C. over night and about 2 gm of a crystalline protein in the form of plates appeared. The activity of this preparation is about one third that of the previously described crystalline trypsin with respect to the digestion of hemoglobin or casein. It is much less active than trypsin in liquefaction of gelatin but much more active in clotting milk. It does not clot blood and contains no amylase or lipase activity. The digestion of casein is carried much further than by the crystalline trypsin. The enzyme is evidently quite distinct from the trypsin previously isolated and may represent the "pancreatic rennet" of Vernon.

The chymo-trypsinogen has been recrystallized ten times and shows constant optical activity and constant proteolytic activity after activation by trypsin. Some samples showed a very slight proteolytic activity without activation which was equivalent to about 1/5000 of that of the activated material. This trace of activity is variable and is probably due to the presence of a small amount of active material.

The chymo-trypsin has been recrystallized three times and all fractions show constant optical activity and constant proteolytic activity as measured by digestion of hemoglobin, casein or gelatin, or rennet action.

There is reason to believe, therefore, that these preparations represent pure proteins, and that the proteolytic activity is a property of the protein molecule.

M. KUNITZ
JOHN H. NORTHROP
LABORATORIES OF THE ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH
PRINCETON, N. J.

DEER AS CARRIERS OF ANAPLASMOSIS

THE possibility that deer might serve as a reservoir for anaplasmosis has been considered for some time. Through the assistance of Mr. Ronald P. Harville, the Division of Fish and Game furnished two deer—one, a southern black-tailed buck, *Odocoileus columbianus scaphiotus;* the other, a Rocky Mountain mule deer, *Odocoileus hemionus hemionus* (Raf.). Both these animals were obtained in areas from which no anaplasmosis has been reported in cattle.

The southern black-tailed deer, number 1, was brought to the laboratory in a very weak condition. It was heavily infested with ticks, of which a large number were removed and identified as *Dermacentor occidentalis, Dermacentor parumapertus* and *Ixodes ricinus californicus.* After removal of the ticks, the animal speedily recovered, but a microscopic examination of this deer's blood made some time afterwards revealed a few bodies resembling *Anaplasma.* To prove the animal a carrier, 5 cubic centimeters of its blood were injected into calf 2642 on December 6, 1932. No symptoms, blood changes nor marginal bodies were observed in the calf. Later, it received blood containing *Anaplasma* from carrier cow 454, and promptly developed the disease, showing mild symptoms and blood changes. Deer 1, therefore, was not proved to be a carrier at this time.

Immediately after this deer was bled for the injection of calf 2642 on December 6, it received 10 cubic centimeters of blood from cow 265, which later succumbed to anaplasmosis. Deer 1 never developed any symptoms or blood changes characteristic of the disease.

On March 22, 1933, 13 cubic centimeters of blood were taken from this deer and injected into bull 133. This animal exhibited none of the symptoms or blood changes observed in anaplasmosis. Later, it was infected with blood from carrier cow 2603 and developed a mild case.

On July 1, 1933, 20 cubic centimeters of blood were taken from deer 1 and inoculated into cow 842. This cow presented symptoms and blood changes typical of anaplasmosis and eventually recovered.

In the blood of the mule deer, number 2, no *Anaplasma* were observed; consequently, it was assumed that this animal was not a carrier. On April 15, 1933, 20 cubic centimeters of blood were injected into this deer from cow 2607, which died of the disease shortly afterwards. As a control, cow 2603 received 10 cubic centimeters of blood from 2607 at the same time. On May 3, 1933, numerous marginal bodies were observed in the blood of control cow 2603. The infection ran a severe course, but the cow recovered. Deer 2 never developed symptoms of anaplasmosis, and, of several blood examinations, no marginal bodies were noted except in one instance and they were too few to be regarded as other than suspicious.

To determine whether deer 2 would transmit the infection, blood was taken from it on May 24, 1933, and injected into cow 2615. This cow exhibited a typical blood picture; numerous marginal bodies were observed, the red corpuscle count dropped from 6,160,000 to 1,440,000 and other changes, such as anisocytosis and punctate basophilia, were noted. The symptoms were characteristic: constipation, rapid pulse and respiration, and progressive weakness, terminating in death on June 28, 1933. The autopsy showed the usual lesions: icterus in the subcutaneous tissue and peritoneum; pale, viscous blood; petechial hemorrhages on the epicardium; markedly enlarged and jam-like spleen; slightly icteric liver; a gall bladder distended with thick, dark-green bile; and mucus-covered fecal pellets in the colon. From all these findings, therefore, the diagnosis of anaplasmosis is conclusive.

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.

WILLIAM HUTCHINS BOYNTON
GLADYS M. WOODS
DIVISION OF VETERINARY SCIENCE
UNIVERSITY OF CALIFORNIA

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.*

ANNIE M. HURD-KARRER
BUREAU OF PLANT INDUSTRY
DEPARTMENT OF AGRICULTURE

1 Helm, "Beitrag zum Anaplasmen Problem," *Zeit. Infektionskr.*, 25: 199, 1924.

1 C. A. Cameron, "Preliminary Note on the Absorption of Selenium by Plants," *Sci. Proc. Roy. Dub. Soc.*, 2 (n. s.): 231–233, 1880.

2 E. M. Nelson, A. M. Hurd-Karrer and W. O. Robinson, "Selenium as an Insecticide," SCIENCE, 78: 124, 1933.

BOOKS RECEIVED

DEBYE, P. *Struktur der Materie.* Pp. 50. 21 figures. S. Hirzel, Leipzig. RM. 3.

ELLWOOD, CHARLES A. *Methods in Sociology; A Critical Study.* Pp. xxxiv + 214. Duke University Press. $1.50.

GOUGH, HERBERT J. *Crystalline Structure in Relation to Failure of Metals—Especially by Fatigue.* Edgar Marburg Lecture. Pp. 111. 53 figures. American Society for Testing Materials. $1.00.

GUDGER, EUGENE W., Editor. *Archaic Fishes: Article V, The Natural History of the Frilled Shark.* Pp. 74. 31 figures, 5 plates. American Museum of Natural History.

HOGBEN, LANCELOT. *Nature and Nurture.* Pp. 143. Norton. $2.75.

JAEGER, EDMUND C. *The California Deserts.* Pp. x + 207. Illustrated. Stanford University Press. $2.00.