On July 1, 1933, another cow, 501, received 20 cubic centimeters of blood from deer 2. As the infection progressed in this cow, an abundance of marginal bodies were noted in the blood; a severe anemia developed, no signs of regeneration appeared and the animal died on August 8, 1933. The pathological changes revealed at autopsy were characteristic of anaplasmosis, as in the case of the previous transmission.

From these experiments, it is evident that certain species of deer, at least, are carriers of anaplasmosis without presenting any symptoms or other definite signs of the disease themselves. By injecting cattle with blood from the southern black-tailed deer following its infection, one of two attempts to transmit the disease was successful. The animal which failed to become infected, 133, proved susceptible to infection later, although it developed only a mild type, suggesting that a certain small degree of resistance may have been conferred upon it by the injection of deer blood. In the case of the mule deer, both transmissions were successful, terminating in fatal cases of anaplasmosis.

Since these experiments prove that certain deer may be carriers and since deer in the wild state harbor a variety of ticks, some of which have been found to be vectors[1] of *Anaplasma,* the obvious conclusion is that deer may be a potential source of anaplasmosis.

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INHIBITION OF SELENIUM INJURY TO WHEAT PLANTS BY SULFUR

WHEAT plants grown in soil to which selenium in the form of sodium selenate is added at the rate of 15 p.p.m., or even less under some conditions, become characteristically chlorotic. The young leaf blades are often almost snow white, with green tips and midveins. A suggestion by Dr. J. E. McMurtrey that the chlorosis might be a symptom of sulfur deficiency (inferable from the view that absorbed selenium replaces sulfur in organic compounds of the plants[1]), and that decreasing the available sulfur might therefore accentuate the injury, led to the discovery of a striking effect of sulfur on the toxicity of selenium to wheat.

In both sand and solution cultures the toxicity of a given amount of sodium selenate varied according to the relative amount of sulfate in the nutrient solution. Where there was no sulfate present the plants died in the early seedling stage; where its concentration was high compared with that of the selenate the plants developed normally without visible injury. It was obvious that the death of the plants in the no-sulfur cultures and the injury of those with moderate amounts of sulfur were due to the presence of selenium rather than to a deficiency of sulfur, for the only effect of sulfur deficiency, as shown by the controls without selenium, was a paler green color. Evidently, these controls had received some extraneous sulfur, possibly from fumigants used in the greenhouse, or from impurities in the chemicals, which were of c. p. grade but not specially purified. The pH values of the nutrient solutions were not correlated with their toxicity.

Plants grown in water cultures with various selenium concentrations up to 28 p.p.m., the highest tried, were uninjured where the proportion of selenium to sulfur was 1:12 or less. Some of the leaves were chlorotic wherever the ratio was as high as 1:8, the injury being progressively more pronounced as the ratio increased. Where it was as high as 1:2 growth was largely inhibited. It is not assumed that these ratios are constant for wheat under all conditions although they have shown a surprising reproducibility under the conditions of these experiments.

Elemental sulfur as well as ammonium sulfate completely inhibited visible injury to wheat plants in soils to which sodium selenate was added. This inhibition of symptoms suggests that the entrance of selenium into plants and the consequent toxicity of such plants for animals[2] may be conditioned by the amount of available sulfur in the soil.

A paper presenting the evidence for the foregoing statements is being submitted to the *Journal of Agricultural Research.*

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