butions to the knowledge of plague, particularly in the matter of the importance of the pigmy vole (*Lagurus curtatus*) as a reservoir host.

In June 1936, the Idaho Department of Public Health initiated its own survey unit which operated until July 1938. It was reactivated during 1941 and 1942.

The Oregon State Board of Health began its plague surveys in August 1936. These have continued to the present time.

In April 1937, the Utah State Department of Health organized a survey unit which continued working until July 1938.

The Montana State Board of Health initiated plague surveys in May 1937. These were continued until 1946.

Three States have carried on plague surveys with financial assistance from the Communicable Disease Center. In 1946, following the first demonstration of wild rodent plague in Texas, the Texas State Department of Health and the Communicable Disease Center set up a plague study headquartered at Brownsfield. This study continued until July 1, 1949. During 1948 and 1949, plague surveys were conducted in Colorado and Utah as a joint effort of the Communicable Disease Center and the health departments of those States.

In 1949, New Mexico experienced the first of six human cases of plague. To date, plague has been found in 20 of the 31 counties. Except for California, New Mexico has reported the largest number of human cases of plague, the largest number of wild rodent plague foci, and the largest number of counties involved. For these reasons, the New Mexico Department of Public Health, in cooperation with the Western Communicable Disease Center Laboratory, has been operating its own survey unit since 1950.

It is somewhat surprising, at first glance, to note that so many of the western State health departments have actively participated in these plague surveys for such a long time. The actual number of human infections from wild rodent sources has been small in relation to other more important causes of human diseases. Yet plague still remains a potential threat of unknown proportions. There are indications that it can still cause deaths in spite of the remarkable efficacy of antibiotics in its treatment, and no one knows when another epidemic may start. For these reasons, it is hoped that Western States will continue their support of plague surveys within their boundaries.

Serologic Titers in Rickettsial Infection as Affected by a Course of Antibiotics

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The question of what effect antibiotics have upon the Weil-Felix and complement fixation titers in rickettsial infections has concerned laboratory workers for some time. Although numerous studies have been made on the subject, the data as a rule have been reported as parts of other studies. Such information was compiled and is presented here.

In 1945 Rose, Duane, and Fischel (1) studied the treatment of Rocky Mountain spotted fever with para-aminobenzoic acid. Although treatment was started early in the disease (third day of onset), serologic specimens taken at regular intervals showed a rise in titer which reached 1:1280. Sadusk, Hjerpe, and Freedman (2) studied the effect of para-aminobenzoic acid upon the clinical course of typhus in the guinea pig. Administration of para-aminobenzoic acid to guinea pigs infected with murine typhus generally prevents the appearance of clinical signs of this infection but permits appearance of rickettsia in the circulating blood and the formation of complement-fixing antibodies. Complement-fixing antibodies and rickettsemia develop in a comparable degree in both treated and untreated control animals at about the same time. The rise in titer

during the period of convalescence is also similar in both groups of animals.

When antibiotics replaced para-aminobenzoic acid as the drug of choice, a somewhat different picture was obtained. Cox (3) states that in mild rickettsial infections or in those cases where the disease was diagnosed quite early, aureomycin modified the course of infection, rendering it exceedingly mild, or even aborted the infection. Under these conditions, experimental animals as well as man may show no demonstrable complement fixation antibodies. If guinea pigs are infected with massive doses of rickettsia and then treated early with aureomycin, clinical signs of the disease may be prevented although complement fixation antibodies may appear in the blood.

Angstein, Whitney, and Beninson (4) studied the protective effect of aureomycin in experimental Rocky Mountain spotted fever and typhus. Complement fixation tests were made on guinea pigs infected with spotted fever. Guinea pigs were given aureomycin 2 hours after intraperitoneal infection and were bled on the fourteenth day. A low complement fixation titer of 1:16 and 1:32 was found in guinea pigs treated with 2.0 mg./dose, as compared to higher titers (1:128 - 1:256) of the group treated with 1.0 mg.

Schoenbach, Bryer, and Long (5) treated a number of patients ill with Rocky Mountain spotted fever (eastern type) with aureomycin. The treatment was started after hospital admission at about the third day of the disease. Weil-Felix tests on one patient at about the fifth day were positive at 1:80 dilution and 1:1280 on the thirteenth day. Similarly, another patient with a Weil-Felix titer of 1:20 on the day of admission was 1:160 on the seventh day after.

Smadel (6) studied the effect of chloromycetin on scrub typhus. Results with the Weil-Felix test indicated that the average titer was one dilution lower with treated patients as compared to untreated patients. There was no significant difference in the time of appearance of Weil-Felix antibodies in treated and untreated patients. However, a group of volunteers whose disease was terminated within less than 3 days after onset had a delay in the appearance of Weil-Felix antibodies but had an average titer of 1:640. Complement fixation tests with scrub typhus could not be compared since the incidence of positive reactions were approximately 50 percent in treated and untreated cases.

In conclusion, it is evident that the introduction of chloromycetin, aureomycin, and other antibiotics for the treatment of rickettsial infection has greatly changed the course of the disease in humans and animals. The serologic picture has also changed but to a lesser degree. Experiments have shown that the Weil-Felix test will detect antibodies in rickettsial infections in untreated as well as treated patients. Treated patients, however, will show a somewhat lower titer. Results with the complement fixation test give a more variable picture and are less predictable. It is seen that a patient must be severely infected in order to show complement fixation antibodies. Complement fixation antibodies usually develop on the fourteenth day after the infection, accompanied by a corresponding rise in titer with time. The Weil-Felix antibodies, however, will develop on the fifth day, accompanied by smaller rise in titer. Weil-Felix antibodies can be detected when the infection is of a lighter nature such as that modified by antibiotics, whereas the complement fixation antibodies may not be demonstrable. From this, it would appear that the importance of the Weil-Felix test in the serologic analysis of the rickettsial infections has by no means decreased.

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