but the absence of the latter is atoned for by the presence of a table of contents just before the introduction. As in most of Dr. Mortensen's publications the illustrations are all that could be desired and are of the greatest service to the user of the book, while the text is entirely free from ambiguities and shows the customary positiveness of the writer. The whole appearance of text and plates is admirable and the Carlsberg Fund, no less than the author, is to be congratulated on this very important contribution to our knowledge of echinoderms.

HUBERT LYMAN CLARK

SPECIAL ARTICLES

A BACTERIAL WILT OF THE BEAN CAUSED BY BACTERIUM FLACCUMFACIENS NOV. SP.

A NEW bacterial disease of navy beans has appeared in South Dakota. The grower on whose farm the disease was discovered reports that what he believes to be the same disease killed 90 per cent. of his 1920 crop. In 1921 he planted the seed harvested from the remainder and lost about 25 per cent. of his crop. Some of this 1920 Dakota seed planted at Arlington, Virginia, also produced a large proportion of diseased plants, many of which never survived the seedling stage. The disease is characterized by a wilting of the leaves of seedlings sometimes accompanied by a discoloration, and by dwarfing, reduction of yield and the death of some of the shoots, if the plant survives the early stages of growth.

Plants from South Dakota, received in the Laboratory of Plant Pathology, Bureau of Plant Industry, August 6, 1921, were found to contain bacteria in the vessels of the stem often accompanied by a browning of the vascular ring. The writer suspected the presence of *Bacterium solanacearum* Erw. Sm. but when petri dish poured-plates were made from the diseased stems a yellow organism was isolated. This, when pricked into vigorously growing King of the Mountain bean seedlings produced the wilt in every case. From these infected plants the yellow organism has been reisolated and has produced the wilt in Great Northern beans. King of the Garden lima and Ito San soy-beans have also become infected as the result of pure culture inoculations.

The same organism has been isolated from the Arlington, Virginia, plants and with it the writer has reproduced the disease.

The discoloration mentioned above may consist of a dull green or brownish green, greenish brown or reddish brown area sometimes bordered with yellow. The discolored area is flabby at first and later dry and papery. In many cases the whole leaf blade and petiole become flabby and droop without any discoloration at all, whereas in others a portion of the leaf becomes flabby and discolored while the rest of the blade and the petiole is turgid for a time at least. It is presumably a question of the number of vessels plugged by the bacteria. This same phenomenon has occasionally been observed in secondary infections of young leaves by *Bacterium phaseoli*.

The wilt of the seedlings in some respects suggests the "systemic disease" of beans ascribed by Burkholder to *Bacterium phaseoli* but the parasite under consideration is very different from *Bacterium phaseoli*. For example, its very moderate, often scanty growth on potato cylinders, due to its very slight diastasic action, is in marked contrast to the exceedingly copious prolonged growth and marked diastasic action of *Bacterium phaseoli* Erw. Sm. The color on potato is Ridgway's mustard or primuline yellow (Color Standards and Nomenclature, plate XVI, 2nd ed., 1912) and there is usually a marked graying of the potato. The difference in the colonies are less marked but plates of the two organisms when compared are easily distinguishable. The colonies of *Bacterium phaseoli* are much more wet-shining and of a much more syrupy consistency. Both *Bacterium phaseoli* and the South Dakota organism reduce the litmus in litmus milk in 4 to 7 days but the cultures of the latter finally become acid and the behavior of the two organisms is very different in regard to the manner and time of the other changes taking place in the milk. Cultures of *Bacterium phaseoli* begin to clear in 1 to 6 days, and a very soft mobile curd is formed, a partial peptonization

of the casein preventing the formation of a solid coagulum. In cultures of the South Dakota organism, on the other hand, there is usually no visible change in plain milk for about three weeks, after which, in varying lengths of time, the fluid becomes solid. Some days after coagulation there is an extrusion of whey and finally peptonization begins, progressing very slowly however. *Bacterium phaseoli* produces tyrosin crystals in abundance in milk but none have been observed in cultures of the South Dakota organism. The latter produces a wide, deep yellow rim (Ridgway's primuline yellow loc. cit.) which is very striking.

Both organisms liquefy gelatin but *Bacterium phaseoli* does it rapidly whereas the South Dakota organism does it so slowly that for the first month there is little or no liquid gelatin present, evaporation taking place almost as rapidly as the liquefaction.

Another good medium for differentiating these two organisms is Congo Red agar. Both organisms take up the stain to a greater or less degree and finally change the agar to a purplish color but *Bacterium phaseoli* makes a very thick, smooth, wet-shining growth and the South Dakota organism only a very meager one. This medium is prepared as follows: 1000.00 c. c. distilled water; 10.00 g. saccharose; 1.00 g. dipotassium phosphate; 0.20 g. magnesium sulphate; 15.00 g. agar flour; 0.10 g. Congo red (Grübler's). Steam the water and salts one half hour, then add Congo red. Filter through cotton and tube. Autoclave tubes fifteen minutes at 115° C.

The bacterium causing the wilt is a polar flagellate rod 2-3 to 3μ by 1-3 to 1-2μ occurring singly or in pairs, and has been named *Bacterium flaccumfaciens* nov. sp.

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THE PERIPHERAL CIRCULATION IN MUSCLE INJURY SHOCK

THE following experiments were undertaken in an attempt to determine the influence of the peripheral tone in the production of the low blood pressure initiated by muscle injury. Evidence has been presented by a number of observers indicating that the vasomotor center is still active in shock produced by the exposure of the viscera or aortic occlusion, and that some peripheral tone is maintained.[1] A recent paper by Erlanger, Gesell and Gasser[2] presents results of a series of experiments in which, in these types of shock, the condition of peripheral constriction was directly determined by the rate of perfusion through the arterioles and capillaries. They show that during the development of shock the peripheral resistance is increased, and that only after the arterial pressure has fallen is there a loss of vasomotor tone, and consequently that a loss of tone is not the primary cause of shock. Our results are in accord with these findings and are presented as evidence indicating that the nervous factor is of minor importance in the causation of the low blood pressure following muscle injury, as in other forms of shock.

The method of determining the relative condition of vasomotor tone was that described by Bartlett,[3] and used by Erlanger, Gesell and Gasser. The rate of inflow of a fluid at constant pressure through the femoral artery of one hind limb was determined at intervals during the development of shock. The inflow cannula was placed in a side branch of the femoral artery or low down on the main branch directed towards the heart. With this arrangement, through the use of clips on the arteries, it was possible to shift quickly from the natural blood supply of the area supplied by the intact branches of the femoral artery to the perfusion fluid and *vice versa*. The perfusion fluid was

[1] Porter: *Am. Jour. Phys.*, 1907, XX: 399. Porter and Storey: *Ibid.*, 1907, XVIII: 181. Porter and Quinby: *Ibid.*, 1908, XX: 500. Seelig and Lyon: *Jour. A. M. A.*, 1909, LII: 45; also *Jour. Surg. Gynecol. and Obstet.*, 1910, 146. Seelig and Joseph: *Jour. Lab. and Clin. Medicine*, 1916, I: 283. Mann: *Johns Hopkins Hosp. Bull.*, 1914, XXV: 205. Morison and Hooker: *Am. Jour. of Phys.*, 1915, XXXVII: 86.

[2] Erlanger, Gesell and Gasser: *Am. Jour. of Phys.*, 1919, XLIX: 90.

[3] Bartlett: *Jour. Exp. Med.*, 1912, XV: 414.