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EPIDEMIOLOGICAL NOTES IN CONNECTION WITH THE 1954 OUTBREAK OF YELLOW FEVER IN TRINIDAD, B.W.I.[1]

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Yellow fever was well known in Trinidad during the eighteenth and nineteenth centuries. The last urban outbreak occurred in Port-of-Spain in 1907 (Trinidad and Tobago, 1908). In 1914, the last reported cases of yellow fever for the island were reported among American oil workers at La Brea in southern Trinidad (Trinidad and Tobago, 1915). This group of people lived in screened bungalows in a zone where mosquito control measures were in force, but worked in the jungle. In retrospect, it seems probable that this latter epidemic represented an outbreak of jungle yellow fever, not transmitted by *Aëdes aegypti.* These early yellow fever accounts are reprinted in the *Caribbean Medical Journal,* Vol. 16 (1955), pp. 60-86.

Trinidad provided some early clues which might have helped in the solution of the riddle of jungle yellow fever, but which were apparently submerged in the medical literature for twenty years and more.

Charles Kingsley (1871), in his book *At Last: A Christmas in the West Indies,* makes the statement (p. 98) that the monkeys in Trinidad died, possibly of yellow fever "the year before last, sensibly diminishing their numbers near the towns." The year in question is probably 1869, when a yellow fever outbreak is recorded for Trinidad.

Balfour (1914) mentions dead monkeys in connection with the 1913-1914 Trinidad yellow fever outbreak near Brighton and La Brea, and speculates upon their connection with the disease.

These are thought to be the earliest known items concerning the possible role of monkeys in yellow fever epidemiology, and both episodes originated in Trinidad.

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Yellow fever was not recorded from Trinidad in official reports in the interval between 1914 and 1954. Personnel of the Trinidad Regional Virus Laboratory took many blood specimens in 1953 and early 1954 from Trinidad residents. These specimens were tested by the Rockefeller Foundation Virus Laboratories in New York, in neutralization tests employing several viruses in a general exploratory program.

Yellow fever neutralization tests were thus performed on 694 sera. In addition to a high percentage of immunes in older people, eight yellow fever immunes were encountered in young people between 15 and 20 years of age from east-central Trinidad. This challenging finding was reported to health department authorities and further exploration was planned. One hundred and fifty additional tests were run on children between 5 and 15 years of age from the same region and no positives were encountered.

Plans for further investigations were abruptly interrupted in April, 1954 by the finding of a yellow fever case. The virus was isolated from the blood of a young male forest worker from Cumaca, in northeastern Trinidad. This man was seen for mild undiagnosed fever in the Arima District Hospital (Anderson, Spence, and Downs, 1954). Following this, no further cases were seen until early in August. In August, September, and early October, 15 additional human cases were seen. Included in these were four fatal cases, of which one was diagnosed by pathological examination of a liver specimen and the others by isolation of virus from liver tissue after death, as well as by histopathological examination (Anderson and Wattley, 1955). In each of the 12 confirmed non-fatal cases seen, virus was isolated from the blood. In one instance the isolation was made on the twelfth day of disease (Downs, Anderson, and Spence, 1955).

Additional human cases were seen, diagnosis being dependent upon serological evidence only. Many of these cases are still being studied serologically. While exact numbers cannot be stated, it is apparent that the epidemic was much more extensive than anyone realized at the time. Indeed, there is enough evidence on hand to indicate that there were hundreds of cases of unrecognized yellow fever in Trinidad during 1954.

Following the finding of a human case, it was possible to determine that monkeys were dying in the forests. During July, August and September, 1954, six recently dead howler monkeys (*Alouatta seniculus insulanus* Elliott) were brought to the laboratory by hunters. These monkeys had been picked up dead or dying in the forests. A seventh monkey was received in January, 1955.

Two of these animals were quite badly decomposed, yet yellow fever virus was isolated from the livers of all seven (Anderson and Downs, 1955). Available evidence suggests that there was an extensive epizoötic in 1954 among howler monkeys of the island, killing off large numbers. Hunters also furnished two stories of dead, white face or capuchin monkeys (*Cebus apella*

Linn). No histories of dead monkeys prior to January, 1954, were elicited in questioning over a hundred hunters and woodsmen, except for two stories of dead animals seen about 1933, some two decades earlier.

Neutralization tests were performed on the sera of 73 monkeys shot expressly for such testing from various forested regions of the island. Nineteen of 61 *Alouatta* sera showed yellow fever neutralizing properties and 7 of 12 *Cebus* sera showed similar evidence of immunity. Whether this immunity represents infection in the remote or recent past has not yet been determined.

Extensive collecting of forest mosquitoes was carried out at Cumaca, Melajo Forest, and Charuma Forest. Mosquito suspensions were inoculated intracerebrally into weaned white mice. Yellow fever virus was isolated from 22 different pools of *Haemagogus* mosquitoes captured in the Melajo Forest and Charuma Forest areas and once from a mixed pool of 17 species of mosquitoes. The infected mosquitoes were in collections made in August and September. Most of the infected *Haemagogus* were collected at ground level rather than in trees (Downs, Aitken, and Anderson, 1955).

Aëdes aegypti inspections were made in houses of confirmed yellow fever cases as well as in houses of undiagnosed fever cases. In several instances where actual captures of mosquitoes were made, no virus was isolated from these mosquitoes.

At least three of the Trinidad human cases from which virus was isolated appear on epidemiological grounds to be *Aëdes aegypti* transmitted, one of these being from urban Port-of-Spain. The remaining cases were probably all forest acquired. Nevertheless, the occurrence of *Aëdes aegypti* in suitable breeding containers and in houses, not only in urban areas but also scattered widespread throughout the island, with indices in rural houses exceeding 70 per cent, makes it possible that some of the cases seen in forest workers and their families in rural areas may have been *Aëdes aegypti* transmitted cases.

To summarize, between April and October, 1954, yellow fever virus was recovered 6 times from *Alouatta* monkeys, 23 times from mosquitoes, and 15 times from human beings. With an additional monkey isolation in January, 1955, this is a total of 45 isolations of virus. Each strain isolated has been confirmed in specificity tests using known monkey serum from yellow fever immune animals.

Dr. Anderson, the virologist of the laboratory staff, had had considerable previous experience with yellow fever in Colombia, but for Dr. Aitken, the entomologist, and for Dr. Spence and myself, epidemiologists, this represented our first direct contact with yellow fever. It was particularly in the field of the diagnosis of yellow fever that we encountered our greatest difficulties and were tripped up quite smartly several times. Since the diagnosis of yellow fever is the first problem encountered by the medical and public health pro-

fessions, either in combatting an existing epidemic or in guarding a vulnerable frontier against introduction of the disease, the details of a couple of our blunders may serve to highlight the problem of diagnosis.

The so-called classical cases of yellow fever, with jaundice, albuminuria, bloody vomit, collapse, and death should offer no difficulties in diagnosis. Lest we be misled into thinking this to be so obvious as to require no further mention, it may be emphasized that none of the first three deaths in the Trinidad epidemic was initially diagnosed as yellow fever (Wattley, 1955) although clinicians in the hospitals had been alerted and although all of these cases were reasonably close to the classical picture. The fourth case was recognized as a yellow fever case before death. (While Wattley states that all these cases were forest acquired, we feel that the fourth case (Ba) described in detail below was probably an *Aëdes aegypti* infected case, infected in the home.)

Difficulties compound, however, with the milder cases, when jaundice is not evident. In a region where dengue, typhoid fever, and malaria are frequently seen, a patient complaining of fever, plus headache, backache, and aching joints attracts but little attention if he is only mildly or moderately ill. Our Trinidad laboratory staff has seen some 600 cases of fever, mostly falling in the mild undiagnosed fever group, before, during, and after the yellow fever outbreak. I am as yet unable to distinguish the mild yellow fever case from other common febrile illnesses on clinical grounds alone.

Following are examples from our clinical and epidemiological records.

On July 27, one of our laboratory technicians went to Charuma Forest acting on a story of dead monkeys related by some oil workers. He returned with a monkey skull with flesh still adhering, and brought with him an oil worker who had become ill with temperature of 102.5 the same day. Serum from this man infected mice and the virus was later proved to be yellow fever. While this was still but a presumptive lead, it was decided to establish a mosquito collecting station in the same forest. The same laboratory technician asked if he could borrow a truck on Sunday, August 8, to revisit the forest with a couple of other technicians. Permission was granted, the boys took the trip as a holiday outing, and we thought no more of it. On August 13, five days later, the same technician said his seven-year-old son had become ill on August 12 with fever and loss of appetite. It developed that the technician, unauthorized, had taken his unvaccinated boy (S.B.) with him on the August 8 excursion.

The boy was brought in. Aside from temperature of 104°, he did not look seriously ill. A blood specimen was taken and the boy was referred to the Colonial Hospital as a yellow fever suspect. Here we made our blunder: we sent him without a covering letter. The busy doctor at the admitting desk of the Colonial Hospital took a brief look at the boy, diagnosed tonsillitis,

and sent him home. The boy recovered uneventfully. We recovered yellow fever virus from the blood specimen. When the case was notified to the Health Department, an exhaustive search was made for secondary cases in the Port-of-Spain suburb in which the child lived. Houses surrounding this potential focus were sprayed with insecticide. No secondary cases were seen.

In retrospective study of this case, the father said the boy never left the roadside in the forested area where the truck was parked. And from another angle, during August and September, yellow fever virus was recovered 13 times from pools of ground captured *Haemagogus spegazzinii* from this immediate vicinity.

In this instance, although yellow fever was suspected, the boy, circulating virus, was permitted to return to a potentially *Aëdes aegypti* infested locale. We may have suspected yellow fever, but we really did not think it was a case. Because we lacked conviction, a public health blunder, fortunately a harmless one, resulted.

Another episode occurred at Brother's Road, near Tabaquite, in southern Trinidad.

A seven-year-old East Indian boy (Ba) fell ill on September 28 and died in the San Fernando hospital on September 30. Yellow fever virus was isolated from his liver.

We visited his home on October 6. It was located in a moderately heavily populated agricultural region, with cocoa, sugar cane, and rice under cultivation, and was some seven miles beeline from the yellow fever infected Charuma Forest.

By good fortune, an efficient public health nurse was at the boy's house the same day, and had a dozen members of the family and neighbors assembled on the front veranda. These people had heard the message broadcast from the sound truck which toured the country regions urging yellow fever immunization, but had not been sufficiently stirred to visit the immunization center, some three miles distant, when it was operating three weeks earlier.

The nurse had taken temperature readings on the assembled people and had two on hand with temperatures reading 99° F., and three more fever cases located in nearby homes.

The dead boy's father and brother were both forest workers in the Charuma Forest, so our attention was focused on them. The father denied any recent illness. The brother said he had had a slight fever about a month before his brother became ill. Blood specimens were taken on both of these men. Both specimens neutralized yellow fever virus. However, the father's serum was negative on complement fixation test with yellow fever antigen, indicating an infection in the more remote past, while the brother's serum

fixed complement in high titer, indicating an infection in the recent past. Thus evidence points toward the older brother's carrying a forest-acquired infection to his home, infecting *Aëdes aegypti* there, and in this way serving as the indirect cause of his brother's illness and death.

Two girls, aged 9 and 16, cousins of the dead boy, and living a quarter of a mile away, were on the front porch. The nurse indicated that both had temperature readings of 99°. The girls were examined cursorily. They did not know they had fever and had no complaints except for mild headache in one. Blood specimens were taken as routine. Both specimens yielded yellow fever virus. Later follow-ups established that both girls recovered rapidly and uneventfully.

We went up the road to the home of a small boy who was too ill to come to the gathering. I felt he was a yellow fever case, yet no virus was isolated and the later serological studies failed to support this diagnosis.

Next we visited the home of the dead boy's uncle and aunt, about half a mile away. Both were ill, the uncle prostrate with high fever and severe headache, the aunt less ill. Blood specimens were taken and the nurse was urged to get these people into the San Fernando hospital. They were taken there the next day and both recovered uneventfully. We recovered no virus from these two persons but, upon serological evidence, the seriously ill uncle did not have yellow fever and the less seriously ill aunt showed a strong conversion with both neutralization and complement fixation tests.

Dr. Aitken, the entomologist, found *Aëdes aegypti* breeding on the premises although no adults were found. We have belabored ourselves in retrospect for not searching the girls' house. Prospects there would appear to have been excellent for making an isolation of yellow fever virus from *Aëdes aegypti*. Excuses are that distances were long, the hour late, and the ice hamper already full of materials to get back to Port-of-Spain. The bare truth is, of course, that we did not really think those girls had yellow fever and did not attach enough importance to them.

The Brother's Road episode related above yielded the original yellow fever death, the probable source of infection, and three additional secondary cases, plus a lost opportunity of catching *Aëdes aegypti* actually at work. Such an opportunity may not offer itself again to yellow fever workers for another several decades, particularly with *Aëdes aegypti* eradication programs progressing so well in so many regions of the world.

The origin of the 1954 yellow fever outbreak in Trinidad is obscure. Maybe infection "hopped over" from nearby Venezuela, which is reported to have had cases in the Orinoco Delta region in 1953 and 1954. Or possibly the disease remains in Trinidad, smouldering, with occasional flare-ups.

The evolution of the 1954 yellow fever outbreak permits of interesting speculation. The Health Department of the Government of Trinidad and Tobago started an intensive anti-*Aëdes* campaign in June, 1954. This was a few days after indisputable laboratory confirmation of the first case seen in mid-April. An island-wide immunization program was begun in July, directed first toward forest dwellers. By the time the epidemic had reached its apparent peak, during August and September, 1954, a large proportion of the residents in the forested areas of the island had received immunizations with 17 D vaccine and extensive *Aëdes aegypti* control had been accomplished in the urban areas. Before the Health Department program was started, the island population in large part had never been immunized against yellow fever, except for the considerable immunization of older inhabitants as a result of earlier epidemics. *Aëdes aegypti* house indices were high, both in urban and rural areas. The stage was set for an explosive outbreak of yellow fever. Whether the Health Department's prompt and vigorous campaign headed off a serious epidemic will never be known. How many minor localized epidemics, such as the Brother's Road outbreak narrated above, may have been averted, is likewise an unknown story. Any one of these could have been the starting point for extensive, serious trouble. The fortuitous finding of the case in April, 1954, permitting a control program to be well under way before the epidemic peak was reached, may easily have served to prevent a major outbreak.

As is the case in most public health work today, credit for an intangible victory rests with many organizations, among them the Trinidad Regional Virus Laboratory and the Rockefeller Foundation Laboratories in New York for early recognition and later following of the epidemic; the Health Department of the Government of Trinidad and Tobago for prompt institution of control measures; and the Pan American Sanitary Bureau for immediate detailing of experts and for supplying large amounts of 17 D vaccine, the latter being sent from the Instituto Oswaldo Cruz in Rio de Janeiro and the Instituto de Estudios Especiales "Carlos Finlay" in Bogota.

SUMMARY

A brief history of the 1954 yellow fever outbreak in Trinidad, B.W.I., is presented. Special emphasis is laid upon difficulties in recognition of mild cases of yellow fever in the field. It is suggested that prompt recognition of the epidemic threat and early institution of control measures averted a serious epidemic.

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