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.

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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.

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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.