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.

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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.

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[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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