

**A Virus in the Forest:
Yellow Fever, West Africa, and the Remaking of Alliances**

Among Living Things, 1900-1950

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Introduction

In 1946, the American naturalist Marston Bates looked back upon twenty years of a massive, coordinated effort by the Rockefeller Foundation, an endeavor that spanned three continents, to map, control, and eradicate yellow fever. In 1925, scientists from the Rockefeller Foundation's West Africa Yellow Fever Commission arrived in Lagos, Nigeria, confident in their ability to eliminate the disease from the Western hemisphere. Their presumption was bolstered by a set of anthropocentric assumptions: that yellow fever was a "purely human disease", that it was transmitted from human to human by the human-loving *Stegomyia* mosquito, and that it prevailed in urban centers along the Atlantic coast of West Africa, South America, and the Caribbean where human and mosquito populations flourished. Within two decades, the Rockefeller Foundation had given up on its original goal. The complex life cycles of the yellow fever pathogen, which extended far beyond the intertwined worlds of humans and mosquitoes to include a multiplicity of beings, made eradication an impossible task.¹

In those intervening years, a new understanding of yellow fever emerged as a human-centered presumption transformed into a multispecies reality. Consequently, "the clear concept of yellow fever epidemiology that prevailed in 1925 has become as extinct as the dodo", noted

Bates from his field laboratory in a sparsely populated frontier town in the Llanos region of Colombia.² Even the term epidemiology had become problematic. With its focus on the pathogens of *demos* (people), epidemiology had largely centered its inquiry on the determinants, distribution, and prevalence of disease in human populations. But, as the natural history of parasites revealed, microbes bound humans and animals together in what Darwin called an “entangled bank” out of which “endless forms most beautiful and most wonderful have been, and are being evolved”.³ Broadly defined, epidemiology was, in Bates’s view, “the ecology of pathogenic organisms”.⁴ As such, epidemiology’s task was to follow a pathogen’s ecology, wherever it might lead.

Viruses, in the parlance of biology, are obligate parasites. Derived from the ancient Greek word *παράσιτος* [parásitos], parasite literally means “a person who eats at the table of another.” Capable of reproducing only in the cells of other organisms, they inhabit a liminal world between animate and inanimate existence. The virus’s parasitic nature – in particular, its need of other organisms to propagate itself – offered a means for scientists in the early twentieth century to make visible its prevalence and behavior through the lives of others upon which it relied. Cross-species exchanges of blood and fluids – of humans, monkeys, mosquitoes, mice, viruses, and numerous other species – arose or were facilitated both in the Rockefeller Foundation’s effort to map the prevalence of yellow fever across geopolitical divides of West Africa and in the industrial work to produce a vaccine. Making visible the multiple ecologies of the yellow fever virus marked an important moment in what George Canguilhem described as a “complete redefinition of the alliance of living things”.⁵

Over the last two decades, historians and anthropologists have increasingly attended to zoonosis and disease emergence as a subject of inquiry. Such work has greatly enriched our

understanding of the historical epistemologies, as well as affects, logics, and infrastructures of global health security, that have shaped knowledge and belief about the ecology of diseases and their control.⁶ In this chapter, I draw upon approaches from multispecies ethnography, environmental and medical history, and political ecology to explore how infrastructures of extraction, critical to the economics and logics of empire, facilitated a key epistemic rupture in the knowledge of a pathogen and its being in the world. The collection and circulation of human blood, animal species, and viruses on a planetary scale were materially beholden to the search for, and the development of, oil, timber, cacao, rubber, and other commodities on the continents of Africa and South America. The building of roads, railways, and air transportation routes in the pursuit and commercialization of commodities constituted what virologist Stephen Morse described in 1990 as “viral traffic”. They served as new “viral highways and byways” that in Morse’s words enabled existing viruses to “conquer new territory” and “gain access to new host populations”.⁷ But they also made visible how a virus, namely, yellow fever, once thought to infect only humans, also lived in animal reservoirs in tropical forest regions in South America and West Africa. Resource frontiers and their accompanying infrastructures, I argue, aided the extraction of a new commodity (viruses) and made a region (the Guinean Forests of West Africa) into a lucrative site in advancing the professional careers of Western scientists in the hunt for the yellow fever virus across West Africa.

The work of Rockefeller Foundation scientists sedimented West Africa as a “hotbed” of yellow fever and southwestern Nigeria as an endemic center.⁸ The very term “hotbed” conjured up nineteenth-century metaphors of seeds, soil, and fermentation that placed diseases in specific locales, fueled, as Jacob Steere-Williams and others argue, by colonialism.⁹ Spatialization of blame accompanied the making of the Guinean Forests of West Africa into a disease reservoir

and the region as an endemic zone of endangerment.¹⁰ Notions of endemicity precipitated strategies of containment and protection, built on racist logics, inequitable exchanges, and different valuations of life that drove and sustained extractive economies of empire.

The remaking of yellow fever and its ecologies from an urban disease into a sylvatic one turned the forests of West Africa, or more precisely, their fluid edges into what Natalie Porter describes as risky zoographies.¹¹ Disease ecology and colonial conservation aligned in marking the rural in West Africa, where slash-and-burn agricultural practices brought the farm and forest into intimate relation, as a zone of endangerment, one that threatened the health of people, plants, and wildlife. Notions of containment traveled from the urban to the rural. But in the spaces of the rural, containment became a double-edged colonial sword, whereby public health and conservation were used to facilitate violent acts of enclosure. In “spillover” hypotheses and zoonotic disease diagrams, the forest’s fluid edge was made into a morally charged and pathologized space of interspecies minglings, whose boundaries needed to be more clearly delineated and controlled.¹² Among the local peoples of Yorubaland, upon whose knowledge of and relationships with the forest Rockefeller scientists relied, being in good relation with the plants, wildlife, and spirits of the forest was, for them, a precondition of human flourishing. But, as I argue in this chapter, such notions of community livelihood and well-being were at odds with ideas and values of the forest and health that Western scientists and British colonial officers sought to impose. In the search for sylvatic yellow fever, knowledge and ways of being collided in a struggle over who had access to, and control of, the forest and its resources.

Cross-species exchanges

In 1920, members of the Rockefeller Foundation's Yellow Fever Commission to West Africa were confident that the "theatrically prompt eradication" of yellow fever "in great centers of endemicity such as Havana, Panama, and Rio de Janeiro", made consideration of a possible animal reservoir of the disease unnecessary.¹³ The Rockefeller Foundation's International Health Board (IHB), later renamed the International Health Division (IHD), had selected General William Gorgas to head the commission. Gorgas had rid Cuba, the Panama Canal zone, and parts of Latin America, of yellow fever by controlling the insect responsible for its transmission: the mosquito, *Stegomyia fasciata* (later renamed *Aedes aegypti*) first suspected as a source of yellow fever transmission in 1881 by the Cuban physician, Carlos Finlay.¹⁴ Gorgas believed that attacking the disease in "its ultimate stronghold", West Africa, would achieve the Rockefeller Foundation's goal: to eradicate yellow fever from the face of the earth.¹⁵ His death from a stroke in London before the team departed for Lagos did not dampen the IHB's hubristic ambitions.¹⁶

No philanthropic institution in the twentieth century rivaled the investments made by the Rockefeller Foundation in medicine and public health. In 1911, when an anti-trust lawsuit broke up Standard Oil Company, the largest petroleum company in the world, John D. Rockefeller Sr., the company's founder and chairman, was the richest man on earth. Backed by 72,000 shares of Standard Oil Company, valued in today's dollars at \$1.6 billion, John D. Rockefeller, his only son, John D., Jr., and his long-term advisor, Frederick Gates, established in 1913 the Rockefeller Foundation, a non-profit philanthropic organization dedicated to "the acquisition and dissemination of knowledge, in the prevention and relief of suffering" in the United States and throughout the world.¹⁷

The sizable business interests of the Rockefeller family in Latin America had long made the area a focus for its philanthropic arm. But in the 1920s, West Africa was a new region of

interest for the Rockefeller Foundation. With tropical Africa “taking its place in the world’s economy”, noted Rockefeller Foundation scientist Oscar Klotz, the “demand for action in the yellow fever problem is becoming more insistent each year”.¹⁸ The 1920 Rockefeller Foundation Yellow Fever Commission to West Africa lasted only three months, from July to October. Unable to find any active yellow fever outbreaks in British, French, or Belgian colonies, it concluded nevertheless that the disease was “endemic and epidemic on the coast” and recommended the IHB establish a commission in West Africa of unlimited duration, which it did five years later.¹⁹

In 1925, the Rockefeller Foundation dispatched a small team of doctors, scientists, and laboratory technicians to establish a research headquarters, in Yaba, a suburb of Lagos, Nigeria, with a field outpost in Accra, Ghana, then the Gold Coast.²⁰ The choice of Lagos was not coincidental. British colonial medical officers considered Lagos and towns lying north a likely endemic center of yellow fever in West Africa. Steps toward eradication, however, could not begin until Rockefeller scientists were certain that yellow fever in West Africa and South America was one and the same disease and its geography and prevalence along the West African coast was known.

Infrequent outbreaks of yellow fever hindered the team’s initial investigative efforts. Henry Beuwkes, the disgruntled head of the commission, sought a reliable nonhuman host that could sustain the disease-causing organism in the lab. Returning to Africa from New York City, he stopped in Hamburg, Germany to meet with the famed animal collector Carl Hagenbeck.²¹ Hagenbeck, whose private zoo pioneered modern animal displays, offered monkeys, including marmosets from Brazil, and rhesus and crown monkeys from India, in large numbers at 40 marks each – half the cost in the United States. The former Army Medical Corps physician promptly

purchased fifteen marmosets, fifteen rhesus macaques, and ten crown monkeys and arranged for shipment to West Africa.²² Beeuwkes bought an additional six chimpanzees from Pastoria, a newly created Pasteur Institute, in the French West African colony of Guinea. The Pastoria primate purchase was not without controversy. The Lieutenant Governor of French Guinea expressed reservations in supplying chimpanzees to a foreign colony, indicating the geopolitical divides that Rockefeller Foundation scientists increasingly found themselves needing to navigate across West Africa.²³ Beeuwkes secured three more chimpanzees from Sierra Leone through Frank Buck, an American celebrity whose best-selling book and film, *Bring 'Em Back Alive*, recounted his world travels in search of exotic wildlife. Beeuwkes arrived in Accra, accompanied by University of London pathologist, Adrian Stokes.²⁴ Only three of the six chimpanzees on board had survived. The physicians and chimps arrived at a serendipitous time. Yellow fever had surfaced in Accra and an epidemic had erupted in Larteh, a village thirty miles inland. With a partial shipment of Hagenbeck's monkeys in port, Stokes decided to depart ship with two of the chimpanzees, while Beeuwkes and the third chimpanzee went on to Lagos.²⁵

With their menagerie gathered, and an epidemic nearby, a novel experiment could proceed: to see whether a nonhuman primate was susceptible to a parasitic organism thought only to infect humans. At the time, Rockefeller scientists were simply looking for a suitable animal besides humans to experimentally aid in the diagnosis of yellow fever and identify the causative agent of the disease. No evidence suggests they were looking for an animal reservoir of the disease in these initial trials. Blood from infected yellow fever patients injected into marmosets and chimpanzees yielded disappointing results. Experiments with crown monkeys at first looked more promising, but proved otherwise.²⁶ When the Accra group learned of a yellow fever outbreak in Kpeve, a small village on the border of the Gold Coast and Togoland, Dr. A. F.

Mahaffy went to collect blood specimens. A twenty-eight-year-old villager presenting mild yellow fever symptoms, referred to only as Asibi by the Rockefeller staff, gave his blood, willingly or not, to the white doctors.²⁷ In Accra, one rhesus macaque, one marmoset, and two guinea pigs received injections of Asibi's citrated blood. The rhesus macaque, labeled 253A, died five days later.²⁸ To firmly establish the identity of the disease, the team needed to transmit yellow fever from an infected monkey to a healthy one, not by injection, but by a mosquito bite. Local authorities in Accra forbid yellow fever experiments using mosquitoes, fearing risk to the local population, much to Stokes' dismay. The Yaba team in Nigeria faced no such restrictions. Heart blood and kidney and liver fluids drawn from the dead macaque were injected into a healthy monkey, 253B, who was then expedited to Yaba. There, mosquitoes dined on 253B's blood before the animal died. Over the next few weeks, the team successfully established a chain of yellow fever transmissions between monkeys through both syringe injections and mosquito bites. They also demonstrated that the organism responsible for yellow fever was not the bacillus *icteroides*, as the bacteriologist Hideyo Noguchi proclaimed, but a filterable virus.²⁹

Cross-species exchanges in blood and fluids were the stuff of early virology research. Investigating unknown and unseen disease agents was a Wild West frontier, and could be deadly. Brightened by the cloudburst of discoveries that accompanied Lagos's rainy season, the atmosphere of Yaba quickly turned dark when Adrian Stokes fell ill on September 15, 1927. Beeuwkes suspected yellow fever but ruled out the possibility of an escaped mosquito as the cause in the lab's chaotic flurry of experiments. "Monkeys are rather wild and difficult to handle", Beeuwkes admitted.³⁰ Ten days earlier, one had bit Stokes' finger, leaving an open wound. By day two, racked with headache, body pain, and fever, his tongue coated dirty white, Stokes begged his colleagues to draw his blood and let mosquitoes feast upon him to sustain a

new batch of inoculations in the lab. By day five he was delirious. A weakening pulse and jaundice had set in. The Rockefeller team, desperate, tried convalescent serum therapy, injecting into his arm 20cc of blood serum from a recovered yellow fever patient, to no avail. Stokes' autopsy confirmed yellow fever.³¹ His demise was followed by a string of deaths among laboratory staff in Lagos and Accra, including Noguchi. These deaths shocked the international scientific community and gave officials at the Rockefeller Foundation pause.³²

But a new frontier had been opened. Yellow fever was not only a human disease. Viruses shared through bodily fluids exchanges in the lab demonstrated the existence of multispecies disease relationships through industrial ecologies of virus research and the possibility of such relationships in the wild.³³

Mapping immunity

Within months of their discovery of cross-species transmission, the Yaba team mobilized rhesus macaques as a tool of disease mapping. British imperial networks facilitated access to macaques, native to India, in the large numbers needed by Rockefeller scientists.³⁴ The so-called protection test they developed could determine the presence or absence of yellow fever antibodies in human blood. Protective antibodies against the virus in individuals without yellow fever symptoms indicated past infection. Yellow fever antibodies in the blood thus became proxies of past epidemics in the landscape, provided individuals who tested positive hadn't migrated from a different region.

To perform the test, a person's blood serum was mixed with live virus and injected into two monkeys. If both monkeys died, the human lacked the antibodies that provided yellow fever immunity. The protection test records, contained in thirty-four volumes labeled the Monkey

Books, are a chilling record of death on an industrial scale as rhesus macaques became a critical cog in a growing virus research enterprise (Fig. 1). Hundreds of tabs in each book show the identity number of a monkey and almost every number is underlined in red, indicating the animal died.³⁵ As many as 2,500 died each year until Rockefeller scientists developed a cheaper test that used mice instead of monkeys. The mouse protection test came to Yaba in 1931.³⁶

Figure 1. A cover and page from volume 15 of the Monkey Books. The death of *M. rhesus* 1950, inoculated with the serum of Dr. Rice, a physician of the Firestone Plantations Company located in Liberia, was proof that Rice had not been infected with the disease during a 1929 yellow fever outbreak in Monrovia. Courtesy of the Rockefeller Archive Center.

In 1929, the Yaba team launched a massive seroprevalence study, then the largest of its kind. Collecting blood from a wide set of an area's population and testing it for disease exposure mapped the extent of yellow fever's range in West Africa. What was the extent of immunity to the disease on the African continent? How recently had epidemics occurred? Was yellow fever endemic in West Africa, as many scientists believed, and, if so, where? Making visible the epidemiology and geography of yellow fever in a region of growing interest to colonial powers and commercial firms was valuable, both scientifically and economically. Such knowledge was critical for implementing policies of disease control to aid commerce and trade exchanges around the globe.³⁷

Immunological mapping of a disease required tens of thousands of imported monkeys and mice for use in conducting protection tests. It required thousands of human blood samples taken from children in mission and government schools and from adults housed in hospitals and

prisons, employed on concessions, and among the population at large.³⁸ Children's bodies, like tree rings, offered clues to the timing of past events. When a child's blood yielded a negative protection test, it indicated the absence of a yellow fever outbreak where the child lived during its lifetime.³⁹ Scientists also collected blood samples in commercial centers and towns located along rail, automobile, river, and caravan routes. The Yaba team reasoned that yellow fever could most easily travel from presumed endemic zones to new population centers along corridors of communication and commerce.⁴⁰

Initially focused on Nigeria and the Gold Coast, the Rockefeller scientists utilized additional arteries of colonial extraction and commerce to harvest and transport blood and other bodily fluids around the globe.⁴¹ As a self-proclaimed humanitarian, politically neutral, philanthropic organization, the Rockefeller Foundation was able to more readily coordinate work across Belgian, British, French, and Portuguese colonies (Fig. 2). In Liberia, the Firestone Plantations Company assisted the mapping project. The workforce of the newly developed rubber plantations, composed in 1928 of 18,000 Liberian laborers, was an abundant source of blood for Rockefeller scientists, who, in turn, aided Firestone in diagnosing yellow fever and advising on its control.⁴²

Figure 2. A 1931 map displaying the reach of seroprevalence studies undertaken by the RF West African Yellow Fever Commission across the geopolitical divides of Central and West Africa. Courtesy of the Rockefeller Archive Center.

By the early 1930s, the seroprevalence immunity surveys conducted by the Yellow Fever Commission had mapped southwestern Nigeria as an endemic center of yellow fever. But maps

of endemicity, as historian Jennifer Tappan notes, were also “maps of susceptibility”.⁴³ Even in areas of supposed high endemicity, the number of African children immune to yellow fever ranged from nine to sixty-eight percent.⁴⁴ In other words, a large, vulnerable population, with no protective immunity, lived in a region where yellow fever was a recurrent disease.

After a decade of yellow fever research, the Commission no longer envisioned “radical campaigns looking toward the eradication of yellow fever in West Africa”.⁴⁵ Instead, Beeuwkes argued that large inland urban centers, poor-quality sanitation, and the “low cultural level of the masses” made any program aimed at elimination or control futile.⁴⁶ Southwestern Nigeria became a risky geography. Increasingly, the Rockefeller Foundation’s International Health Division focused on identifying zones of endangerment, an effort aimed at protecting foreigners, invariably white foreigners, and their interests. French officials faulted the British for failing to contain yellow fever outbreaks in their own West African colonies, which, they claimed, resulted in yellow fever “infections appearing sporadically in French territories”.⁴⁷ Beeuwkes blamed the persistence and exacerbation of yellow fever outbreaks instead on Africans themselves, whom, he argued, lacked the “sanitary standards” as well as “the intelligent and sympathetic participation of the public” in public health campaigns necessary to stamp out the disease.⁴⁸ Attitudes of cultural and racial superiority shaped accusations of blame.

The scope of what Beeuwkes described as “hotbeds” of yellow fever shook colonial powers with holdings across Africa. Writing in July 1932 on behalf of the Office International d’Hygiène Publique (OIHP), a precursor to the World Health Organization, Colonel S. P. James, a former British officer in the Indian Medical Service, expressed grave concern to IHD director Frederick Russell about the Yaba team’s findings. “The protection-test results,” James wrote, “have seriously alarmed the delegates of various infectible but as yet uninfected countries”.⁴⁹

Quarantine, he argued, was an ineffective response to reports of disease or identified areas of endemicity. The opening of air passenger routes from London, England, to Cape Town, South Africa, with planned expansion service to West Africa amplified fears. Such anxieties were very much a part of discussions at the Sanitary Conference of the Health Organization of the League of Nations, held in Cape Town in November 1932, where Wilbur Sawyer, Director of Laboratories of the Rockefeller Foundation's International Health Division, was in attendance as "a neutral expert".⁵⁰ One stowaway mosquito might carry disease from West Africa to East Africa and on to India where yellow fever was not known. Some members of the OIHP Yellow Fever Commission, which James directed, questioned the reliability and veracity of protection test results. Others found comfort in mistaken ideas of racial immunity. James asked Russell if just as yellow fever appeared "inoffensive" to "the native of Africa" who "lodges, sustains, and transports the virus," might not the same apply to the native of British India.⁵¹ James's wishful – but wrong – thinking was blind to the death toll yellow fever wrought upon African populations. He and others misconstrued the acquired immunity of individuals who survived childhood exposure to the disease as immunity based on an already problematic biological definition of racial types. Belief in racial immunity was a convenient scapegoat for holding different valuations of life that shaped health policies and actions. Since, James believed, yellow fever was only "an important cause of sickness and death" among whites in Africa, wouldn't "preventive inoculation or vaccination of white races" be a "sufficient prophylaxis?", he asked Russell.⁵² As Rockefeller Foundation dreams of yellow fever eradication faded in the early 1930s, Russell began devoting significant staff, staff, and funding to such a prophylaxis; namely, vaccine development.

Industrial ecologies, vaccine inequities

In the late 1920s, another hotbed of yellow fever virus was fermenting, but in the temperate zone. The International Health Division opened its Yellow Fever Laboratory on the east side of Manhattan, New York City, in June 1928, initially occupying two rooms of the Rockefeller Institute, at 66th street and York Avenue. Monkeys, some infected with yellow fever, were housed in the largest of the rooms. Thomas Norton, a twenty-year-old premed student working as a lab technician, described monkeys “as more important than the men who merely worked on them”. He also related risks he and others encountered daily. Staff and visitors entered the lab through a screened vestibule, meant to prevent mosquitoes escaping and carrying yellow fever to city residents. The laboratory director, Harvard-trained physician Wilbur Sawyer, instructed visitors to keep their hands in their pockets to avoid contamination.⁵³

In the monkey-filled “dirty room”, staff in white trousers and lab coats and wearing rubber gloves and aprons worked long hours every day to conduct protection tests and to maintain the virulent Asibi strain in an ever-rotating colony of rhesus macaques.⁵⁴ The monkeys did not take kindly to either captivity or the jabs and prods of syringes and thermometers. One monkey, Norton recalled, incensed by a researcher’s scolding, grabbed a “cherished pipe” from the doctor’s mouth and smashed it to the ground.⁵⁵ Biting and throwing food, or other matter, were among the tactics macaques employed to express resistance to an association that reliably ended in death. Other risks, Norton disclosed, included “infectious blood unknowingly being sprayed or spattered about from sick monkeys with bleeding gums or bloody stools”.⁵⁶ In the first two years of operation, Norton, Sawyer, and five other lab personnel caught yellow fever. Miraculously, all survived.⁵⁷

The dangers of laboratory research and an ever-expanding geography of yellow fever across West Africa, visualized by seroprevalence mapping, spurred a global race for vaccine. The geopolitics of empire and the arrogance of Western science shaped it. Since the 1880s, rival claims of the discovery of yellow fever's aetiological agent propelled triumphant pronouncements and dashed hopes of a yellow fever vaccine.⁵⁸ By the spring of 1928, two strains of the virus had been isolated for research use. One was the strain collected from Asibi Dagomba by the Yaba team. The other originated in French West Africa when Andrew W. Sellards, an assistant professor in Harvard University's Department of Tropical Medicine traveled to the Pasteur Institute in Dakar, Senegal, with the financial support of Firestone, and met with French colleagues, Jean Laigret and Constant Mathis. Together, they isolated a strain of the yellow fever virus from a Syrian patient, François Malayi. The Pasteur team managed to maintain their virus in a rhesus colony in Dakar for three months. To transport the virus to Harvard, Sellards killed a monkey at the most virulent stage of infection, froze liver samples, and escorted the tissue, containing dormant yet live yellow fever virus, aboard ship to London and Boston.⁵⁹ At Harvard, Max Theiler, Sellards' colleague, successfully transmitted the Dakar strain to the common white mouse by injection into the brain. In mice, unlike monkeys, the yellow fever virus localizes in the nervous system. Infected mice died of encephalomyelitis, a pathological manifestation of the virus previously witnessed in neither humans nor monkeys.⁶⁰

In the New York Yellow Fever Laboratory, Sawyer and his staff used Theiler's mouse results to engineer an in-house vaccine for laboratory workers. Sawyer also recruited Theiler to work for the Rockefeller Foundation's IHD. Beginning in the early 1930s, IHD employees working with yellow fever received a two-part protective treatment: an inoculation of the French strain, weakened through serial passage in mice, and an injection into the abdomen of antibody-

rich human blood serum collected from laboratory personnel who had survived yellow fever. Theiler and other American and British scientists regarded the mouse-adapted French strain, with its neurotropic properties, too dangerous for a mass vaccination program. Theiler, with Rockefeller colleagues, instead cultured the Asibi strain in minced chicken embryos lacking nervous tissues. After more than one hundred subcultures, a variant of the Asibi strain of virus, labeled 17-D, resulted in a “safe” vaccine that stimulated antibody production in monkeys and did not elicit fatal encephalitis in mice.⁶¹

In 1937, production of the 17-D vaccine in the New York laboratory began. Vaccine mass production entailed an industrial ecology of multispecies relationships. The laboratory, which had taken over an entire floor of the Institute, consumed three to four thousand mice per week, one thousand monkeys per year, and countless chicken embryos. Roughly thirty-four million vaccine doses produced by the close of the Second World War mostly went, at no cost, to protect American, British, and other Allied troops.⁶²

British and French colonies in Africa used contrasting methods to supply and distribute yellow fever vaccines. In 1938, with vaccine production increasing in New York, Sawyer wondered if the IHD might engage the British in a plan for yellow fever control through vaccination in West Africa, where four years earlier the IHD Yaba operation in Nigeria had closed.⁶³ Fred Soper, the administrative head of the Rockefeller Foundation’s programs in South America, where a 17-D vaccination rollout had begun, brazenly rebuffed Sawyer with disdain for Africans. “Yellow fever in the native is of little or no importance”, Soper insisted.⁶⁴ He doubted Sawyer could get the British colonial government to use the costly vaccine “in the absence of proof of the importance of yellow fever as a killing disease among the natives”.⁶⁵ A 1940 outbreak of yellow fever in the Nuba Mountain region of Sudan resulted in an estimated 20,000

cases and 2,000 deaths, undermining Soper's claim of mild disease among Africans. The IHD, prompted by Sawyer, supplied the British with more than one million doses of 17-D vaccine to help control the Sudan epidemic. During the war years, the British immunized military personnel east of Nigeria and civilian populations in Eritrea and along the Kenyan coast. Roughly three million doses of the vaccine were also distributed in Uganda. But the mass vaccination program that Sawyer envisioned to protect both African and British subjects in British West Africa never transpired.⁶⁶

The French did adopt a mass vaccination strategy. In 1941, under the orders of the Vichy regime, a combined smallpox and yellow fever scratch vaccine became mandatory across French West Africa (AOF). Outbreaks of yellow fever in the military, coupled with fears of Africans as potential reservoirs of the virus, prompted collective action.⁶⁷ The AOF's fascist regime, federated system of direct rule, and stronger assimilation policies help account for the biopolitical differences apparent in the French versus British vaccination strategies. In the following decade, an estimated twenty million people of African and European origin living in the AOF received the vaccine, many twice. The vaccine relied on the highly neurotropic virus strain isolated in Dakar by Sellards, Laigret, and Mathis. Unlike the 17-D vaccine, the Laigret-Sellards version did not require cold chain refrigeration and was cheaper to produce. But it had well-known, troubling side effects. Laigret dismissed these, thinking them far less adverse than "the dangers of yellow fever".⁶⁸ By the 1950s, under a new French Constitution that granted greater, albeit limited political and citizenship rights to its African colonies and subjects, reports of serious complications from the vaccine – encephalitis and death, particularly among children under the age of ten – were mounting. One estimate is that the vaccine killed up to 3,000

children. The vaccine was withdrawn from use on European children in 1951, but a differential valuation of life let its use on African children continue until 1960.⁶⁹

Hazards of the forest

The International Health Board entered West Africa in the 1920s confident that the yellow fever problem in South America had been brought under control.⁷⁰ Within a decade, however, those much-touted triumphs were fading. Brazil and Colombia saw new outbreaks, including some in remote rural regions, which puzzled Rockefeller scientists, since yellow fever was thought to be an urban disease.⁷¹ In the isolated emerald mining area of Muzo, located in Columbia's Llanos frontier, yellow fever cases appeared frequently. Yet, the mosquito *Aedes aegypti* was nowhere present. Hoping to unlock this yellow fever anomaly, the IHD established a lab in the frontier town of Villavicencio.⁷²

In the 1930s, the Llanos region lay outside the reach of the Colombian nation state. Seventy-five miles to the southeast of Villavicencio, where an island-like mesa, the Cordillera Macarena, rose abruptly from the tropical lowlands, workers contracted by foreign firms in search of oil and mineral deposits opened areas in the dense forest.⁷³ In January 1940, the head of the Villavicencio lab, John Bugher, learned of a Shell Oil employee, Hernando Ríos, who was presenting yellow fever-like symptoms at a local clinic. Protection tests confirmed yellow fever. Disease in a remote forested region far from an urban center was just the opportunity the virus hunter Bugher had been looking for.⁷⁴

Bugher was a bacteriologist and pathologist by training and a good "field man". Working with his Colombian colleague, Jorge Boshell-Manrique, rhesus macaques, mice, and equipment were airlifted to a patch of savanna and carried by mules across rivers and through dense forest

to establish a base camp. Bugher boasted of “collecting every form of life they could find”.⁷⁵ Insects of all classes were caught, pulverized, and injected into the monkeys and mice. Wild monkeys were shot and bled for protection tests. The mosquito *Haemogogus capricornii*, which lives high in the forest canopy, was shown to carry the yellow fever virus. Studies to determine the insect’s ecology and behavior began. Marmosets and saimiri monkeys proved susceptible to the yellow fever strain transmitted by *Haemogogus capricornii*, and were considered important reservoirs. This work detailed a sylvatic cycle of yellow fever in the tropical forest. The potential for zoonotic spillover of yellow fever from monkey to human arose when workers came to topple towering trees, which brought infected mosquitoes down from the canopy for a meal of human blood. Infected workers traveling to nonimmune, mosquito-ridden population centers brought the possibility of urban outbreak (Fig. 3). Viruses moved in the paths of extraction infrastructures – of commerce and of science – to make new alliances across species divides.⁷⁶ The intertwined relations of human and nonhuman beings inhabiting a rural, forested region upended a key assumption that motivated the Rockefeller Foundation’s eradication program; namely, that yellow fever was strictly an urban, human disease. A new scientific object, “jungle” or “sylvatic” yellow fever came into being.⁷⁷

Figure 3. A diagram depicting the sylvatic cycle of yellow fever and its leap to an urban cycle in South America. Note the sharp boundary between urban and jungle. From George K. Strode, ed., *Yellow Fever* (New York: McGraw-Hill Book Co., 1951), p. 536.

If monkeys in the tropical forests of South America could serve as hosts of the yellow fever virus, might the same be true in the Guinean Forests of West Africa? In 1943, the

Rockefeller Foundation sent Bugher to the former Yaba laboratory to find out.⁷⁸ At Yaba, Bugher hoped to demonstrate what Rockefeller colleagues in a field station in Bwamba, Uganda had begun to suspect: a sylvatic cycle of yellow fever existed in the equatorial forests of western and central Africa, which circulated among monkeys and mosquitoes, independent of humans.⁷⁹

“All of the evidence of yellow fever in recent years in Nigeria is limited to the southern forested areas where the monkey population is abundant”, Bugher wrote.⁸⁰ “It is because of the implications of such hypothesis that we are now engaged in a survey of monkey immunity throughout the whole of southern Nigeria”.⁸¹ Looking for yellow fever among monkey and mosquito species in the forests of southwestern Nigeria, Bugher believed, was far more valuable than continuing to investigate its prevalence in human populations.

In a fragment of the Guinean Forest, not far from the railway line that connected Yaba to Abekouta, Bugher, aided by the British colonial forestry service, began work on a monkey immunity survey. Violent acts of enclosure by the British in 1901, and again in 1938, had alienated Yoruba land from its peoples in the creation of the Ilaro Forest Reserve.⁸² When the British seized control of the Ilaro forest in 1901, local chiefs objected to restrictions placed on use rights to the forest that British law imposed. One chief bitterly complained to the District Commissioner of the seizure by the government of “over a hundred kola nut trees in one portion of the forest” that he had inherited from his ancestors.⁸³ The usurpation of forest use rights by the British in the name of conservation live in the memories of local people almost a century later. An elder from the neighboring village of Ipake, for example, recalled how his ancestors were forcibly displaced from the forest, where they settled and farmed, gathered fruits, nuts, and healing plants, harvested wild game, and paid tribute to a forest-dwelling orisha that brought plenty to their lives. The Ilaro forest was a mix of village, farm, and forest when the British took

control, he told me.⁸⁴ Bugher admitted as much. The Ilaro Forest Reserve offers “a range of vegetation”, but could not “be termed undisturbed nor” is it “anything approaching virgin or climax forest [...] suitable for the establishment of a permanent field station from the Ecological aspect”, Bugher observed.⁸⁵

Notions of pristine nature, undisturbed by humans, shaped Bugher’s vision of risky zoographies. In a diagram illustrating the virus jump from jungle to urban yellow fever in Africa, the fluid edges of forest and farm, where domesticated and wild species meet, becomes a potential pathological space. Zoonotic disease diagrams, Christos Lynteris argues, function as an “epidemiological Rosetta stone [...] rendering infection intelligible as a relation that spans the species divide”.⁸⁶ In the depiction of the sylvatic cycle of yellow fever in Africa, it is “the proximity of the house to the forest” that determines human vulnerability to infection. Given that “a large portion of the population of Central West Africa lives in contiguity with forested regions”, noted Bugher, “the forests must be recognized as a hazard which will endure into the indefinite future”.⁸⁷ Cultivated crops, such as bananas, attracted monkeys from the interior to the unruly edges of the forest, where mosquito species, like *A. simpsoni*, did not discern between human and non-human primate blood. A resulting spillover could burn through a non-immune human population “like a crown forest fire”.⁸⁸ Absence of yellow fever “in the coastal and interior communities”, argued Bugher, did not “preclude the sudden appearance of the disease in an epidemic wave”.⁸⁹ In the diagram, cultivation of domestic crops on the forest’s edge is rendered as the primary route of infection between monkey and human. Local farming practices on the forest’s edge, not colonial industrial extraction, are implicated in “geographies of blame”, which, Lynteris notes, are common to zoonotic disease diagrams (Fig. 4).⁹⁰ It is not so much

being in nature, but being in nature in the wrong way, that becomes the threat in this moral tale of disease ecology that stigmatized Yoruban relationships with the forest.

Figure 4. A diagram depicting the sylvatic cycle of yellow fever and its leap to an urban cycle in Africa. Note the much more fluid boundary between urban and jungle than that in the South America illustration. From George K. Strode, ed., *Yellow Fever* (New York: McGraw-Hill Book Co., 1951), p. 537.

And, yet, to make visible the ecology of a virus, Bugher was himself dependent upon the skills of a local Ilobi hunter, Nosiru, in addition to the help of countless other Africans, in gathering insects and mammals in the forest.⁹¹ Eighty-six-year-old Oba Saliu Adekokun Ajibade (Oba is a title designating a position of leadership and authority in Yorubaland) recounted to me with his razor-sharp memory, how American scientists came to his village of Ilobi on the edge of the Ilaro Forest reserve. No one knew what they were doing, he told me, and then one day they just vanished. The Oba, who had been a young boy at the time, remembered Nosiru as “the fellow who catches mosquitoes”.⁹² Nosiru, the Oba recalled with a smile, had a big pet monkey that the villagers enjoyed, and whom they would help by cutting up bananas to put in live traps. The traps were placed in trees to capture the furry Softly-Softly creatures, or pottos, in a forest where people dared not go for fear of British retribution, said the proud keeper of Ilobi history.⁹³ In the forest, amid ruins of iron ore pits, an extant shrine to the forest god Ogun, master of iron, creator and destroyer, speaks to what Robyn d’Avignon describes as “ritual archives” of human-spirit pacts.⁹⁴ In the Guinean Forests of West Africa, dwellers had, and have, ways of being in relation with one another: human inhabitants, forest denizens and occupiers of liminal worlds

beyond those of a virus. These ways of relation prescribed moral tales of order and well-being, though they were quite different from those which Rockefeller scientists believed important to the health of people and the forest.⁹⁵

Bugher searched in vain for the virus in every species of monkey and mosquito they could capture in forests in and around southwestern Nigeria. Pottos, inoculated with the virus in the lab, produced antibodies.⁹⁶ Still, his findings constituted no smoking gun. Frustrated, he thought to prove his jungle yellow fever hypothesis with an experiment. In June 1947, Bugher and colleagues, accompanied by five rhesus monkeys and 600 mice, traveled to the mountainous region of British Cameroon. In a forest reserve that encompassed the rim of an extinct volcano above a nearby town surrounded by oil palm and banana plantations, Rockefeller scientists attempted to experimentally spark a yellow fever outbreak among monkeys in the wild.⁹⁷

The team first vaccinated approximately 4,000 people in a five-mile radius around the volcano with a little-used experimental 17-D scratch vaccine developed at Yaba. It was the first large-scale trial of that vaccine. Next Bugher inoculated a rhesus monkey with yellow fever virus and lifted it to the forest canopy, hoping it would initiate an outbreak among the forest's monkey inhabitants. When that animal showed no yellow fever symptoms, the team injected another monkey, releasing it into the forest. It promptly died. Fifty boys from the nearby Catholic Mission – all of whom, Bugher emphasized, as if to thwart potential criticism, were vaccinated – were tasked with roaming the forest at night to net mosquitoes. Traps were set for small animals. Human and monkey blood samples were collected from as far away as twenty-five miles. To Bugher's disappointment, yellow fever did not spread through forest monkeys. He reasoned that the area's small mosquito population foiled the experiment.⁹⁸ The ecology of jungle yellow fever proved more complex than the Rockefeller scientists discerned. What was learned, Bugher told

foundation officers, “was that so-called bacteriological warfare is not so easy”.⁹⁹ What was missed was the demonstration of appalling arrogance and plain disregard of human life in pursuit of a hypothesis.

Conclusion

In 1951, the Rockefeller Foundation’s International Health Division concluded more than twenty-five years of yellow fever research. In his summary assessment of the massive yellow fever program, IHD director Richard Taylor counted the capturing, classification, and natural history investigations of “thousands of vertebrates and hundreds of thousands of arthropods” in the tropical forests of South America and Africa as among the yellow fever program’s great achievements.¹⁰⁰ The effort brought ecology and virology together in ways that remade epidemiological understanding. Assumptions that yellow fever was strictly an urban, human disease were shattered by the revelation that the virus also circulated among wild animals in the forest. The recognition of a sylvatic cycle of yellow fever exposed an arrogant anthropocentric vision that sought to eradicate a microbe from the face of the earth.¹⁰¹ That quest, to extinguish the existence of a single-stranded infectious RNA molecule wrapped in a protein coat and needing another being to reproduce, consumed untold money and countless lives—both human and nonhuman. It failed, but in the process, new, and unexpected, assemblages of species were found in the field and created in the laboratory.

Misplaced pride in technological prowess was tempered again in 1942 when some 78 percent of 420,000 American soldiers became infected with hepatitis B. The cause was determined to be the 17-D vaccine, which then included human serum, unknowingly contaminated with virus, as an ingredient. The incident remains the largest vaccine-induced

epidemic outbreak of disease in American history.¹⁰² Sawyer initially refused to believe but ultimately accepted the evidence that vaccine was the cause.

The emergence of a multispecies understanding of yellow fever did not foster more equitable exchanges in the power relations that shaped the chasing of ecological understanding across viral divides. Trafficking in blood and viruses, critical to the work of Rockefeller scientists, rode on infrastructures of empire, commerce, and extraction. And of course it was the threat that yellow fever posed to empire and the economic expansion of multinational corporations across the globe that motivated efforts to fund and organize the work of Rockefeller scientists. Inequitable and unjust relations were built into the industrial ecologies of virus research and vaccine production. Disregard of yellow fever as a deadly disease among Africans, collection of blood under unknown conditions of consent, and unequal access to vaccines are just a few of the ecologies of injustice that arose.

Scientific legacies and moral tales persist in Rockefeller maps depicting yellow fever endemicity and in more recent disease-risk visualizations produced by EcoHealth Alliance. Such legacies and tales combine to shape the methods and logics that inform today's ideas of the Guinean Forests of West Africa as a global hotspot of biodiversity and emerging disease.¹⁰³ Similarly, origin stories of epidemics, too easily sculpted with unreliable assumptions about a place and its people, can stigmatize local human-forest relationships in the search for blame, as when origin stories of the 2013–2016 Ebola outbreak in West Africa criticized West African forest-use practices such as bushmeat hunting and subsistence agriculture.¹⁰⁴

Tales of scientific knowledge buttressing power and control, as when the British, in the name of conservation, usurped Yoruba use rights in the Ilaro forest, are not new. Tales of fear of disease and dispensing of blame, depicted in the zoonotic disease diagram of Rockefeller

Foundation scientists that turns the forest's fluid edge into a pathologized space of risky interspecies encounters where the leap from "jungle" to "urban" yellow fever occurs, are not new. But these old tales can be worked in new ways. In the history of yellow fever research in West Africa, we see the nascent convergence of conservation biology, disease ecology, and virology. In the decades that followed, these areas of scientific endeavor have intersected further to bolster the efforts of global North scientists, government agencies, and environmental NGOs with reasons, biodiversity conservation and pandemic preparedness among them, to control access to and use of the forest by local peoples in West Africa. In these undertakings, economy, ecology, and power are at play in the remaking of alliances among living things.

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¹ Bates, "The Natural History of Yellow Fever in Colombia," 43.

² *Ibid.*, 43.

³ Darwin, *On the Origin of Species*, 489-490.

⁴ Bates, "The Natural History of Yellow Fever in Colombia", 42.

⁵ Canguilhem, "Foreword", xi.

⁶ There is now a large literature on the history and anthropology of zoonosis, disease emergence, and pandemic preparedness. See, e.g.: Anderson, "Natural Histories of Infectious Diseases"; Anderson, "Postcolonial Ecologies of Parasite and Host"; Caduff, *The Pandemic Perhaps*; Dentinger, "Patterns of Infection and Patterns of Evolution"; Greatrex, "'Back to the Jungle'"; Hinchliffe, "More than One World, More than One Health"; Jephcott, "Propagating Visions of a Forest Reservoir"; Honigsbaum, *The Pandemic Century*; Jones and Amramina, "Entangled Histories of Plague Ecology in Russia and the USSR"; Fearnley, *Virulent Zones*; Kelly, Keck and Lynteris (eds.), *The Anthropology of Epidemics*; King, "The Scale Politics of Emerging"; Lakoff, *Unprepared*; Méthot and Dentinger, "Ecology and Infection"; Mitman, "Forgotten Paths"; Nading, *Mosquito Trails*; Roth, "Researching the Ebola Reservoir With the Heuristic of the Fetish in Guinea"; da Silva, "Between Deserts and Jungles".

⁷ Morse, “Regulating Viral Traffic,” 81-82. For an interesting look at how the concept of viral traffic prepared the ground for more recent gain-of-function studies, see Lakoff, “The Routes of Viral Traffic”.

⁸ Beeukwes and Mahaffy, “The Past Incidence and Distribution of Yellow Fever”, 69.

⁹ Steere-Williams, “Endemic Fatalism and Why It Will Not Resolve COVID-19”. On seed and soil metaphors in the early history of germ theory, see Christos Lynteris, “*Pestis Minor*”; Worboys, *Spreading Germs*.

¹⁰ On the spatiality of blame, see Lynteris, “Afterword”; Rudge, “Thinking Beyond the ‘Wild’ Pandemic”.

¹¹ Natalie Porter, “Risky Zoographies”.

¹² Rockefeller Foundation scientists first used the term “spill over” in 1945 to explain the likely cause of an epidemic wave of yellow fever in the State of Goiás in Brazil. See Strobe (ed.), *Yellow Fever*, 496. We do not yet know how early this term was used in outbreak narratives.

¹³ Rockefeller Archive Center (hereafter RAC), Rockefeller Foundation (hereafter RF), International Health Board/Division (hereafter IHB/D), RG5, Subseries 3_495 O, box 214, f 2650, Yellow Fever, Report No. 7548, July 19-October 30, 1920, 45.

¹⁴ Finlay, “El mosquito hipoteticamente considerado como agente de trasmision de la fiebre amarilla”. For the best account of this history, of which there are many, see Espinosa, *Epidemic Invasions*.

¹⁵ *The Rockefeller Foundation Annual Report, 1924*, New-York, 61 Broadway 1925, 91.

¹⁶ Klotz, *Yellow Fever in West Africa*, 15.

¹⁷ For an entry into the vast literature on the history of the Rockefeller Foundation and international health, see Marcos Cueto (ed.), *Missionaries of Science*; Farley, *To Cast Out Disease*; Palmer, *Launching Global Health*; Stepan, *Eradication*.

¹⁸ Klotz, *Yellow Fever in West Africa*, 29.

¹⁹ RAC, RF, IHB/D, RG5, Subseries 3_495 O, box 214, f 2650, Yellow Fever, Report No. 7548, July 19-October 30, 1920, 25.

²⁰ On the history of the the Rockefeller Foundation's yellow fever research program at Yaba, see Megan Vaughan, “A Research Enclave in 1940s Nigeria”.

²¹ RAC, RF, RG1, SG 1.1, Series 300-833, Subseries 495, box 5, f 29, Beeuwkes, Henry, “West African Yellow Fever Commission-Diary,” Vol. 3, 1927, 500.

²² *Ibid.*, On Hagenbeck, see Rothfels, *Savages and Beasts*.

²³ See: Archives de l’Institut Pasteur de Kindia, Guinea, Lieutenant Gouverneur de la Guinée Française A Monsieur le Directeur de l’Institut Pasteur, 1^{er} Juillet 1927, and the exchange between Beeuwkes and Robert Wilbert, Director of Pastoria. I am grateful to Marion Thomas for kindly sharing this correspondence with me.

²⁴ RAC, RF, RG1, SG 1.1, Series 300-833, Subseries 495, box 5, f 29, Beeuwkes, Henry, “West African Yellow Fever Commission-Diary”, Vol. 3, 1927, 501.

²⁵ *Ibid.*, 502.

²⁶ *Ibid.*, 509-516. See, also, Stokes, Bauer and Hudson, “The Transmission of Yellow Fever to *Macacus Rhesus*”.

²⁷ RAC, RF, RG1, SG 1.1, Series 300-833 (FA386B), Subseries 495, box 5, f 29, Beeuwkes, Henry, “West African Yellow Fever Commission-Diary”, Vol. 3, 1927, 538.

²⁸ *Ibid.*, 538-539.

²⁹ Ibid., pp. 539-543. Stokes, Bauer and Hudson, “The Transmission of Yellow Fever to Macacus Rhesus”.

³⁰ Ibid., 617.

³¹ Ibid., 617-621.

³² RAC, RF, RG1, SG 1.1, Series 300-833, Subseries 495, box 5, f 30, Beeuwkes, Henry, “West African Yellow Fever Commission-Diary”, Vol. 4, 1928, 761-765. See also Davis, “Noguchi a Martyr to Medical Research”.

³³ For an important intervention in thinking about the biomedical and technical infrastructures that have shaped cross-species fluid bonding in virus research and vaccine development, see Jain, “The WetNet”. On the contribution of medical anthropology to the turn to multispecies ethnography, see Brown and Nading, “Introduction”.

³⁴ On the biomedical trade in rhesus macaques, see Suri, *Selling Simians*; Ahuja, *Bioinsecurities*.

³⁵ RAC, RF, Field Staff, 1913-1974, Laboratory Records of the West African Yellow Fever Commission, Series 1, African Monkey Books.

³⁶ See, RAC, RF, IHB/D, RG5, Subseries 3_495 O, box 215, f 2662, “Annual Report of the West African Yellow Fever Commission, 1931”. Sawyer and Lloyd, “The Use of Mice in Tests of Immunity Against Yellow Fever”.

³⁷ On the importance of yellow fever’s control in global commerce and trade, see Harrison, *Contagion*.

³⁸ Information on where samples were collected have been compiled from the African Monkey Books and annual reports of the West African Yellow Fever Commission located at the RAC.

³⁹ In Brazil, the Rockefeller Foundation’s focus on typical cases of yellow fever in adults who moved to endemic zones in coastal cities skewed their attention to yellow fever as an urban

disease. Brazilian scientists, in contrast, argued that yellow fever was prevalent in rural areas, particularly among children, whose symptoms were indistinguishable from malaria and typhoid fever. Development of the protection test offered a more precise diagnostic marker, but it did not shift the Rockefeller Foundation's sampling focus away from urban centers in West Africa. See Löwy, "Epidemiology, Immunology, and Yellow Fever"; Ilana Löwy, *Virus, moustiques et modernité*.

⁴⁰ Beeuwkes, Bauer and Mahaffy, "Yellow Fever Endemicity in West Africa"; Beeuwkes and Mahaffy, "The Past Incidence and Distribution of Yellow Fever in West Africa".

⁴¹ On arteries of commerce and disease, see Harrison, *Contagion*.

⁴² On Firestone, see Mitman, *Empire of Rubber*.

⁴³ Tappan, "Wandering Epizootics and Zones of Emergence", 3.

⁴⁴ RAC, RF, IHB/D, RG5, Subseries 3_495 O, box 215, f 2662, "Annual Report of the West African Yellow Fever Commission, 1931", 9.

⁴⁵ *Ibid.*, 2.

⁴⁶ *Ibid.*, 2.

⁴⁷ RAC, RF, RG1, SG 1.1, Series 300-833, Subseries 495, box 5, f 27, Beeuwkes, Henry, "West African Yellow Fever Commission-Diary", vol. 1, 1925, 9.

⁴⁸ "Annual Report of the West African Yellow Fever Commission, 1931," 2.

⁴⁹ RAC, RF, IHB/D, RG5, Series 4, Rockefeller Institute Virus Lab, box 36, f 403, James to Russell, 14 July 1932.

⁵⁰ RAC, RF, IHB/D, RG5, Series 4, Rockefeller Institute Virus Lab, box 36, f 403, Sawyer to Rajchman, January 31, 1933. On commercial air traffic fears and the Cape Town conference, see

Bell, *Frontiers of Medicine in the Anglo-Egyptian Sudan, 1899-1940*; Borowy, *Coming to Terms with World Health*.

⁵¹ RAC, RF, IHB/D, RG5, Series 4, Rockefeller Institute Virus Lab, box 36, f 403, James to Russell, 14 July 1932.

⁵² *Ibid.*

⁵³ RAC, RF, RG1.1, Series 100, International Health Division—Laboratories – History 1942-1950, box 11, f 92, ‘Recollections of the Yellow Fever Laboratory,’ October 1946, 16.

⁵⁴ *Ibid.*, 17.

⁵⁵ *Ibid.*, 18.

⁵⁶ *Ibid.*, 17.

⁵⁷ *Ibid.*, 18.

⁵⁸ Löwy, “Yellow Fever in Rio de Janeiro and the Pasteur Institute Mission (1901-1905)”.

⁵⁹ Mathis, Sellards, and Laigret, “Sensibilité du *Macacus rhesus* au Virus de la Fièvre Jaune”; Sellards and Hindle, “The Preservation of Yellow Fever Virus”. On the development of the French vaccine, see Velmet, *Pasteur’s Empire*.

⁶⁰ Theiler, “Studies on the Action of Yellow Fever in Mice”.

⁶¹ RAC, RF, RG1, SG1.1, International, Series 100, International Health Division—Laboratories – History 1942-1950, box 11, f 92, “The Foundation’s Research Center in New York,” March 1, 1946, and RAC, RF, RG1.1, International, Series 100, International Health Division—Laboratories – History 1942-1950, box 11, f 92, ‘Recollections of the Yellow Fever Laboratory,’ October 1946.

⁶² Animal estimates are from RAC, RF, RG1, SG1.1, International, Series 100, International Health Division—Laboratories –1937-1938, box 10, f 81, Bauer, ‘Monkeys Imported from

India,' April 28, 1938, and RAC, RF, RG1, SG1.1, International, Series 100, International Health Division—Laboratories – Minutes, 1932-1941, box 9, f 77, 'Of Mice and Men – And Others'.

Note these estimates also include animals used in IHD's work on influenza and malaria, as well as yellow fever. On the history of the 17-D vaccine, see Bazin, *Vaccination*; Farley, *To Cast Out Disease*; Theiler, "The Virus".

⁶³ RAC, RF, RG1, SG1.1, Series 300-833, Subseries 477 O, box 3, f 20, Sawyer to Soper, April 8, 1938.

⁶⁴ RAC, RF, RG1, SG1.1, Series 300-833, Subseries 477 O, box 3, f 20, Soper to Sawyer, April 18, 1938, f 20.

⁶⁵ Ibid.

⁶⁶ Bell, *Frontiers of Medicine in the Anglo-Egyptian Sudan*.

⁶⁷ On the history of the vaccine in the AOF, see Velmet, *Pasteur's Empire*.

⁶⁸ Quoted in Bazin, *Vaccination*, 450.

⁶⁹ Bazin, *Vaccination*, 451-452.

⁷⁰ See Stepan, *Eradication*.

⁷¹ On Soper's initial skepticism of "rural" yellow fever and acknowledgement that "yellow fever is not necessarily an urban disease," see Soper et. al., "Yellow Fever Without Aedes Aegypti", 584.

⁷² The Colombia scientist, Roberto Franco, and two of his students investigated an outbreak of fever in the Muzo region in 1907, which they described as "yellow fever of the forests". In 1932, two IHD scientists, Jerome Austin Kerr and Luis Patiño Camargo, confirmed Franco's results, although their publication in English was suppressed by Soper. See: Quevedo et. al., "Knowledge and Power".

⁷³ For an account of the region at the time of oil exploration, see Gilliard, “The Cordillera Macarena”. On the history of the Llanos, see Rausch, *A Tropical Plains Frontier*.

⁷⁴ RAC, RF, RG1, SG1.1, Series 300-833, Subseries 311.0, box 13, f 113, Bugher Service Diary, 2-10.

⁷⁵ RAC, RF, RG1, SG1.1, Series 300-833, Subseries 311.0, box 11, f 100, Bugher to Sawyer, January 27, 1940.

⁷⁶ RAC, RF, RG1, SG1.1, Series 300-833, Subseries 311.0, box 13, f 113, Bugher Service Diary.

⁷⁷ See, Soper, “The New Epidemiology of Yellow Fever”. The term “sylvatic” was not unique to yellow fever. It was also applied to plague during the same time period, as scientists began to consider the possibility of plague existing among certain animal populations in “the wild.” See da Silva, “Between Deserts and Jungles”.

⁷⁸ RAC, RF, RG1, SG1.1, Series 300-833, Subseries 3_495 O, box 3, f 17, Bugher to Warren, Dec. 11, 1943.

⁷⁹ On Bwamba, see Cummiskey, ““An Ecological Experiment on the Grand Scale””.

⁸⁰ RAC, RF, IHB/D, RG5, Routine Reports, Series 3, Routine Reports – West Africa – Yellow Fever Subseries 3_495 O, West Africa (Nigeria) – Yellow Fever, Annual Report, 1945, 14.

⁸¹ Ibid.

⁸² On the history of British forest policy in Nigeria, see Adeyoju, *Forestry and the Nigerian Economy*.

⁸³ Anon., “The Ilaro Forest Reserve Agreement”, *Lagos Weekly Record* (August 3, 1901).

⁸⁴ Elder interview, Ipake, Nigeria, June 14, 2023.

⁸⁵ RAC, RF, IHB/D, RG5, Routine Reports, Series 3, Routine Reports – West Africa – Yellow Fever Subseries 3_495 O, West Africa (Nigeria) – Yellow Fever, Annual Report, 1945, 100.

⁸⁶ Lynteris, “Zoonotic Diagrams,” 465.

⁸⁷ RAC, RF, RG1, SG1.1, Series 300-833, Subseries 3_495 O, box 3, f 18, Bugher to The Honourable, November 28, 1944.

⁸⁸ Taylor, “Epidemiology”, 488.

⁸⁹ RAC, RF, RG1, SG1.1, Series 300-833, Subseries 3_495 O, box 3, f 18, Bugher to The Honourable, November 28, 1944.

⁹⁰ Lynteris, “Zoonotic Diagrams”, 477.

⁹¹ RAC, RF, IHB/D, RG5, Routine Reports, Series 3, Routine Reports – West Africa – Yellow Fever Subseries 3_495 O, West Africa (Nigeria) – Yellow Fever, Annual Report, 1945, 100.

⁹² Interview with Oba Saliu Adekokun Ajibade, Ilobi, Nigeria, June 20, 2023.

⁹³ Ibid.

⁹⁴ d’Avignon, *A Ritual Geology*.

⁹⁵ Interview with Oba Saliu Adekokun Ajibade.

⁹⁶ RAC, RF, IHB/D, RG5, Routine Reports, Series 3, Routine Reports – West Africa – Yellow Fever Subseries 3_495 O, West Africa (Nigeria) – Yellow Fever, Annual Report, 1945, 5.

⁹⁷ RAC, John C. Bugher Papers, box 1, f 10, Kumba Diary Notes. For an excellent account of the Kumba experiment, see Vaughan, “A Research Enclave in 1940s Nigeria”.

⁹⁸ RAC, John C. Bugher Papers, box 1, f 12, “Studies in the Cameroons”.

⁹⁹ RAC, RF, RG1, SG1.1, Series 300-833, Subseries 3_495 O, box 3, f 23, Bugher to Warren, October 13, 1948.

¹⁰⁰ Taylor, “Epidemiology,” 466.

¹⁰¹ Bugher, “Jungle Yellow Fever”.

¹⁰² Marr and Cathey, “The Yellow Fever Vaccine Misadventure of 1942”.

¹⁰³ See, e.g., visualizations in Allen et. al., “Global Hotspots and Correlates of Emerging Zoonotic Diseases”. The term “hotspot” first originated among conservation biologists in the 1980s to prioritize and safeguard regions with high endemicity and biodiversity in an effort to stave off mass species extinction. It was then taken up by disease ecologists in the 2000s to identify high risk areas of emerging infectious disease threats where increased disease surveillance would allegedly prevent the next pandemic. Key individuals and institutions bridged both communities. For the two most cited papers, see Myers et. al., “Biodiversity Hotspots for Conservation Priorities”; Jones et. al., “Global Trends in Emerging Infectious Diseases”. On hotspots, see Brown and Kelly, “Material Proximities and Hotspots”.

¹⁰⁴ See, e.g., Fairhead, “Technology, Inclusivity, and the Rogue”; McGovern, “Bushmeat and the Politics of Disgust”; Pooley et. al., “No Conservation Silver Lining to Ebola”; Roth, “Researching the Ebola Reservoir”.