was a member of the Royal Society of London, and all his scientific work was done and published there. The author cites the sixth edition (1873) of the "Origin of Species" as the place of acknowledgment by Darwin of the priority of Wells's publication of the concept of natural selection, whereas Darwin first published this in the fourth edition (1866, p. xiv). Wells was not only a pioneer in evolution and physics (of dew) but also in ophthalmology (theory of vision) and epidemiology.

Scientists are peculiarly subject to environmental influences. Their best work has been done under the aegis of intellectual freedom. Wells spent his energies in Carolina in political turmoil. Fortunately, science knows no race, nationality nor region.

CHARLES A. KOFOID

SPECIAL ARTICLES

YELLOW FEVER VIRUS IN JUNGLE MOSQUITOES[1]

UNTIL recent years yellow fever was regarded as a house disease, transmitted solely by the domestic mosquito, *Aedes* (*Stegomyia*) *aegypti* (Linnaeus). The existence of a special epidemiological type of the disease, now known as jungle yellow fever, became evident when it broke out under rural and jungle conditions in the Valle do Chanaan, Espirito Santo, Brazil, in 1932. During the course of the epidemic no trace of the classical vector could be found in the infected area.[2]

Since then over 20 similar outbreaks have been observed in various parts of South America (Colombia, Peru, Bolivia, Paraguay and most of the Brazilian states.)[3] Study has shown that during such outbreaks man is generally infected only while in contact with the forest or jungle. Household infections are not common, except where the house stands within the jungle.

It has been shown experimentally that a number of Brazilian mosquitoes other than *Aedes aegypti* can become infected under laboratory conditions. Successful transmission by bite, however, has been obtained only with three species: *Aedes scapularis* (Rondani), *Aedes fluviatilis* (Lutz) and *Haemagogus capricorni* (Lutz).[4,5,6,7,8]

The 1938 outbreak of jungle yellow fever in the state of Rio de Janeiro, Brazil, afforded an opportunity to demonstrate the presence of yellow fever virus in mosquitoes caught in the jungle. Mosquitoes collected alive at points near where human infection had occurred were forwarded daily to the laboratory in Rio de Janeiro. They were first classified by species or groups of species and then allowed to bite non-immune rhesus monkeys.[9] After feeding on the monkeys the insects were tested for the presence of yellow fever virus by a method previously described[11] but briefly summarized as follows: They were killed, ground finely, suspended in a diluent and centrifuged, and the supernatant fluid was injected into mice intracerebrally. This report is based on results obtained with 24,304 mosquitoes sent in from the state of Rio de Janeiro over a period of eleven weeks, the following species being represented:

LIST OF WILD-CAUGHT MOSQUITOES TESTED FOR THE PRESENCE OF YELLOW FEVER VIRUS

Group	Species	Used	Fed	Remarks
1	*Aedes scapularis* (Rondani)	180	100	
2	*A. leucocelaenus* (Dyar and Shannon)	4,671	2,270	Positive by bite
3	*Haemagogus capricorni* (Lutz)	1,216	646	Positive by bite
4	*Psorophora ferox*	253	143	
	P. albipes	15	7	
	A. fulvus (Wiedemann)	8	5	
	A. serratus (Theobald)	1,570	545	
	A. terrens (Walker)	503	58	
5	*Saebethes*, 3 species	1,092	369	
6	All other sabethines (*Sabethoides, Limatus, Wyeomyia, Goeldia* and *Trichoprosopon* (*Joblotia*), totalling about 20 species	14,796	3,443	Positive by injection

Positive results were obtained from the following: (1) *Aedes leucocelaenus* (D. and S.). On February 22, Monkey 5, on which a total of 16 *A. leucocelaenus*

[1] From the Cooperative Yellow Fever Service of the Ministry of Education and Health of Brazil and the International Health Division of The Rockefeller Foundation, Rio de Janeiro.

[2] F. L. Soper, H. A. Penna, E. Cardoso, J. Serafim, Jr., M. Frobisher, Jr., and J. Pinheiro, *Am. Jour. Hyg.*, 18: 555–587, 1933.

[3] F. L. Soper, *Quart. Bull. Health Org., League of Nations*, 5: 1–50, 1936.

[4] We are indebted to Dr. P. C. A. Antunes for the recent information that *Haemagogus janthinomys* (Dyar) 1921 is a homonym of *H. capricorni* (Lutz) 1904.

[5] N. C. Davis and R. C. Shannon, *Jour. Expt. Med.*, 1: 803–808, 1929.

[6] N. C. Davis and R. C. Shannon, *Am. Jour. Trop. Med.*, 11: 21–29, 1931.

[7] L. Whitman and P. C. A. Antunes, *Am. Jour. Trop. Med.*, 17: 803–823, 1937.

[8] L. Whitman and P. C. A. Antunes, *Am. Jour. Trop. Med.*, 17: 825–831, 1937.

[9] The methods used for collecting, shipping, classifying and feeding will be dealt with in a subsequent paper.[10]

[10] R. C. Shannon, "Methods for Collecting and Feeding Mosquitoes in Jungle Yellow Fever Studies." *In preparation.*

[11] L. Whitman, *Jour. Exp. Med.*, 66: 133–143, 1937.

from Affonso Arinhos had fed on February 7, 9, 10, 11 and 14, was found prostrate in its cage with a subnormal temperature and was sacrificed. This animal showed fever on the 17th, 18th, 19th and 21st, but was not bled because of its previous irregular temperature. Microscopic examination of the liver showed typical lesions of yellow fever, including inclusion bodies, and the blood drawn on the day of death gave positive mouse-protection test results. While it was impossible to fix the date of infection of Monkey 5, experience with other animals suggested that infection probably occurred on the 14th, when only one specimen of *A. leucocelaenus* fed.

(2) *Haemagogus capricorni* (Lutz). Between February 7 and 11, six *H. capricorni,* also collected at Affonso Arinhos, were allowed to feed upon Monkey 4. Between February 24 and 26, twelve additional insects of the same species, collected near Bemposta and Pedro do Rio, were allowed to bite the same animal. The monkey first showed fever on February 28 and died three days later. Autopsy findings followed by microscopic examination of the liver indicated that the animal died of yellow fever. Blood drawn on the first day of fever caused typical yellow fever encephalitis in mice. Blood drawn shortly before death produced fatal encephalitis in one of six mice, and also gave positive mouse-protection test results.

It is believed that Monkey 4 became infected from a lot of four mosquitoes which fed on it on February 24. An emulsion of these mosquitoes injected into mice caused typical yellow fever encephalitis. Monkey 3 was inoculated with second-passage mouse-brain material, with the result that virus was demonstrated in the blood stream and specific yellow fever antibodies were afterward found to be present in the serum.

The supernatant fluid from an emulsion of 118 *H. capricorni,* collected in the same infected areas of Bemposta and Pedro do Rio on March 10, produced encephalitis in mice. Transfer of infective brain material resulted in infection of Monkey 6, with demonstrable circulating virus, and the production of specific immunity as shown by the mouse-protection test. Fifty-eight of the mosquitoes included in this emulsion had fed on Monkey 8 without infecting it.

(3) *Sabethine Mosquitoes.* Owing to the numerous species in the sabethine group and the difficulty of naked-eye classification, it proved impractical during this preliminary study to separate the sabethines by species previous to feeding and injection.

Twenty-one of 88 specimens in a mixed group of *Sabethoides, Limatus, Wyeomyia, Goeldia* and *Trichoprosopon,* caught together with the first lot of infected *Haemagogus* at Bemposta and Pedro do Rio, February 24, fed on Monkey 9. This monkey developed no evidence of infection or immunity. The inoculation of the supernatant fluid from an emulsion of the 88 mosquitoes, however, produced encephalitis in mice. Second mouse-passage material produced infection in Monkey 4A with circulating virus and the production of specific immunity.

These results show that one or more of the 88 specimens in this group had fed on an infected person or animal before capture. The negative results in Monkey 9 may have been due to failure of infected mosquitoes to feed, to the non-completion of the incubation period of the virus in the mosquito or possibly to inability of the infected species to transmit, even though capable of conserving the virus for long periods of time.

The evidence presented incriminates two species of forest-inhabiting mosquitoes, *Aedes leucocelaenus* (D. and S.) and *Haemagogus capricorni* (Lutz), as natural vectors of yellow fever, and indicates that one or more species of sabethine mosquitoes may harbor the virus of the disease without definitely implicating them in actual transmission.

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THE TREATMENT OF SPONTANEOUS TUMORS IN DOGS BY THE INJECTION OF HEPTYL ALDEHYDE[1]

FOLLOWING the observation that the addition of heptyl aldehyde to the otherwise normal diet of mice harboring spontaneous carcinomas of the mammary gland brings about liquefaction and certain retrogressive changes of the tumors without injury to the animal by such treatment,[2] it seemed logical to test out the possibility that dogs suffering from spontaneous tumors might respond in a similar manner. The amount of material needed, however, to bring about retrogressive changes in tumors in mice by the addition of the material to the diet would be entirely too great for practical purposes in dogs. Consequently, the subcutaneous injection of small amounts of heptyl aldehyde has been injected into dogs (from 0.10 to 1.00 cc at a time). Injection of the material into mice was found to be impractical for the reason that ulceration at the site of injection invariably occurred. Even though liquefaction of tumors in mice was brought about by the injection of heptyl aldehyde at remote spots, this local reaction interfered with subsequent injections and even with complete recovery of the mouse from a single injection. Due, perhaps, to the tougher skin of the dog, local sloughing of tissue is not pro-

[1] This experiment has been made possible by grants from the International Cancer Research Foundation, the Anna Fuller Fund and from the Jane Coffin Childs Fund. The dogs have been kept under normal outdoor conditions in the Whitney kennels at Orange, Connecticut.

[2] L. C. Strong, SCIENCE, 87: 144, 1938.