multiplication takes place. After several weeks a swarm of fork-tailed *cercariae* emerge from the snail, swim about in the water and in case mammalian species enter this "infected water," the cercariae adhere to the skin of the mammal, drop their tails, penetrate through the skin and by way of the afferent and systemic circulation eventually reach the portal system, where they develop into male and female worms.

This infection, which is relatively common in parts of Africa, particularly in the Nile Valley, was brought to the New World with the importation of slaves and became established in Brazil, Venezuela and the Guianas. It has also been reported from Peru, Panama and Costa Rica, although it has probably not become established in the latter two countries. It is known to be present in the Lesser Antilles, especially in Antigua, Guadeloupe, St. Kitts, Martinique and Barbadoes. It also occurs in Puerto Rico, where it was first reported by Gonzalez-Martinez in 1904, three years before the species was differentiated from *Schistosoma haematobium* (the vesical blood-fluke), with which it is frequently coextensive in Africa. No autochthonous case of *S. mansoni* infection is known for North America.

In view of the limited size of the Island of Puerto Rico, and because of the economic importance of the disease on the island, a unique opportunity is afforded for study of the infection.

Important preliminary studies on the epidemiology, biology, pathology and clinical aspects of the disease have been made by members of the staff of the School of Tropical Medicine at San Juan. The present investigation will be confined to the following program:

BIOLOGICAL ASPECTS

(1) Ways by which the viable eggs reach the waterways where the intermediate host lives.

(2) Hatching phenomena and infection of the snail. (An abundance of viable eggs is needed to determine experimentally if species of *Planorbis* in the United States can be readily infected).

(3) Length of time required for complete development in the appropriate snail, and period of discharge of viable cercariae from the snail.

(4) Route of migration of the young worm through the human body, once it has penetrated through the skin.

(5) Number of eggs in the uterus of the female worm.

(6) Method of deposition of eggs in the gut wall. Do eggs migrate in the tissues?

PATHOLOGICAL AND CLINICAL ASPECTS

(1) Does schistosomiasis splenomegaly constitute an important clinical entity in Puerto Rico?

(2) Can splenomegaly and hepatic cirrhosis develop in experimental animals harboring only one sex of the worm (male or female)?

(3) Types of ulceration and papillomata developing in experimental animals (monkeys, rabbits, etc.).

(4) The reliability of precipitin and other serological tests as a means of diagnosis.

(5) Factors determining selection of mesenteric and rectal veins by worms.

EPIDEMIOLOGICAL ASPECTS

(1) How and in what locations do the people of Puerto Rico expose themselves to infections?

(2) Natural reservoir hosts of the infection in Puerto Rico. The types of canals and ponds where the snails abound and afford an opportunity for carrying on the life cycle.

(3) Determine the best method or methods for attacking the problem of prevention in Puerto Rico.

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TRANSMISSION OF INFECTIOUS EQUINE ENCEPHALOMYELITIS IN MAMMALS AND BIRDS

IN 1931, Meyer, Haring and Howitt[1] reported the recovery of a filtrable virus from the brain of a horse suffering from an acute form of encephalomyelitis which had been particularly prevalent among horses and mules of rural sections of California during the years 1930 and 1931. They have conducted extensive experiments with this and subsequently recovered strains of the virus and reported horses, mules, guinea pigs, monkeys, white rats, white mice and rabbits[2] as being susceptible to the infection. Guinea pigs have been found to be admirably adapted to the investigation of the experimental disease.

In 1932 the writers recovered a strain of encephalomyelitis virus from the brain of a field case which occurred in South Dakota where the disease was epizootic. In the course of experimental work with this virus, calves (2), sheep (2), dogs (4) and cats (2) have been tested for evidence of susceptibility to intracerebral inoculation. In these preliminary trials, the three last named species have been found to be definitely refractory.

The inoculated calves developed a febrile reaction which persisted for several days. Five days following inoculation of a 0.5 cc dose of encephalomyelitic guinea pig brain emulsified 1 part in 10 parts (approximate) of physiological saline, both animals displayed anorexia, difficult swallowing, stupor, incoordination, grinding of the teeth, localized myopalmus and photophobia. The intensity of these symptoms

[1] K. F. Meyer, C. M. Haring, B. Howitt, "Etiology of Epizootic Encephalomyelitis of Horses in the San Joaquin Valley," SCIENCE, n. s., 74: 227–8, 1930.

[2] K. F. Meyer, C. M. Haring, B. Howitt, "Newer Knowledge of the Neurotropic Virus Infections of the Horse," *J. A. V. M. A.*, n. s., 32: 3, 1931.

in each case varied during the succeeding days and the general attitude varied from extreme lassitude and drowsiness on the one hand to hyperesthesia and clonic spasms on the other. The height of the clinical reaction occurred on the 7th to 9th days, retrogressing to normalcy on the 14th day with the animals remaining normal on the 20th day.

Using a very active sample of the same virus, White Leghorn cockerels (3) about 4 months of age and mature common pigeons (4) were inoculated intracerebrally while under light chloroform anesthesia. A 0.2 cc dose of approximately a 1/50 dilution of virulent guinea pig brain was used. The cockerels have remained normal for 20 days.

The four pigeons so inoculated developed general weakness, ataxia and marked tremors on the third day; dying on the third to fourth day after inoculation. The brains of these pigeons produced the typical disease in guinea pigs. Pigeons receiving normal guinea pig brain were in no way affected by the inoculation.

These inoculations, though too limited for final conclusions, indicate that calves are not entirely refractory to encephalomyelitis. A 100 per cent. "take" in the case of pigeons indicates the possible use of these birds in routine examination of tissues from field cases. The possible relationship of the calf and the pigeon to the epizootiology of the natural disease is worthy of consideration.

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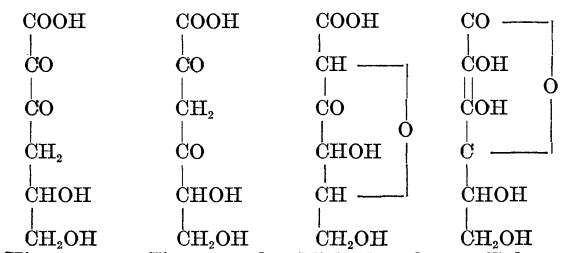
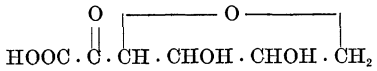
PRELIMINARY NOTE ON THE STRUCTURE OF ASCORBIC ACID (VITAMIN C)

THE following formulae have been advanced to express the structure of ascorbic acid:



Hirst et al I. Karrer et al II. Micheel et al III. v. Euler IV.

And a fifth possible one may be suggested:



V.

It should be possible to choose the correct expression for the structure of ascorbic acid on the basis of the results of hydrogenation. Structures I and II require four atoms of hydrogen for complete reduction, and the rest only two atoms. The hydrogenated products thus obtained should differ in the number of hydroxyl groups, I and II having four, III and V three, and IV five.

Structures I and II may be differentiated by the ability of the reduction product of II to form a γ lactone, while that of I can not. Likewise, the reduction products of III and V may be differentiated by the ability of that of V to form a lactone, whereas that of III should not, inasmuch as the known 2, 5-anhydrohexonic acids do not form lactones.

On the basis of these considerations, ascorbic acid was exhaustively hydrogenated. Only two atoms of hydrogen were absorbed, and the resulting acid formed a stable lactone. In order to ascertain the number of hydroxyl groups, the product was acetylated, but unfortunately, the acetyl derivative could not be crystallized. The analysis of the amorphous product did not permit a definite conclusion as to the number of hydroxyl groups, inasmuch as the various possible acetyl derivatives do not present sufficiently striking differences in elementary composition. It is hoped, however, that methylation of the hydrogenated ascorbic acid will afford accurate information as to the number of hydroxyl groups present. The experiments will be performed as soon as the necessary material is available.

The present indications would seem to favor IV or V as the probable structure of the ascorbic acid.

The material for the present experiments was generously placed at our disposal by Professor Szent-Györgyi, to whom we wish to express our great indebtedness.

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