of such a correlation scheme depends not alone upon the extent to which the foraminifera can be found in beds wherein their presence previously has not even been suspected, but also upon whether such primitive types change sufficiently during a relatively short elapse of geologic time to be really diagnostic. The tremendous geologic range of such species as the modern *Ammodiscus incertus* (d'Orbigny) might seem to indicate that they do not, but on the other hand there is no difficulty in separating Alexandrian Brassfield samples from the Niagaran Bainbridge sediments solely on the basis of differences in their foraminiferal remains. That a number of other mid-Paleozoic formations may yield foraminifera when the unsoluble residues are studied is suggested by the fact that we have also found the Helderbergian Bailey formation of southeastern Missouri to contain their abundant remains, though the assemblage has not yet proven to be particularly diversified. This new fauna is being described by Mr. Hunter.

In addition to the very definite discoveries mentioned above, it may be well to list the less positive and the entirely negative results of our studies. The Oriskanian Little Saline limestone pretty certainly contains these microscopic organisms, although our specimens are thus far both rare and discouragingly fragmentary. The mid-Devonian Grand Tower, Beauvais and St. Laurent formations have been carefully examined, thus far with only negative results. The Ordovician Joachim formation apparently does not contain foraminifera, but there is evidence, still far from certain, that the Plattin, Kimmswick and Fernvale may yet yield unquestionable specimens. A number of older formations of the Ozarks, such as the Gasconade, Jefferson City, Cotter and Powell, also have been studied, but without positive results. As H. S. McQueen, of the Missouri Bureau of Geology and Mines, however, has run literally thousands of careful mineralogical studies on the insoluble residues of these sediments without noticing foraminifera, we may at least assume that very few of the organisms were preserved in these strata.

CAREY CRONEIS
PAUL H. DUNN
DAVID HUNTER

WALKER MUSEUM,
UNIVERSITY OF CHICAGO

TREATMENT OF COTTON ROOT-ROT WITH AMMONIA

A TOXIC effect of ammonia on the root-rot fungus, *Phymatotrichum omnivorum* (Shear) Duggar, has been determined as a result of a series of experiments in growing the fungus in different media, during the season of 1931. When ammonium salts were used as the source of nitrogen in Duggar's solution, the growth of the fungus was notably restricted, and this indication was confirmed by subsequent tests. It was found that both the mycelial and sclerotial stages of the fungus were killed by short exposure to the gas, and where dilute concentrations of the hydroxide were applied under field conditions, the fungus was killed in the tissues of cotton roots.

In the cultural experiments with ammonium nitrate and ammonium sulfate, when used at a concentration to yield approximately 12.4 grams of nitrogen per liter, very little growth of mycelium appeared after intervals of 11, 18 and 31 days, whereas the other sources of nitrogen, *viz.*, calcium nitrate, potassium nitrate and sodium nitrate, produced an abundant growth.

After pH readings had revealed that the suppression of growth with the ammonium salts was not attributable to an acid reaction of the media, the apparent toxic effect was studied. The procedure was as follows: fresh cultures were prepared in 125 cc Erlenmeyer flasks containing 50 cc of neutral carrot agar, and after growth was well established over the media, the mycelium in each of four flasks was exposed for 20 minutes to dilute ammonium hydroxide (.1, .5 and one per cent. by volume, prepared from 28 per cent. ammonia water). Four untreated flask cultures were reserved as checks. After the above exposures, transfers were made immediately from the treated and check flasks to neutral carrot-agar slants in test-tubes. In every case, the inoculum from the cultures treated with ammonium hydroxide failed to grow, but growth occurred in all cases from the check inoculum.

Other flask cultures were subjected to ammonia treatment for 30 seconds by generating the gas from 500 cc of 28 per cent. ammonia water and allowing this to enter the culture flask by means of glass tubing. The gas treatment also completely inhibited growth of the fungus when transfers were made. Further tests were made with root-rot sclerotia by exposing them to ammonia, chlorine and formaldehyde gas for short intervals (10, 15 and 20 seconds) with the ammonia treatments showing complete mortality.

Roots obtained from freshly wilted cotton plants were exposed to the same gases for intervals of 30 seconds and of 1 and 2 minutes and later tested for viability of the fungus in moist chambers. The ammonia treatments prevented growth, while the formaldehyde and chlorine were only partially effective. Good growth occurred in all of the checks.

Field tests have also been conducted at various times during the season to compare the effectiveness of 6 per cent. solutions of ammonium hydroxide, formalin and sodium hypochlorite when applied in the soil to the roots of infected cotton plants. In

these experiments large mature plants were selected and treated with the different disinfectants by saturating the soil about the roots of the plant. After forty hours the plants were removed from the soil and the roots placed in moist chambers. Observations were made at one-day and five-day intervals. Plants treated with ammonium hydroxide showed no growth of mycelium; the plants treated with formalin, slight growth; those treated with sodium hypochlorite, good growth; the checks, good growth.

The disinfection with ammonia appears to be more complete than with other chemicals used in comparative tests, and the danger of killing adjacent plants, as by formalin treatments, is avoided. The possibility of utilizing ammonia or ammonium compounds for the control of the disease in cotton fields as well as for protecting ornamentals or shade trees is suggested, and further experiments are being made.

D. C. NEAL
R. E. WESTER
K. C. GUNN

BUREAU OF PLANT INDUSTRY,
U. S. DEPARTMENT OF AGRICULTURE,
GREENVILLE, TEXAS

PREVENTION OF BLOOD COAGULATION BY CYSTEINE[1]

IN 1928[2] one of the writers reported that when oxidation in Berkefeld filtrates of the Rous chicken tumor was prevented by means of cysteine, the spontaneous loss of infectivity of the filtrates which takes place rather quickly with this virus is very markedly inhibited. At the same time a precipitate which usually forms under aerobic conditions, varying in degree from a slight opalescence to a definite flocculation, no longer occurs, and the filtrates remain water clear for days, as long as they are protected from the air. It was at first believed that this precipitate was in some way connected with the filterable virus, since "infectivity" disappeared more or less parallel with its formation. Control experiments, however, in which Berkefeld filtrates of embryonic chick tissues were prepared, showed this precipitate to an even more marked degree than tumor filtrates, and here, too, its formation was largely or completely prevented by reducing with cysteine.

The precipitate appeared to consist of protein, and the possibility was obvious that it resulted from a slow coagulation of some of the saline soluble tissue proteins and that oxidation played a part in its formation. It was logical to inquire whether oxidation might enter into the chain of events resulting in the coagulation of blood. There has been in the past some evidence to this effect, but as far as we could find from a hasty review of the recent literature, nothing very direct.

It was a simple matter to withdraw a few cc of human blood in an oiled syringe and transfer 1 cc quantities to three test-tubes, each containing two glass beads. One tube served as control, containing 0.5 cc of saline. The second tube contained 0.1 g of cysteine hydrochloride, neutralized with NaOH in 0.5 cc saline, and the third tube 0.1 g of alanine in saline as a control on the NH_2 and COOH portion of the cysteine. The blood in each tube was quickly covered with 2 cm of melted vaseline. By tilting the tubes at intervals, it was possible to observe when coagulation took place by the movement of the glass beads. Tubes 1 and 3 coagulated in about ten minutes. Tube 2 remained completely uncoagulated for twenty-four hours, was then opened, the blood drawn off, and found to be quite fluid, and was discarded. Repetition of the experiment showed that 0.01 g of cysteine hydrochloride was insufficient to prevent the coagulation of 2.0 cc of blood, but that 0.05 g would do so.

Further experiments indicated that thorough aeration of the cysteinized blood, leading to oxidation of the cysteine to cystine, would result after some time in coagulation.

The further analysis of the mechanism of this phenomenon is definitely outside the scope of a bacteriological department, but the experiment can be so easily repeated that it seems worth while to describe it in the hope that by means of it some further light may be shed on the obscure question of blood coagulation.

J. HOWARD MUELLER
SOMMERS STURGIS

[1] From the Department of Bacteriology and Immunology, Harvard University Medical School, Boston.

[2] J. H. Mueller, SCIENCE, 68: 1752, 88, July 27, 1928.

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