Cucumber and Tomato Canker. (*Mycosphaerella citrullina*, Grossenb.)

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the borders of cornfields, suggest its accidental introduction with seeds from the Mediterranean region. Some authors prefer the name *P. nodosa*, L., to *P. bulbosa*, L., but Hackel explained in 1880 (*Cat. rais. Gram. Portug.* p. 2) why *P. bulbosa* has to stand, and it is hardly necessary to repeat his arguments.

How far the claims of the grass as a first-class fodder grass are justified is still to be seen, but the reports from Australia as well as from Natal are undoubtedly promising.

XXXVIII.—CUCUMBER AND TOMATO CANKER.

(*Mycosphaerella citrullina*, Grossenb.)

(With Plate.)

G. MASSEE.

Dr. Grossenbacher has recently described a disease caused by *Mycosphaerella citrullina*, Grossenb., which proves very destructive to melons in the United States. The disease is first indicated by the wilting of the leaves, after which the entire plant collapses and dies. The nodes of the stem, especially those nearest the root, present a water-logged appearance, often accompanied by an exudation of gum. At a later stage the diseased patches change to an ashy-grey or whitish colour, and the epidermis becomes broken up and studded with numerous perithecia belonging to the conidial stage of the fungus. This is followed by the production of the ascigerous condition of the fungus when the host is dying or dead. Infection experiments proved that the spores of the fungus could infect uninjured melon plants, also other allied plants, although curiously the cucumber proved immune, and resisted all attempts at infection in the United States.

Quite recently specimens of diseased tomato plants were received at Kew, from Waltham Cross. In each case the base of the stem was considerably shrunk, and the cortex destroyed, and studded with numerous minute perithecia, which on examination proved to be identical with the conidial or *Ascochyta* stage of the American melon disease. In some specimens the nodes of the stem were also attacked. Plants attacked as above promptly succumbed; a grower's statement being that they fell over like nine-pins.

Almost simultaneously with the above, a diseased cucumber plant was received from Gloucestershire, which showed the whitish diseased nodal portions of the stem, characteristic of the American melon disease. Numerous minute perithecia, which proved on examination to be those of the *Ascochyta*, were present on the diseased patches.

Cucumber plants attacked by this disease are stated to be quickly killed outright. Experiments conducted at Kew showed that the spores from diseased cucumber plants would infect young tomato plants, and that spores from a tomato plant would infect vegetable marrow plants. In both instances the plants infected showed the conidial (*Ascochyta*) stage of the fungus within a fortnight, and in both instances the lesions formed were characteristic.

TOMATO CANKER.

Both in this country and in the United States the conidial form of fruit is responsible for the rapid extension of the disease.

The simultaneous occurrence of this well-known American disease at two widely separated localities in this country is difficult to explain, as living melon plants are not imported, and the fungus does not attack the fruit.

Conidial form. Pycnidia depressed-globose, wall thin, parenchymatous, pale brown, mouth minute, distinct, 90–160 μ diam., produced under the epidermis, then bursting through; conidia hyaline, cylindrical, becoming 1-septate and slightly constricted at the centre, 9–16 $\times$ 4–6 μ .

Ascigerous form. Perithecia depressed-globose, rough, dark coloured, mouth papillate, becoming almost superficial, 100–165 μ diam.; asci cylindric-clavate, 8-spored; spores hyaline, oblong-fusoid, 1-septate, distal cell often largest, usually more or less constricted at the centre.

The result of preventive experiments conducted in this country are not yet recorded. According to Grossenbacher spore inoculations conducted in the field were almost complete failures, and it is considered that the very moist, warm environment of the greenhouse is essential for the infection and development of the fungus. Further experiments showed that the spores of the fungus are not killed by exposing infected soil to weathering conditions of winter, nor by fumigation with hydrocyanic gas. It seems that an epidemic may be prevented by spraying thoroughly with Bordeaux mixture before the disease appears, and repeating at intervals, to keep the plants covered with the solution.

J. G. Grossenbacher; N.Y. Agric. Expt. Sta., Technical Bull. No. 9 (1909).

XXXIX.—LIGNUM NEPHRITICUM.

(*Eysenhardtia amorphoides*, H.B.K.)

OTTO STAFF.

Early in the present year an inquiry was addressed to the Director of the Royal Botanic Gardens, Kew, concerning the origin of *Lignum nephriticum*, a wood which formerly, but mainly during the seventeenth century, attracted a good deal of attention on account of its medical as well as physical properties, but subsequently was completely lost sight of. It had been referred by authors to various species; but it was quite clear that so far as the pharmacological literature was concerned, nothing definite was known as to its origin. The most remarkable character of the wood, and the one on which all the earlier writers insisted, was that a watery infusion of the wood assumed after a certain time a blue colour. In the Kew Museum the only sample professing to represent the product was a specimen which had been obtained from Messrs. Menier & Cie., Paris, as Bois néphrétique, in 1851. It was not named botanically, but had the appearance of a sapotaceous wood, and did not give the characteristic reaction with water. Linnaeus referred *Lignum nephriticum* to