geographical work. The publications also include a technical series and reproductions of maps and charts of historical interest.

The society has always been concerned in advancing British cartography by maintaining a high standard of draughtsmanship and lettering and by employing modern methods of reproduction for the maps accompanying papers in the *Geographical Journal,* and special maps published from time to time. The map of Europe and the Middle East recently prepared for the British Council embodies several developments in this field. The advice of the society is at the disposal of official departments when requested, and has been formally submitted, *e.g.,* as evidence to a committee considering the future of the Ordnance Survey.

Similarly the society has contributed much to raise the level of geographical education and to secure the recognition of geography as an independent discipline at the universities. Its initiative resulted in the foundation of schools of geography at Oxford in 1899 and at Cambridge in 1903.

During the war the work of the society has naturally been curtailed in some directions, but despite damage to the house by enemy action its main features have been maintained. Many members have been able to apply their geographical experience and training in the national interest. The library and map collection have been constantly used by service and other government departments and by representatives of the Allied nations. At the society's meetings, subjects concerning post-war planning have been discussed from the geographical aspect.

The basis of the society's work thus lies in the association of those directly qualified for exploration, travel and research with the general body of members who are interested in the geographical background of countries and peoples, and who by the support of the society enable it to advance geographical knowledge. The Society can thus act as the recognized representative of all British geographers, serve as a repository of experience and information, and encourage adequate standards in the science.

G. R. CRONE,
Librarian

ROYAL GEOGRAPHICAL SOCIETY,
LONDON, ENGLAND

SPECIAL ARTICLES

RECOVERY OF EQUINE ENCEPHALOMYELITIS VIRUS (WESTERN TYPE) FROM CHICKEN MITES[1,2]

DURING the past fifteen years three immunologically distinct types of equine encephalomyelitis virus have been discovered, and the importance of these viruses as a cause of disease in man is now well established.[3, 4, 5] An epidemic of encephalitis occurred among horses in Texas during the summer and fall of 1944, together with at least three human cases due to the Western equine virus.[6]

A recent article by Smith, Blattner and Heys[7] concerning the isolation of the St. Louis encephalitis virus from chicken mites prompted us to conduct epidemiological investigations. The similarity in the mode of transmission of equine encephalomyelitis and St. Louis encephalitis is evidenced by the fact that neutralizing antibodies against both viruses have been demonstrated in the serum of fowl. Only recently Hammon and his associates[8] demonstrated neutralizing antibodies against equine encephalomyelitis virus in the serum of chickens in the Southwest. In correlated field and laboratory studies made in the State of Washington, antibodies to both St. Louis and Western equine viruses were found in as high as 50 per cent. of apparently healthy domestic fowl. It seemed likely, as pointed out by Smith and her associates,[7] that some blood-sucking vector which does not necessarily bite man was transmitting the disease to fowl. Furthermore, evidence is conclusive that reservoirs of equine virus exist in wild and domestic animals (including birds), and at least two blood-sucking insects have been found in nature harboring the virus.[9, 10]

With the cooperation of two local veterinarians, a survey was made in Dallas County, Texas, to locate farms and ranches where known cases of equine encephalomyelitis had occurred. With this information available, chicken mites (*Dermanyssus gallinae*), fowl ticks (*Argus persicus*) and sera from barnyard fowl were collected from nine of these ranches and

[1] Preliminary report.

[2] These studies are being supported by a grant from the Rose Lampert Graff Foundation, Los Angeles, Calif.

[3] J. P. Leake, *Pub. Health Rep.*, 56: 1902, 1941; F. W. Jackson and others, *Can. Pub. Health Jour.*, 33: 241, 1942.

[4] R. Feemster, *Am. Jour. Pub. Health*, 26: 1403, 1938; L. D. Fothergill, J. H. Dingle, S. Farber and M. L. Connerley, *New England Jour. Med.*, 219: 411, 1938.

[5] J. Casals, E. C. Curnen and L. Thomas, *Jour. Exp. Med.*, 77: 521, 1943; E. H. Lennette and H. Koprowski, *Jour. Am. Med. Asn.*, 123: 1088, 1943.

[6] S. E. Sulkin, to be published.

[7] M. G. Smith, R. J. Blattner and F. M. Heys, SCIENCE, 100: 362, 1944.

[8] W. McD. Hammon, W. C. Reeves and J. V. Irons, *Texas Rep. Biol. and Med.*, 2: 366, 1944.

[9] W. McD. Hammon, W. C. Reeves, B. Brookman and E. M. Izumi, SCIENCE, 94: 328, 1941.

[10] C. H. Kitselman and A. W. Grundmann, *Kans. Agric. Exper. Sta., Tech. Bull.*, No. 50, p. 1, 1940.

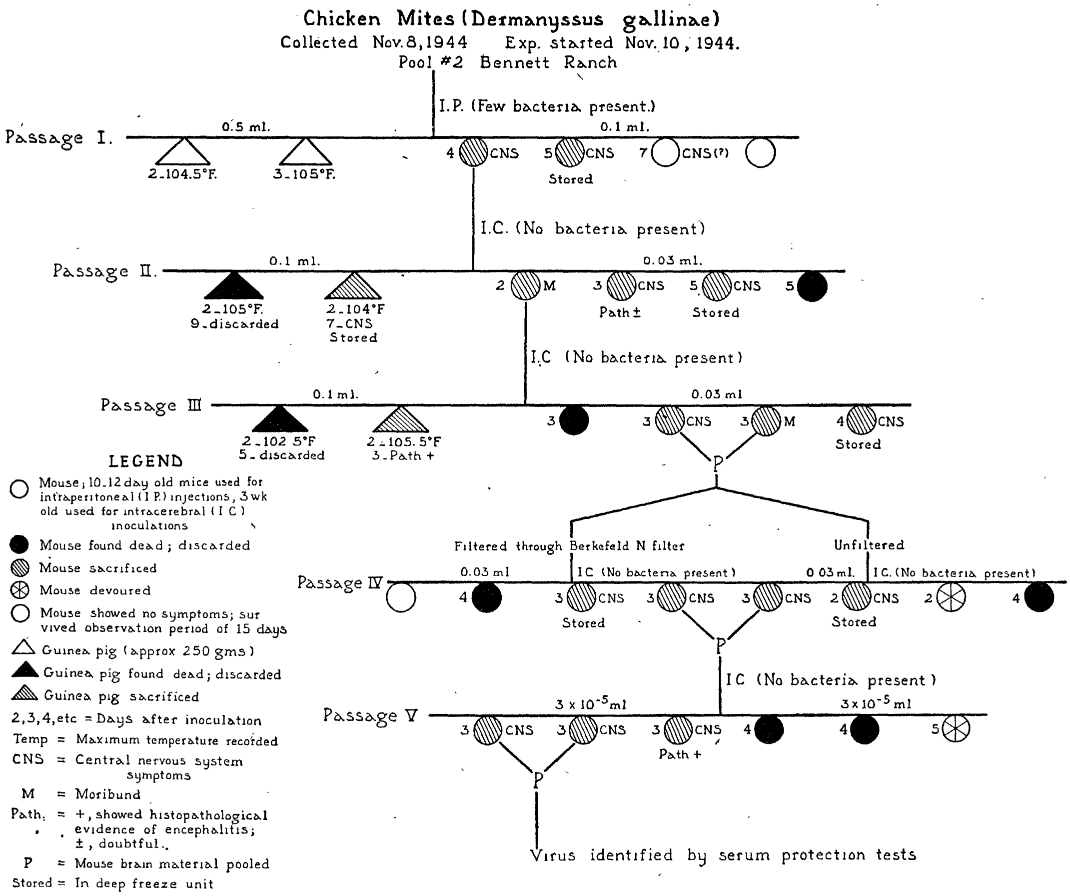

FIG. 1

farms. The mites and ticks were kept in test-tubes for several days without feeding. The results of a successful experiment in which the Western equine virus was demonstrated in chicken mites are summarized in Fig. 1. Chicken mites were collected from the Bennett ranch on November 8, 1944, and two days later these experiments were started. A pool of approximately 100 mites was triturated in broth in an agate mortar, centrifugalized at 5,000 r.p.m. for ten minutes in an angle centrifuge, and the supernatant fluid, which was cultured aerobically and anaerobically, was inoculated intraperitoneally into each of four 10- to 12-day-old Swiss mice and two 250-grain guinea pigs. Both guinea pigs showed a temperature rise on the second and third day, respectively. Two of the four mice exhibited neurological symptoms on the fourth and fifth days, respectively, after the inoculation, and the brain from one was used for subsequent intracerebral inoculation into other animals. With each passage aerobic and anaerobic cultures were made and brain tissue from animals showing central nervous system symptoms were examined for histopathological evidence of encephalitis.[11] After the third passage, filtration experiments were conducted with the pooled brains of mice showing symptoms. Identification of the filtrable agent of the fifth mouse passage as the Western type of equine encephalomyelitis virus was made by neutralization tests with serum of rabbits immunized to known strains of Western and Eastern equine virus and St. Louis encephalitis virus. The filtrable virus recovered from the chicken mites was neutralized by the Western equine immune serum but was not affected by the serum of rabbits immunized to the Eastern strain of equine virus or the virus of St. Louis encephalitis. Similar experiments with chicken mites collected from the Bennett ranch were conducted after the mites were kept in test-tubes for nine days without feeding. Again virus could be recovered suggesting that the agent was present within the bodies of these insects. Three of seven serum samples from chickens on the Bennett ranch contained neutralizing antibodies for the Western equine virus. Experiments with the fowl ticks collected from the same ranch were negative.

All attempts to isolate virus from specimens collected at the remaining eight farms were negative. Attempts to demonstrate virus in chicken mites or

[11] The author is indebted to Dr. Charles Ashworth for examining these tissues.

fowl ticks collected at a farm where a proved fatal case of Western equine encephalitis in a child had occurred[6] were likewise negative. The above procedures were repeated in each case. Tests for neutralizing antibodies in the serum of the chickens on the various farms have not been completed.

These studies are being extended and will be repeated during the summer and fall months when the common chicken mite is more prevalent. Meanwhile, studies are in progress to determine whether, under experimental conditions, hereditary transmission of the Western equine virus in chicken mites can be effected.[12]

SUMMARY

The Western type of equine encephalomyelitis virus has been isolated from chicken mites (*Dermanyssus gallinae*) in nature during an outbreak of the equine disease in the Southwest.

S. EDWARD SULKIN[13]

DEPARTMENT OF BACTERIOLOGY AND IMMUNOLOGY,
SOUTHWESTERN MEDICAL COLLEGE,
DALLAS, TEXAS

THE ANTIBACTERIAL PROPERTIES OF DICUMAROL

DESPITE the considerable amount of work done on dicumarol, it has not been recognized up to now that this compound has marked antibacterial properties. The growth inhibitory effect of dicumarol was studied against fourteen bacterial species and the results are represented in Table 1.

TABLE 1

	Growth inhibitory concentrations of dicumarol
Staphylococcus aureus	1 : 100,000
Streptococcus pyogenes	1 : 100,000
Streptococcus viridans	1 : 100,000
Streptococcus fecalis	Not inhibited at 1 : 25,000
Bacillus anthracis	1 : 100,000
Bacillus subtilis	Not inhibited at 1 : 25,000
Corynebacterium diphtheriae	1 : 25,000
Clostridium welchii	Not inhibited at 1 : 25,000
Brucella abortus	1 : 25,000
Eberthella typhosa	Not inhibited at 1 : 25,000
Escherichia coli	Not inhibited at 1 : 25,000
Salmonella paratyphi A	Not inhibited at 1 : 25,000
Proteus vulgaris	Not inhibited at 1 : 25,000
Pseudomonas pyocyanea	Not inhibited at 1 : 25,000

Since naphthoquinones having Vitamin K activity are the physiological antagonists of dicumarol, it was of interest to determine whether naphthoquinones would antagonize the antibacterial properties of this compound. This possibility was tested in the case of *Staphylococcus aureus*. It was found that methyl-1,-4-naphthoquinone failed to antagonize the growth inhibition caused by dicumarol.

[12] S. E. Sulkin and C. L. Wisseman, Jr., to be published.

[13] With the technical assistance of George C. Patterson.

Dicumarol was first isolated by Link and associates[1] from spoiled sweet clover. One may be allowed to speculate on the possibility that if it could be shown that the spoilage of sweet clover was due to the action of microorganisms, dicumarol could be considered a naturally occurring antibiotic.

ANDRES GOTH

SOUTHWESTERN MEDICAL COLLEGE,
DALLAS, TEXAS

THE MECHANISM OF GROWTH INHIBITION BY HEXENOLACTONE

A POTENT growth-inhibitor, supposedly parasorbic acid, occurs in a variety of natural sources, notably malt, yeast, orange peels[1] and the ripe berries of the mountain ash, Sorbus.[2] The inhibitor suppresses the germination of seeds and pollen as well as the growth of certain microorganisms and animal tissues.[1,3] Among the bacteria are some (lactic acid bacteria, *Streptobacterium plantarum*) which, like animal epithelia (chick epithelia, Ehrlich carcinoma), are relatively unresponsive; and others (*Staphylococcus aureus*) which, like fibroblasts and mesenchymal cells, are very sensitive to the inhibitor.[1,3,4] Kuhn and Jerchel[2] have recently identified parasorbic acid (I) in extracts of Sorbus berries and established proof for its structural configuration. Synthesis was reported independently by two groups of investigators.[1,2]

In the form of a relatively simple and comparatively stable molecule, *i.e.*, an unsaturated delta-hexenolactone, a potential tool is thus provided for the elucidation of the mechanism of an antibiotic activity.

Because of structural resemblance between hexenolactone (parasorbic acid) and pantolactone, which reacts with beta-alanine to form pantothenic acid, Medawar, Robinson and Robinson[1] suggested a possible interference of the inhibitor with pantothenic acid metabolism. In experiments designed to test this hypothesis[3,5] pantothenic acid did not weaken the activity of hexenolactone. On the other hand, both alpha-alanine and beta-alanine as well as glutathione were shown by Hauschka[5] to inactivate the inhibitor, while glycine, iso-leucine and glutamic acid had no such effect. Further experiments, to be summarized here, have established cysteine, but not cystine, as antagonistic to hexenolactone. This antagonism was demonstrated by bio-assay and its mechanism elucidated in part by colorimetric and spectrophotometric methods.

[1] M. S. Stahmann, C. F. Huebner and K. P. Link, *Jour. Biol. Chem.*, 138: 513, 1941.

[1] Medawar, Robinson and Robinson, *Nature*, 151: 195, 1943.

[2] Kuhn and Jerchel, *Ber. Chem. Ges.*, 76B: 413, 1943.

[3] Kuhn, Jerchel, Moewus, Moller and Lettre, *Naturwiss.*, 31: 468, 1943.

[4] De Lor and Means, *Rev. Gastroenterol.*, 11: 319, 1944.

[5] Hauschka, *Nature*, 154: 769, 1944.