about a highly concentrated urinary output, containing large amounts of both chlorides and urea, while the hormone of the adrenal cortex induces a large urinary volume having a relatively low concentration of urea and chlorides. It is noteworthy, too, that the concentrations of both urea and chlorides in the urine tend to vary together, in one direction or the other, according to the hormone used.

A true diuretic substance is said to be one which will "consistently and reproducibly elevate the urine flow, as determined by catheterized specimens in well-controlled experiments, from moderate rather than very low levels; the diuresis should reach a rate at least comparable to that observed after a moderate dose of water, and it should persist for a period of at least 30 minutes."[4] In our experiments the hormone of the adrenal cortex as a diuretic factor fulfils these conditions in every respect.

The hypothesis that the cortico-adrenal hormone acts as a diuretic agent and in antagonism to the antidiuretic hormone of the posterior lobe of the pituitary explains the puzzling fact that hypophysectomized animals show only a transient polyuria, while animals from which the posterior lobe alone is removed show a persistent diuresis. When the posterior lobe only is excised, lack of its antidiuretic hormone plus the presence of the diuretic adrenal hormone would lead to polyuria through decreased water reabsorption from the renal tubules. When both lobes are removed, the uncompensated activity of the diuretic cortico-adrenal hormone would persist until the adrenal cortex (in the absence of the corticoadrenotropic hormone of the anterior lobe) becomes atrophic, at which time both posterior-lobe antidiuretic hormone and cortico-adrenal diuretic hormone would be absent from the bloodstream and polyuria would disappear.

From many considerations, now omitted for lack of space, it appears that while post-pituitary action may be exerted chiefly in the direction of inhibition of the aqueous portion of urinary excretion by the kidney, the influence of cortico-adrenal extract (in so far as its renal action is concerned) is directed towards increased water elimination. Thus the present experiments seem to establish the physiological antagonism of the post-pituitary and cortico-adrenal principles in their action on urinary secretion.

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[4] H. W. Smith, "The Physiology of the Kidney," New York, 1937, p. 229.

ISOLATION OF THE FUNGUS CAUSING THE RED STELE OR RED CORE DISEASE OF STRAWBERRIES

A SERIOUS root rot of the strawberry was described in Scotland in 1926, under the name "Lanarkshire disease."[1] The term "red core root" was proposed for the disease in 1929,[2] as being descriptive of its most characteristic symptom. A disease presumably identical with "red core root" was first reported in the United States in 1935 from Illinois, under the name "black-stele root rot."[3] In 1937[4] the "black stele" or "red stele" disease was reported from Virginia, Maryland, New Jersey, New York and Michigan, in addition to Illinois. This disease now appears to be spreading in the Middle Atlantic States and is becoming decidedly injurious in restricted areas where soil and climate are favorable to its development.

The red-stele disease may be easily distinguished from other strawberry root rots by the fact that the causal organism, while completely destroying fine, fibrous roots, advances up the central cylinders of larger roots, killing the interiors and causing them to turn dark red, while the surrounding cortex still appears alive and healthy. The fungus eventually advances to the stem of the plant, but apparently does not invade it. The larger roots die gradually, beginning at the distal ends, some time after the steles have been killed. As a consequence of the injury to the root system, affected plants wilt, older leaves die, and newly formed leaves remain very small with conspicuously short petioles. Badly diseased plants often succumb during the heat of spring and summer, but lightly affected plants grow normally through the summer, only to suffer a renewed attack by the fungus late in the season.

Red stele was first attributed to such causes as poor cultural conditions and parasitism by mycorrhizal fungi and by Pythium species. Since 1929[5] there appears to have been general agreement that the disease is caused by an undetermined species of Phytophthora. Oospores of the Phytophthora type occur commonly and abundantly in diseased roots, and sporangia and zoospores characteristic of the species may be induced by submerging recently infected young fibrous roots in cool water for a period of 18 hours

[1] Claude W. Wardlaw, "Lanarkshire Strawberry Disease, a Report for the Use of Growers (with preface by Professor J. M. F. Drummond)". Carluke (Scot.). Printed by J. Bell, 1926. Issued by Botany Department, University of Glasgow.

[2] Nora L. Alcock, Gard. Chron., 3 (86): 14–15, illus., 1929.

[3] H. W. Anderson, *Phytopathology*, 25: 5, 1935.

[4] J. B. Demaree and G. M. Darrow, U. S. D. A. *Plant Dis. Rptr.*, 21 (22): 394–399, December 1, 1937.

[5] Nora L. Alcock, *op. cit.*

or more. However, a survey of the literature discloses that previous attempts to isolate the fungus and grow it in pure culture have been unsuccessful. In the absence of pure cultures of the organism, it has necessarily been impossible to prove conclusively that the associated Phytophthora causes the disease.

The present note reports successful isolation of the Phytophthora from strawberry roots, proof of its pathogenicity, and some comments on its behavior in culture.

The fungus was isolated in November, 1937, from roots of diseased strawberry plants growing on the U. S. Horticultural Station farm at Beltsville, Md. Fall roots were well developed at the time, and the fungus was actively attacking the distal fibrous roots and had advanced a short distance up the steles of some of the main roots. Large roots with diseased steles but healthy cortex were thoroughly washed in sterile water, the cortex was carefully removed under aseptic conditions, and the upper 2 cm of the diseased zones were cut in pieces about 5 mm long. The pieces were mounted in a hardened drop of water agar on a coverglass, covered with an additional drop of agar, and inverted over a Van Tieghem cell. Growth of hyphae was followed under the microscope, and either developing hyphae or the entire root pieces, whichever seemed advisable from appearances, were transferred to test-tubes. Approximately 50 per cent. of the pieces yielded Phytophthora in pure culture, or contaminated only with bacteria, which were later eliminated from the cultures. A similar series of isolations from diseased plants collected by Dr. H. W. Anderson near Vermilion, Ill., late in March, 1938, yielded only 4 cultures of Phytophthora out of 45 isolations, most of the others giving a species of Pythium which evidently follows the Phytophthora very closely in the roots.

Pathogenicity was proved by inoculating mycelial cultures and zoospores to roots of potted strawberry plants which had been previously rooted from runners in autoclaved soil. Zoospore inoculation proved to be the simplest method. Abundant infection and some oospore production in fine roots was present six days after inoculation. Under greenhouse conditions wilting began three weeks after inoculation, and at this time the root systems were almost completely destroyed, showing all the characteristic symptoms of naturally infected plants. The organism was recovered in pure culture from inoculated plants by the method used in the original isolations, from the reddened steles of affected larger roots. Uninoculated check plants remained healthy.

The identity of the species is still being investigated. As had previously been determined[6] on its host plant, the fungus belongs to the group of Phytophthoras having large, non-papillate sporangia, comparatively large oospores and predominatingly amphigenous antheridia; but cultural and cross-inoculation studies have so far failed to place it accurately in any described species.

In culture, the Phytophthora grows best on oatmeal agar and in canned pea broth,[7] and somewhat less rapidly on lima-bean agar. Growth is unsatisfactory on all other media tried, including cornmeal agar. Neither sporangia nor oospores have occurred in the culture media tested up to the present time, but sporangia and zoospores develop in abundance when small blocks of mycelium, grown in thin layers of lima-bean agar, are shallowly irrigated with cool tap water. The fungus has a relatively low temperature range, mycelium being killed in one week at 30° C., while zoospores are produced at 10° C., but are inhibited at 22° C., the range of maximum production lying between 14° and 18° C. It is perhaps needless to point out that, together with mechanical distribution of diseased plant parts, zoospore dispersal constitutes the principal means of spreading the disease in the field.

Spore and sporangial measurements of the American red-stele organism come well within the range described for the Phytophthora associated with the Lanarkshire disease in Scotland, and since the symptoms are also similar in most respects, it appears probable that the two diseases are identical.

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THE MEASUREMENT OF THE EXTERNAL FORCE IN WALKING

FOR an adequate study of human locomotion it is necessary to measure with considerable precision the force exerted on the substratum by the foot at each instant during progression. Since this force is the resultant of the force of gravity and the reaction to the acceleration of the body at the moment, its measurement provides a means of determining the acceleration and so of the force concerned in the movement of the body as a whole. Three measurements are necessary if the force is to be known both as to magnitude and

[6] Ibid.

[7] L. H. Leonian, *W. Va. Agr. Exp. Sta. Bull.*, 262 (p. 13), 36 pp., 1934.