of callus at the advancing edge of the lesion; second, the *diffuse type*, in which the fungus spreads more rapidly, killing the tissues so quickly that the formation of callus is not possible. Apparently it was only the conspicuous *canker type* that was observed and described by Buckhout.

In the well-defined canker there may be more or less zonal growth (fig. 7), as the result of the repeated formation of callous tissue at the advancing edge, but it is the exception for the callus to check the advance of the fungus. The lesion is generally extended more rapidly lengthwise of the trunk than transversely, thus giving rise to cankers of somewhat elliptical outline (figs. 2 and 3). Cankers five feet or more in length have been observed on trunks of chestnut trees 6-8 inches in diameter

that had not yet been girdled. In old cankers there is likely to be more or less destruction of tissue at the center, so as to leave a somewhat open wound exposing the decaying wood (figs. 3 and 5). Old cankers are likely also to show the work of woodpeckers and insect larvæ.

When a well-defined canker is not produced the extent of the lesion may be indicated by an elliptical discolored area which is generally somewhat depressed. Even when the bark of this area is not destroyed it is rougher than surrounding portions due to more or less shrinking and cracking. In all cankers the characteristic black nodules may be noted on those portions on which the bark has remained intact.

In some infections there is a pronounced enlargement or hypertrophy of the entire invaded area (fig. 6), instead of atrophy or lessening of growth. In the open cankers there may be more or less enlargement or distortion of the trunk (figs. 3, 5 and 7), due to the stimulating effect of the parasite. Such effects appear to be more frequent on the oaks than on the chestnut.

The disease is least evident in its *diffuse form*. In this type there is an absence of well-defined cankers and the fungus appears to spread so rapidly that little or no callus is developed. In such cases the young tree is killed before time has been given for the development of a canker. This points to the fact that the larger trees showing well-defined cankers represent the few individuals that have offered the most resistance to the attacks of the parasite. The diffuse type of the disease has not yet been found on anything but young trees up to 3-4 inches in diameter. It should not be understood that there are two entirely distinct types of the disease, for there are all gradations between the pronounced canker types and the diffuse types; in the intermediate types the lesion may be marked by a more or less concentric zonation (figs. 2, 4 and 7), while in the extreme diffuse type no zonation is evident (fig. 8).

The disease appears to start in a branch axil. In all young infections or old cankers this relation is an invariable rule, and in lesions of any age the dead stub of a branch marks the center

of the infection (fig. 7). After the branch has been killed the fungus grows more rapidly through its tissues, and soon produces the characteristic fruiting pustules over its surface in the form of dark brown, powdery, and erumpent nodules 1-3 mm. ( $\frac{1}{25}$ - $\frac{1}{8}$  inch) in diameter (fig. 8).

The production of sprouts or "suckers" just below a lesion is one of the marked characteristics in either the canker or semi-diffuse forms of the disease (figs. 11 and 12). This production of vigorous sprouts occurs as soon as the trunk has been girdled, and they may be few in number or sufficiently numerous to make a conspicuous clump.

The final result of the disease is the death of the tree, but in the extreme canker type there is a rather prolonged struggle. No cases have been observed where the advance of the parasite has been permanently checked. It is apparently the diffuse type of the disease that is exacting the larger toll, if we may judge from observations in certain localities where the disease is especially prevalent. The disease is especially serious since there is a marked tendency to the production of body or trunk cankers only. They may occur at any point from the ground up but they appear to be more common in the lower portions of the trunk. As soon as girdling has been completed the parts beyond the canker must succumb, but even before such a fatal termination, the trunk may be so weakened by the inroads of the fungus as to be unable to withstand the force of strong winds. Many fallen oaks which have been broken at a lesion are silent witnesses of the destructiveness of this trouble (fig. 12). Unlike the chestnut bark fungus, the organism in question grows equally well in both bark and wood. It is this disintegration of the wood by this parasite and not by secondary decays that causes the pronounced weakening of the trunk in the region of a canker. The extent to which the fungus invades the wood even in the canker type is shown in Fig. 14, the light area being the portion infected. In Fig. 13 the only normal wood remaining shows a light color also.

### THE CAUSAL ORGANISM.

A single species of fungus is invariably associated with this disease. It has repeatedly been isolated from all of the different types of the disease on a variety of hosts.<sup>1</sup> The fungus grows well in ordinary culture media, but 3% dextrose agar has been used in making most of the isolations. Tissue transfers made by planting small fragments of diseased bark or affected wood in agar plates give pure cultures in the majority of cases. Typical isolation plates are shown in Figs. 15 and 16. The fungus produces a cottony aerial mycelium and this turns to a dirty gray after one to three weeks. The under side of the colony shows a dark brown or black coloration which first becomes noticeable at the center and gradually spreads towards the periphery of the colony. Different strains show great variation in the amount and intensity of darkening (fig. 17). No spore formation has been observed in any cultures.

The fungus in question, *Strumella coryneoidea* Sacc. & Wint., is not a new one, but is an old species which has not previously been accused of being a parasite. It was first collected at Perryville, Mo., in 1883, by C. H. Demetrio and described by Saccardo and Winter.<sup>2</sup> The fungus has also been collected at London, Ontario, on oak bark by J. Dearness (No. 94, June, 1889). The two following collections referred to this species were apparently incorrectly determined:

- 1.—Flora of Washington. No. 307. Collected Jan. 28, 1894, on dead branches of *Rhus diversiloba* J. and G., by W. N. Suksdorf. Columbia River, W. Klickitat Co.
- 2.—Flora Ludoviciana. No. 1799. Collected Mar. 30, 1889, on young dead twigs of *Carya olivaeformis* by A. B. Langlois. St. Martinsville P. O., La.

A careful comparison of our specimens with those of Demetrio (Ellis and Everhart. North American Fungi, No. 1653), and Dearness (No. 94), has failed to reveal any morphological

1. Many of the isolations were made by Mr. R. C. Walton, who was associated with the writers during the earlier work on this disease.

2. Saccardo and Winter. Rabenherstil Fungi europael extraeuropael. No. 2984. Hedwigia, 1883: 175-176. Ellis and Everhart. North Am. Fungi, No. 1653, June 1885.

differences, so we feel justified in referring the parasite to the species originally described by Saccardo and Winter. The causal relation is based not only on the constant association of the fungus with the disease but also upon successful inoculations in the field. That *Strumella coryneoidea* was not connected with the disease by Dr. Buckhout<sup>3</sup> is evident from his statement that the disease is "apparently of the same nature if not identical with *Nectria ditissima*, the canker of the apple tree."

*The fruiting stage.*—During the time previous to the girdling and consequent death of a branch or trunk, the fungus produces only mycelium and aborted or rudimentary fruiting pustules. As soon as the girdling has been completed, however, an abundance of fertile pustules appear in the form of brownish erumpent powdery cushions, the *sporodochia*, scattered either sparsely or abundantly over the surface of the invaded portions and also beyond the extent of the original lesion (figs. 8 and 18).

The sporodochia vary in size from 1-3 mm. in diameter (fig. 19), and each consists of a dense aggregate of fungous tissue which bursts through the external cork layer in the form of a rounded nodule which produces over its entire free surface a multitude of erect, branched, spore-bearing hyphæ or conidiophores. Spores are developed in profusion from the terminal portions of the brownish conidiophores (fig. 20), and either loose or attached, give the granular or powdery appearance to the fruiting pustules (figs. 8 and 18). The spores (fig. 21) are light or very dark brown, single-celled, vary from nearly globular to pear-shaped or irregular in form, and vary in size from 5.1—13x4—7.3 mikrons. No ascus stage has been found in the life cycle of this fungus, but the profusion of conidiospores would appear to offer an effective means of dissemination. As the accumulated spore mass is powdery when dry it seems probable that wind dissemination is one of the important means of spread of the fungus. The loose spores are undoubtedly washed down by rains. The latter statements are based however on observational evidence rather than on direct experimental evidence.

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3. Loc. cit. p. 250.

### HOST RELATIONS.

This disease must be viewed with some additional concern since it is not confining its attacks to a single species. As a disease of the chestnut it is much less virulent than the *Endothia* blight but it occurs on the chestnut beyond the present range of the chestnut bark disease. Various species of oaks are affected and in some localities the disease has made such headway as to cause some alarm. In the short experience with this disease up to date it has been found attacking the following species:

American chestnut, . . . . .	<i>Castanea dentata</i> (Marsh.) Borkh.
Chestnut oak, . . . . .	<i>Quercus Prinus</i> L.
Black oak, . . . . .	“ <i>velutina</i> Lam.
Red oak, . . . . .	“ <i>ruba</i> L.
Scarlet oak, . . . . .	“ <i>coccinea</i> Muench.
White oak, . . . . .	“ <i>alba</i> L.

The disease appears to be more severe on the red and black oaks, and also produces more pronounced cankers on these species. The most typical development of the disease upon oaks has been observed in fairly dense stands in which a single species predominates. The disease is more obscure on the white oak than on any other species of oaks. This is due to the very rough character of the normal bark, which may render inconspicuous lesions of some extent. The *Strumella* cankers on the chestnut (fig. 9) are rarely as conspicuous as those caused by *Endothia*. In some *Strumella* infections in this species the lesion may reach a considerable size without showing much discoloration or depression of the bark. There is but rarely any indication of concentric zonation in lesions on this species, while it may be recalled that this character is especially marked in the oak species. In old cankers in all species there is generally more or less corrosion at the center, so as to leave a rough, open wound. It seems probable that all species of oaks will be found susceptible to this disease.

## GEOGRAPHICAL DISTRIBUTION.

The *Strumella* disease has been studied from specimens collected at widely separated points in Pennsylvania. The first specimens studied were collected in Somerset county in the southwestern part of the State. In this region the disease was prevalent on both chestnut and chestnut oak. A little later the trouble was found on chestnut oak in Mifflin county in the central part of the State. It is common on the red and black oaks, and occasional on the white oaks in the State forest in Huntingdon county. It has been observed in Elk county in the northwestern part of the State, where it is especially severe on the chestnut in a number of localities. Studies made in Carbon county in the eastern part of the State show that the trouble is fairly common. It has also been reported from Pike county. The known occurrence of the disease at localities in the southern, middle, northern and eastern portions of Pennsylvania suggest that a more detailed scouting of our forests will show a rather general distribution throughout the State.

The causal fungus, *Strumella coryneoidea*, has been collected in the fruiting stage on both chestnut and oak species in several of the localities studied. The specimens received from some of the localities represent only the canker type, probably due to the fact that only conspicuous examples of the trouble were observed by the collectors. In the localities worked in detail by the writers, the fruiting stage has always been found. In this connection reference may be made to the first collections of the fungus in Missouri, and its later collection from Ontario, Canada, as indicating a wide range. All of the evidence at present points to the parasite as a native of the northeastern portion of North America. If this supposition is correct, the disease is less likely to prove as virulent as though it were introduced from some foreign country. The behavior of the chestnut bark disease is prophetic of what might happen if the *Strumella* disease should be introduced into new localities.

The supposition concerning the wide distribution of the disease has been further substantiated by the collection of authentic

specimens on both the black and white oak in Massachusetts by Mr. R. G. Pierce. It is hoped that this brief description will aid and stimulate further observations and study on this interesting tree disease, so that we may have more complete information, as to its range and prevalence.

### EXPLANATION OF PLATES.

All of the photographs used for illustrating this bulletin were made under the writers' supervision from specimens collected in Pennsylvania.

#### PLATE I.

Fig. 1.—A young *Strumella* infection of the "amoeboid" type on chestnut oak 3 inches in diameter. The minute black nodules scattered over the surface distinguish this superficially from a young chestnut blight lesion.

Fig. 2.—A semi-diffuse type of infection showing three distinct zones of growth, and the characteristic dark nodules scattered over the invaded area. The cut stub of a branch shows at the center, and its lighter peripheral portion indicates the depth to which the fungus had penetrated the wood.

#### PLATE II.

Fig. 3.—A well-defined canker on red oak 2.5 inches in diameter. The advance of the fungus was checked on the right by a marked formation of callus. In the central part the bark was completely destroyed so as to leave an open wound exposing the wood. (See also Fig. 13 for transverse section through this canker).

Fig. 4.—The same specimen shown in Fig. 3 viewed from the opposite surface. The zonal advance of the fungus may be noted. Only a narrow bridge of uninfected tissue remains. (See also Fig. 13).



**PLATE III.**

Fig. 5.—Canker on a black oak 3 inches in diameter showing considerable hypertrophy and also the destruction of tissue to form an open wound.

Fig. 6.—A young lesion on chestnut oak 1 inch in diameter showing hypertrophy, with characteristic longitudinal cracking of the bark. The fungus has completely encircled the trunk.

**PLATE IV.**

Fig. 7.—A canker on a scarlet oak 2.5 inches in diameter showing pronounced zonal growth and the stub of a branch at the center. (See Fig. 14 for transverse section through this canker).

Fig. 8.—Portion of a small black oak tree 1.5 inches in diameter killed by the diffuse type of the disease. The characteristic fruiting pustules, or sporodochia, are very numerous over the entire surface.

**PLATE V.**

Fig. 9.—Typical *Strumella* canker on a chestnut trunk 6 inches in diameter. Note that on this species the extent of the lesion is indistinct.

Fig. 10.—A body canker from a young white oak tree. The elliptical outline of the canker was especially noticeable in its earlier stages, and callus-zones mark its progress.

**PLATE VI.**

Fig. 11.—Portion of a small black oak tree killed by the *Strumella* disease. Vigorous sprouts, or "suckers" were produced below the lesion which was of the semi-diffuse type. The center of the infection is marked by a short stub of a branch.

## PLATE VII.

Fig. 12.—A black oak which has been broken by the wind at the lesion. The copious sprouting below the lesion is evident and the canker also shows the work of woodpeckers.

## PLATE VIII.

Fig. 13.—Transverse section through the middle of the canker shown in Fig. 3. The light area on the left is the only normal tissue that remains.

Fig. 14.—Transverse section through the middle of the canker shown in Fig. 7. The lighter area above the organic center has been invaded by the fungus and the wood disintegrated. This affected wood is soft and punky.

## PLATE IX.

Fig. 15.—An isolation culture of *Strumella coryneoidea* on 3% dextrose agar, made by tissue transfers. The small fragment of diseased tissue is still noticeable at the center of each colony.

Fig. 16.—An isolation culture of *Strumella coryneoidea* on 3% dextrose agar made by transferring marked colonies from poured plates.

## PLATE X.

Fig. 17.—A series of cultures of *Strumella* on dextrose agar taken from the under surface to show the variation in the development of the black coloration characteristic of the species. Each culture represents a different strain.

## PLATE XI.

Fig. 18.—A piece of bark slightly magnified to show the characteristic fruiting pustules, or sporodochia, in the form of dark brown, powdery, erumpent nodules.

Fig. 19.—Vertical section through a typical sporodochium. The dense aggregate of fungous tissue has broken through the periderm, and produced a superficial zone of branched conidiophores, in the terminal parts of which spores have been formed. Semi-diagramatic from a camera drawing. X-75.

#### PLATE XII.

Fig. 20.—A single branched conidiophore showing characteristic form and septation. The spores are loosely attached and easily separate from the conidiophores. X-800.

Fig. 21.—Terminal portion of a conidiophore showing the origin of spores. X-800.

Fig. 22.—Camera drawing of spores showing the variation in form. X-800.



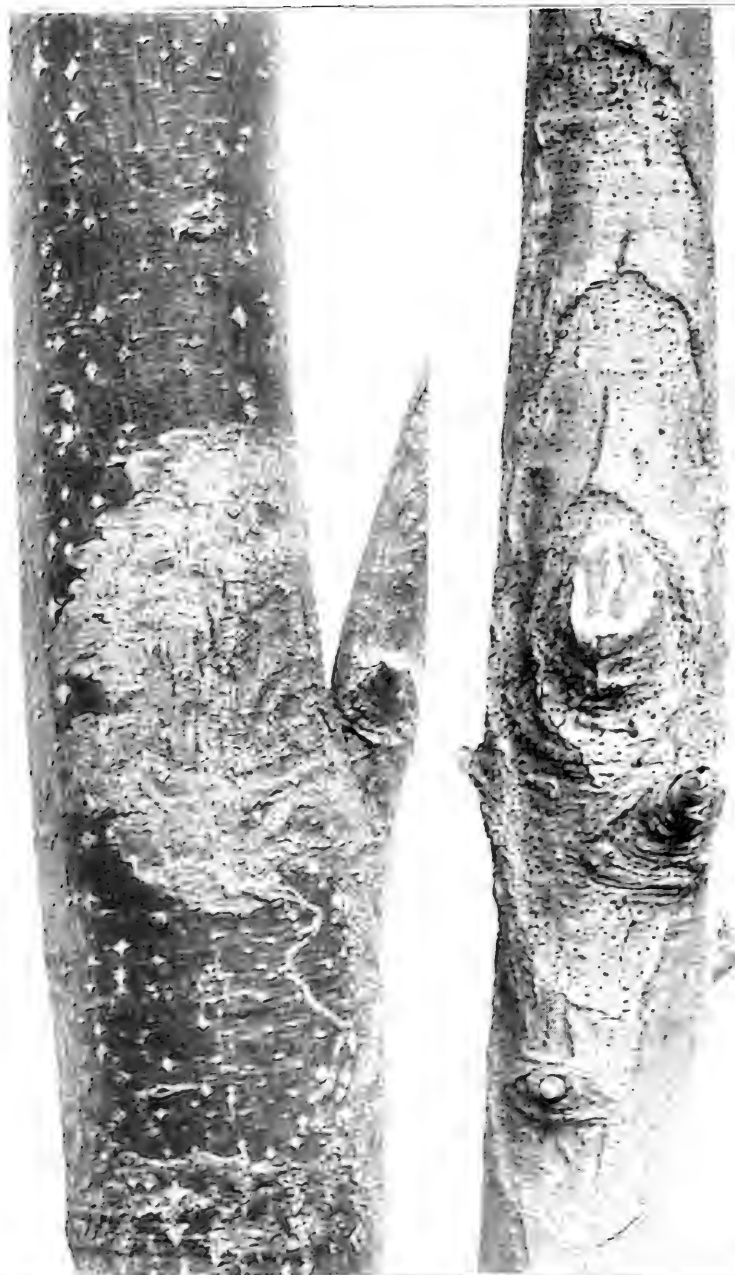


FIG. 1

FIG. 2

PLATE II.



Fig. 3.

Fig. 4.

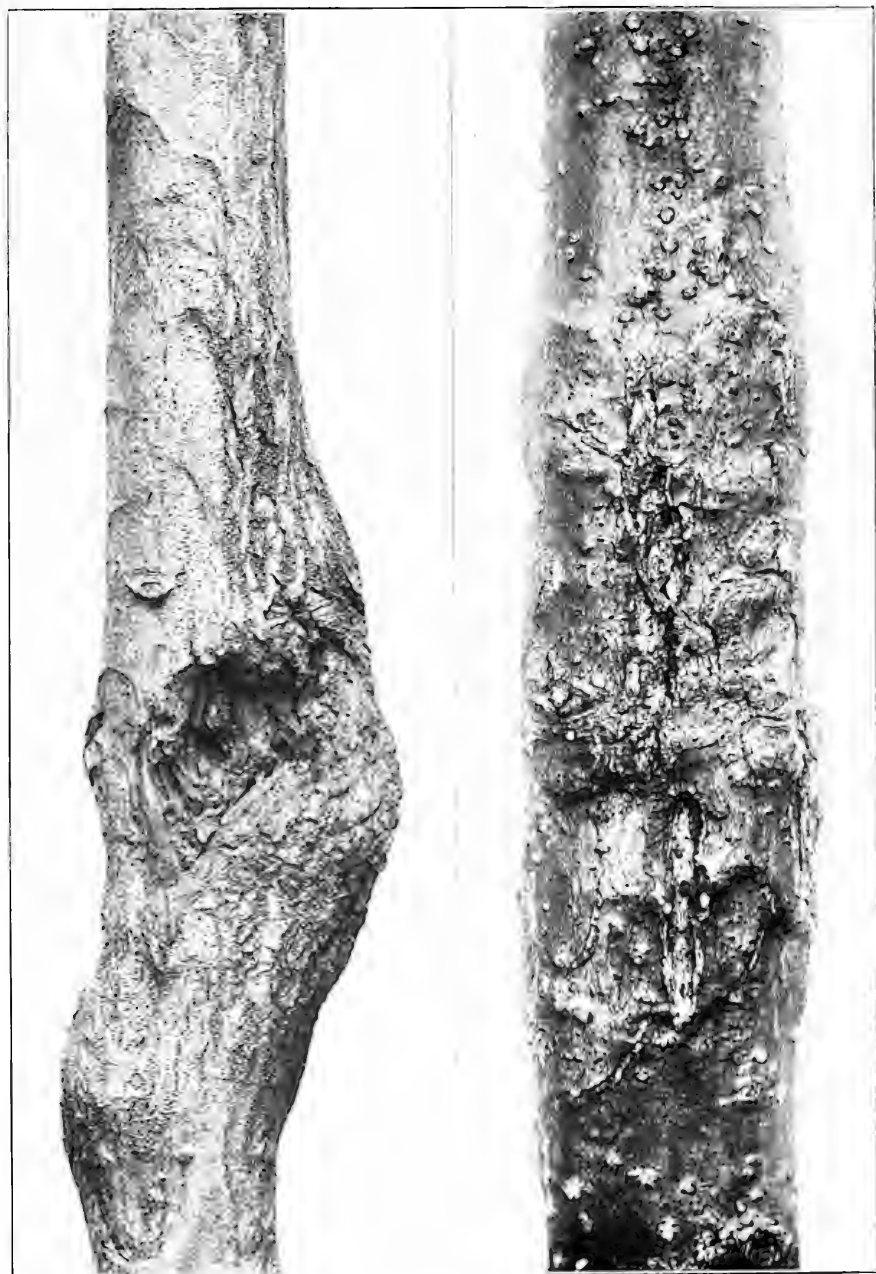


Fig. 5.

Fig. 6.

PLATE IV.

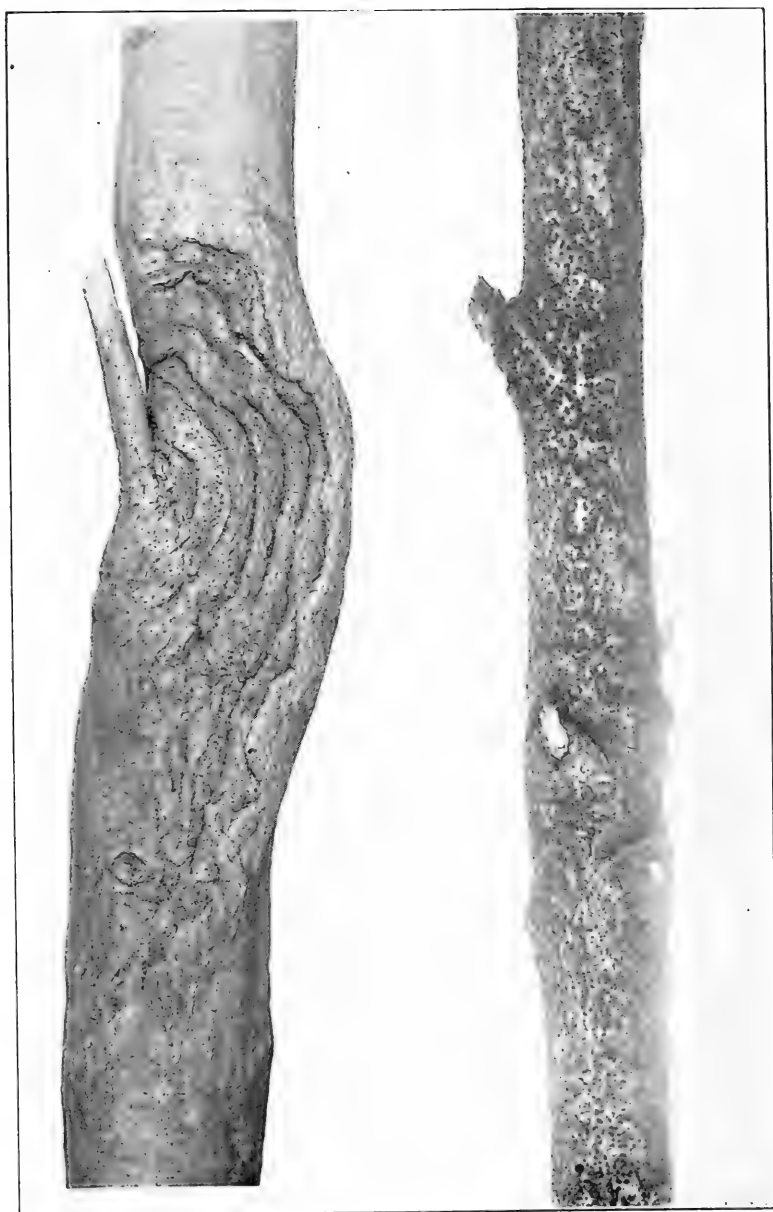


Fig. 7.

Fig. 8.



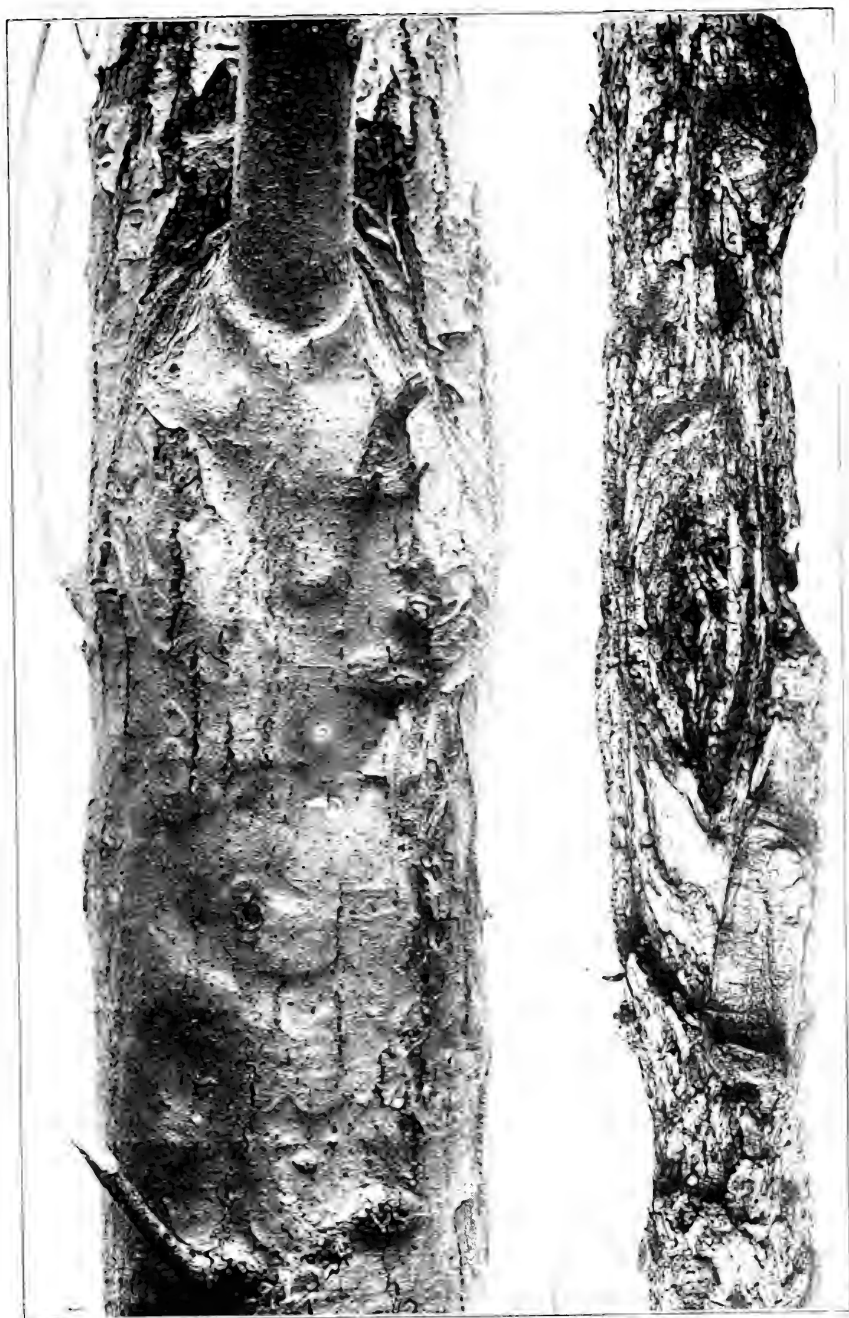


Fig. 9.

Fig. 10.

PLATE VI.



Fig. 11.

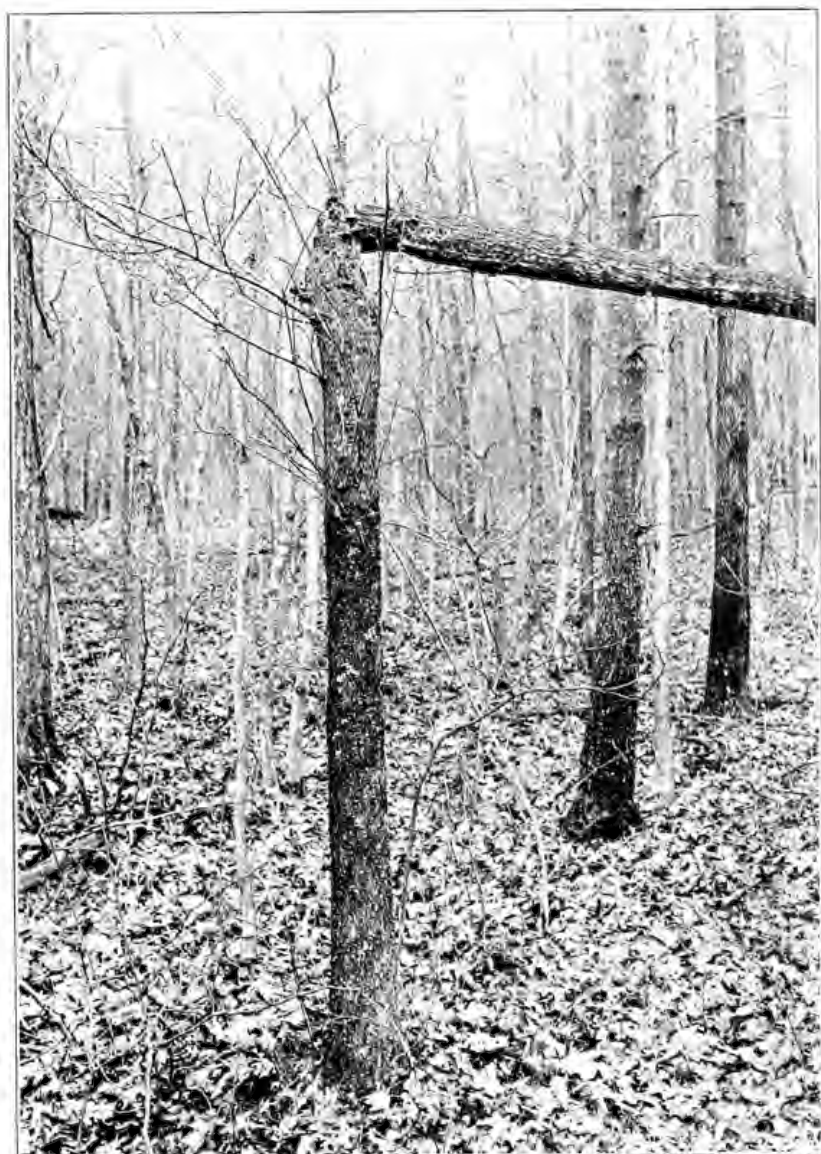


Fig. 12.

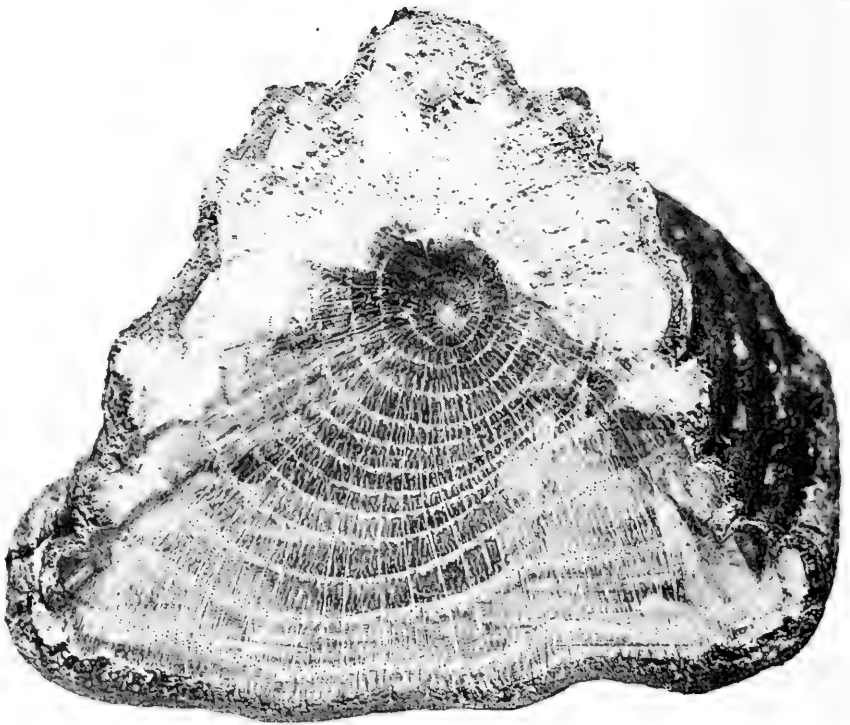
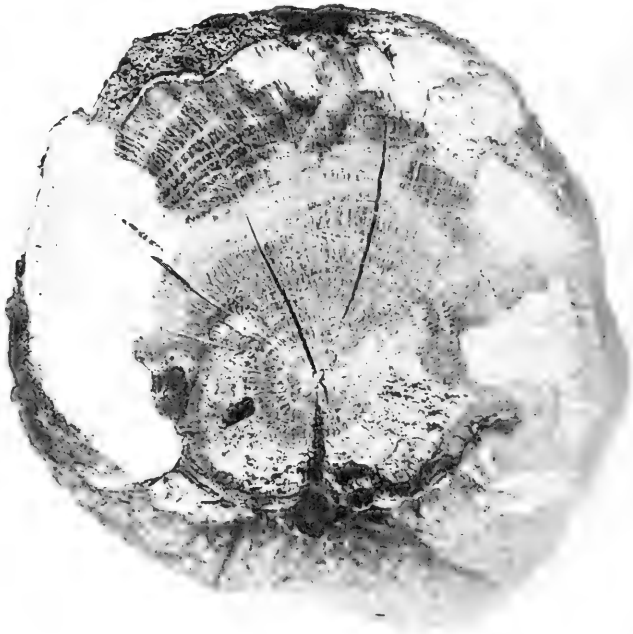


Fig. 13.

Fig. 14.

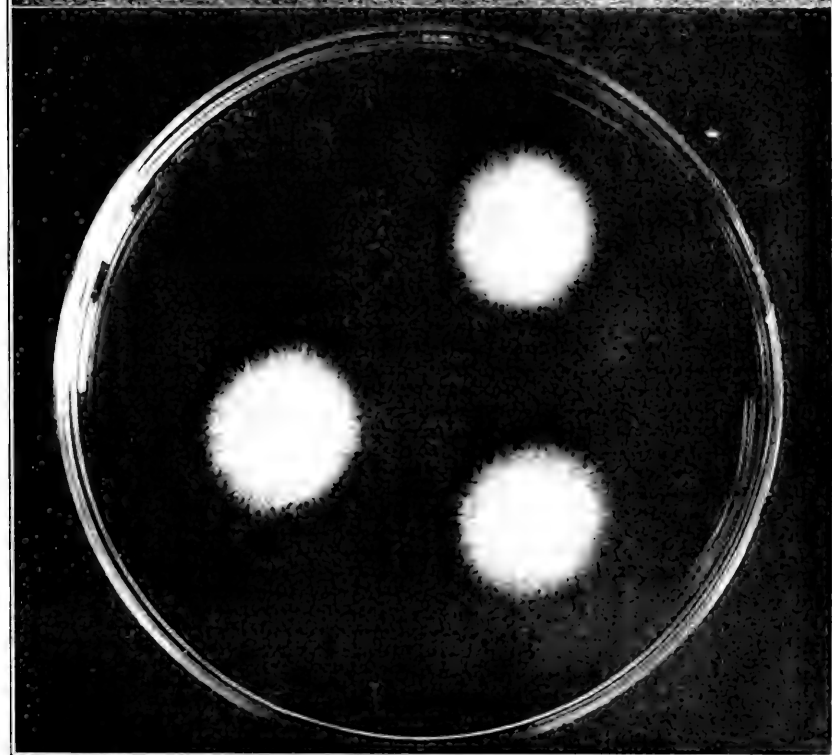
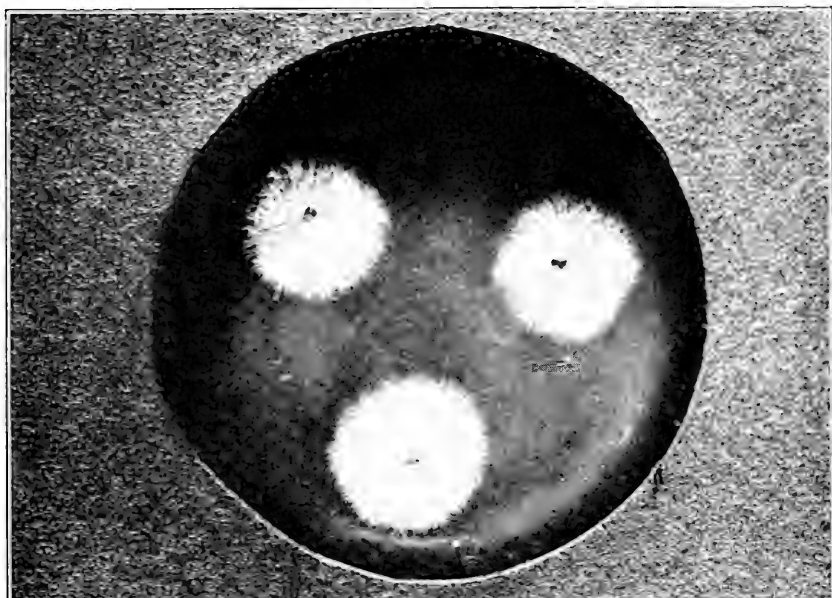


Fig. 15.  
Fig. 16.

PLATE X.

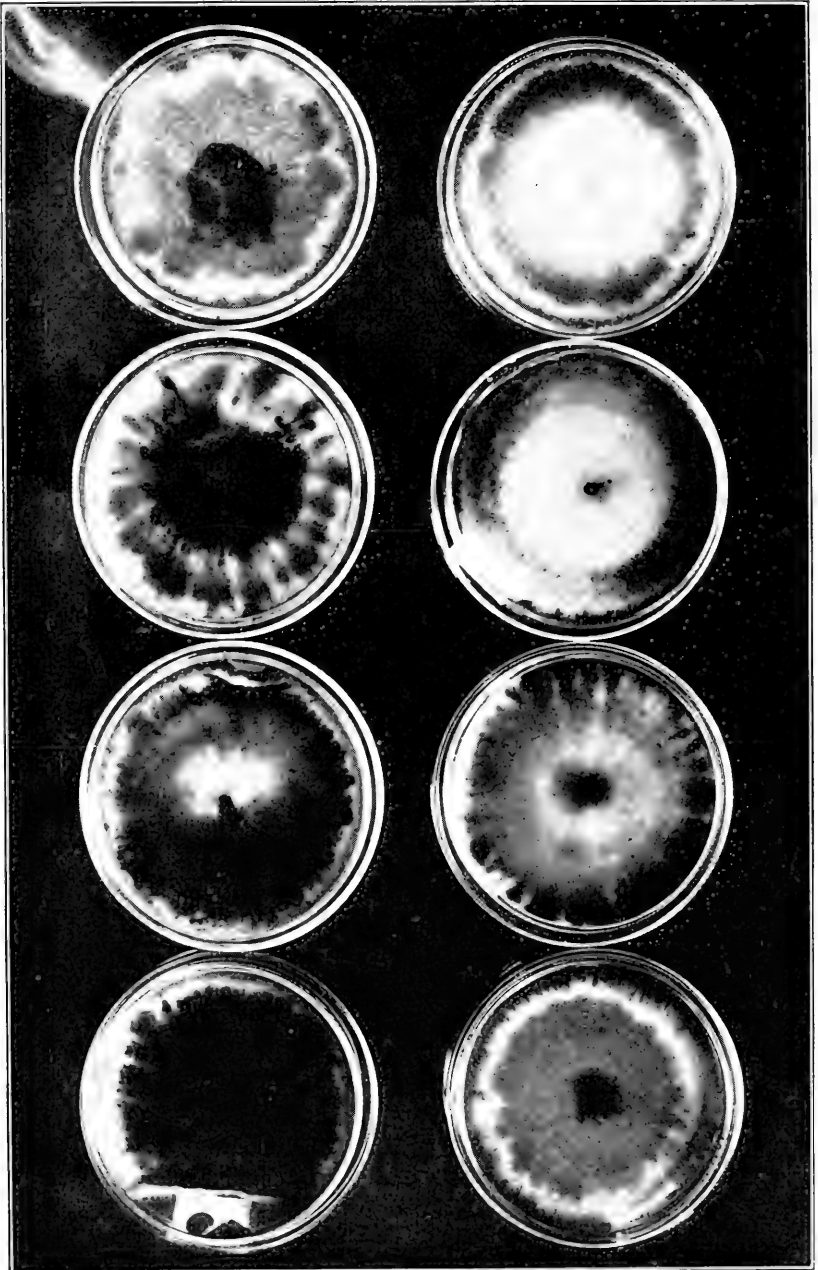


Fig. 17.

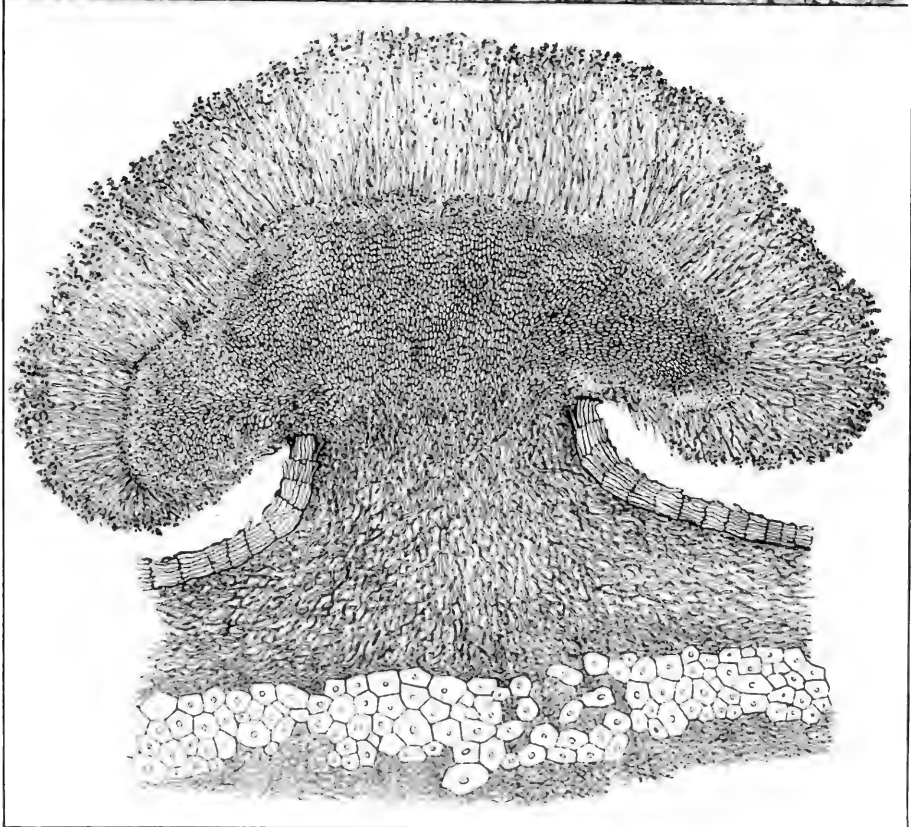
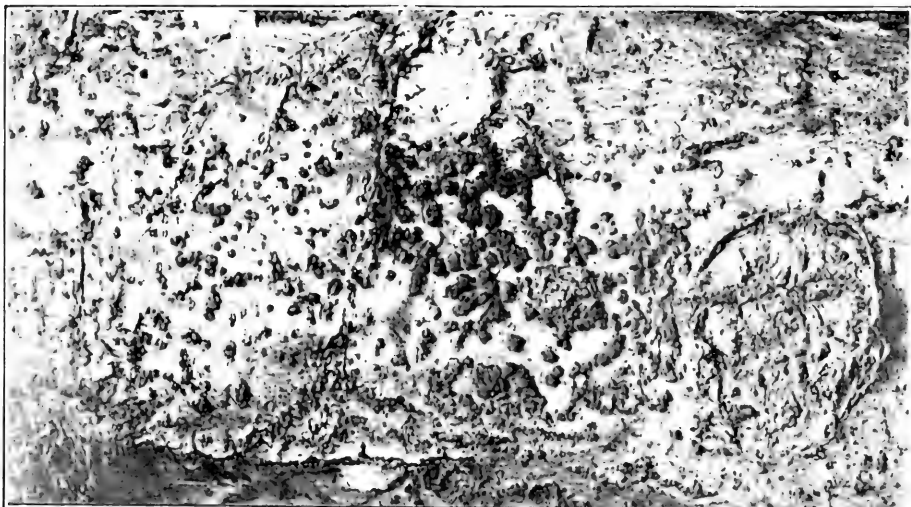


Fig. 18.

Fig. 19.

PLATE XII.

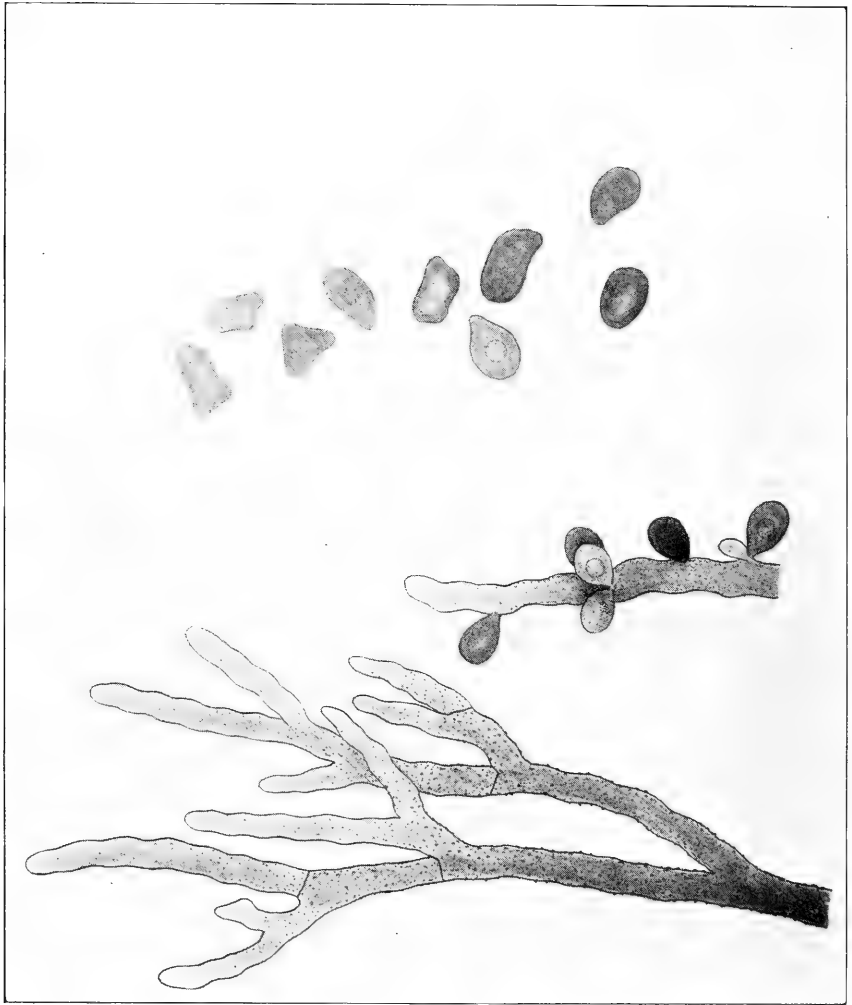


Fig. 22.

Fig. 21.

Fig. 20.

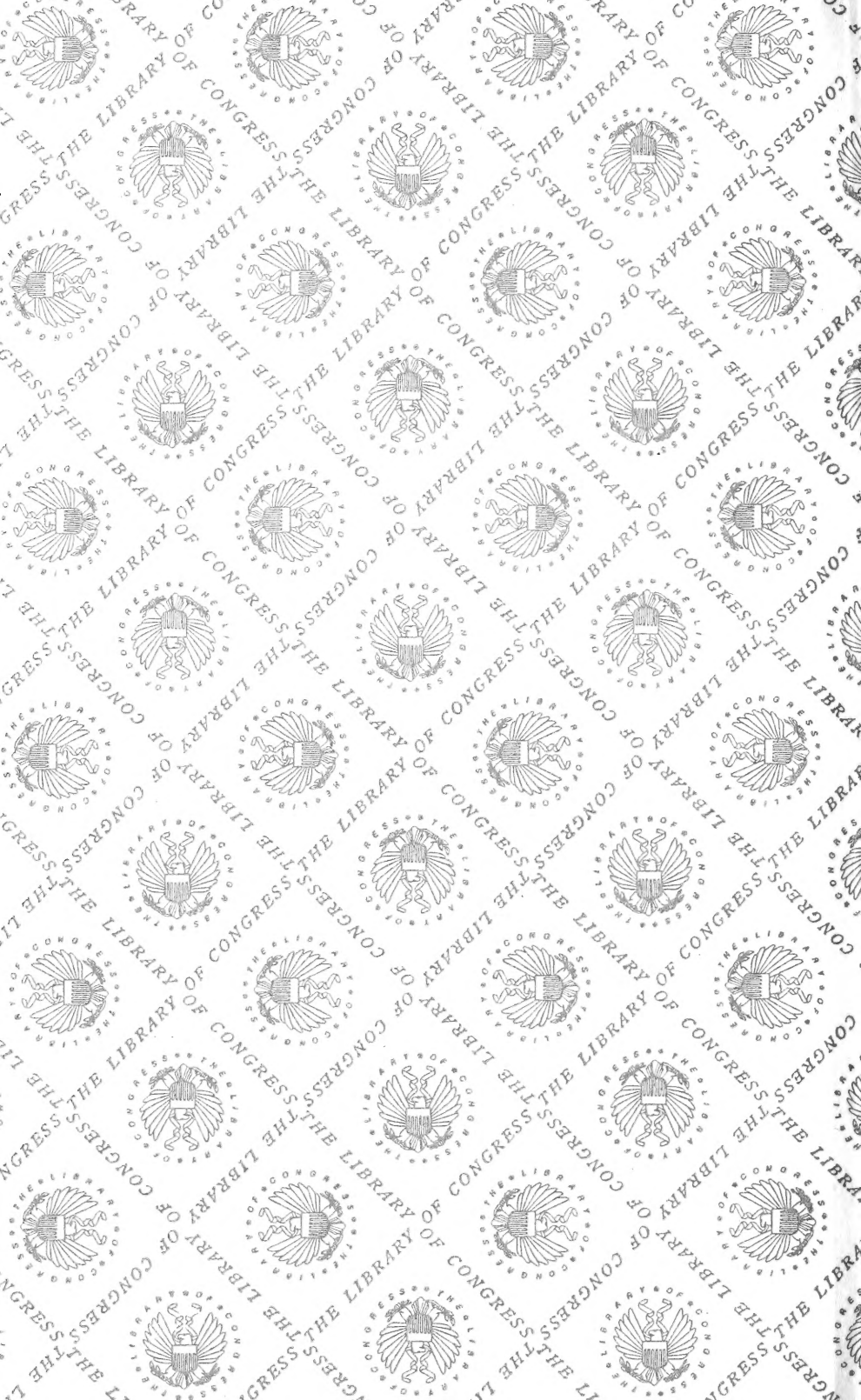
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