PLACING GLOBAL SCIENCE IN AFRICA: INTERNATIONAL NETWORKS, LOCAL PLACES, AND VIRUS RESEARCH IN UGANDA, 1936-2000

by
Julia Ross Cummiskey

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Abstract

"Placing Global Science in Africa: International Networks, Local Places, and Virus Research in Uganda, 1936-2000" analyzes six decades in the history of the Uganda Virus Research Institute (UVRI) as a site of knowledge production in order to show the connections between place, scientific research, and the history of Uganda and nearby parts of East Africa. It repositions Africans and African institutions at the core of the narrative, re-centering our understanding of the relationship between global science and African science.

Using archival sources, published articles, and over sixty oral history interviews collected during fifteen months of field work, it explores the events that led to the establishment of Africa, and Uganda in particular, as a center of biomedical research, much of it focused on HIV/AIDS. It adds a historical dimension to a body of literature on medical research in Africa that has been dominated by anthropologists and shows how Uganda was a hub of virus research long before the AIDS epidemic. The project takes advantage of the longevity of the UVRI (previously known as the Yellow Fever Research Institute or YFRI and the East African Virus Research Institute or EAVRI) to study the changes and continuities in research practices between colonial, post-colonial, and post-Civil War periods of Ugandan history and to trace changing ideas about the relationship between disease, health, and place; the role of African skilled labor; the place of African institutions in the global community; and the ways African natural and social environments are investigated and represented for different audiences. All of this material serves to refine our understanding of what the "local" of local partnerships in international medical research collaborations signifies and how it shapes major international medical research projects.

Dissertation Committee

Randall M. Packard (thesis advisor)
Jeremy Greene
Graham Mooney
Sara Berry
Pontiano Kaleebu

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Abbreviations

AIDS	Acquired Immune Deficiency Syndrome
ACP	AIDS Control Programme
ARV	Antiretroviral Therapy
CDC	United States Centers for Disease Control (Formerly the Communicable Diseases Center)
CMS	Colonial Medical Service
CMRS	Colonial Medical Research Service
EAC	East African Community (Kenya, Tanzania, and Uganda)
EAVRI	East African Virus Research Institute
EBV	Epstein-Barr Virus
EID	Emerging Infectious Diseases
HIV	Human Immunodeficiency Virus
NIH	National Institutes of Health (United States)
IARC	International Agency for Research on Cancer
ICRF	Imperial Cancer Research Fund
IHD	International Health Division (of the Rockefeller Foundation)
KS	Kaposi's Sarcoma
MISS	Maternal-Infant Supplementary Study
MRC	Medical Research Council (United Kingdom)
NCI	National Cancer Institute (United States)
NLM	National Library of Medicine (United States)
OH	Oral History Interview
OIHP	*Office International d'Hygiène Publique*
PCR	Polymerase Chain Reaction
PEPFAR	President's Emergency Fund for AIDS Relief (United States)
RAC	Rockefeller Archive Center
RCCS	Rakai Community Cohort Study
RF	Rockefeller Foundation
STD	Sexually Transmitted Disease
TNA	The National Archives (United Kingdom)
UCI	Uganda Cancer Institute
USAID	United States Agency for International Development
UTM	Universal Transverse Mercator
UVRI	Uganda Virus Research Institute

VCP	Viral Cancer Program
WHO	World Health Organization
YFRI	Yellow Fever Research Institute

Introduction

The Uganda Virus Research Institute

Africa has long been associated with infectious disease. Recent events appear to have confirmed longstanding suspicions about the pervasiveness of infectious diseases, both familiar and novel, in Africa. From 2012-2013 it was impossible to discuss travel in any part of Africa, however distant from the epicenter of the Ebola outbreak, without answering questions about risk of infection. In 2014, when news first broke that the epidemic of microcephaly in infants born in Latin America might be linked to Zika virus—a previously little-known mosquito-borne virus first identified in 1947 in Uganda—the disease was quickly identified as an African disease that had "escaped" to the Americas. Almost immediately, a chorus of voices began asking why so many viruses seem to come from Africa and, even more specifically, Uganda. The short answer is, as David Serwadda and others have stated, because scientists have been looking for them in Uganda for a long time. This thesis is the long answer.

When the Zika Forest virus appeared on the radar of Americans with the link between infection and microencephaly in Latin America, the *New York Times* revisited the discovery of the virus at the Yellow Fever Research Institute (YFRI) in 1947 under the headline "In a Remote Ugandan Lab, Encounters with Zika and Mosquitoes Decades Ago".[1] That "Remote Ugandan Lab" is the Uganda Virus Research Institute (UVRI). Calling the laboratory remote only makes sense from a particular frame of reference. Even when the Institute was founded in 1936, Entebbe was a hub of international travel. Today, only 40km along a crowded highway from Kampala, and host to a United Nations Regional Service Centre serving approximately 16,000 personnel, Entebbe hardly feels remote.[2]

[1] Josh Kron, "In a Remote Ugandan Lab, Encounters with Zika and Mosquitoes Decades Ago," *New York Times* April 6, 2016, http://www.nytimes.com/2016/04/06/world/africa/uganda-zika-forest-mosquitoes.html?_r=2, accessed May 26, 2016. The version of Kron's article in the same day's print issue ran under the headline "Revisiting the African Backcountry Where a Virus First Came to Light."

[2] Even Zika Forest itself is practically a peri-urban space. The sign marking the entrance to the forest and declaring it as the property of the UVRI is a short walk from the Kampala-Entebbe road, the busiest thoroughfare in the country. A

In 1936, the Rockefeller Foundation's International Health Division (IHD), in partnership with the colonial government of Uganda, opened the Yellow Fever Research Institute (YFRI) on the premises of the former Trypanosomiasis Research Institute on a hilltop overlooking Lake Victoria in Entebbe. The Institute, later renamed the East African Virus Research Institute (EAVRI) and then the Uganda Virus Research Institute, has operated continuously since that time. The UVRI is typical of many sites of medical research in Africa in that it is a national institution, under the jurisdiction of the Ministry of Health, but almost entirely funded independently of state expenditure.[3] Historically it has always been at least partly a government institution. At its founding, it was a joint venture of Uganda's colonial government and the Rockefeller Foundation IHD. In 1950, the IHD withdrew its support and the British Colonial Medical Service (CMS) took over responsibility for its staffing and operation. In the late 1950s it came under the jurisdiction of the East African Common Service's Organization in preparation for the transition to Independence at which point it was enveloped in the East African Community.

Today, the UVRI campus includes a main building, constructed in the early 20th century in which the core staff (those employed by the UVRI-Ministry of Health) offices are located. These have been unevenly updated over the past several decades to accommodate increased power requirements, telephone lines, internet connections, etc. They are not air conditioned, though many open windows provide a cool breeze on most days. Only steps away is the shiny new building housing the Uganda offices of the United States Centers for Disease Control and Prevention (CDC)—a multi-story, air conditioned facility that wouldn't be out of place in any major American city. It is located behind and a bit below the original building on the hill so that

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university is on the far side of the highway and between the forest and the highway lie a brightly-painted primary school and several small *shambas*. Only ten hectares in size, the remaining forest is routinely encroached upon by human activity in the form of recreation, dumping of waste, and extraction of resources. Tamie Jovanelly, Julie Johnson-Pynn, and James Okot-Okumu, et al., "Pioneering water quality data on the Lake Victoria watershed: effects on human health," *Journal of Water and Health* 13, no. 3 (2015): 920-930.

[3] P. Wenzel Geissler, "Introduction: A Life Science in Its African Para-State," in *Para States and Medical Science: Making African Global Health*, P. Wenzel Geissler, ed., (Durham: Duke University Press, 2015), 1-44.

from the entrance to the campus the colonial building still dominates the view, but a visitor could be excused for assuming that this is the most important building.[4]

To date, there has been virtually no historical account of the work of the Institute. It appears briefly in accounts of the Rockefeller Foundation's International Health Division[5] and in works on the histories of diseases such as yellow fever and Burkitt's lymphoma.[6] In 2002, then-director Sylvester D. K. Sempala published a three-page profile of the Institute and its history in the journal *TRENDS in Microbiology*.[7] At various points, the Annual Reports of the Institute have summarized and reflected on the Institute's history and its significance. Recently, with viruses discovered at the Institute hitting the headlines in North America (West Nile and Zika) journalists have "rediscovered" the Institute and its work.[8] But there has been no sustained historical study of the Institute.

In this dissertation, I consider episodes in the history of the Institute in order to explore themes common to the history of science and medicine and African history. The first of these themes is the position of biomedical research in the history of Africa and, reciprocally, the role of Africa and Africans in the history of biomedical research. This includes the notion of Africa as a laboratory for various disciplines and the processes that led to Africa, and Uganda in particular, as a site of intensive research on HIV/AIDS in the late 20th and early 21st centuries. Secondly, the dissertation considers the significance of place as a category of historical analysis. Place, and imagination of place, helps us to understand what is unique or particular about virus research in Africa, or more specifically, Uganda. Third, the dissertation attends to the tension between

[4] In this respect and many others, the UVRI resembles the site described by P. Wenzel Geissler in "What Future Remains? Remembering an African Place of Science," in *Para-States and Medical Science: Making African Global Health*, P. Wenzel Geissler, ed. (Durham: Duke University Press, 2015): 142-175.
[5] John Farley, *To Cast Out Disease: A History of the International Health Division of the Rockefeller Foundation (1913-1951)* (Oxford: Oxford University Press, 2004).
[6] George Strode, ed., *Yellow Fever* (New York: McGraw-Hill Book Company, Inc., 1951).
[7] Sylvester D. K. Sempala, "Institute Profile: The Uganda Virus Research Institute," *TRENDS in Microbiology* 10, 7 (2002): 346-348.
[8] Andrew Green, "Uganda Discovered the Zika Virus. And the Solution for it," *Foreign Policy* February 10, 2016, http://foreignpolicy.com/2016/02/10/uganda-discovered-the-zika-virus-and-the-solution-for-it/, accessed May 26, 2016; Josh Kron, "In a Remote Ugandan Lab, Encounters with Zika and Mosquitoes Decades Ago," *The New York Times,* April 6, 2016, http://www.nytimes.com/2016/04/06/world/africa/uganda-zika-forest-mosquitoes.html?_r=2, accessed May 26, 2016.

locality and universality or globality as it played out in the work of researchers at the Institute in different periods. Finally, the dissertation traces the changing status of African expertise and skill in the work of the Institute in the colonial, post-colonial, and late 20th century periods.

Health, Healing, and Medical Research in the History of Africa

Historical work on health, healing, and medical research has yielded important insights into African history. The earliest accounts of colonial medicine were consistent with colonial apologetics, emphasizing the benefits that Western medicine held for colonized peoples though acknowledging that other aspects of colonial projects were detrimental. A later generation of historians of medicine soon countered with the argument that, far from being an apolitical gift to colonized subjects, colonial medicine was in fact a tool of colonial states used to make the tropics safe for European settlers and offering little in the way of material benefits to indigenous populations.[9] Some historians went further and argued that colonial medicine, when it did impact indigenous people, was less about improving their health than about disciplining their minds and bodies; in other words it was part and parcel of attempts to impose hegemonic colonial rule.[10] Other historians portrayed medicine and public health as "battlefields" where imperial administrators and indigenous populations contested ideas about health, governance, and power.[11] Most recently, medicine in the colonial period and beyond has been interpreted as one of many sites in which we can see the dynamic exchange of ideas and knowledge about bodies, the environment, governance, expertise, and community. For example, Deborah Neill has argued that in Africa, tropical medicine as an "epistemic community" linked European doctors and scientists from different, even competing, countries in tightly-knit networks through which shared ideas

[9] David Arnold, ed., *Imperial Medicine and Indigenous Societies* (Manchester, England: Manchester University Press, 1988); Roy M. Macleod and Milton J. Lewis, eds., *Disease, Medicine, and Empire: Perspectives on Western Medicine and the Experience of European Expansion* (London: Routledge, 1988).
[10] David Arnold, *Colonizing the Body: State Medicine and Epidemic Disease in Nineteenth-Century India* (Berkeley: University of California Press, 1993).
[11] Megan Vaughan, *Curing their Ills: Colonial Power and African Illness* (Stanford: Stanford University Press, 1991).

about disease, the relationship between place and disease, and the best methods of disease control traveled.[12]

Other historians have pointed to the entanglements between medical research, economics, and governance. Randall Packard's book on tuberculosis in the South African mines implicated medical researchers in the larger enterprise of resource extraction and exploitation of African labor.[13] Luise White's focus on the blood-collection practices of a variety of medical workers and researchers highlighted the connections between those practices and the larger extractive enterprises of colonial states.[14] Her work emphasized the importance of taking seriously African discourse around medical research practices and, like Packard, considering them in the context of the politics and economies of colonial projects.

Over the last several years, a growing amount of historical and ethnographic attention has focused on the issues, practices, and consequences of postcolonial global health projects in Africa. This newest generation of histories of medicine in Africa comes from individuals trained in the history of science and medicine who have been busily situating post-colonial medical projects in Africa in the context of global pharmaceutical markets, the outsourcing of medical experimentation, the competition between infectious and non-infectious and acute and chronic diseases for resources, and the paradigm of emerging infectious diseases.[15]

Recent historical and ethnographic work has also emphasized the experimental or research-driven aspects of biomedical practice in Africa.[16] A common theme of these works is

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[12] Deborah Neill, *Networks in Tropical Medicine: Internationalism, Colonialism, and the Rise of a Medical Specialty, 1890-1930* (Stanford: Stanford University Press, 2012).
[13] Randall Packard, *White Plague, Black Labor: Tuberculosis and the Political Economy of Health and Disease in South Africa* (Berkeley: University of California Press, 1989).
[14] Luise White, *Speaking with Vampires: Rumor and History in Colonial Africa* (Berkeley: University of California Press, 2000).
[15] Adriana Petryna, *When Experiments Travel: Clinical Trials and the Global Search for Human Subjects* (Princeton: Princeton University Press, 2009); Julie Livingston, *Improvising Medicine: An African Oncology Ward in an Emerging Cancer Epidemic*, (Durham: Duke University Press, 2012); Kristin Peterson, *Speculative Markets: Drug Circuits and Derivative Life in Nigeria* (Durham: Duke University Press, 2014); Abena Osseo-Asare, *Bitter Roots: The Search for Healing Plants in Africa* (Chicago: University of Chicago Press, 2014).
[16] Maureen Malowany, "Unfinished Agendas: Writing the History of Medicine in Sub-Saharan Africa," *African Affairs* 99, no. 395 (2000): 325-49; Melissa Graboyes, "Incorporating Medical Research into the History of East Africa," *International Journal of African Historical Studies* 47, no. 3 (2014): 378-399.

emphasizing the connectedness of Africa to the rest of the world and challenging earlier portrayals of Africa as cut off from global networks and exchanges. One of the guiding metaphors in these works is Africa as a laboratory.[17] They suggest that American and European experts in a variety of fields have used sites in Africa to test experimental principles, methods, and substances since the early colonial period. On the one hand, the metaphor emphasizes the otherness of Africa, a place that is isolated from the complexities of life in the modern world and amenable to great levels of manipulation and control. On the other hand, the metaphor highlights the implicit acknowledgment that relationships and phenomena observed in Africa should have some relevance in other parts of the world.

This project builds on and complicates narratives of Africa as a laboratory. While the metaphor of a laboratory suggests a number of constructive ways of thinking about how Africa has been made into a site of knowledge production, it overemphasizes the degree of control aspired to, let alone achieved, by many research projects. At the same time, by making all sites of knowledge production metaphorical laboratories, it erases the specifics of actual laboratories, like the one in Entebbe, and the ways in which they aspired to placelessness in the tradition of the Latourian laboratory that produced facts untainted by traces of their origins.[18] The situation of an actual laboratory in Entebbe was significant for, as Crane argues, "Laboratories … are significant not simply in their ability to translate patient bodies into scientific data but also in their physical locations. The geography of laboratories is the geography of scientific networks."[19]

I propose that, like the Institute itself, Uganda has been constructed as a combination of a laboratory and a field site—a place where scientists can alternately emphasize and capitalize on the artificiality and the naturalness of disease patterns and experimental sites. The history of the

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[17] Helen Tilley, *Africa as a Living Laboratory: Empire, Development, and the Problem of Scientific Knowledge, 1870-1950* (Chicago: University of Chicago Press, 2011); Lundy Braun and Evelynn Hammonds, "Race, Populations, and Genomics: Africa as Laboratory," *Social Science & Medicine* 67, no. 10 (2008): 1580-1588.
[18] Bruno Latour and Steve Woolgar, *Laboratory Life: The Construction of Scientific Facts* (Princeton: Princeton University Press, 1986).
[19] Johanna Tayloe Crane, *Scrambling for Africa: AIDS, Expertise, and the Rise of American Global Health Science* (Ithaca: Cornell University Press, 2013), 105.

Institute's field work is connected to larger questions about the knowledge and governance of place in colonial and post-colonial Uganda. As Lynette Schumaker wrote, "The field science perspective brings to the history of colonial science in Africa the ability to ground that science in its African context and thus to understand what is African about science in Africa."[20] This perspective is not limited to the colonial period. Increasingly historians and social scientists interested in global health and medical research have inquired about what is African about global health in Africa. They have emphasized the ways that technologies and methods intended for universal use get modified, reinterpreted, and repurposed by African users.[21] Examining the field sites of global health research in Africa offers the opportunity to consider the ways that Africa and Africans have shaped biomedicine in the 21st century. Thinking of Africa as a field site as well as a laboratory for biomedical research also highlights the ways that research projects have made and remade publics and other forms of community in Africa.[22]

The Institute also provides the opportunity to add a historical perspective to recent ethnographic work on the "scramble" for field sites, especially in Uganda, in which to conduct AIDS research. Africa, and Uganda in particular, have been identified as sites of increasing competition among researchers in Europe and the United States "for relationships with Ugandan researchers and their patients."[23] To paraphrase an apt definition of primatological field sites, Uganda has become a place that answers questions about AIDS.[24] Most accounts date this development to the early days of the AIDS epidemic and the alacrity with which the Museveni

[20] Lynette Schumaker, "A Tent with a View: Colonial Officers, Anthropologists, and the Making of the Field in Northern Rhodesia, 1937-1960," *Osiris* 11 (1996): 238.
[21] Marianne de Laet and Annemarie Mol, "The Zimbabwe Bush Pump: Mechanics of a Fluid Technology" *Social Studies of Science* 30, no. 2 (2000): 225-263; Caroline H. Bledsoe, *Contingent Lives: Fertility, Time, and Aging in West Africa* (Chicago: University of Chicago Press, 2002); Duana Fullwiley, "Discriminate Biopower and Everyday Biopolitics: Views on Sickle Cell Testing in Dakar," *Medical Anthropology* 23 (2004): 157-194.
[22] Vinh-Kim Nguyen, *The Republic of Therapy: Triage and Sovereignty in West Africa's Time of* AIDS (Durham: Duke University Press, 2010); Ann Kelly, "Progress of the Project: Scientific Traction in The Gambia," in *Differentiating Development: Beyond an Anthropology of Critique*, ed. Soumhya Venkatesan and Thomas Yarrow (New York: Berghahn Books, 2012), 65-83; Ruth Prince, "Situating Health and the Public in Africa: Historical and Anthropological Perspectives," in *Making and Unmaking Public Health in Africa: Ethnographic and Historical Perspectives*, ed. Ruth Prince and Rebecca Marsland (Athens, Ohio: Ohio University Press, 2014), 1-51.
[23] Crane, *Scrambling for Africa*, 102.
[24] Amanda Rees, "A Place that Answers Questions: Primatological Field Sites and the Making of Authentic Observations," *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences* 37, no. 2 (2006): 311-333.

administration acknowledged the crisis and invited researchers to come do something about it.[25] I argue that it is part of a much longer history. In this project I demonstrate that in fact, Uganda was deliberately made into a place that answered questions about viruses more generally, decades before AIDS was discovered.

Space, Place, and the Geographic Imagination in African Science

At the entrance of the Uganda Virus Research (UVRI) in Entebbe hangs an old map, brittle inside its frame and darkened with age. It shows the West Nile District of northwestern Uganda and is overlaid with a number of red dots. The first time I visited UVRI in 2012 I barely noticed it. But over the following several years, as I encountered more and more maps in institutional archives, publications, personal papers, and current project offices I was forced to recognize the centrality of maps in the Institute's work. Having prepared for the project with a course of readings on laboratory technique and studies, why was I suddenly in need of a crash course in geography and cartography?

The ubiquity of maps in the materials related to the work of the Institute over the past 8 decades indicates that place and spatiality as an analytic framework was critical to the ways in which virus researchers understood and conducted their work in Uganda as well as a historical understanding of the significance of the Institute and its work. Maps functioned as a way for researchers to consolidate their knowledge of the various factors they believed to be related to disease, to communicate their findings to outside audiences, and to make findings from particular sites generalizable to larger types of spaces. Map-making in the pursuit of knowledge about viruses is a perfect example of "knowledge-making itself as a form of communicative action."[26] But the maps are also key to a historical understanding of the ways in which medical researchers created ways of knowing places in Africa that interacted with and sometimes conflicted with

[25] Jan Kuhanen, "The Historiography of HIV and AIDS in Uganda," *History in Africa* 35 (2008): 301-325.
[26] James A. Secord, "Knowledge in Transit," *Isis* 95, no. 4 (2004), 661.

existing ways of knowing places. The task of making knowledge about viruses in Uganda (and elsewhere in East Africa) was inseparable from the task of making knowledge of particular places in Uganda.

Place, and imagination of place, helps us to understand what is unique or particular about virus research in Africa, or more specifically, Uganda. Literature in the field of the history of science and medicine has firmly established the importance of place for understanding the practices, priorities, and even findings of medical research projects. Sometimes called the "spatial turn,"[27] this emphasis incorporates an awareness that it is not just the *where* that matters when it comes to place, but also the *what* and the *who*.[28] Place is made meaningful by consideration of the "actants" who occupy it and their expectations, goals, and anxieties.[29] With this in mind, this project considers the significance of the Institute as a place of knowledge production by looking at the people conducting research and the tools they used, their objectives, the obstacles they faced, and the strategies they employed. In fact I am looking at three types of places: the laboratory itself, which like labs everywhere aspired to a placeless space-like quality that would make the results it produced travel seamlessly to other locations; the field sites where the Institute collected data and raw materials and observed ecological systems; and Uganda as a place in which people lived, worked, and sometimes died, a place which was variously known by Africans and Europeans in their distinct "socio-spatial imaginaries".[30] Each of these places had to be made and re-made to suit the goals of scientists, politicians, and others occupying the spaces.[31]

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[27] Diarmid A. Finnegan, "The Spatial Turn: Geographical Approaches in the History of Science," *Journal of the History of Biology* 41, no. 2 (2008): 369-388; Charles W. J. Withers, "Place and the 'Spatial Turn' in Geography and in History," *Journal of the History of Ideas* 70, no. 4 (2009): 637-658.
[28] John Agnew, "Space and Place," in *The SAGE Handbook of Geographical Knowledge*, ed. John Agnew and David Livingstone (London: SAGE Publications, 2011), 316-330.
[29] Agnew, "Space and Place".
[30] Agnew, "Space and Place," 2011.
[31] Latour, "Give me a laboratory."

Many of the better-known contributions to the literature on science and place have come from studies of Enlightenment-era scientific projects. [32] But this spatial turn also has the potential to inform conversations about what is "colonial" about colonial medicine or what is "tropical" about tropical medicine.[33] The connections between geography and history, as Charles Withers observed, include "consideration of … place as social practice and of placing as a process in accounting for the uneven movement of ideas over space and time".[34] I further suggest that the location of the Institute in Uganda did indeed have consequences for its ability to produce, export, and receive knowledge from other places, but that its location was not simply a natural and static quality. The Institute actively constructed the places in which it researched in order to maximize their ability to produce knowledge that would have currency outside Uganda.

Julie MacArthur's recent book on cartography and community in colonial western Kenya expertly links mapping practices with what she calls the "political imagination" of both colonizers and indigenous populations as they established boundaries and constructed both places and ethnicities in the late 19th and early 20th century. This project undertakes a similar task with respect to the scientific imagination, or more specifically the biomedical imagination.[35] Instead of using maps to imagine communities in the sense suggested by Benedict Anderson and MacArthur, the maps made by the Institute allowed it to imagine ecologies which encompassed humans, insects, other animals, and viruses. These maps were critical to the development of a widespread perception of Uganda as both a place riddled with exotic viruses and a place where researching those viruses was possible. For many people in the west, Africa is a place riddled with disease. This pathological imagination of Africa as "the white man's grave" has long roots in

[32] E.g. Simon Schaffer and Steven Shapin, *Leviathan and the Air-Pump: Hobbes, Boyle, and the Experimental Life*, (Princeton: Princeton University Press, 1985); Charles J. Withers, *Placing the Enlightenment: Thinking Geographically about the Age of Reason* (Chicago: The University of Chicago Press, 2007).
[33] Shula Marks, "What is Colonial About Colonial Medicine? And What has Happened to Imperialism and Health?" *Social History of Medicine* 10, no. 2 (1997): 205-219; Warwick Anderson, "Where is the Postcolonial History of Medicine?" *Bulletin of the History of Medicine* 72, no. 3 (1998): 522-530.
[34] Withers, "Place and the 'Spatial Turn'": 639.
[35] Mari Webel took a similar approach in "Ziba Politics and the German Sleeping Sickness Camp at Kigarama, Tanzania, 1907-14," *International Journal of African Historical Studies* 47, 3 (2014): 399-423.

the experience of early European exploration and attempts at settlement. More recently, with the late 20th century discourse about emerging infectious diseases (EID), this imagination has been revived.[36] The UVRI gives us the opportunity to consider close to a century of virus research in Uganda and the ways in which it manufactured places in Uganda that would yield information about viruses, both known and unknown.

The significance of place has evolved as the practices and meaning of virology has changed. During the course of the Institute's existence, the field of virology also underwent major changes. When the Institute opened, the term virus was still sometimes placed in quotation marks and was defined as an infectious agent that was small enough to pass through the finest filters. No consensus had been reached about the precise nature of viruses; they were defined by exclusion.[37] Colloquially it was still common for the term to be applied indiscriminately to unknown pathogens.[38] The tools and procedures for doing virological research were precise but relatively simple. In 1936 the Institute was equipped with a variety of glass laboratory ware, tools for collecting blood, centrifuges, needles, other miscellaneous small instruments, and, most importantly, a vast mouse colony. This was the state of the art. By the end of the century virologists hoping to make major breakthroughs required access to delicate instruments costing thousands of dollars to perform polymerase chain reactions (PCR), electron microscopy, etc. Not only can today's virologists see viruses, they can take them apart, modify them, and put them back together. With increasingly sophisticated and expensive technologies applied to virology, vast improvements in the ability to preserve and transport tissue specimens, and the changing research agendas that follow from and drive these developments, the significance of place has changed at the Institute. On the one hand, proximity to areas where viruses can be found is no longer so critical to the location of laboratory facilities. On the other hand, the politics of place

[36] The discourse of emerging infectious diseases suggests that diseases have natural homes from which, due to changing human behavior or the environment (global climate change, etc.), they sometimes "emerge" or escape. Nicholas King, "Security, Disease, Commerce: Ideologies of Postcolonial Global Health," *Social Studies of Science* 32, 5-6 (2002): 763-789; Nicholas King, "The Scale Politics of Emerging Diseases," *Osiris*, 19 (2004): 62-76;
[37] Thomas Rivers, "Recent Advances in the Study of Viruses and Viral Diseases," *JAMA* 107, no. 3 (1936): 206-210.
[38] Rivers, "Recent Advances".

continue to influence decisions about where science can and should be conducted. Researchers continue to make arguments about the importance of locating virus research in Uganda, though on different grounds in the 21st century than those advanced in the early decades of the 20th century.

The Local and the Global in Longitudinal Perspective

Historians have demonstrated the interconnectedness of early international health programs and colonial health and medicine.[39] Randall Packard's history of global health argues that colonial forms of knowledge constituted the foundation upon which later international and global health practice and science were built.[40] But there are few historical studies that span the successive periods. This dissertation aims to connect some of the concerns of these different groups of scholars by taking a longitudinal perspective. The history of the UVRI allows me to consider both the continuities and the changes in the ways that scientific research was practiced and transmitted under the colonial regime, the post-colonial period of international health, and the late 20th century dawn of "global medicine". I argue that the Institute reveals the role played by geographic pathology (and other overlapping efforts to link place and disease like medical geography, etc.) in the transition from a tropical hygiene and medicine paradigm to an international health paradigm and further to the paradigm of global health.[41] These three paradigms have overlapped and been invoked in sometimes confusing and inconsistent patterns. But, as Vincanne Adams recently wrote, "The shift from *international health development* to *global health* in the sixty-year-old postcolonial infrastructure of transnational health aid is not a

[39] Warwick Anderson, *Colonial Pathologies: American Tropical Medicine, Race, and Hygiene in the Philippines* (Durham: Duke University Press, 2006); Tilley, *Africa as a Living Laboratory*, Neill, *Networks in Tropical Medicine*, Steven Palmer, *Launching Global Health: The Caribbean Odyssey of the Rockefeller Foundation* (Ann Arbor: University of Michigan Press, 2010); Sunil Amrith, *Decolonizing International Health: India and Southeast Asia, 1930-65* (New York: Palgrave Macmillan, 2006); Nancy Leys Stepan, *Eradication: Ridding the World of Diseases Forever?* (Ithaca: Cornell University Press, 2011).
[40] Randall Packard, *A History of Global Health: Interventions into the Lives of Other Peoples* (Baltimore: Johns Hopkins University Press, 2016).
[41] This ancestry of global health is from Jeffrey Koplan, T. Christopher Bond, Michael H. Merson, et al., "Towards a Common Definition of Global Health," *Lancet* 373, no. 9679 (2009): 1993-1995. See also Packard, *A History of Global Health*.

simple case of new bottles for old wine."[42] Nor was the transition from tropical medicine to international health merely rhetorical, though it did roughly coincide with the attempt to reframe colonial medicine as international and thus less weighted with the baggage of imperialism that was losing favor in the decades after the Second World War.

In addition to shedding light on the international or global scale of biomedical research, the history of the UVRI offers a lens through which to consider the local history of Entebbe and the people that inhabited the Institute. One of the advantages of studying the history of an Institute that has persisted over the long-term is that it gives us the opportunity to explore the lives of everyday Ugandan people during periods best known for the acts of a few exceptional individuals, such as Idi Amin in the 1970s. Documents in the UVRI's archive offer glimpses into strategies for surviving in periods of shortage, such as the records of the sugar-buying clubs formed at the Institute in the 1970s and '80s. Employee files with resumes, photographs, and correspondence put faces and partial biographies to the abstract phenomenon of Africanization in the 1960s and '70s. Correspondence between the Institute, Makerere University, the Ministry of Health, international donor organizations and partners, and other peer institutions shed light on the workings of intra- and international cooperation, negotiation, and competition.

Global or transnational history is in vogue. The tension between the local and the global has preoccupied scholars in recent years. It has even led to the growing adoption of the neologism "glocal" in health as well as studies of religion, literature, and economics.[43] By focusing on one set of locales connected to the Institute, I am critically examining the category of "local", asking who defines the local in contrast with the regional, the national, the international or the global; what they know about the locality; and what the significance of the local is for studies of different viral diseases in different periods. This project engages simultaneously with the work on social studies of science, the ethnographic work on international health research programs, and the

[42] Vincanne Adams, ed., *Metrics: What Counts in Global Health* (Durham: Duke University Press, 2016), 1.
[43] A Google search for the term "glocal" gave about 410,000 results on May 13, 2016. According to Google ngram, the term rose in prominence starting in 1960 and grew steeply in the 1990s and early 2000s.

political and social history of Uganda. In short, it attempts to situate the historical work of the virus research institute in Entebbe into its geographic, social, and political, context.

In this project I look at the ways in which the local and the global existed in tension with one another in a very real way for historical actors at the Institute as well as being a dilemma for the historian. As Latour wrote, "The global is part of local histories."[44] In this study, the Institute in Entebbe is implicated in "a series of transnational processes in which the histories of diverse places become connected and interdependent."[45] Moreover, this dissertation challenges the model of assuming that "local partners" in global health research are African and "international partners" are North American or European. Scientists at the Institute self-consciously made themselves into both international and local partners to further particular aims at particular moments in time and often inhabited both roles simultaneously. Their locality or internationality was not a simple function of their location or national origin, but had to be constructed and performed with demonstrations of particular forms of knowledge, ability to access both global and local networks, and fluency in the various vernaculars of the different groups they needed to win over. This work was akin to the boundary work performed by scientists as described by Gieryn, who have to police the lines between the scientific and the non-scientific because the distinctions are not always self-evident.[46] As scientists, the Institute's researchers had to distinguish their labor from that of unskilled technicians and assistants, amateur naturalists, and professionals whose expertise in forest and game management might overlap with their own. They also had to make strategic decisions about when and how to represent themselves as local or not. These decisions, and the advantages or disadvantages of locality, were contingent upon the changing politics of Uganda as a protectorate, an independent state, a failed state, or a state in crisis. They were also contingent on the agenda of the person at any point in time: whether he was

[44] Bruno Latour, "Spheres and Networks, Two Ways to Reinterpret Globalization," *Harvard Design Magazine* 30 (2009): 142.
[45] Lynn Hunt, "Reframing History," *The Chronicle of Higher Education* August 11, 2014, http://chronicle.com/article/Reframing-History/148175/.
[46] Thomas F. Gieryn, "Boundary-Work and the Demarcation of Science from Non-Science: Strains and Interests in Professional Ideologies of Scientists," *American Sociological Review* 48, no. 6 (2014): 781-795.

making a claim on resources controlled by an outside agency; making a claim for participation in international conferences, publication, or policy; or seeking participants for research trials. In the 1930s and 40s, the YFRI's position was only precariously international and its researchers went to great lengths to emphasize their cosmopolitanism. In the 1960s and 70s, with Uganda's efforts to Africanize its civil service and research infrastructure, locality as something other than race had to be emphasized by expatriate scientists. In the 1990s, in the context of the AIDS crisis, scientists dealing with sensitive questions of health, sexuality, and politics, emphasized their locality, their identification with the subjects of their research, and their investment in the greater interests of the Ugandan public. But in each of these periods, there were compelling reasons to be both local and international.

One of the chief aims of this project is to historicize the category of "local partner". This serves to nuance our understanding of what the "local" of local partnership signifies and how it shapes major medical research projects.[47] Terry Eagleton wrote that "the rich are global and the poor are local."[48] In global health the tacit assumption is often that the rich partners are global and the poor or "developing" or "underresourced" partners are local. In some cases locality becomes a euphemism for poor and, implicitly, less expert. In many instances, the inclusion of local partners in global health projects is intended to protect the interests of study subjects or at least to maintain an appearance of collaboration. However, most of these constructions reduce the categories of local and global to euphemisms or overly simple binaries. Instead, I take seriously the suggestion that different partners in research have different relationships to the places in which the research takes place and different ways of knowing about those places. Past analyses have privileged the interaction between science and the politics and social networks that characterized institutions

[47] There is excellent ethnographic literature interrogating the significance of the "partner" part of the term. Johanna Tayloe Crane's ethnographic study of AIDS research projects in Uganda calls into question the presumed distinction between the "partnerships" of contemporary global health and the relationships that characterized research projects in earlier periods and fields termed tropical medicine or international health. As Crane says, "the espousal of partnership—while a noble aspiration—runs the risk of obfuscating both the enduring and novel forms of inequality that shape the transnational relations of global health." Crane, *Scrambling,* 170.
[48] Terry Eagleton, *After Theory* (New York: Basic Books, 2003), 22.

based in the global north, primarily the U.S. and Western Europe. The example of the Virus Research Institute in Entebbe shows that the social and political context in which the "local" partner institutions operate is equally significant for understanding the outcomes of cross-national research projects.

Recent scholarship has shed light on the changes in the character, intentions, and methodologies of what is now called global health or, as Randall Packard has defined it, "interventions into the health of other peoples".[49] For the most part these historical and anthropological studies have looked at the changing nature of the institutions, organizations, and individuals from donor countries that have designed and implemented the programs. But the changing nature of the local or recipient partners has been less well understood. How have the institutions, organizations, and individuals in recipient countries, specifically Uganda, responded to, anticipated, or driven changes in the work of colonial, tropical, and global health? This dissertation uses the example of the UVRI and the analytic of place to explore how the local partners in colonial, international, and global health initiatives represented themselves as appropriate recipients of outside money for the purposes of medical research. The ability of the Institute to endure changing scientific, political, and social tides stemmed in part from its ability to translate place-specific local knowledge into data with currency in the international health market.

The "international" and the "local" don't always map onto the expected people and places.[50] In the 1930s and 40s, American and British researchers stationed in Entebbe were resolutely international in their outlook and understanding of the source of their own expertise. But as some of them established lengthy careers in Entebbe and claimed expertise in forms of knowledge that were highly specific to particular localities in Uganda, they began to identify as

[49] Packard, *A History of Global Health*.
[50] Ruth Prince has described the way that Kenyans "map" categories of local and global onto the therapeutic landscape in Kenya. "Precarious Projects: Conversions of (Biomedical) Knowledge in an East African City," *Medical Anthropology* 33, no. 1 (2014) 68-83.

local experts vis-à-vis their London- or New York-based colleagues. Uganda-based British scientists in the 1960s were "local" with respect to their collaborators in London and New York. Ugandan-born scientists in the same period were seen as limited by their locality until they succeeded in establishing their international credentials through international fellowships and publication in international journals. Even then, there were limits to the stature of their international credentials. Ugandan-born scientists in the 1980s and 1990s had to perform both locality for their US-based funders and internationality for scientific critics. They also had to demonstrate both simultaneously for their research subjects. Comparing oral history accounts, published scientific papers, and unpublished documents makes it possible to see the ways in which these Ugandan researchers experienced these dual identities as coherent or otherwise in different situations.

Sources and Methods

In order to tell a story that spans the colonial, post-colonial, and contemporary eras in Uganda I rely on published literature, archives, and oral history interviews. I have collected nearly 1000 journal and newspaper articles reporting on the research and findings of the Institute's scientists. In addition, I have collected a substantial amount of "grey" literature composed of internal reports to the various public and private agencies that have funded the Institute's work including the Rockefeller Foundation, the World Health Organization, the International Agency for Research on Cancer, and the U.S. National Institutes of Health.

My primary sources are a mixture of formal archival material, memoirs, scientific publications, and unprocessed documents taken out of file cabinets and deposited in "archives". This project capitalizes on the previously unstudied archival holdings of the UVRI. From these combined holdings, I have assembled something akin to what Luise White has called a

"hodgepodge archive" or a "messy archive".[51] The locations of the more formal archives are indicative of the breadth of the network that the Institute has cultivated over the years and the diversity of relationships it has had with other institutions around the world; I have consulted archive collections in Scotland, England, Switzerland, New York, and Uganda. These archives offer insights into the official and the personal lives of the scientists, their families, and the support staff of the Institute. They allow me to portray the Institute not only as a site of scientific research, but also the nexus of social and sometimes political life for several generations of investigators. Most importantly, they shed light on the routine tasks that went into the projects that are described in general terms in the published literature. They reveal the obstacles overcome on the way to the results that are published and the alternate paths abandoned before choosing the strategies reflected in the published work.

In addition to archival work, I conducted interviews with nearly 40 past and present members of the Institute's staff or that of its partners and over 30 interviews with individuals living in the communities where the Rakai Project operates. By including residents of the area in which the HIV/AIDS research is conducted, I was also able to incorporate the perspective of the subjects of scientific research in a way that is far more difficult in the earlier periods.[52] These interviews do not constitute either a random or a representative sample of residents in the villages where the RHSP works or has worked, but they were useful in challenging and confirming some of the conclusions I had reached from my interviews with RHSP employees, the published articles, and unpublished documents. They also drew my attention to the ways in which the project maps and their significance for determining which households were included in study activities actively constituted particular qualities of the local and changed the communities they were studying.

[51] Luise White, "Hodgepodge Historiography: Documents, Itineraries, and the Absence of Archives," *History in Africa* 42 (2015): 309-318.

[52] These interviews were conducted with the assistance of a research assistant, Mr. Charles Ssekyewa, in Luganda and transcribed by two other research assistants, Ms. Eve Kirabo and Ms. Doreen Kibi. Interview participants were selected with the assistance of community outreach workers from the RHSP and local government leaders (LC1s).

Overall, my methodology brings the published, archival, and oral sources into conversation with one another so that the goals of individual scientists, the prerogatives of funding agencies, the contingencies of laboratory and field practice, and the social and political networks at local, regional, and international levels make up a comprehensive picture of scientific life at the UVRI from the late colonial period to the end of the 20th century.

Outline of the Dissertation

This dissertation is organized into three parts of two chapters each. In *Part One: Yellow Fever, 1936-1950*, Chapter One considers the reasons that the colonial government of Uganda and the IHD decided to establish a yellow fever research laboratory in Entebbe and its main goals and activities during the period between its establishment and the isolation of yellow fever virus in western Uganda in 1942. This chapter argues that IHD scientists had to actively create an understanding of Uganda as a legitimate site for the production of knowledge about viruses in terms of its geography, climate, ecology, labor, and politics. They did this in a way that illustrates the tensions between the imperatives of colonial medicine and the discourses of tropical medicine on the one hand and the ascendant language of internationalism and international health on the other. As Packard has observed, yellow fever "is a good place to begin to understand both the nature of colonial medicine and its entanglement with the emerging field of international health."[53] The IHD team worked to establish the laboratory in Entebbe as a space for international research that would not be limited by its location in Uganda but which would profit from its proximity to the kinds of places where yellow fever was believed to be active. At that time, the institute was part of the Rockefeller Foundation's tropical medicine program and the British empire's colonial medical apparatus in East Africa. They were primarily interested in yellow fever, the paradigmatic disease of the international health model that characterized the early years of trans-national medical collaboration to curb the spread of infectious diseases. The growth of

[53] Packard, *A History of Global Health*, 17.

international air travel raised the specter of rapid transport not only of people and goods but also bacteria, parasites, viruses, and the vectors that spread them, and caused public health officials to revisit quarantine regulations and modes of preventing the spread of diseases like yellow fever and the Institute's remit was to study the natural ecology of the virus in order to inform policy decisions. This chapter highlights the tension between the Institute's claims to international status and relevance and its dependence on and adaptation to local labor, environmental conditions, and politics.

Chapter Two describes some of the ways in which one member of the team in particular, Alexander Haddow, embraced the possibilities of the places in Uganda where he conducted research. One of the differences between Haddow and his lab-based colleagues was his willingness to stake his career on his claims to knowledge about a particular set of places, rather than the kinds of techniques that would be applicable more broadly. This chapter covers the period from Haddow's recruitment in 1942 to the closure of the Institute's main field site in Bwamba in 1950, when the Institute's administration was taken over by the colonial government and the IHD withdrew. It argues that the field was a site of diverse forms of knowledge production, including the experimental, and that it required ongoing efforts to discipline nature in order to make it produce scientific results. In order to capitalize on the authority of field research as arising from its quality of "unadulterated reality, just now come upon,"[54] Haddow had to downplay the work he did to render the field legible. But that work was, in fact, substantial. I argue that Haddow's work epitomizes colonial efforts to impose order and discipline on African places colonial officials perceived as chaotic and that this work was critical to the establishment of Bwamba as a site of intensive virus research leading to the discovery of a number of previously unknown viruses.

[54] Thomas Gieryn, "City as Truth-Spot: Laboratories and Field-Sites in Urban Studies," *Social Studies of Science* 36,1 (2006): 5-38, quotation on 6.

After Uganda's Independence in 1962, the Institute had to define its role as an "Africanized" institution and demonstrate that it was still a relevant research center in the rapidly changing field of virology. The shift from imperial tropical medicine to international health represented, for the Institute, a major decrease in the security of its finances and future. As Chakrabarti observed, "tropical medicine developed out of a strong metropolitan involvement,"[55] and while that involvement was sometimes a double-edged sword, declining metropolitan involvement presented challenges for the institute. As one former Institute scientist remarked decades later, the degree to which African research institutes were able to maintain strong ties to institutions in metropolitan centers was directly related to their stability after Independence.[56] Rather than capitalizing on the institutionalized interest of the colonizing government of Britain (and to some extent Belgium) or the economic imperial interests of the RF in tropical medicine, the Institute had to learn how to see and make seen Uganda as a place where questions of interest to international health funders could be answered. Burkitt's lymphoma (BL) offered an opportunity for the Institute to join the burgeoning field of cancer virology. In *Part Two: Burkitt's Lymphoma, 1961-1979*, Chapter Three considers the Institute's investigation of a viral etiology for BL between 1961 and 1969 with the Imperial Cancer Research Fund. The Institute's location in Uganda at the epicenter of lymphoma research made its site an asset rather than a disadvantage at a time when some believed that "Entebbe was finished"[57] as a place for cutting edge virus research. This chapter argues that early attempts to interpret the apparently idiosyncratic distribution of the tumor syndrome as indicative of an arbovirus-related etiology involved multiple ways of relating place and disease informed by models of investigation developed in the yellow fever studies. Chapter Four follows the Institute's cancer investigations to the northwestern Ugandan district of West Nile, where it conducted a series of cohort studies

[55] Pratik Chakrabarti, *Medicine & Empire 1600-1960* (New York: Palgrave Macmillan, 2014), 149.
[56] William H. R. Lumsden, "Impact of Independence and Nationalism on Tropical Medicine," *Bulletin of the New York Academy of Medicine* 51 (1975): 595-607.
[57] Memorandum on the visit of Dr. J. Weir of the Rockefeller Foundation, December 16, 1964, TNA FD 7/1509.

between 1968 and 1979. It shows that efforts to translate the Institute's international standing under expatriate leadership to equal standing under African leadership were curtailed by political developments in Uganda, at the same time that efforts to transform West Nile into an experimental space like Bwamba in an earlier period were similarly interrupted.

Uganda's civil war interrupted most work at the Institute between 1979 and 1986. In *Part Three: HIV/AIDS, 1985-1998,* I discuss how international funding for HIV/AIDS research gave Uganda an opportunity to reanimate its virus research capacity after Yoweri Museveni assumed the presidency in 1986. Chapter Five, "Rakai District was put on the map because of all the deaths," begins with the discovery of a new virus ravaging the Rakai District of Uganda in 1985 and explores the Rakai Project's initial work measuring the impact of the HIV epidemic and its dynamics between 1986 and 1994. This work firmly established Rakai as "a place that answered questions" about HIV. This chapter argues that the concentration of international HIV research in Uganda was facilitated by earlier generations of work constituting Uganda as a constructive place for virus research. Along with the mobilization of vast resources to fight AIDS, the Institute benefited from the new politics of global health which, in an attempt to distinguish itself from the paternalist traditions of international health, emphasized the importance of "local partners". In this period the Institute, and more particularly the Rakai Project, one of the projects affiliated with the Institute, set about making themselves over as the consummate local partners with a rich knowledge of the places in which HIV research was to be conducted and where it was hoped it would have an impact. Chapter Six is an in-depth study of the Rakai Project's community-randomized controlled trial of mass STD treatment for HIV prevention from 1994 to 1998 and argues that the maps used in this study not only made the places and communities in Rakai visible to researchers but actively constituted them. It also shows how the controversial results of this trial forced the project to explicitly defend Rakai as a valid site of HIV knowledge production.

In each generation of virus research at the Institute, scientists investigated and constructed the nature of places in Uganda as sites conducive to the study of viruses. The forms

of investigation possible in the colonial, early post-colonial, and Museveni-era periods of Uganda's 20th century history help historians to understand the relationship between scientific knowledge production, politics, and place that have given rise to the current landscape of HIV/AIDS projects in Uganda and elsewhere in Africa.

Chapter 1
Human Immunity Surveys and Virus Isolation, 1936-1941

Introduction

In 1936, a pair of maps appeared in an article by two Rockefeller Foundation scientists, Wilbur Sawyer and Loring Whitman [Figure 1].[1] One map included large hatched sections indicating the areas of Africa covered by serosurveys over the previous 5 years. Those were largely areas where physicians had observed cases of yellow fever and the investigations were intended to identify the limits of the areas where the disease was endemic. The second, more detailed map showed the results of the most recent survey covering central East Africa. That map indicated the locations where serum was collected, and the proportions of both adults and children who tested positive for yellow fever antibodies in each location. These maps challenged the known clinical picture of the disease in Africa, which included no history of yellow fever outbreaks as far eastwards as these surveys indicated the disease had appeared. This puzzle—how to explain the apparently contradictory evidence of clinical observation and laboratory investigation and what it implied about the nature of yellow fever—prompted a major investment in yellow fever research in East Africa by the Rockefeller Foundation's International Health Division that began in 1936. This research agenda was based in Entebbe, Uganda at the Yellow Fever Research Institute (YFRI) on the premises of the former Human Trypanosomiasis Institute. The decision to establish a laboratory in Entebbe was consistent with the direction Frederick Russell had defined for the IHD before his retirement in 1934, to concentrate on field and laboratory investigations at the expense of health systems development.[2] The YFRI's mission was to outline the extent of yellow fever immunity in East Africa, identify the areas that were vulnerable to yellow fever outbreaks, and decipher the natural history of the virus. These projects were all in service of the larger goal of

[1] Wilbur A. Sawyer and Loring Whitman, "The Yellow Fever Immunity Study of North, East, and South Africa." *Transactions of the Royal Society of Tropical Medicine* 29, no. 4 (1936): 397-412.
[2] John Farley, "The International Health Division of the Rockefeller Foundation: the Russell Years, 1920-1934," in *International Health Organisations andMmovements, 1918-1939,* ed. Paul Weindling (Cambridge: Cambridge University Press, 1995): 203-221.

identifying what, if anything, was preventing the spread of yellow fever eastwards across the continent as far as the coast so that they could, as the Rockefeller Foundation put it, "hold the line against yellow fever." British and American interests were united in hoping to prevent yellow fever, known to be endemic in West Africa and transmitted by *Aedes aegypti* mosquitos, from spreading across the Indian Ocean to South Asia, where no yellow fever outbreaks had ever occurred but where *A. aegypti* were abundant. British colonial officials in particular were anxious to prevent the virus from invading the Indian subcontinent and causing the kind mortality, morbidity, and disruption to trade and commerce that the virus had visited on ports in the Americas.

In this chapter I reposition the YFRI, typically represented as an outpost of the IHD, whose work was centered in New York, at the center of a network of people, practices, and objects. Focusing on Entebbe highlights the tensions between the efforts to incorporate the YFRI into an international space of virus research stretching across the Americas, Europe, Africa, and Asia, and the significant effects of the "local" places in which the YFRI's research was conducted. On the one hand, this chapter considers the YFRI and its research sites as spaces within an international network of scientific knowledge production linking scientists in the Americas, Europe, and the European colonies who sought to create universal knowledge about tropical diseases.[3] Like the facilities in Brazil, the lab in Entebbe was expected to transform blood, liver specimens, and mosquitoes into "quantifiable parameters and codified inscriptions" that would speak the universal nature of yellow fever as well as its local variation and characteristics.[4] People, animals, objects, procedures, and data flowed across vast distances and connected remote locations in a common pursuit of the answers to questions about how yellow fever was transmitted, what factors promoted or impeded its epidemicity and endemicity, and

[3] Deborah J. Neill, *Networks in Tropical Medicine: Internationalism, Colonialism, and the Rise of a Medical Specialty 1890-1930* (Stanford: Stanford University Press, 2012).
[4] Ilana Löwy, "Epidemiology, Immunology, and Yellow Fever: The Rockefeller Foundation in Brazil, 1923-1939," *Journal of the History of Biology* 30, no. 3 (1997): 397-417, quotation on 415.

whether it was likely to spread into previously uninfected parts of the world. On the other hand, the specific qualities of the places where the research was conducted determined what made for a successful investigation in Uganda—what kinds of people thrived there, what kinds of strategies were successful or unsuccessful, and what kinds of objects were required to make yellow fever work possible. I argue that understanding the places where the YFRI worked, particularly in Uganda, is critical to understanding the processes and outcomes of their work. Reciprocally, studying the work of the YFRI offers a unique opportunity to understand the history of late colonial Uganda and the lives and work of Africans who participated in yellow fever research as lab technicians, medical auxiliaries, field assistants, and sources of blood and tissue.

From the beginning, the IHD anticipated that the Institute would eventually be able to undertake a comprehensive investigation into the epidemiology of yellow fever. But initially, as Sawyer wrote to Fred Soper, "The whole emphasis is still centered around isolating and locating the virus."[5] The expectation was that this would not take long. Uganda's relatively well-organized medical infrastructure, the IHD believed, would lend itself to yellow fever investigations and George K. Strode, the Assistant Director of the IHD, confidently predicted "if there is yellow fever in Uganda we should be able to find it without much delay and prove it by isolation of the virus."[6] As it turned out, this would occupy the Institute's energies for its first five years. This chapter will consider the ways in which this goal was approached and the various strategies employed in its pursuit. It begins with an overview of the Rockefeller Foundation's yellow fever program and the developments that preceded the foundation of the YFRI. I will discuss the problem that the YFRI was established to solve—resolving the apparent discrepancy between clinical and laboratory evidence about the geographic distribution of yellow fever and its implications for predicting and controlling the spread of the virus. Then I will discuss the strategies that the Institute employed in the pursuit of that goal, the obstacles researchers faced,

[5] Sawyer to Soper, April 1, 1938, Folder 2, Box 1, Series 477o, RG 1.1, Rockefeller Foundation Records (RF), Rockefeller Archive Center (RAC).
[6] Strode to Sawyer, December 10, 1936, Folder 1, Box 1, Series 477o, RG 1.1, RF, RAC.

including the outbreak of a world war, and the events that led to the resolution of at least one part of the puzzle—whether yellow fever virus was in fact present in East Africa. I will conclude with the Institute's transition to a broader set of investigations intended to tackle the second part of the problem—the epidemiology of the virus's transmission in humans and animals in East Africa. Throughout, I will be highlighting the ways in which place mattered, both to the researchers themselves and for the purposes of understanding their work from a historical perspective.

The Rockefeller's International Health Division and Yellow Fever Research

The International Health Division of the Rockefeller Foundation had been involved in yellow fever research since the Panama Canal raised anxieties about the disease reaching East Asia.[7] In May 1915, the International Health Commission adopted a resolution "to give aid in the eradication of this disease in those areas where the infection is endemic and where conditions would seem to invite cooperation for its control" and appointed General William C. Gorgas as the director of the Commission's yellow fever work.[8] He formed the Rockefeller Foundation Yellow Fever Commission and, despite delays related to the First World War, undertook preliminary steps towards eradicating the disease in Guayaquil, Ecuador—the only place in South America known to have endemic yellow fever.[9] While a decision to expand the work of the Commission to West Africa was reached in 1916, the First World War prevented the expedition from forming until 1920 when the Commission set out to "determine whether the reported yellow fever in that region actually was yellow fever, and, if so, to ascertain whether control measures were feasible."[10] That expedition foundered when Gorgas fell ill and died en route, but in 1925 a new West Africa Yellow Fever Commission headed by Henry Beeuwkes set out to: "(a) to study the characteristics and epidemiology of the disease in West Africa and its relationship to the yellow

[7] Strode, *Yellow Fever*, 12.
[8] Strode, *Yellow Fever*, 14. The International Health Commission (IHC) was established in 1913, renamed the International Health Board (IHB) in 1916 and then renamed the International Health Division (IHD) in 1927. The Rockefeller Archive Center, "100 Years: The Rockefeller Foundation," http://rockefeller100.org/exhibits/show/health/international-health-division, accessed January 17, 2017. In this account I will use the abbreviations for the contemporary names as they occur.
[9] Strode, *Yellow Fever*, 14.
[10] Strode, *Yellow Fever*, 18.

fever of the Western Hemisphere; (b) to isolate the organism that caused the disease; (c) to discover the method of transmission; and (d) to identify those areas in which the disease was continually present."[11]

By 1936, the Yellow Fever Commission had established certain facts about yellow fever. Earlier doubts about whether the disease in West Africa was the same as yellow fever in South America had been largely laid to rest.[12] It was a filterable virus and it could be transmitted by mosquitos from one infected person to another susceptible one.[13] In South America, the mosquito *Aedes aegypti* was the main urban vector, but scientists from the Rockefeller Foundation working in Colombia, most famously Fred Soper, had recently discovered that yellow fever could be sustained in endemic form in animals without infecting humans and was almost certainly transmitted by additional insect vectors as yet unidentified. Soper called this phenomenon jungle yellow fever.[14] The 1936 report of the Rockefeller Foundation observed that while recent developments had "resulted in slight lifting of the veil which previously had covered all but a small part of the epidemiology of disease" and that new findings indicated that yellow fever presented "a darker picture than we had supposed," those same developments were largely the result of improved scientific methods which would permit the Rockefeller Foundation to build on its previous successes.[15] Ongoing research aimed to identify the vector or vectors responsible for yellow fever transmission in Africa and to define the conditions necessary for the perpetuation of the virus in the environment.

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[11] Strode, *Yellow Fever*, 19.
[12] Max Theiler and Andrew W. Sellards, "The Immunological Relationship of Yellow Fever as it Occurs in West Africa and in South America," *Annals of Tropical Medicine and Parasitology* 22: 449-460; Rivers, "Recent Advances."
[13] Rivers, "Recent Advances." A "filterable virus" indicated something that caused disease and was small enough to pass through the smallest filter. At this time the term "virus" was sometimes used indiscriminately to designate any pathogen that couldn't be directly observed.
[14] Fred Soper, *Rural and Jungle Yellow Fever: A New Public Health Problem in Colombia* (Bogotá: Editorial Minerva, 1935).
[15] Rockefeller Foundation, 1936 Annual Report, The Rockefeller Foundation: New York, 15.

Preliminary serosurveys conducted by the West Africa Yellow Fever Commission appeared to show a distribution of YF across Africa that presented "an enigma".[16] While yellow fever immune specimens had been found much further south and east of the known yellow fever endemic region of West Africa, "east of Uganda and on to the Indian Ocean a vast area of Africa appears to be strangely free of the yellow fever virus" despite the abundance of known yellow fever vector mosquitos along Africa's eastern coast.[17] Even more provocatively, while there were positive specimens in Uganda, the mosquito species known to transmit yellow fever were believed to be rare in Uganda. This constellation of research findings led the IHD to state, "Uganda is strategic territory, inasmuch as it seems to present a natural barrier to the spread of the disease. Why? Wherein lies the barrier? Is there some unknown carrier of the virus at work within Uganda and on to the west? And if so, what?"[18] The urgency of the IHD's efforts to answer these questions stemmed from fears that yellow fever would appear in the coastal cities of eastern Africa, multiply rapidly among the profusion of mosquito vectors, cross the Indian ocean, and infect the Indian subcontinent which also had an abundance of vectors but had thus far remained free of yellow fever. These fears were almost identical to those that had followed the sleeping sickness epidemics in Uganda at the beginning of the century.[19] In other words, scientists were expected to establish the nature of the putative (and possibly vulnerable) barrier between yellow fever and the Indian Ocean coastline so that it could be protected.

The 1936 maps of yellow fever disease and immunity distributions suggested two possible scenarios, each alarming in its own way. One possibility was that yellow fever had for some time been present in East Africa but had gone unrecognized by the colonial medical

[16] "Excerpt from Trustee's Confidential Monthly Bulletin," May, 1938, "A Yellow Fever Barrier in Africa," Folder 2, Box 1, Series 477o, RF, RAC.
[17] "Excerpt from Trustee's Confidential Monthly Bulletin," May, 1938, "A Yellow Fever Barrier in Africa," Folder 2, Box 1, Series 477o, RF, RAC.
[18] "Excerpt from Trustee's Confidential Monthly Bulletin," May, 1938, "A Yellow Fever Barrier in Africa," Folder 2, Box 1, Series 477o, RF, RAC.
[19] Maryinez Lyons, *The Colonial Disease: A Social History of Sleeping in Northern Zaire, 1900-1940* (Cambridge: Cambridge University Press, 1992), 71. The IHD Confidential Bulletin of February 1942 described the apparently yellow fever-free but mosquito rich zone picturesquely: "The area [along the Indian Ocean coast of Africa] is thickly infested with Aedes aegypti, but seemingly these mosquitoes found no opportunity to ply their virus-carrying trade." Folder 6, Box 1, Series 477o, RG 1.1, RF, RAC.

authorities. The suggestion by an American agency that British colonial doctors had failed to recognize an epidemic disease under their noses was hardly flattering.[20] The other possibility was that yellow fever was spreading eastwards and that officials could expect to encounter rising numbers of clinical cases in the eastern colonies of Uganda, Kenya, and Tanganyika. In either case the prospect of having to deal with the political and economic consequences of declaring East Africa a yellow fever endemic zone was extremely unattractive. In fact, there was a third possibility, which the IHD was anxious to eliminate: that the newly-developed mouse protection test was an unreliable indicator of true yellow fever immunity and that the maps of serosurvey results did not in fact represent the distribution of yellow fever infection.

The Rockefeller surveys relied on one critical and controversial technique only recently developed, the mouse protection test. While this test appeared to be the ultimate example of the triumph of laboratory epistemology over clinical or epidemiological expertise, the authority of laboratory findings was in fact highly contested.[21] At a meeting of the London Society of Tropical Medicine and Hygiene, supporters and skeptics of this technology spoke about the implications of the survey test results. G. M. Findlay of the Wellcome Bureau of Scientific Research in London, a proponent and user of the test, spoke confidently about a yellow fever endemic zone revealed by antibody tests that stretched across Africa. But Colonel F. P. Mackie, a veteran of Uganda's sleeping sickness commission, expressed reservations:

> It seems very extraordinary to me that silent penetration of new areas in Africa by yellow fever may occur without any clinical evidence of the existence of the disease…It amounts to this: the conclusions [about the endemic areas] are based upon the specificity of the mouse-protection test concerning which, despite its general acceptance, some may have lingering doubts."[22]

To some extent, this disagreement reflected wider tensions between biomedical researchers, whose authority derived from extensive training in metropolitan universities and various tropical

[20] Heather Bell, *Frontiers of Medicine in the Anglo-Egyptian Sudan, 1899-1940* (Oxford: Clarendon Press, 1999), 166.
[21] Bell, *Frontiers of Medicine*, 167.
[22] "Discussion [following Soper's address on the yellow fever situation]," *Transactions of the Royal Society of Tropical Medicine and Hygiene* 32, no. 3 (1938): 328-332, quotation on 326.

settings, and colonial physicians, whose claims to expertise were founded on extended periods of service in a particular colonial territory and incorporation into tightly-woven local networks of government and missionary physicians. Skeptics could reasonably claim that the Rockefeller teams had insufficient knowledge of the local environment in which the specimens were collected to interpret their tests accurately. In order to more fully investigate the situation in Central and East Africa, the IHD decided to open a laboratory in the contested region to conduct more extensive immunological investigations and attempt to isolate yellow fever virus, thereby settling the question once and for all.

Locating the Laboratory

A number of factors contributed to the decision to locate the YFRI in Entebbe. Alternative locations had been explored including the Sudan. But the government of the Sudan was reluctant to allow the investigations to proceed with a laboratory in their territory, fearing that introducing yellow fever virus into the territory for experimental purposes posed a threat, even under controlled laboratory conditions.[23] While Ugandan colonial authorities were cautious about the risks of allowing experiments involving live virus in the Protectorate, they were also sensitive to the danger that the undetected presence of yellow fever transmission posed to them. They were keenly aware of their status as "the Piccadilly Circus of Africa" where strategic lines of traffic converged, and wanted to make sure that yellow fever didn't jeopardize that traffic.[24] Finally, there was a building previously used as a sleeping sickness laboratory on top of one of the hills outside the residential section of town that the government was willing to make available to the IHD for its YFRI. Fred Soper himself, well-known for his yellow fever investigations in South America, inspected the site and declared his opinion that it would be better than alternative locations in Nairobi or Khartoum because of "its isolated position and its proximity to the area of

[23] Bell, *Frontiers of Medicine*, 177.
[24] Excerpt from Strode's diary quoting the governor, March 8, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.

country under investigation, which is readily reached by aeroplane in an hour or two and by motor car in five hours."[25] With some work to make it mosquito proof, add water and power, and renovate some of the space, the building was well suited to its new purpose.[26] Strode was pleasantly surprised by the facilities in Entebbe, noting in his diary that the Institute was "a larger and better place than I had imagined" and calling the location "superb".[27] IHD Assistant Director Andrew Warren likewise described the renovated compound as "a large, excellent structure of 20 rooms that has been adapted to our use … [with] excellent facilities for the mouse colony, monkey cages and guinea-pigs."[28] The goal was to make the YFRI a world-class laboratory that happened to be situated in proximity to the sources of research specimens: Central and East African animals and people. For the purposes of attracting suitable staff, it was also helpful that Entebbe had a climate that many Americans and Europeans found attractive.

Personnel

Staffing the Institute was even more complicated than finding a suitable site. The qualities of the initial group of researchers and subordinate staff, those they shared and those they lacked, give us some insight into the anticipated and actual demands placed on them. The IHD selected Dr. Alexander Francis Mahaffy, known to friends and colleagues as "Tiny", to run the Institute in Entebbe. Mahaffy was a veteran of the West African yellow fever investigations and part of the team that had isolated the Asibi strain of the virus. Before deploying him to Uganda, the IHD sent him to Brazil for two months in order to study the methods used in Rio de Janeiro by Soper and his colleagues to investigate yellow fever.[29] In particular, he was expected to observe the organization and administration of the project, methods for controlling yellow fever

[25] Unsigned draft dispatch, n.d. 1935, Uganda National Archives, J-101-II "Yellow Fever".
[26] Strode diary, March 3, 1937, RG 12, RF, RAC, digitized, accessed Jan. 15, 2015.
[27] Strode diary, March 3, 1937, RG 12, RF, RAC, digitized, accessed Jan. 15, 2015.
[28] Excerpt from Warren's diary, Feb. 8, 1939, Folder 3, Box 1, Series 477o, RG 1.1, RF, RAC.
[29] Sawyer to Farrell, April 25, 1936, Folder 1, Box 1, Series 477o, RG 1.1, RF, RAC.

transmission, the viscerotomy service, and work on the epidemiology of the newly-discovered jungle yellow fever cycle of transmission.[30]

Mahaffy was joined in Entebbe by bacteriologist John Harland Paul, previously stationed in Colombia, and Alexander W. Burke, who was transferred to Uganda from yellow fever work in Brazil. Burke at least was reported to be very pleased with the new posting and got along well with Mahaffy.[31] In a diary entry from July 1936, a few months before the Uganda lab opened, he reported facetiously that over a shared dinner at the staff house during Mahaffy's study visit, he and Mahaffy "had practically all the problems of yellow fever transmission solved."[32] Paul had several years of experience with yellow fever, having worked on it with the IHD in New York, Lagos, Brazil, and Colombia between 1932 and 1936.[33] He was responsible for transporting the lab's supply of rhesus monkeys to Entebbe.[34] J. O. Harper, a government entomologist in Kenya, was seconded to the project in order to conduct surveys of potential vectors in areas where transmission was suspected.[35] Paul was aided in the lab by Mr. E.G. Gibbins of the Uganda Medical Service and 12 African staff members provided additional support as laborers, drivers, messengers, clerks, and attendants.[36] For Europeans, some of these positions proved to be somewhat fluid; in 1939 Gibbins replaced Harper as the project's entomologist before being called up for military service.[37] There was a rigid distinction between so-called skilled and unskilled labor, however, with Africans exclusively limited to the unskilled roles. Consideration

[30] Annual Report for 1936 Paris Office - International Health Division. Section 4: Uganda Yellow Fever Research Institute, Folder 2905, Box 241, Series 700, RG 5.3, RF, RAC. Viscerotomy was a procedure used to retrospectively diagnose cases of yellow fever infection after death from a brief illness consistent with yellow fever. A small instrument (viscerotome) was used to extract a small piece of liver from a human cadaver without requiring a full post-mortem examination and autopsy. The liver section was then analyzed for signs of yellow fever pathology.
[31] Sawyer to Mahaffy, August 17, 1936, Folder 1, Box 1, Series 477o, RG 1.1, RF, RAC.
[32] AWB (Alexander W. Burke) diary, July 6, 1936, Box 57, RG 12, RF, RAC.
[33] Rockefeller Foundation, 1950 Annual Report, New York: Rockefeller Foundation, 19.
[34] GKS (George K. Strode) diary, March 3, 1937, RG 12, RF, RAC, http://dimes.rockarch.org/a1164613-7fc3-41d3-894e-ed8584219f2d, accessed Jan. 15, 2015.
[35] "Annual Report for 1936 Paris Office - International Health Division. Section 4: Uganda Yellow Fever Research Institute," Folder 2905, Box 241, Series 700, RG 5.3, RF, RAC.
[36] "Annual Report for 1936 Paris Office - International Health Division. Section 4: Uganda Yellow Fever Research Institute," Folder 2905, Box 241, Series 700, RG 5.3, RF, RAC.
[37] Excerpt from AJW Diary, Feb. 12, 1939, RAC RF RG1.1 Series 477o Box 1 Folder 3. There was at least one other example of a European in a relatively junior position rising significantly through the ranks at the Institute with additional education and training (Gillett).

of the work they did operating the mouse protection test (more detail on this later in the chapter), however, shows that a great deal of skill was in fact involved in their labor.

The qualities of the places in which research on tropical medicine was conducted had a selective effect on the people who joined and stayed in the networks of tropical medicine. Most Americans and Europeans found Entebbe quite pleasant. Strode described his first impression of the town as "a lovely town, trees everywhere, spacious lawns and brilliant flowers surrounding pleasant looking homes and the great Lake Victoria stretching miles to the south."[38] But not everyone was capable of capitalizing on the opportunities presented by work in Uganda and the IHD struggled to recruit and retain people suited for the work. The first lab man, John Harland Paul failed to thrive in Uganda and was seen as an impediment to the progress of the laboratory work. Strode wrote, "The consensus of opinion is that there is an unusually fine opportunity for research here but that Dr. Paul is not quite up to it and does not fully appreciate it."[39] Of Paul's replacement, Strode continued, "No man not fundamentally a research man would do."[40] Ideally they would build up the lab capacity with "at least one person of broad bacteriological experience."[41] Ultimately the IHD identified two such people: Kenneth C. Smithburn and Thomas Patrician Hughes.

However, choosing staff for the Uganda laboratory involved more than just professional considerations, though those of course were important. The place where the laboratory was located, Entebbe, imposed some constraints on the personnel selection. Men—and they were exclusively men—with school-age children were avoided because the opportunities for educating children were limited in Uganda.[42] They had to be suited to or willing to adapt to life in the tropics and, if they were married, so did their wives. Unmarried men were considered ideal

[38] GKS diary, March 3, 1937, RG 12, RF, RAC, digitized, accessed Jan. 15, 2015.
[39] Strode to Warren, March 20, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.
[40] Strode to Warren, March 20, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.
[41] Sawyer to Soper, April 1, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.
[42] Sawyer to Soper, May 20, 1936, Folder 1, Box 1, Series 477o, RG 1.1, RF, RAC. Smithburn's predecessor at the YFRI, J.H. Paul, was chosen over another possible candidate, Henry Kumm, in part because Kumm had school-age children. Sawyer to Soper, May 1936, Folder 1, Box 1, Series 477o, RG 1.1, RF, RAC.

though, as Warren put it, "Whether a man is married or not would be of no great consequence if the reactions of his wife were favorable. For example Mrs[.] Burke thinks that the Sudan is not a fit place for a white woman and on the other hand Mrs. Mahaffy loves it."[43] One candidate for a position in Entebbe, Bequaert, withdrew himself from consideration because "his wife considers it certain suicide and a form of desertion of his family".[44] Part of Paul's failure to thrive in Entebbe was blamed on his wife who was "bored".[45] A third addition to the YFRI's staff in 1939, epidemiologist Henry Jacobs, didn't last very long. In 1941 Mahaffy complained that he appeared to dislike living in the bush and was frequently ill when working in the field. He suggested that, "his successor should be made to understand that he will have to spend the greater part of his time on safari."[46] Not every scientist with an interest in diseases of the tropics was cut out for life in the forests of western Uganda.

While the IHD's selection of men to send to Uganda demonstrates the real impact of the specific location of the lab, the process of orienting Smithburn to yellow fever work illustrates the degree to which the IHD still understood medical research as occurring in a transnational space in which the particularities of any given place were no more significant than the universal principles of virology. Hughes had seven years of experience with yellow fever at the IHD labs in New York, but Smithburn's first task with the IHD was to get up to speed on virus work.[47] His previous experience was in bacteriology and immunology and he was unfamiliar with some of the laboratory techniques central to virus work.[48] More specifically, before becoming a critical part of the YFRI team Smithburn had to get up to speed on yellow fever. To achieve that purpose he trained first in the New York lab of the IHD and then proceeded to Rio de Janeiro to train in yellow fever work under Fred Soper.[49]

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[43] Warren to Sawyer October 26, 1938, RAC RF RG1.1 Series 477o Box 1 Folder 2.
[44] Hughes to Andrew [no last name], July 30, 1941, Folder 5, Box 1, Series 477o, RG 1.1, RF, RAC.
[45] Strode to Warren, March 20, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.
[46] Mahaffy to Warren, March 20, 1941, Folder 5, Box 1, Series 477, RG 1.1, RF, RAC.
[47] Rockefeller Foundation, 1950 Annual Report: New York: The Rockefeller Foundation, 19.
[48] Sawyer to Mahaffy, April 30, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.
[49] Sawyer to Soper, April 28, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.

At the end of March, Smithburn left Brazil and sailed to Europe where he had several meetings in London and Paris with key IHD and colonial office personnel to further prepare him for his work in Entebbe. Here he met the first of several explanations for why the work he observed in Brazil couldn't be entirely exported to Entebbe. Dr. Warren met with Smithburn and advised him that viscerotomy, while certainly desirable in East Africa, was impracticable due to the isolation of many villages and the resistance of local residents, among other reasons.[50] Other strategies would have to be employed in order to define and demonstrate the limits of endemic yellow fever in East Africa. Warren himself had recently visited the Institute. He was positive about their potential but cognizant of the challenge facing them. In a letter to Sawyer he wrote, "In Uganda I was particularly impressed with the difficult problem with which the group there is confronted in their efforts to isolate the yellow fever virus. The virus is unquestionably present in an endemic form but due to the lack of communication and to the habits of the natives, isolation of a virus in a given period of time will be a matter of chance."[51] He and other senior members of the IHD were more cautious about predicting a rapid resolution of the fundamental problem of yellow fever distribution than they had been only a few years earlier.

It quickly became evident that, unlike his predecessor, Smithburn was an excellent match for the Entebbe laboratory. He was, by all accounts, fiercely dedicated to the pursuit of the laboratory's goals and immersed himself in the work. Having made the transition to arbovirus work, he embraced it whole-heartedly. According to one of his colleagues, Smithburn could

[50] Kenneth C. Smithburn (KCS) diary, April 4, 1939, Folder 101, Box 11, Series II, Kenneth C. Smithburn Papers, RAC. Even the liver slides obtained through viscerotomy were subject to interpretation—there were frequent disagreements between qualified scientists about whether pathological specimens were indicative of yellow fever infection. Experts acknowledged that it was difficult to say with certainty whether a specimen was positive or not for yellow fever. Wrote Strode of one French doctor, "I also got the impression that B. feels quite sure of his ability to diagnose yellow fever by examining the stained liver section. This may be due to the relatively small number of specimens he has examined." Strode diary, RAC RF RG12 Reel M Str 2, Frame 711 accessed online Jan. 13, 2015. The *East African Medical Journal* quoted Soper as saying that for pathological interpretation of a viscerotomy sample he would "insist on the examination being made by someone who has been doing nothing but looking at livers for a considerable period of time." "Editorial: Yellow Fever" *East African Medical Journal* 19, no. 4 (1942): 106 quoting from *Transactions of the Royal Society of Tropical Medicine and Hygiene* for November, 1938.
[51] Warren to Sawyer, April 5, 1939, Folder 3, Box 1, Series 477o, RG 1.1, RF, RAC.

identify the genus of a sample of ground up mosquitos by their smell alone.[52] He was also not averse to self-experimentation when the opportunity arose. Upon accidentally cutting himself with a broken ampoule of mouse brain infected with Bwamba fever virus, he wrote "The damage was done – so I did not wash off the virus from my hand for about 10 minutes – and then only with water. [Mahaffy] took a blood sample at once to serve as a control in case anything happens. I shall take my temperature twice daily and do daily tests for circulating virus. We do not know what to anticipate, and nothing may happen, but if anything does we shall try to learn what we can about it."[53]

One of Smithburn's first tasks was training a cadre of African laboratory assistants. A handful of international health experts couldn't run the laboratory alone. The estimates for 1940 included salaries for one European laboratory assistant, four African laboratory assistants, three laboratory cleaners, and three African field assistants.[54] By 1942 the lab employed twenty-five African lab workers including one who was allegedly nicknamed "he who strikes with a needle" for his inoculation skills.[55] Very little is known about them as individuals, but as a group they were critical to the laboratory's operation. A man named Eriza was hired in 1939 and Smithburn trained him as a section cutter, making the fine slices of organ tissue that could be examined under a microscope for evidence of yellow fever infection, observing, "He seems to have good capacity to learn and understand the methods. He reads English well."[56] Another new hire, Zavuga, was a good worker, Smithburn wrote, but slower than Eriza "and thus far less to be depended on to avoid errors."[57] Just as not all American and European scientists could adapt to life and work in Entebbe, not all Ugandans could adopt the practices and behaviors expected of

[52] Undated note, Folder Gillett/06/08, John David Gillett Papers, Archives of the London School of Hygiene and Tropical Medicine (LSHTMA). Apparently *Aedes gambiae* had an "earthy" smell.
[53] KCS diary, March 6, 1943, Box 439, RG 12, RF, RAC.
[54] Folder 3, Box 1, Series 477o, RG 1.1, RF, RAC.
[55] Excerpt from Trustees Confidential Bulletin Feb. 1942 issue "Africa on Guard Against Yellow Fever", Folder 6, Box 1, Series 477o, RG 1.1, RF, RAC.
[56] KCS diary May 16, 1939, Folder 101, Box 11, Series 2, KCS Papers, RAC.
[57] KCS Diary May 21, 1939, Folder 102, Box 11, Series 2, KCS Papers, RAC. Years later, Kitchen named Zavuga as one of only two Africans he knew that earned his pay. Kitchen to Smithburn, March 4, 1949, Folder 34, Box 4, Series 1, KCS Papers, RAC.

laboratory technicians. A third assistant, Grace, was slow to learn the cutting and staining techniques Smithburn taught, partly because of his limited command of the English language.[58] After less than two weeks he was given the remainder of the month's pay and dismissed.[59] While the competent work of technicians doing even the most skilled work is largely taken for granted in the published reports of the YFRI's work, there are frequent mentions of the incompetence or deceit of African employees in the internal reports of Smithburn and his European and American colleagues. Times when African employees stole animal feed, misplaced laboratory keys, and failed to manage routine tasks like taking the temperatures of laboratory monkeys exasperated Smithburn.[60] They also may be subtle indications of the way that African subordinate staff took advantage of employment at the Institute to meet their own needs, expressed frustration or anger over their treatment, or simply manifested a very different understanding of the laboratory's work and its importance.

The Mouse Protection Test & Mass Serum Collection

For the most part, the African laboratory technicians and animal colony attendants were occupied by tasks related to the mouse protection test, the central immunological technique employed by the YFRI. Viscerotomy was one way of making yellow fever visible to researchers in the body of deceased victims. The mouse protection test was a way to make it visible to researchers in the bodies of survivors. Created by Max Theiler of the Rockefeller labs in New York and subsequently refined by the staff of the Institute in Entebbe, the mouse protection test was an affordable, relatively simple way to determine whether a human (or animal) possessed the antibodies that indicated previous exposure to yellow fever virus. In brief, the mouse protection test required scientists to mix the serum from a person whose immune status was unknown with

[58] KCS Diaries, May 2, 1939, Folder 101, Box 11, Series 2, KCS Papers, RAC.
[59] KCS Diaries, May 10, 1939, Folder 101, Box 11, Series 2, KCS Papers, RAC.
[60] KCS Diaries, August 31, September 28, and October 10, 1945, KCS diary, RG 12, RF, RAC.

live yellow fever virus and then to inject the mixture into the brain of a mouse. Figure 2 shows what the test looked like in operation.

If the mouse lived, then the test was positive. A living mouse indicated that the virus in the mixture had been neutralized by antibodies in the human serum, thus revealing that the person from whom the serum was taken had been exposed to the virus at some point in the past. It was, as one Rockefeller Foundation report put it, "telltale evidence of a past encounter" with the virus.[61] On the other hand, if the mouse died, the test was negative. The virus had survived in the mixture, indicating that the human serum contained no antibodies to the virus and thus that the source of the serum was a person who had never been exposed to the virus.[62]

While designed to meet the need for a definitive and universal test for yellow fever immunity, the mouse protection test did in fact need to be modified to fit local conditions. Smithburn wrote extensively about the modifications to the test as it was conducted in New York and Brazil to meet the exigencies of Ugandan conditions and the scale of the YFRI's project.[63] For example, the test also depended on a consistent and voluminous supply of baby mice. In the words of the Rockefeller Foundation, "mice became as indispensable as men and virus" for the project.[64] Maintaining productive mouse colonies proved to be a challenge for men trained in virology, bacteriology, and laboratory techniques but called upon to wrestle with vagaries of altitude, temperature, and breeding behaviors in order to cultivate this critical resource. The Institute's mice were descendants of a line originally from New York and obtained in 1936 from

[61] Excerpt from Trustees Confidential Bulletin Feb. 1942 issue "Africa on Guard Against Yellow Fever", Folder 6, Box 1, Series 477o, RG 1.1, RF, RAC.
[62] Bell points out that interpretation of the test was not always as straightforward as this description suggests. Not all infected mice died immediately. Determining whether experimental animals were ill and whether their illness was yellow fever or another infection was challenging and involved tacit knowledge that the laboratory technicians and the supervisors must have developed over time. Bell, *Frontiers of Medicine,* 167.
[63] Kenneth Smithburn, "Experimental Studies on the Yellow Fever Protection Test," *Journal of Immunology* 51, no. 3 (1945): 173-189. The relative merits of standardization of protection test procedures versus allowing laboratories to innovate and research improved methods were still being debated at the WHO in the 1950s. See F.N. Macnamara, "The Mouse Neutralization (Protection) Test for Yellow Fever," WHO/YFV/13, Accessed Sept. 2, 2015 and Kenneth Smithburn, "The Mouse Protection Test for Yellow Fever," WHO/YFV/2, Accessed Sept. 2, 2015.
[64] Excerpt from Trustees Confidential Bulletin Feb. 1942 issue "Africa on Guard Against Yellow Fever", Folder 6, Box 1, Series 477o, RG 1.1, RF, RAC.

the IHD labs in Brazil.[65] Genetically, therefore, they were "comparable" to the mice in Brazil. [66] This was important because interpreting the results of the test required mice of consistent susceptibility to yellow fever infection (which varied among mouse strains), size, and health.[67] Hypothetically, these mice, like scientists, should have been able to perform identically in any laboratory. The movement of mice from New York to Brazil to Entebbe traces the contours of a transnational experimental space that was ideally free of the vagaries of particular places. But, as with people, the reality of moving mice from place to place was more complex. While they may have been genetically identical to their brethren in Brazil, the environment in Entebbe caused them to behave differently.

As the YFRI staff quickly discovered, these mice couldn't be treated in quite the same way as their relatives in Brazil. After transporting the initial mouse stock from Rio de Janeiro to Entebbe, the YFRI staff found themselves stymied by "relatively poor condition of the mice, low pregnancy rate and the failure to care for the young."[68] By gradually increasing the amount they fed the mice until they were eventually giving them roughly double the amount recommended by the staff in Brazil, they were able to improve the quality of the colonies until they appeared to have eliminated the problem of neglected or eaten litters. They speculated, "The acclimatization apparently involves change in metabolism rate and higher food requirements."[69] By the end of 1939 they anticipated total production of 10,000 mice per month.[70] The births, deaths, and mating habits of the mice were meticulously observed and documented by a large staff who were also responsible for maintaining the cleanliness of the colony and protecting it from contamination by infectious agents or wild rodents. In addition to maximizing the fertility of the colonies, the mice

[65] Smithburn, "Experimental Studies," 174.
[66] Smithburn, "Experimental Studies," 174.
[67] Alexander Mahaffy, Wray Lloyd, and H. A. Penna, "Two Years' Experience with the Intraperitoneal Protection Test in Mice in Epidemiological Studies of Yellow Fever," *American Journal of Epidemiology* 18, no. 3 (1933): 619.
[68] Annual Report, 1936, page 24, Folder 2905, Box 241, Series 3, 700, RG 5, RF, RAC. The Entebbe mouse diet in 1936 consisted of 2 buckets each containing: 2000cc milk, 4000cc boiled water, 700g bran, 1400g bread, 30g calcium carbonate, 15g sodium chloride, and 20cc cod liver oil.
[69] Annual Report, 1936, page 24, Folder 24, Box 241, Series 3, 700, RG 5, RF, RAC.
[70] "Africa-Yellow Fever Investigations," Folder 3, Box 1, Series 477o, RG 1.1, RF, RAC.

had to be maintained under the most sterile conditions possible to avoid epidemics of mouse typhoid or other infections that could wipe out whole colonies or interfere with the interpretation of antibody tests.[71] Periodically disease would strike or unexplained factors would lead to a drop-off in the fertility or survival of the colonies and the scientists would have to tinker with diet, containers, etc. to restore the colonies' productivity, as in May 1942 when Smithburn returned from leave to find his first task was to improve the mouse colonies.[72]

In addition to vast numbers of mice, the project of identifying areas in which the virus was or had recently been active also required enormous numbers of human serum samples. First, the human serum had to be collected from hundreds, eventually thousands of African so-called donors [Figures 3-5]. Sometimes collection was on the basis of signs of illness, as when Mahaffy and his wife would take tours of nearby villages during which she would "fix the babies' eyes and […] dish out quinine and aspirin […] while Tiny [Mahaffy] has a look round for yellow fever suspects." Sometimes they went in search of individuals reported to be ill. Mrs. "Jimmie" Mahaffy described one such trip to a village called Kinyara:

> When we got to Kinyara we found it to be a small village and mission school. They put out a row of chairs for us to sit on. Then the school formed a sort of body guard, squatting all around, and I don't think any of them took their eyes off us for a second. We took the blood specimen from the man who had been sick. Couldn't find anyone else who needed treatment so off we pushed home again and if anyone could have produced a couple of bottles of really cold beer when we got off those bicycles, we would cheerfully have given him their weight in gold![73]

More often, the YFRI staff relied on the European and African dispensary staff to notify them of cases where a patient had an unexplained high fever. When the YFRI staff was away, they would ask dispensary staff to draw blood from these patients and inoculate it into mice.[74] Local "dressers", African medical auxiliaries, in the Masaka district in 1937 were expected to conduct

[71] Mahaffy, Lloyd, and Penna, "Two Years' Experience," 618-628.
[72] KCS diaries, May 19, 1942, Box 439, RG 12, RF, RAC.
[73] Mrs. A.F. Mahaffy to Miss R.V. Reed, excerpted from IHD Newsletter July 1937, Folder 1, Box 1, Series 477o, RG 1.1, RF, RAC.
[74] Excerpt from Strode's diary, March 4, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.

home visits and look for cases of acute fevers.[75] The plan was that blood taken from such a case would be injected into a mouse, which, if it became ill, would be sacrificed, its brain emulsified and injected into another mouse, which would in turn be transported to Entebbe, where its brain would be emulsified and injected into a rhesus monkey. In the event that virus was isolated from the procedure, "it will be frozen and dried and sent to New York for further study."[76] Successful collection and analysis of these specimens required a web of connections linking rural Uganda to New York via medical auxiliaries, drivers, laboratory technicians, animal attendants, and air travel.

Most specimens were collected in mass operations. This was partly because the researchers didn't believe that Africans were reliable reporters of their own disease experiences. The Rockefeller Foundation explained to its trustees, "To the African native, disease is a fairly unitary experience and all fever is regarded as malaria."[77] Under those circumstances, an objective measure of past infection was critical. The mouse protection test was that measure, and was successful precisely because it "helped to further sever disease identity from the experience of people feeling unwell."[78] Since researchers couldn't rely on individuals reporting disease, they simply aimed to survey as many members of the at-risk population as possible. Individuals of various ages were targeted for the collections in order to identify with some precision the date of the most recent outbreak of yellow fever. If a 40-year old adult tested positive for antibodies, he or she could have been exposed at any time in the previous four decades. But if a seven-year-old child was positive, an outbreak must have occurred in past seven years. Initially, European and American doctors drew the blood, but as the scale of operations increased, so did the necessity to

[75] GKS Diary March 7, 1937, RG 12, RF, RAC, digitized, accessed Jan. 15, 2015.
[76] GKS Diary March 7, 1937, RG 12, RF, RAC, digitized, accessed Jan. 15, 2015.
[77] Excerpt from Trustees Confidential Bulletin Feb. 1942 issue "Africa on Guard Against Yellow Fever", Folder 6, Box 1, Series 477o, RG 1.1, RF, RAC.
[78] Bell, *Frontiers of Medicine*, 196.

recruit and train skilled African staff who gradually took over some of the blood collection [Figure 6].[79]

It was necessary for the YFRI to integrate their work into the politico-geographic organization of the Protectorate. One of the purposes of Warren's 1939 visit to Uganda was to liaise with the colonial authorities and make sure he was fully conversant with the ways in which space and people were divided and subdivided for the purposes of governance, service delivery, and, now, research.[80] From the beginning, the IHD had been deeply embedded within the British empire. The yellow fever serosurvey was one of many medical research programs observing African bodies and extracting biological specimens from African people for the purpose of conducting research. For the YFRI, this created opportunities to secure specimens for yellow fever protection tests. One such opportunity was the periodic examination of natives for sleeping sickness by the colonial medical authorities. In 1938 Strode detailed such a procedure in his diary:

> The sleeping sickness inspection has been used as a means of finding suspicious cases of Y.F. [yellow fever], by taking temperatures of all those who attend the inspection. In this way AWB [Burke] has examined many thousands of natives in this district [West Nile]. All individuals who have temperatures over 100°F are set aside; their bloods are examined for malaria parasites and those negative are bled and their serums injected intracerebrally into white mice.[81]

For local people subjected to these examinations and extractions, these encounters with colonial biomedicine would have been far less sanguine. That the extraction of blood was an experience laden with meaning has been well established by a number of historians and anthropologists.[82] Moreover, it was one of the most ubiquitous experiences of colonial medicine for Africans:

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[79] This practice was possible in the interwar period and immediately after WWII but by the 1960s and 1970s it was considered highly inadvisable for a research protocol to permit white personnel to draw blood from Ugandan donors. Edward H. Williams to Director of the EAVRI, November 13, 1969, WTI/EHW/G/3 Wellcome Library Archives (WLA).

[80] Excerpt from Warren's Diary, Feb. 8, 1939, Folder 3, Box 1, Series 477o, RG 1.1, RF, RAC.

[81] GKS Diary March 12, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC. It was such a procedure that led to the isolation of what came to be known as West Nile Virus.

[82] E.g. White, *Speaking with Vampires*; Jennifer Tappan, "Blood Work and 'Rumors' of Blood: Nutritional Research and Insurrection in Buganda, 1935-1970," *International Journal of African Historical Studies* 47, no. 3 (2014): 473-495; P. Wenzel Geissler, Ann Kelly, Babatunde Imoukhuede, et al., "'He is now like a brother, I can even give him some blood'—Relational ethics and material exchanges in a malaria vaccine 'trial community' in The Gambia," *Social Science & Medicine* 67, no. 5 (2008): 696-707.

"Whether as part of an organized survey, a diagnostic procedure, or private research, the requirement that they surrender tissue had become one of the ways Africans experienced colonial-era medicine."[83] The proliferation of projects involving the sampling of blood from Africans has been compared to geographic explorations in that sometimes "blood seems to have been taken for no reason other than the sheer exuberance of exploration."[84] But in this case, at least, it served a very particular purpose. It wasn't only Africans who invested blood with additional meaning. For the people drawing the specimens, blood was a way to transform foreign and potentially hazardous parts of Africa into experimental sites, sites of production, and new markets.[85] While there is little direct evidence of Africans' reactions to being bled for yellow fever testing, it seems likely that this would have been an ambiguous experience at best and a sinister one at worst. In order to secure the cooperation of the individuals and communities whose blood they required, the researchers had to accommodate some local practices and sometimes to employ the coercive power of the British state.

In order to connect the results of protection tests on individual serum samples to the larger question of where in East Africa yellow fever virus was found, the Institute had to translate laboratory results to geographic data. This required meticulous data management so that specimens could be tracked from their point of collection through the determination of the protection test results. Then the results were mapped. Making the maps was a painstaking task that occupied not only the staff the Institute but even, in some cases, their wives. Florence Smithburn, an accomplished artist whose watercolors delighted visitors to Entebbe, spent time in

[83] Patrick Malloy, "Research Material and Necromancy: Imagining the Political-Economy of Biomedicine in colonial Tanganyika," *International Journal of African Historical Studies* 47, no. 3 (2014), 425-443, quotation on 430. Malloy continued, "blood can be understood as having become a quasi-currency in an elaborate and unequal exchange that allowed medical experts to extract knowledge from African bodies."
[84] Malloy, "Research Material and Necromancy," 430.
[85] Gregg Mitman and Paul Erickson, "Latex and Blood: Science, Markets, and American Empire," *Radical History Review* no. 107 (2010): 45-73.

1943 making the maps of serum results that illustrated a publication on the distribution of positive samples in East and Central Africa published in 1946.[86] [Figure 7]

The piggybacking of colonial projects, particularly in regions far from the urban centers, was common and made it much easier for researchers in a number of fields to complete their projects. The YFRI's research was most easily and efficiently conducted in the places where the colonial state was already assembling people for other purposes. By 1938, the Institute had established a field camp in the Semliki Forest in the Bwamba district of western Uganda in order to take advantage of the concentration of African laborers at work on a road construction project. As a report to the Rockefeller Foundation trustees put it: "the illnesses of the workers and their families provide rich opportunities for study."[87] This was especially the case for workers that were not indigenous to the area and who would be less likely to have previously acquired immunity to yellow fever and other viruses believed to be especially prevalent in the region. The project also offered the opportunity to capitalize on the unusually high level of medical surveillance of workers and their families to attempt to isolate virus from a person with an acute infection. The procedure was described as follows:

> The native doctors are able to care for all ordinary diseases [among the workers]; but whenever a patient shows symptoms of an unusual character, the native doctor hurries to report the case to [the YFRI] camp. If none of the white doctors is present, the native himself takes a sample of the patient's blood, using one of the sterilized receptacles which have been provided. Such samples are injected into mice. If the mice show interesting symptoms the injection is transferred to other mice which are then sent to the Institute at Entebbe for study."[88]

Similar strategies were employed by the YFRI in the southern part of the Anglo-Egyptian Sudan where they took advantage of sleeping sickness inspections and another road project (the Meridi-

[86] KCS diary, May 17, 1943, Box 439, RG 12, RF RAC; Alexander F. Mahaffy, Kenneth Smithburn, and T. P. Hughes, "The Distribution of Immunity to Yellow Fever in Central and East Africa," *Transactions of the Royal Society of Tropical Medicine and Hygiene* 40, no. 1 (1946): 57-82. Several of Florence Smithburn's paintings from East Africa can be viewed online courtesy of the Indianapolis Museum of Art. http://collection.imamuseum.org/results.html?query=smithburn+florence+bartley accessed January 27, 2017.

[87] Excerpt from Trustee's Confidential Monthly Bulletin, May, 1938, "A Yellow Fever Barrier in Africa," Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.

[88] Excerpt from Trustee's Confidential Monthly Bulletin, May, 1938, "A Yellow Fever Barrier in Africa," Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC. The "native doctors" were not indigenous healers but African men trained to act as medical auxiliaries.

Yei road).[89] In these ways, the YFRI was deeply embedded in the local political and economic environment of British East Africa.

"Hitherto Unknown Viruses"

While the agenda of the IHD for the YFRI was relatively narrow (though ambitious), the experiences of the researchers in Uganda led them to important scientific discoveries they hadn't anticipated. Occasionally blood specimens collected from ill patients in the hopes of isolating yellow fever "betrayed the presence of infectious agents which as yet have not been identified and which in fact may prove to be important causes of disease now unknown to science."[90] During the extended period YFRI researchers undertook the fruitless quest to isolate yellow fever virus, the discovery of previously unknown viruses in the course of these surveys, as Mahaffy put it, "added greatly to the zest of the work."[91] These viruses offered the opportunity to establish the importance of an East African virus research institute while also keeping the lab men occupied. As Strode observed to Soper, they served the important role of being "something interesting and useful to explore while carrying on the negative work which is inevitable in this search for a needle in a haystack."[92] With fieldwork related to the search for the yellow fever virus curtailed by the war, laboratory-based investigations of these hitherto unknown viruses collected between 1936 and 1938 came to occupy more of the time of the staff in Entebbe.[93] Announcement in the *Lancet* of one of these new agents, West Nile Virus, led the journal to assert, "The last twenty years of the nineteenth century will be remembered as the golden age of bacteriological discovery; the period between the two world wars deserves equal credit in regard to virus

[89] Bell, *Frontiers of Medicine*, 183.
[90] Excerpt from Trustee's Confidential Monthly Bulletin, May, 1938, "A Yellow Fever Barrier in Africa," Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.
[91] AFM Diary March 2, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF RAC.
[92] Sawyer to Soper, April 1, 1938, Folder 2, Box 1, Series 477o, RG 1.1, RF, RAC.
[93] Jacobs to Warren, October 15, 1939, Folder 3, Box 1, Series 477o, RG 1.1, RF, RAC.

research. Not only many long-known diseases but also many new ones have been traced to virus infection."[94]

The isolation of these viruses also provided validation of the methods being used by the Institute and allayed concerns that its failure to isolate yellow fever was due to poor methods or implementation.[95] In addition, as yellow fever had never been a clinically-recognized problem in Uganda, demonstration that the Institute could identify and possible offer some control suggestions for other diseases causing recognizable morbidity was valuable to the colonial state. Mahaffy reported of Uganda's director of medical services, William H. Kauntze, "he has told me that in his opinion the viruses now being studied here are probably of more importance as far as Uganda is concerned than yellow fever."[96] Securing the continued support of the officials in Uganda required some acknowledgment of their priorities for local medical research.

Nuba Mountains Outbreak[97]

The validation of the mouse protection test came when an outbreak was finally observed in progress in the Nuba Mountains of Anglo-Egyptian Sudan in October 1940. Believed to have originated in May in Tira Limon, "an isolated plateau not far from Talodi," it was detected only months later.[98] Findlay of the Wellcome Institute, a sometimes rival of the YFRI, suggested that "In all probability the epidemic would have burnt itself out on Tira Limon and nothing would have been heard of the outbreak" (because the local people were notorious for avoiding encounters with dispensary staff and other health officials) if crop damage in nearby areas hadn't led occupants of other villages to wander into the area looking for food.[99] Liver sections were

[94] "Two New Virus Diseases of Man," *Lancet* 236, no. 6117 (1940): 657-658.
[95] Alexander J. Haddow, "Studies on the Natural History of Yellow Fever in East Africa With Notes on Other Insect-borne Infections" (DSc thesis, University of Glasgow, 1957), 17.
[96] Mahaffy to Warren, May 18, 1939, Folder 3, Box 1, Series 477o, RG 1.1, RF, RAC.
[97] Bell provides a detailed account of this epidemic from the perspective of the medical authorities in the Anglo-Egyptian Sudan (and to some extent to the people living in the region) in *Frontiers of Medicine*. My account concentrates on the developments directly related to the YFRI in Uganda.
[98] Findlay, Memorandum on Yellow Fever in Africa, February 1, 1941, TNA, CO 859/63/8.
[99] Findlay, Memorandum on Yellow Fever in Africa, February 1, 1941, TNA, CO 859/63/8, page 24.

used to confirm the diagnosis of yellow fever and in November Mahaffy and the YFRI team succeeded in isolating two strains of virus from clinical cases.[100]

Isolation wasn't simple. By the time the YFRI team got to work in November of 1940 the epidemic was waning and the best way to find cases was to go from home to home in the remote hill areas.[101] In order to isolate the virus, they needed to find patients in the early stages of infection, before they had begun to recover as their immune systems overwhelmed the virus. Because the researchers didn't trust patients to report the duration of their illness they collected specimens from all patients that had a fever and other suggestive symptoms. These specimens were put to two uses: a small portion of each serum specimen was injected into a batch of five or six mice that were then transported to Entebbe for observation; the remaining serum was saved and later tested for protective antibodies.[102] Two patients, referred to as "prospective virus-donors" yielded virus: Case S 5, a young woman examined on November 22, 1940, and Case S 6, a Sudanese laboratory assistant who became ill on the same day in another part of the region.[103] The laboratory assistant was observed throughout his illness and convalescence but the young woman could not be located for follow-up observation after the first time she was examined and bled.

Once they had arrived in Entebbe, the inoculated mice were observed for signs of illness. Sick mice that had been inoculated with serum from cases S 5 and 6 were sacrificed and their brains were processed into a suspension for intracerebral inoculation into new batches of mice. The virus appeared to increase in virulence over the course of the passages such that it affected an increasing percentage of mice inoculated with each passage.[104] The affected mice were further

[100] Findlay, Memorandum on Yellow Fever in Africa, February 1, 1941, TNA, CO 859/63/8; Extract from Minutes of the 413th Meeting of the Colonial Advisory Medical Committee, June 24, 1941, TNA, CO 859/63/8.
[101] Alexander F. Mahaffy et al., "The Isolation of Yellow Fever Virus in the Anglo-Egyptian Sudan," *Annals of Tropical Medicine and Parasitology* 38, no. 2 (1941): 141-148.
[102] Mahaffy et al, "The Isolation of Yellow Fever Virus," 142.
[103] Mahaffy et al, "The Isolation of Yellow Fever Virus," 142.
[104] Mahaffy et al, "The Isolation of Yellow Fever Virus," 143-4.

examined after sacrifice and lesions were observed in their brains that were consistent with yellow fever infection.

Technicians also inoculated the brains of mice from those originally inoculated with serum from cases S 5 and 6 into rhesus monkeys—the only non-human primates known to show symptoms of yellow fever infection. The inoculated monkeys either succumbed to the disease or recovered and were shown to have developed antibodies to yellow fever virus.[105] Lesions in the livers of the 7 monkeys that died after inoculation were also consistent with yellow fever infection.[106]

These results were considered definitive evidence that yellow fever virus had been isolated from humans in the Nuba Mountains and seemed to lay to rest most, if not all, of the doubts about the accuracy of the mouse protection test surveys. Moreover, there was no evidence to suggest that the virus thus isolated differed in any way from strains isolated in West Africa or South America.[107] The isolation of the virus was cause for great celebration and relief in Entebbe as well as at the headquarters of the Rockefeller Foundation International Health Division in New York. Of course, medical authorities in East and Central Africa were much less sanguine than the IHD scientists, since the test results seemed to suggest that they had been unable not only to stop, but even to observe, an epidemic. As Colonel Crouch of the Sudan Medical Service put it:

> [T]his epidemic has come upon us like a bolt from the blue. And it is this sudden and unexpected appearance of the disease in a country where the medical authorities have been on the alert and have taken the recognised precautionary measures to guard against its introduction which creates, in my opinion, disquieting possibilities in any territory where the vector is present and where mouse protection tests have proved positive.[108]

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[105] Mahaffy et al, "The Isolation of Yellow Fever Virus," 144.
[106] Mahaffy et al, "The Isolation of Yellow Fever Virus," 147.
[107] Mahaffy et al, "The Isolation of Yellow Fever Virus," 148.
[108] Colonel Crouch, Appendix, Proceedings of the Conference on Yellow Fever Held Under the Auspices of the Conference of East African Governors at the Medical Research Laboratory, Nairobi, December 9, 1940, TNA CO 859/63/8.

The first part of the enigma presented by the maps showing the discordant distributions of yellow fever cases and yellow fever immunity in Africa had been resolved. The editors of the *East African Medical Journal* asserted confidently:

> Now an epidemic has occurred, and it has occurred in an area where mouse protection tests had been found to be positive, and in circumstances which almost certainly preclude the possibility that the infection had recently been introduced from the West. It, therefore, follows that whatever views may hitherto have been held with respect to the specificity of the mouse protection test in these areas, there is now the very strongest evidence in support of the contention, not only that the infection has long been present in the Sudan, but that it is probably still present in some form, or other, in every region where mouse protection tests have yielded positive results, and that 'flaring up' of the disease may occur in any of these regions at any time.[109]

At the same time, the clinical characteristics of the outbreak did justify some of the confusion that had seized the medical community due to the apparent disagreement between the laboratory evidence of yellow fever activity in the region and the utter absence of clinical observation of any cases. Many of the cases in the Nuba Mountains outbreak were indeed quite mild and medical officers were gratified to confirm that they had probably not been overlooking cases of the virulent "yellow jack" type of yellow fever infection.[110] As Kirk of the Stack Medical Laboratories in Khartoum and one of the principle investigators of the epidemic observed:

> The very large preponderance of mild cases which was observed during this epidemic…suggest[s] one explanation for the failure to discovery cases in some regions where mouse-protection tests show that the virus has been present. The 'typical case' of yellow fever as observed in this epidemic bore little resemblance to the text-book descriptions of the disease…In sporadic cases of this type clinical diagnosis would be well-nigh impossible. By the time that malaria had been excluded and the physician had turned to the differential diagnosis of other tropical fevers, the disease would be over; or, if antimalarial treatment had been given, recovery would appear the normal result of this. The occurrence of a sufficient number of severe and fatal cases to attract attention in the present instance was due only to the magnitude of the epidemic.[111]

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[109] "Yellow Fever and East Africa," *East African Medical Journal* 17, no. 10 (1941): 403-4.
[110] Bell, Frontiers of Medicine, 193.
[111] Robert Kirk, "An Epidemic of Yellow Fever in the Nuba Mountains, Anglo-Egyptian Sudan," *Annals of Tropical Medicine and Parasitology* 38, no. 1 (1941): 102-3.

Moreover, he continued, antibody testing after the epidemic indicated that even with the heightened awareness of all doctors during the outbreak many mild cases were never diagnosed.[112]

The outbreak appeared to bring the preponderance of evidence firmly on the side of the specificity of the mouse protection test, but it did not in and of itself answer the second question raised by the maps described at the beginning of the chapter—whether yellow fever had been present and undetected in the so-called silent areas for a long time or whether it was penetrating further east over time. In the months following the epidemic, Findlay attempted to resolve this question using historical accounts of fevers that might be yellow fever reported by Arabs and Europeans in their accounts of travel through the region.[113] While his evidence was tenuous at best, he concluded, "There is no evidence to suggest that yellow fever has been recently introduced into the Sudan."[114] While this may have been reassuring to some, authorities were by no means complacent about the risk of yellow fever spreading beyond its known boundaries, especially in the context of troop movements and population disturbances due to the war.

While the outbreak seemed to satisfy the greater scientific community that the serosurveys were accurate, researchers at the time of the outbreak were unable to determine which insect vector was responsible for the outbreak or even whether this was a "classical" outbreak of yellow fever or whether it was something more like "jungle" yellow fever, defined by Soper as yellow fever in the absence of *Aedes aegypti*.[115] Entomological studies at the time of the epidemic were inconclusive but suggested that *Aedes aegypti* was indeed present in the Nuba Mountains villages but was not definitely the only or even primary vector.[116] Colonial authorities and the Rockefeller Foundation agreed, more research was necessary.

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[112] Kirk, "An Epidemic of Yellow Fever," 103.
[113] G. M. Findlay, "Yellow Fever and the Anglo-Egyptian Sudan: Historical," *Annals of Tropical Medicine and Parasitology* 32,1 (1941): 59-65.
[114] Findlay, "Yellow Fever," 65.
[115] F. L. Soper, "Yellow Fever: The Present Situation (October, 1938) with Special Reference to South America," *Transactions of the Royal Society of Tropical Medicine and Hygiene* 32, no. 3 (1938): 300.
[116] Kirk, "An Epidemic of Yellow Fever," 106.

Vaccine Distribution Work

Despite the new energy for studies on the epidemiology of yellow fever in East Africa, during the war years much of the effort in the labs was necessarily diverted into activities to support the allied war effort, especially testing and distributing vaccine. After the Nuba Mountains epidemic had convinced even the most ardent opponents of the mouse protection test that yellow fever was a real threat in East Africa, demand for yellow fever vaccine rose precipitously.[117] As the 1941 semi-annual report put it, "Following the Sudan outbreak all neighboring countries became 'yellow fever conscious'."[118] With military mobilization bringing large numbers of non-immune Africans and Europeans into the endemic zone, the Allies scrambled to secure access to vaccine stocks.[119] Even before the United States entered the war in 1941, the IHD was lending substantive support to the war effort through the YFRI by using the Institute as a major depot for yellow fever vaccines that had been donated by the IHD. Eritrea and the Kenyan coast were prioritized, "In order that yellow fever should not interfere with the war effort and in order to prevent the spread of the disease to India and the Far East".[120] By the end of 1941 they had distributed over 800,000 doses and in 1942 they distributed an additional 759,660 doses. Between 1941 and 1945 the Entebbe Institute distributed 3,145,760 doses of vaccine.[121] Of those the largest number (38%) went to the Armed Forces. Another 22% went to Kenya, 12% went to the Anglo-Egyptian Sudan and Eritrea, 8% each to Uganda and the Belgian Congo, and the remainder was divided between French Equatorial Africa, Tanganyika, Egypt, Northern and Southern Rhodesia, Nyasaland, Nigeria, Zanzibar, Spanish Guinea, Palestine, Portuguese East Africa, and the Union of South

[117] An address on the diseases that might affect troops in East Africa given in 1940 didn't mention yellow fever. J. R. Gregory, "The Medical Profession and the War," *East African Medical Journal* 17, no. 1 (1940): 2-13.
[118] YFRI first Semi-annual report 1941, Folder 2952, Box 244, Series 700, RG 5.3, RF, RAC.
[119] For the history of the development of the yellow fever vaccine, see Strode, *Yellow Fever,* 36-37, 606-619 and John Farley, *To Cast out Disease: A History of the International Health Division of the Rockefeller Foundation (1913-1951)* (Oxford: Oxford University Press, 2004), 169-182.
[120] Confidential Report of Studies, 1939-1946, Yellow Fever Research Institute, Entebbe, Uganda, Folder 2608, Box 211, Series 477o, RG 5.3, RF, RAC.
[121] Confidential Report of Studies, 1939-1946, Yellow Fever Research Institute, Entebbe, Uganda, Folder 2608, Box 211, Series 477o, RG 5.3, RF, RAC.

Africa.[122] These vaccine doses traced new connections between the Institute in Entebbe and sites across Africa and beyond, as the Institute "became a testing and distribution center for the whole of Africa east of Nigeria, and for the Middle East."[123] In addition, tens of thousands of doses were used in assays to verify potency. Each batch was titrated and each titration required a minimum of 60 mice, meaning that the laboratory's already high need for mice was strained considerably by this requirement for thousands more.[124] In 1942 Smithburn observed in his diary that the staff person on lab duty "under present arrangements…is a somewhat unglorified shipping agent."[125] This was a source of frustration to Smithburn and others who, though they acknowledged its importance, regretted interference with their research agendas. Venting some of this frustration, Smithburn wrote in 1945, "A great deal of this day—like too many others—was spent in handling matters of little moment which no stretch of the imagination could bring under the heading of Y.F. investigations."[126] That year alone they distributed nearly half a million doses, bringing the total doses distributed by the YFRI to 3,145,760, of which 38% went to the armed forces.[127] The vaccine work, however, established a positive reputation for the Institute and elicited the colonial government's gratitude, which would stand it in good stead when it came to rely exclusively on support from government sources after 1950.

[122] Confidential Report of Studies, 1939-1946, Yellow Fever Research Institute, Entebbe, Uganda, Folder 2608, Box 211, Series 477o, RG 5.3, RF, RAC. The number distributed to the Army represented those doses known to have been used for the military but an unknown proportion of the other distributions were also destined for military use. From 1940 until 1946, the IHD manufactured, shipped, and distributed, largely through the YFRI, roughly 4,000,000 vaccine doses Smithburn, "The Yellow Fever Research Institute at Entebbe," 1951, Folder 54, Box 5, Series 1, KCS Papers, RAC.
[123] Confidential Report of Studies, 1939-1946, Yellow Fever Research Institute, Entebbe, Uganda, Folder 2608, Box 211, Series 477o, RG 5.3, RF, RAC.
[124] Confidential Report of Studies, 1939-1946, Yellow Fever Research Institute, Entebbe, Uganda, Folder 2608, Box 211, Series 477o, RG 5.3, RF, RAC.
[125] KCS diary, June 20, 1942, Box 439, RG 12, RF, RAC.
[126] KCS diary, August 13, 1945, Box 439, RG 12, RF, RAC. 1945 was the last year that the YFRI held major responsibilities for vaccine testing and distribution. By the end of that year laboratories in South Africa had taken over the production and distribution of the vaccine in the region. The YFRI also had found it impossible to meet the conditions for vaccine potency and preservation introduced by UNRRA. YFRI Annual Report 1945, UVRI Archive (UVRIA).
[127] YFRI Annual Report 1945, UVRIA.

Isolation of Yellow Fever from Bwamba

The Nuba Mountains outbreak had mostly overcome the reservations of critics of the mouse protection test and its use to detect the presence of yellow fever transmission.[128] However, the YFRI was still anxious to isolate the virus in Uganda, one of the territories where people were most skeptical about the disease's presence. Experts still debated the existence of so-called "silent" areas where individuals had antibodies to yellow fever but no cases of yellow fever. Mahaffy expressed his opinion that there were no places that were truly "silent", but that in some places the clinical cases simply hadn't been observed.[129] In the spring of 1941 Hughes's program of repeated testing of the same individuals (whose identities were verified by thumbprints affixed to blood specimens and read by an officer formerly of Scotland Yard[130]) paid off.[131] Specimens drawn from previously negative individuals in Bwamba were found to have seroconverted. [132] Subsequently efforts to find active cases in that region intensified. Evidence that the virus had been active in the region so recently spurred the Protectorate to assign medical inspector and trained entomologist Dr. J.D. Gillett to the YFRI's project and he took the lead on the Bwamba-based investigations.[133] Gillett's entomological investigations yielded large numbers of only one species of mosquito in the affected area: *Aedes simpsoni*.[134] The YFRI intensified its work collecting human specimens in the area and attempting to transmit virus from *Aedes simpsoni* to

[128] Of course there were still some who remained unconvinced. Smithburn encountered one of these, a Dr. Percy Ross, in Kisumu in September 1944 who referred to the Entebbe group as the "Rockefeller Racketeers." But he was in the minority and Smithburn apparently didn't even consider it worthwhile to challenge him in the presence of more enlightened colleagues. KCS Diary September 15, 1944, Box 439, RG 12, RF, RAC.
[129] Proceedings of the Conference on Yellow Fever Held Under the Auspices of the Conference of East African Governors at the Medical Research Laboratory, Nairobi on Monday the 9th of December, 1940, TNA CO 859/63/8.
[130] "Africa on Guard Against Yellow Fever," Excerpt of the RF Trustee's Confidential Bulletin, February 1942, Folder 6 ,Box 1, Series 477o, RG 1.1, RF, RAC.
[131] This was a strategy Hughes proposed in 1939 when the war made it more difficult for the YFRI to invest in large-scale blood collection activities. Instead, he suggested offering a selected group of non-immune individuals a cash incentive to participate in repeat bleedings. Then, by limiting their tests to samples from individuals with at least two separate specimens, the laboratory could maximize the efficiency of their immune testing. The plan was to use these serial tests to identify an area in which yellow fever transmission was occurring and then to undertake an intensive project to isolate the virus in that particular area. A similar strategy would be employed a decade later with the sentinel monkey program. Hughes to Warren, October 15, 1939, Folder 3, Box 1, Series 477o, RG 1.1, RF, RAC.
[132] YFRI semi-annual report 1941, Folder 2952, Box 244, Series 700, RG 1.1, RF, RAC.
[133] YFRI semi-annual report 1941, Folder 2952, Box 244, Series 700, RG 1.1, RF, RAC.
[134] YFRI semi-annual report 1941, Folder 2952, Box 244, Series 700, RG 1.1, RF, RAC.

laboratory animals. Viscerotomy, previously seen as too offensive to local opinion to be a good investment of time and energy, was undertaken in the area, though it failed to turn up any positive cases.[135] In addition, the Institute deployed a cadre of "temperature scouts"[136]:

> African Assistants trained in the use of the thermometer were sent throughout the district to search for persons suffering from a febrile illness. All cases found by this or by any other means were investigated and if their illness was considered suspect and was not of more than 4 days duration a blood specimen was taken for examination by the protection test.[137]

None of these specimens yielded evidence of yellow fever infection.[138] Finally the team isolated yellow fever virus from a woman from whom Mahaffy had taken blood in Bwamba.[139] Shortly thereafter, the laboratory succeeded in isolating two different strains of yellow fever virus from experimental monkeys inoculated with Aedes simpsoni mosquitoes captured in Bwamba.[140] Reporting on these isolations, Smithburn confided to Warren, "This result is a bit of a relief, since we can now get on with more important investigations."[141]

Following these developments, Uganda's medical department began carrying out vaccinations in the neighboring Toro district, a program of "blockade vaccination", to ensure that any epidemic activity in Bwamba didn't spill over onto its Eastern neighbors and the area where infections were detected was quarantined.[142] Between June and August of 1941 the Medical Department vaccinated about 145,000 people in western Uganda and the area was quarantined.[143] Smithburn expressed the ambivalence of the YFRI staff about this turn of events:

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[135] There is no evidence that the researchers or the colonial state had any reason to believe that viscerotomy would be less objectionable than it had been in the past, but under the circumstances of a possible epidemic, they appear to have weighed the risk of violent resistance and the cost of intensive pursuit of specimens from possible cases less heavily against the benefit of more definitive identification of yellow fever victims.
[136] Africa on Guard Against Yellow Fever," Excerpt of the RF Trustee's Confidential Bulletin, February 1942, Folder 6, Box 1, Series 477o, RG 1.1, RF, RAC.
[137] YFRI semi-annual report 1941, Folder 2952, Box 244, Series 700, RG 1.1, RF, RAC.
[138] YFRI semi-annual report 1941, Folder 2952, Box 244, Series 700, RG 1.1, RF, RAC.
[139] Smithburn to Warren, 18 August 1941, Folder 5, Box 1, Series 477o, RG 1.1, RF, RAC.
[140] Smithburn to Warren, 18 August 1941, Folder 5, Box 1, Series 477o, RG 1.1, RF, RAC.
[141] Smithburn to Warren, 18 August 1941, Folder 5, Box 1, Series 477o, RG 1.1, RF, RAC.
[142] YFRI semi-annual report 1941, Folder 2952, Box 244, Series 700, RG 1.1, RF, RAC. The phrase "blockade vaccination" is from a letter from Smithburn to Warren, June 20, 1941, Folder 5, Box 1, Series 477o, RG 1.1, RF, RAC.
[143] Confidential Report of Studies, 1939-1946, Yellow Fever Research Institute, Entebbe, Uganda, Folder 2608, Box 211, Sereis 477o, RG 5.3, RF, RAC; "Africa on Guard Against Yellow Fever," Excerpt of the RF Trustee's Confidential Bulletin, February 1942, Folder 6, Box 1, Series 477o, RG 1.1, RF, RAC.

> I hated to see this done, but I think it was the thing to do under the circumstances. I think it does not necessarily mean that epidemiological investigations in the area will be useless, and there is, moreover, an area in the adjoining Congo which we believe to be like Bwamba from the point of view of yellow fever studies.[144]

Indeed, as we will see in the next chapter, investigations in Bwamba, particularly on zoonotic infections and mosquitos, were of critical importance for the Institute throughout the 1940s.

Conclusions: "All that is to be known about the epidemiology of yellow fever is not yet known."[145]

By taking Uganda as the point of focus for a study of yellow fever work in this period, the contingency of the work comes to the fore, as does the unique constellation of skills necessary to carry out that work. Uganda was not merely the site for the Institute, it was the place that had to be made known by cartographers, entomologists, lab workers, and even a Scotland yard fingerprint expert in order to support the kind of investigations the IHD envisioned. Most of this knowledge was the result of a decidedly colonial apparatus and discounted the kind of local knowledge that would come to the fore in their more detailed epidemiological studies over the next decade. The isolation of yellow fever was taken by the members of the IHD to be a triumph of the application of a universal technology (the mouse protection test) over the obstacles to its implementation. While they had to make adjustments in everything from the diet of their mice to the methods of collecting specimens, in the end they felt justified in declaring the victory of international health expertise over local conditions and expertise.

In 1941 another pair of maps was made showing the disparity between the range of places where cases of yellow fever infection had been observed clinically and places where there was immunological evidence that infection had occurred, the so-called "silent areas" or

[144] Smithburn to Warren, 18 August 1941, Folder 5, Box 1, Series 477o, RG 1.1, RF, RAC.
[145] Mahaffy, Proceedings of the Conference on Yellow Fever Held Under the Auspices of the Conference of East African Governors at the Medical Research Laboratory, Nairobi, December 9, 1940, TNA, CO 859/63/8.

"predicted zone".[146] Even after five years of work dedicated to validating the results of the mouse protection tests, the Institute had barely made any progress in explaining the second part of the question provoked by these maps—what factors determined the limits of yellow fever distribution. The identification of a region that apparently experienced ongoing endemic yellow fever at the most easterly known limit of yellow fever distribution gave scientists the opportunity to figure out what were the characteristics of that region which made it conducive to yellow fever transmission and thus to determine how transmission could be prevented in the future. In 1941, the *East African Medical Journal* observed that more work would need to be done to determine whether control measures against *Aëdes aegypti* would be either possible or sufficient to control yellow fever outside the urban centers and concluded, "it will be necessary to rely in the first place on inoculation until our entomologists and the research workers at the Institute which owing to wise foresight was established at Entebbe some years ago, can tell us more."[147] As Sawyer and Whitman had put it in their 1936 synthesis of the previous investigations in Africa:

> The zone of high prevalence of immunity affords an exceptional opportunity for an intensive study by epidemiologists, pathologists, bacteriologists, entomologists and zoologist to determine (1) the symptomatology and pathology of the disease produced by the immunizing infection (2) the characteristics of the prevailing strain of yellow fever virus, (3) the identities and habits of the blood-sucking arthropod vectors, and (4) the presence or absence of warm-blooded animal hosts other than man. Persistent studies along these lines should make it possible to estimate the extent of the danger from yellow fever in Central Africa and the probability of its spread to the eastern coast. Such studies should also help to determine what precautionary measures are required."[148]

After the isolations in Sudan, and at the time that isolation from Bwamba was in progress, Warren, on behalf of the IHD, gave Mahaffy and his team the green light to shift their focus away from efforts to isolate yellow fever virus to a broader study of yellow fever epidemiology:

> We are all in agreement that it is no longer necessary to devote practically all of your attention to the isolation of virus and that the epidemic in the Sudan was convincing enough proof that yellow fever, as it occurs in Central Africa, is

[146] "Africa on Guard Against Yellow Fever," Excerpt of the RF Trustee's Confidential Bulletin, February 1942, Folder 6, Box 1, Seresi 477o, RG 1.1, RF, RAC.
[147] "Yellow Fever and East Africa," 408.
[148] Sawyer and Whitman, "The Yellow Fever Immunity Study," 412.

> identical to the disease in other parts of the world. We are also in agreement that the epidemic in Sudan should serve to remove the last traces of doubt in the minds of those individuals who still retain a bit of skepticism as to the validity of the mouse protection test. We are in agreement, therefore, that the next step seems to be an intensive epidemiological study[.][149]

Of course, what they had found was that the disease was not, in fact, identical; the cases in Sudan were much milder from a clinical perspective than those understood to be typical in the Americas. But the point for Mahaffy, Warren, and the rest of the IHD team was that they had ascertained that the virus itself was the same and that the diagnostic tests used to identify yellow fever and make it visible were reliable.

In fact, after so much anticipation of the isolation of yellow fever virus, the event itself seemed anticlimactic for some of the IHD staff. Sawyer, Warren, and Hughes met in the spring of 1941 and agreed "that the program should be continued as otherwise very little would have come out of the whole venture…the emphasis has been too exclusively on finding virus and […] real epidemiological studies should now begin."[150] These epidemiological studies would occupy the Institute throughout the 1940s and are the topic of the next chapter.

[149] Warren to Mahaffy, May 27, 1941, Folder 5, Box 1, Series 477o, RG 1.1, RF, RAC.
[150] WAS Diary May 5, 1941, Folder 5, Box 1, Series 477o, RG 1.1, RF, RAC.

FIGURES

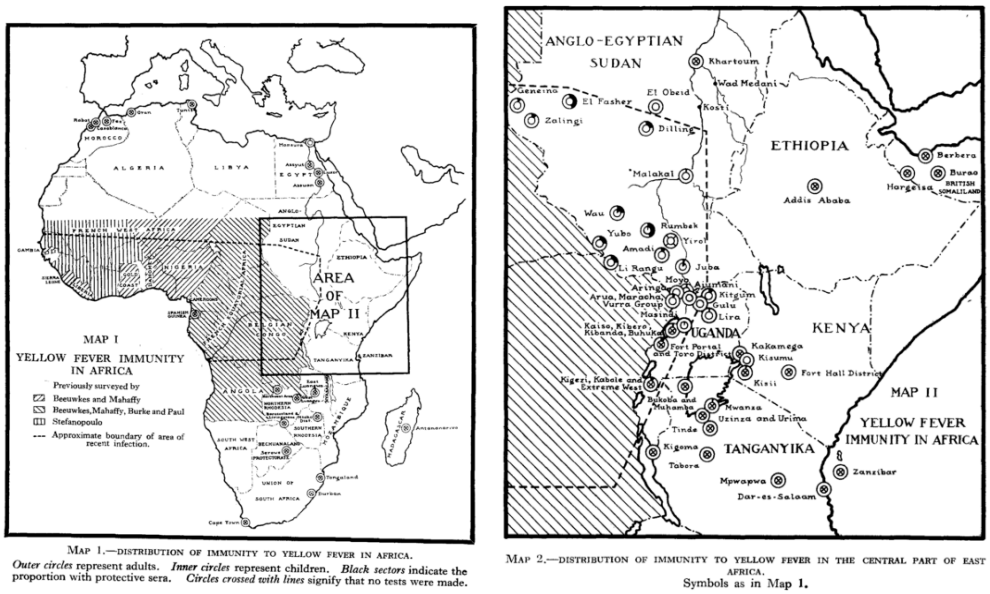

Figure 1a (left) and 1b (right): Maps showing the distribution of immunity to yellow fever in Africa (left) and East Africa (right). From W. A. Sawyer and Loring Whitman, "The Yellow Fever Immunity Study of North, East, and South Africa," *Transactions of the Royal Society of Tropical Medicine* 29, no. 4 (1936): 402-403.

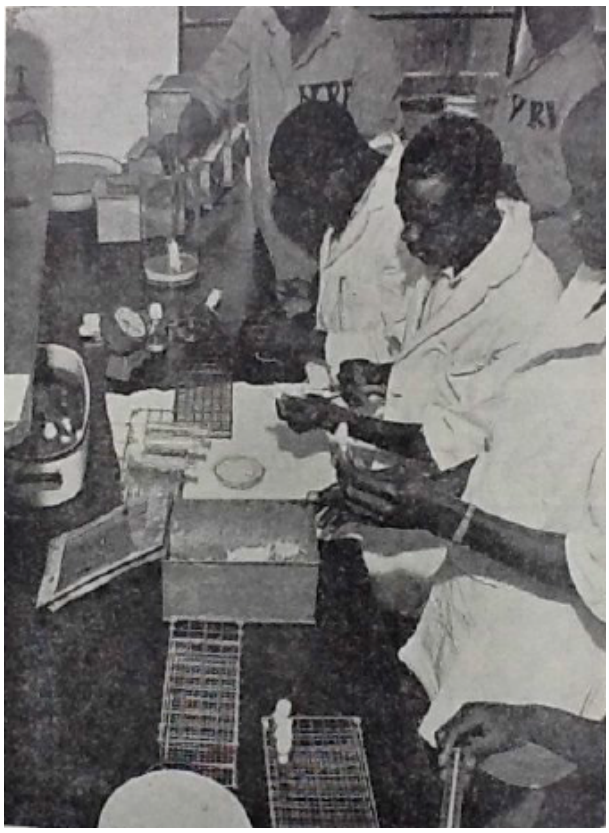

Figure 2: The mouse protection test. The technician at the very bottom of the picture labels the vials containing the serum-virus mixture so that the test could be linked to the location of the sample collection. At the top of the picture a mouse is anesthetized in a jar of ether. Next, a technician injects a starch solution into the mouse's brain and then another technician injects the serum-virus solution. Finally the mouse is placed in a box and the label from the vial is attached to the box. From the *East African Virus Research Institute Annual Report* 1953.

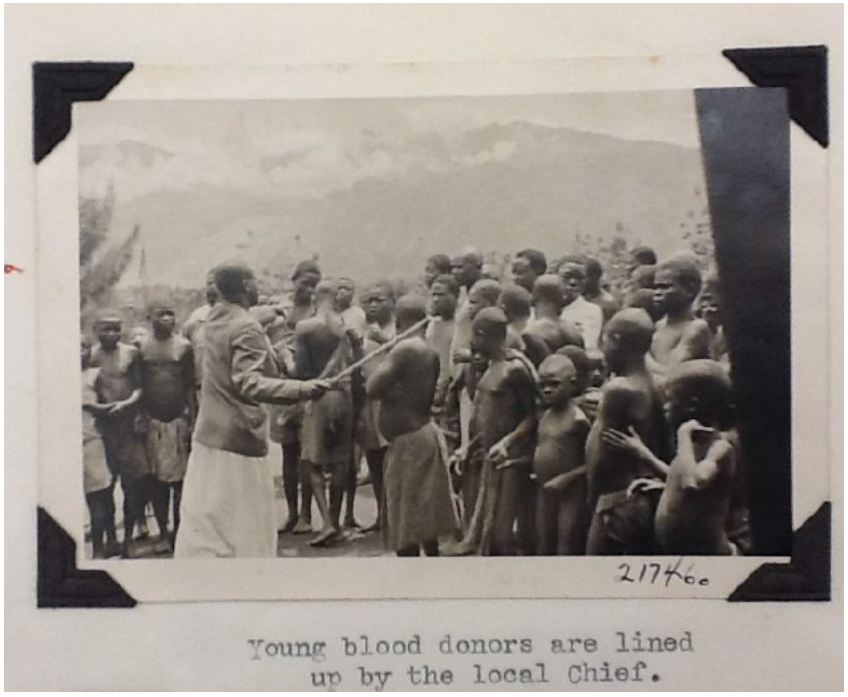

Figure 3: "Young blood donors are lined up by the local Chief." Courtesy of the Rockefeller Archive Center[151]

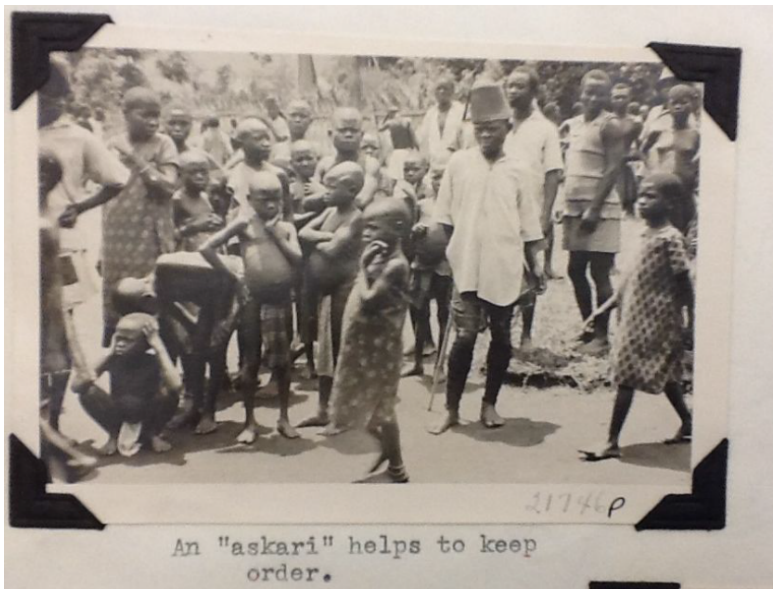

Figure 4: "An 'askari' helps to keep order." Courtesy of the Rockefeller Archive Center.[152]

[151] YFRI Annual Report 1940.
[152] YFRI Annual Report 1940.

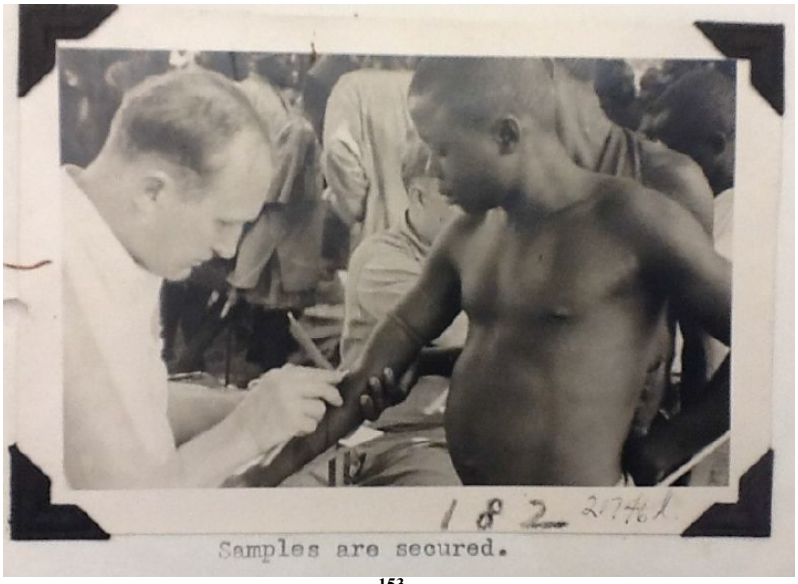

Figure 5: "Samples are secured."[153]

Figure 6: "collecting blood specimens for immunity surveys in Uganda, East Africa."[154]

[153] YFRI Annual Report 1940.
[154] Strode, *et. al, Yellow Fever.* New York: McGraw-Hill Book Company, 1951.

FIG. 8. DISTRIBUTION OF IMMUNITY TO YELLOW FEVER IN UGANDA.
Solid circles denote immunity; blank circles denote no immunity.

Figure 7: Map of the results of immunity tests in Uganda, drawn by Florence Smithburn for a publication by the YFRI staff. Mahaffy, Smithburn, and Hughes, "The Distribution of Immunity."

Chapter Two
"An ecological experiment on the grand scale": Creating an Experimental Field in Bwamba, Uganda, 1942-1950

Introduction

During a visit to Entebbe, Uganda, Dr. Wilbur Downs of the Rockefeller Foundation wrote rapturously, "From the laboratory door one can look out over lacustrine swamps and swamp forests which I know are hopping with viruses."[1] Among the arguments made in favor of establishing the Yellow Fever Research Institute (YFRI) in Entebbe, as discussed in the previous chapter, was its proximity to such forests "hopping with viruses". One such forest, located rather further from the laboratory door, was located in Bwamba District, approximately 350 km west of Entebbe. [Figure 1] This chapter focuses on the field investigations in Bwamba that the YFRI conducted between 1942, the year they succeeded in isolating yellow fever for the first time in East Africa, and 1950 when the Institute was transferred from the Rockefeller Foundation's International Health Division (IHD) to the newly-formed British Colonial Medical Research Service (CMRS). These field studies, largely directed by Scottish medical entomologist Alexander J. Haddow (about whom more will be said below), focused on the nature of relationships between mosquitos, monkeys, humans, and the yellow fever virus in the Bwamba district of western Uganda. Close attention to the material practices of Haddow's research in Bwamba reveals the intensive local engagement between the researchers, the place they studied, and the people that lived there required for the establishment of a productive research environment. In this chapter I will describe the ways that Haddow went about making Bwamba into a suitable place for experimental virology work, including some methods he developed in earlier work in Kenya. I will also discuss how African field assistants, most of whom remain anonymous in the historical record, were critical partners in this scientific place-making enterprise. In this work, like the contemporaneous work of the laboratory in Entebbe, the local

[1] Downs to May, May 20, 1960, Folder 58, Box 7, Series 477, Record Group 1.2, Rockefeller Foundation (RF), Rockefeller Archive Center (RAC).

contingencies of work in Bwamba existed in tension with Haddow's ambition to produce scientific results that would hold up well beyond the limits of Bwamba. Transforming parts of the district into laboratory-like experimental spaces was his way of attempting to control the local qualities of the place so that it could serve as a site of international health knowledge-making.

The metaphor of Africa as a laboratory, first used in the late 19th century, has recently gained currency among historians of the continent.[2] Additionally, there has been growing interest in the history of Africa as an experimental space, where colonial and post-colonial experts alike implemented investigational medical, agricultural, and economic programs.[3] In most cases, these studies have concentrated on human beings as the subjects of these experiments. The history of the field research in Bwamba complicates the narrative of experimentation in Africa by also considering the non-human components of experimental systems in virus research and how those systems linked humans, non-human primates, and mosquitos in an ecological understanding of a particular African place. But in order to disentangle the complexities of these relationships, scientists found it necessary to transform natural places into experimental spaces by importing non-native species, manufacturing artificial dwellings, and manipulating the behavior of human residents.

Haddow's vision of Bwamba was profoundly influenced by the growing science of ecology and the school of thought that emphasized biological systems and the relationships between their component parts. Attention to the physical environment in which disease was transmitted was not new. Existing work on medical research in Africa, particularly the sleeping sickness campaigns of the early 20th century, shows the intimate relationship between environment and health in the colonial period and the ways in which colonial investigations into

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[2] Helen Tilley, Africa as a Living Laboratory: Empire, Development, and the Problem of Scientific Knowledge, 1870-1950 (Chicago: University of Chicago Press, 2011).

[3] Christopher Bonneuil, 'Development as Experiment: Science and State Building in Late Colonial and Postcolonial Africa, 1930-1970,' *Osiris* 15 (2000): 258-281; Melissa Graboyes, *The Experiment Must Continue: Medical Research and Ethics in East Africa, 1940-2014* (Athens, Ohio: Ohio University Press, 2015); Lundy Braun and Evelynn Hammonds, 'Race, Populations, and Genomics: Africa as Laboratory,' *Social Science & Medicine* 67, no. 10 (2008): 1580-1588; Vinh-Kim Nguyen, *The Republic of Therapy: Triage and Sovereignty in West Africa's Time of AIDS* (Durham: Duke University Press, 2010).

local ecologies informed (or failed to inform) their policies and interventions.[4] What Haddow emphasized in addition to these widely held ideas about environment was an interest in experimental manipulation of the living parts of the environment in order to test hypotheses about the transmission of yellow fever and other viruses. In the case of the YFRI's yellow fever research in Uganda, the very act of investigating Bwamba's ecology involved intervening in it—the field site had to be constructed in particular ways to yield useful observations—and African field workers were instrumental in this work as well. People had to be made to enter parts of the forest they normally avoided, different species of monkey had to be introduced to treetops and homesteads in the district, and mosquitoes had to be removed from the forest and transported to the laboratory.

In Bwamba, Haddow conducted collecting work that was critical to the function of the experimental work of the Entebbe laboratory and also undertook investigations that others in the discipline were just beginning to refer to as ecosystems research. But Haddow's approach to this work was not exclusively observational—from the very beginning he attempted to impose experimental conditions on his fieldwork that would, ideally, permit him to reach more universal conclusions from a very particular set of observations in Bwamba.[5] Using published and archival sources to consider the various forms of knowledge production employed in Bwamba and the ways that Haddow attempted to represent the local relationships between viruses, arthropods, and mammals on maps, I argue that Haddow's work transformed Bwamba into a particular kind of place, a place constructed to represent nature and natural relationships, but also one characterized by a number of artificial relationships introduced by Haddow himself. Bwamba was made into a place in which yellow fever, invisible in East Africa, would be visible. This place, in turn, shaped

[4] African auxiliary staff played crucial roles in these investigations, shaping the ways in which British administrators and scientists came to know their African territories. Mari L. Webel, "Mapping the Infected Landscape: Sleeping Sickness Prevention and the African Production of Colonial Knowledge in the Early Twentieth Century," *Environmental History*, Forum: 'From "Natural" to "Artificial" Disease Environments: Technology, Ecology and Human Health 1850-2010,' 20, no. 4 (2015): 722-735.

[5] Following Bruno Strasser, who has suggested that historians of science avoid conflating place with practices and styles of scientific investigation. Bruno Strasser, "Collecting Nature: Practices, Styles, and Narratives,' *Osiris* 27, no. 1 (2012): 310.

his research findings. Haddow's research work in Bwamba was a fundamentally colonial project, in which the organizing, disciplining and reconfiguration of people, their dwelling places, mosquitos, and monkeys was undertaken for the purposes of rendering them into experimental spaces but was also about colonizing Bwamba.

Haddow and the Creation of Experimental Fields

Born in Scotland on December 27, 1912, Haddow showed an early interest in the natural world and its connection to human health and disease. At the age of seven he drew a detailed sketch of a mosquito and titled it "Stegomia auther [sic] of yellow fever greatly magnified."[6] He earned his BSc in zoology from the University of Glasgow in 1934 and completed his MB ChB in 1938, a degree he obtained in order to gain "a broader outlook on the subject [medical entomology]" rather than an intrinsic interest in practicing medicine.[7] He was selected for a three-year Medical Research Council (MRC) junior research fellowship to study tropical medicine at the London School of Hygiene and Tropical Medicine under P.A. Buxton who was working on insect physiology and particularly the relationship between insects and climate.[8] In 1941 he traveled to Kisumu, Kenya where he worked with P.C.C. Garnham and with C.B. Symes, the medical entomologist of Kenya, studying mosquito behavior and the effects of various mosquito repellant substances.[9] In 1942, upon the recommendation of the renowned British medical entomologist P. A. Buxton, Alexander Mahaffy, the director of the YFRI, hired Haddow to run the Institute's field station in Bwamba in 1942 for a yearly salary of 660 pounds.

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[6] P. C. C. Garnham, "Alexander John Haddow, 27 December 1912-26 December 1978," *Biographical Memoirs of Fellows of the Royal Society* 26 (1980): 225-254, quotation on 226.
[7] Garnham "Alexander John Haddow," 225. As another obituary put it, "From the start of his career he [Haddow] was determined never to let his qualifications in medicine interfere with his entomology." J. D. Gillett "Alexander John Haddow 1912-1978," *Antenna* 3, no. 2 (1979): 54.
[8] V. B. Wigglesworth, 1956, "Patrick Alfred Buxton. 1892-1955," *Biographical Memoirs of Fellows of the Royal Society* 2: 69-84.
[9] Garnham, "Alexander John Haddow"; Records of Mosquito Work in Experimental Huts at Kisumu, Books I-III, Papers of Alexander John Haddow, Boxes DC 68/17- 68/19, University of Glasgow Archive Services.

Haddow's subsequent efforts to transform Bwamba into a place that would produce reliable data about virus transmission was apparently influenced by trends that favored the types of experimental authority the lab produced and, like many contemporary biologists, he attempted to bring some of the discipline of experimental science to the field. To use Kohler's vocabulary, he displayed traits of "border biologists" who "operated in the field but by the rules of the lab."[10] Benefitting from the experience of the first generation of such investigators, Haddow never seemed to struggle to reconcile these tendencies, but rather emphasized their mutual utility.

Haddow's experimental field techniques were evident in his earliest work in Africa, before he arrived in Uganda. In the course of his Medical Research Council (MRC)-funded studies of mosquito feeding in the huts occupied by the native population of Kisumu, Kenya in relation to malaria, Haddow was frustrated by the inability to make comparisons between existing huts because "no two were exactly alike."[11] Moreover, the huts themselves were inconvenient for the mosquito collection, crowded with the owners' possessions and built with roofs that were difficult for the catchers to reach.[12] Kenneth Smithburn's wife Florence, an accomplished artist whose work on the YFRI's maps I mentioned in chapter one, painted a picture of two huts in Uganda that illustrates how different these huts, similar to those in western Kenya, could be from one another as well as the form of their roofs which would make mosquito catching work difficult.[13]

Much like other colonial projects, Haddow's experimental designs were intended to impose order on sites and arrangements he perceived to be disorderly. In order to regulate his unruly Kenyan field site Haddow designed experimental huts, which were intended to replicate the types of huts occupied by the local population while incorporating design elements to

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[10] Robert Kohler, Landscapes and Labscapes: Exploring the Lab-Field Border in Biology (Chicago: University of Chicago Press, 2002), 176.
[11] Alexander J. Haddow, "The Mosquito Fauna and Climate of Native Huts at Kisumu, Kenya," *Bulletin of Entomological Research* 33, no. 2 (1942): 91-142, 93.
[12] Alexander J. Haddow, "Studies on the natural history of yellow fever in East Africa with notes on other insect-borne infections," (D.Sc. Thesis, Glasgow University, 1957), 4.
[13] Florence Smithburn, "Old hut and new hut," watercolor on paper, 1946, http://collection.imamuseum.org/artwork/60032/, accessed January 29, 2017.

facilitate measurement and collection while also guaranteeing that comparisons between huts could be made with confidence.[14] Using thermographs and hygrographs, temperatures and humidity in the experimental huts were scrupulously documented, as was the relationship between these conditions and the occupation of the huts.[15] Anemometer readings at the nearby Kisumu airport allowed him to account for changes in the wind as they might relate to the experiments in progress.[16] These experiments concerned a range of factors that Haddow and his colleagues proposed might relate to the population and biting behavior of various mosquito species inside the huts, including rainfall, ambient temperature and humidity, the number, size, and age of the hut's inhabitants, whether the inhabitants were more or less dirty, and whether sweat on unwashed clothing attracted mosquitoes even when there was no human bait.[17] They were also used to measure the effect of pyrethrum powder on indoor mosquito populations.[18] The results of these investigations, he noted, could be of importance both for disease control measures and for the design and conduct of further research.[19] Of course it also meant that what he was measuring was not, in fact, the behavior of mosquitos in native huts, but the behavior of mosquitos in simulacra of native huts. This distinction does not appear to have concerned

[14] Haddow, "The Mosquito fauna and climate," 93. These huts were so successful that they led to a variety of other investigations related to the indoor behavior of mosquitos. This effort to regulate indigenous domestic spaces was consistent with colonial projects across Africa and beyond. Nancy Rose Hunt, *A Colonial Lexicon: Of Birth Ritual, Medicalization, and Mobility in the Belgian Congo* (Durham, Duke University Press, 1999); Bernard Cohn, *Colonialism and its Forms of Knowledge: The British in India* (Princeton: Princeton University Press, 1996); Alison Bashford, *Imperial Hygiene: A Critical History of Colonialism, Nationalism and Public Health* (New York: Palgrave MacMillan, 2004); Warwick Anderson, *Colonial Pathologies: American Tropical Medicine, Race, and Hygiene in the Philippines* (Durham: Duke University Press, 2006). Public health in England also had a long history of advocating for the orderliness of domestic spaces. Graham Mooney, *Intrusive Interventions: Public Health, Domestic Space, and Infectious Disease Surveillance in England, 1840-1914* (Rochester: University of Rochester Press, 2015).
[15] "Record of Mosquito Work in Experimental Huts at Kisumu," University of Glasgow Archives, DC 68/17, DC 68/18, and DC 68/19; Haddow, "The Mosquito fauna and climate," 99.
[16] Haddow, "The Mosquito fauna and climate," 99.
[17] Haddow, "The Mosquito fauna and climate," 116-122. In at least one case the Africans serving as human bait refused to participate in an experiment that involved measuring mosquito populations in a hut occupied by more than 10 people as the 'nuisance factor' of the large mosquito population (and presumably the discomfort of being crowded by other people) was too much. Haddow, "Studies on the natural history," 7. But in other cases human baits withstood conditions that Haddow reported as "(subjectively) […] miserable". Haddow, "Studies on the natural history," 7, 82.
[18] In these experiments African assistants was positioned as "bait" in a control hut and in an experimental hut dusted with the insecticide. C. B. Symes, J. McMahon, and A. J. Haddow, "Pyrethrum Powder: A Preliminary Note on its Use in the Control of Insect Vectors of Disease," *The East African Medical Journal* 18 (1942): 360-375. Insecticide research was a major concern as Allied troops moved through parts of the globe where malaria and other insect-borne diseases were endemic.
[19] Haddow, "The Mosquito fauna and climate," 121.

Haddow who was apparently satisfied that his experimental huts were sufficiently similar to the 'natural' huts for his results to be relevant. This was the first case of Haddow's quest to impose sufficient standardization on ostensibly "natural" field sites for them to efficiently generate knowledge that could be generalized to other, similar sites. This was an aspect of his approach to scientific research that would shape his work in Bwamba in subsequent years.

In January 1942 Haddow traveled to Entebbe to join the staff of the YFRI.[20] At the time, the YFRI was expending large amounts of effort on testing and distributing yellow fever vaccine for Allied troops in Africa. The war strained their resources, but their ongoing studies were still a priority as they were expected to help curb the spread of the virus beyond its existing limits. Like the overall mission of the Institute, which was to understand yellow fever epidemiology for the purposes of protecting American and European interests in Africa and the Far East, the vaccine program was principally about protecting Allied troops, not African communities in endemic regions. Mahaffy's agenda for Haddow's first month was to "spend the remainder of the month in Entebbe becoming yellow fever minded […] and generally preparing himself for the field problem in Bwamba."[21] In part, becoming "yellow fever minded" involved becoming more familiar with the work of the laboratory-based side of the Institute's research. In the fall of 1942 Haddow spent several weeks in Entebbe clerking in the laboratory's mouse room in order to familiarize himself with that aspect of the research program.[22] Haddow made a good first impression on his laboratory-based colleague Kenneth Smithburn, who wrote that Haddow was "a very valuable addition to our staff."[23] An effective field operation was critical for Smithburn's laboratory-based efforts, and he was hopeful that Haddow would be able to run such an

[20] Research on the prevention and control of insect-vectored diseases in general, and yellow fever in particular, was considered critical to the war effort, with large numbers of susceptible individuals being mobilized for service in north, central, and east Africa.
[21] AFM (Alexander Francis Mahaffy) Diary, Monday January 5, 1942, Box 275, Record Group 12, Rockefeller Foundation (RF), Rockefeller Archive Center (RAC).
[22] AJH (Alexander John Haddow) Diary, 12-30 November 1942, Box 191, Record Group 12, RF, RAC.
[23] Smithburn to Warren, July 14, 1942, Folder 6, Box 1, Series 477o, RG 111, RF, RAC.

operation.[24] His time in the laboratory also informed Haddow's approach to experimental work in the field.

Putting Bwamba on the Map

Before Bwamba could be made into a place where viruses were investigated, it had to be defined. From the beginning, Bwamba as a place was an artificial colonial product. Haddow himself noted that it really comprised at least three distinct ecological zones, only one of which he thought was inhabited densely enough to merit study.[25] Neither was it a coherent political entity. Bwamba was home to several different groups of people, many of whom were closely related to people on the far side of the border in the Belgian Congo.[26] In fact, until 1912 when the boundary between Uganda and the Belgian Congo was finalized, it occupied a "no-man's land" between the two colonies, though the British included it within the Toro kingdom.[27] The presence of Europeans in Bwamba was minimal in the 1930s and '40s. Occasional visits from a Catholic priest and various colonial officers were the exception and the only Europeans who stayed for extended periods during this time were the YFRI employees.[28]

Bwamba's selection as a field site for the YFRI was prompted by the discovery of a large proportion of immune individuals in the first years of the survey and a large project to construct a road from Fort Portal to Bundibugyo in the late 1930s. Since the early 1930s the IHD had been testing individuals across the region for yellow fever immunity in an effort to determine where the disease was endemic. They used a relatively new technique, the mouse protection test, to

[24] Smithburn was using mosquitos collected in the field to attempt to determine which species carried yellow fever. After the mosquitos were sorted by species, they were ground up and injected into experimental mice, which were then observed for the characteristic signs of yellow fever infection. Other experiments attempted to establish which infected mosquitos could infect mice through their bites and thus were suspects for transmission. These experiments were intended to determine which mosquito species were most important for yellow fever transmission.
[25] Haddow, "On the Mosquitos of Bwamba County I," 1-2.
[26] Edward H. Winter, *Bwamba: A Structural-Functional Analysis of a Patrilineal Society* (Cambridge: East African Institute of Social Research, 1956); Edward H. Winter, *Beyond the Mountains of the Moon: The Lives of Four Africans* (Urbana: The University of Illinois Press, 1959).
[27] Winter, *Beyond*, 3.
[28] Winter, *Bwamba*, 7.

detect yellow fever virus antibodies in human serum.[29] The presence of many immune individuals of different ages in Bwamba indicated recent virus transmission. The road project brought large numbers of laborers into areas of the forest where they would be exposed to viruses to which they were not already immune. It also permitted the YFRI to keep those individuals under fairly close observation.[30] The YFRI hoped to take advantage of the concentration of a large number of yellow fever-susceptible men in an area with known circulation of the virus and the means to observe them on a regular basis in order to isolate yellow fever virus in a person with an active infection. This effort was unsuccessful, but the completion of the road made Bundibugyo a much more attractive research site than it had been when it was only accessible by foot.[31]

The new field station, located at Bundibugyo in the Bwamba district of western Uganda in the foothills for the Rwenzori Mountains, officially opened on March 6, 1942.[32] One of Haddow's first tasks when he first arrived in Uganda was to map Bwamba with "sufficient accuracy for epidemiological purposes."[33] Haddow's maps allowed him to control for and manipulate the variations in natural and human geography in Bwamba that affected his experimental work. Previous maps were approximate at best and indicated only the major landmarks such as main roads and towns.[34] In April and May of 1942 Haddow led the effort to create a map at a scale of 1:50,000 covering the whole of the region. By the end of 1942 the map was in its 5th revision.[35] In 1943 Haddow collaborated with the Forest Department to conduct a

[29] For more on the mouse protection test, see Chapter One.
[30] Kennth C. Smithburn, Alexander F. Mahaffy, and John H. Paul, "Bwamba Fever and its Causative Virus," *American Journal of Tropical Medicine and Hygiene* 21, no. 1 (1941), 75.
[31] Annual Report for 1973, Paris Office-International Health Division, Section III-Uganda Yellow Fever, Folder 2920, Box 242, Series 700, RG 5.3, RF, RAC.
[32] Bwamba was selected as a site for intensive field studies because it yielded a number of yellow fever antibody-positive human serum samples in the late 1930s. Bwamba was loosely bounded by the Rwenzori mountain range to the east, its watershed to the southeast, the Lamia River to the west, the grassland of the Semliki Flats to the north, the Semliki River marking the border with the Belgian Congo to the west, and the Lamia River (also the international border) to the south. Winter, *Beyond*, 2.
[33] "Yellow Fever Research Institute, Entebbe, Uganda, Annual Report 1942," Folder 2601, Box 211, Series 477o, RG 5.3, RF, RAC.
[34] "Yellow Fever Research Institute, Entebbe, Uganda, Annual Report 1939," in the "European Annual Report 1939" vol. II. Folder 2941, Box 243, RG 5.3, RF, RAC.
[35] "Uganda Yellow Fever Research Institute 1st Semi-Annual Report for 1942," Folder 2600, Box 211, Series 3, RG 5, RF, RAC.

more detailed survey of the forest around the Semliki River.[36] Map-making consistently figured high among Haddow's priorities throughout his time in Bwamba. The volume of his cartographic products and the amount of time he committed to them makes it important to consider their significance for his research program and the means by which he produced them.

Maps are instruments of power, particularly in imperial and colonial sites. They were also central to the way tropical medicine came to be understood and investigated.[37] Just as modern statecraft requires maps to make populations "legible", the YFRI's research relied on maps to make Bwamba intelligible to foreign scientists.[38] Haddow's work was predicated on the belief that knowledge of which zoological, botanical, and meteorological phenomena coincided with immunity to yellow fever could direct field and laboratory investigations in the right direction. He and his colleagues saw their research as an extended project that would elucidate the nature of yellow fever (and other arboviruses) by discerning the relationships between the virus, its invertebrate vectors, and its animal and human hosts. Maps were critical for this work, allowing him to visually inspect the relationships between various data related to human, primate, and insect immunity, the physical landscape, and other factors.[39] Haddow's maps demonstrate an intimate acquaintance with Bwamba district. For example, he drew one map showing the locations of children who tested positive for yellow fever immunity in relation to the distribution of forest cover (as opposed to swamp, cultivated land, and other types of landscape).[40] Other maps showed YFRI campsites, rest houses, dispensaries, physical features like forests, swamps, rivers, and peaks, and native paths [Figure 1]. The richness of these maps is especially notable

[36] Haddow, "Studies on the natural history," 69.
[37] Warwick Anderson, "Natural histories of infectious disease: ecological vision in twentieth-century biomedical science," *Osiris* 19 (2004): 39-61; Historians have been especially attentive to the use of sleeping sickness maps. Kirk Arden Hoppe, *Lords of the Fly: Sleeping Sickness Control in British East Africa, 1900-1960* (Westport, CT: Praeger Publishers, 2003); Helen Tilley, "Ecologies of Complexity: Tropical Environments, African Trypanosomiasis, and the Science of Disease Control in British Colonial Africa, 1900-1940," *Osiris* 19 (2004): 21-38; Webel, "Mapping the Infected Landscape."
[38] James Scott, *Seeing Like a State: How Certain Schemes to Improve the Human Condition Have Failed* (New Haven: Yale University Press, 1998).
[39] Webel describes similar "sketch maps" used by sleeping sickness researchers in German East Africa though it is not clear that they contained a comparable amount of local detail. Webel, "Mapping the Infected Landscape," 727.
[40] Figure 1 in Haddow, Smithburn, Mahaffy, and Bugher, "Monkeys in Relation to Yellow Fever in Bwamba County, Uganda," *Transactions of the Royal Society of Tropical Medicine and Hygiene* 40, no. 5 (1947): 677-700.

when contrasted with a contemporary map of the Bwamba District by the Directorate of Colonial Surveys showing only rivers and roads with some paths and tracks, based on air photography "without ground control."[41] Haddow's far more detailed maps permitted him to define Bwamba as an experimental space, imposing a new reality on the landscape distinct from that of the indigenous residents or colonial administrators.

Mosquito Investigations

Haddow and his team conducted extensive investigations into mosquito behavior. As scientists with extensive field experience, Haddow and Gillett were more cautious than many of their colleagues about extrapolating laboratory findings to nature. Remarking on the problems with many lab-based studies of mosquito predation, Gillett wrote, "Conditions in the laboratory usually bear extraordinarily little resemblance to those in nature."[42] For them, the experiments they conducted in the field occupied a position closer to true nature than those conducted in the lab. For example, as part of his ongoing work to describe the habits and life-cycles of potential yellow fever vector insects, Haddow arranged experiments to compare the attractiveness of children, goats, monkeys, baboons, and fowl for different species of feeing mosquitoes.[43] As he explained one such experiment in his official diary, "[I d]ecided to make a catch using a unit of 6 fowls," to complement an earlier study comparing mosquito feeding on "our smallest toto, two baboons, and 2 small goats."[44] He described this addendum to the protocol as "not quite critical, as the catch will not be simultaneous with the others already done, but if the result is fairly definite I think it can be accepted. Shall do this catch at the same place and hour as the previous catches. Shall then repeat the whole thing using all four baits together."[45] Experiments could be

[41] Map of Bwamba District, Sheets 1-4 Directorate of Colonial Surveys, 1948, British Library.
[42] Gillett, *Mosquitos*, 132.
[43] AJH Diary, 10 June 1942, Box 191, RG 12, RF, RAC.
[44] AJH Diary, June 8 and June 10 1942, Box 191, RG 12 RF RAC. A "toto" (from the Swahili word *mtoto*) is a child. Haddow and other YFRI scientists referred to African children as "totos" and frequently referred to the African men they employed as "boys".
[45] AJH Diary, 10 June 1942, Box 191, RG 12, RF, RAC.

designed to confirm observations made casually during fieldwork. For example, while working along the Semliki River in 1941 Gillett observed that *A. simpsoni* seemed to prefer biting him and his African companions on the head.[46] In June of the following year, Haddow arranged an experiment with six totos "to determine whether simpsoni really prefers the head or whether it merely likes to bite well above the ground. The catch showed that it bites the head in preference to the body in any posture—even if the bait is lying down."[47] The results of these experiments held greater epistemic power for Haddow and his colleagues than simple observations of mosquito activity near children, goats, and other animals in their everyday activities. Like Haddow's experimental huts in Kenya, these experiments represented efforts to coax what he sometimes called "critical" results from disorderly field conditions by standardizing and organizing apparently disorderly arrangements of people, animals, and places.

At the same time, Haddow's maps also connected the lab to the field, linking the Bwamba-based mosquito collection work to the laboratory-based work of Smithburn and his colleagues in Entebbe. These mosquitoes, once tested for yellow fever virus in the laboratory and documented on the maps, allowed the YFRI team to "see" areas of yellow fever activity in Bwamba when they couldn't detect signs of sickness in people or other animals. The workers at Bwamba were responsible for supplying the raw material for much of the lab's testing. Mosquitos were collected, identified, separated by species, and sent to the lab to test for yellow fever virus. The locations in which the mosquitos were collected were carefully documented so that any positive batches could be properly attributed to their origin and further investigations could take place in those locations. Haddow refined the methods used for collecting mosquitos over time. At first he recruited large groups of people, up to 100, to serve as "stationary bait" while trained catchers moved among them and captured mosquitos as they attempted to feed.[48] Subsequently he found that it was equally effective and far more efficient to train catchers who collected on a

[46] Gillett, *Mosquitos*, 87.
[47] AJH Diary, 26 June 1942, Box 191, RG 12, RF, RAC.
[48] Haddow, "Studies on the natural history," 53.

mobile basis, collecting mosquitoes that were attracted by their disturbance of the brush and undergrowth.[49] Far more than passive bait, these catchers gained considerable skill at their given task and after several months the team of 20 had increased their catching rates from 50-100 mosquitoes/day to up to 1000 mosquitoes in only a morning.[50]

Invisible Entomologists

Haddow was able to make yellow fever visible by employing a large staff of largely unacknowledged ("invisible") African assistants. Haddow invested time and energy in training local staff in his methods so that they could assist him in the creation of experimental environments in Bwamba. Haddow's outstanding dedication to his work and enthusiasm for long stretches of time in the field notwithstanding, his research agenda was entirely dependent on the work of skilled African assistants. The work of African employees permeates the records of daily activity at the Institute, both in the field and in the laboratory. One man in particular, Yovani Ssenkubuge, came to be Haddow's right-hand-man, taking on increasingly responsible roles in the field station, managing the mosquito catches and managing the field station in Haddow's absence. Ssenkubuge alone appears regularly in the records of Haddow's activities. He was a co-author on eight papers published between 1962 and 1976 and was acknowledged by name in many more.[51] One of the Institute's entomologists, J.D. Gillett described him as "a tireless and experienced worker whose splendid example did so much to ensure the success of…experiments in the forest."[52] Luis Mukwaya, the Institute's first Ugandan entomologist, credits Ssenkubuge with training him in mosquito work, allowing the Institute to maintain some continuity in skills

[49] Haddow, "Studies on the natural history," 54. Smithburn and Haddow, "Isolation of yellow fever virus from African mosquitoes," *The American Journal of Tropical Medicine and Hygiene* 1, no. 3 (1946): 261-271.
[50] Haddow, "Studies on the natural history," 54.
[51] E.g. Philip S. Corbet, "Entomological Studies from a High Tower in Mpanga Forest, Uganda. IV. Mosquito Breeding at Different Levels in and Above the Forest," *Transactions of the Royal Entomological Society of London* 113, no. 11 (1961): 275-283.
[52] Gillett, *Mosquitos*, 95.

and methods when most of the expatriate scientific staff left in the mid-1960s.[53] In another exceptional case, American anthropologist Edward H. Winter interviewed one of Haddow's Amba workers for a life history.[54] But the majority of African fieldworkers remained anonymous in the written records of the Institute.

In June of 1942 Haddow reported that he had spent the last month training three of the more senior assistants in identification of adult mosquitos and the results were encouraging enough for him to contemplate a more basic course for some of the other assistants.[55] Sometimes the question of training was more about acculturating Africans to the scientific lifestyle as it was about learning particular techniques. Haddow's decision to conduct 24-hour catches in the forest ran up against a major obstacle when he found that his African field workers were deeply averse to spending the night in the forest. The fieldworkers' reluctance to spend the night in the forest is a reminder that local communities had their own forms of knowledge of places in Bwamba. The forest represented a site of both physical and spiritual danger in the cosmology shared by the Amba with their neighbors in the Belgian Congo.[56] Without, perhaps, fully understanding the reasons for their hesitation, Haddow undertook a series of shorter night-time catches for the purposes of gradually making them accustomed to the practice.[57] In order to make the night-time forest a site for research he had to alter the relationship between people and the location. Because humans weren't part of the cycle that Haddow was investigating at the time, he did not apparently consider this a problem.

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[53] Gladys Kalibbala, "Dr. Mukwaya, Uganda's most renowned malaria researcher," *New Vision,* April 26, 2012, accessed April 30, 2014, http://www.newvision.co.ug/news/630627-dr-mukwaya-uganda-s-most-renowned-malaria-researcher.html.

[54] Winter, *Beyond the Mountains of the Moon.* Mpuga (a pseudonym Winter gave to the worker) described the circumstances in which he came to work for the project, his understanding of the project's objectives, his responsibilities, and the relationship between temporarily hired laborers, more senior African field superintendents, and Haddow. Winter conducted his interviews in the early 1950s in the abandoned premises of the YFRI field station and his interpreter, Felix Rwambarali, was Mpuga's former supervisor at the YFRI complicating any interpretation of Mpuga's account. Nonetheless, the life history offers one of very few opportunities to capture an indigenous perspective on the work of the YFRI team in Bwamba.

[55] AJH Diary, 4 June 1942, Box 191, RG 12, RF, RAC.

[56] Randall Packard, *Chiefship and Cosmology: An Historical Study of Political Competition* (Bloomington: University of Indiana Press, 1981).

[57] Haddow, "Studies on the natural history," 65.

Haddow rarely reflected on the very real changes his own experimental interventions occasioned in the relationships between people, animals, viruses, and the environment. However, Haddow was keenly aware that changing human behavior could have real consequences for yellow fever epidemiology. Moreover, places in Uganda were changing rapidly in this period and Bwamba was no exception. By the mid-1940s Haddow had observed that, there were a number of non-immune 'squatters' arriving in the region from the Congo who would be vulnerable to yellow fever infection.[58] Not content to passively observe the changing dynamics of human-monkey-mosquito ecologies in Bwamba over time, Haddow concocted an experiment that he thought the YFRI could conduct to simulate these changes in a controlled environment and so answer the question, "What happens when a native moves into a patch of primary forest, clears it, builds his hut and plants his crops?" He suggested establishing an experimental strip from which preliminary baseline measurements of mosquito prevalence and behavior and primate population that would then be divided into a control zone and an experimental zone. Then, Haddow suggested:

> Employ a local native with wife and children to move into the experimental strip. Let him begin clearing and building his hut. Study any changes in the fauna—Do the arboreal mosquitoes increase in incidence at ground-level when the trees are felled? Do they persist in the clearing or move back to the forest? Do they spread through the clearing by night? Do new species appear?

and a long series of additional questions such an experiment could address.[59] He was careful to recommend that the Institute not employ any of its own African assistants as "they have been so much in contact with our own ideas that they may not behave quite as other natives would under the same circumstances."[60] Haddow was aware that his own influence on the behavior of his field staff was changing the way they interacted with their own environment. While there is no

[58] AJH to AFM September 7, 1945, Folder 9, Box 2, Series 477o, RG 1.1, RF, RAC. Haddow's use of the term "squatters" overlooks or ignores the fact that the Amba population on the Ugandan side of the Semliki was closely related to a large Amba community on the Belgian side of the border. Edward Winter, "The Aboriginal Political Structure of Bwamba," in *Tribes Without Rulers: Studies in African Segmentary Systems,* John Middleton and David Tait, eds. (London: Routledge & Kegan Paul, 1958), 137. During the time that Haddow worked in Bwamba, there was a large flow of immigrants from the Belgian Congo into Bwamba. Winter, *Bwamba*, 233-234.
[59] AJH to AFM September 7, 1945, Folder 9, Box 2, Series 477o, RG 1.1, RF, RAC.
[60] AJH to AFM September 7, 1945, Folder 9, Box 2, Series 477o, RG 1.1, RF, RAC.

evidence that he ever conducted this experiment, Haddow's proposal highlights what would be a persistent concern for virus researchers in Uganda—how the relationship between human disease and geography could continue to be studied when people were always moving. For Haddow, humans could only meet the experimental requirements of his work if they were employed to perform particular tasks, even if those tasks were designed to mimic "natural" human behavior. Left to their own devices, humans didn't behave with sufficient predictability for his purposes of using Bwamba as a laboratory for yellow fever studies. By 1951 Haddow and his colleagues were inclined to rely less on human immunological surveys for the very reason that humans, unlike monkeys, could not be relied upon to be "truly resident" in the area in which they were tested.[61]

Vertical Reconnaissance

In order to describe the "natural" relationship between people, other primates, and the forest environment, Haddow had to manipulate the forest canopy and the place of humans and monkeys in it. In late 1943, having received a "progress note" from the IHD group in Colombia reporting that yellow fever was routinely found in mosquito species inhabiting the forest canopy, Haddow worked to reorient his own work in Bwamba.[62] He designed an experiment to test the prediction that one of the species of mosquitos known to transmit the virus would be found feeding on primates in the canopy.[63] Just as the 24-hour catch allowed him to see which mosquito species intersected with immune monkey species chronologically, Haddow needed a strategy that would permit him to assess which species intersected spatially—specifically in terms of vertical distance from the ground. The first obstacle he encountered in the study of mosquito behavior in the forest canopy was the reluctance of his Amba staff to climb trees. Faced with the truly vertiginous

[61] Alexander Haddow, Kenneth Smithburn, Alexander Mahaffy, et al., "Monkeys in Relation to Yellow Fever in Bwamba County, Uganda," *Transactions of the Royal Society of Tropical Medicine and Hygiene* 40, no. 5 (1947): 677-700. The mobilization of adult men during WWII and travel in search of wage labor contributed to the perceived displacement of African people in this period, which made it difficult for YFRI scientists to locate foci of yellow fever activity using human serosurveys.
[62] Haddow, "Studies on the natural history," 85.
[63] J.D. Gillett, *Mosquitos*, 95.

prospect of ascending into the canopy, they balked.[64] Adopting an approach similar to his gradual introduction of night work, he decided to begin work on low platforms and gradually increase their height.[65] This project was more than just an observational exercise—it was, as entomologist J.D. Gillett wrote, "a true experiment in the usually accepted scientific sense, as rigorous in its discipline as any carried out at the bench—an ecological experiment on the grand scale."[66] Gillett was responding to the universalizing ambition of Haddow's program—Haddow was not satisfied to report on a limited set of observations but wanted to be able to make a statement about the behavior of particular mosquito species in general.

By the end of 1942 the field station had collected a total of 25,038 mosquitoes and Haddow had trained a cadre of skilled African assistants, finished building the camp, established systems for transporting specimens and materials between Entebbe and the field station, and made a map of the district.[67] Twelve months later, field work in Bwamba had also led to what Haddow considered to be a fundamental change in the conceptualization of yellow fever in the IHD. From an anthropocentric picture of a virulent virus which sometimes manifested in milder forms, scientists had moved to a picture of the virus as essentially, "a mild enzootic infection of monkeys which, from time to time 'escapes', as it were, into the human population, more or less by chance."[68] This reorientation of yellow fever studies shifted much of the focus from human serological results to animal studies.

Wild Monkeys

Wild monkeys played a variety of roles in the YFRI's research. For one thing, they were a source of blood for protection tests that would inform the maps of yellow fever prevalence.

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[64] This wasn't an irrational fear—Haddow's colleague William Lumsden described one man's fall from a height of about 40 feet, though he claimed that Tomasi Nkbuwa, the man in question, was "little injured except for his pride." WHL diary, August 6, 1948, Box 273, RG 12, RF, RAC.
[65] Haddow, "Studies on the natural history," 86. Later he refined this technique by investing in a pair of towering steel structures that penetrated all the way to the top of the forest canopy and permitted simultaneous catching on various levels.
[66] John D. Gillett, *Mosquitos* (London: Weidenfeld and Nicolson, 1971), 96.
[67] Haddow, "Studies on the natural history," 58.
[68] Haddow, "Studies on the natural history," 73.

These maps, versions of which Haddow prepared in his first year at the Institute, relate the human serological results to the distribution of red tail monkeys, colobus monkeys, and other "minor" monkey species.[69] The data on these maps are the result of not only meticulous record keeping and laboratory procedure, but also skilled hunting.[70] In order to conduct tests into the immunity of monkeys in the forests of Bwamba (and their role in transmission of yellow fever to humans) it was necessary to get blood specimens from large numbers of monkeys. At first Haddow and his team attempted to trap or purchase living monkeys, but they found it very difficult to lure monkeys to a baited trap when food was locally abundant.[71] Soon they turned to hunting, and much ink was spilled on the methods needed to kill a monkey and secure a blood specimen before the heart stopped beating.[72]

By 1951 the staff of the Institute had performed yellow fever protection tests on blood from 1,049 monkeys procured by purchase, capture, or hunting across southern and western Uganda.[73] An inventory of species, the location in which they were collected, and the individuals responsible for their capture indicates that most of the European senior staff of the Institute, as well as a number of "native trappers" contributed to this endeavor at one time or another. It seems likely that this was as much a form of recreation for the Europeans as a scientific enterprise and was often undertaken "after hours".[74]

The contents of Haddow's field kit in a list he published for the information of other zoological researchers illustrate his efforts to bring some degree of laboratory precision to his field work: containers for blood specimens, instruments for labeling samples, a variety of knives and scissors for dissecting the monkey carcasses and equipment to sharpen them in the field, a

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[69] Figures 2,3, and 4 in Haddow, Smithburn, Mahaffy, and Bugher, *Transactions of the Royal Society of Tropical Medicine and Hygiene,* 1947. YFRI Annual Report for 1942.
[70] Haddow, Smithburn, Mahaffy, and Bugher 1947.
[71] Haddow, "Studies on the natural history," 58.
[72] Haddow, Smithburn, Mahaffy, et al., "Monkeys in Relation."
[73] Alexander Haddow, George W. A. Dick, William H. R. Lumsden, et al., "Monkeys in Relation to the Epidemiology of Yellow Fever in Uganda," *Transactions of the Royal Society of Tropical Medicine and Hygiene* 45, no. 2 (1951): 189-224. Results from 20 monkeys taken by non-Institute staff brought the total results analyzed to 1069 unique specimens.
[74] Haddow, Dick, Lumsden, and Smithburn, "Monkeys in relation," 189-224.

spring balance, a measuring tape, aluminum tags for the skull, skin, and other large specimens, tubes with preservative solutions for pathological specimens, microscope slides and alcohol to fix the blood films, twine, and a record book.[75] With the proper tools, Haddow believed, the field, like the lab, could be controlled sufficiently to produce data that would travel unimpaired by the local particularities of its collection.

Sentinel Monkeys

One of the most ambitious of Haddow's interventions in Bwamba was the sentinel monkey program. The Institute's extensive surveys of immunity among various monkey species strongly suggested a 'jungle' or non-human cycle of yellow fever.[76] The isolation of yellow fever virus from a species of mosquito known as *Aedes africanus* established it as the prime suspect of transmission among monkeys. However, conclusive evidence of the cycle's dynamics had to come from the observation of an epizootic, or an epidemic among animals, in progress. By the mid-1940s, the YFRI team was stymied in its efforts to isolate yellow fever virus from indigenous primates by the fact that those animals didn't display symptoms of infection that would alert observers to the existence of an epizootic. In 1945 the YFRI team made one last intensive effort to isolate virus from mass collections of mosquitoes, even transferring some of the duties of the Entebbe lab to the field station when specimen transport became a major obstacle.[77] But the effort was in vain and the team needed to find a better way forward. As Smithburn, put it, "The principle obstacle to the solution of the problem lay in finding a locality in the forest where the virus was active at any particular time. There were no natural indicators to point to such an area,"

[75] Alexander J. Haddow, "Field and laboratory studies on an African Monkey, *Cercopithecus ascanius schmidti* Matschie," *Proceedings of the Zoological Society of London* 122, no. 2 (1952): 301.
[76] The YFRI staff generally avoided using the term "jungle yellow fever" with reference to their studies because, strictly speaking, jungle yellow fever was defined as yellow fever in the absence of *anopheles*, which were abundant in Bwamba. However, they were looking for an analogous cycle of yellow fever that would explain its persistence in the absence of susceptible human hosts—one that would involve mosquitoes, monkeys, and possibly another vertebrate host. It was common for people to refer to any such cycle as a jungle cycle.
[77] Haddow, "Studies on the natural history," 103.

so the Institute had to make its own.[78] They decided to deploy a set of rhesus monkeys as "sentinels" in various locations throughout Bwamba including farms, alongside roads, and on platforms in the treetops.

In short, in order to expose Bwamba's naturally-occurring yellow fever epizootics, the YFRI had to introduce an entirely new species of monkey as well as people to tend to it. Their presence in the forest was intended to simulate reality but in such a way that would make the interaction between virus, mosquitos, and monkeys visible. The YFRI initiated the sentinel monkey program in August 1945. The sentinel monkeys were canaries in the coalmine for yellow fever researchers.[79] The idea was to establish a system for rapidly identifying areas where yellow fever was being actively transmitted. Like sensor systems that alert seismologists to earthquakes, the sentinel monkeys alerted the YFRI staff to regions where there was current viral activity. Unlike the protection test results or the testing of batches of mosquito collections for virus, the sentinel monkeys could make an outbreak of yellow fever visible in real time, not only in retrospect. The sentinels were all rhesus monkeys, bred at the Institute or imported, because local monkey species showed no observable effect of yellow fever infection while rhesus monkeys would develop a fever and other signs of illness when infected.[80] They were stationed in forests on tree platforms in a wide variety of forest types at fixed points where pairs of young men—"sentinel monkey boys"—fed them, cleaned their cages, and took their temperatures so that any sign of fever would be observed and reported immediately [Figures 2 and 3].[81]

The investment of time and resources in the sentinel program was typical of what Haddow viewed as an entirely rational shift from studies of human serum immunity to greater

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[78] Kenneth Smithburn, Alexander Haddow, and William Lumsden, "An Outbreak of Sylvan Yellow Fever in Uganda with *Aedes (Stegomyia) africanus* Theobald as Principal Vector and Insect Host of the Virus," *Annals of Tropical Medicine and Parasitology* 43 (1949): 74-89.

[79] For a wide-ranging and thought-provoking set of essays on the role of sentinels in different scientific projects see *Limn* Issue 3, June 2013, http://limn.it/issue/03/.

[80] One outcome of the sentinel monkey program was the discovery that while rhesus monkeys would become sick when exposed to yellow fever in the forest, they did not become as acutely ill as they did when infected with a laboratory strain of the virus. Haddow, "Studies on the Natural History," 127.

[81] Smithburn, Haddow, and Lumsden, "An Outbreak," 75-77; Haddow, "Studies on the Natural History," 114-115.

attention to immunity in other primates. One major advantage of the sentinel program was that it freed the field teams from routine large-scale mosquito collections for mass testing and allowed Haddow to plan a number of ambitious field studies in and beyond Bwamba in the mid-1940s.[82] As he wrote to Mahaffy in 1945, the various vaccine programs, the return of numbers of Africans from wartime service in other parts of the world, and the discovery that yellow fever could be active in monkeys without causing any evident illness in nearby human settlements all made it imperative to shift the focus to monkeys.[83] While important work had been done by other teams in the description of mosquito and monkey distributions in Central and East Africa, Haddow noted that much of it was done by museum teams and, while that work was of high quality, "We must look much more to the ecological and bionomical sides—to put it simply, yellow fever work must depend much more on natural history and field observation than on systematics."[84]

But what exactly did Haddow mean when he encouraged more "ecological" studies? As a description of the study of the relationships between organisms and their shared environment, the term dates back to the late 18th century.[85] In the interwar period, experts debated the exact meaning of ecological work and the degree to which it implied a novel approach to the study of the natural world.[86] After WWII, several leaders in the relatively new subfield of population ecology were emphasizing mathematical methods—quantitative approaches to describing and even predicting the future behavior of complex systems.[87] Yellow fever epidemiology was certainly one such complex system and Haddow's emphasis on doing the kind of mosquito catches that would allow him to make generalizations about the populations of various species in different habitats as well as their behavior shows the influence of this kind of thinking. For example, in his extensive studies on mosquito breeding in plant axils (the part of the plant where

[82] Haddow to Mahaffy, September 7, 1945, Folder 9, Box 2, Series 477o, RG1.1, RF, RAC.
[83] Haddow to Mahaffy, September 7, 1945, Folder 9, Box 2, Series 477o, RG1.1, RF, RAC.
[84] Haddow to Mahaffy, September 7, 1945, Folder 9, Box 2, Series 477o, RG1.1, RF, RAC.
[85] Oxford English Dictionary, s.v. "ecology".
[86] Tilley, *Africa as a Living Laboratory*, 162.
[87] S. E. Kingsland, *Modeling Nature: Episodes in the History of Population Ecology* (Chicago: The University of Chicago Press, 1985).

the leaf meets the stem and where, in some plants, water collects), he faulted an earlier study's methodology of measuring the overall percentage of axils wherein any larvae were found as "a quite inadequate basis for quantitative work."[88] In his own surveys he used both the number of larvae found in each type of plant axil as well as the relative proportion of plants in an area to estimate the importance of various plant species.[89] Still, he was modest in his own claims to quantitative authority, acknowledging that the variety, even on a local level, in the sizes of plants, the numbers of axils per plant, the sizes of the axils, the quantity of water held by the axils, and plant reproduction, any attempt to quantify the relationship between plants and mosquito reproduction was "crude" at best.[90] But this was sufficient to make decisions about how best to design further fieldwork.

The term ecology also points to a difference between the kind of activity Haddow was engaging in with his maps and an earlier tradition of medical geography that tended to be deterministic, constant, and relatively straightforward in its depiction of the relationship between disease and the places in which disease was found.[91] Haddow was doing a kind of disease ecology that Warwick Anderson describes as "the spatial imaginary of disease ecology [which] was more abstract and biologically animated than medical geography" whereby "the processes it described usually were visible only to experts, not readily discerned or experienced by the general public."[92] For Haddow this meant that far from simply correlating the presence of a given species with the presence of yellow fever immunity in a given location—a correlation that amounted to circumstantial evidence at best—the Institute needed to understand the feeding habits of mosquitos and the likely interactions between potential mosquito vectors, potential non-human primate hosts, and humans. The sentinel monkey program would, hopefully, point researchers to a location where they could expect to isolate the virus "in action", but only careful entomological

[88] Haddow, "Studies on the natural history," 33.
[89] Haddow, "Studies on the natural history," 33.
[90] Haddow, "Studies on the natural history," 34.
[91] Warwick Anderson, 'Natural histories of infectious disease: ecological vision in twentieth-century biomedical science,' *Osiris* 19 (2004): 39-61.
[92] Anderson. "Natural histories of infectious disease," 42.

and zoological studies of an ecological bent ahead of time would prepare them to interpret their results in a meaningful fashion.

It wasn't until 1947 that the sentinel program bore fruit. The apparently simple task of stationing sentinel monkeys and waiting for them to fall ill was unexpectedly complex. At first the monkeys were caged, but it was observed that *Aedes africanus* mosquitoes wouldn't enter an enclosed area to feed.[93] The original set-up of the experiment had been, as Mahaffy put it, "doomed to failure".[94] So the monkeys had to be leashed and left in the open. This left them vulnerable to attack by predators, including eagles and other primates, which killed several sentinels.[95] Then a series of false alarms in 1946, where monkeys with elevated temperatures failed to show any other signs of infection, led to an investigation. Haddow concluded that overzealous attendants had artificially elevated the temperatures of the sentinels through the stress of unnecessarily frequent rectal temperature measurements. As he wrote in his official diary:

> Someone […] seems to have impressed the field boys that 103° constitutes reportable fever in a monkey. Doubtless last week's sackings have toned the boys up a trifle. As a result, when a temperature of 103° is recorded the wretches begin taking the temperatures again and again until it jolly well does go up. […] Anyone who has seen a bunch of nervous and clumsy boys dealing with a rhesus will not be surprised.[96]

Training monkey boys to intervene into the "natural" state of the rhesus monkey only as much as was necessary to detect the signs of infection without intervening so often that they artificially created those signs themselves was a delicate balancing act.

Finally, in September 1947 one of the sentinels in Zika, near Entebbe, fell ill and a chain of events began which ended in the Institute stating conclusively that they had identified the

[9393] Haddow confirmed this hypothesis by confining one of the smaller monkey attendants who was also a skilled mosquito catcher, in one of the monkey cages where he did not catch a single *A. africanus* mosquito though another catcher positioned outside the cage caught several. Haddow, "Studies on the natural history," 126.
[94] Alexander F. Mahaffy, "The epidemiology of yellow fever in Central Africa," *Transactions of the Royal Society of Tropical Medicine and Hygiene* 42, no. 6 (1949): 521.
[95] Haddow, "Studies on the natural history," 127.
[96] AJH diary, April 21, 1946, Box 191, RG 12, RF, RAC.

forest mosquito vector of yellow fever virus.[97] In 1948 several of the sentinels fell ill in Bwamba at nearly the same time permitting the Institute to focus its field investigations in the areas immediately surrounding those sentinel locations. Serum from the infected monkeys was subinoculated into lab animals and transmitted. Infection was further confirmed by post-mortem examination of the affected sentinel monkeys. Finally, intensive insect catching in the areas where the sentinels were infected resulted in numerous isolations of virus from *Aedes africanus* mosquitoes but none from any other species.[98] This was considered satisfactory evidence that the mosquito *Aedes africanus* was the vector responsible for maintaining the forest cycle of yellow fever in Uganda. In combination with data on rural and urban human-mosquito transmission, these findings led to a comprehensive model for yellow fever epidemiology in Africa.

Conclusions

Early in 1950, Haddow, who had spent and enjoyed so much time at the field station, criticized the new director of the Institute for not redirecting the resources used to maintain the station for more productive fieldwork. With the resolution of the questions of the forest cycle of yellow fever Haddow and his colleagues were more interested in turning their attention to the problem of yellow fever in drier climates such as those found in the northeast and northwest of the territory. To a degree, the success with which they had framed Bwamba as a site representative of a particular type of natural environment—the tropical African forest—limited the types of questions that they could investigate there. In 1950, as previously agreed in keeping with their general practice, the IHD withdrew its staff from the YFRI and turned over administration of the Institute to the East African High Commission. At the end of the year the Institute's annual report announced that the Bwamba field station, "having fulfilled its purpose", had been closed.[99] Under

[97] Alexander J. Haddow, K. Smithburn, G. Dick, et al., "Implication of the Mosquito *Aedes (Stegomyia) africanus Theobald* in the Forest Cycle of Yellow Fever in Uganda," *Annals of Tropical Medicine and Parasitology* 42 (1948): 218-223.
[98] Smithburn, Haddow, and Lumsden, "An outbreak"; Haddow, "Studies on the natural history," 132-141.
[99] *Virus Research Institute Annual Report* (Nairobi: Government Printer, 1951).

new direction, the Institute, renamed the East African Virus Research Institute, shifted its research priorities. Subsequent field studies would be handled on a less permanent basis and would cover a wider range of habitat types depending on the research program.

While Haddow and his colleagues aspired to an understanding of yellow fever's dynamics in its "natural state", they had to make Bwamba into a place where those dynamics could be observed, quantified, and then translated for application in the laboratory, other field sites, and the boardrooms of international public health agencies. It was not sufficient to observe the complex web of relationships between people, animals, mosquitos, and plants in the place—the place itself had to be remade in order to make yellow fever visible. This place-making required the concerted effort of the scientist, his field staff, and support from the home laboratory to translate field observations into generalizable statements of fact. For Haddow, the field, particularly Bwamba, was not something that existed in opposition with the laboratory as a place in which an alternative form of knowledge was produced; it was an extension of the laboratory's knowledge-production capacity. Haddow's efforts in Bwamba proved productive beyond their initial goals—as they attempted to isolate yellow fever, the YFRI team isolated three entirely new viruses in Bwamba: Bwamba virus, Semliki Forest virus, and Bunyamwera virus. The capacity for intensely studied and manipulated field sites to produce a wide range of medical information would incentivize researchers to continue setting up these types of sites elsewhere in Uganda.

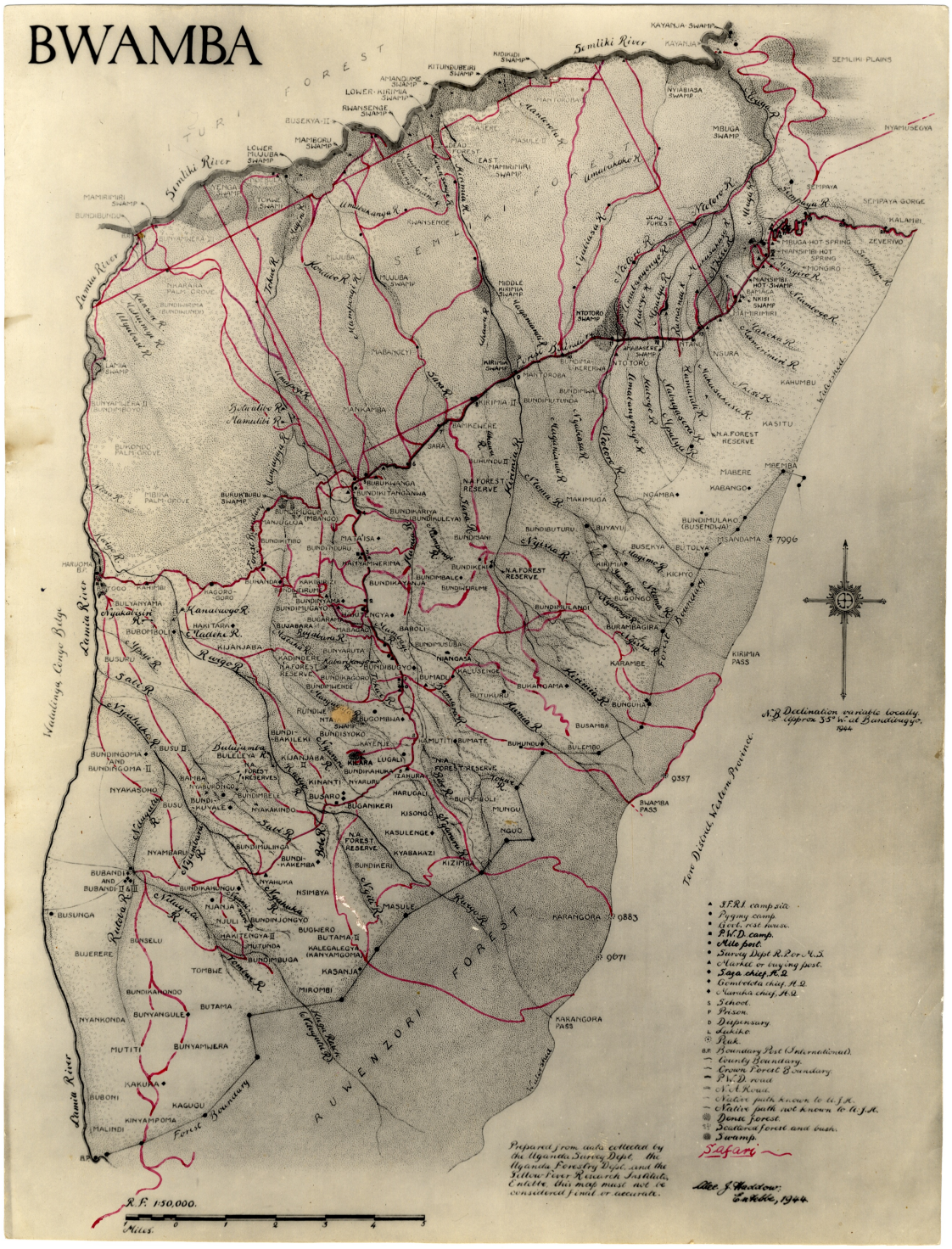

Figure 1: Map showing the routes of Haddow's safaris in Bwamba, 1944. Estate of AJ Haddow and University of Glasgow Archives & Special Collections, Papers of AJ Haddow, GB248 DC 068/84.

Figure 2: Field worker on one of the ladders to the canopy platforms. Estate of AJ Haddow and University of Glasgow Archives & Special Collections, Papers of AJ Haddow, GB248 DC 068/80.

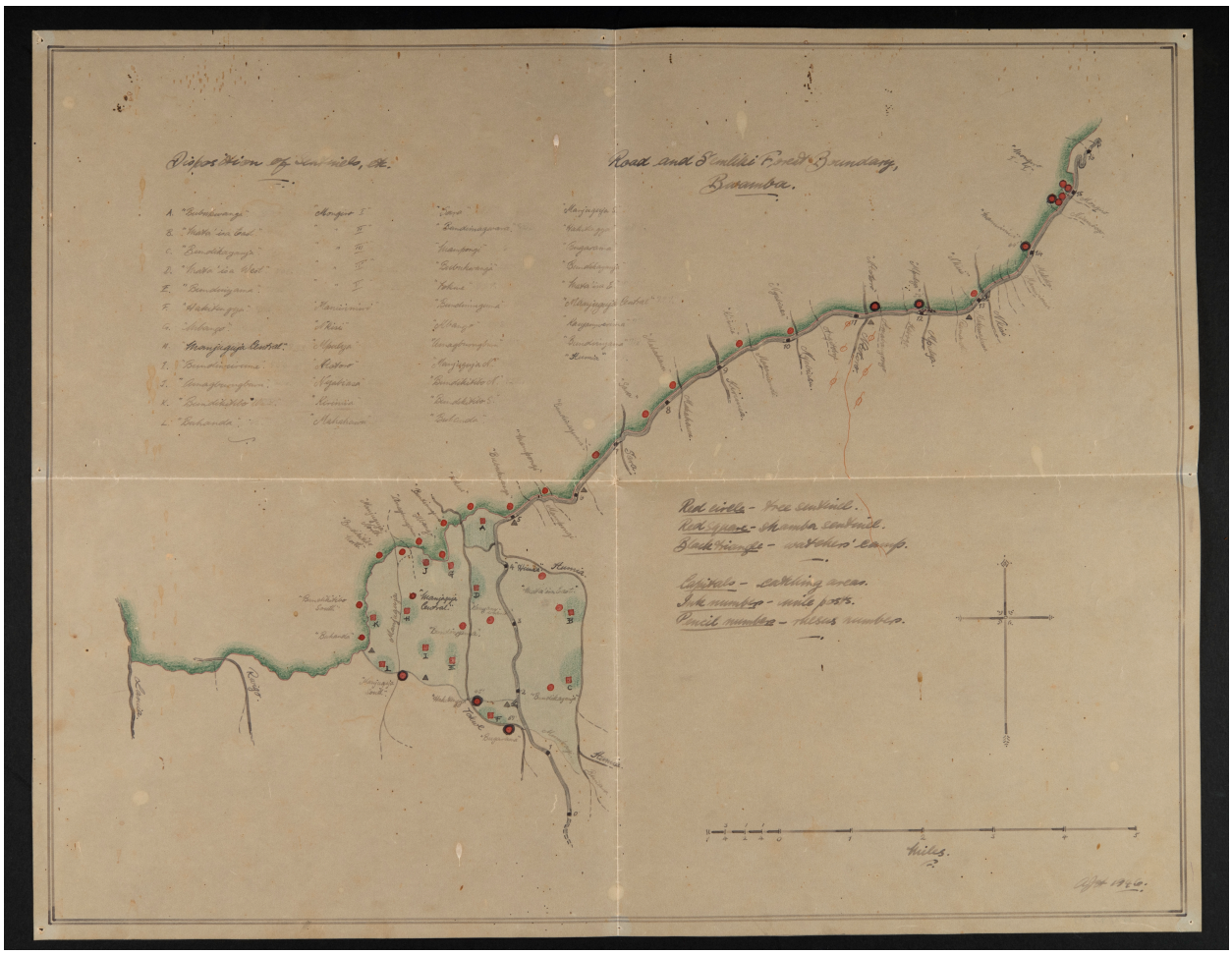

Figure 3: Haddow's map of the sentinel monkey locations in Bwamba, 1946. Estate of AJ Haddow and University of Glasgow Archives & Special Collections, Papers of AJ Haddow, GB248 DC 068/84.

Chapter Three
Collaborating on Cancer: Burkitt's Lymphoma Investigations at the EAVRI, 1961-1969

Introduction

Throughout the 1950s the Institute underwent a number of changes, as did Uganda as a whole. After the Rockefeller Foundation withdrew from the everyday running of the Institute in 1950. It was placed under the aegis of the East African Medical Research Council (EAMRC), a division of the East African Common Services Organization (EACSO), with funding from Uganda, Kenya, and Tanganyika territories, and renamed the East African Virus Research Institute (EAVRI). When Alexander Haddow became the EAVRI's acting director in 1952 and permanent director in 1953, he inherited an institution with a world-class reputation but an uncertain future. Among the challenges Haddow and his colleagues faced was to define the mission of the Institute as it expanded its purview beyond yellow fever. He had to recruit new stakeholders and demonstrate the EAVRI's value to a set of funders with more limited means and greater expectations of accountability and short-term results than the IHD.

This and the following chapter examine the ways in which the Institute adjusted to forces that made Uganda less attractive as a site for some forms of research, including the departure of many expatriate physicians and researchers and the transition to short-term funding opportunities and partnerships with metropolitan donor organizations. Researchers at the Institute invested in projects that capitalized on Uganda's potentially unique contributions to newly emerging fields of virus research. As the ties that linked the Institute to centers in New York and London weakened, it capitalized on the ties it had to individuals and institutions operating in Uganda and the wider region. It was these relationships that led the EAVRI into the burgeoning field of cancer virology.

Specifically, this chapter explores the relationship between the EAVRI and a team from the Imperial Cancer Research Fund (ICRF) housed at the Institute between 1961 and 1969 as they attempted to link Burkitt's lymphoma to a viral agent. Like Chapter One, it demonstrates the

tension between the norms and expectations of international science and the local conditions of research in Uganda in the periods immediately preceding and following Independence. It also builds on the theme of researchers attempting to make a virus, one that existed only in theory, "visible" using maps and other tools. Finally, it considers the ways in which the EAVRI sought to market itself as a desirable local partner to international cancer research teams while also maintaining enough independence to make autonomous scientific contributions and get recognition for them.

The relationship between the EAVRI and the ICRF, like the relationship between the Yellow Fever Research Institute (YFRI) and the Rockefeller Foundation International Health Division (IHD), required negotiating the material constraints of work in a tropical laboratory, competing priorities, and personality conflicts. However, unlike the YFRI and the IHD, the EAVRI and the ICRF were not integrally connected. The ICRF could have chosen other local partners and the EAVRI could have pursued other collaborations or areas of research. Their partnership was always understood to be temporary and of uncertain duration. The ways in which each partner understood the advantages and disadvantages of the relationship were dynamic, and sometimes caused conflict. As in the case of yellow fever investigations, the ability of the EAVRI team to command and communicate a superlative knowledge of their local turf—Entebbe and Uganda more broadly—was essential to its position as a desirable partner for research into Burkitt's lymphoma. Maps were involved, as was the Institute's relationship to local government representatives; the physicians on the ground in various districts; its ability to maintain a working laboratory environment; and the accumulated results of 25 years of research on the virological, entomological, and zoological ecologies of the region. The work of the Institute in this period was critical for the establishment of Uganda as a site that would continue to yield important scientific findings about viruses in the post-colonial period.

Like the story of the YFRI and yellow fever investigations, the story of the Institute's work on Burkitt's lymphoma begins with a set of maps. Denis Burkitt was an Irish surgeon who

came to Uganda in 1946 with the Colonial Medical Service after having spent some time in the country during his stint as a surgeon in the Royal Army Medical Corps during the Second World War.[1] Burkitt was well known to the EAVRI team. A previous director, Kenneth Smithburn, had been treated by Burkitt and corresponded with him, even exchanging Christmas cards and gifts, and the European medical community in the Kampala-Entebbe area was small enough that they and their colleagues would have mingled socially as well as professionally.[2] In 1961 Haddow, a medical entomologist who had worked at the Institute since 1942, saw one of Burkitt's tumor maps. He observed that the geographical distribution of the cases appeared to correspond to zoogeographical zones such as those described by naturalist James Chapin in 1923 as well as to the distribution of some mosquito species.[3] According to a letter Haddow wrote to Himsworth assessing the value of Burkitt's largely anecdotal data points, "if by such crude and empirical methods one can arrive at a reasonable map of the tumour distribution then that distribution is almost certainly a true ecological one."[4]

While Burkitt and others have remarked on how the comparison of the lymphoma maps and the maps from the EAVRI influenced their work, this juxtaposition was equally productive of new lines of inquiry for Haddow and his colleagues at the EAVRI. The suggestion that the tumor might be related to an arbovirus stimulated two decades of projects at the EAVRI in pursuit of such a pathogen. The period of time in which these studies unfolded, from 1961 to 1979, was one in which the EAVRI was engaged in the wider push for "Africanization," while attempting to maintain their international scientific credibility and to demonstrate their value to the local and regional governing bodies.

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[1] Owen Smith, "Denis Parsons Burkitt CMG, MD, DSc, FRS, FRCS, FTCD (1911093) Irish by Birth, Trinity By the Grace of God," *British Journal of Haematology* 156, no. 6 (2012): 770-776.
[2] Burkitt to Smithburn, September 28, 1948 and September 1, 1951 Folder 124, Box 14, Series 3, KCS Papers, RAC.
[3] Alexander J. Haddow, "An Improved Map for the Study of Burkitt's Lymphoma Syndrome in Africa," *East African Medical Journal* 40, no. 9 (1963): 429-432; Alexander J. Haddow, "Epidemiological Evidence Suggesting an Infective Element in the Aetiology," in *Burkitt's Lymphoma*, ed. Denis Burkitt and Dennis H. Wright (Edinburgh: E & S Livingstone, 1970), 198-209. Davies had noted that the lymphoma maps bore a resemblance to the maps of yellow fever maintained at the EAVRI.
[4] Haddow to Himsworth qtd. in Emm Barnes and Joanna Baines, *The Changing Faces of Childhood Cancer: Clinical and Cultural Visions since 1940* (Basingstoke: Palgrave Macmillan, 2015), 74-75.

Haddow's interest in linking Burkitt's lymphoma to a virus, particularly an arbovirus, may have been related to the larger situation of the Institute. After 1950, when the Institute officially embraced a wider mission and changed its name, there was not a clear path for the Institute to define itself or advocate for funding in the same way that yellow fever had provided a clear problem to solve and stakeholders. The Institute was still well regarded for its work in arbovirology and epidemic investigation and attracted funding for equipment, studies, and training from the World Health Organization, the Rockefeller Foundation, and the Colonial Development and Welfare program.[5] But its core funding was precarious and not always sufficient to fill all of its scientific and technical lines.[6] At no point in the course of the studies related to Burkitt's lymphoma was this the only matter under investigation at the Institute. But it commanded the time and attention of a large number of its workers, its animal resources, and its space.[7] It also seemed to offer the greatest opportunity for dramatic results.

Mapping a Tumor

Before turning to the Institute's stake in cancer research, this chapter will review the work that went into creating the original tumor maps in order to emphasize how intimately this project was connected to the local and regional networks of physicians and scientists in Uganda and, to a lesser extent, across sub-Saharan Africa. Understanding the way that Burkitt's lymphoma research unfolded in this period requires understanding the networks that linked hospitals, mission stations, clinics, and research institutions in East Africa to one another and how those networks shifted and reformed as the British colonial regime gave way to independent African nations. And these networks, not the ones linking the Institute to London, were the ones that led Haddow and his colleagues to cancer research, through their formal and informal ties to Burkitt

[5] Alexander J. Haddow, "Introduction," *EAVRI Report* January 1954-June 1955, 1; A.J. Haddow, "Introduction," *EAVRI Report* July 1955-June 1956, 1; William H. R. Lumsden, "Introduction and Summary," *EAVRI Report* July 1956-June 1957, 3.
[6] Alexander J. Haddow, "Introduction," *EAVRI Report* 1953, 1.
[7] Alexander J. Haddow, ed., *EAVRI Report* July 1962-June 1963; Alexander J. Haddow, ed., *EAVRI Report* July 1963-December 1964.

and his colleagues at Mulago Hospital in Kampala. Nurturing the connections between local institutions and individuals was part of the work Haddow and his colleagues at the EAVRI did to construct late-colonial and early post-colonial Uganda as a good site for virus research. [8]

In the 1950s, Burkitt led the effort to describe a syndrome of tumors in children that appeared to be limited to particular parts of East Africa. Burkitt was a consulting surgeon at Mulago Hospital, the teaching hospital of Makerere University and Kampala's national referral hospital, when he first became interested in what appeared to him to be a tumor complex manifesting itself most noticeably in large, disfiguring, and aggressive tumors in the jaws of children. By his own account, it was a referral from a colleague, Hugh Trowell, that introduced him to the first of these children.[9] At that time, he was apparently not impressed, but when he encountered a child with a similar tumor of the face in Jinja, several hours east of Kampala, something struck a chord. Burkitt began actively seeking information about such tumors. The syndrome was not new – once he began inquiring, Burkitt ascertained that colleagues had been seeing patients with tumors that fit the profile for years. Indeed, Burkitt consulted the notes of Albert Cook, who had maintained meticulous records of the patients he saw at Mengo Hospital in Kampala in the first half of the 20th century, and found evidence that such tumors were known to Cook as well.[10] However, no one had previously posited an etiologial connection between the tumors.

Using a combination of personal observations, questionnaires mailed to physicians and surgeons practicing in various parts of Africa, and consultation of the existing tumor registry in Kampala and other historical records, Burkitt set out to map the distribution of the tumor. The results of these inquiries outlined what he called the "tumor belt" extending across sub-Saharan

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[8] Brendan Clarke has made an argument about understanding the maps and the repurposing of existing medical infrastructure for research on Burkitt's lymphoma as "small science" contrasted with the "big science" typically associated with cancer research. I found his analysis useful, but argue that placing the Burkitt's lymphoma research projects at the Institute in the larger chronological context of virus research in Uganda highlights the importance of place-making, an aspect of the work that Clarke does not address. Brendan Clarke, "Mapping the Methodologies of Burkitt lymphoma," *Studies in the History and Philosophy of Biological and Biomedical Sciences* 48 (2014): 210-217.
[9] Denis P. Burkitt, "The Discovery of Burkitt's Lymphoma," *Cancer* 51, no. 10 (1983): 1777-1786.
[10] Burkitt, "The Discovery," 1778.

Africa and dipping southwards along the eastern coast. Burkitt's surgical orientation is evident in the metaphor he used for his analyses to more exactly delineate the boundaries of this region; he called his work a series of "geographical biopsies," comparable to the work of a surgeon working in tandem with a pathologist to identify the precise point at which healthy tissue gives way to malignancy.[11] Like the YFRI before him, Burkitt was trying to pinpoint the line or lines that separated areas with the disease from areas without it, what he would later call "pathologic frontiers."[12] He thought this work was a first step to discerning the "responsible environmental factors" and he believed that the frontiers dividing "tumour-bearing" and "tumour-free" places most distinctly were the best areas to look for those factors.[13]

Burkitt's tendency to take a wider geographic approach rather than a narrower pathological approach may have been informed by his father's work studying the migratory patterns of robins in Ireland.[14] An article about the scope of medical geography, published a few years later, remarked that while the idea of connecting epidemiology with geography dated back at least as far as John Snow's famous map of cholera cases, what was novel in the middle of the 20th century was the degree to which scientists were collaborating to make these maps.[15] In 1955, in a practice reminiscent of the collective investigations of the late 19th century in Europe, Burkitt began circulating flyers and questionnaires to hospitals and physicians serving various parts of tropical Africa, and meticulously indexing the responses.[16] Burkitt worked mainly in Kampala's large, urban referral hospital and did not have the intimate knowledge of Uganda's backcountry that Haddow developed in Bwamba, but he did have something else: access to an extensive

[11] Denis P. Burkitt, "Determining the Climate Limitations of a Children's Cancer Common in Africa," *British Medical Journal* 2, no. 5311 (1962), 1019. It was a metaphor he developed in 1962 to describe his work including the safaris and would continue using into the 1980s. Burkitt, "The Discovery," 1779.
[12] Denis P. Burkitt, "A Great Pathologic Frontier," *Postgraduate Medical Journal* 42, no. 491 (1966): 543-547.
[13] Burkitt, "A Great Pathologic Frontier," 546; Burkitt, "Determining the Climate Limitations," 1019.
[14] Davis Coakley, "Denis Burkitt: An Irish Scientist and Clinician Working in Africa," A Trinity Monday Memorial Discourse delivered at Trinity College, Dublin, 2011, https://www.tcd.ie/Secretary/FellowsScholars/discourses/discourses/2011_D Coakley on D Burkitt.pdf It was also an approach Burkitt had applied to his studies of hydroceles in the 1940s according to Burkitt, "The Discovery," 1777.
[15] N. D. McGlashan, "The Scope of Medical Geography," *The South African Geographical Journal* 47 (1965): 35-40.
[16] Harry Marks, "'Until the Sun of Science…the True Apollo of Medicine has Risen': Collective Investigation in Britain and America, 1880-1910," *Medical History* 50 (2006): 147-166. Wellome Library Archives (WL) WTI/DPB/B.4; WL WTI/DPB/B.3/2;

network of government and mission medical providers across the continent. Burkitt's first posting upon arriving in Uganda had been to the upcountry district hospital in Lira, where he and one African doctor were responsible for the 100-bed hospital, as well as the population of 250,000 in a 7,000 square mile area, an experience which may have helped him connect with other upcountry doctors in the course of his investigation.[17] Burkitt's questionnaires were illustrated with photographs of children with the characteristic tumors and asking people who believed they had seen cases of what Burkitt called "malignant lymphoma" to supply details to Burkitt at Mulago's department of surgery. The questionnaire, apparently intended for circulation with the Uganda protectorate, shows two passport-sized photos of children's heads, the first showing a child with an early stage tumor and the second showing a child with a late stage tumor. Beneath the photographs a short typed paragraph informs readers, "The above photographs illustrate Sarcomas of the jaws in children. These are in fact virtually the only jaw tumours we have seen in children. They frequently involve more than one quadrant of the jaw." The flyer goes on to request any reader who encounters such a jaw tumor in a child to attempt to collect information on the patient's age, sex, tribe, first symptom, the location of the tumor, whether the teeth are affected, and whether the patient has tumors elsewhere in the body, and to forward such information to Burkitt. Furthermore, the flyer suggests sending biopsy or post mortem tissue specimens to the Protectorate Laboratory for Histology.[18]

The second flyer produced and circulated in the same period, is printed on a single side of one page. Along the left hand side are three photographs illustrating a tumor of the left maxilla, a tumor in the right mandible and right maxilla invading the orbit of the eye, and a tumor involving all four quadrants of the jaw with related displacement and loss of teeth. The text, running down the right-hand side of the page announces, "THE COMMONEST CHILDRENS' [sic] CANCER in East Africa, and possibly right across Tropical Africa is MALIGNANT LYMPHOMA." The

[17] Coakley, "Denis Burkitt," 7; Denis P. Burkitt, Edward H. Williams, and J. Lester Eshleman, "The Contribution of the Voluntary Agency Hospital to Cancer Epidemiology," *British Journal of Cancer* 23, no. 2 (1969): 269-274.
[18] WL WTI/DPB/B.3/5.

flyer indicates the age range of patients and symptoms presented by afflicted children, including jaw tumors, abdominal tumors, and paraplegia. At the bottom, in italics, the flyer concludes, "*The Department of Surgery, Makerere College, Kampala, would be grateful for any information indicating where these tumours have been recognized.*"[19] Burkitt used the resulting data, along with data culled from hospital records in Kampala, to plot the locations of cases on a large map of Africa and reached a preliminary conclusion that the tumor was limited to certain areas within tropical Africa.

Mobilizing Scientific Enthusiasm and Material Support

In 1958 the *British Journal of Surgery* published Burkitt's description of the tumor, identified as a sarcoma for the time being.[20] (Eventually pathological investigation would identify it as a lymphoma). At this stage his analysis was limited to clinical, histological, and pathological descriptions of the cases he had identified in Uganda; an age distribution of those cases; and brief citations of publications from other parts of Africa that appeared to describe the same syndrome. It included only a short section on the geographical distribution, which suggested the tumor's widespread occurrence rather than any confined distribution. This article was largely ignored at the time it was published and relatively little attention was paid to the putative syndrome outside Uganda.[21]

Three years later, Burkitt and John Davies, a pathologist at Mulago Hospital, published a brief article summarizing the investigations into the tumor syndrome (now believed to arise from lympho-reticular cells, making it a lymphoma) that had taken place in the intervening period.[22] This article noted, "From its earliest recognition in Uganda it has been clear that this tumour was geographically distributed in an unusual way…The distribution in the African continent is

[19] WL WTI/DPB/B.3/4.
[20] Denis P. Burkitt, "A Sarcoma Involving the Jaws in African Children," *The British Journal of Surgery* 46, no. 197 (1958): 218-223.
[21] Burkitt, "The Discovery," 1779.
[22] Denis P. Burkitt and John N. P. Davies, "Lymphoma Syndrome in Uganda and Tropical Africa," *The Medical Press* 245 (1961): 367-369.

extremely interesting" and speculated briefly on the possible boundaries of its range.[23] The closing section of the article, subtitled "speculation and suggestions," offered the first suggestion that a virus might be involved and that at the very least a "*prima facie* case" for such a factor justified further investigation. As they put it, "If this speculation is permitted then there are some interesting possibilities disclosed by the geographical distribution of this tumour…the boundary appears to stop short of the 'frost line'…In other fields of work this would immediately suggest that an insect vector might be involved."[24]

More discussion of the geographic distribution was included in an article by Burkitt and Gregory O'Conor published the same year. This article was the first to include a map of reported cases and contained a full page on the geographical distribution of the tumor [Figure 1].[25] Haddow published his own "improved" map of the tumors and possible factors related to their distribution in 1963 [Figure 2].[26] In Haddow's map, arrived at by layering tissue-paper maps showing regions where the mean rainfall and temperature were conducive to mosquito reproduction and hence arbovirus transmission, he predicted the areas where the tumor might develop if it was caused by an arbovirus. He then incorporated a final layer showing the places where tumors had, in fact, been observed.[27]

Burkitt and Davies closed their 1961 article with a plaintive plea for professional collaboration on the puzzle of the tumor syndrome combined with an extremely tentative etiological proposal:

> Clearly there is much further work to be done and help is needed. It is not an unreasonable hypothesis that this might be a virus induced neoplasm of the reticulo-endothelial system and it occurs under conditions which make it not impossible that it might be a vector borne virus disease. We are certain of certain basic facts in the

[23] Burkitt and Davies, "Lymphoma Syndrome," 368.
[24] Burkitt and Davies 1961, 369.
[25] Denis Burkitt and Gregory T. O'Conor, "Malignant Lymphoma in African Children: I. A Clinical Syndrome," *Cancer* 14, no. 2 (1961): 258-169.
[26] Alexander J. Haddow, "An Improved Map for the Study of Burkitt's Lymphoma in Africa," *East African Medical Journal* 40, no. 9 (1963): 429-432.
[27] The original drafts of these maps are in the University of Glasgow Archives (UGA) DC 68/77. Haddow also consulted a 1952 map by P. F. Mattingly of the distribution of *Aedes simpsoni*, one of the species of mosquito that the Yellow Fever Research Institute had linked to yellow fever transmission.

> situation. The speculations we have raised on these may well be wrong but we would plead for further investigation of a disease which is not only of interest because of the misery and suffering it causes in Africa, but has aspects of interest to all concerned in cancer research."[28]

Burkitt and Davies were suggesting a possible causal pathway that was outside their respective areas of expertise, surgery and pathology. They used both the pathos of children's suffering and the potential for being involved in identifying the first definitive link between a virus and a human cancer, one of the most sought after prizes in medical research at the time, to emphasize the urgency of dedicating additional resources to its study.

After several decades on the margins of mainstream cancer research, the search for cancer viruses, or oncoviruses, was gaining prestige and momentum in the early 1960s following the demonstration that viruses could induce cancers in laboratory mice, a more convincing model for human oncology in the opinion of many researchers than the chickens used in earlier experiments with viruses causing tumors.[29] At the same time, the definition of a virus was evolving with new insights from electron microscopy and molecular biology, to consolidate around the idea of "an entity containing genetic material (DNA or RNA) surrounded by a protein coat."[30] These discoveries, combined with the renewed enthusiasm for vaccine research generated by the success of the polio vaccine, drove enormous funds and energy in the direction of cancer virus research.[31] Haddow was eager to claim a portion of those funds and energy for the EAVRI.

Like Burkitt and Davies, Haddow also adopted the language of pathos when it served his purposes. Apparently there was some question of whether the ICRF project should be hosted at the EAVRI, and Haddow did not hesitate to invoke the specter of suffering children to advocate for the ICRF's location at the Institute, though obliquely. In January of 1961 he wrote to the Ministry of Health's Director of Medical Services, C. W. Davies, notifying him that the VRI's

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[28] Burkitt and Davies 1961, 369.
[29] Gregory J. Morgan, "Ludwick Gross, Sarah Stewart, and the 1950s Discoveries of Gross Murine Leukemia Virus and Polyoma Virus," *Studies in History and Philosophy of Biological and Biomedical Sciences* 48 (2014): 200-209.
[30] Ton van Helvoort, "'Virus & Cancer Studies'—Still Fascinating after all these Years," *Studies in History and Philosophy of Biological and Biomedical Sciences* 48 (2014): 258-259.
[31] Robin Scheffler, "Following Cancer Viruses Through the Laboratory, Clinic, and Society," *Studies in History and Philosophy of Biological and Biomedical Sciences* 48 (2014): 185-188.

steering committee had approved the Institute's participation in investigations into the lymphoma's aetiology.[32] In the letter Haddow expressed an uncharacteristically impassioned plea to keep the suffering of the patients in the foreground of all conversations about the proposed research projects. "As I understand it," he wrote,

> the investigation is to be made in the hope of alleviating, or preventing the occurrence of, a tumour which is particularly horrifying and rapidly fatal. Such being the case, the study cannot be carried on in a spirit of academic detachment. It is not, after all being made for the honour and glory of any particular institution or group of workers, but as a basic attack on a particularly dreadful disease."[33]

That said, he argued, the decision of where to locate the study should be made by the group most responsible for the implementation of the project—in this case, the International Cancer Research Fund.[34] Knowing that the ICRF already favored the Entebbe location, he reiterated: "the unavoidable conclusion is that the work should be carried out where it can be done most effectively. To anyone who has seen an advanced case, this should, I feel, be the only consideration, whether the matter be considered from a professional or from a moral standpoint."[35] Haddow's appeal to ignore the potential for "honour and glory" aside, he clearly recognized the potential benefits of hosting this research at the EAVRI if it resulted in important discoveries in the field of cancer virology.

The Tumor Safaris

The story of Burkitt's tumor safari is so familiar that it bears reminding that this was not the most obvious approach to studying a tumor. [36] On October 7, 1961, after a year of planning,

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[32] Letter from Haddow to C.W. Davies, January 17, 1961, UVRI Archive (UVRIA), GC 15 "Cancer File—ICRF".
[33] Letter from Haddow to C.W. Davies, January 17, 1961, UVRIA, GC 15 "Cancer File—ICRF".
[34] Letter from Haddow to C.W. Davies, January 17, 1961, UVRIA, GC 15 "Cancer File—ICRF".
[35] Letter from Haddow to C.W. Davies, January 17, 1961, UVRI Archive, GC 15 "Cancer File—ICRF."
[36] The safaris (especially the "long" safari) have featured in numerous scientific and popular accounts including Denis Burkitt, "A 'Tumour Safari' in East and Central Africa," *British Journal of Cancer* 16,3 (1962): 379-386; Bernard Glemser, *Mr. Burkitt and Africa* (New York: The World Publishing Company, 1970); Lawrence K. Altman, "Medical Missionary in a New Role: Medical Missionary is Seeking New Role in the Modern World," *The New York Times* November 10, 1971, 49, 51; Denis Burkitt, "The Discovery of Burkitt's Lymphoma," *Cancer* 51,10 (1983): 1777-1786; Brian Kellock, *The Fibre Man: The Life Story of Dr Denis Burkitt* (Herts, England: Lion Publishing plc, 1985); Ethel R. Nelson, *Burkitt, Cancer, Fiber: How a Humble Surgeon CHANGED THE WORLD!* (Brushton, NY: TEACH Services, Inc., 1998). Emm Barnes and Joanna Baines point out the ubiquity of the term safari to designate any long

Burkitt set out to more precisely define the geographic distribution through a more active investigation. He invited two friends and colleagues to accompany him on his "tumour safari," intended to cover approximately 9000 miles in East and Southeastern Africa.[37] Missionary doctor E.H. "Ted" Williams, who ran a hospital near Arua in the West Nile district of Uganda, met Burkitt in Kampala. Williams and Burkitt set off and stopped en route in western Tanzania to pick up Dr. Clifton "Cliff" Nelson, a Canadian family physician who had worked for the Colonial Medical Service in Uganda before resigning to join the African Inland Mission.[38] In 1961 he was stationed in western Tanzania. Burkitt, Williams, and Nelson packed a station wagon with clothing, personal items, camping supplies, spare tires, petrol, and food as well as an album of photographs of children with various tumors Burkitt believed to be part of the syndrome.[39] This album, currently in the collection of the Wellcome Library, is a thick, hand-bound volume of photographs of children with horrifically disfiguring tumors. Some of the children are obviously close to death. Many are stoic, staring blank-faced into the camera. Others have clearly visible tears streaking their cheeks. Scars cross some of their faces where surgical excision was attempted but incomplete. Tumors break the skin or erupt from the orbits. The suffering is inescapable. Even those pictures where the tumors are relatively small and do not appear to be causing great pain are troubling once the viewer knows that Burkitt's tumors were characterized by their rapid growth and that the prognosis for any of these patients in the first years of Burkitt's investigations was grim. But the album served a practical purpose on the tumor safari. It was intended to jog the memories of the physicians and surgeons in the village dispensaries and hospitals that the safari visited. Burkitt and his colleagues hoped that these visual reminders would bring to mind cases, even those seen years before, which might fit the syndrome profile.

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journey in East Africa and argue, "Illness safaris should not, then, be understood as morbid searches to capture rare or wild diseases. Rather, Burkitt's safaris, like Livingstone's trips, were designed to discover what was going on in Africa's interior: to disprove western assumptions about the nature of life in the continent." Barnes and Baines, *The Changing Faces of Childhood Cancer*. I take their point, but the way Burkitt and others have described the safaris after the fact certainly plays on the romantic and exotic visions of the stereotypical African safari.

[37] WL WTI/DPB/B.8, 7/10/61.

[38] Nelson, *Burkitt: Cancer, Fiber*, 99.

[39] Burkitt, "Determining the Climate Limitations," 1019; Burkitt, "The Discovery," 1780.

Burkitt was not the only person taking such an approach at this time. In the middle of the century interest in the overlapping fields of geographic pathology and medical geography was on the rise. By the 1970s the relative failure of traditional epidemiological and laboratory methods to shed light on the question of cancer etiology was leading some to suggest that "docile attention to nature's own experiments" in the form of geographical pathology might be a more fruitful avenue to pursue.[40] As we saw in the case of yellow fever research at the Institute, the notion that *where* you found cases of a disease could be a means of approaching the question of *what* caused the disease was not entirely new. In fact, the YFRI's yellow fever work inspired a new stage in the tumor investigations.

From the Maps to a Vectored Virus

At a 1963 cancer research symposium Burkitt offered a concise explanation of the path of reasoning that led from his initial observations to the search for a virus: "(1) if this tumor which we have been investigating is climate-dependent, it would appear to be dependent on an insect vector, and (2) if it is dependent on an insect vector, it is for that reason that we assume the tumor to be virus-induced."[41] The idea that the tumor might be related to an arbovirus suggested possible candidates among the many geographic features encoded in the maps for consideration for an etiological role. Climate, including both temperature and rainfall, was known to be critical to the distribution of the insect vectors of tropical arboviruses.

What these maps concealed is as important as what they revealed. The earliest maps use circles to indicate locations in which tumors had been detected or reported. In some of the later maps, however, the tumor belt appears as a solid swathe across the continent [Figure 3]. Within this region, the text of the articles suggests and the unpublished notes and data tables makes even

[40] Harold Himsworth, "Introduction," in Burkitt and Wright *Burkitt's Lymphoma*, v.
[41] Burkitt, *Viruses, Nucleic Acids, and Cancer: A Collection of Papers Presented at the Seventeenth Annual Symposium on Fundamental Cancer Research, 1963*, 639. Apparently an earlier suggestion of a parasitic precursor to the lymphoma had been dismissed by Burkitt by this point. Gregory. O'Conor, "Malignant lymphoma in African children. II. A pathological entity," *Cancer* 14, no. 2 (1961): 270-283, 282.

more clear, there were areas that did not appear to be affected by the tumor and far larger areas about which insufficient information was available to conclude one way or the other. Also invisible on the maps, even those with greater resolution, is the variation in the sources and quality of the data represented.

The timing of this investigation was critical, according to Burkitt. Major changes were taking place in Africa, which would very shortly, Burkitt believed, make such an investigation impossible. For one thing, European and American doctors were leaving the various countries of Africa in large numbers in anticipation of and following the transitions to independent governance. Burkitt's investigations depended on the formal and informal networks of expatriate medical professionals operating across the continent with similar training and a shared understanding of biomedical research practices. As he wrote in 1962, the year Uganda gained its Independence:

> A survey there [in Ruanda and Urundi] was considered of crucial importance and considerable urgency…The urgency was imposed by the threatened exodus of many doctors, all of whom were expatriates, owing to the expectation of almost immediate independence. The departure of most trained medical observers with their fund of experience would have made an inquiry of the nature proposed almost valueless.[42]

In other words, a whole population of people upon whom Burkitt relied as local partners were about to relocate. There was not an obvious cohort of people to replace them in experience, education, or connections to other networks in the region and beyond. While Uganda had had a medical school since the early 1920s, the number of trained African physicians was insufficient to replace all of the departing expatriates. In addition, they were not integrated into the same networks as the British physicians with whom Burkitt was connected.

Burkitt also anticipated that the changes in African governance and society would render the kind of investigation he proposed into the relationship between geography and disease less possible for other reasons. In the 1960s, Burkitt believed that, unlike people in "technologically

[42] Burkitt, "Determining the Climate Limitations," 1020-21.

advanced countries" where risk exposure was more or less constant (with some occupational or cultural variations), in East Africa, "there are still many groups of people living in circumscribed communities in different geographical circumstances and exposed to widely varying nutritional, social, economic, and other environmental factors."[43] This presented an opportunity for the researchers willing to do the footwork involved in collecting information about various disease incidences among these different groups.

The hypothetical link between the tumor and a virus, specifically a vectored virus, rested on two main legs. The first was its geographical distribution, which was first linked to altitude and then further refined to a combination of temperature and rainfall as limiting factors.[44] Given the numerous examples of other diseases in Africa that were linked to climate and were spread by insect vectors, including malaria, yellow fever, and trypanosomiasis, the hypothesis that this geographic distribution could be explained by a vectored agent was an obvious one. Haddow pushed this comparison beyond known diseases and observed that the tumor distribution also overlapped significantly with that of mosquito species known to transmit viruses, including *A. simpsoni*, *A. gambiae* and *A. funestus*.[45] However, none of the investigators with a stake in this puzzle were naïve enough to think this was the only possible explanation.

Their confidence in the hypothesis, however, was increased by the second set of data and its implications: the age distribution. Along with McCallum of the East African Meteorological Department, Haddow analyzed the age distribution of Burkitt's lymphoma in Burkitt's records of 363 cases from Uganda, Tanganyika, Mozambique, and Nigeria in order to determine whether it

[43] Michael S. R. Hutt and Denis P. Burkitt, "Geographical Distribution of Cancer in East Africa: A New Clinicopathological Approach," *British Medical Journal* 2, no. 5464 (1962): 719.
[44] Alexander J. Haddow, "Malignant Lymphoma in African Children: Bioclimatic Distribution," *East African Virus Research Institute Report*, July 1960-June 1961, 30; Burkitt, "Determining the Climate Limitations," 1020; Haddow, "An Improved Map," 429-432. There is some disagreement about the exact order of these events in different accounts.
[45] Haddow, "Epidemiological Evidence," 206. The possibility that the unknown viral agent causing the tumor could be transmitted by a combination of different vectors, as is the case in many other diseases including yellow fever, malaria, and o'nyong-nyong, made it unnecessary to prove a contiguous distribution of any single vector and the tumor.

appeared to support or undermine the proposed link with an arbovirus.[46] They noted that there was a steep increase in cases between birth and age five-six, which "rather resembles G.W.A. Dick's figures for the age incidence of yellow fever antibodies in the sera of children from Bwamba County, Uganda."[47] After age six, they found that "approximately 25 per cent of the population at risk becomes affected in each succeeding year," which they compared to the 27 per cent age-incidence of yellow fever immunity in monkeys in Bwamba.[48] Finally, even when the series was expanded to 578 cases by the end of the decade, there were still no recorded cases in infants under 12 months old, which was consistent with the transitory immunity to arboviruses that immunized mothers give their children.[49]

Haddow and McCallum suggested a narrative that would explain the age incidence in the context of rural Ugandan society:

> African children—particularly in country districts—may accept mother's milk up to and even beyond age 2 as a supplement to a diet which is frequently deficient. They may receive a little antibody from this vehicle. At about age 3 they become much more independent, and begin to wander from the home, among the plantations, and to accompany their parents to the well and the forest. By age 5 boys may be goat-herding and girls are also moving freely in quite a wide area around their homes. It is interesting that it is after age 3, when the child begins to wander by himself outside the home, that the great rush of cases begins also.[50]

This analysis added a social dimension to the correlations between the disease, geography, climate, and age. And it relied on observations made by people familiar with the ways of life in West Nile, observations casual observers on brief visits to the area were unlikely to make.

Miscellaneous observations about the known cases, such as the apparent lack of correlation between the tumor incidence and racial or tribal categories, did not necessarily support the arbovirus theory in particular, but directed attention away from genetic factors and towards

[46] A.J. Haddow and D. McCallum, "Preliminary Statistical Work on Mr. Burkitt's Records," *EAVRI Report* July 1961-June 1962, 31-36; Haddow, "Age Incidence in Burkitt's Lymphoma Syndrome," *East African Medical Journal* 41,1 (1964), 1.
[47] Haddow and McCallum, "Preliminary Statistical Work," 32.
[48] Haddow and McCallum, "Preliminary Statistical Work," 33.
[49] Haddow, "Epidemiological Evidence Suggesting," 207.
[50] Haddow and McCallum, "Preliminary Statistical Work," 35. It is interesting that this set of observations was excluded from the article Haddow published in the *East African Medical Journal* in 1964, which is otherwise quite similar to the write-up in the Institute report. A.J. Haddow, "Age Incidence in Burkitt's Lymphoma Syndrome," *East African Medical Journal* 41,1 (1964): 1-6.

environmental factors.[51] Haddow and McCallum conceded that much of these analyses of age distribution, etc. were only "armchair epidemiology" of rather small subgroups, and that statistical experts were already suggesting alternative modes of analysis; but their goal in sharing these initial findings was "to stimulate discussion and to point out that, up to the time of writing, nothing has been found to preclude the possibility that Burkitt's syndrome could be caused by an arthropod-borne virus."[52]

Situation of the EAVRI

While Haddow and his colleagues took pains to get up to speed with international developments in cancer virus research, the way that the EAVRI approached the Burkitt's lymphoma problem was profoundly influenced by the particular situation in which it found itself in the years immediately preceding and following Uganda's Independence in 1962. In the years following independence, a fundamental shift took place in the way the EAVRI and other research institutes were staffed. There was growing pressure to "Africanize" staff positions. However, a dearth of qualified African scientists meant that many expatriates still occupied leadership positions, albeit with temporary contracts and uncertain futures. As one former medical officer recalled at the end of the century, there was a real difference between the scientists like Haddow who spent most of their professional lives in Uganda with every expectation that they would work out their days at their home institutions, and the younger scientists who arrived in the early 1960s knowing that there was no long-term future for them in Uganda.[53] As he put it, "After the early 1960s we did have lots of new expatriate scientists coming along, full of new ideas, but they couldn't be committed in the same way".[54] This was not a distinct change that happened at the moment of independence, or even a few years earlier when it became clear that independence was inevitable.

[51] Haddow, "Epidemiological Evidence Suggesting," 207.
[52] Haddow and McCallum, "Preliminary Statistical Work," 36.
[53] Roger Whitehead, in L.A. Reynolds and E.M. Tansey (eds), *British Contributions to Medical Research and Education in Africa after the Second World War*, Wellcome Witnesses to Twentieth Century Medicine, vol. 10 (London: Wellcome Trust Centre for the History of Medicine at UCL, 2001), 57-58.
[54] Whitehead in Reynolds and Tansey, *British Contributions*, 57-58.

It was a gradual change in the assumptions and futures envisioned by scientists. Jack Woodall, who arrived in Entebbe in 1959, described what he assumed was the common expectation of scientists: "we were there for life as far as we knew" and "that if you stayed there long enough you'd become director or vice-director." By the mid-1960s, however, (he dates it to Independence, which he recalled as being in 1965, not 1962) "it was quite clear…that we were the wrong color. That we would never get promoted."[55] Woodall may have been one of the people Haddow had in mind in the late 1970s when he recalled the challenges of running the Institute around the time of Independence: "There being no longer any prospect of a prolonged career, expatriate staff tended to join an institute for three or four years and then to move on elsewhere – just when they were becoming really useful."[56] While he was careful not to place all the blame on nationalist movements, Haddow joined Lumsden in observing that for many expatriate scientists, what was lost at the time of Independence was the connection with a "home base" in Europe or the United States.[57] He wrote, "Few of us would have joined the various services to which we belonged had we thought for a moment that we would be transferred from a service base in Britain to African authorities who now became responsible for our salaries, pensions and conditions of work."[58]

Even before Uganda became independent in 1962, changes in the administration and structure of the British colonial research services had created challenges for recruiting and keeping skilled researchers. Virologists were among the most difficult to hire and keep on staff in any of the overseas postings—Haddow referred to them as "scarce creatures" and Himsworth agreed that they were "rare birds" who were reluctant to pursue careers in the tropics.[59] The Institute's Assistant Director, entomologist J. D. Gillett cited the uncertainty of his pension as

[55] Jack Woodall oral history.
[56] Alexander J. Haddow, "A Talk on Research Institutions Delivered at the Symposium on Health & Medicine in Africa During the Colonial Period, held in New College, Oxford, March 21-23, 1977," UGA 68/77.
[57] Haddow, "A Talk on Research Institutions"; William H.R. Lumsden, "Impact of Independence and Nationalism on Tropical Medicine," *Bulletin of the New York Academy of Medicine* 51 (1975): 595-607.
[58] A.J. Haddow, A talk on research institutions delivered at the Symposium on Health & Medicine in Africa during the Colonial Period, held in New College, Oxford, March 21-23, 1977, Glasgow University Archives, 68/77.
[59] Haddow to Himsworth April 16, 1959 and Himsworth to Haddow May 1, 1959, TNA FD 7/1509.

among the factors leading to his decision to resign in 1961.[60] But there were also changes in the expectations of researchers in anticipation of Independence and Africanization of state institutions including the EAVRI. Haddow, for example, resented the imperative to train up African scientists to meet the need for skilled researchers announcing, with some chagrin, "Most of us, I believe, felt that this was the function of the universities and few of us had come to Africa with any idea of the education of others. I for one, would have stayed at home had an educational career been my objective."[61] While Haddow had an insatiable appetite for scientific work, he needed a project that promised discovery—not merely pedagogy.

In this period of uncertainty for expatriate scientists and apparently great potential opportunity for young African scientists, the Institute needed a guiding mission. While not explicitly articulated by Haddow or his colleagues, it seems likely that he recognized that a successful, high-profile line of investigation would help the Institute keep expatriate scientists for longer periods; ensure that the expatriates, should they have to leave the Institute, would find good jobs elsewhere; improve the ability of the Institute itself to attract foreign resources; and guarantee that a rising generation of African investigators would have credibility and would be taken seriously on the world stage.

Burkitt's lymphoma, and its putative viral link, provided just such an investigation. Soon after Burkitt described the tumor's distribution and he and others began to posit a link to a human virus, multiple teams had mobilized in the U.S., Europe, and Africa to be the first to positively link a human cancer to a virus. As historians of cancer research in the second half of the 20th century have observed, "Burkitt's discovery of the first human cancer to apparently be caused by a virus came at a time when faith that such patterns of causation would be found, if only sufficient money and talent were set to the challenge of seeking these tiniest of carcinogens, was

[60] J. D. Gillett to Lewthwaite, November 25, 1961, TNA FD 7/1509.
[61] Haddow, "A Talk on Research Institutions."

at its peak [especially in the U.S. and the U.K.]."[62] In 1970, Harold Himsworth, former secretary of the Medical Research Council, was the first (but not the last) to suggest that Burkitt's lymphoma would be a Rosetta Stone for understanding cancer writ large, not just a small subset of pediatric cancers. As he put it, "No explanation of cancer…will ever be adequate unless it satisfactorily accounts for this particular tumour, its distribution, its age incidence, its response to therapy and its relation to other diseases."[63] Being part of the search to decipher this Rosetta Stone was a great opportunity for the Institute. As the Annual Report for 1961-1962 put it, "This investigation is among the most exciting and important yet carried out here."[64] At the same time, the issue of cancer in Africa, long held to be rare and unimportant among "primitive" peoples, was increasingly recognized as a significant cause of morbidity and mortality on the continent, though there was some disagreement about whether this was a new phenomenon or only a newly recognized one.[65]

Imperial Cancer Research Fund Collaboration

In 1960, James Stuart Porterfield of the National Institute for Medical Research suggested to the ICRF that they might be interested in pursuing the connection between Burkitt's lymphoma and a virus.[66] The suggestion was not unanimously supported—at least some ICRF members believed that the cost of "the Africa project" should be borne by the Colonial Office rather than the ICRF—but, with funding from the Leverhulme Trust, the MRC and, indeed, the Colonial Office, the project was approved and a team from the ICRF arrived in Entebbe in December 1961.[67] The ICRF and the EAVRI initially envisioned a close collaboration, at least on paper. In his letter of December 1961 introducing the ICRF team to Haddow, Harris spoke of Haddow as the "*pater*

[62] Barnes and Baines, *Changing Faces of Childhood Cancer*, 72.
[63] Himsworth, forward to *Burkitt's Lymphoma*, v.
[64] EAVRI Report July 1961-June 1962, 2.
[65] G.T. O'Conor and J.N.P. Davies, "Malignant Tumours in African children: With Special Reference to Malignant Lymphoma," *The Journal of Pediatrics* 56, no. 4 (1960), 526.
[66] The Marquess of Salisbury, "The President's Address," *Imperial Cancer Research Fund Fifty-Ninth Annual Report and Accounts 1960-1961*, 9.
[67] Harris to Haddow, February 24, 1961, UVRIA, GC 15 "Cancer File-ICRF."

familias" of the project.[68] He was expected to facilitate the adjustment of the ICRF team to the conditions of life and work in Entebbe. That team consisted of Dr. Peter Simons and two laboratory technicians (one of whom was his wife, Margery).[69]

The ICRF team had to modify some of its plans to adapt to local conditions in Uganda. For one thing, while the team initially intended to rely on human fetal tissue cultures to test biopsy material for viruses, "it was soon found that, because of local customs, the supply of human foetal material is very limited."[70] Instead they turned to a combination of sources for their tissue cultures including monkey kidneys, hamster embryos, and HeLa cells.[71]

Less than a year after the team arrived, Haddow was excited to report that the Institute believed they had isolated a virus from tissue from a Burkitt's tumor.[72] The work that led up to this development actually preceded the partnership with the ICRF; the EAVRI had begun inoculating tissue from biopsies into mice in February, 1960.[73] These procedures were drawn from their extensive experience with arbovirus studies and proceeded from the hypothesis that the tumor might be related to a vectored virus.[74] The goal was not necessarily to demonstrate the inoculum would produce tumors in the experimental animals. As Haddow acknowledged, "the production of a tumour in mice after such an inoculation would only be accepted with the greatest reserve by workers in the cancer field," but, he argued, "should an arthropod-borne virus be

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[68] Harris to Haddow, December 11, 1961, UVRIA, GC 15 "Cancer File-ICRF."
[69] Harris to Haddow, December 11, 1961 and Haddow to Harris, December 14, 1961, UVRIA, GC 15 "Cancer Research-ICRF." One of the letters in this file suggests that there had been some possibility that Paul Weinbren would be on this team and that Haddow had vehemently opposed this placement. Harris made haste to assure Haddow, "Obviously we should not think of asking anyone to go to Entebbe without your expressed consent and approval." Harris to Haddow, February 24, 1961, UVRIA, GC 15 "Cancer Research-ICRF."
[70] "Epidemic Lymphoma in Children in Africa," *Imperial Cancer Research Fund Sixtieth Annual Report and Accounts, 1961-1962*, 27-28.
[71] "Epidemic Lymphoma in Children in Africa," 27-28.
[72] Haddow to Timms, July 11, 1962, UVRIA, GC 15 "Cancer File-ICRF."
[73] Haddow to Timms, July 11, 1962, UVRIA, GC 15 "Cancer File-ICRF"; EAVRI Annual Report 1960-61, 30. They were also collecting blood from lymphoma patients and their relatives for serum protection tests against known arboviruses including Nairobi sheep disease, Sindbis, Chikungunya, West Nile, Ntaya, Zika, and Bunyamwera viruses. Only the last of those gave positive results. Williams and Woodall, "Preliminary Virus Studies," *EAVRI Report* July 1960-June 1961, 31.
[74] EAVRI Annual Report, 1960-61, 30.

responsible and present at the time of biopsy, it would be more likely to produce encephalitis in the mice after a few days than a tumour after weeks or months."[75]

This first virus isolated from a tumor came from a biopsy taken from a 10-year-old girl in West Nile district on June 18, 1962, and sent to the EAVRI by a Dr. D. A. Hyslop of the Arua Government Hospital.[76] On June 20th, workers at the Institute inoculated samples from the biopsy material into 20 newborn mice, which began showing "a typical arbovirus appearance" including partial paralysis on the 24th and 25th of June.[77] Not only were cancer specialists perhaps unlikely to accord great importance to such inoculations, they also probably could not have interpreted outcomes like "a typical arbovirus appearance" that required the kind of tacit knowledge EAVRI workers had achieved over the previous several decades. Studies related to the new agent were being pursued in tandem with the Institute team working with mosquitoes and mice and the ICRF team concentrating on tissue cultures and hamsters.[78] Haddow was emphatic in his report to the Medical Research Secretary, Geoffrey Lowe Timms, that the partnership was going well: "Throughout the investigation the outstanding feature has been the effective and pleasant cooperation between all the units concerned—I.C.R.F., Makerere College, the Ministry of Health and the Mission Hospitals, and ourselves."[79]

But that "effective and pleasant cooperation" was soon strained by differing institutional norms and competing priorities between the ICRF and the EAVRI. Though Haddow had written quite optimistically in private letters (or as Lewthaite put it, with "very justifiable restrained excitement and optimism"[80]) about the potentially novel virus isolated from the tumor, he was aware of the risk of making overambitious claims in advance of sufficient evidence. His counterparts in the ICRF, Director of Scientific Research Dr. G.F. Marrian and Head of the

[75] Haddow to Timms, July 11, 1962, UVRIA, GC 15 "Cancer File-ICRF".
[76] J. P. Woodall, M. C. Williams, D. I. H. Simpson, and A. J. Haddow, "The Isolation in Mice of Strains of Herpes Virus from Burkitt Tumours," *European Journal of Cancer* 1, no. 2 (1965): 137-40.
[77] Haddow to Timms, July 11, 1962, UVRIA, GC 15 "Cancer File-ICRF".
[78] Haddow to Timms, July 11, 1962, UVRI Archives, GC 15 "Cancer File-ICRF."
[79] Haddow to Timms, July 11, 1962, UVRI Archives, GC 15 "Cancer File-ICRF."
[80] Lewthwaite to Haddow, July 18, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."

Division of Experimental Biology and Virology, Dr. R.J.C. Harris, were even more sensitive to the risks of premature publication. Only a little over a week after writing a letter to Timms emphasizing the harmony between all the interested parties, Haddow received a letter from R. Lewthwaite in the Department of Technical Cooperation about Marrian and Harris's fears that the reputation of the ICRF would be harmed if research that appeared to link a virus to a human cancer was published prematurely.[81] Simons's suggestion that a preliminary note publishing the initial findings was not well received.[82]

In addition, their concern appears to have been exacerbated a mention of the work being done by the EAVRI and the ICRF in a speech by Uganda's Minister of Health.[83] Only a few months away from the transfer of power to an independent Ugandan government, the colonial government was attempting to consolidate its legacy of important biomedical discovery in Uganda. Haddow was sympathetic to the desire to link Uganda with what might turn out to be "a notable discovery."[84] The minister also, as Haddow pointed out, "wished to show that his surgeons and physicians were playing a part in this combined operation."[85] In other words, government, as well as the private research foundations, was playing a part in important work on cancer.

Lewthwaite appears to have been trying to placate Marrian and Harris while also assuring Haddow that he trusted and respected Haddow's judgement. He wrote, "I am sure you are much too experienced not to be aware of the risk of premature publication…But I feel it incumbent on me to stress the views which Marrian and Harris feel so strongly, viz. that, from both the scientific and the publications aspects, undue publicity at the present stage of the investigation

[81] Lewthwaite to Haddow, July 20, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."
[82] Haddow to Marrian and Harris, July 20, 1962 and Haddow to Lewthwaite, July 24, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."
[83] Haddow to Marrian and Harris, July 20, 1962 and Haddow to Lewthwaite, July 24, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."
[84] Haddow to Lewthwaite, July 24, 1962, UVRIA, GC 15 "Cancer Research-ICRF."
[85] Haddow to Lewthwaite, July 24, 1962, UVRIA, GC 15 "Cancer Research-ICRF."

could be very harmful."[86] Anticipating that Haddow would be affronted by the letter, Lewthwaite appended a handwritten note at the end of the typed letter, writing "I trust that this letter will not give offence. But Marrian and Haris are deeply concerned lest anything should be published on this matter which may have to be retracted later."[87]

Haddow had already written to Marrian and Harris conceding that the enormous pressure from the press and other interested parties to share the progress of the work was "an entirely new experience for me, but I believe that it is a familiar one in the cancer field."[88] He wrote more fully on the matter a few days later in a long letter to Lewthwaite copied to Timms, suggesting that he thought Marrian and Harris's concerns were exaggerated and their expectations of secrecy unrealistic. He pointed out that the EAVRI was collaborating with dozens of people on the investigation.[89] All in all, he concluded, "I think it would be fair to say that the whole thing is being followed stop by step by half the medical profession of this area, apart from which you know how everyone in a small town knows everyone else's business."[90] Haddow resented what he described as Marrian and Harris's efforts to blame him for any leaks of information and defended his responsibility to keep the government of Uganda, which provided much of the EAVRI's budget, informed of its activities.[91]

The network of collaborators that Haddow and the EAVRI had tapped into in Uganda was substantial and possibly unique in the region. They included government and mission surgeons, the pathology department at Mulago; the Scientific Council for Africa South of the Sahara (SCA)/Commission for Technical Cooperation in Africa (CCTA); mathematicians from the Uganda meteorological department (who were working on the age distribution analysis), and the Animal Health Research Center, which was doing the bacteriological controls on all

[86] Lewthwaite to Haddow, July 20, 1962, UVRIA, GC 15 "Cancer Research-ICRF."
[87] Lewthwaite to Haddow, July 20, 1962, UVRIA, GC 15 "Cancer Research-ICRF."
[88] Haddow to Marrian and Harris, July 20, 1962, UVRIA, GC 15 "Cancer Research-ICRF."
[89] Haddow to Lewthwaite, July 24, 1962, UVRIA, GC 15 "Cancer Research-ICRF." Lewthwaite replied that, "As for leakage, they [the ICRF] haven't a clue as to the 101 different gaps through which the most confidential knowledge seeps in the Tropics." Lewthwaite to Haddow, July 27, 1962, UVRIA, GC 15 "Cancer Research-ICRF."
[90] Haddow to Lewthwaite, July 24, 1962, UVRIA, GC 15 "Cancer Research-ICRF."
[91] Haddow to Lewthwaite, July 24, 1962, UVRIA, GC 15 "Cancer Research-ICRF."

specimens.[92] Uganda had an unusually rich academic and research community and the EAVRI was well integrated into it on both formal terms via the EAC and more informal, personal terms. The ICRF was perhaps blind to the contributions made by some of these informal and formal partners as well as to the expectations that they would have had in terms of data sharing and transparency. Haddow, who had invested the better part of a career in cultivating these relationships and was now capitalizing on them, had a much stronger sense than his temporary partners at the ICRF of the importance of these local partnerships in making the lymphoma research possible.

In addition, Haddow took issue with the standards that Marrian and Harris required of experiments to demonstrate any link between the virus and the tumor. There was a tension between the international standards Marrian and Harris expected should apply to biomedical research regardless of its site and the realities, political, logistical, and epidemiological, of the research site in Uganda on which Haddow considered himself the authority. Haddow wrote of the stipulations for any publication, which Marrian and Harris sent to Simons:

> The list of necessary checks far exceeds Koch's postulates. They include the production of tumours at least in hamsters (but who knows that hamsters will develop this tumour) and the isolation of the same virus from several other cases (might take ten years unless we were lucky.) They further state that all cases must be shown to have antibodies. This, I think, is quite unsound (on the basis of African swine fever and herpes simplex.) My own guess would be that the section of the community which get the tumour could well consist of people in whom antibody formation is irregular or intermittent.[93]

Haddow was speaking in his capacity as a local expert and struggling to reconcile the standards of western biomedical research with his own experience of work in Uganda and his sense of responsibility to represent the interests of the government of Uganda. His assessment of the benefits and risks of publishing the preliminary findings was informed by his sense of the EAVRI's obligations to local stakeholders and his different expectations of the level of certainty

[92] Haddow to Lewthwaite, July 24, 1962, UVRIA, GC 15 "Cancer Research-ICRF."
[93] Haddow to Lewthwaite, July 24, 1962, UVRIA, GC 15 "Cancer Research-ICRF." Other partnerships between foreign scientists interested in cancer and Institutions based in East Africa had soured in the past. Haddow reminded Lewthwaite of Gilbert Dalldorf's work in the region and his secrecy, which "caused a great deal of resentment among the people on whom he was dependent for material, laboratory space, etc." Haddow to Lewthwaite, August 1, 1962, UVRIA, GC 15 "Cancer Research-ICRF."

that it would be possible to establish in a timely manner. Having seen the work on yellow fever stretching into two decades, he believed he knew better than the ICRF what kind of data justified publishing in the virus world and how long it could take to meet the kinds of criteria they were proposing, if indeed it was ever possible. Moreover, while he required an exacting standard of replicability and precision in his field-based work, he felt that men who had experience only in laboratory facilities could not appreciate the complexity of real-world investigations. As he remarked years later, "the investigation of complex field situations with their endlessly interacting factors should never be equated with the deliberate, planned and controlled study of a finite problem in the laboratory."[94] Despite all of his efforts to transform Bwamba into a laboratory-like experimental space in the 1940s, Haddow recognized a qualitative difference between laboratory work and work conducted *in situ* in Uganda. And Haddow, as the Director of the Institute, had a vested interest in affirming the importance and intrinsic value of locating research projects in places where they could incorporate all of those "endlessly interacting factors" rather than reducing virological research to those questions that could be answered in a laboratory located anywhere in the world.

Lurking under the question of the timing of any publication was also a deeper schism between the ICRF and the EAVRI about the ownership of data produced by their collaborative efforts. As Haddow wrote:

> Now while ICRF indulge in pretty fulsome publicity (when it suits them) you know that they prefer to work under a shield of secrecy, a method which personally I don't care for. You will remember that when you [Lewthwaite], [Miles] Williams and I went out to Mill Hill [the home of the ICRF] six months ago we could barely get it across that any information we dug up was not for the exclusive use of ICRF. Harris said that it must be understood that any epidemiological data we got must be for their use, not for BECC [British Empire Cancer Campaign], etc., and I said that any information we had on that or any subject would be given freely to any reputable person who asked for it. I don't think this point ever did get home properly.[95]

[94] Alexander J. Haddow, "Mosquito-Borne Viruses—The Need for a Balanced Team," presented at the Royal Society of Tropical Medicine and Hygiene, Centenary of Medical Entomology Symposium held in London, November 23-25, 1977, UGA, DC 68/77.

[95] Haddow to Lewthwaite, July 24, 1962, UVRIA, GC 15 "Cancer Research-ICRF." In this period the EAVRI was, in fact, collaborating to some extent with the BECC as they were housing several suckling monkeys purchased by M. Anthony "Tony" Epstein of the British Empire Cancer Campaign and working with him to inoculate them with human

Haddow did not stop there. He suggested that, in fact, the ICRF's exaggerated concerns about premature publication were, in fact, a cover for their discomfiture at having been beaten to the identification of a virus by the EAVRI team. Or, as he put it, "Obviously they have missed the boat, are mad about it, and are lashing out."[96] Haddow noted that Simons had apparently not been told that the EAVRI was already at work on serological testing before the ICRF team arrived and that Simons had not immediately started tissue inoculation when the specimen in question was received. Simons only began work on the agent after the EAVRI team had already succeeded in inducing illness in mice. Haddow reminded Lewthwaite that at the time he had written at length to Timms in order to make "a clear claim for priority, namely that the Institute isolated the virus, using methods which had been tried on the same tumour a considerable time before the ICRF team even came out."[97] Haddow might have been willing to accommodate the ICRF team, but, as he wrote, "I am equally determined that the Institute is not to be bulldozed into the background. We would have got this agent whether the ICRF had come here or not."[98] As the EAVRI became less secure in any particular source of international funding, it was also more reluctant to surrender control over its findings and data. While the YFRI was sufficiently under the umbrella of the RF to not resent the RF claiming credit for its work, the EAVRI had a much more guarded relationship with the ICRF and subsequent international partners.

Nonetheless, Haddow took a far more conciliatory tone with Marrian and Harris and the partnership held enough promise for both parties that they attempted to find some common ground. Haddow and his colleagues at the YFRI had enjoyed a great deal of independence under the IHD and they had to adjust their strategies for dealing with new international research

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tumor material. John P. Woodall, David Ian Hewitt Simpson, and Miles C. Williams, "Monkey Inoculations," *EAVRI Report* July 1963-December 1964, 25. In 1964 Epstein and Woodall were authors on a *Lancet* article reporting that observations of the inoculated monkeys seemed "to show that a form of human malignancy can be transmitted experimentally by an infectious agent." M. Anthony Epstein, John P. Woodall, and Andrew D. Thomson, "Lymphoblastic Lymphoma in Bone-Marrow of African Green Monkeys (*Cercopithecus Aethiops*) Inoculated with Biopsy Material from a Child with Burkitt's Lymphoma," *The Lancet* 284, 7354 (1964): 288-291. That work continued throughout 1965, "Burkitt's Lymphoma," *EAVRI Report* January-December 1965, 27.

[96] Haddow to Lewthwaite, July 24, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."

[97] Haddow to Lewthwaite, July 24, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."

[98] Haddow to Lewthwaite, July 24, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."

partners who controlled access to resources while also attempting to maintain control over the research. Harris thanked Haddow for his letter assuring them that the Minister had disclosed nothing of import and that the speech had received very little attention. He further explained that his anxieties about premature publication stemmed from his observation of other cancer researchers who were overeager to publish promising findings only to be "red-herringed."[99] Marrian similarly sought to assure Haddow that he had not meant to impugn Haddow's discretion or justice, but emphasized, "This could be such a very big thing if it comes off that it seemed to us to be very dangerous to risk bringing the whole project into disrepute by publicity about a single virus isolation which might conceivably be a red herring."[100] He also noted that he was not even informing the ICRF Council of the isolation yet in order to minimize the risk of leaked information.[101]

By the beginning of August the situation had cooled off and work proceeded on the unidentified agent with attempts to ascertain its size by passing it through viruses of various pore-sizes and serum protection tests which ruled out 28 of the viruses for which the EAVRI held antisera. Haddow estimated that this represented approximately one third of the known African arboviruses.[102] They also began inoculating local rats with the assistance of a rat-trapper from the Game Department whose field station had closed and hoped to expand their collection of other "peri-domestic" rodents as Harris suggested, but that work was delayed by the theft of nearly all of their rat traps.[103] At the same time the EAVRI was also continuing their entomological work, their role as a W.H.O. reference laboratory, and their isolations of viruses from mosquitoes and human patients at their own clinic and other hospitals in the region.[104] Haddow urged Lewthwaite

[99] Harris to Haddow, July 26, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."
[100] Marrian to Haddow, July 25, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."
[101] Marrian to Haddow, July 25, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF."
[102] These included Bunyamwera which had previously given a positive reaction and prompted hopes that it might be the agent they were seeking. Haddow to Lewthwaite, August 1, 1962.
[103] Haddow to Lewthwaite, August 1, 1962, UVRIA, GC 15 "Cancer Research-ICRF".
[104] EAVRI Annual Report July 1961-June 1962. The Institute first opened an outpatient clinic in October 1956 staffed by Dr. Ellen Mary Knight, in order to supply the lab with specimens from patients with fevers. The objective of the program was "to attempt to determine if virus agents are importantly concerned in the causation of fevers among humans in the lakeshore region of Uganda, and, if so, to determine which agents are mainly concerned. Also it is hoped

and the ICRF to consider the work on the virus isolated from the tumour in the broader context of virology, not just as a potential etiology for the lymphoma:

> even if the virus is a mere 'passenger' in the tumour this could be a highly important finding in our particular field. The question of the maintenance of arbor viruses [sic] in interepidemic periods is one of major importance in tropical medicine, and the presence of this agent in a girl who was circulating over two logs of antibody and who had no virus in circulation strikes me as a finding of very considerable intrinsic interest, quite apart from the cancer aspect.[105]

While Haddow expounded on the ancillary benefits of the tumor research in terms of general scientific interest, he no longer appealed to the pathos of suffering cancer patients as he had in the earliest stages of the work. He was tailoring his approach to his understanding of the needs and motivations of the particular group upon whose funding he relied.

By late October it appeared quite likely that the virus they had been working on was a herpes virus, not an arbovirus. Dr. R. R. Dourmashkin of the ICRF had examined cultures of the virus under the electron microscope and observed that they resembled herpes virus.[106] Subsequent cross neutralization tests and complement-fixation tests confirmed his findings.[107] The inability of anyone to imagine a scenario in which a herpes virus would have the kind of distribution that had been observed of the tumor led both Harris and Haddow to conclude reluctantly that it was unlikely to be the etiological agent they had been looking for.[108]

These findings were not published until 1965 when, according to a plan suggested by Haddow, the two teams published their findings separately but in tandem in an issue of the *European Journal of Cancer*.[109] By that time more promising avenues were being pursued in

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to locate areas which would appear to be suitable for more detailed studies designed to elucidate the mechanisms of transmission and survival of these agents." Ellen M. Knight, Miles C. Williams and P. J. Mason, "Studies on Pyrexias in Man in Uganda," EAVRI Report July 1956-June 1957, 4.

[105] Haddow to Lewthwaite, August 1, 1962, UVRIA, GC 15 "Cancer Research-ICRF."

[106] Peter J. Simons and M. G. R. Ross, "The Isolation of Herpes Virus from Burkitt Tumours," *European Journal of Cancer* 1, no. 2 (1965): 135-6.

[107] Simons and Ross, "The isolation of Herpes Virus," 135-6.

[108] Harris to Haddow, October 25, 1962; Haddow to Harris October 31, 1962, UVRIA, GC 15 "Cancer Research-ICRF."

[109] Haddow had heard rumors that Simons and Harris were planning to publish the herpes isolations and feared that they would fail to credit the EAVRI's contributions. Haddow to Lewthwaite, April 2, 1964, UVRI Archives, GC 15 "Cancer Research-ICRF". As a preemptive measure he wrote to Harris and proposed that EAVRI and ICRF submit

Entebbe, but the two articles still ended on rather different notes. The paper authored by Simons and Ross of the ICRF concluded, "The ubiquitous nature of herpes simplex virus …suggests that herpes virus is probably not involved in the aetiology of the Burkitt tumour."[110] The paper authored by the EAVRI team, on the other hand, ended with the observation that:

> The cumulative incidence of the Burkitt tumour suggests an attack rate in children comparable to that of herpes virus. The lack of demonstrable immunity in some cases may be due to a failure in immune response such as occurs in a proportion of patients with other forms of lymphoma. If carcinogenesis is a two-stage process, then herpes virus could be the initiating agent of the Burkitt tumour, provided that the promoting agent were a climatically or geographically limited factor.[111]

But, they conceded, herpes virus in a jaw tumor could just be a consequence of coincidental oral infection and at least some pediatric cancer cases secreted greater quantities of herpes virus than children without cancer so, "in the absence of further evidence, the presence of herpes virus in these tumours cannot be regarded as significant."[112] Overall, the EAVRI version of the paper seems far less dismissive of the potential significance of the herpes virus.[113]

In the meantime, by the end of 1962, major doubts were growing about the suitability of Peter Simons to life and work in Entebbe. As was the case at the YFRI, not all capable scientists were able to adapt to life and work in Entebbe. Even when all was well in the laboratory itself, some individuals and their families struggled to make themselves at home in Uganda, and this had an impact on their work. In the case of Simons, a persistent fungal infection that impaired his ability to operate in the laboratory was viewed as the symptom of a larger problem. Marrian and Haddow came to believe that the condition, was psychosomatic in origin.[114] Haddow felt that part of the problem was the sense of intellectual isolation that investigators based a great distance

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separate papers "to avoid conflict about authorship, priority, etc." Haddow to Harris April 2, 1964, UVRI Archives, GC 15 "Cancer Research-ICRF".

[110] Simons and Ross, "The isolation of herpes virus," 136.

[111] Woodall, Williams, Simpson, and Haddow, "The Isolation in Mice," 137-40.

[112] Woodall, Williams, Simpson, and Haddow, "The Isolation in Mice," 137-40.

[113] A few years later, when Epstein published his discovery of the virus that would be linked to Burkitt's lymphoma (Epstein-Barr virus or EBV), a herpes virus, Woodall wondered whether they had, after all, isolated it first. But by then he was in Brazil and it is not clear whether a strain of the virus isolated at EAVRI was ever tested against EBV. Woodall Oral History.

[114] Marrian to Haddow, October 15, 1962; Haddow to Marrian November 7, 1962; Marrian to Haddow November 12, 1962, UVRI Archives, GC 15 "Cancer Research-ICRF". Specifically they agreed that his wife was "a man-eater of the worst description."

from their parent institution and operating in someone else's facilities might reasonably feel. He suggested that requiring Simons to keep a diary on the model that the IHD had used might be useful. It could be combined with a monthly progress report, which Simons would have to submit to the ICRF to keep them informed of his plans and to give them the opportunity to supply the consistent guidance Haddow felt Simons required.[115]

But the problem was not entirely about feeling professionally remote. Entebbe was less of a center for expatriate social life than it had been in the 1930s. Simons himself requested a transfer out of Entebbe, complaining that the location offered nothing but work to him and his wife.[116] Marrian assured Haddow that he considered such a complaint "the purest nonsense, since as Bob [Harris] and I know very well the people in scientific circles in Entebbe and Kampala are extremely friendly and sociable, and the opportunity for relaxation and amusement are infinitely better in Uganda than they ever could be in suburban London."[117] Haddow himself never seemed at a loss for convivial entertainment, but it is likely that Entebbe in the early 1960s might indeed have less to offer a young British couple than suburban London.

Despite Marrian and Haddow's best efforts, Simons did not reconcile himself to the location and at the end of his 21-month "tour" in Entebbe he transferred back to Mill Hill. By the spring of 1963 Marrian and Harris had identified his replacement: Dr. Thomas Bell formerly of the University of Aberdeen.[118] Haddow was pleased with Bell and his team-mates, Mr. M. G. R. Ross and Mr. A. Massie, and at the end of the year wrote:

> I can't tell you how delighted we all are with your new team. They have rapidly become 'integrated' and we do greatly appreciate hearing about what is going on… I am sure that there are going to be false starts and that many 'hares' will be started. On the other hand I

[115] Haddow to Marrian, November 7, 1962. Haddow noted that while the diary might sound like a "nuisance," he and the other IHD researchers had not found it to be one and that "It provided a very useful link and record of the work." He would, he recalled, rely on Smithburn's and Mahaffy's diaries when he returned to Entebbe from Bwamba in order to be able to "pick up all the strands of lab work." Moreover, he wrote, he still consulted his old diaries from time to time "to settle a point."
[116] Marrian to Haddow November 12, 1962, UVRIA, GC 15 "Cancer Research-ICRF."
[117] Marrian to Haddow, November 12, 1962, UVRIA, GC 15 "Cancer Research-ICRF." This seems to me to be somewhat hyperbolic and I am not convinced Marrian was entirely sincere and not just appealing to Haddow's pride in Entebbe and his own company.
[118] Marrian to Haddow, May 28, 1963, UVRIA, GC 15 "Cancer Research-ICRF."

> now feel confident that if something does turn up it will be taken in hand rapidly and efficiently.

The ICRF also seems to have been pleased with the direction the collaboration was taking. The Chairman of the Fund, Sir Cecil Wakely, visited Entebbe and took advantage of his opportunity in his introduction to the Fund's Annual Report "to correct the impression given by recent Press reports and to re-assert that the widest possible exchange of information occurs between cancer research workers throughout the world."[119]

In early 1964, the investigations appeared to be entering a new phase after Bell isolated a reovirus from three cases of Burkitt's lymphoma. Bell was eager to publish his findings immediately and drafted a manuscript. But once again, conflict with the ICRF home office arose regarding the proper timing for such publication. Upon being advised by Harris that he and Marrian thought it was too soon to move towards publication, Bell wrote a heated letter to Marrian in which he stated, "While I am in full agreement that bad papers should not be published, in comparison with the recent efforts of Dalldorf and Epstein this one is brilliant."[120] He emphasized the evidence in support of the identification of the isolate as a Reovirus type 3 and argued, "This is the first isolation of <u>any</u> Reovirus in East Africa, and as far as we can see from the literature [sic], the first isolation of a Reovirus in the Continent of Africa. As an epidemiological and clinical virologist I am able to say that this communication is worth publishing irrespective of an aetiological relationship with Burkitt's Lymphoma."[121] Bell was aware that other teams in the United States, Europe, and Australia were working with reoviruses and was anxious to be the first to publish on its possible link to Burkitt's lymphoma. In addition, he was suspicious that Harris had verbally communicated some of Bell's findings at a meeting in the United States, findings which were then adopted into the work of Epstein and Dalldorf.[122] In March of 1964 Epstein, Bert Achong, and Yvonne Barr had published a preliminary

[119] Sir Cecil Wakely, "The Chairman's Report," Imperial Cancer Research Fund's *Sixty-First Annual Report and Accounts, 1962-1963*, 9.
[120] Bell to Marrian, April 4, 1964, UVRIA, GC 15 "Cancer Research-ICRF."
[121] Bell to Marrian, April 4, 1964, UVRIA, GC 15 "Cancer Research-ICRF."
[122] Haddow to Lewthwaite, April 10, 1964, UVRIA, GC 15 "Cancer Research-ICRF."

communication in *The Lancet* documenting their isolation of a virus that appeared similar to herpes simplex from cultures of cells from Burkitt's tumors.[123] That virus, named Epstein-Barr virus (EBV), became the most prominent suspect in the search for a cause for Burkitt's lymphoma.

Haddow was sympathetic to Bell's desire to establish priority and gain recognition which would also accrue to the EAVRI. He wrote to Lewthwaite expressing his exasperation at the ICRF's reluctance to publish and suggested that it was possible that the ICRF was even deliberately suppressing the publication of any paper that would reveal methods that other teams might then put to use.[124] He reiterated his earlier concerns about the ICRF's fondness for secrecy:

> I.C.R.F. must recognize that various bodies are collaborating with them, who do not agree with their outlook. We, for instance, find it difficult and far from pleasant to 'collaborate' with Makerere on the surface, while holding back from them most of the crucial information…In general I cannot tell you how very tired I am of all this cloak-and-dagger business. The big Cancer groups have such a suffocating preoccupation with their own prosperity and future that research and progress must inevitably suffer. 'It is all right if we make the discovery, all wrong if you do, and the devil take the patients'.[125]

While Makerere may have seemed like a relatively insignificant player from the vantage point of London, for Haddow, the local institutions and individuals with whom he collaborated were critical partners in the EAVRI's work.

Haddow, and the Institute itself, were in a sort of intermediary position between the international agencies such as the ICRF based in Europe and the Africans rising through the ranks to senior scientific positions in Uganda. On the one hand, Haddow shared many of the ICRF personnel's prejudices about Africans and was pessimistic about the future of the EAVRI without expatriate staff. On the other hand, he had a much deeper appreciation of the realities of life and work in Uganda than the ICRF staff on temporary posting to Entebbe and he demonstrated a deep loyalty to the Institute and its interests. When Bell threatened to resign from the ICRF if Marrian

[123] M. Anthony Epstein, Bert G. Achong, and Yvonne M. Barr, "Virus Particles in Cultured Lymphoblasts from Burkitt's Lymphoma," *The Lancet* 283, no. 7335 (1964): 702-703.
[124] Haddow to Lewthwaite, April 10, 1964, UVRIA, GC 15 "Cancer Research-ICRF."
[125] Haddow to Lewthwaite, April 10, 1964, UVRIA, GC 15 "Cancer Research-ICRF."

and Harris did not approve the submission of the manuscript, Haddow tried to persuade him that, not only did he risk either embarrassing himself over a false lead or leaving an investigation just before it reached its most important stages, but also that if Bell resigned and published independently, it would not be possible for any member of the EAVRI staff to appear as an author, "which would be disappointing to us."[126] Whether Bell had overreacted to the ICRF's hesitations or the ICRF was persuaded to change their mind, Harris did in fact submit the manuscript to the *BMJ* a few days later with some minor changes.[127] He sent a copy to Haddow along with a note indicating he thought Bell was "in some form of mental crisis" and requesting that Haddow "cool him off."[128]

As it transpired, Haddow had not been in a position to soothe Bell's ruffled feathers. He replied to Harris indicating that he hoped he could exert some influence over Bell in the future, but that for the previous several months the relationship between Bell and the EAVRI team had been strained by Bell's accusations that someone at the EAVRI had "leaked" information that allowed Epstein to culture the cells Bell was working on. Haddow informed Harris, "In the course of a discussion at my house (which went on till the early hours) he suggested that someone in the Institute had either sent rather detailed information to Epstein or had actually sent material."[129] While Haddow pointed out that there were a number of ways in which information might have traveled from Entebbe to Epstein without anyone at the EAVRI deliberately "leaking" it, Bell was unconvinced, and at last Haddow agreed that the EAVRI team would, for a period of one month, work "blind"—meaning they would process material for Bell without knowing which were experimental specimens and which were controls. Unsurprisingly, Haddow reported, "Woodall and particularly Williams resented this arrangement but did abide by it, albeit reluctantly."[130] Not

[126] Haddow to Harris, April 14, 1964, UVRIA, GC 15 "Cancer Research-ICRF."
[127] Harris to Haddow, April 14, 1964, UVRIA, GC 15 "Cancer Research-ICRF." The manuscript was published as Thomas M. Bell, Alexander Massie, M. G. R. Ross, and Miles C. Williams, "Isolation of a Reovirus from a Case of Burkitt's Lymphoma," *British Medical Journal* 1964: 1212-1213.
[128] Harris to Haddow, April 14, 1964, UVRIA, GC 15 "Cancer Research-ICRF."
[129] Harris to Haddow, April 17, 1964, UVRIA, GC 15 "Cancer Research-ICRF."
[130] Haddow to Harris, April 14, 1964, UVRIA, GC 15 "Cancer Research-ICRF."

long after this arrangement was made, Bell discovered that Wright in Mulago's department of pathology might have accidentally included Bell's media in the specimens he sent to Epstein, "thus enabling a start with culture in London."[131] In any case, the damage was done and Bell ceased accusing the EAVRI of sabotaging his work. As Marrian remarked to Haddow, "the whole situation has been complicated by our friend E. [Epstein] This is something you and I, Bell and Bob [Harris] have got to learn to live with."[132] By mid-April Haddow could report to Harris that the "coolness existing between the staffs on account of the suspicion that we had been helping Epstein surreptitiously… is now over, and there is full collaboration."[133]

These interpersonal tensions represented larger tensions between the imperatives of running a research station in Uganda on the one hand and supporting visiting representatives of larger, wealthier organizations on the other. Haddow's, and the EAVRI's, interests were best served by making visible the contributions they could make to important research projects such as the connection between a local cancer and a newly-discovered virus. They also had to navigate the local politics of the late colonial regime looking for successes to champion. The interests of the ICRF, on the other hand, were best served by being conservative. They had a reputation to protect and far more to lose from premature claims than the EAVRI. In order for the Institute survived beyond Independence, its senior management had to become adept at balancing these competing interests and others that came with international partners.

Transitions

The mid-1960s brought new challenges to the Institute. It continued to negotiate the terms on which it, as a part of an independent African government apparatus, could and should collaborate with European and American institutions. And its staffing shortages became even more acute. On April 30, 1965, after 23 years at the Institute and 13 years as its Director, Haddow retired and

[131] Haddow to Harris, April 14, 1964, UVRIA, GC 15 "Cancer Research-ICRF."
[132] Marrian to Haddow, April 20 1964, UVRIA, GC 15 "Cancer Research-ICRF."
[133] Haddow to Harris, April 14, 1964, UVRIA, GC 15 "Cancer Research-ICRF."

moved from Entebbe back to his home in Glasgow. Jack Woodall (virologist) had left at the beginning of the year to work for the Rockefeller Foundation in Brazil and David Simpson (Medical Research Officer) left in June for a post at Queen's University, Belfast.[134] This led to what J. W. Kibukamusoke, the Chairman of the East African Medical Research Council (EAMRC), described to Marrian as "a very difficult period" for the Institute.[135] The remaining senior scientific staff was reduced to a single virologist (Miles Williams), who was now directing the Institute. Already, visitors from London had commented that the previous several years had seen very interesting developments at the Institute connected to the vectors of virus disease but rather little work on the clinical dimensions of human virology.[136] Kibukamusoke was anxious for Marrian to dispel the rumors that Bell might also be leaving Entebbe and emphasized how important Bell's work was to the continuity of research on Burkitt's lymphoma at the Institute. He implored Marrian to either leave Bell in Entebbe or replace him with another virologist.[137] He assured Marrian, "It is our intention that the Virus Research Institute should not close despite the difficulties it is experiencing at the present time. Arrangements are being made to resuscitate the Institute and it should not be long before it is fully running again. My main concern, however, is that work on Burkitt's lymphoma should continue."[138]

Bell did stay, but in the later years of EAVRI-ICRF collaboration, conflict frequently arose around three main issues: housing, laboratory space, and the secondment of African medical graduates. Nonetheless, Williams, as the Director of the EAVRI, was keenly aware of the importance of keeping the ICRF on board as a partner. In the course of a rather tense negotiation over housing allocations in June 1965, Williams wrote to Bell:

> I would like to state that I and this Institute and in fact the whole of the E.A. Medical Research Council are anxious to maintain the good relations that have existed between the Imperial Cancer Research Fund and ourselves in the past. We recognize the value of

[134] M.C. Williams, "Introduction and Summary," *EAVRI Report* January-December 1965, 4-5.
[135] Kibukamusoke to Marrian, April 1, 1965, UVRIA, GC 15 "Cancer Research-ICRF."
[136] Letter from G. Macdonald and G.M. Bull to Kibukamasoke [sic], January 30, 1965, TNA FD 7/1509.
[137] Kibukamusoke to Marrian, April 1, 1965, UVRIA, GC 15 "Cancer Research-ICRF."
[138] Kibukamusoke to Marrian, April 1, 1965, UVRIA, GC 15 "Cancer Research-ICRF."

> the contribution that has come from the Imperial Cancer Research Fund both scientifically and otherwise.[139]

Throughout the EACSO, there was growing awareness that research programs were having enormous difficulties recruiting and retaining experienced scientists, and that in all likelihood such problems were going to persist for some time. Under the circumstances, the Social and Research Services Ministerial Committee drew attention to the potential benefits of "the type of arrangement where our research institute collaborates with a foreign institute in our research project."[140] Nonetheless, there were limits to the concessions the Institute and the EAMRC were willing to make to the ICRF and the degree of authority they were willing to cede. After consulting with Kibukamusoke on the heated question of housing allocations for the ICRF team, Williams agreed to Bell's request for three houses for two years, but included an admonition from Kibukamusoke to improve his attitude and allow Williams to run the Institute "without too much difficulty."[141] One of the contributions the ICRF made to the Institute in 1965 was the offer to train an African doctor in virology.[142] In November, the EAVRI welcomed the newly appointed trainee, Dr. Germano Munube, who was immediately seconded to the ICRF team to assist and be trained by Bell.[143] Munube was also expected to act as the liaison between the ICRF project and Mulago Hospital.[144] Between 1965 and 1966 the staff was augmented by a number of new hires. In August Munube was joined by Louis Mukwaya, a trainee entomologist.[145] Peter M. Tukei was brought on as a virologist in July 1966 at the same time A.W.R. McCrae came in to head the Department of Entomology.[146] In December, American virologist Brian Henderson of the Centers for Disease Control was seconded to the Institute.[147] Gradually the permanent scientific staff of

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[139] Williams to Bell, June 16, 1965, UVRIA, GC 15 "Cancer Research-ICRF."
[140] Memo from D.M. Wako, December 31, 1965, UVRIA, GC 15 "Cancer Research-ICRF."
[141] Williams to Bell, June 16, 1965, UVRIA, GC 15 "Cancer Research-ICRF."
[142] Munube to Kibukamusoke July 27, 1965 and Kibukamusoke to Williams, July 30, 1965, UVRIA, GC 15 "Cancer Research-ICRF."
[143] Miles C. Williams, "Introduction and Summary," *EAVRI Report* January-December 1965, 5.
[144] Williams, "Introduction and Summary," 5.
[145] Williams, "Introduction and Summary," 5.
[146] Williams, "Introduction and Summary," 4.
[147] Williams, "Introduction and Summary," 4.

the EAVRI was becoming more African in its composition, but Europeans and Americans still held the most senior positions.

One of the characteristics of the EAVRI-ICRF partnership was its limited and uncertain duration, a challenge that would plague future partnerships as well. There was always an awareness on the part of the Institute's staff that the agencies and individuals partnering with EAVRI had finite investments in the infrastructure, professional development, and overall future of the Institute. Periodically, when agreements were due for reconsideration and renewal, old points of contention (housing and lab space, publication, the sharing of information) resurfaced. Additionally, in the mid 1960s, there appears to have been some anxiety on the part on the ICRF that the EAVRI would take advantage of the training offered by the ICRF to their seconded staff, particularly Munube, to duplicate or compete with the work being done by the ICRF team.[148] At the same time the EAVRI still resented the "secrecy" and autonomy of the ICRF team.[149] While the directors of the YFRI had been accountable to the IHD and the CMRS, they could be confident that all of the activity taking place in their laboratories and field sites was under their direction. With the ICRF, there apparently were concerns that in the ICRF-controlled lab rooms discoveries were being made without the knowledge of the EAVRI Director and that there was less-than-full transparency between the teams. Understandably this undermined the sense of partnership between the two agencies.

In early 1967 negotiations for the ICRF to work for two more years in Entebbe nearly foundered on these familiar issues. Williams was concerned enough about striking the proper tone in his correspondence with the ICRF that he sent a draft of a letter to Marrian in February to Benjamin Lush of the U. K. Medical Research Council for his comments.[150] In the draft letter, Williams expressed his desire for the ICRF to continue its work at the EAVRI until July 1969, but

[148] Draft of a letter from M.C. Williams to Marrian February 17, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."
[149] UVRI Archives, GC 15 "Cancer Research-ICRF". Letters between Anderson, Lush, M.C. Williams, Bell, and Kibukamusoke in 1967, 1968.
[150] Williams to Lush, February 17, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."

warned that there were obstacles to making the ICRF's desired laboratory and housing allocations. He further stated that he did not intend to extend Munube's secondment to the ICRF and that he had some concerns about the independence that the ICRF team tended to exercise from the overall operation of the Institute. He also called for "no secretive work."[151] Lush suggested eliminating much of the detail around the space issues and all references to personnel and secrecy issues explaining, "These I think would be much better brought out in discussion rather than being put on the record, where they might arouse premature antagonism."[152] Overall Lush's revised draft adopted a far more conciliatory tone than Williams's initial one.[153] Williams discussed Lush's suggestions with Dr. Anderson of the EAMRC and then sent the revised draft to Marrian.[154]

Williams's first draft of the letter, though not sent to the ICRF, sheds some light on the exact nature of the tensions between the ICRF and the EAVRI. In it he wrote that he had "grave doubts in view of the previous difficulties if it is a good arrangement to second one of our officers to a totally independent group within the building."[155] He referred to Bell's expressed concern about potential clashes of interest between the two research teams and, in the interest of avoiding them, outlined the Institute's planned research program:

> The Institute's research program will include the development of studies in West Nile related to cancer, studies on reovirus in mosquitoes, serological survey and the investigation of minor outbreaks of disease to give serological controls for Burkitt's tumour and other studies. The tissue culture section, the expansion of which was delayed initially to show that no competition was intended with the visiting ICRF group, must now be expanded and officers including Dr. Munube will be given a free hand to work with such viruses as are considered of interest including reoviruses. There could be potential clash in such studies particularly as Dr. Bell has trained Dr. Munube. It is to be hoped that during the two year period if the ICRF stay that the Institute will gain the experience to take over certain of the aspects of Burkitt's tumour study which may continue over a longer time by collaboration with the ICRF, and that a longer term association may continue.[156]

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[151] Draft of a letter from Williams to Marrian, February 17, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."
[152] Lush to Williams, February 23, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."
[153] Draft II of a letter to be sent from Williams to Marrian, UVRI Archives, GC 15 "Cancer Research-ICRF."
[154] Williams to Marrian, February 27, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."
[155] Draft of a letter from Williams to Marrian, February 17, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."
[156] Draft of a letter from Williams to Marrian, February 17, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."

He went on to say Bell, should he stay, "should work with our staff, along agreed lines, which can be reviewed as required. Under these circumstances there would have to be no secretive work."[157]

The question of the degree to which the ICRF could and should be required to operate with transparency continued to be discussed among Williams, Anderson, and Lush. Lush and Anderson sympathized with Williams's frustration, but Anderson was also anxious to avoid alienating the ICRF or other visiting research teams. He forwarded a memo from D. M. Wako of the Social and Research Services Ministerial Committee from December 31, 1965 to the Directors of Research Institutes and Organisations under the EACSO in which the terms of collaboration with foreign donor governments were laid out.[158] Among other things, they included a commitment from the EACSO not to "interfere in the actual conduct of research work of visiting research teams."[159] He further noted that Wako had confided in him that when he visited Europe in 1965 to attempt to recruit assistance in the form of visiting research teams, "the governing bodies of these teams made it quite clear that their visiting teams would not come under the control of E.A.C.S.O. Directors in the actual content of research."[160] Under the circumstances, therefore, Bell was not required to collaborate with the EAVRI staff on his actual research work unless he chose to do so. As Anderson summarized the situation, "It is a matter of personal relationship and confidence."[161] Anderson also made it clear that the imperative to extend some privileges to visiting teams was not limited to the ICRF; East Africa could look forward to a time in the future when it could supply its own scientific research expertise, but it would take time. He counseled:

> Consequently, however much it goes against the grain, we shall have to depend on outside workers for a considerable period of time until our own scientists and technicians have leveled up to the task. The outside workers will gain from us as much as they give. It is to our advantage in East Africa that there are teams of men desirous of using the facilities of our establishments to work and at the same time keep us abreast of

[157] Draft of a letter from Williams to Marrian, February 17, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."
[158] Memo from D.M. Wako, December 31, 1965, UVRI Archives, GC 15 "Cancer Research-ICRF."
[159] Memo from D.M. Wako, December 31, 1965, UVRI Archives, GC 15 "Cancer Research-ICRF."
[160] Anderson to Williams, March 2, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."
[161] Anderson to Williams, March 2, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."

> developments elsewhere. Consequently, our best recommendation to others are those that have worked in our laboratories.[162]

Williams was under extraordinary pressure to establish a good working agreement for the following two years because in 1968 he would retire and turn the reins over to director-designate George W. Kafuko, the Institute's first African director. Williams and Kafuko consulted with each other frequently on the best way to move forward with the ICRF, and Williams reminded Kafuko to keep his eye on the long-term prospects of the Institute, noting in one letter, "you will require a well trained and responsible team in 1969 not Dr. Bell."[163] Anderson, too, counseled Kafuko to think about positioning the Institute for the future and added, "personally I look forward to the day when we can do this sort of thing from our own resources. We shall not only be trully [sic] independent but mature and able to return in kind if not in cash to those who have helped us."[164]

Williams continued discussions with the ICRF in person in London while on home leave. He wrote to Kafuko describing his meeting with Marrian and Harris in which he emphasized "that there would be a need for a change from the emphasis on independence to an emphasis on collaboration—in particular as we expected some 50% of the time of our staff to be spent on cancer studies in particular Burkitt's lymphoma studies, and as also we planned to continue the work after 1969."[165] Finally in June a final agreement was reached outlining the terms under which the ICRF team under Bell's direction would stay in Entebbe through 1969.

In the meantime, Bell was struggling to reconcile his work on the reovirus with persistent isolations of herpesvirus in tumor tissue. He proposed in 1967 that perhaps the reovirus and a

[162] Anderson to Williams, March 2, 1967, UVRI Archives, GC 15 "Cancer Research-ICRF."
[163] Williams to Kafuko, n.d. [but around April 3, 1967], UVRI Archives, GC 15 "Cancer Research-ICRF."
[164] Anderson to Kafuko, April 15, 1967, UVRIA, GC 15 "Cancer Research-ICRF."
[165] Williams to Kafuko, n.d. UVRIA, GC 15 "Cancer Research-ICRF."

herpes virus were "co-carcinogens" in which case, "The high incidence in tropical Africa may then be related to the arthropod-vectoring of the reovirus."[166]

Conclusions

By 1970, when Burkitt and Wright published their collection of articles summarizing the findings of various approaches to the study of the tumour, Uganda was more than ever fully established as being "on the fringes of the scientific world."[167] The reovirus isolated in Entebbe was still under investigation in connection with Burkitt's lymphoma, but the best candidate for the causal agent was widely believed to be the herpes-type virus isolated in Anthony Epstein's lab in the U.K. and under study at Gertrude and Werner Henle's lab in New York.[168] Developments in cell culture production had overcome the necessity of locating major research projects in Africa or at least in close contact with someone who could regularly supply fresh specimens, and investigators interested in the cellular- and tissue-level carcinogenic processes had no great incentive to partner with African institutions. Moreover, as virology relied more and more on high-precision and high-cost instruments such as electron microscopes, the labs at Entebbe appeared more and more antiquated.

Not everyone was convinced that the tumor was caused by a virus, or even that the tumor was in fact a novel disease entity. A team of researchers from Sloane-Kettering in New York and the Medical Research Laboratory in Nairobi published an article in 1964 suggesting that the epidemiological data from Kenya, as well as cases that had been identified over the previous few years outside Burkitt's initial geographical limits, pointed to the tumor being "an old familiar one,

[166] Thomas Bell, "Review of the Evidence for a Viral Aetiology for Burkitt's Lymphoma," *Treatment of Burkitt's Tumour: Proceedings of a Conference organized by the Chemotherapy Panel of the International Union Against Cancer,"* 1967.
[167] Himsworth in Burkitt and Wright, *Burkitt's Lymphoma*, v.
[168] Bell, "Isolations of Reovirus Type 3," in Burkitt and Wright, *Burkitt's Lymphoma*, 222-230; Epstein and Achong, "The EB Virus," in Burkitt and Wright, *Burkitt's Lymphoma*, 231-248.

only modified in form and course."[169] In that case, they added, "The modifying factor, or factors, rather than the primary cause would then be sought in terms of an anophiline vector."[170] The modifying factor they suggested was "a noteworthy and exceptional form of malaria."[171]

Lewthwaite's prediction to Haddow in 1965 that "the persistent relentless work which you and your staff are doing alongside Dr. Bell will figure largely in the story of the unraveling of its aetiology when it is ultimately revealed" did not entirely come to pass.[172] In retrospect, Bell's anxiety to establish priority with early publications (even before all the information was in) was understandable—cancer research turned out to be very much a race-to-the-finish, winner-takes-all-proposition. The collection of Burkitt's lymphoma-related essays published by Burkitt and Wright in 1970 includes a chapter by Bell on the reovirus isolations, which gives substantial credit to his partners at the EAVRI.[173] But later accounts have largely written Entebbe out of the story. Even Bell has been largely overlooked in the history of the virus-tumor connection and Epstein and the Henles (along with Burkitt, of course) are the main protagonists of most narratives for popular and professional audiences. According to this widely cited narrative, Epstein chanced upon a lecture by Burkitt at Middlesex, was intrigued, and offered to cover the costs of shipping specimens to the U.K. for testing.[174] Then he isolated an agent, which he shared with the Henles, who had been advised by Everett Koop that it was a good candidate for a cancer

[169] Gilbert Dalldorf, C.A. Linsell, Frances E. Barnhart, and Ruth Martyn, "An Epidemiologic Approach to the Lymphomas of African Children and Burkitt's Sarcoma of the Jaws," *Perspectives in Biology and Medicine* 7 (1964): 435-449. Haddow cited this article as an example of one of the clues he and others missed due to a "blind spot" among the teams that "stood too close" to the arbovirology question in the early years of the Burkitt's lymphoma investigations in his lecture "Mosquito-Borne Viruses—The Need for a Balanced Team," presented at the Royal Society of Tropical Medicine and Hygiene, Centenary of Medical Entomology Symposium held in London, November 23-25, 1977, Glasgow University Archives, DC 68/77.
[170] Gilbert Dalldorf, C.A. Linsell, Frances E. Barnhart, and Ruth Martyn, "An Epidemiologic Approach to the Lymphomas of African Children and Burkitt's Sarcoma of the Jaws," *Perspectives in Biology and Medicine* 7 (1964): 435-449.
[171] Dalldorf, Linsell, Barnhart, and Martyn, "An Epidemiologic Approach," 435-449.
[172] Lewthwaite to Haddow, June 2, 1964, UVRI Archive, CG 15 "Cancer File—ICRF."
[173] Bell, "Isolations of Reovirus Type 3," in Burkitt and Wright, *Burkitt's Lymphoma*, 222-230.
[174] A lecture by Dr. Denis Burkitt at the Wellcome Trust on July 26, 1988, audiocassette, WL; "Sir Anthony Epstein in conversation with Denis Burkitt," Oxford, March 20, 1991, Audiocassette and transcript, WL.

virus.[175] The Henles then serendipitously identified is as the cause of infectious mononucleosis when one of their lab technicians fell ill.

In retrospect, the search for a single arbovirus that could account for the lymphoma was largely disregarded as a bit naïve. But the arbovirus theory was still productive. As cancer researchers have continued to build their understanding of the complexity of oncogenesis, some of the work from the Institute has received renewed appreciation. As a group of medical researchers recently reflected:

> The search for an arthropod borne virus was unsuccessful, yet a ubiquitous herpes virus was discovered in [Burkitt's lymphoma] cells whose role in the pathogenesis of the disease is still not fully understood. The concept of an arthropod-borne transmissible agent has nevertheless been most fruitful as it paved the way for the identification of holoendemic malaria as the most important cofactor for the development of BL in endemic areas in Central Africa and New Guinea.[176]

But overall, the collaboration between the EAVRI and the IARC has been relegated to a footnote in most accounts of the Burkitt's lymphoma story.

However, the Institute was not finished with research on Burkitt's lymphoma in 1969. If anything, the previous decade had taught them the value of membership in the elite group of cancer research bodies and the depth of interest in the African lymphoma problem. Over the course of the 1970s they would continue to work on the lymphoma, though with a change of focus and a new cast of international partners. Chapter Four will take up the investigations based in the West Nile District in combination with a variety of local and international partners to further untangle the relationship between the various factors implicated in the lymphoma question.

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[175] "Sir Anthony Epstein in Conversation with Denis Burkitt," Oxford, March 20, 1991, Audiocassette and transcript, WL.

[176] Sebastian Grömminger, Joseph Mautner, and Georg W. Bornkamm, "Burkitt Lymphoma: The Role of Epstein-Barr Virus Revisited," *British Journal of Haematology* 156, no. 6 (2012): 719-729, quotation on 726.

Figure 1: From Burkitt and O'Conor 1961. (with permission)

FIG. 4. Map of Africa in which the white areas are those within which the mean temperature is above 15°C and the annual rainfall more than 20 inches.

Figure 2: A map based on the one Haddow developed. From Burkitt 1983. (With permission)

FIG. 1. "The Lymphoma belt" in Africa which showed the approximate distribution of the tumor.

Figure 3: From Burkitt 1983. This version of the map was widely reproduced in popular coverage of the tumor investigations. (With permission)

Chapter Four
Maintaining Experimental Stability in Unstable Times: Burkitt's Lymphoma Investigations in West Nile, 1965-1979

Introduction: Zooming in on West Nile

The discovery of Burkitt's lymphoma, a cancer that appeared to be limited to particular parts of Africa and caused by a virus, attracted a range of researchers with a stake in the field of cancer viruses to Uganda. As an article in *The New York Times* put it, "The importance of the inquiry [into Burkitt's lymphoma] is obvious from the procession of cancer specialists from many parts of the world to Kampala and Entebbe."[1] At the same time that they were partnering with the Imperial Cancer Research Fund (ICRF) project in Entebbe (described in Chapter Three), the East African Virus Research Institute (EAVRI) was also collaborating with experts at Makerere University and rural physicians to conduct its own investigations into the etiology of the tumor. Health officials in Uganda and the rest of the East African Community (EAC) saw cancer, and its potential connection with arboviruses and other conditions of the tropics, as a way to demonstrate that independent African nations could continue to generate important medical knowledge for the rest of the world. The history of investigations led by the EAVRI team in the West Nile District highlights the crucial role of African scientists in designing and implementing internationally funded research projects in Uganda in the 1970s, a role they would increasingly play in the conduct of viral research in Africa in the ensuing decades.

While the first developments in Burkitt's lymphoma research were accomplished by mapping the distribution of the tumor on a continent-wide basis, subsequent investigations were more local. Denis Burkitt, the surgeon who first described the eponymous tumor, used a laboratory metaphor to explain his own shift, first from a world-wide view, then to an African, a regional, and finally a Ugandan view:

> Viewing the world first and Africa subsequently might be compared to examining a microscope slide initially through a low-power lens and then using a higher magnification to examine a smaller segment. Using this simile, we turned

[1] John Hillaby, "Study of African Cancer Indicates Link to Virus," *The New York Times* November 25, 1962.

> next to East Africa, since we had studied the tumor intensively throughout all of the East African territories. Using a still higher powered objective, once again we found a closer relationship between tumor incidence and intensity of malaria. Finally we focused on Uganda and used the oil immersion.[2]

Similarly, former EAVRI director Alexander Haddow observed in 1970 that Uganda "forms a microcosm which illustrates in miniature the relationship between tumour distribution and environmental factors as they apply elsewhere in Africa."[3] But even Uganda was too large an area for the kind of cohort study that the EAVRI hoped would provide definitive evidence of a cause of the tumor syndrome. The EAVRI chose West Nile as the location of the intensified investigations for several reasons. Arua, the capital of the district, was easily accessible by bus and the district as a whole had comparatively good communication infrastructure.[4] Perhaps the most important factor in the choice of West Nile as a study location was that Ted Williams, one of the doctors who had accompanied Burkitt on his "long safari" in 1961, operated a mission hospital in the district where he had been keeping careful records and was enthusiastic about collaborating on the tumor research.

The investigations that followed, on which the EAVRI, Williams, and the World Health Organization's International Agency for Research on Cancer (IARC) collaborated, represented a major shift for the Institute. The Institute, largely staffed with entomologists, had previously concentrated on arboviruses and regarded people as relatively minor players in the viral transmission cycles. While they still believed that a mosquito-borne virus was implicated in Burkitt's lymphoma, their prospective studies had to incorporate human geography to a greater degree than their earlier yellow fever work. Establishing an intimate understanding of the locality of the West Nile district required a different set of skills than those used to solve the puzzle of yellow fever transmission in the forest of Bwamba district. Chief among these was the ability to translate knowledge of the social, biological, and physical environment of West Nile into

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[2] Denis P. Burkitt, "The Discovry of Burkitt's Lymphoma," *Cancer* 51, no. 10 (1983): 1777-1786, quotation on 1782.
[3] Alexander Haddow, "Epidemiological Evidence Suggesting an Infective Element in the Aetiology," in *Burkitt's* Lymphoma, eds., Burkitt and Wright, 198-209.
[4] M. C. Pike, Edward H. Williams, and Barbara Wright, "Burkitt's Tumour in the West Nile District of Uganda, 1961-5," *British Medical Journal* 2, no. 5549 (1967): 395-399.

cartographic representations amenable to statistical analysis. Once again, EAVRI researchers employed maps to make visible the etiology of a disease. This chapter shows the process by which "local" knowledge of place, disease, and human behavior was collected, evaluated, and translated into "objective" data that would be accepted as evidence of a causal relationship between Epstein-Barr virus and Burkitt's lymphoma in the court of international scientific opinion.

This chapter also examines how medical researchers in Uganda maintained their investigative work and adjusted to rapidly changing circumstances in a period of national chaos and tragedy. The Institute capitalized on its international reputation, achieved largely through its yellow fever investigations (Chapters One and Two), to leverage public and private resources to sustain a large cohort study for most of the 1970s, despite the growing wave of state-sponsored violence and exodus of expatriates from Uganda following Idi Amin's coup in January 1971. For some of the EAVRI investigators, the Burkitt's lymphoma work was an opportunity to make connections with international research agencies, some of which provided avenues out of the country when the situation got most precarious at the end of the decade.[5] For other researchers, the work was seen as a once-in-a-lifetime opportunity to observe a sort of natural experiment that justified the risks involved. The chapter explores the apparent paradox of the years of Idi Amin's regime being recalled as "golden years" at the EAVRI.[6] During this period, the Institute had to develop new strategies for attracting and cultivating partnerships with international agencies, strategies that would prepare them for the transformed landscape of biomedical research in Uganda following the discovery of AIDS.

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[5] George Olwitt Oral History (OH).

[6] Sylvester Sempala, "Institute profile: The Uganda Virus Research Institute," *Trends in Microbiology* 10, no. 7 (2002): 346-348. Historians have recently been giving new attention to the 1970s in an effort to add nuance to the history of a period often described in hyperbolic language. E.g. Godfrey Asiimwe, "From Monopoly Marketing to Coffee Magendo: Responses to Policy Recklessness and Extraction in Uganda, 1971-79," *Journal of Eastern African Studies* 7, no. 1 (2013): 102-124; Holger Bernt Hansen, "Uganda in the 1970s: A Decade of Paradoxes and Ambiguities," *Journal of Eastern African Studies* 7, no. 1 (2013): 83-103; Anneeth Kaur Hundle, "Exceptions to the Expulsion: Violence, Security and Community Among Ugandan Asians, 1972-79," *Journal of Eastern African Studies* 7, no. 1 (2013): 164-182; Alicia C. Decker, *In Idi Amin's Shadow: Women, Gender, and Militarism in Uganda* (Athens, Ohio: Ohio University Press, 2014).

The chapter begins with a description of the circumstances that led to West Nile being selected for further investigations and the assumptions that went into choosing it as a site of epidemiological research in the late 1960s and 1970s. It continues by describing the unique contributions of Edward "Ted" Williams to knowledge about cancer in the district and how his work came to the attention of the EAVRI and other international researchers. Then it describes the design and implementation of the joint EAVRI/IARC cohort study intended to provide definitive evidence of a link between Burkitt's lymphoma and Epstein-Barr virus, as well as possible co-factors in the development of the tumor. Finally, it closes with the collapse of the project in 1979, the results of the study, and the reasons that it was marginalized in subsequent accounts of the history of Burkitt's lymphoma. Like previous chapters, this chapter highlights the tensions between local knowledge and international expertise. It also profiles the tools used to make viruses, and the diseases they caused, visible to researchers.

People Fixed in their Places

The West Nile cancer studies were based on a particular view of Africa and Africans as diverse but static. The link between environmental exposure and cancer was a prominent topic in the 1960s. But investigations into the links between particular types of exposure and cancer were difficult to conduct—it was impossible to control for all variables in a sufficiently large population to find relatively rare outcomes. People were simply too likely to move between different environments, encountering too many potential carcinogens. The studies conducted on Burkitt's lymphoma in West Nile were explicitly dependent on "bodies in place"—people with disease or at risk for disease who could be identified with a single, persistent location.[7] Burkitt and his colleagues believed that the lives and health of people in sub-Saharan Africa were more fundamentally diverse than those of people in "technologically advanced countries" where "the majority of people live under very similar circumstances apart from specific hazards associated

[7] Crane, Scrambling for Africa, 157.

with special industries and individual customs."[8] This lack of diversity in "advanced countries" was due to people's mobility. In East Africa, on the other hand, they claimed, "there are still many groups of people living in circumscribed communities in different geographical circumstances and exposed to widely varying nutritional, social, economic, and other environmental factors."[9] This meant that East Africa represented a kind of natural experiment that researchers should be able to use to observe the different health outcomes of people living under different conditions. It also meant that it should be possible to track individuals and communities over relatively long periods of time without worrying about loss to follow-up. This perspective vastly underestimated the mobility of African individuals and populations both before and under colonial rule.[10] The idea of African societies as isolated, immobile, and immutable was little more than a colonial imaginary adopted by the medical researchers. Nonetheless, it was a powerful imaginary that underlay the approach of Burkitt, Williams, the EAVRI researchers, and their collaborators at the IARC. These assumptions made detailed cartography a natural choice for investigating the tumor in West Nile and dictated the use of researchers who could reliably identify the unique conditions of various places in the district—local researchers. Maps would make visible the differences between populations exposed to different environmental conditions.

Williams and his colleagues also believed that these "circumscribed communities" in East Africa were not likely to last for long. In the late 1960s, Williams told Bernard Glemser, who wrote a book on the discovery of Burkitt's lymphoma, "We have perhaps ten years left here in which to do the detailed mapping of cases of cancer."[11] As Glemser explained:

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[8] Michael Hutt and Denis Burkitt, "Geographical Distribution of Cancer in East Africa: A New Clinicopathological Approach," *British Medical Journal* 2, no. 5464 (1965): 719-722, quotation on 719.
[9] Hutt and Burkitt, "Geographical Distribution of Cancer," 719.
[10] This has been widely documented. Pre-colonial societies in Uganda and elsewhere were linked with one another and the wider Atlantic and Indian Ocean worlds by trade. Under colonial governments, vast numbers of Africans migrated for wage labor on settler plantations, in mines, and in cities. Wars, famines, and other causes of forced displacement also account for significant episodes of mobility in all regions of Africa. For Uganda and the region, see David E. Apter, *The Political Kingdom in Uganda: A Study of Bureaucratic Nationalism* (Princeton: Princeton University Press, 1961); Henri Médard and Shane Doyle, eds, *Slavery in the Great Lakes Region of East Africa* (Athens, Ohio: Ohio University Press, 2007); Neil Kodesh, *Beyond the Royal Gaze: Clanship and Public Healing in Buganda* (Charlottesville, Virginia: University of Virginia Press, 2010).
[11] Quoted in Bernard Glemser, *Mr. Burkitt and Africa* (New York: The World Publishing Company, 1970), 100.

> The reason is that virtually all underdeveloped countries are now in [the] process of development. In most parts of Africa the pattern of life is changing with extraordinary speed. Until recently, for example, the population of a fairly remote region like the West Nile District was relatively stable. Few people ever traveled more than a few miles from the village where they were born, simply because the means for travel were unavailable…Now there is a daily bus to Kampala, and it has been estimated that about thirty thousand West Nilers travel to Kampala every year…People will move freely from place to place in Uganda, as they do anywhere else in the world' and as a consequence, keeping track of disease will become increasingly difficult.[12]

Thus, while medical geography promised to shed light on the etiology of Burkitt's lymphoma, if scientists waited too long to conduct their studies, the opportunity could slip away.

Ted Williams and the Kuluva Cancer Records

Medical map-making, as we have seen, was a central tool in virus research in Uganda. However, Williams's choice of cartography as a tool for cancer research may in part have been a result of observing his father's work. Williams was born in 1915 to John Hammond Williams and Marion Lucy Wheeler Williams. At the time they were living in Nairobi where John Williams was employed in the Land and Surveys Department, rising eventually to the post of Chief Computer of Maps.[13] The family relocated to England in 1929, where Williams did his medical training at Bart's Hospital Medical School.[14] In 1940 he married Muriel Francis, a nurse, and the two applied to and were accepted by the Africa Inland Mission.[15] They arrived in West Nile in July 1941 where they set a about establishing Kuluva Hospital.[16] Their early decision to issue patients with individual record numbers that they kept throughout their lives, rather than reissuing numbers each year as at other hospitals, later permitted them to use patient records for longitudinal research purposes.[17] It also suggests that the Williamses viewed their patients as part of a community with some permanence, not just cases requiring attention while they were at the hospital. Williams came to be intimately familiar with the geography of the West Nile District,

[12] Glemser, Mr. Burkitt and Africa, 100.
[13] Edward H. Williams, "It Came to Pass," Wellcome Library Archives (WLA), WTI/EHW/D/12, page 2.
[14] Williams, "It Came to Pass," 4-5.
[15] Williams, "It Came to Pass," 14.
[16] Williams, "It Came to Pass," 24.
[17] Williams, "It Came to Pass," 27.

largely through his work on leprosy.[18] He also described to Glemser his process for making sure that he knew exactly where his pediatric cancer patients lived:

> With every one of my cases, after I've treated the child I take him, or her, home myself. So I know exactly where that child came from. And this *detailed plotting* is of tremendous importance. It is something that should be done by all missionary doctors who live and word for a lengthy period in underdeveloped countries.[19]

He believed that doctors needed to know about the places and conditions in which patients lived in order to understand their health or illness. He also wanted to be able to follow up with patients who didn't return to the clinic. This knowledge of the location in which each Burkitt's lymphoma patient was living at the time his or her symptoms developed was critical for the type of study the EAVRI and the IARC envisioned.

Williams's experience on Burkitt's tumour safari had sparked his interest in the question of medical research in Africa. He was frustrated by the tendency of his colleagues to write about Africa as a homogenous whole when it came to epidemiology and disease patterns. As he wrote in a letter to the editor of the *BMJ*:

> The tendency to make epidemiological generalisations is common in trying to explain disease patterns in developing countries…But I want to emphasize that in my opinion it is not correct to look on Africa as a homogenous whole from the point of view of disease patterns…My feeling is that epidemiology in Africa has now exhausted the value of generalisations, and that more attention should be paid to studying localized disease patterns and the reasons for the differences.[20]

Using his own observations from decades of practice in West Nile, Williams set out to do just that kind of research. As Williams put it to fellow physicians, "many of us have succumbed to his [Burkitt's] flattering prospect of being 'World authorities on cancer in our own areas.'"[21] Upon his return from the "long safari," Williams began keeping careful records of all of the cases of cancer in his practice.[22] Using printed form that included a sketch map of West Nile on which he

[18] Williams, "It Came to Pass," 66.
[19] Quoted in Glemser, *Mr. Burkitt and Africa*, 99. Emphasis in Glemser.
[20] Letter from Edward H. Williams to The Editor, *British Medical Journal*, October 9, 1973, WLA WTI/EHW/B/8.
[21] Edward H. Williams, "Observations on Tumours in the West Nile District," Sir Albert Cook Memorial Lecture delivered October 30, 1969, WLA WTI/EHW/B/8.
[22] Williams, "Observations on Tumours."

would indicate the location of the patient's home, Williams began to collect data points for the Kuluva Cancer Registry [Figure 1].

Doing what he later referred to with some self-deprecation as "signpost research," Williams constructed a collection of maps relating his observations of the tumor with the population distribution of the district and factors he surmised might be causal factors.[23] These included geographical formations and tobacco curing facilities [Figure 2].[24] Other maps related space and time to the tumor distributions, appearing to show a "drift" in lymphoma cases across the district [Figure 3].[25] When he showed his early maps to statisticians Richard Doll and Malcolm Pike in November of 1965, such as the map of what Williams believed to be a cluster of cases along the Anyau River [Figure 4] they were intrigued, though they expressed reservations about Williams's back-of-the-envelope statistical analysis and intuitive identification of patterns and relationships. What followed was a collaboration between Williams and Pike that led to a pair of papers on the distribution of tumor cases in West Nile District in the British Medical Journal and the British Journal of Cancer in 1967 and 1969.[26] It also resulted in Williams becoming educated in biostatistical methods. As he put it, "He [Pike] impressed on me the significance of space-time clustering as a clear indication of an infective agent involved in the aetiology of Burkitt's lymphoma, although I confess I failed to follow him into the intricacies of the statistical calculations involved."[27] Williams's growing sensitivity to the nuance of statistical vocabulary is evident in one of the maps he made showing a what he originally labeled a "clustering in time and place" of tumor cases but later revised to indicate simply "groups" [Figure 4—see the altered text

[23] Williams to Corry Van den Bosch, April 29, 1992, WLA WTI/EHW/G/2. He went on to explain that he meant, "it was merely making some observations which to a large extent depended on my being long resident in the area, which others were able to interpret in ways which at first were very surprising to me."
[24] WLA WTI/EHW/G/3.
[25] WLA WTI/EHW/G/3.
[26] Malcolm C. Pike, Edward H. Williams, and Barbara Wright, "Burkitt's Tumour in the West Nile District of Uganda, 1961-5," *British Medical Journal* 2, no. 5549 (1967): 395-399; Edward H. Williams, P. Spit, and Malcolm C. Pike, "Further Evidence of Space-Time Clustering of Burkitt's Lymphoma Patients in the West Nile District of Uganda," *British Journal of Cancer* 23, no. 2 (1969): 235-246.
[27] Williams, "Observations on Tumours."

in the upper right hand portion of the image.][28] Clustering, he must have been informed, had a very particular meaning in the world of cancer statistics.

Cancer clusters were the subject of considerable interest in the mid-1960s. In the United States, an apparent concentration of childhood leukemia cases in the suburban town of Niles, Illinois drew the attention of an investigator from the Communicable Disease Center.[29] His investigation of this apparent "cluster" of cancers was the first of 108 that the (renamed) Centers for Disease Control (CDC) would conduct over the next two decades across 28 states and 5 foreign countries.[30] Later clusters were identified near waste disposal sites and industrial facilities, though most were not found to be statistically significant after investigation.[31] These clusters tended to set off disputes between experts and highly-engaged lay communities who mobilized mapping, statistical analysis, and community organizing to demand investigation and remediation of perceived carcinogens.[32] By the late 1980s, cancer clusters were controversial and many epidemiologists viewed reports of clusters with a great deal of skepticism.[33] But in the early 1960s, when Williams showed Pike evidence of his cancer "cluster" in West Nile, they were of great interest. Seeing parallels to methods applied successfully to infectious disease investigations, Alexander Langmuir, a prominent epidemiologist at the CDC, for example, regarded "the orderly study of any grouping of cases [as] probably a worth-while procedure […] likely to be productive if carried out imaginatively by an enterprising epidemiologist."[34] Williams may not have been a formally-trained epidemiologist, but he was certainly enterprising.

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[28] WLA, WTI/EHW/G/2.
[29] Clark Heath and Robert J. Hasterlick, "Leukemia Among Children in a Suburban Community," *American Journal of Medicine* 34, no. 6 (1963): 796-812.
[30] Glyn G. Caldwell, "Twenty-Two Years of Cancer Cluster Investigations at the Centers for Disease Control," *American Journal of Epidemiology* 123, supplement 1 (1990): S43-S47.
[31] Phil Brown, "Popular Epidemiology: Community Response to Toxic Waste-Induced Disease in Woburn, Massachusetts," *Science, Technology & Human Values* 12, no. 3/4 (1987): 78-85.
[32] Brown, "Popular Epidemiology," 78.
[33] K. J. Rothman, "A Sobering Start for the Cluster Busters' Conference," *American Journal of Epidemiology* 123, supplement 1 (1990): 6-13.
[34] Alexander D. Langmuir, "Formal Discussion of: Epidemiology of Cancer: Spatial-Temporal Aggregation," *Cancer Research* 25, no. 8 (1965): 1384-1386.

The maps published in 1967 and 1969 show an evolution of Williams's medico-cartographic sensibilities. They have axes in eastings and northings, using the Universal Transverse Mercator (UTM) coordinate system [Figures 4-6].[35] This facilitated the calculation of distances between points on the map representing tumor cases, for the purposes of identifying clusters. Pike and Williams used these coordinates to calculate whether the proximity of any pairs of cases in place and time were greater than would be expected to occur by chance if they were not caused by a common factor. They concluded that, indeed, "the disease possesses the epidemic characteristic of 'drift'—patients whose dates of onset were close together tended to live closer together than could be expected on the basis of chance alone."[36]

But Williams was adamant that the pathological, virological, and statistical studies could only be conducted and given meaning with a proper appreciation of the culture of the communities being studied. After leaving Uganda permanently, he took the opportunity of a talk at the London School of Hygiene and Tropical Medicine on "Geographical Pathology in the West Nile District of Uganda" to admonish his audience: "I have found that my own understanding of the pathology of the West Nile has only come with an appreciation of cultural background, both old and new."[37] He was an advocate of the kind of knowledge of a place and its people that could only be achieved at a local level. Though not a Ugandan, Williams's long service in the West Nile district allowed him to speak as a particular kind of local expert. The African scientific staff of the EAVRI commanded a different form of local expertise, which they would leverage to attract research funding in the 1970s.

Constructing an Epidemiological Laboratory in West Nile

[35] The UTM system was developed by the U.S. Army in 1947 and subsequently used widely for military and other purposes. R. B. Langley, "The UTM Grid System," *GPS World* 9, no. 2 (1998): 46-50.
[36] Pike, Williams, and Wright, "Burkitt's Tumour in the West Nile," 398. "Drift" like "cluster" was a term used to describe a convergence of cases in time and place that appeared to exceed what would be expected if cases were randomly assorted.
[37] Edward H. Williams, "Geographical Pathology in the West Nile District of Uganda," A talk given at the London School of Hygiene and Tropical Medicine, n.d., WLA, WTI/EHW/B/8.

By the late 1960s, the results of a series of small investigations based on informal arrangements between Ted Williams in Kuluva, pathologist M. S. R. Hutt at Makerere University, and Malcolm Pike at the Medical Research Council (MRC) had convinced the EAVRI to seek funding for a permanent field station in West Nile. Just as Haddow had attempted to transform Bwamba into an experimental place for yellow fever research, the EAVRI researchers hoped to establish West Nile as a site in which they could conduct epidemiological research on Burkitt's lymphoma—a kind of population laboratory. In 1967 and 1968, the EAVRI applied for funds from the MRC and the Wellcome Trust to improve the infrastructure for their Burkitt's lymphoma investigations and other cancer studies in West Nile.[38] They proposed to locate a senior technician in the district full-time to be responsible for coordinating with local medical authorities, follow-up with cases, collect specimens, and provide on-the-spot assistance to the scientific directors of the study based in Entebbe and Kampala.[39] A small grant from the Children's Research Fund enabled them to start some preliminary work in the expectation that additional funding would be available if there were interesting results.[40]

In fact, the West Nile field station was part of a larger plan on the part of the EAVRI to ensure their continuing viability as a participant in the production of international biomedical knowledge. Part of their pitch was the possibility that establishing a population cohort to study Burkitt's lymphoma might then enable further studies on entirely different diseases. As Brian Henderson, an American virologist temporarily seconded to the EAVRI by the CDC, wrote to the Institute's director, "Once a group of people is under observation I think any other studies such as hepatitis, respiratory diseases, etc can be more easily tackled."[41] By using international interest in

[38] Michael S. R. Hutt and Miles C. Williams, "Request for a Grant to Assist Investigations Concerning Burkitt's Tumour and Other Cancers in the West Nile District, Uganda," Submitted to the Medical Research Council, June 28, 1967 UVRIA GC20 "West Nile Cancer Project"; George Kafuko, "Request for a Grant to Assist Investigations in Burkitt's Tumour, Other Caners and Diseases of Scientific Research Interest in West Nile District, Uganda," May 11, 1968, UVRIA GC 20 "West Nile Cancer Project."
[39] "Request for a Grant to Assist Investigations Concerning Burkitt's Tumour and Other Cancers In the West Nile District, Uganda", June 28, 1967, UVRIA, GC 20 "West Nile Cancer Project".
[40] Sir Wilfrid Sheldon to H. Greenwood of The Children's Research Fund, 15 August 1967 and 28 November 1967, UVRIA GC20 "West Nile Cancer Project".
[41] Brian [Henderson] to Kafuko, June 20, 1968, UVRIA GC20 "West Nile Cancer Project.

Burkitt's lymphoma to generate funds to establish a field station and the infrastructure to conduct population studies, the EAVRI could position itself to be an attractive partner for international research on a number of different diseases.

There was some skepticism that a large population-based cohort study could be sustained in West Nile.[42] Using a $12,000 annual contract from the U.S. National Cancer Institute (NCI) Viral Cancer Program (VCP) with Makerere University, the EAVRI did a pilot project to establish the feasibility of a larger study.[43] Neither the researchers nor their funders took the feasibility of such a study for granted. Longitudinal cohort studies were still relatively unusual in Africa. Only a handful of them are listed in PubMed from the 1960s with a growing number in the 1970s. Earlier studies that likewise followed a group of healthy individuals in order to observe the development of disease involved far smaller numbers of people and tended to be clinic-based.[44] In November 1968 a team of researchers from the EAVRI, the IARC, and Makerere University Medical School collaborated with Ted Williams to collect blood specimens from 1122 children in the West Nile District. The specimens were sent to Gertrude and Werner Henle's laboratory at Children's Hospital in Philadelphia for measurement of EBV levels. 18 months later the study team was able to account for all of the original 1122 children, 97% of whom were available for further testing.[45] The study concluded that the stability, cooperation, and

[42] International Agency for Research on Cancer, "Report of a work group meeting to plan a prospective sero-epidemiological study of Burkitt's lymphoma in East Africa: Entebbe, Uganda, 16-18 November 1970.
[43] Viral Oncology Program, "Special Virus Cancer Program Progress Report #8," Bethesda, 1971, 207.
[44] P. D. Marsden, "The Sukuta Project: A Longitudinal Study of Health in Gambian Children from Birth to 18 Months of Age," *Transactions of the Royal Society of Tropical Medicine and Hygiene* 58 (1964): 455-489; L. J. Bruce-Chwatt, "A Longitudinal Survey of Natural Malaria Infection in a Group of West African Adults," *The West African Medical Journal* 12 (1963): 141-173. Prospective cohort studies were still a relatively new tool in epidemiology in general. One of the most famous early cohort studies, the Framingham Heart Study, was started in 1948 but, as historian Robert Aronowitz observed, only came to embody what would come to be recognized as the modern cohort study over the first several years of its implementation. Robert A. Aronowitz, "The Framingham heart study and the emergence of the risk factor approach to coronary heart disease, 1947-1970," *Revue d'Histoire des Sciences* 64, no. 2 (2011): 263-295. Precursors of the cohort study form such as longitudinal studies of a group of individuals over time without a clear mechanism for establishing causality were also known in Africa, though still uncommon in the 1960s. See, for example, C. A. M. Wenne-van der Mey, "A Longitudinal Study of Children at Igbo-Ora, Nigeria," *Tropical and Geographical Medicine* 20, no. 4 (1968): 335-341. Mervyn Susser, "Epidemiology in the United States After World War II: The Evolution of Technique," *Epidemiologic Reviews* 7 (1985): 147-177.
[45] George Kafuko, Brian Henderson, Barnabas Kirya, *et al*, "Epstein-Barr Virus Antibody Levels in Children from the West Nile District of Uganda: Report of a Field Study," *Lancet* 229, no. 7753 (1972): 706-709.

immunological profile of the District's population were all consistent with the requirements for a long-term cohort study in West Nile.[46]

Beyond the Virus: Malaria Studies and the Search for Co-Factors

While the IARC-funded project focused rather narrowly on measuring the relationship between EBV virus antibodies and the tumor, other investigations took a broader approach to the problem Following the publication of Williams's observations about Burkitt's lymphoma cases in West Nile, the IARC convened a meeting in Nairobi in December 1968 to discuss the status and future of research on Burkitt's lymphoma and its connection to Epstein-Barr virus (EBV).[47] One main theme of the conference was the need to consider cofactors in the development of the tumor, given that EBV was virtually ubiquitous and certainly not limited to the areas where the tumor was found. An editorial in the *East African Medical Journal* (*EAMJ*) reported on the discussions at the meeting:

> Recent studies cast doubt on the likelihood of this being a simple vector-borne virus disease; while the tumour is found under certain climatic conditions, its distribution is not entirely coincident with this. More precise mapping has shown that areas in which the tumour is endemic are areas where malaria is hyperendemic.[48]

The possibility of a relationship between malaria and Burkitt's lymphoma had first been suggested by Gilbert Dalldorf, an American pathologist and virologist working with data from the Kenya Cancer Registry in the early 1960s, as part of his argument that perhaps the BL was not a novel clinical entity but merely a particular manifestation of the familiar lymphoblastic leukemia.[49] Dalldorf's reluctance to accept the lymphoma as a distinct clinical entity gave him a

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[46] Kafuko et al, "Epstein-Barr Virus Antibody Levels," 709.
[47] "The Burkitt Lymphoma, Infectious Mononucleosis and the Epstein-Barr Virus," *East African Medical Journal* 46, 7 (1969): 400-401.
[48] "The Burkitt Lymphoma, Infectious Mononucleosis and the Epstein-Barr Virus," 400-401. The editor of the *EAMJ* at the time was Dr. H. M. Cameron, a pathologist.
[49] Gilbert Dalldorf, "Lymphomas of African Children with Different Forms or Environmental Influences," *JAMA* 181, no. 12 (1962): 1026-1028; Alice Stewart, J. N. P. Davies, Gilbert Dalldorf, et al, "Malignant Lymphomas of African Children," *Proceedings of the National Academy of Sciences* 70, no. 1 (1973): 15-17; Gilbert Dalldorf, C. A. Linsell, Frances E. Barnhart, et al, "An Epidemiologic Approach to the Lymphomas of African Children and Burkitt's Sarcoma of the Jaws," *Perspectives in Biology and Medicine* 7, no. 4 (1964): 435-449.

different perspective on the possible causes of the characteristic tumors than Burkitt and his colleagues in Uganda. As Dalldorf and his co-authors put it:

> Judgment necessarily hinges on interpretation of the nature of the disease. If one believes it to be a unique disease, one naturally searches for a unique etiology. Its geographical distribution suggests that it is probably mosquito-transmitted. If, on the other hand, the disease is an old familiar one, only modified in form and course, it follows that the primary cause need not be unique but may be widely shared by other peoples. The modifying factor, or factors, rather than the primary cause would then be sought in terms of an anophiline vector. Among the factors which fit the geographical distribution is a noteworthy and exceptional form of malaria.[50]

Malaria was not the only factor Dalldorf and his colleagues in Kenya considered. They remarked on the similarities and differences between the cultural practices of tribes that occupied areas with the highest and lowest incidence of the tumor in Kenya, such as the age children were weaned (similar), diet (different), and oral hygiene (inconclusive). They also studied the environments in which tumors were rare or relatively common, capturing rodents for virus studies, measuring radioactivity, and observing the construction of domestic spaces. But it was malaria, specifically holoendemic malaria, that appeared to be the most plausible co-factor.

At the time Dalldorf's conjectures about malaria and Burkitt's lymphoma were published, most of the resources of the EAVRI and its partner, the ICRF, were still directed towards the search for a single virus that would satisfy Koch's postulates and prove to be the unique cause of the tumor. It wasn't until the intensive search for a single arbovirus that could cause the cancer had been abandoned and intensive studies of the serology of children with and without the lymphoma or in areas characterized by high or low tumor incidence had been undertaken that the malaria hypothesis was revisited.[51] After the failure of the ICRF program to isolate a single virus that could be found in Burkitt's lymphoma patients and not in people without the tumor, the EAVRI team was ready to consider a more complicated etiology.

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[50] Dalldorf, Linsell, Barnhart, et al, "An Epidemiologic Approach," 446. Alexander Haddow later recalled the lack of attention paid to this article as one of the "blunders" along the route to discovering the causes of Burkitt's lymphoma. Alexander J. Haddow, "Paper for the Centenary of Medical Entomology Congress," UGA, DC 68/77.
[51] Haddow, "Paper for the Centenary."

In a way, the more complicated the etiology, the better for the African-based researchers. As they struggled to find a way to maintain the EAVRI without the relative security of colonial funding, research programs that emphasized factors best investigated on location in Uganda offered them a competitive advantage. While viruses could be isolated from tissue sent to labs across the world, studies on the possible co-factors that led to BL had to be conducted *in situ* in the communities where the tumor was found and by researchers attuned to the lives of the study population and willing to commit to lengthy investigations. Having been "scooped" in the matter of EBV by American and British scientists with superior laboratory resources, access to electron microscopy, and sophisticated tissue culture facilities, the EAVRI may have welcomed the suggestion that in order to really understand the relationship between the virus and the tumor investigations would have to move beyond outside the laboratory. The Institute's new director, Dr. George Kafuko, was especially well poised to investigate the link between Burkitt's lymphoma and malaria.

Kafuko was the Institute's first African director. Kafuko had been recruited to serve as director-designate under the previous director, Miles Williams, in 1966. Before coming to the EAVRI, Kafuko had worked for the Uganda Ministry of Health (MOH) and the Uganda Malaria Eradication Pilot Project, a joint program of the MOH and the World Health Organization (WHO). In 1968 he assumed the directorship and Miles Williams spent the last few months of his time in Uganda as a scientific consultant to the Institute to complete the transition before retiring later that year. Later that year, the Institute's clinician Ellen Knight died, leaving only two expatriates among the senior scientific staff: Brian Henderson and Angus McCrae, an entomologist. As Kafuko wrote in that year's annual report, "This is the first report to be written about work done by this Institute under the direction and supervision of an East African Director with the majority of the scientific and technical staff being East Africans. This marks another

milestone in the historical development of the Institute."[52] While the Africanization of the Institute was a source of pride for Kafuko and other Ugandans, it also meant the further erosion of ties connecting the Institute to wealthier institutions in the United States and Great Britain.

Like Haddow before him, Kafuko collaborated with Denis Burkitt on a new interpretation of the tumor distribution maps. Relying on existing data, they compared the regions of malaria endemicity in Uganda with the locations of Burkitt's lymphoma cases to create a new map highlighting the relationship between the two diseases [Figure 7]. Their co-authored article in the *International Journal of Cancer* concluded, "If the EB [Epstein-Barr] virus is an aetiological agent, malarial infection provides the additional factor responsible for its high incidence in certain tropical areas."[53] But most international resources were still directed at settling that "if" question—if EBV really was an etiological factor in the tumor.

The IARC and the West Nile Cohort Study

In November 1970, representatives from the EAVRI, IARC, the NIH, the NCI, and the VCP met in Entebbe to organize a prospective cohort study that would, they hoped, provide definitive evidence of EBV as a precursor to Burkitt's lymphoma.[54] Drs. Geser and Day of the IARC introduced their proposal. The purpose of the projected study was to test the link between EBV and Burkitt's lymphoma and, if there was a connection, to approximate the period between EBV infection and tumor development for the purposes of a future vaccine trial.[55] Based on preliminary information about Burkitt's lymphoma age incidence in West Nile, EBV antibody titers in West Nile, and evidence favoring a short latency period for Burkitt's lymphoma tumours, the proposal settled on a series of testable hypotheses. The null hypothesis was that there was no relationship at all between EBV immunity and Burkitt's lymphoma. The second possible

[52] EAVRI Annual Report 1968, page 1.
[53] George Kafuko and Denis Burkitt, "Burkitt's Lymphoma and Malaria," *International Journal of Cancer* 6, no. 1 (1970): 1-9, quotation on 8.
[54] National Library of Medicine (NLM) "IARC Internal Technical Report 71/001".
[55] NLM, "IARC Internal Technical Report 71/001".

conclusion was that they would observe that EBV infection preceded development of tumors after a relatively short latent period of 12-24 months. The third possible outcome would be that tumors would only develop in individuals with "long and heavy exposure with EBV."[56] The study authors acknowledged that, in fact, there was a fourth possible outcome that the proposed study would not be able to distinguish from the null hypothesis—that the relationship between EBV and Burkitt's lymphoma was "more complicated than in previous hypothesis, involving for example a long and variable latent period, or a delicate relationship between EBV infection and possible co-factors."[57] Possible co-factors, such as malaria, would distort the appearance of a relationship between EBV antibody titers and tumor formation.

Not everyone in the meeting was content to ignore the possibility of co-factors. Professor J. W. Kibukamusoke, Professor of Internal Medicine and Chairman of the Uganda Research Council from Makerere Medical School observed that the ubiquitous EBV could not possibly be solely responsible for the extremely rare BL. As Williams and his coauthors had remarked on the same observation in 1969, "Any hypothesis relating EBV infection to the development of Burkitt's lymphoma must therefore be slightly tortuous."[58]

The attendees agreed that malaria parasite load data would be valuable and it was suggested that it might be possible to collect malaria slides from each child, later studying the slides from lymphoma cases when they were detected. Still, not everyone believed that such a study would produce conclusive results even if the link between malaria and Burkitt's was real. As early as 1970, Gregory O'Conor, in an article outlining the mechanisms by which malaria might, with co-factors such as EBV infection, lead to BL, expressed pessimism about the likelihood that a field study could confirm the association:

> Although it may be more appropriate, it is usually extremely difficult to test hypotheses in the field and with human populations. The association of holoendemic malaria with the distribution of Burkitt's tumor is so well

[56] NLM, "IARC Internal Technical Report 71/001".
[57] NLM, "IARC Internal Technical Report 71/001".
[58] Williams, Spit, and Pike, "Further Evidence," 242.

> documented that there is little more one can do beyond lending full support to malaria eradication campaigns and wait for the expected disappearance of Burkitt's tumor.[59]

As had been the case with yellow fever, the capacity of field-based investigations to provide definitive proof of causal links was in doubt.

Ultimately the group decided to move forward with the study in West Nile and to collect additional information that might elucidate the role of possible co-factors, including malaria. They speculated that, in the event of a more complex etiology, that information might be informative or that they could help to untangle the mechanisms by which EBV infection led to tumor growth. These data included medical histories (including histories of malaria), information on living conditions, diet, agricultural production, rainfall, and geographic features of the homes in which the children lived (including proximity to rivers, sources of drinking water, etc.).[60] Because such data couldn't be reliably collected retrospectively for Burkitt's lymphoma patients, the project would visit each child enrolled in the cohort several times a year.

The final West Nile project was really a set of closely related studies, each taking a slightly different approach to the question of BL etiology. The main study, involving the largest number of subjects, was the prospective cohort study, which aimed to demonstrate the chronology of EBV infection with regard to BL diagnosis. The IARC proposal outlined the protocol for building that cohort:

> All children under the age of six in the West Nile Counties of Maracham, Terego and Aringa (possibly excepting the sparsely populated sub-county of Kei) will be registered. Children in this area born within five years after the initial registration will be registered. These children will be visited every six months for five years for treatment and vaccinating programmes, and at each visit an account will be obtained of significant medical events since the last visit. All children will be bled as soon as possible after reaching age two, and rebled two or three years later. The continuity of medical surveillance, together with a well-informed

[59] G. T. O'Conor, "Persistent immunologic stimulation as a factor in oncogenesis, with special reference to Burkitt's tumor," *The American Journal of Medicine* 48, no. 4 (1970): 279-285, quotation on 284.
[60] NLM, "IARC Internal Technical Report 71/001"; EAVRI Annual Report for 1972. Several years earlier, Lameck Hazel Goma, an entomologist with the EAVRI, had observed that children with Burkitt's lymphoma in West Nile and Buganda tended to live in places near permanent or semi-permanent surface water in which mosquitos could breed. Lameck Hazel Goma, "The Environmental Background to Cases of Burkitt's Lymphoma Syndrome in Uganda," *East African Medical Journal* 42, no. 2 (1965): 62-66.

> medical infrastructure, will ensure the reporting, or discovery, of the [Burkitt's lymphoma] cases.[61]

A stable and cooperative study population was absolutely critical to such a project.

In addition, a smaller number of children were enrolled in a longitudinal sub-study of several clusters of households, which were bled at 6-month intervals. The longitudinal sub-study was intended "to identify environmental factors (virus infection, malaria infection, malnutrition or pollution of drinking water) involved in the causation of BL" and to measure "The seasonal variation of these factors and changes over time" over a three-year period.[62] The EAVRI's particular strength in entomology also came into play with the collection and identification of mosquitos in both tumor-positive and tumor-negative areas of West Nile. The collected mosquitos known to bite man were preserved and sent to an unnamed laboratory for attempts to isolate EBV nucleic acids.[63]

Kafuko and Williams provided local expertise to the group of international experts. For example, four field teams were proposed to cover 35,000 children in the first two years, but Williams convinced the group that five teams would be needed to account for time and effort lost to staff illness and leave. Kafuko also weighed in on the composition of the team, suggesting a medical assistant, nursing assistance, and trained medical staff for bleeding and treatment, all under the direction of a medical officer. The attendees agreed, based on the outcome of the preliminary bleeding project, that identifying children for rebleeding "would be no more than a minor issue."[64] But Williams objected when he heard that the project protocols would call for 20 milliliters from thousands of young children in the district. As he wrote, "Knowing the reluctance of West Nilers to the taking of blood, I jibbed at this and said they would only succeed if they

[61] NLM, "IARC Technical Report 71/001," Annex 3, 7.
[62] EAVRI Annual Report for 1975, 37.
[63] EAVRI Annual Report for 1975, 38.
[64] NLM, "IARC Technical Report 71/001."

took just 3 mls. I refused to budge on this and it was eventually agreed."[65] On this and similar matters, the international experts seemed ready to defer to the local experts.

Thus, the EAVRI had embarked on an ambitious project to subject children and their families in portions of the West Nile District to an extraordinary degree of surveillance in order to produce valuable data that couldn't be generated anywhere else. In August of 1971 the Institute received 42,245 USD from the NCI and the IARC for the BL work in West Nile District.[66] The funds were received too late in the year to begin enrollment for the planned 35,000-member cohort, but Kafuko reported that during the final quarter of the year the Institute had recruited and begun training the staff that would be dedicated to the West Nile District work.[67]

Implementation and Insecurity

The coup by Idi Amin, notoriously a native of West Nile, in January of 1971 must have shaken the confidence of some of the study's architects. Certainly over the next few years both Ugandans and expatriates had reason to avoid coming to the attention of the General or his regime. Ted Williams recalled that in the 1970s "there were spies watching us all the time. We learned to be careful talking with anyone in Arua town, because there would often be some byestander [sic] listening to our conversation."[68] But, if anything, Amin's association with West Nile seems to have enhanced the ability of the researchers to continue the project during the ensuing years of progressively declining security and governance and decreasing tolerance of foreign nationals. During the expulsion of Asians from Uganda in 1972, IARC researcher Dr. Dharm Beri reportedly posted a notice that he was working for the WHO and claimed that he encountered no harassment.[69] Dr. George Olwitt, who joined the Arua team as a medical officer several years

[65] Williams autobiography, WTI/DPB/D/12, 101.
[66] EAVRI Annual Report for 1971, 3.
[67] EAVRI Annual Report for 1971, 63.
[68] Williams, "It Came to Pass," 84.
[69] "Asian scientist works on in Uganda," *New Scientist* 72, 1030 (1976): 603.According to this report, Beri was a former colonial medical officer and once treated Amin as a patient. Beri was quoted in the magazine as saying, "There

later, was a member of the Lango tribe, one of the groups targeted for state terrorism under Amin, and he recalled feeling vulnerable in West Nile, too vulnerable in fact to let his family join him there. But he believed that his relationship to the project, and particularly to their French collaborators who would periodically visit, offered him protection.[70] The connection between Amin's home district and a team of researchers from prestigious international agencies may have been a source of pride for the General. In any case, the EAVRI's records are almost completely silent on the question of politics or security in this period.

In September 1974 the project wrapped up the primary sera collection. Altogether they collected 38,161 specimens from children between 0 and 5 years old, the original study population. They also expanded sera collection to 3,179 children between 6 and 8 years old because some cases were observed in that age range as well. Finally they collected 3,895 samples from mothers of infants.[71] With the bulk of the enrollment and collection work complete, the project laid off nearly half of its staff and concentrated on detection of BL cases and follow up of known patients in the District.[72]

The case detection work was conducted under the supervision of a Medical Officer and a Medical Consultant (Williams). The case detection team visited hospitals and clinics in the study catchment area "to encourage workers in these places to keep a vigilant search for BL cases" and to report any suspected cases to the Project Office for further investigation.[73] They ordered and distributed throughout the district calendars featuring "pictures showing patients before and after treatment preferably those from West Nile who are still alive."[74] They also introduced a system of

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are many Asians coming back to run sugar mills and so on. They are welcome." It seems likely that he was being politic. The following spring he left the country.

[70] Olwitt OH.

[71] EAVRI Annual Report for 1974, 29.

[72] EAVRI Annual Report for 1974, 29.

[73] EAVRI Annual Report for 1974, 30.

[74] East African Virus Research Institute Quarterly Report No. 28, April 9, 1975, UVRIA, G.C. 20 "West Nile Cancer Project."

"pink chits" in 1974, through which medical workers and chiefs could notify the case detection team of any children that might have BL.[75]

Between 1966 and 1973, a total of 133 cases had been reported in the West Nile District, an average of 16.6 per year ranging from 10 in 1968 to 22 in 1973.[76] But in 1974 only six cases were detected.[77] The team speculated that it was possible that the implementation of a BCG campaign in the district in 1972 which reached between 60 and 70 percent of children up to 10 years old had somehow led to the decline in BL cases. The existence of unrelated public health programs like this one highlight the limits of the researchers' ability to control their experimental zone. They could not even control all of the medical interventions that study participants received.[78] Another possible explanation was that in 1972 the team had begun dispensing the antimalarial chloroquine during blood collection sessions in the study area and malaria suppression may have been related to the dearth of BL cases. It is unclear why the researchers decided to introduce a variable related to one of the factors whose impact they were attempting to measure. It is possible that malaria treatment was one of the ways that they maximized participation in the study. The team indicated its intention to "vigorously" study these possibilities in 1975.[79] But they also noted that it was entirely possible the low number of cases in 1974 was merely an anomaly, especially since it had already been established that cases often occurred in "clusters in 'time and space'".[80] The good news was that three of those cases were in children who had been bled as part of the prospective cohort study, permitting analysis of the longitudinal relationship between EBV antibodies and tumor development.[81] By the following year the number of detected cases had returned to the expected level and the team was please to

[75] EAVRI Annual Report for 1975, 37.
[76] EAVRI Annual Report for 1974, 30.
[77] EAVRI Annual Report for 1974, 30.
[78] That does appear to have been something they were aware of in the planning phases.
[79] EAVRI Annual Report for 1974, 30.
[80] EAVRI Annual Report for 1974, 30.
[81] EAVRI Annual Report for 1974, 30.

note that 5 of the 21 cases diagnosed in 1975 had been included in the preliminary collection of blood samples, permitting analysis of change in serum status.[82]

Also in 1974, the Arua-based team continued studies on the geographic distribution of malaria parasitemia in sample populations of children up to 5 years old enrolled in the in-depth study. The data collected did not seem to indicate any geographic patterns in the distribution of malaria prevalence or severity. It did, however, suggest that while young boys and girls were infected at approximately equal rates, boys had significantly higher parasite counts than girls. Regarding this observation, the team reported:

> The reason for this phenomenon is not clear, but it seems to be a persistent observation among the young children in the West Nile. It may be explained by the different behavioural patterns of boys and girls. The boys tend to follow domestic animals to pastures at times (morning and evening) when anopheles mosquitoes are very active and have a higher risk of exposure to mosquito bites than girls who tend to stay with the mother near smoking fires as she cooks meals for the family.[83]

While researchers in the U.K. or the U.S. might be able to do the laboratory studies that related antibody levels and carcinogenesis, local knowledge of peoples' behavior was necessary to interpret some of the findings. The role of the EAVRI as the local partner in this international collaboration was to provide that knowledge.

The beginning of the end

All sections of the Institute began to feel the strain of the political situation by the late 1970s. During this period, the Institute was attempting to maintain a diverse set of virus investigations in addition to its work on Burkitt's lymphoma. Its annual reports continued to summarize research on yellow fever, other arboviruses, and routine virus identification work for Mulago and a variety of clinics. The entomology department continued to conduct a variety of projects on the various vectors and their behavior. But staffing the various departments was always challenging and got worse after the collapse of the EAC in 1977. For the second half of that year the Institute operated

[82] EAVRI Annual Report for 1975, 37.
[83] EAVRI Annual Report for 1974, 32.

without a budget. Some employees of the Institute and the West Nile project, employed under EAC agreements, left when the Institute was transferred to the Uganda Public Service.[84] Others left the country because they feared for their safety. Peter Tukei, who had assumed the directorship of the Institute when Kafuko retired in 1973, fled the country in February 1977.[85] Beri, the IARC researcher in charge of the Arua station, left the country in May.[86] Two other members of the EAVRI's scientific staff failed to return from training programs in the United Kingdom and the United States.[87] Louis Mukwaya, an entomologist who had worked at the EAVRI since 1965 and who took over as Acting Director of the Institute after Tukei's departure, wrote in the 1977 Annual Report, "The period under review is perhaps the most difficult the Institute has gone through in its history."[88]

Amidst all of this instability, the project staff relied on their relationships with local officials and communities in West Nile to carry on, though preparations were being made to conclude the project earlier than originally planned. As of September of 1978, Olwitt reported that case detection was continuing without interruption. He reported on the particulars of suspected Burkitt's lymphoma cases brought to the team's attention by a variety of informants, including patients' family members, medical officers of local mission hospitals, and local dispensaries.[89] Apparently the researchers still had the trust of at least some of the people in the district. But later that fall the situation took a dramatic turn for the worse. Reacting to widespread mutinies in his army, Amin sought to unify his troops by redirecting their dissatisfaction towards a common enemy: Tanzania.[90] Ugandan troops committed a brief but spectacularly violent

[84] George Olwitt, "Report of Burkitt's Lymphoma Project in West Nile Arua: July to September, 1978," UVRIA "Annual Report (1978) Ent/Zool". I have been unable to locate any copy of the Institute's 1978 Annual Report.
[85] Letter from D. P. Beri to Peter Tukei, March 31, 1977, UVRIA G.C. 20 "West Nile Cancer Project".
[86] EAVRI Annual Report for 1977, 9.
[87] EAVRI Annual Report for 1977, 5-8.
[88] EAVRI Annual Report for 1977, 1.
[89] George Olwitt, "Report of Burkitt's Lymphoma Project in West Nile Arua: July to September, 1978," UVRIA "Annual Report (1978) Ent/Zool". Marissa Mika has described the lengths to which investigators based at the Uganda Cancer Institute went to maintain continuity of case management for children with Burkitt's lymphoma in this period. Marissa Mika, "Research is our Resource: Surviving Politics and Experiments at an African Cancer Institute," PhD diss., University of Pennsylvania, 2015.
[90] Decker, In Idi Amin's Shadow, 150.

occupation of Tanzania's Kagera Salient to which Tanzanian forces responded by invading Uganda.[91] Together with members of the Ugandan resistance in exile, they proceeded through Masaka district, north of the Tanzanian border and on the western shore of Lake Victoria, and gradually made their way to Kampala.[92] In April 1979 they occupied Kampala and Amin fled into exile in Libya.

The consequences in West Nile were immediate and brutal. Having enjoyed some favor under Amin's regime, the West Nile region suffered a severe backlash after his overthrow. Many from the region were among the estimated 130,000 Ugandans who fled into South Sudan fleeing violence enacted on communities perceived as having supported Amin.[93] In March of 1979 both the IARC Burkitt's lymphoma team and the WHO Team for Special Studies in Virology based in Entebbe terminated their Ugandan projects and withdrew from the country, leaving behind assorted laboratory equipment but no staff.[94] Olwitt, who was one of the last EAVRI researchers in Arua at the end, called it a "get away operation." One of his last memories of the field station was seeing the road outside covered with what looked like snow—shredded records from the project.[95]

Conclusions: The Failure of a Place-Making Project

In many ways, the story of Burkitt's lymphoma research in Uganda is another instance of the expertise of African researchers and doctors who spent their careers in Africa, whose

[91] Decker, *In Idi Amin's Shadow*, 151-152.
[92] The violence and disruption of the fighting in Masaka district in this period was later invoked as one of the likely factors in the early AIDS epidemic in the region.
[93] Peter Woodward, "Uganda and southern Sudan: peripheral politics and neighbor relations," in *Uganda Now: Between Decay and Development*, ed. Holger Bert Hansen and Michael Twaddle, (London: James Currey, 1988), 224-238, 234.
[94] A. Geser, G. de-Thé, G. Lenoir, et al, "Final case reporting from the Ugandan prospective study of the relationship between EBV and Burkitt's lymphoma," *International Journal of Cancer* 29, 4 (1982): 397-400, 397; UVRI 1979 Annual Report.
[95] Olwitt interview. One of the challenges in doing the history of this project is the rather sparse collection of surviving documents. While Olwitt recalls that copies of all of the documents kept (and destroyed) in Arua were also sent periodically to Nairobi and Entebbe, I have not been able to locate very many.

contributions have largely been excluded from the official and popular histories.[96] The publication summarizing the final findings of the West Nile study didn't include a single Ugandan author, or even acknowledge the Institute or its staff.[97] Ultimately the relationship between malaria and Burkitt's lymphoma was confirmed, but not until many years after the end of the West Nile study.[98] Virologist Dorothy Crawford published a book about EBV and cancer in 2014, which has two chapters about the research projects in Uganda. She acknowledged that in the 1960s "many local African doctors and scientists were now studying Burkitt [sic] Lymphoma," but didn't name any Ugandan scientists, mention the EAVRI, or otherwise deviate from a standard account of the role of international, mainly British and American scientists in unraveling the tumor's etiology.[99] Closer attention to the primary sources, both published and unpublished, however, reveals the intimate connections between international and local researchers that permitted the research to move forward. Time and again observations made locally were given one interpretation by international scientists, then modified with local expertise, and tested using a combination of methods developed inside and outside of Uganda. In particular, the maps, which are ubiquitous in the published and archived papers produced about Burkitt's lymphoma, offer irrefutable evidence of the critical contributions of Uganda-based scientists and physicians.

One consistent thread linking the EAVRI's during it's ventures in cancer virology to its earlier research on yellow fever was the recognition that the Institute's greatest asset was its location, in "the middle of Africa… 'home' of many diseases, particularly those transmitted to man by mosquitos."[100] Facing the crisis in staffing and funding the institute at the end of 1977, Mukwaya reflected on the potential contributions the Institute could make, and the conditions that would be necessary to permit its survival:

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[96] Abena Dove Osseo-Asare, *Bitter Roots: The Search for Healing Plants in Africa* (Chicago: University of Chicago Press, 2014).
[97] Geser et al, "Final case reporting."
[98] E.M. Molyneaux et al, "Burkitt's lymphoma," *The Lancet* 379, 9822 (2012): 1234-1244.
[99] Crawford, *Cancer Virus*, 49.
[100] EAVRI Annual Report for 1977, 3.

> The future of this Institute will largely depend on the new policies and objectives. It should be remembered, however, that since its foundation this Institute has been run by somewhat independent organizations catering for national and international interests. The professional staff as a whole has always been recruited internationally. One would wish the same policy to continue. It will be a pity if one day the standards of the Institute drop and instead of being a research laboratory, this Institute becomes a diagnostic laboratory. Success of any research institution much depends on the quality of the staff, right from the top to the bottom. Perhaps caution should be exercised when drawing up the new objectives and policy of the Institute so that the strong foundation set up by our predecessors is not ruined. Starting it again would be too difficult and expensive.[101]

Throughout the 1970s, the Institute worked to establish West Nile as a place that could produce authoritative, conclusive information about cancer and its relationship to viruses. But a variety of factors combined to thwart this effort. For one thing, while the story of Burkitt's lymphoma is often illustrated with Burkitt's tumor maps and even occasionally the climate maps, the maps from West Nile are almost never cited in retrospective accounts of the Burkitt's lymphoma research. Ultimately it appears that while geography and cartography were instrumental in the initial years, by the late 1970s more powerful epistemological tools were found in the laboratories of Gertrude and Werner Henle, Anthony Epstein, and others than in the maps of the EAVRI. The observational work that the EAVRI was especially well equipped to provide was less valuable in the 1970s than it had been in the 1940s, when they were able to link it to laboratory results in their own institution.

The political crisis that overtook the project also frustrated the Institute's larger scientific place-making aspirations. Not only did it lead to the hasty and incomplete conclusion of the trial, but it forever marked West Nile as a place that was principally significant for its association with Idi Amin, his army, and their brutality. Mark Leopold, who conducted a book-length study of West Nile's image and experience in the twentieth century, suggests one reason that the West Nile studies of the EAVRI became so marginal:

> Put crudely, then, the effects of the postcolonial era among the people of West Nile were to reinforce the economic marginality of the district, established under

[101] EAVRI Annual Report for 1977, 3.

> colonial rule, and then, under Amin, to divert the human resources of the region (especially young males) into the army and thus into a freebooting, pillaging lifestyle. The strange fantasies about Amin cultivated by both international and Ugandan commentators were based on pre-existing ideas about his home district, and grew to determine how the inhabitants of the area were seen by other Ugandans […] The West Nilers became inelucatably, 'Amin's people'.[102]

This identification of West Nile was powerful enough to occlude its significance in the cancer and virus research communities.

However, the work in West Nile and the collaboration with the IARC was formative for the EAVRI, soon to be nationalized and renamed the Uganda Virus Research Institute, and many of its Ugandan scientists. Barnabas Kirya, for example, an arbovirologist and key member of the Burkitt's lymphoma research team at the EAVRI, went on to the be first Ugandan head of the department of microbiology at Makerere University and later served as the President of the Uganda Medical Association and in the diplomatic corps as Uganda's High Commissioner to the United Kingdom.[103] Germano Munube, who worked with international teams investigating Burkitt's lymphoma at the EAVRI and took advantages of training opportunities in the United States that they offered, later worked with the WHO in Nigeria and as a WHO consultant for more than 20 years. The connections made by these men and others at the Institute served to cultivate relationships between the newly Africanized Institute and partners in Europe and the United States that would enable it to survive, if not prosper, during the uncertain years of Amin's presidency and the subsequent civil war. Thus, when Uganda's AIDS epidemic emerged in the late 1980s, the Institute was poised to take a leading role.

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[102] Mark Leopold, *Inside West Nile: Violence, History, and Representation on an African Frontier* (Oxford: James Currey, 2005,) 66.
[103] Joel Ogwang, "Uganda's top expert on insect viruses," *New Vision* August 7, 2012, http://www.newvision.co.ug/new_vision/news/1304879/uganda-expert-insect-viruses, accessed November 29, 2016.

Figures

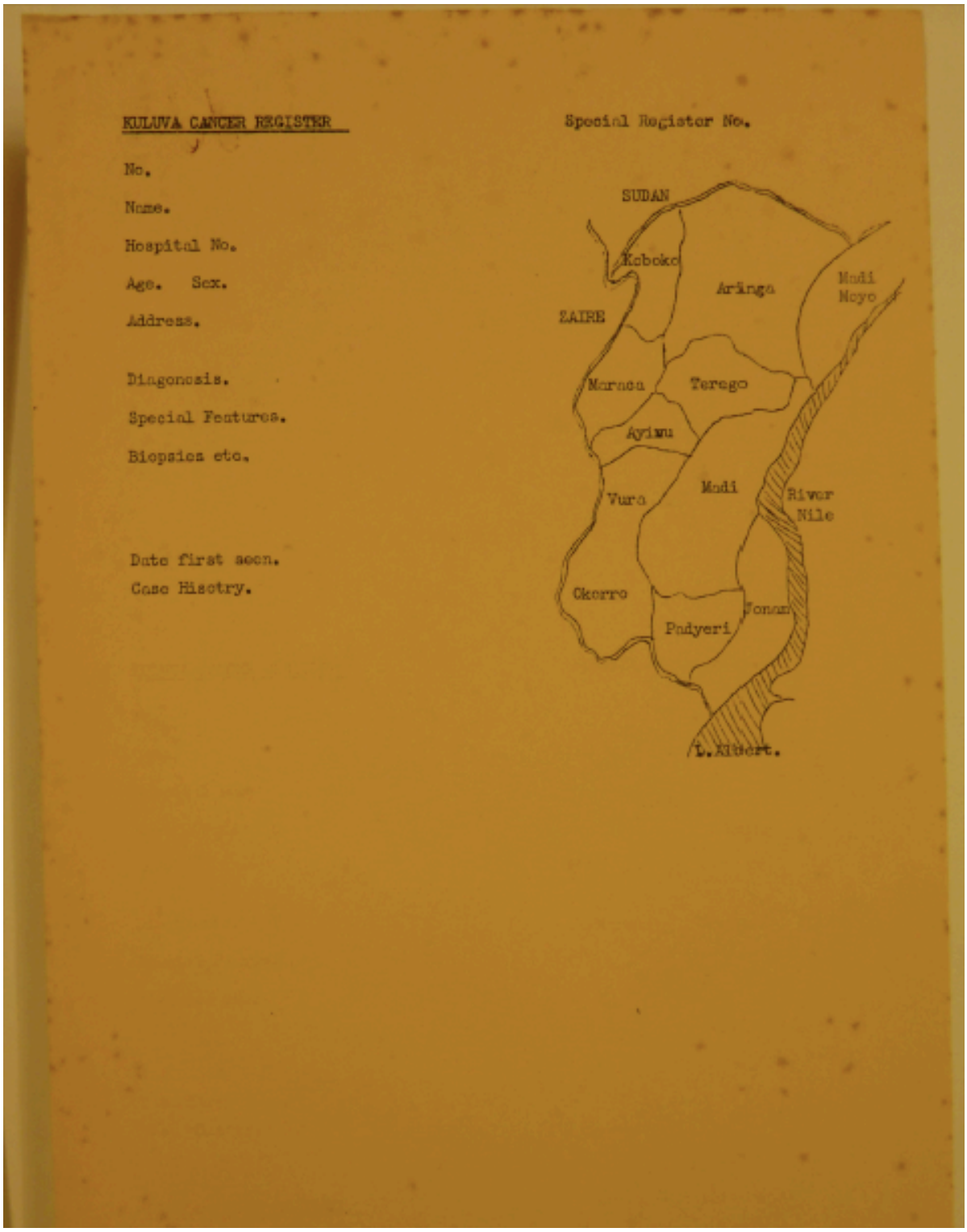

Figure 2: A blank page from the Kuluva Cancer Registry maintained by Edward H. Williams. Courtesy of the Wellcome Library.

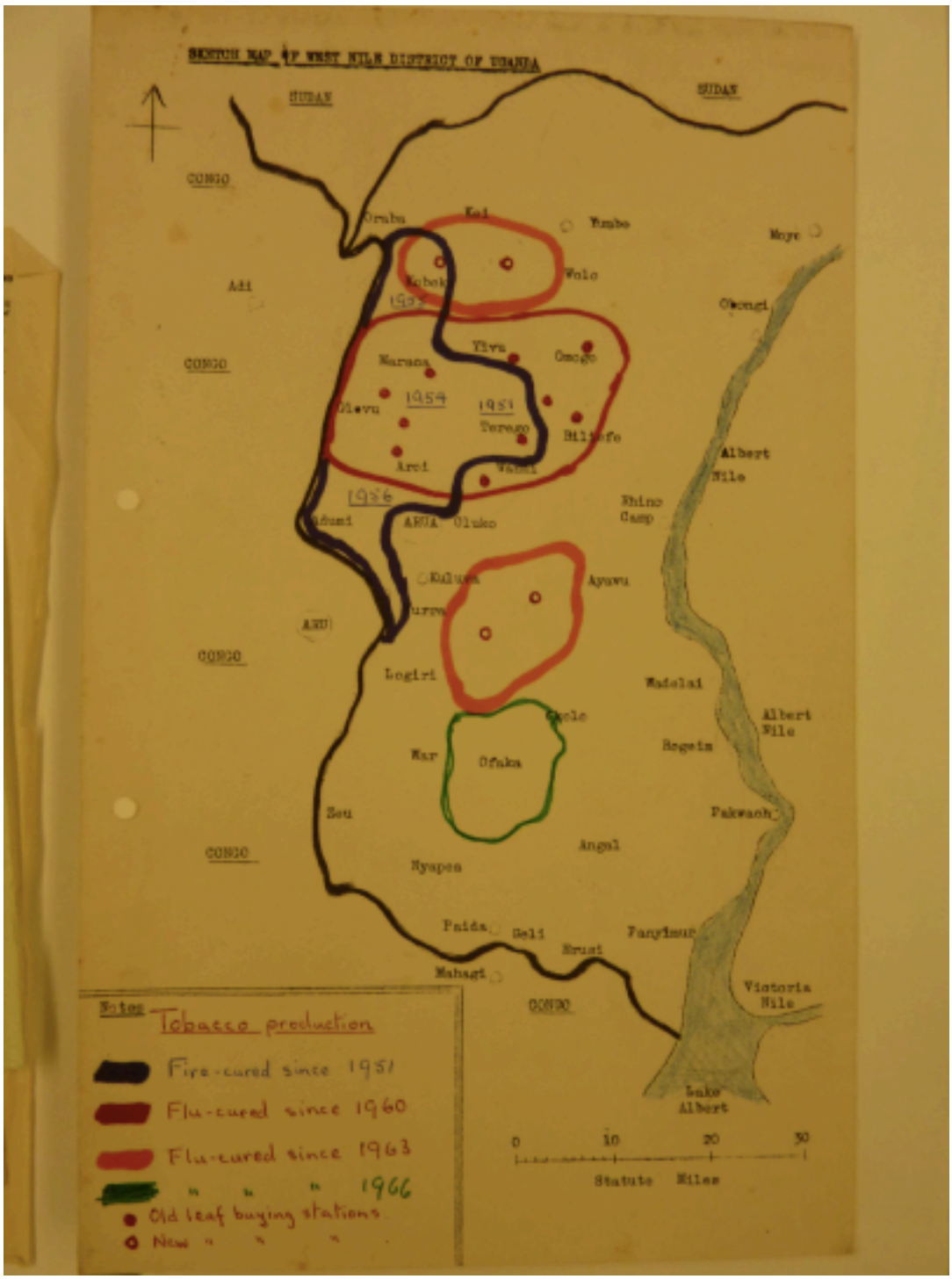

Figure 2: A map of West Nile District by Edward Williams showing the locations of tobacco production, one of the factors he suspected could be linked to cancer cases. Courtesy of the Wellcome Library.

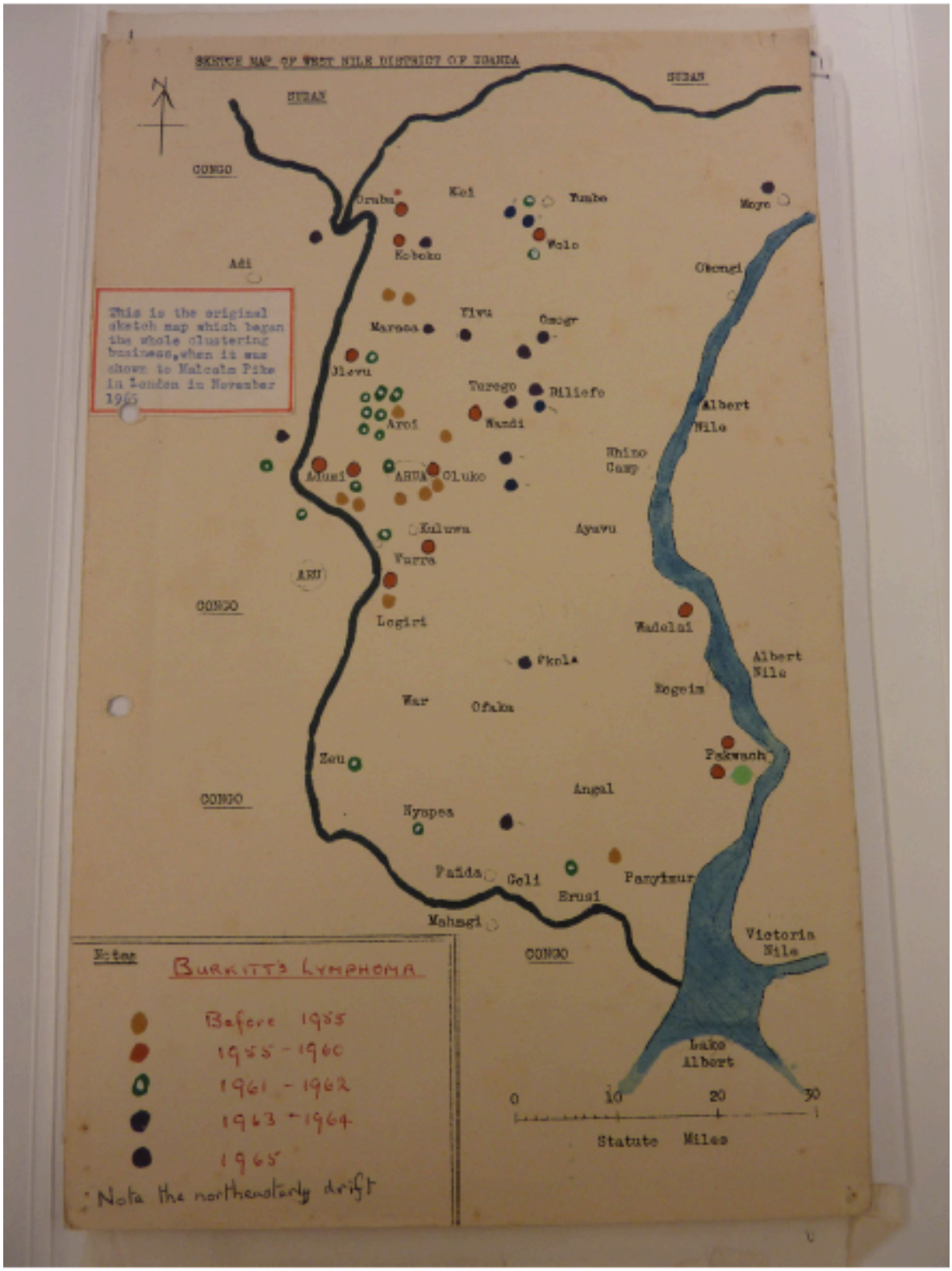

Figure 3: A map of West Nile District by Edward Williams showing the "drift" of Burkitt's lymphoma cases. Courtesy of the Wellcome Library.

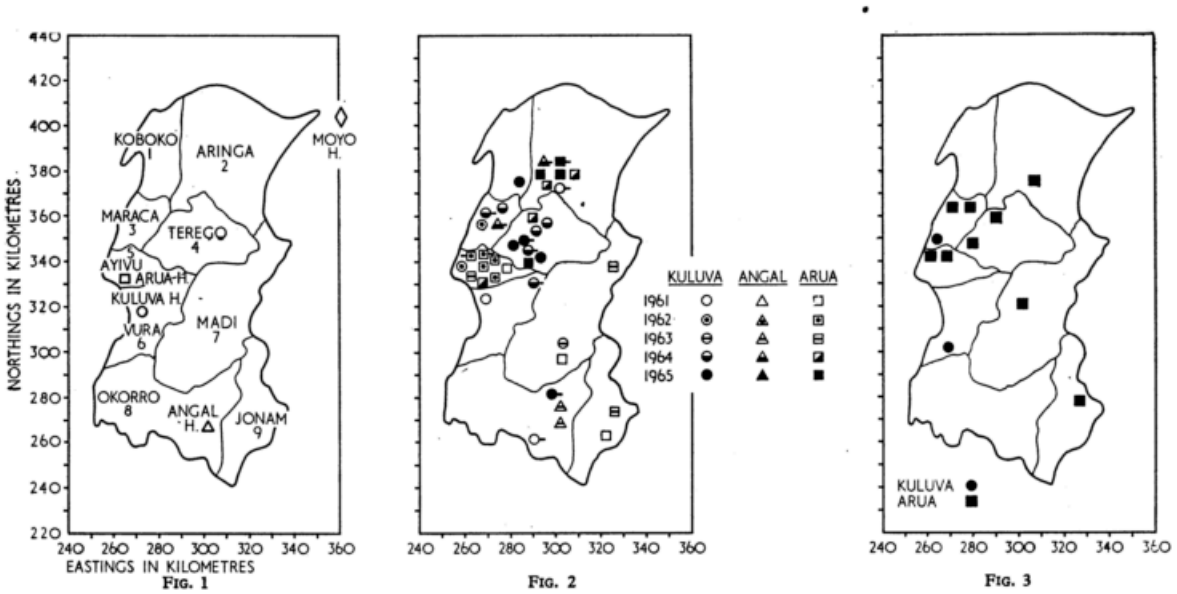

FIG. 1 FIG. 2 FIG. 3

FIG. 1.—Counties (with reference numbers) and hospitals of West Nile District. FIG. 2: All known and suspected cases of Burkitt's lymphoma, with known addresses in West Nile District and with dates of onset between 1 January 1961 and 31 December 1965. A tail attached to the symbols signifies a non-microscopically proved case. FIG. 3: All known and suspected cases of Burkitt's lymphoma in West Nile District, with dates of onset between 1 January 1966 and 31 September 1966.

Figure 4: Reproduced, with permission, from Malcolm Pike, Edward Williams, and Dennis Wright, Burkitt's Tumour in the West Nile District of Uganda, 1961-5," *British Medical Journal* 2, no. 5549 (1967): 396.

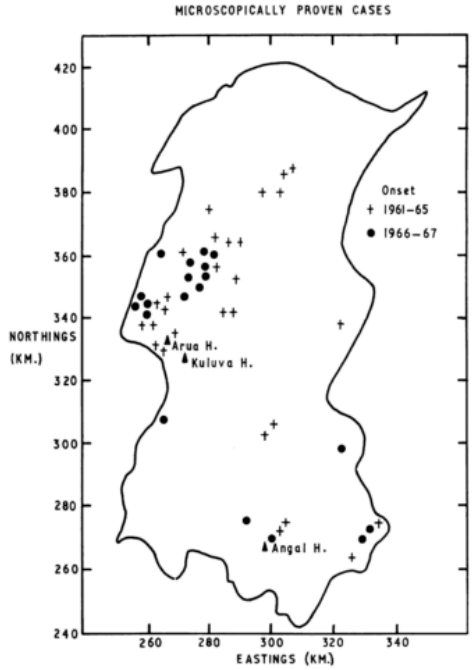

FIG. 2a.—All microscopically proven cases of Burkitt's lymphoma with known addresses in West Nile District and with dates of onset between January 1, 1961, and December 31, 1967.

Figure 5: Reproduced, with permission, from Edward Williams, P. Spit, and Malcolm Pike, "Further Evidence of Space-Time Clustering of Burkitt's Lymphoma Patients in the West Nile District of Uganda," *British Journal of Cancer* 23, no. 2 (1969): 238.

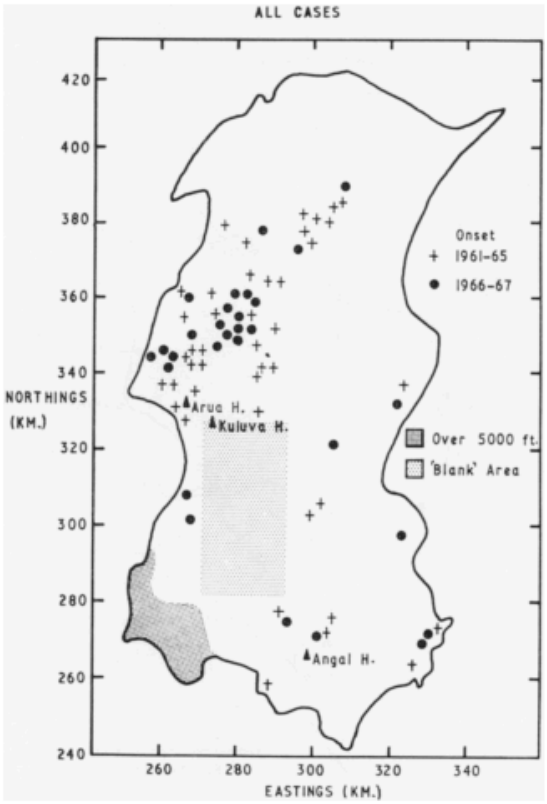

FIG. 2b.—All known and suspected cases of Burkitt's lymphoma with known addresses in West Nile District and with dates of onset between January 1, 1961, and December 31, 1967. Heavily shaded area is that part of the district over 5000 feet in altitude. Lightly shaded area is the " blank " area described in the text.

Figure 6: Reproduced, with permission, from Edward Williams, P. Spit, and Malcolm Pike, "Further Evidence of Space-Time Clustering of Burkitt's Lymphoma Patients in the West Nile District of Uganda," *British Journal of Cancer* 23, no. 2 (1969): 239.

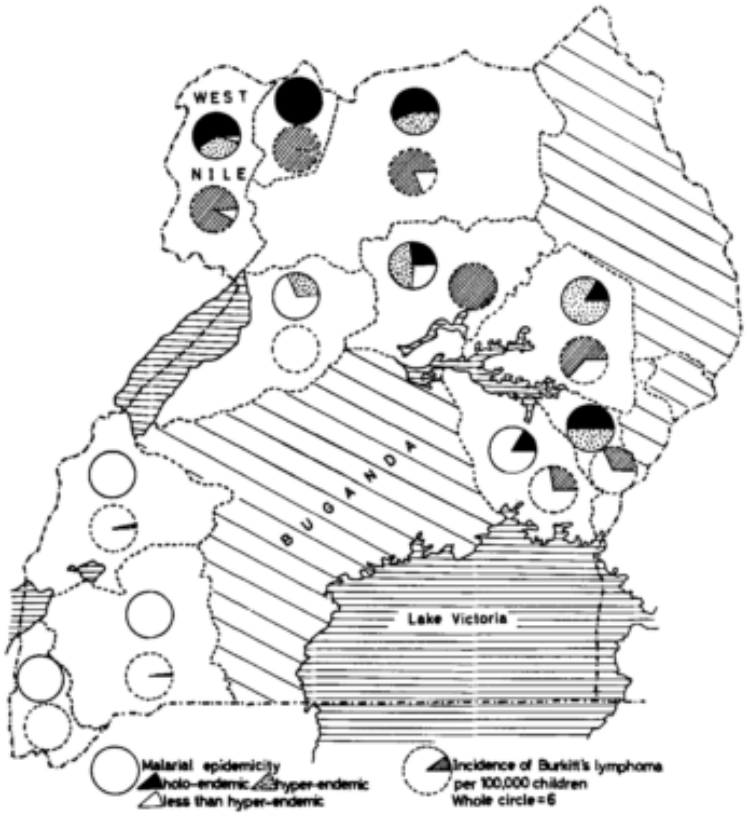

FIGURE 3

Degree of malarial endemicity in different regions of Uganda compared with the incidence of BL. Insufficient information is available from Buganda and from the extreme East (diagonal hatching). The population in the north-east is nomadic and it is not yet possible to relate location of patients to malaria.

Erratum: In the key to symbols "Malarial epidemicity" should read "Malarial endemicity".

Figure 7: Reproduced, with permission, from George Kafuko and Denis Burkitt, "Burkitt's Lymphoma and Malaria," *International Journal of Cancer* 6, no. 1 (1970): 5.

Chapter Five
Putting Rakai on the Map: The Rakai Project and the Production of a Rural African AIDS Epidemic, 1986-1994

"Rakai District was put on the map because of all the deaths"-Tropical Fish: Tales from Entebbe by Doreen Baingana[1]

Introduction

For most people outside of Uganda, Rakai, if it means anything at all, is synonymous with AIDS research. Among AIDS researchers today, Rakai is a well-known location—home of one of the longest-running cohorts for HIV research in the world. The Rakai Project (more recently renamed the Rakai Health Sciences Program or RHSP) has produced hundreds of articles, pioneered numerous preventive and treatment interventions, and launched the careers of dozens of Ugandan investigators. As one of the earliest and most influential programs dedicated to the study of AIDS in rural Africa and the evaluation of interventions to prevent and treat the disease, the Rakai Project has had an enormous impact on global understandings of HIV and the AIDS pandemic. While its place in the universe of AIDS-related trials in sub-Saharan Africa is well-established, historical accounts of the AIDS epidemic in Africa have not paid much attention to the particulars of the Project's establishment or to the crucial role of Ugandan physicians and researchers in its genesis and operation. The early history of the Rakai Project demonstrates that these individuals, and the larger African medical community, were instrumental in defining what we have come to know as the African AIDS epidemic and much of the basic science on HIV transmission, the natural history of HIV infection, and the efficacy of various techniques and tools for preventing and treating the disease. But Ugandan scientists were as susceptible to prevailing ideas about the peculiarities of African sexuality and so-called risk behaviors as their European and American colleagues. Assumptions dating back to the colonial period about African sexual promiscuity, the risks of urbanization, and the absence of indigenous homosexual behaviors all shaped the

[1] Doreen Baingana, *Tropical Fish: Tales From Entebbe* (New York: Harlem Moon, Broadway Books, 2006), 84.

questions Ugandan scientists asked about AIDS in Rakai and the types of answers they were prepared to find.

The early history of the Rakai Project sheds light on the ways that early knowledge about the African AIDS epidemic was created and consolidated. On the one hand, it highlights the foundational contributions of Ugandan scientists in the design, implementation, and interpretation of an influential international research program. On the other hand, it demonstrates that Ugandan scientists were not immune to the stereotypes and prejudices about African sexuality and its relationship to HIV risk that were prevalent among social scientists and epidemiologists. These preconceived ideas, and the social and political environment of late 1980s and early 1990s Uganda, led the Rakai Project scientists to privilege certain forms of investigation, like over-sampling roadside communities, while foreclosing others, like questions about homosexual behavior and iatrogenic transmission. These ideas were embedded in the design of the Rakai Project an its study communities and reproduced in their results. Those results were influential enough to frame virtually all subsequent understandings of AIDS in rural sub-Saharan Africa.

This chapter will begin by discussing the discovery of the AIDS epidemic in the Rakai District of Uganda in the 1980s, a discovery led by Ugandan physicians and health workers in close collaboration with a small group of expatriate physicians and researchers. It will describe how the establishment of the Rakai Project was contingent on the cooperation of a number of agencies and individuals in Uganda and the United States and especially on the history of Uganda as a center of international virus research activities. In particular, the existence of the Uganda Virus Research Institute (UVRI), even in a depleted condition, provided a crucial framework, politically and materially, for the new research program.

Sounding the Alarm: Slim Disease in Rakai

In 1983, Joseph Ssembatya, a young health inspector from Kalisizo, Rakai District in Uganda, was going about his routine work promoting hygiene and disease prevention in Kakuuto, a small

trading village near Lake Victoria not far from the border with Tanzania when he began to observe something new taking place among the residents.[2] As he recalled some 30 years later, "[people] came to my office and said, 'Oh, *musawo*[3], look. People in that village, we've had so many people dying.'" They described people dying after having severe diarrhea and weight loss. Ssembatya visited the villages where the disease was being reported and at first thought he was seeing a cholera outbreak, but didn't think the patients he saw looked like they had cholera. His next thought was typhoid. He rode his bicycle from Kyeebe to Kalisizo (approximately 60 km) to discuss the situation with his boss, District Medical Officer Dr. Anthony Lwebaga. As Ssembatya was aware, declaring an epidemic of either cholera or tyhoid was fraught with consequences, as both were notifiable diseases for the World Health Organization. Lwebaga traveled to Kasensero, one of the villages with a large number of sick and dying individuals located on the shore of Lake Victoria and a center of fishing and illegal trade (*magendo*) with Tanzania. Lwebaga didn't believe the epidemic was either cholera or typhoid but agreed that something serious was going on and reported to the Ministry of Health. Ssembatya recalled Lwebaga making a vague reference to "funny viruses" he had studied in medical school that might be similar to what they were seeing in Rakai. The Ministry of Health directed him to send specimens to UVRI, per standard practice.[4] The Ministry of Health next reached out to the Makerere School of Public Health to conduct further investigations, but before the nascent investigation, planned for 1984, could

[2] The dates for the earliest events in this narrative are difficult to establish definitely. Ssembatya began work in Rakai in 1981 and recalls these events as taking place only a year or two after he started the job. Wilson Carswell's unpublished memoir dates Lwebaga's notification of the Ministry of Health to November 1984 (chapter 2 page 2). Both Serwadda and Carswell recall that the newspaper article highlighting the situation in Rakai came out in December 1984. But it isn't until the Ministry of Health investigation in early 1985 that there is enough documentation to state dates with much certainty. This part of the story of AIDS in Uganda has been told and retold many times and most of the principal participants have recounted their version of events numerous times. John Kinsman has written a thorough account of the chain of events that led from the first observations of atypical KS patients in Kampala to the gradual recognition that AIDS was already ravaging Rakai. John Kinsman, *AIDS Policy in Uganda: Evidence, Ideology, and the Making of an African Success Story* (Palgrave MacMillan, 2010).

[3] *Omusawo* is the word for doctor in Luganda, the language spoken most widely in Rakai. Ssembatya was not a medical doctor, but was afforded the honorific title as the local embodiment of the state health apparatus. Individuals associated with medical research projects in the district, regardless of their formal qualifications, are typically addressed as *Musawo* by people living in Rakai.

[4] Joseph Ssembatya Oral History (OH).

proceed all work was halted with the rise in hostilities that preceded the Museveni administration.[5]

In the meantime, physicians at Mulago Hospital and the Uganda Cancer Institute (UCI) in Kampala were also noticing what appeared to be a new epidemic. Young physician David Serwadda, a clinical medical research officer at the UCI was among the first to connect Rakai to the HIV/AIDS epidemic. As a young student, Serwadda had excelled in the sciences and was guided towards a career in medicine.[6] He graduated from the Makerere University Medical School in 1982 and entered the profession at a time when many of the Ugandan physicians who had risen to senior positions after expatriates fled the Amin regime were themselves leaving the country. Serwadda estimates that approximately half of his graduating class left Uganda and didn't return.[7] He completed his internship in obstetrics and gynecology at Nsambya General Hospital in Kampala in 1983 and joined the staff of the UCI as a clinical medical research officer.

As Serwadda tells it, he was part of a journal club at the UCI wherein physicians discussed articles describing the outbreak of Kaposi's sarcoma (KS) among homosexual white men in the United States. Serwadda was familiar with KS as a highly treatable tumor mostly affecting women—nothing like the persistent, aggressive clinical presentation described in the gay men. Serwadda connected with Wilson Carswell, a surgeon at Mulago, and Anne Bayley, a surgeon working in Zambia who had been studying the connection between atypical KS and the virus then called HTLV-III.[8] At their suggestion, Serwadda collected serum from about a dozen of the patients with KS at the UCI and sent them for analysis to their colleague Robert Downing, then at Porton Downs in the United Kingdom. Upon receiving word that four of the patients had

[5] Ssembatya OH.
[6] David Serwadda OH.
[7] Serwadda OH.
[8] Robert G. Downing, R.P. Eglin, Anne C. Bayley, "African Kaposi's Sarcoma and AIDS," Lancet 323, no. 8375 (1984): 478-480; Anne C. Bayley, R. Cheingsong-Popov, A. G. Dalgleish, et al, "HTLV-III Serology Distinguishes Atypical and Endemic Kaposi's Sarcoma in Africa," *Lancet* 325, no. 8525 (1985): 359-361.

tested positive for HTLV-III, Serwadda was astonished. All four of the positive patients were from Rakai.[9]

The serology results were not enough to convince most people in Uganda that the mysterious disease affecting gay white men in San Francisco and New York had anything to do with cancer patients from Rakai. As Serwadda described his experience as a junior medical officer presenting the results at a meeting of the Uganda Medical Association,

> Everybody was looking at me like, what am I smoking? In everybody's mind, this was a disease among white homosexual[s]. The common question was—we don't have homosexuals here. This disease couldn't be here. It's just been discovered. How come it is here? The third one was, you're such a young doctor; I think you don't know how to make your diagnosis. You may be mistaking it for something else. In and among the medical colleagues, it was a denial—I mean disbelief.[10]

It was an article in the newspaper the *Star* in December 1984 that finally galvanized a public response to the growing epidemic in Rakai.[11]

Another young doctor, Nelson Sewankambo, had also noticed an unusual pattern of illnesses on the Mulago Hospital medical wards. The son of a member of the board of medical technologists at Mulago Hospital, he decided to study medicine while in secondary school.[12] He graduated from Makerere Medical School in 1976 and, after serving his internship year in Kampala, Sewankambo was posted to a small rural hospital in Kabale, near the southwestern border with Rwanda.[13] In Kabale, Sewankambo got to practice a wide range of medical and surgical skills, and became very fond of his rural community of patients and colleagues. After 18 months, however, he decided that he needed to return to Kampala if he wanted to accomplish his goals of further professional development and specialization. So he returned to Makerere and

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[9] Serwadda OH. David Serwadda, Wilson Carswell, W. Ayuko, et al, "Further experience with Kaposi's sarcoma in Uganda," *British Journal of Cancer* 53 (1986): 497-500.
[10] Serwadda OH.
[11] Kinsman quotes the article at length. Serwadda mentioned it, but couldn't remember the title, in his interview with me. I have not been able to find a copy of the article itself though it is frequently referenced in accounts of the early epidemic. "Mysterious Disease Kills 100 People in Rakai," *Star*, December 1984.
[12] Serwadda and Sewankambo attended the same secondary school, though Sewankambo was a few years ahead of Serwadda. Sewankambo OH.
[13] Sewankambo OH.

earned the Master of Medicine degree in 1981, before taking a junior faculty position at the University.[14] Sewankambo noticed a rise in patients in the general medical wards with a particular constellation of symptoms beginning in the early 1980s:

> severe diarrhea on and off, extreme weight loss, emaciation, extreme weight loss, *severe* weight loss in most cases. Skin rash, which was fairly itchy and patients kept scratching themselves from time and you felt sorry for them when you saw them scratching themselves and recurring fevers. Fevers, fevers, fevers, high temperatures on and off. That was kind of [the] typical picture and we saw these cases and they did not conform to any particular kind of picture. They resembled a few things but were not quite the same.[15]

He discussed them with a friend from medical school, Anthony Lwebaga, the District Medical Officer in Rakai who supervised Joseph Ssembatya.[16] A coalition of Ugandan physicians was beginning to build around the need to investigate this apparently new syndrome.

The government's response to the publicity around the 1984 *Star* article was to dispatch a team of senior doctors to Rakai to investigate the reported epidemic. Serwadda, a very junior doctor, was not included in that trip, but he says that he went to see them "and told them it must be AIDS. I've seen people in the Cancer Institute who are coming with clinical signs. They never believed me." Instead, after their visit to Rakai the group concluded that what Ssembatya and Lwebaga had reported was an outbreak of typhoid.[17] Serwadda recalls being immediately skeptical about this conclusion and said "we decided to put up an expedition of our own." The people who put together the second "expedition" included Anne Bayley, who was in Kampala for the Association of East African Surgeons, Robert Downing, Wilson Carswell, Nelson Sewankambo, and Roy Mugerwa.[18] Together they traveled from Kampala to Masaka, where they set up a temporary headquarters from which they visited several health centers in Masaka and

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[14] Sewankambo OH.
[15] Sewankambo OH.
[16] Sewankambo OH.
[17] Serwadda OH. Carswell's memoir names George Kirya and Eric Kigonya, and Mathew Kakande as members of this committee. He recalls that they diagnosed one of the "slim" patients with typhoid and another with "an obscure medical condition" but were unable to reach a diagnosis on nine other patients. (Chapter 2 page 3).
[18] Serwadda OH and Carswell memoir chapter 2 page 6. Sewankambo recalls that Edward Katongole-Mbidde was also on this trip, but he is not mentioned in any of the other accounts. Sewankambo's recollection is also that Downing wasn't actually on this trip though he was involved in the planning and tested the specimens they collected afterwards. Roy Mugerwa went on to work on the AIDS/tuberculosis syndemic in partnership with Case Western Reserve University. Sewankambo OH.

Rakai District looking for patients with the characteristic wasting and other symptoms of slim disease and collecting urine, sputum, blood, and stool specimens.[19]

In June of 1985, Carswell presented the results of the expedition to Rakai and the subsequent serological findings at a meeting of surgeons in Blantyre, Malawi "to alert doctors from other African countries to the this [sic] new way in which AIDS could present in Africa, and to give some idea of the extent of the problem of slim disease."[20] And Serwadda took the lead on writing up the results for what would be a landmark *Lancet* article. The article summarized findings from the examinations of 29 patients with slim disease in Masaka and Rakai and 42 slim patients seen in Mulago (most of whom were from Masaka or Rakai). The 29 patients examined in Masaka and Rakai all tested positive for HTLV-III antibodies, as did 34 of the 42 patients tested in Kampala. In addition, the team tested 410 healthy "controls"—employees of Mulago Hospital, of whom 10% also tested positive.[21] But in the 1985 *Lancet* article that marks the beginning of the collaboration that would grow into the Rakai Project, one of the most important HIV cohort studies in Africa, the authors waffled on the question of whether slim disease was really AIDS. In the article's summary, they wrote, "Although slim disease resembles AIDS in many ways, it seems to be a new entity."[22] But in the discussion section of the article, after beginning with the statement that "slim" is "not unlike AIDS", they proceed to discuss slim as a different epidemiological form of HTLV-III, the same virus that caused AIDS elsewhere. Even this lukewarm endorsement of the connection between slim disease and AIDS caused consternation in Uganda. Serwadda says that the attitude at the Ministry of Health was that he and his colleagues were "spoilers". He recalls skepticism about the tests, the methodology, and the validity of the results reported in the *Lancet*.[23] Their work was interrupted in July when General Tito Okello deposed President Milton Obote, ruling for six months before being ousted by

[19] Carswell memoir chapter 2 page 7; Serwadda OH; Sewankambo oral history interview.
[20] Carswell memoir chapter 2 page 8.
[21] David M. Serwadda, Nelson Sewankambo, J. Wilson Carswell, et al., "Slim Disease: A New Disease in Uganda and its Association with HTLV-III Infection," *The Lancet* 326, no.8460 (1985): 849-852.
[22] Serwadda, Sewankambo, Wilson, et al., "Slim Disease," 850.
[23] Serwadda OH.

Yoweri Museveni's National Resistance Army.[24] It wasn't until Museveni took over the government that there was a real commitment on the part of the government.

The 1985 *Lancet* article gives a snapshot of the many unanswered questions outstanding about what aspects of AIDS in Africa might be uniquely or at least particularly African. The first possible mode of transmission mentioned was "heterosexual promiscuity"—"Although the subjects in our study deny overt promiscuous behaviour, their sexual behaviour is, by Western standards, heterosexually promiscuous."[25] Even the finding that some children were infected was explained away with the statement that "sexual immaturity is not a reliable indication of sexual inactivity."[26] No evidence explaining this statement is offered. The other characteristically "African" explanation for the epidemic was a mosquito transmission. The authors even invoked the example of the studies linking Burkitt's lymphoma and malaria to illustrate some of the possible explanations for slim disease: "Malaria was identified as a major risk factor for Burkitt's lymphoma because the lymphoma did not occur at altitudes where the *Anopheles* mosquito did not occur. Since all our subjects came from the lowlands, we are not able to draw conclusions about an association of HTLV-III infection with altitude or a specific type of mosquito."[27]

The 1985 article also documented some potential risk behaviors that were conspicuously absent from most subsequent publications on AIDS in Uganda, including male homosexual behavior. According to the article, the itinerant traders on whom much of the early investigations were focused "admitted to both heterosexual and homosexual casual contacts."[28] However, later papers included no mention of homosexual behavior in Uganda, instead representing Rakai as the site of a heterosexual epidemic. The foreclosing of particular lines of inquiry deemed far-fetched or simply unacceptable in Uganda happened very quickly.

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[24] Jennifer Bakwaya, "Uganda: Disbelief, then Dawning Horror: How AIDS First Came Here," *The East African*, January 20, 2003, 5-7.
[25] Serwadda, Sewankambo, Carswell, et al., "Slim Disease," 852.
[26] Serwadda, Sewankambo, Carswell, et al., "Slim Disease," 852.
[27] Serwadda, Sewankambo, Carswell, et al., "Slim Disease," 852. Ronald Gray points out that this is incorrect—Masaka and Rakai have endemic malaria. Email communication, February 24, 2017.
[28] Serwadda, Sewankambo, Carswell, et al., "Slim Disease," 852.

Establishing the Rakai Project

Convinced that there was important work to be done on the epidemiology of AIDS in Rakai, Sewankambo and Serwadda began to pursue outside funding. With some technical assistance from the CDC, they submitted a proposal to conduct a community-based survey of HIV prevalence in Rakai.[29] Serwadda recalls that the CDC seemed like a natural place to seek funding because they were already investing in AIDS research in Zaire.[30] But the CDC turned down the application for funding for a 1000-person cohort study, in part because they believed it would not be feasible. Sewankambo explained, "We were young researchers with no history of doing cohort studies and here we are proposing setting up a cohort in the communities and following up these people to see what is happening. Very ambitious study."[31] But Jonathan Kaplan of the CDC was impressed by the proposal and forwarded it to a colleague at USAID who in turn directed it to Columbia University, which had some unspent funds from a family planning grant.[32]

Maria Wawer first heard about the project that Sewankambo and Serwadda were seeking to fund while on vacation in Italy in August of 1987. As she recalled twenty years later, "My enthusiasm was underwhelming…Most of the news coming out of Uganda for the past 15 years suggested it was a burnt-out basket case."[33] Her employer, Columbia University, was contemplating using some outstanding funds from a USAID contract to support a small study proposed by "some Ugandan doctors".[34] Columbia asked Wawer to visit Uganda, meet with Serwadda and Sewankambo, and determine whether the project was feasible—whether the Ugandan doctors were capable of conducting the sort of research they were proposing and whether any research at all could be conducted in Rakai. The three of them all fondly recall their

[29] Wawer OH.
[30] Serwadda OH.
[31] Sewankambo OH.
[32] Serwadda OH.
[33] "Brief History of the Rakai Health Sciences Program (RHSP), 1987-2004, *Rakai Health Sciences Program: 20 Years of Improving Health Through Research: Status Report-February 2007*, II.
[34] "Brief History of the Rakai Health Sciences Program (RHSP), 1987-2004, *Rakai Health Sciences Program: 20 Years of Improving Health Through Research: Status Report-February 2007*, I.

initial impressions of one another—Serwadda and Sewankambo were inclined to think she was too small and young to survive the "rough and tumble of Uganda" with its road blocks, poor infrastructure, and Wawer feared the soft-spoken young Ugandans were too shy to get the project off the ground.[35] But they traveled together to Rakai where Wawer was impressed both by the scale of the need for some kind of intervention and by the seriousness of her prospective colleagues.[36]

What the recollections of all three of the original collaborators emphasize is the degree to which the Rakai Project, and early AIDS research in Uganda in general, was an African-led project from the very beginning. It was Ugandan physicians and community health workers who identified the problem, collected data to support their claims, and sought out the international resources needed to pursue a research program in hopes of finding a solution. While the intellectual and financial contributions of American partners was critical to the growth of the RP, this was not a case of American scientists driving a research program that just happened to be located in Uganda or capitalizing on Ugandan suffering to serve a foreign research agenda.

The UVRI and the Rakai Project

While most of the Ugandan principal investigators were (and still are) affiliated with Makerere University, the RP was administratively located under the umbrella of the UVRI for several reasons. At the time the project began, the UVRI had the only facilities in the country for processing HIV tests. In addition, unlike the university, the UVRI was part of the Ministry of Health with its network of outposts throughout the country. There was an existing chain of command linking local medical officers to the Ministry of Health and it was more consistent with practice to link with UVRI. In addition, the Ministry of Health (MOH) was familiar to people in the rural villages and was generally seen by people in the district as an institution that existed to

[35] Seradda OH, Wawer OH.
[36] Wawer OH.

represent their interests. The university, by contrast, had very little connection to the rural areas. As Sewankambo put it:

> We knew, as Ugandans, that if we wanted to do more research on HIV, or HTLV-III as it was known then, and out in the communities of Rakai and Masaka, we needed the backing of the Ministry of Health. There was no way we would get credibility linking up with the communities here [in Rakai] and coming from the University at Kampala as academicians...we may succeed to some extent but not to the level that potentially we could. So we thought that the Ministry of Health must be part and parcel. First of all they must give a blessing to this and as much as possible should be partners.[37]

Finally, the UVRI had experience handling large quantities of potentially pathogenic material. When the Rakai Project was starting up, the principal investigators approached the Ministry of Health to inform them of their plan to conduct additional research in the District and the Ministry of Health asked them to coordinate their specimen processing through UVRI. As Serwadda recalled, there was concern about the infectiousness of the blood specimens and it was believed that UVRI's extensive experience handling viral agents would be better suited to the project than Makerere.[38] Serwadda noted, "They didn't want us having three refrigerators and things full of these viruses here [at Makerere]."[39] As earlier chapters of the dissertation have shown, the UVRI had built and maintained a reputation for safe and significant virus research over the course of several decades.

The Rakai Project also represented a major opportunity for the UVRI. At the time the Rakai Project was established, the UVRI had suffered more than a decade of neglect. When Benon Biryahwaho joined the Institute as a medical officer in April 1980, the Institute was dominated by entomological work and needed major investments in equipment and training of personnel in order to equip it to return to work on human virology.[40] Musagara claimed that for the Institute, "Rakai was looked at like a savior…Because government had no money. So Rakai was looked at like a savior. Number one, we took over staff, which were redundant. Second, I

[37] Sewankambo OH.
[38] Serwadda OH.
[39] Serwadda OH.
[40] Benon Biryahwaho OH.

think there was some little overhead, which was given to the Institute. Then also the infrastructure—we helped improving some of the infrastructure…we rehabilitated many offices."[41] For example, one of the Rakai Project's first NIH contracts included rental of office space at UVRI including the space itself, electricity, water, and the services of a telephone receptionist, maintenance personnel, and security personnel at a rate of $5000/year in year one with 6% increases in the second and third years of the study.[42] Additionally it budgeted $1000 to upgrade the office space at UVRI with furniture and an air conditioning unit.[43] Serwadda agreed that for the director of the UVRI at the time, Dr. Sylvester Sempala, the Rakai Project represented "one way of sort of revamping activities on the [UVRI] campus."[44] Moreover, the partnership between UVRI and RP provided a model for other collaborations. "Rakai laid the foundation. So, after Rakai came, then MRC also came. Then other collaborators also came. So Rakai laid the foundation."[45] Rakai hired many of its first staff members, especially data entrants, from among people previously employed by the UVRI but whom the government could no longer afford to pay.[46] In 2002 the UVRI's acting director, Dr. Miph Musoke, echoed Musagara's claims about the significance of the Rakai Project for the Institute's future saying, "This collaboration [with RP] has brought scientific achievements to UVRI and has assisted the Institute [to] regain its international image."[47] His successor, Director Edward Katongole-Mbidde, made a similar claim five years later:

> [The Rakai Project] was among the first institutions to collaborate with the government of Uganda and the UVRI on HIV/AIDS, thus paving the way for other collaborators including the Medical Research Council (UK), Centers for Disease Control and Prevention (CDC), and International AIDS Vaccine Initiative (IAVI). This is one happy family collaborating for the good of Ugandans and the International community in general.[48]

[41] Makko Musagara OH.
[42] Sexual Networks proposal, 18.
[43] Sexual Networks proposal, 17.
[44] Serwadda OH.
[45] Musagara OH.
[46] Musagara OH.
[47] "A word form [sic] Dr. Miph Musoke, Acting Director, 2002," *Rakai Health Sciences Program Annual Report 2003-2004*, 5.
[48] "A Word From Dr Edward Katongole-Mbidde, MBChB," *Rakai Health Sciences Program Status Report—February 2007*, 1.

The Rakai Project's partnership with the UVRI was formative in making Entebbe, and Uganda more broadly, a site of intensive AIDS research and Ugandan scientists were the leaders in forming that partnership.

The First Study

Columbia University reached an agreement with the Uganda Ministry of Health and the Uganda AIDS Control Programme (ACP) in the beginning of 1988 to apply leftover USAID funds to a project entitled "Study of the Effect of Health Education on the Transmission of Human Immunodeficiency Virus Infection in the Rakai District of Uganda". At this stage the project was housed at the ACP with the UVRI, in its capacity as a WHO AIDS Reference Laboratory, conducting serology.[49]

Columbia committed to funding the study up to the amount of $167,759 and to provide technical assistance to the ACP "in the implementation and evaluation of the project."[50] The project proposal called for two phases: the first, to be funded with the USAID money, was expected to last 18 months and consisted of a baseline survey to establish "population-based data on the prevalence of HIV infection and on the population's current knowledge, attitudes and practices regarding AIDS prevention."[51] The second phase was to consist of three years of follow-up visits at 6 month intervals to "collect information with which to determine the incidence of HIV infection, risk factors for transmission, and the natural history of HIV infection among those infected" as well as to "gather KAP [knowledge, attitudes, and practices] data on the effects of AIDS prevention in the form of education and condom distribution."[52] From the very beginning of the Rakai Project, the problem of AIDS was understood to be a behavioral problem closely

[49] UP/010/95: USAID Proposal.
[50] Operations Research Proposal: Study of the Effect of Health Education on the Transmission of Human Immunodeficiency Virus Infection in the Rakai District of Uganda, UP/010/95: USAID Proposal.
[51] UP/010/95: USAID Proposal.
[52] UP/010/95: USAID Proposal.

related to the sexual practices of Ugandans and subsequent investigations reflected and reproduced this association.

The proposal gives some indication of what was known, assumed, and uncertain about the epidemic in Uganda as of the late 1980s. The proposal cited studies that found that men and women were infected at approximately the same rates and concluded that the epidemic was primarily driven by heterosexual transmission with iatrogenic transmission relatively unimportant.[53] No mention was made of homosexual behavior or transmission, though the 1985 article by Serwadda and colleagues indicated that men reported casual homosexual contacts.[54] By extrapolating from other countries, the Rakai Project team predicted that between 10 and 30 percent of asymptomatic HIV-positive individuals would develop AIDS within 5 years. Because there was very little testing and there were "no defined risk populations" in Uganda, the proposal emphasized the importance of population-wide education about behavior change for the purposes of preventing infection.

At the time that the cooperative agreement was signed, funding was only guaranteed for the first year of the project (Phase I). But the team expressed confidence that funding for Phase II would be forthcoming because of "the importance of this project and the interest it will generate."[55] The proposal also outlined what it envisioned as the greater significance of the project, both in Uganda and beyond. "The rationale for the relatively intense research activity is the marked lack of understanding of the dynamics of the HIV infection epidemic in Africa, a lack which hinders effective program development. Results of this project will provide important insights not only for Uganda, but for program development in other East African countries." Moreover, the proposal noted, the Ugandan government was already onboard with the project and indeed encouraging "rapid implementation." Given the political resistance to acknowledging the AIDS epidemic in many other countries, they pointed out "It is unlikely that such rapid indication

[53] UP/010/95: USAID Proposal.
[54] Serwadda, Sewankambo, Carswell, et al., "Slim Disease," 852.
[55] UP/010/95: USAID Proposal.

[sic—implementation?] of a major community based study will be feasible in many other African settings."[56] In this way and others, the Rakai Project team was making a case that Rakai was a uniquely appropriate site for virus research, a claim that would become more and more true as they built their research infrastructure in the following years.

The proposal also cited the results of a pilot study conducted in February 1987 to demonstrate the project's feasibility.[57] The pilot, which involved two villages and 47 study participants had yielded some insight into the challenges researchers would face as well as potential solutions. For example, they noted that "the visit would have proceeded more smoothly had village authorities been notified beforehand,"—a protocol they were meticulous about observing thenceforth. They also noted, "Interviews proceeded well when introductory information and details of medical history preceded questions concerning sexual behaviour."[58] Even such small logistical issues such as the size and type of tubes used for blood collection were tweaked in response to the experiences of the pilot study.[59]

Inventing the Community

One of the foundational tasks of the Rakai Project was inventing the community for a community-based study. Unlike clinic-based studies, which recruit participants from attendees at clinics, hospitals, and dispensaries, community-based studies aim to reach a sample of the population that is more representative of the total population at risk, without the selection bias of a clinic-based study. The dry language of the sampling strategy outlined in the initial USAID proposal belies the significance of the decisions being made about who was and was not included in the study community being built by the Rakai Project.

[56] UP/010/95: USAID Proposal.
[57] UP/010/95: USAID Proposal.
[58] UP/010/95: USAID Proposal.
[59] UP/010/95: USAID Proposal.

Before the AIDS epidemic, Rakai was a relatively obscure part of the country. Under the British Protectorate it was incorporated into the Kingdom of Buganda, which covered most of central Uganda, including the capital city of Entebbe and the seat of the Buganda kingdom in Kampala. Until the mid-1970s it was part of Masaka District.[60] By the late 1980s, it had acquired a reputation for death. Makko Musagara, born in Mityana District, west of Kampala, and educated largely in Kampala, described his surprise at finding Rakai to be occupied at all given the stories about death and destruction in the district due to war and illness: "there are so many stories about Rakai…I was surprised to find people there."[61] In 1992, when Fred Nalugoda visited Rakai for the first time after being invited to join the study team, he was cautioned by a friend who grew up in Lwamaggwa (in Rakai District) not to shake hands with anyone and warning him, "You don't have to go to Rakai. Once you go there, you're going to die."[62] His own initial observations appeared to confirm the grim warning: "driving through Rakai, it was quite terrifying. Because you would find almost every home…there is a funeral. And then, of course the people you would find, you'd find like everyone is sick…their hair was almost getting off, skin's different."[63]

The Rakai Project had to create a way to make the district manageable for the purposes of a research study. This involved grafting their particular epidemiological geographical vision onto the existing political and social geography of the district. Between April 1988 and March 1989, using the money Columbia contributed from a USAID grant, the Rakai Project investigators designed a preliminary study of the community, which would serve as a baseline for future project proposals. They based their samples on a unit of local government, the Local Council

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[60] http://www.ubos.org/onlinefiles/uploads/ubos/2009_HLG_%20Abstract_printed/Rakai%20district%20statistical%20abstract.pdf.

[61] Musagara OH.

[62] Nalugoda OH.

[63] Nalugoda OH.

(LC).[64] Museveni's government organized LCs in tiers of successively larger catchments. LCs consist of approximately 100 households in a village and the surrounding countryside. In Rakai, LCs were the latest version of a longstanding political organization that had been called different things under Buganda and English colonial rule.

In the initial USAID proposal the study was designed to include 24 clusters divided into three strata based on the size of the population and their proximity to roads. At the time of the baseline study, there were 744 RC1s in Rakai District. The first stratum, or cluster type, consisted of 5 clusters in a single town: Kalisizo, the largest settlement in Rakai district with a population of approximately 30,000. Kalisizo was designated an "urban center" though the use of scare quotes in the proposal suggests that the writers considered urban to be an overstatement.[65] This cluster was predicted to be "intermediate risk". The second stratum was the "trucking towns"—villages located along roads used by long-distance truckers. 36 of Rakai's LC1s were located along such roads and 9 of them were to be included in the study as the trading center stratum. The clusters in this stratum were expected to yield the highest prevalence of HIV infection. Finally, the clusters in the third stratum, "rural villages", were located at a distance from major roads and were assumed to be at relatively low risk of HIV infection. 10 of the District's 708 rural RC1s were to be included in the study. Already by this period, understandings of the epidemiology of

[64] Resistance committees were the smallest level of government created by the ruling National Resistance Movement political organization. Yoweri Museveni's National Resistance Army had been fighting a bush war for nearly a decade before he was declared president in 1986. His revolutionary forces had been organized into Local Resistance or Revolutionary Councils (RCs), a structure that came to assume geographic and civic dimensions in the post-civil war nation-state. Local councils, or LCs, are their peacetime incarnation, legislated in 1987 (they are often referred to in the early 1990s as RCs, but the terms are interchangeable). These built on the existing system instituted under Amin of *mayumba kumi* (literally "10 households"), small groups of households within a village, which facilitated village management. The local council system was established by legislation in 1987 and 1997. There is a useful table comparing the Buganda, colonial, and NRM organizations in Abwoli Y. Banana, Nathan D. Vogt, Joseph Bahati, et al., "Decentralized Governance and Ecological Health: Why Local Institutions Fail to Moderate Deforestation in Mpigi District of Uganda," *Scientific Research and Essay* 2, no. 10 (2007): 436. LC1 corresponds roughly to the Buganda *butongole* or the colonial village. In Rakai District there are 780 LC1s, 14 of which are in villages or towns consisting of more than one LC1. LC2s consist of several LC1s, etc.

[65] UP/010/95: USAID Proposal.

AIDS and its connection to transportation and truck drivers had convinced researchers that oversampling the communities located near roads was useful.[66]

The study was not designed to precisely determine the prevalence and incidence of HIV in subpopulations believed to be at low risk of infection, such as older adults, because logistics precluded enrollment of a large enough population to yield significant results in these subgroups. But the proposal insisted, "the samples are ample to determine the public health significance of HIV infection, and to guide the development of program strategies."[67] In order to secure funding and show the greatest results with limited resources, the RP concentrated on the segment of the population perceived to be most at risk based on pre-existing assumptions about African sexuality and modes of HIV transmission, and gave up the opportunity to measure alternative patterns of infection, such as iatrogenic or homosexual transmission.

Ultimately the baseline study included only 21 clusters: 9 of the roadside LC1s, located along the district's asphalt roads, labeled the "trading center stratum" and 12 of the rural LC1s—the "rural stratum" [Figure 1].[68] Roads were the only aspect of the physical geography of the district that mattered enough to feature on the map used in the study proposal and later in the published article summarizing the study's findings. For 10 of the clusters the Rakai Project was able to secure a complete list of all households from which an index household was randomly selected. For the rest of the clusters, a single household was "randomly selected on the basis of a grid map" and designated as the index household for the purposes of sampling.[69] Then the enumeration team, which visited each location a few weeks in advance of the survey team, made a detailed map of the index household and 39 other households "in a continuous concentric distribution" around the index household. [70] This sampling design was intended to generate a random sample of households representative of the clusters and thus of the district as well as "to

[66] UP/010/95: USAID Proposal.
[67] UP/010/95: USAID Proposal
[68] DHHS Grant Application, "HIV Dynamics and Prevention, Rakai District Uganda", 58.
[69] DHHS Grant Application, "HIV Dynamics and Prevention, Rakai District Uganda", 58.
[70] DHHS Grant Application, "HIV Dynamics and Prevention, Rakai District Uganda", 58.

facilitate mapping and the identification of the same households during subsequent survey rounds."[71] In the event that, at the time of the census was conducted and informed consent secured, members of a selected household refused to participate the next home was added.[72]

Making the distinction between rural and trading center clusters required some tacit knowledge. When I asked him if the difference would have been obvious to a stranger visiting Rakai for the first time, Stan Musgrave explained:

> Well…it [the knowledge] built up. And I'm not sure that I could have done it—I certainly couldn't have done it at the very beginning. Among other things you talked to Joseph Ssembatya, the district medical—health education officer. And the DMO [District Medical Officer]. And others who know their community very well. And as you go out it's kind of like, okay, are there none up to three shops here? Okay, that's rural. Or are there—oh there are 25 shops here. Oh, that's a trading center. Someplace in the middle, you've got to draw a line. And I don't recall an exact rule for that…Partly there are so many rural communities that as you started doing the sampling, the cases on the borderline [between rural and trading center] weren't a problem just because they weren't chosen. And, so, trading centers were pretty obvious. Places that would have a market, as I think about it, was the other bit that—people would come to there even if only once a week or something.[73]

Even Joseph Ssembatya, the health education officer on whom the team relied heavily for local knowledge, told me that the exact limits of villages were not evident to outsiders. Unlike parishes, which would be the catchment area of a church, or sub-counties, which were typically bounded by a swamp, forest, or other natural feature, in order to know where a village stopped and started you had to ask someone in the village itself with no guarantee that two individuals would provide the same answer.[74]

The decision to stratify the clusters in this way, and the decision to oversample the trading centers, was directly related to what the researchers believed they already knew about the virus and how it was transmitted in Rakai. David Serwadda explained the rationale behind stratification this way:

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[71] DHHS Grant Application, "HIV Dynamics and Prevention, Rakai District Uganda", 58.
[72] UP/010/95: USAID Proposal, 13.
[73] Musgrave OH.
[74] Ssembatya OH.

> It was clear that we needed to stratify into trading center and rural…simply because at the time we started, we knew this was a sexually-active disease just like many sexually-active diseases we know that tend to concentrate in trading centers. So if we did not stratify and sample in each stratum, the trading centers would be underrepresented.[75]

They modeled their understanding of how AIDS was probably transmitted on existing data about syphilis and gonorrhea.[76] A table of sociodemographic characteristics of the study communities by stratum published in 1991 gives some insight into the differences between the strata, though it isn't clear which characteristics were definitional and which were only observed after the stratification had been completed [Table 1].[77] Later, a third "intermediate" stratum was added, accounting for the fact that as the smallest towns grew and added things like bars and guesthouses, they would gradually acquire characteristics—namely HIV incidence and prevalence—that fell between the highest and the lowest strata.[78]

Serwadda's statement that he and his colleagues "knew this was a sexually-active disease" treats the heterosexual transmission of HIV as a given. Indeed, by the late 1980s, there was wide consensus that AIDS in Africa was a heterosexual epidemic. Ed Hooper wrote, "It is not difficult to deduce that sexual activity is the principal source of infection in the area [Kasensero], for nearly all recorded victims are either in the 16-45 'sexually active' bracket or are the young children of infected parents."[79] But the relationship between the age distribution of an infection and its mode of transmission is not that straightforward.[80] Nor are all people between 16 and 45 equally likely to be "sexually active". Hooper didn't trouble to cite evidence for the claim that (hetero)sexual activity was the primary risk factor for HIV infection in Kasensero. He even dismissed as "fear and superstition" local claims that mosquitos and other biting insets might be

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[75] Serwadda OH.
[76] Serwadda OH.
[77] Maria Wawer, David Serwadda, Stanley Musgrave, et al., "Dynamics of Spread of HIV-1 Infection in a Rural District of Uganda," *British Medical Journal* 303, no. 6813 (1991):1305.
[78] Serwadda OH. Ronald Gray visited Rakai in 1989. He recalls, "after studying the prevalence data and maps, [I] decided that there was a spatial epidemiologic pattern suggesting an intermediate prevalence community structure." Ronald Gray, email communication, February 24, 2017.
[79] Ed Hooper, "AIDS in Uganda," *African Affairs* 86, no. 345 (1987): 469-477, quotation on 472.
[80] Randall Packard and Paul Epstein, "Epidemiologists, Social Scientists and the Structure of Medical Research on AIDS in Africa," *Social Science and Medicine* 33 (1991): 771-794.

spreading the disease, a possibility seriously entertained by scientists only two short years earlier.[81]

Once the study sample had been selected, the work of recruitment began. The practice of working in some randomly selected villages and not others led to some suspicion on the part of local leaders who wondered whether the selection of villages was an indicator of where the disease was or wasn't present.[82] The team was also concerned about the possibility that participation in a study related to HIV might be stigmatizing for the communities and individuals they approached—they specified in the study protocol that "In order to avoid stigmatizing the study subjects as persons being examined solely for HIV infection, the survey team will stress the fact that the questions and blood samples can be used to study a number of health conditions."[83]

Making the Dynamics of HIV in Rakai Visible

In 1989 Wawer submitted a proposal to the NIH for a grant entitled "HIV Dynamics and Prevention, Rakai District Uganda", known among project staff as the HIV Dynamics Study. This was the grant that would support the second phase of the research begun with the USAID funds. The proposal was for a four-year grant, running from Jan. 1, 1990 to Dec. 31, 1993 at a total cost of $2,461,671.[84] The study was intended to build on the preliminary cross-sectional study in order "to obtain descriptive epidemiological data on HIV-1 prevalence, annual seroconversion rates, patterns of transmission (including sexual and perinatal), progression to clinical disease, cofactors and coinfections, and behavioural risk factors." It also used knowledge, attitudes and behaviors (KAB) indicators "to evaluate the effects of health education and condom distribution programs."[85] Finally, the project was designed to permit the incorporation of novel prevention technologies and strategies, should they become available. While the preliminary study had

[81] Hooper, "AIDS in Uganda," 472; Serwadda, Sewankambo, Carswell, et al., "Slim Disease," 852.
[82] Ssembatya OH.
[83] UP/010/95: USAID Proposal, 15.
[84] DHHS Grant Application, "HIV Dynamics and Prevention, Rakai District Uganda", 1.
[85] DHHS Grant Application, "HIV Dynamics and Prevention, Rakai District Uganda", 2.

enrolled 840 households in 21 clusters (divided into 9 "roadside trading centers" and 12 "rural villages"), the expanded study was planned to double the total study population to approximately 6800 individuals with four more years of follow-up.

The nature of households, like the difference between community types, could be opaque to the uninitiated. Because enrollment was planned at the household level, the project assumed an average household size of approximately 5 individuals in order to determine the number of households that should be enrolled in each cluster. Households were defined as people living together and sharing meals.[86] But apparently simple questions on the census form such as the relationship between different members of the households could stymie researchers, particularly those from outside Uganda. Tom Lutalo, who was hired by the UVRI in 1988 as a statistical research officer and who began working with the RP in the early 1990s, described the challenges American-based researchers had in interpreting some of the Rakai household data. Polygamy, multi-generational households, households that included apparently unrelated children, and the location of households in relation to one another apparently confused some researchers. Lutalo described the difficulty of making sense of the data without intimate knowledge of the places in Rakai the study subjects inhabited, the politics of infrastructure development (or lack thereof), and the subtleties of land ownership and utilization.[87] Part of his job was to help the foreign researchers make sense of the data so that they could recognize data entry errors and attach meaning to the patterns that they were describing. And sometimes he had to persuade them that what looked like problems in the community that they could "fix" were more complicated than they realized. For example, Lutalo recalled a conversation he had had with a Chinese-American researcher visiting Rakai for the first time:

[86] Musgrave OH. This "classic" household definition was useful for the purposes of the early study, but as the RP and similar studies have invested more heavily in network-based analyses, it has imposed some limits when it comes to linking individuals, particularly highly mobile individuals. For these reasons, there has been some discussion of restructuring the cohort along different lines, but, as Musgrave put it, "once it's set up, it's a bear to change". Musgrave OH.
[87] Lutalo OH.

> 'I don't understand, Tom. What's the value of being scattered like this? Why don't you talk to them?' 'They construct houses near each other so that the water will be brought easily, telephone will-and then they till that other land together.' He had not imagined that this is the distribution of the household because he could see that the distance to the well was this. 'Why do you continue having those long distances to the well? Why don't you construct houses together and you team up and you bring water?' Then I asked, 'But who is going to give you the capital for that water—from which source? Their pipes—whatever is involved, it requires the intervention of the government.' 'Then why doesn't the government do it?' Ah, now you have to explain to him the political situation—the land issues. You get me?[88]

Lutalo's job on paper was to manage the statistical data—but in practice he is also an interpreter of the places in which the research was conducted.

The HIV Dynamics study included key personnel from Columbia University (Wawer, Musgrave, Zena A. Stein, and Patrick E. Shrout), infectious disease specialist Michael Lange, from St. Luke's/Roosevelt Hospital, and four Ugandans: Serwadda, Sewankambo, Joseph Konde-Lule from Makerere and Makko Musagara of the Uganda AIDS Control Programme.[89] In 1990, the two main men on the ground in Entebbe were Musagara and Stanley Musgrave..Musgrave was a 35-year old American physician who had previously worked in Ethiopia and Haiti before joining the Center for Population and Family Health at Columbia University where he encountered Wawer and the Rakai Project. As the Resident Advisor to the AIDS Control Program (ACP) of the Uganda Ministry of Health for the Rakai Project, Musgrave was Wawer's representative in Uganda while she was absent. Musagara was only 30 years old and had been working as a health educator with the AIDS Control Program and consulting for the National Population Program of the Uganda Ministry of Planning and Economic Development for two years. Before attending university he had been employed as an enumerator in the 1980 National Population Census and after earning his B.Sc. in geography from Makerere University in 1985 he went to work as a research assistant a the Institute of Statistics and Applied Economics at Makerere. Then the WHO sponsored him for a program at the University of Exeter for his M.A. in Population Research specializing in reproductive health research. At Exeter he read accounts of

[88] Lutalo OH.
[89] DHHS Grant Application, "HIV Dynamics and Prevention, Rakai District Uganda", 2.

the AIDS epidemic in Uganda in the WHO's *Population Reports* and upon his return applied to work at the ACP.[90]

Musagara was working in the office next door to Stan Musgrave when Musgrave began working on the first questionnaire for the RP study. Musagara was interested and offered his services as a trained population researcher and Luganda speaker to translate the questionnaires. The MOH approved his request to assist full-time with the RP and he participated in the field studies planning, eventually becoming the field supervisor.

Together, Musgrave and Musagara mapped the study clusters, organized the survey teams, dealt with the logistics of transportation, payroll, etc. They made maps on F4 (210x330mm) papers showing the study communities in great detail. As Musagara described them, "you had to draw each house, each household. Then we drew the roads, the vegetation if possible. We had to even number those houses."[91] These maps served practical purposes for the research team members, allowing them to navigate unfamiliar villages, keep track of the movement of people from one place to another, and document their decisions about the boundaries of villages. They were also a tool for making visible the etiology of the virus in Rakai.

It is important to recall what was known and what was not yet known about AIDS in Uganda at the time the HIV Dynamics study was launched. In the 1989 journal *AIDS* "Cumulative bibliography of the current world literature on AIDS," there were only 51 entries in the section on "Aids in Africa." [but this only includes one year of publications. There is also a 1988 edition but I can't find an earlier one that would cover 1986.][92] The most high-profile studies on AIDS in Africa were being conducted in Kinshasa at Mama Yemo hospital by *Projet SIDA*, a collaboration between several international scientists established in 1984 with funding from the CDC.[93] Prevalence data for Kampala was based on male blood donors (15% HIV-

[90] Musagara OH.
[91] Musagara OH. I have not been able to locate any of these original maps, though they sound very similar to those made at the beginning of the STD study, described in Chapter Six.
[92] "Cumulative bibliography of the current world literature on AIDS," *AIDS* 3,1 (1989): A1-A53.
[93] Jon Cohen, "The rise and fall of Projet SIDA," *Science* 278, no. 5343 (1997): 1565-1568.

positive in 1987) and antenatal clinic attendees (24% HIV-positive in 1987).[94] As of December 1988, the Ugandan national surveillance system had reported 6772 cases of AIDS of which just over half were in women. The distinction between HIV-1 and HIV-2 had been recognized and it was known that the Ugandan epidemic was of HIV-1. Furthermore, it was known that prevalence of the infection was rising quickly in central and eastern Africa, based on data from serial serosurveys, population surveys, clinic surveys, and studies of prostitutes. Most of the non-sexual modes of transmission had been largely discounted based on assumptions about the link between AIDS and sexual behavior, but were not yet so out of the question as to go without saying—the HIV Dynamics study proposal made a point of stating "There is no evidence that HIV-1 is vector borne."[95]

The unique contribution of the Rakai Project was to be the nature of its study population. As the HIV Dynamics proposal argued, "Most African data have been gathered from urban and clinical settings, and from selected groups (blood donors, prostitutes, STD patients)."[96] By contrast, the RP would shed light on the epidemic in the general rural population. As they put it,

> There are a number of compelling reasons for examining the HIV-1 epidemic in rural and roadside trading areas in Africa. Eighty percent of the African population is rural and represents a potentially vast reservoir for infection. HIV spread within rural regions will influence the impact of the epidemic on mortality and population dynamics in sub-Saharan Africa. Transmission by road travellers (and by prostitutes in trading center stops) is thought to represent a principal mode of HIV spread in Africa. Patterns of transmission, the prevalence of cofactors, sexual behavior, and the natural history of HIV-1 infection may be different in rural than in urban areas.[97]

It is no coincidence that, despite these points, most of the research on AIDS in Africa up until this point had been conducted in cities. Rural African communities were difficult to access by African researchers and seen as almost insurmountably inaccessible to international researchers. In the aftermath of Idi Amin's regime and the devastating civil war that followed his overthrow, roads

[94] HIV Dynamics proposal, 53.
[95] HIV Dynamics proposal, 54.
[96] HIV Dynamics proposal, 56.
[97] HIV Dynamics Proposal, 56.

were in disrepair, sanitation infrastructure was nearly absent outside the big cities, and telephones were rare. But the RP described the advantages of going to the Rakai District to conduct AIDS research:

> The district offers unique opportunities to study non-urban HIV-1 patterns since it includes agrarian villages and towns on the major international trading routes from Tanzania in the south, Rwanda in the west, and Lake Victoria to the east. AIDS in Uganda was first identified in Rakai. The District presents the possibility of examining the dynamics of the epidemic in both high prevalence communities (trading centers) and in those with low to moderate rates (rural villages). Moreover, the high prevalence of HIV-1 in Rakai facilitates the establishment of cohort studies because surveillance can be maintained in smaller populations than in areas of low prevalence.[98]

Furthermore, the Rakai Project team justified its funding by arguing that their work would produce data that could be applied to "other rural African settings."[99]

The decision to oversample clusters with major trading centers had important consequences for the nature of the cohort. This was in contrast to a very similar cohort established shortly after the beginning of the RP by the UK's Medical Research Council (MRC) in Masaka, the district immediately to the north of Rakai. The MRC's Masaka project was also headquartered at the UVRI. In 2010 anthropologist Jan Kuhanen had this to say about the sometimes disparate findings of the two cohorts:

> Rakai is a predominantly rural district in south-west Uganda, bordering Tanzania, and the home of the first longitudinal community-based research project on HIV and AIDS in Uganda. Another such project was established in the neighbouring Masaka District and, though similar research took place at both sites, the results have been remarkably different, with lower incidence and prevalence levels and a greater degree of reported behaviour change documented in Masaka than in Rakai. The differences in results are partly due to the composition of the populations, as the initial population cohort in Rakai consisted of 12 rural villages and nine peri-urban trading centres with 40 households in each, while the Masaka cohort consisted of a cluster of 15 villages in one rural sub-county located 35 km away from the town of Masaka and 16 km from the main highway. Masaka had a larger population in absolute terms (about 10,000 people), but did not include urban or peri- urban communities. Given the greater proportion of townspeople in the Rakai cohort, the occupational and income differences and the types of social activity, it is clear that the levels of mobility, sexual networking and HIV incidence and prevalence tend to be more variable there

[98] HIV Dynamics Proposal, 56.
[99] Sexual Networks Proposal, 63.

> than in the purely rural Masaka cohort. The Rakai cohort is therefore more representative of the whole area, whereas the Masaka cohort may only be representative of the rural population of the Masaka district.[100]

However, as Kuhanen went on to point out, results from Rakai were often reported as findings from a study of rural Uganda. The enormous influence of Rakai's work in the field of early HIV studies in Africa mean that this particular construction of a rural study community came to represent what epidemiologists understood to be rural Africa.

The Rakai Project wasn't the only attempt to measure the extent of the epidemic in Uganda. In 1987 the Ugandan government had launched a national serosurvey designed to yield "a random probability sample of the population".[101] But it excluded "regions that were insecure"—namely the north and the eastern regions of the country where Museveni's government had not yet established control.[102] Moreover, it was, at best, a snapshot of the epidemic in a particular moment in time, whereas the Rakai Project was always intended to supply longitudinal data about the *dynamics* of the epidemic.

Specifically the project was designed to fill two types of "gaps in knowledge": population-based information on the transmission of HIV, progression of HIV-related disease, and HIV cofactors on the one hand and the behavioral data needed to design, implement, and evaluate preventive measures.[103]

Mapping Sexual Networks

In August 1991 the Rakai Project submitted another proposal to the NIH for a project entitled "Ugandan Sexual Network/Behaviors Study for HIV Prevention" to run from January 1992 through December 1994, overlapping with the end of the HIV Dynamics study.[104] Maria Wawer

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[100] Jan Kuhanen, "'Balinsalamu embawo?' AIDS and the context of sexual behaviour adjustment in Rakai, Uganda, c. 1975-90," *Journal of Eastern African Studies* 4, no. 1 (2010): 20-43, fn 17.
[101] Seth Berkley, Warren Naamara, Samuel Okware, et al, "AIDS and HIV infection in Uganda—are more women infected than men?" *AIDS* 4, no. 12 (1990): 1237-1242, quotation on 1238.
[102] Berkley, Naamara, Okware, et al, "AIDS and HIV infection in Uganda," 1238.
[103] HIV Dynamics proposal, 58.
[104] Sexual Network proposal.

was the principal investigator and Janet Edmonston was the resident behavioral special advisor in Uganda. Other co-investigators on the project included Martina Morris, a sociologist at Columbia University, Sewankambo, and Edward Kirumira, a sociologist at Makerere University.[105] The study was designed to build on the initial findings from the HIV Dynamics study which indicated that "geographic location, mobility and related variables influence sexual behavior and the structure of sexual networks in the study population, contributing in turn to the rapid spread of HIV infection."[106] The proposal outlined plans "to define sexual networks and behaviors critical to the transmission of HIV infection, and to elucidate their determinants, including place of residence, geographic mobility and social/ethnic status. A specific objective will be to determine whether there are potential behavioral barriers to HIV spread from high to low prevalence areas, and whether certain communities act as bridging populations facilitating HIV transmission into lower prevalence villages."[107]

The project was divided into three components: a set of in-depth interviews, focus groups, and a structured sexual network and behaviors survey. The study design called for in-depth interviews and focus groups in six to eight villages in Rakai district. The study sites were to be divided between trading centers, intermediate trading villages, and rural villages.[108] This stratification was based on the findings from the very first pilot study as well as the ongoing HIV dynamics study that "Rates of infection vary dramatically across three geographic strata (agricultural villages, small trading villages, and trading centers) in the district."[109]

The sites were also to be evenly divided between villages already included in the HIV cohort study (operating under the HIV dynamics study funding) and villages not already included in data collection by the Rakai Project.[110] The proposal elaborated:

[105] Sexual Network proposal, 2.
[106] Sexual Network proposal, 2.
[107] Sexual Network proposal, 2.
[108] Sexual Network proposal, 52.
[109] Sexual Network proposal, 52.
[110] Sexual network proposal, 52.

> The rationale for including a number of the villages enrolled in the cohort study is to acquire more in-depth understanding of behaviors in setting[s] where we have detailed information regarding the distribution of HIV infection and current incidence rates. However, since reported or actual behaviors may have changed as a result off the study and its associated preventive program, communities outside the cohort will also be selected for comparative purposes.[111]

Already in the first years of the Rakai Project, the paradoxical tension between the value of a long-term cohort and the problem of accounting for the effect of the study itself on the behavior and characteristics of the population it studied was apparent.

Beyond the stratification of study communities, the Sexual Networks Study also described certain types of individuals that were of particular interest. The study design was based on, and therefore produced data consistent with, existing assumptions about who was at risk for AIDS. The in-depth interviews were to include subgroups representing "group[s] of interest": "village and community leaders, CSWs [commercial sex workers]/bar girls; clients of CSWs; hotel/bar owners and managers; truck drivers and their assistants; local health personnel; young women (15-24) in rural and trading villages and main road trading centers; young men (15-24) in the three settings; older adults (25+) in these settings; long term residents, recent migrants, and returning travellers (work related and other travel)."[112] The focus groups were set up to include people grouped by age, sex, socioeconomic status and "characteristic(s) of interest, such as occupation or migration."[113]

Overall, the survey component of the study was designed to include 1600 members of the district's adult population in addition to 450 individuals "selected by quota on the basis of characteristics of interest" if they were not adequately represented in the general random sample.[114] Such individuals included "CSWs, truck drivers, shopkeepers, travellers/migrants and other persons identified in the [in-depth interviews] and [focus groups] as being important

[111] Sexual network proposal, 53.
[112] Sexual network proposal, 54.
[113] Sexual network proposal, 56.
[114] Sexual network proposal, 58.

bridging populations in trading centers, intermediate trading villages and rural communities."[115] By focusing on the "characteristics of interest" the RP made it more likely that they would find evidence that affirmed and perhaps refined the existing understanding of AIDS transmission patterns in sub-Saharan Africa, but made it less likely that they would observe any other patters of transmission.

Conduct of the sexual networks study built on the early success of the RP in recruiting both skilled personnel and study subjects. The proposal emphasized the RP's 2.5 years experience obtaining high response rates in serosurvey work, focus group participation, and enlisting the support of local medical and health education workers, who had "become adept at motivating villagers to participate."[116]

Relating Rakai Project Data to the Global Geographies of AIDS

The earliest publications to come out of the Rakai Project were largely descriptive and provided some of the earliest data about the scope and nature of the epidemic in rural Uganda (which was extrapolated to East Africa and, sometimes, to sub-Saharan Africa. The first journal article to list the Rakai Project as the institutional affiliation of some of its authors was published in 1990 in *AIDS* and suggested that the findings from Rakai were inconsistent with WHO's model of Patterns I, II, and III epidemics.[117] According to this typology, based on a mixture of geographic and epidemiologic criteria, the Pattern II epidemic was located primarily in sub-Saharan Africa and was propagated primarily through heterosexual intercourse and perinatal transmission.[118] Part of the description of Pattern II epidemics, the assertion that in Pattern II countries men and

[115] Sexual network proposal, 58.
[116] Sexual networks proposal, 61.
[117] Berkley, Naamara, Okware, et al., "AIDS and HIV infection in Uganda."
[118] Jonathan Mann and James Chin, "AIDS: A Global Perspective," *New England Journal of Medicine* 319, no. 5 (1988): 302-303.

women were infected at approximately equal rates, was contrary to what was being observed in Uganda where women had a rate of infection 1.4 times greater than men.[119]

The Rakai Project's early work in the HIV Dynamics and Sexual Networks studies was consistent with what Cindy Patton has described as the contradictory tropical and epidemiological "thoughtstyles" in early international AIDS research which were simultaneously shaped by an understanding of disease as rooted in place while also visualizing the movement of diseased bodies between places.[120] The Rakai Project was fully implicated in what Patton describes as the Global Programme on AIDS's effort to reorient thought about the disease from a tropical model based on dividing up parts of the world into healthy and diseased towards an epidemiological model based on transmission patterns. As Patton demonstrates, this program was almost immediately undermined by its own discourse which "grafted" the Pattern I, II, and III epidemics back onto maps with obvious links to the tropical disease maps of the previous century. Thus Pattern II AIDS, defined as an epidemic principally transmitted by heterosexual intercourse, almost immediately became more commonly known as "African AIDS".[121]

Silences in the Research

While the Rakai Project aimed at developing a comprehensive set of longitudinal community-based data related to HIV/AIDS incidence and prevalence, there were areas it declined to cover. It deliberately avoided topics such as "homosexuality, masturbation, and anal intercourse" with the explanation that "Other African data, the physical evidence from the Kampala STD clinic, focus group research in the Rakai project, and preliminary data from the Rakai and MRC counseling programs all suggest that such practices are rare in our population."[122] While they had not yet developed a questionnaire at the time they submitted the sexual networks proposal, they included

[119] Berkley, Naamara, Okware, et al, "AIDS and HIV infection in Uganda," 1239.
[120] Cindy Patton, *Globalizing AIDS* (Minneapolis: University of Minnesota Press, 2002).
[121] Patton, *Globalizing AIDS*, 59-60.
[122] Sexual networks proposal, 62.

a questionnaire used in Thailand in April 1991, which included a variety of explicit questions about specific sexual behaviors including questions about anal intercourse and homosexual sex. Because of the aforementioned understanding about the lack of such behaviors in Uganda, the Rakai Project proposed the following:

> Given this information, we may test a strategy of asking some of the very detailed sexual practices questions of only a subsample of our network respondents to determine respondent reaction and the effects on the interview. If the practices are indeed not part of the local sexual repertoire, the amount of information lost by truncating the practices section of the questionnaire may not be great compared to the enhancement of project feasibility and acceptance by the community.[123]

In other words, unless they were much more common than believed, the cost of asking about highly-stigmatized behaviors outweighed the possible benefits of documenting that they were taking place. Condom use behavior was explicitly noted as an exception to this proposed policy of avoiding explicit questions about sexual practices.[124] This strategy, while politically and practically expedient, limited the ability of the Rakai Project to produce data that might challenge pre-existing notions of sexuality in Rakai and rural Africa more broadly.

Challenges and Opportunities in International Collaboration

Veterans of the early RP are almost unanimous in their accounts of mutually respectful interactions between American and Ugandan collaborators.[125] But there are indications the PIs were sensitive to the perils of international collaborations. In 1990 Serwadda and Edward Katongole-Mbidde coauthored a viewpoint article in the *Lancet* about the challenges facing researchers on AIDS in Africa.[126] They lamented the frequent preoccupation of foreign researchers with establishing the "origin" of AIDS in Africa—a project that they described as "pejorative and unfortunate" and which lost researchers the goodwill of many African

[123] Sexual networks proposal, 62.
[124] Sexual networks proposal, 62.
[125] It is likely that people with more negative recollections of the early years of the project were less likely to be still affiliated with it by the time of my research and thus less likely to have been interviewed by me.
[126] David Serwadda and Edward Katongole-Mbidde, "AIDS in Africa: Problems for Research and Researchers," *Lancet* 335, no. 8693 (1990): 842-843.

governments.[127] As a result, they claimed, "the motives of foreign researchers were viewed with extreme suspicion—suspicion that was readily transferred to their African coworkers."[128] They also pointed out that "HIV seroprevalence figures from small local surveys are often quoted as representative of a whole country, and may even be extrapolated to apply to the whole continent", a misuse of data which Serwadda and Katongole-Mbidde said not only inflamed government resistance to research but also made "patients and local populations reluctant to participate in further hospital or community-based studies, especially in areas for which high seroprevalence rates have been described"—such as Rakai.[129] Most damningly, the two Ugandan doctors said that "it sometimes seems that researchers (and funding agencies) from overseas find it easier than their local colleagues to overlook the suffering caused by AIDS to individuals and communities in Africa."[130] But Serwadda and Katongole-Mbidde were not discouraging international collaborations on AIDS research in Africa—on the contrary, they made it clear that they believed the urgency of the epidemic made it imperative to apply all available resources and acknowledged that "Africa needs financial, medical, and scientific support to cope with the clinical effects of HIV infection and for further research."[131] Part of the solution, they concluded, was for research programs funded by foreign agencies to commit greater resources towards programs that would benefit study populations and towards training of African "local collaborators."[132]

This was an objective that the RP was mindful of in its early proposals. The Sexual Networks study described the role of Janet Edmondson, the anthropologist listed as the "resident behavioral science advisor," as critical "Given the paucity of trained and experienced Ugandan behavioral scientists" and indicated that "Dr. Edmondson's role will include strengthening the behavioral science infrastructure in Uganda through her work with project counterparts on faculty

[127] Serwadda and Katongole-Mbidde, "AIDS in Africa," 842.
[128] Serwadda and Katongole-Mbidde, "AIDS in Africa," 842.
[129] Serwadda and Katongole-Mbidde, "AIDS in Africa," 843.
[130] Serwadda and Katongole-Mbidde, "AIDS in Africa," 843.
[131] Serwadda and Katongole-Mbidde, "AIDS in Africa," 843.
[132] Serwadda and Katongole-Mbidde, "AIDS in Africa," 843.

at Makerere University."[133] The proposal was also explicit about prioritizing opportunities for Ugandan scientists to participate in international conferences, noting that 4 of the 6 trips budgeted for international conferences would be taken by Ugandan researchers.[134]

Suffering and Despair: The Experiences of Local Scientists

As Serwadda and Katongole-Mbidde's pointed out, foreign researchers didn't always (and perhaps couldn't) share the same empathy for their research subjects that local researchers experienced. One of the common threads in many of my interviews with Ugandan members of the Rakai Project team was their personal connection to the AIDS epidemic and the suffering it wrought in Uganda in the 1980s and 1990s. Joseph Ssembatya described times in 1988 when he might attend five burials in a single day.[135] Another physician-researcher who grew up in Rakai told me "In my village back in the late 1980s, I witnessed lots of death when I was a boy. Almost every week there was somebody being buried in the village…there were lots of deaths."[136] The scale of devastation in Rakai district was something many RP employees witnessed first-hand before ever becoming involved in research. For project staff members who live in Rakai district and have family and friends in many of the study communities, the data tables represent more than statistics. And project members don't have to look beyond the staff of the research program itself to see the impact of AIDS: Serwadda recalls that before antiretroviral therapies became widely available, many of the Rakai Project's first employees died quite young, often of AIDS.[137]

For physicians, the motivation that drove them to epidemiologic research stemmed in part from a sense of helplessness as clinical providers. In 1990 a *New York Times* article entitled "Scenes from a nightmare" described the conditions at Mulago where Serwadda and his colleagues were struggling to manage overwhelming numbers of desperately ill AIDS patients.

[133] Sexual Networks Proposal, 6.
[134] Sexual Networks Proposal, 9.
[135] Ssembatya OH.
[136] Kagaayi OH.
[137] Serwadda OH.

The reporter quoted Serwadda saying, "I've got to get out of clinical medicine. It's too depressing…if I'm ever going to make any difference, I have to go into public health…Ninety percent of the patients I treated last year in the department of medicine were terminal. And young: 20, 22, 26 years old. It's so depressing. So depressing."[138] Dr. Benon Biryahwaho made a similar observation about his decision to leave clinical practice for research a decade earlier after an internship at Mulago in pediatrics in the late 1970s: "Some admission days you have 30, 40 kids admitted [with] raging fever and diarrhea and vomiting. But you have no IV fluids. So the next morning, you spend half a day writing death certificates for those who passed on. After six months of that period, I decided no, I'm not going to be a clinician. My whole orientation changed totally."[139] In both periods, when Uganda's health care sector was struggling to deal with crumbling infrastructure, drug scarcities, and poor morale, virus research was an attractive avenue for physicians who wanted to see their work make a difference.

Exporting the Stratified Geography of Rakai

The first major publication to come directly out of the Rakai Project, "Dynamics of Spread of HIV-1 Infection in a Rural District of Uganda," appeared in 1991 in the *British Medical Journal*.[140] The HIV Dynamics paper, as it was called by members of the project, put Rakai on the radar of AIDS researchers worldwide.[141] In the paper, the authors present the results as an important contribution to knowledge about the epidemic in rural sub-Saharan Africa because of their distinction between different types of rural communities. What they observed was

[138] Kathleen Hunt, "Scenes From a Nightmare," *New York Times* August 12, 1990.
[139] Biryahwaho OH.
[140] Wawer, Serwadda, Musgrave, *et al*, "Dynamics," 1303-6. The Rakai Project has consistently favored publication in British journals, which are more widely read by physicians in East Africa than the American journals. In particular, studies from the *Lancet* are often reported by the BBC and thus reach a large audience in Uganda. Serwadda OH.
[141] According to Web of Science, the paper was cited 66 times in the decade following publication. Google Scholar lists 79 citations in the same period. Both databases were queried on August 6, 2016.

"substantial variations in HIV seroprevalence" within the district population.[142] The mean seroprevalence by cluster ranged from 1.2% to 52.8%.[143]

The following year another paper appeared in AIDS with an even greater emphasis on the 3-tiered stratification of the study population.[144] Entitled "HIV risk factors in three geographic strata of rural Rakai District, Uganda," the paper concluded that heterosexual intercourse was the primary risk factor across the district, but that the degree of risk associated with sex varied widely between strata and that this knowledge should inform prevention programs. In the final line of the paper, the authors suggest that district residents themselves should be encouraged to think about the risk of their sexual encounters with respect to the strata: "We should ensure residents of rural villages know that sexual contact with individuals from trading centers or intermediate trading villages can substantially increase the risk of exposure to HIV infection."[145]

The first papers described essentially static data—it was only after observing the cohort for several years that the project could begin producing longitudinal data. And because the questionnaire didn't attempt to link sexual partners and so couldn't directly observe infection transmission. But the authors of the paper interpreted the differences between the three strata as indicative of larger transmission patterns. As they put it, "it is possible to infer the spread of infection by examining differences in HIV seroprevalence by cluster and by type of cluster. The differences noted between main road trading centres, rural trading villages, and rural agricultural villages…suggest that HIV transmission tends to follow lines of communication along main and secondary roads, as had been found with other sexually transmitted disease."[146]

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[142] Wawer, Serwadda, Musgrave, *et al*, "Dynamics," 1305.
[143] Wawer, Serwadda, Musgrave, *et al*, "Dynamics," 1305.
[144] David Serwadda, Maria Wawer, Stanley Musgrave, et al., "HIV Risk factors in Three Geographic Strata of Rural Rakai District, Uganda," *AIDS* 6, no. 9 (1992): 983-989.
[145] Serwadda, Wawer, Musgrave, et al, "HIV Risk Factors," 989.
[146] Wawer, Serwadda, Musgrave, *et al*, "Dynamics," 1306.

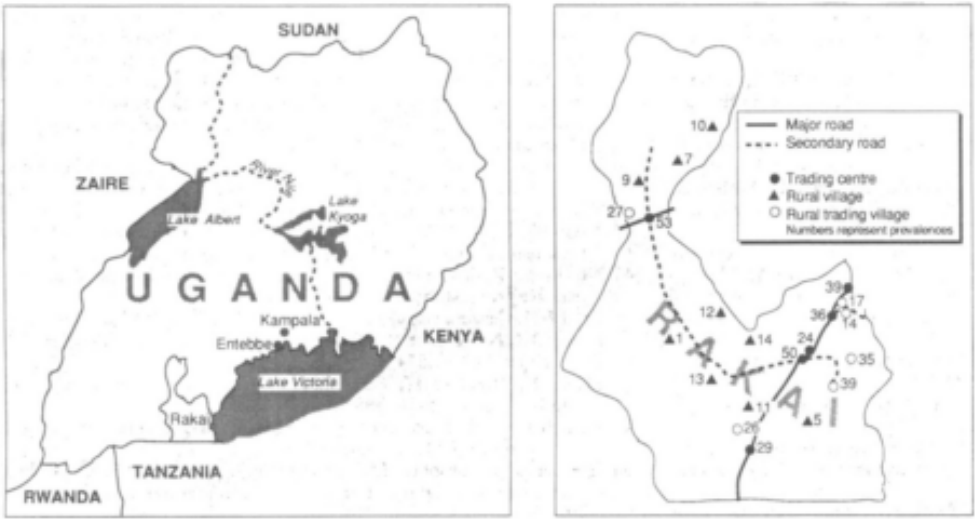
Conclusions

In the early 1990s, the Rakai Project was attempting to cobble together enough funding in relatively small amounts to sustain the cohort. Despite recognition from national and international groups as "a prototype for future ...programs"[147], its future was uncertain. When the Rakai Project's funding for the cohort expired at the end of 1992, the remaining funding sources were insufficient to support the cohort in its entirety. While select studies continued doing work among subpopulations of the study community, two years of data on the cohort as a whole were lost in 1993 and 1994. In order to restart the longitudinal community-based study the Rakai Project had always intended to operate, they needed to find an intervention to test—something that would capture the interest and resources of major funding groups. The groundwork had been laid, but to cement Rakai's reputation as a site of knowledge production for global HIV prevention they needed a high-profile research subject. The next chapter will tell the story of that intervention—STD treatment for HIV prevention.

The story of the Rakai Project's early years offers a new perspective on the way that global knowledge about AIDS in Africa was produced and established in the 1980s and early 1990s. Unlike many popular narratives, it places Ugandan researchers at the center of the story and demonstrates that their personal, professional, and intellectual experiences were decisive in the design and implementation of AIDS research projects. It also illustrates how widespread preconceptions about Africa sexuality and its relationship to HIV risk influenced the design of this seminal research project and, thus, the types of data that it produced. By the mid-1990s they had become part of the accepted wisdom about AIDS in Africa and had an indelible impact on the trajectory of subsequent research on prevention and treatment of AIDS in Uganda and far beyond.

[147] Seth Berkley, "RF Grant #91079, #31, Family and community-based health care initiatives," in "100 Years: The Rockefeller Foundation, http://rockefeller100.org/items/show/4675, accessed April 22, 2013.

Figures and Tables



Maps (left) of Uganda showing Rakai district and (right) of Rakai district showing geographical distribution of clusters and prevalence of HIV-I infections in each cluster

Figure 1: Map showing the location of Rakai District (left) and the locations of the study clusters as well as their respective HIV-1 prevalence rates (right). Reproduced, with permission, from Wawer, Serwadda, Musgrave, *et al*, "Dynamics of Spread of HIV-1 Infection in a Rural District of Uganda," *British Medical Journal* 303, no. 6813 (1991): 1305.

	Main road trading center Number (percent)	Rural trading village Number (percent)	Rural agricultural village Number (percent)
Number of clusters	6	6	9
Number of households	236	238	359
Mean distance of clusters to main road (km)	n/a	4.5	19.8
Presence of lodge (hotel)	6 (100)	0	0
Presence of bar	6 (100)	6 (100)	0
Households with modern building materials	224 (95.0)	185 (77.7)	120 (33.3)

Table 1: Characteristics of study households by cluster stratum. Adapted, with permission, from Wawer, Serwadda, Musgrave, *et al*, "Dynamics of Spread of HIV-1 Infection in a Rural District of Uganda," *British Medical Journal* 303, no. 6813 (1991): 1305.

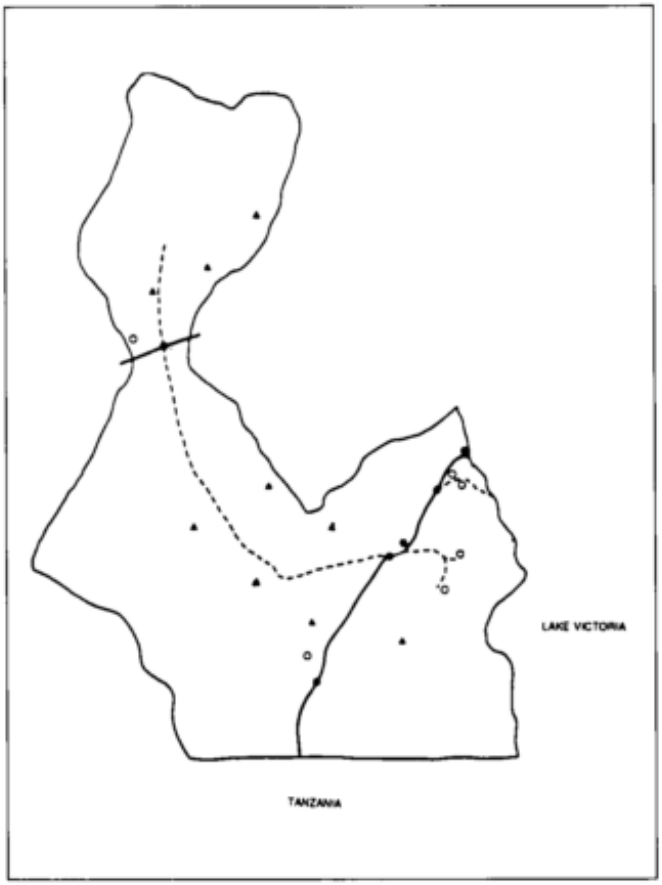


Fig. 1. Map of Rakai district showing the geographic distribution of clusters. Major road (——); secondary road (- - - -); trading center (●); rural village (▲); intermediate center (○).

Figure 2: Reproduced, with permission, from Serwadda, Wawer, Musgrave, *et al*, "HIV Risk Factors in Three Geographic Strata of Rural Rakai District, Uganda," *AIDS* 6, no. 9 (1992): 984.

Chapter Six
Experimental Communities: The Rakai Project Study of STD Control for HIV Prevention, 1994-2000

Introduction

International aid partners such as the United States Agency for International Development (USAID) and the Joint United Nations Programme on HIV/AIDS (UNAIDS) aggressively marketed Uganda as an AIDS success story in the 1990s.[1] But in Rakai, among the Ugandan researchers and their closest collaborators, it was a decade of uncertainty and disappointment in the outcomes of experimental interventions. By the mid-1990s, hopes for rapid vaccine development were fading and the Rakai Project was considering other interventions that would be suitable for trial in their cohort. In 1993 their application to the World AIDS Foundation for a grant to promote vaccine trial acceptance was turned down.[2] Funding was a problem—in 1993 the patchwork of funding that the Rakai Project had assembled in previous years to fund the cohort fell apart and the cohort was discontinued. The Project itself didn't dissolve; limited funding from Rockefeller Foundation funding for HIV prevention activities, and technical assistance support from the Mellon Foundation kept a skeleton crew of project staff employed part-time. But it wasn't until 1994 that the Project was able to secure funding to restart the cohort. This was largely due to their ambitious proposal to test an intervention designed to break the connection between sexually transmitted diseases (STDs) and HIV infection.[3]

Chapter Five described the way that the discovery of the AIDS epidemic in Uganda by a group of physicians and epidemiologists based in Kampala attracted international researchers back to Uganda after a decade of neglect, and how the team working in Rakai began to define the

[1] Jan Kuhanen, "The historiography of HIV and AIDS in Uganda," *History in Africa* 35 (2008): 301-325; Jan Kuhanen, "'No sex until marriage!': Moralism, politics and the realities of HIV prevention in Uganda, 1986-1996," *Journal of Eastern African Studies* 9, no. 2 (2015): 270-288.
[2] Dr. Raymond Dedoner to Dr. Stanley Musgrave, July 19, 1993, RHSP records, "WAF/08/95: WORLD AIDS".
[3] Sexually transmitted diseases or STDs was the terminology used during the trial and the terminology that I will use in this chapter. Today the preferred term is sexually transmitted infections (STIs) reflecting the fact that many STIs don't result in any recognizable symptoms or disease state.

district in terms of its AIDS epidemic in the late 1980s. Work in Rakai and elsewhere in the country focused mainly on "building the capacity for surveillance and research needed for monitoring the epidemic."[4]

In the mid-1990s, the Rakai Project launched a community-randomized clinical trial of mass STD treatment for HIV prevention. This was the next step in transforming the district into a laboratory of sorts for population-based experimental AIDS research. Numerous historians, journalists, and anthropologists have likened sub-Saharan Africa, and Uganda in particular, to a laboratory for HIV/AIDS research.[5] This chapter aims to look more closely at the practices, tools, and people involved in making the cohort in the Rakai District a "population laboratory." Furthermore, it examines the limits of the laboratory metaphor for describing what it was like to live and work in a Ugandan AIDS research site in the 1990s.[6] The case of the Rakai Project's STD trial once again highlights the role of African expertise in establishing these experimental sites, expertise that is often missing from accounts of global public health and biomedicine in Africa.[7] This chapter will highlight the stories of some of the individuals who conducted the STD trial. While African researchers remain anonymous in many accounts of AIDS research in Africa, considering their biographies offers new ways to think about the relationship between scientific work and community building.

In order to conduct the STD trial, the Rakai Project had to fundamentally re-imagine the physical and human geography of the district. Using the data collected during their preliminary observational studies, they constructed a new organization of the district in order to make it work to create knowledge about HIV prevention that could be generalized to rural sub-Saharan Africa. The study also relied heavily on the expertise and skill of African investigators at every level

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[4] Kuhanen, "'No sex until marriage!'," 274.
[5] E.g. Larry Krotz, *The Uncertain Business of Doing Good: Outsiders in Africa* (East Lansing: Michigan State University Press, 2009), 174. The notion of Africa as a laboratory has much broader applications than just HIV/AIDS research. See introduction.
[6] Rakai Health Sciences Program, "Rakai Health Sciences Program (formerly Rakai Project)" Profile on the Indepth network website, http://www.indepth-network.org/dss_site_profiles/RakaiProfile.pdf, accessed January 15, 2016.
[7] Johanna Tayloe Crane, *Scrambling for Africa: AIDS, Expertise, and the Rise of American Global Health Science* (Ithaca: Cornell University Press, 2013), 180.

from data collection to senior management. Many of these investigators had started out at the UVRI, which provided a place for aspiring researchers to get into the system.

While the STD trial put the Rakai Project firmly on the international stage, it also posed new dilemmas.[8] David Serwadda described it as "the trial that gave us the most grief and anxiety."[9] When the trial produced unexpected results, the Project had to explicitly defend the integrity and value of its programmatic organization, scientific protocols, and analytical strategies. A misrepresentation of their methods further led to a serious critique of their ethics. The value of the Rakai team and its parent organization, the UVRI, as a local partner for international AIDS research projects was contested in private meetings, public conferences, and professional journals. In the end, however, the Rakai Project was able to justify its methods and results.

The ability of the Rakai Project to staff and implement such an ambitious study was partly the result of its relationship with the UVRI and the systems that governed virus research developed in the country over the preceding decades. Many aspects of the story told in this chapter, such as the prominence of maps and geographic analysis, and the deliberate and simultaneous performance of local expertise and international credibility by expatriate and Ugandan scientists build on themes explored in earlier chapters. In this chapter, I will describe the rationale for a trial of STD treatment for HIV prevention as well as the design of the Rakai Project's trial. Then I will describe how the UVRI and its legacy of work on international virus research projects were crucial to the trial's implementation. I will show how the people who made the trial possible were a combination of young physicians inspired by Serwadda and Nelson Sewankambo. These were individuals with personal connections to the Rakai District, and a small but significant contingent of expatriates. I will detail the practices and rationale for the mapping of the trial and how the decisions made by the team responsible for delineating the study

[8] Tom Lutalo OH.
[9] David Serwadda OH.

communities had major consequences for the trial and the people it enrolled. Then I will describe the implementation of the trial—the daily practices that went into producing the data required to evaluate the efficacy of the intervention. Finally, I will summarize the trial's results and discuss the impact of those results and some of the controversies that emerged from the unblinding of the study data. Each section of the chapter illustrates the context, people, and practices that went into transforming the Rakai District into a space for producing valid experimental data on the apparent link between STDs and HIV.

Justifying and Designing the Experiment

The link between STDs and AIDS goes back to the very beginning of the known epidemic. One of the early explanations for the cluster of symptoms observed in gay men in the United States was that their promiscuous lifestyle exposed them to an extraordinary level of infection, especially sexually transmitted infections including cytomegalovirus, leading to the collapse of their immune systems. In short, rather than the result of a particular virus, AIDS was thought to be the result of multiple STDs and the unspecified effects of "allogenic semen" overwhelming individuals' immune systems.[10] While this particular theory was generally rejected once HIV was identified as the virus causing AIDS, it was only the first of many that connected AIDS with other STDs. A more specific explanation for the apparent correlation between AIDS and other STDs was that infections leading to genital ulcers, such as herpes simplex virus (HSV) and syphilis, compromised the skin's physical barrier to HIV infection, and increased the concentration of HIV-target cells in the genitals, increasing the likelihood of infection following exposure. This theory suggested that treatment of STDs, in the absence of any other behavior change, would still reduce the intensity of HIV transmission. That is to say, while there was still no vaccine or medication to prevent HIV, there was still a biomedical solution.

[10] Joseph Sonnabend, Steven S. Witkin, and David T. Purtilo, "Acquired immunodeficiency syndrome, opportunistic infections, and malignancies in male homosexuals: A hypothesis of etiologic factors in pathogenesis," *Journal of the American Medical Association* 249, no. 17 (1983): 2370-2374.

At the time the Rakai Project team proposed the trial, the evidence linking STDs with HIV incidence was based on a series of observational studies conducted mainly in clinical settings. As early as 1989, a survey of knowledge, attitudes, and practices (KAP) related to AIDS in two rural sub-districts, Kasangati and Nsangi, located just north and south of Kampala respectively, observed that HIV-infection was correlated with infection with one or more other STDs even after controlling for number of sexual partners and history of injections.[11] A meta-analysis published in 1992 examined 163 studies on the connection between HIV and other STDs and found 15 studies showing that, after controlling for behavior, STDs more than doubled the risk of HIV transmission.[12] The meta-analysis concluded that, for definitive answers about the relationships between HIV and other STDs, there needed to be studies that included control groups.[13] The Rakai Project group set out to do just such a study.

In 1993, Maria Wawer submitted an application entitled "STD Control for AIDS Prevention, Uganda" to the DHHS PHS for a five-year grant in the amount of $7,873,738. The purpose of the grant was to fund a community-randomized, controlled trial of mass STD treatment for HIV prevention. The primary hypothesis to be tested was that "in areas with high prevalence of human immunodeficiency virus (HIV) type-1 and sexually transmitted diseases (STDs), STD[s] enhance HIV transmission and acquisition." This was divided into two specific hypotheses: first "Reductions in STD prevalence and incidence will result in reductions in HIV transmission" and second, "Reduction in STDs can be effectively achieved by mass STD treatment, coupled to active surveillance and case finding." As they put it, mass treatment "represents the only feasible strategy which will provide broad STD coverage in this rural,

[11] Joseph K. Konde-Lule, Seth Berkley, Robert G. Downing, "Knowledge, Attitudes and Practices Concerning AIDS in Ugandans," *AIDS* 3, no. 8 (1989): 513-518. Seth Berkley of the Rockefeller Foundation and an early collaborator of the Rakai Project, summarized the evidence suggesting that the correlation between HIV and other STDs might be more than simply a proxy for high-risk behavior. Seth Berkley, The Public Health Significance of Sexually Transmitted Diseases for HIV Infection in Africa," in *AIDS and Women's Reproductive Health*, ed. Sheldon J. Segal, Lincoln C. Chen, and Jamie Sépulveda Amor (New York: Plenum Press, 1991), 73-84

[12] Judith N. Wasserheit, "Epidemiological Synergy: Interrelationships Between Human Immunodeficiency Virus Infection and Other Sexually Transmitted Diseases," *Sexually Transmitted Diseases* 19, no. 2 (1992): 61-77.

[13] Wasserheit, "Epidemiological Synergy," 72.

underserviced population" while strategies aimed at treating people who sought care in a clinic would miss "many asymptomatic but infectious persons, and can be expected to have substantially lower efficacy in STD control in an area [with] very high endemic rates of STDs, such as Rakai."[14] They went so far as to propose that "STD control by mass treatment…represents the most feasible and effective strategy for the control of STDs, and thus potentially of HIV, in the rural African context."

Rakai wasn't the only sub-Saharan African site testing the connection between STD treatment and HIV prevention. At roughly the same time two other major studies launched, one in nearby Masaka run by the British Medical Research Council (MRC), also headquartered at the UVRI, and one in Mwanza, Tanzania [Figure 1]. The differences between the three study sites and their study populations would be dissected in great detail when they released conflicting results. Given that the nature of the study sites became a critical point in the interpretation of their results, it is useful to take a step back and consider what the study hypotheses assume about Rakai as a study site. Fundamentally it was expected to stand in for all rural African communities with high prevalence of HIV-1 and STDs. While many of the study sites within Rakai did indeed have high HIV and STD prevalence rates, the Rakai Project's own work had shown a range of HIV incidence rates across different villages and towns.[15] Like HIV rates, STD rates varied but were less well studied. In the grant they highlighted two HIV prevalence rates: 12% of people in Rakai over the age of twelve were HIV-positive and the subset of adults between 20 and 34 years old living in "main road trading centers and large trading villages on secondary roads" of whom fully 25% were HIV-positive.[16]

The UVRI and the Rakai Project

[14] "STD Control for AIDS Prevention, Uganda", 102.
[15] "STD Control for AIDS Prevention, Uganda", 105.
[16] "STD Control for AIDS Prevention, Uganda", 105.

One important, but generally unremarked, aspect of the Rakai Project's makeup at the time of the STD trial was its connection with the UVRI. By the early 1990s, Uganda had been a center for virus research activities for more than half a century. After a period of decline in the 1980s, HIV/AIDS-related funding, including the Rakai Project, offered an opportunity for the UVRI to reclaim its place in the international community of virus researchers.[17] For the Project's part, the Institute continued to serve as a vital link to the Ministry of Health, what one member of the STD study team referred to as its "umbilical cord."[18] The UVRI was the source of personnel and infrastructure that made the grant proposal plausible.

One of the key members of the STD trial team was a UVRI employee. Tom Lutalo, a scientific officer at the UVRI, joined the Rakai Project as the chief in-country data manager and analyst in 1994 after having been loaned to the team to work as a data editor during the HIV dynamics study.[19] Unlike many research projects that limited their in-country operations to data collection which was then exported to the donor countries for analysis and publication, Rakai was committed to using and expanding the local capacity to conduct end-to-end research; capacity that existed, albeit in a depleted state, at the UVRI. Born in 1966, Lutalo joined the UVRI in 1988 after earning his bachelor's degree in statistics at Makerere University. Lutalo was raised in Kawanda, in the Luweero triangle north of Kampala, but grew up visiting his father's family in Kayabwe, about halfway between Kampala and Masaka on the main highway where it crosses the equator.[20] As a teenager, he heard stories of the slimming epidemic in Rakai and had seen some local cases.[21] When the armed conflict in the Luweero triangle escalated, Lutalo and his family relocated to Entebbe, to live with his grandfather and study at St. Mary's College where he met the man who would become his mentor and introduced him to statistics.[22] At the time, and while he studied statistics at Makerere, Lutalo saw statistical training as a way to escape Uganda. As he

[17] Sylvester Sempala, Uganda Virus Research Institute Annual Report No. 37, 1995, 1, UVRI Archive (UVRIA).
[18] Fred Wabwire-Mangen OH.
[19] Lutalo OH.
[20] Lutalo OH.
[21] Lutalo OH.
[22] Lutalo OH.

put it, "[At that time, what are you going to be doing? You are surrounded by wailings, people being killed, specially in the center [Central Region] here." While at Makerere he worked with Dr. Leonard Atuhaire who encouraged him to pursue biostatistics, a specialization that would require international training and which might provide opportunities to work with an organization like AMREF where his earliest mentor, Male-Mukasa, worked in Zimbabwe.[23] There was no opportunity for such training immediately, so Lutalo took a position with the Ministry of Planning and Economic Development in Entebbe. But he was dissatisfied with the lack of activity there and when he heard about an opportunity to train in medical statistics on a scholarship supplied by the WHO to the ACP, he inquired with Dr. Sempala, then director of the UVRI, who was a fellow member of the Entebbe Families Network.[24] The scholarship covered the expense of his master's degree in the UK on the condition that he return to Uganda. While his application for the scholarship was being processed he requested and received a transfer to the Ministry of Health and began working at the UVRI. Dr. Jane Kengeya-Kayondo put him to work organizing the Institute clinic's patient records and computerizing clinical records. At the time, the Institute was the only place in Uganda processing HIV tests and Lutalo taught himself how to use the computer procured by the ACP to operate the software package Epi Info and begin organizing the results of the tests.[25] When he returned to the UVRI in 1989 after finishing his master's degree, he was given the responsibility for processing the results of the Institute's ELISA tests—connecting the plate readers to the computers, calculating optical density cut-off values, and identifying positive tests.

Around the same time, the Rakai Project contracted with Rank Consult, a firm started by Atuhaire, Lutalo's one-time mentor, and his partners to handle epidemiological data and statistical analysis. Atuhaire encouraged Lutalo to work on programming the data analysis tools for the Rakai Project studies and soon Lutalo was spending more time on Rakai data than on the

[23] Lutalo OH.
[24] Lutalo OH.
[25] Lutalo OH.

UVRI's tests. Eventually, in 1991, Musgrave and Sewankambo requested that the Ministry of Health formally second Lutalo and another UVRI statistician/data manager, Joseph Mabirizi, to the project on a full-time basis.[26] As the STD trial began, he worked closely with Lainjo Bongs, the expatriate resident advisor and data manager.

The STD grant included a 2.1 million dollar five-year subcontract with the UVRI with Sewankambo and Serwadda as PI and co-investigator respectively. This subcontract was an important part of the efforts the UVRI was making to re-establish its research infrastructure. It included the salary of a to-be-named STD medical specialist as a co-investigator, the salary of Thomas Lutalo as the Ugandan Data Coordinator, four half-time physicians, a senior team supervisor, and a number of other team members including field supervisors, medical assistants, nurses, phlebotomists, nurse interviewers, drivers, health educators, and behavioral researcher workers. The subcontract also included budget items for field supplies, medications to treat basic non-STI diseases (antimalarials, oral rehydration therapy, vitamins, etc.), equipment, supplies, fuel, and "study household gifts".[27] In total the UVRI subcontract included salaries for 53 people, of whom most would be dedicated 100% to the project for a minimum of one year (several positions ranged from 30-50% effort in some project years). Through the UVRI, the Ministry of Health seconded two medical officers, two health educators, four medical assistants, four midwives, one health visitor, one senior nursing officer, and four nurses to the Rakai Project's STD study.[28] The proposal also listed the UVRI as the primary source of laboratory facilities for the project.[29]

Peopling the Project Staff

In addition to the UVRI's Tom Lutalo, the individuals responsible for most of the day-to-day operation of the trial were almost exclusively Ugandans trained at Makerere University. The

[26] Lutalo OH.
[27] "STD Control for AIDS Prevention, Uganda", 30.
[28] Sylvester Sempala, Uganda Virus Research Institute Annual Report No. 37, 1995, 9, UVRIA.
[29] "STD Control for AIDS Prevention, Uganda", 30.

Rakai Project gave them an opportunity to develop research skills that would launch them onto the international stage as well as a chance to work on a disease that had affected many of them personally. In addition to the staff seconded from the Ministry of Health through the UVRI, several of the physicians hired to fill management roles in the new study knew about the Rakai Project from knowing David Serwadda and Nelson Sewankambo at Makerere. Noah Kiwanuka was only a small child when his father Livingston Basuulwa, a District Chairman in Rakai, decided he should be a doctor. He was planning to pursue a career in cardiology or gastroenterology and surgery when, as a third year medical student, he was assigned to Nelson Sewankambo's ward, Ward B, in Mulago Hospital. Kiwanuka was impressed by Sewankambo's practice and dedication to hospital work and education.[30] Around that time he started observing the people bringing packages of vacutainers to Sewankambo and asked his mentor what they were for:

> I asked him, 'Where are these samples coming from?' And he told me they are coming from—that they have some research work they're doing in Rakai. And I told him, 'By the way, I come from Rakai.' And as soon as he said research, I knew it was about HIV because, you know, I had seen HIV from, I should say probably from the first few cases that broke out in Rakai. I said, 'Ah.' So I became a bit interested. At that time I'd been affected by HIV *a lot* because my dad died of HIV. My stepmother—I had two stepmothers, they died of HIV. I lost brothers, sisters, young, old, cousins, friends—even people I went to school with, same classes. I lost my classmates. So anybody who was doing something to control HIV would naturally come in very handy for me because I had seen the explosive part of the epidemic in my home district…When I finished med school and I went for internship, I specifically requested to be on his [Sewankambo's] ward. I went on his ward…And as soon as I finished internship, they had got this money to do the STD trial. So they advertised in newspapers that they were looking for a coordinator…I applied.[31]

As a 26-year-old newly minted physician lacking several of the qualifications the advertisement specified, such as training in epidemiology and management experience, Kiwanuka didn't believe he had much of a chance at the position. Sure enough, shortly after interviewing with Serwadda, Fred Wabwire, Konde-Lule, and senior study coordinator Lynn Paxton, he received word that the

[30] Noah Kiwanuka OH.
[31] Kiwanuka OH.

position had been offered to a candidate with greater experience. He was still contemplating his future when he received an invitation to accompany Sewankambo to Rakai for a week or two to assist with the STD trial training and a pilot study. It transpired that the woman from South Africa who had been offered the coordinator position had arrived but found that the location in Rakai was too remote and declined the position. So Kiwanuka was offered the position. As he said, "it was handy because it was my home district…I mean, I knew the people. I knew the community very well. I would get along with the community. And so that's how I was given the offer of being the field coordinator for the study." Kiwanuka speculated that Sewankambo had suspected the South African candidate might not be a good match for the rural district work and had prepared a backup plan.[32]

Other recruits had local connections to the project, such as Fred Makumbi. Makumbi completed his bachelor's degree in biostatistics with a concentration in development economics at Makerere in 1992.[33] At the time his family was living in Masaka where his father was the director of the Southwestern Integrated Project, a program engaged in several UNICEF-funded public health projects. Through this work he had met Joseph Ssembatya (one of the key players in the early recognition of the epidemic as described in Chapter 5), who passed Makumbi's CV to the Rakai Project administration. After an interview with anthropologist Janet Edmondson, who was initiating a new study, and Robert Ssengonzi, who had completed his degree in sociology at Makerere in the same year that Makumbi graduated and was hired by Edmondson a couple of months before Makumbi.[34] Initially Makumbi was hired to work in the field in Rakai doing data collection—particularly the census-taking and the collection and editing of surveys. The learning curve for fieldwork was steep. Everything from dress code, to choosing food for the field, to the etiquette for refusing the only chair in an interviewee's home, was learned on-the-job by making

[32] Kiwanuka OH.
[33] Fred Makumbi OH.
[34] Makumbi OH.

mistakes and listening to the older, more experienced field workers.[35] After working on the sexual networks study for two years, Makumbi was selected to join Lutalo and Steven Baryahirwa on the data team because of his statistics training. While most of the team was based in Kalisizo, the data team worked out of an office at the UVRI in Entebbe. But Makumbi, Lutalo, and Baryahirwa took turns traveling to Kalisizo so that there was oversight in the data collection and so that they would stay in touch with the conditions in which the data was being produced.[36]

Like Kiwanuka, many of the young physicians who came on to the project team with the STD study grant had grown up in Rakai. For them, AIDS was not simply a research problem. Their decisions to pursue medical careers and then to work in research were directly related to their personal experiences of loss and witnessing the devastation of AIDS in Rakai. As Makumbi put it, while rumors of slim disease had reached other parts of Uganda in the late '70s and early '80s, "It wasn't real. It wasn't real. Apart from those that had lived in Rakai—they knew HIV was real."[37] While David Serwadda and Nelson Sewankambo had first encountered AIDS as young physicians on the wards of Makerere, the younger physicians had seen slim take friends and family members starting in their childhoods.

One of the people Kiwanuka hired was Joseph Kagaayi, a recent graduate of Makerere University Medical School and another native of Rakai District. Born in Kasambya village of Kyotera Town in 1974, Kagaayi resided in the district until he finished primary 3 and then continued his education in nearby Masaka district where his father worked, before moving on to King's College Buddo in Kampala. He described knowing from a very young age that he wanted to be a doctor: "I think I knew I wanted to be a doctor at about four years. The reason was very simple. There was a lot of malaria when I was growing up and every month I used to be at the doctor's office. Every month I used to get injections for malaria. I was sick and tired of being sick

[35] Makumbi OH.
[36] Makumbi OH.
[37] Makumbi OH. Slim was the vernacular disease category that referred to the severe wasting or "slimming" experienced by AIDS patients.

and tired. I think I talked to my mom. I must've been 5 or so. I said, 'I think the doctors don't know what they're doing. How can I be sick every month?' I told her, 'You know, when I grow up, I think I'm going to be a doctor. I'm going to be much better than all these doctors.'"[38] But midway through medical school he started to focus on HIV instead. He had seen HIV as a child in the late '80s and witnessed first hand the scale of the losses it created in the district. By the time he left medical school he had decided to focus on pediatrics and accepted an internship at Rubala Hospital where he was mainly responsible for surgery and pediatrics. Once his internship was completed he took a position at Kalisizo Hospital in Rakai, almost adjacent to the offices of the RP. He had known of the Rakai Project from its earliest days—some of the team members in the first surveys had stayed in a hotel Kagaayi's father owned in Kyotera—the Las Vegas Inn.[39]

Kiwanuka also recruited Godfrey Kigozi, a classmate from medical training who was stationed at the government hospital in Gombe.[40] Kigozi had known and admired many of the senior scientists on the Rakai Project as instructors at Makerere: Serwadda, Sewankambo, and Wabwire.[41] Kiwanuka had been having some difficulty recruiting people to work in Rakai's rural environment, and he appealed to Kigozi's admiration for the senior scientists as well as his desire to work with children. Kiwanuuka wanted Kigozi to manage a sub-study nested within the larger STD trial—the Maternal-Infant Supplementary Study (MISS). The MISS trial was designed to measure the effect of treating pregnant women for STDs on the outcomes of their pregnancies and their risk of transmitting HIV to their infants.[42] Running the MISS trial, Kiwanuka persuaded Kigozi, would be the perfect opportunity to combine his interests in pediatrics and infectious disease.[43] In addition, like Serwadda, Kigozi was frustrated with the limited care he was able to

[38] Joseph Kagaayi OH.
[39] Kagaayi OH.
[40] Godfrey Kigozi OH.
[41] Kigozi, OH.
[42] Women were screened for pregnancy during enrollment in the STD trial and, if found to be pregnant, were enrolled in MISS and, if they were in the treatment arm, given a different combination of drugs that was recommended for pregnant women. In the control arm they received supplements including iron and folate. Ronald H. Gray, Fred Wabwire-Mangen, Godgrey Kigozi, et al., "Randomized trial of presumptive sexually transmitted disease therapy during pregnancy in Rakai, Uganda," *American Journal of Obstetrics and Gynecology* 185, no. 5 (2001): 1209-1217.
[43] Kigozi, OH.

deliver in the hospital and thought that research might allow him to make a more substantial contribution to the fight against AIDS.[44]

The rapid scale-up of the Project's operations entailed a few growing pains. Kiwanuka and Kigozi were very young and had little or no experience in either administration or research when they joined the Rakai Project. They learned as they went how to manage staff, deal with unexpected events, and ensure compliance with all of the study protocols. While the total project staff during the HIV Dynamics study had been approximately 40 people, the STD trial employed around 150.[45] Standard Operating Procedures (SOP) were entirely new to many employees. Even people who had been with the project before the STD study had to adjust to modifications "to match the rigor of the trial."[46]

They were also learning the basics of clinical trials research. It wasn't until the STD trial was well underway that Kigozi and Fred Nalugoda, another young member of the study team who had joined the team as a research assistant in 1992 and was promoted to field supervisor for the STD study, had the opportunity to do their masters training in the United States. Many of the new hires were working on a research project for the first time and weren't aware of all of the procedures, despite thorough and evolving training activities. That is part of the reason that the team also included a small group of expatriates who were expected to ensure that the study protocols were followed and that all practices met the standards of the funders and the American partner institutions. The project had American laboratory coordinators throughout the trial who supervised the local lab manager and staff.[47] The trial as a whole was supervised on site by Lynne Paxton, an American medical doctor who served in the capacity of technical advisor.[48] Occasionally the role of expatriates and their place in the chain of command for trial operation

[44] Kigozi OH. See description of Serwadda's frustration in Chapter Five.
[45] Makko Musagara OH.
[46] Kiwanuka OH.
[47] Denise McNairn was the first laboratory coordinator on the study and was succeeded by Mary Meehan in 1996. Opendi Ochala OH.
[48] Fred Nalugoda OH; Betty Nantume OH.

created tensions and in subsequent projects their role was substantially reduced.[49] But during the STD trial they also represented avenues of connection between Uganda and the professional development resources of the United States for junior researchers.

Mapping and Making Communities

The first task of the STD study team was defining the units of randomization—in this case the communities of individuals and households. In the course of designing and implementing the STD study, the Rakai Project systematically remade the Rakai District, designating communities in order to fit them into an experimental regime. These designations had material consequences for the people living within the study area.[50] Though the project was conducted at the behest of senior researchers based in Kampala, Baltimore, and New York, the work of establishing these boundaries was done by the Kalisizo-based field workers and it was their knowledge and understanding of local travel patterns, living arrangements, and relationships that informed the myriad decisions about who belonged to which community and who was excluded.

In total, the project was designed to enroll 10,400 individuals. Half of them lived in 29 treatment communities and half in 29 "equivalent communities" constituting the control group.[51] These communities were further organized into 10 "superclusters" of five to six communities each.[52] Each supercluster was either composed entirely of treatment communities or of control communities. Between each supercluster was a "buffer zone" intended to ensure that there was little or no cross-contamination of treatment and control groups [Figure 2].[53]

While this is all described as quite straightforward in the grant application and subsequent publications, these communities were not pre-existing, self-evident entities. The

[49] Serwadda OH.
[50] Angela Last, "Experimental Geographies," *Geography Compass* 6, no. 12 (2012): 706-724.
[51] "Rakai STD Control for AIDS Prevention Project, Project Summary," 100 Years: The Rockefeller Foundation, accessed April 22, 2013, http://rockefeller100.org/items/show/4676. Rockefeller Archive Center, Rockefeller Foundation records, unprocessed, box R3106, folder RF 91079 A31.
[52] "Rakai STD Control for AIDS Prevention Project, Project Summary."
[53] Lutalo OH.

Rakai Project team had to define them, to invent them even. The LC1s existed independently of the research program, but all of the other units of organization used by the study (communities, clusters, superclusters) had to be defined by them. Some communities consisted of several LC1s—others were only one LC1 or even a portion of an LC1 if a large LC1 was divided by a highway or another significant barrier.[54] In order to plan the STD study, the Rakai Project PIs decided that they needed to get a comprehensive picture of the district as a whole. They considered commissioning aerial photographs of the district to get a bird's eye view, but decided they couldn't afford it. Instead, Gray, Wawer, Serwadda, Sewankambo, and Nalugoda spent several weeks driving all over the district and noting the different types of villages, the types of homes and businesses that they contained, the ease or difficulty of traveling between them, and they linked those observations to the serological data they had already collected. For example, they knew that in the earlier surveys, villages with a certain number of bars or shops had HIV prevalence in a certain range, so they extrapolated those rates to the villages where no prevalence data was available.[55] Global positioning system (GPS) technology wasn't widely available at the time, so they would use the odometers in the vehicle to measure distances and note them on hand-drawn paper maps along with churches, bars, lodges, major road intersections, and large markets.[56] Local leaders would be consulted about the limits of their villages, but sometimes it wasn't entirely clear how far from the road households should be included. Often it came down to the practicalities of research: "It was a question of how far teams could walk off-road carrying everything they had to carry and how much time it would take to reach remote households."[57]

Nor could every village be included in the STD study. The power calculations that measured the number of participants that would be required to show a particular size effect of the intervention meant that the trial needed to maximize the number of communities that had

[54] Lutalo OH, Nalugoda OH.
[55] Nalugoda OH.
[56] Ronald Gray OH.
[57] Gray OH.

sufficiently high HIV incidence and where the outcomes of interest (new HIV infections) would be common enough but not so high that the effect of the intervention would be overwhelmed.[58] For example, one of the fishing villages where some of the first AIDS cases were detected had a 55% HIV prevalence. The PIs were afraid that with such high prevalence already, the impact of STD treatment would be impossible to measure.[59] So the team looked for villages with a prevalence between roughly 5% and 30%.[60] Finally, they had to determine which villages should be grouped together into either intervention or control clusters. They wanted to minimize the amount of interaction between people in the two arms of the study. As Wawer put it, "just looking at a map you couldn't tell…whether Community X had primary social and presumably sexual contacts with Community Y…because there could have been a swamp in between, there could have been a hill…Or, you know, maybe they were Bakiga or Banyarwanda so they…had less contact with the Baganda or clans or whatever."[61] So they interviewed people asking, "Where do you go to socialize? Say, when you have a festival or go for a disco? Where do you go for marketing? Where do you go to sell your stuff? Where are the schools that your children go to?"[62] Villages where people gave overlapping responses were grouped together.

All in all, people in Rakai were presumed to have extremely limited mobility. Wawer observed that during their first survey in the district, only one household in the entire study reported owning a motorized vehicle.[63] Bicycles were more common, but certainly not ubiquitous and, as Wawer put it, "even then you know you're not going to cross a mountain and a swamp with your bicycle to find somebody to fall in love with. You're going to fall in love with

[58] Nalugoda OH.
[59] Nalugoda OH.
[60] Nalugoda OH.
[61] Maria Wawer OH.
[62] Nalugoda OH. Wawer gave very similar examples of the questions they would ask people in what she called "ad hoc focus group" sessions. Wawer OH.
[63] Wawer OH.

somebody…within walking distance."[64] Or, as Gray put it, "basically, relationships extended as far as it was worth walking."[65]

Then and now, the word that Rakai Project staff most commonly use to designate the units of study for their population-based trials is "community". The staff of the Rakai Project use the word "community" in both a formal and a more casual way. It can designate the particular unit created for the purposes of the STD study and maintained in the Rakai Community Cohort Study (RCCS). But members of the project staff also refer to non-project staff in general as "the community". Sometimes it refers to the total population of the cohort or even to the population of the district that the cohort attempts to represent.

The most deliberate use of the word, however, is to designate the third-smallest unit of the population-based study (after individual and household). As I discussed in chapter five, the communities were inventions of the study team, meant to reflect real patterns of social interaction and closely related to political organization in the LC system, but fundamentally a creation of the research project. I asked Fred Nalugoda whether people in neighboring villages whom the study grouped together into a single community would have understood themselves as sharing a community or not. He told me, "They would identify themselves with a village, like the LC. And they would tell you the leader of the other village is a different one. If you go in with another leader, they say, 'Oh no. For me I stop here.' And then we say, ok, for purposes of our study, let's go together. We are going to get the other one [village leader] as well because for us in the study we take this as one. And they were able to understand that."[66] One of the community mobilizers, a resident of one of the study communities, who helped the Project draw the map of his community explained that an outsider might not be able to identify the limits of the village:

> It is a bit impossible [for a stranger to know the boundaries] because there is no definite line that it's from this line you cross to another area, but there are villages that this is a certain village if—this is a village and this is a village, but

[64] Wawer OH.
[65] Gray OH.
[66] Nalugoda OH.

> here between the two villages there is a gap which people identify that from village when you go to the next village, you're in another area.[67]

Musagara agreed that the boundaries of villages tended to be demarcated in terms of natural boundaries, such as hills, rivers, or forests, and that to an outsider, those boundaries might be impossible to recognize. Only a local informant, such as the LC chairperson, could tell the researchers where each village ended.[68] In the end, the size of clusters ranged from four villages to more than 20 villages.[69] The stakes were high for these determinations; failure to correctly understand the social geography of these communities could lead to cross-contamination between treatment and control communities and nullification of the trial results.

Mapping the social geography of Rakai in this way was a tool for making visible the etiology of AIDS. Up until this point, the mapping exercise had been mainly conceptual—to determine which villages could be included in the study for practical purposes and how they might be grouped.[70] Once a set of villages had been grouped into communities and then further grouped into clusters, the more concrete mapping began. Teams of data collectors performed community surveys, noting the presence of hospitals, shops, bars, schools, markets, hotels, mosques, and main roads each one contained [Figures 3, 4, and 5].[71] Like Haddow's maps of Bwamba, the Rakai Project's maps were instruments for the creation of an experimental space that included and privileged certain aspects of the physical, natural, and social geography and excluded others. Unlike the Bwamba studies, however, the Rakai Project's maps created communities that the residents of the district were very much aware of and experienced as having a material effect on their well-being.

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[67] Anonymous 1 OH. *"Ekyo tekisoboka, olwensonga nti tetuyina* line*, kubanga bagamba nti gundi abeera [*redacted*], gundi abeera [*redacted*]—naye nga awo wakati watera okubaawo akabangirize,* like a gap. For instance *okluva wano okutuuka wali ku* trading center *wotandikidde—awo wosanze omuzikiti bayita wo lugamba n'ente.* Then *ate wano bayita wo [*redacted*], ate wali wosaanze ekubo bayita wo [*redacted*]."*

[68] Musagara OH. I know of no instances in which these designations were a source of explicit conflict between local informants with different answers to these questions, but it seems likely that issues of political influence and land title may have been at play in ways the researchers were not aware of.

[69] Nalugoda OH.

[70] Nalugoda OH.

[71] Nalugoda OH.

Implementing the Study: Working and Living on the Map

With the personnel in place and the maps drawn, the trial itself launched. The trial required a diverse set of practices and the labor of dozens of individuals, though the details of this were typically condensed into brief methods sections in final publications. While historians and ethnographers have placed a great deal of emphasis on the expatriate-operated research projects, the accounts of Rakai Project staff confirm that their study design, as well as its implementation, was largely the product of indigenous expertise. Before each survey round, Lutalo recalled, he, Kiwanuka, the Kigozis (Godfrey Kigozi met and married his wife, Grace, when she was a data editor on the trial), Gray, Wawer, and others would meet in the boardroom at the UVRI and work on the questionnaire. Someone would propose a question and the group would discuss whether it should be included, how it should be phrased, whether it should be split into multiple questions, what kind of variable it would be coded as, etc.[72] Even simple changes had complex consequences with changing skip patterns, making sure that variables were defined consistently between rounds, and that multiple types of data were coded appropriately.[73]

In Rakai mass treatment consisted of the administration of "a highly effective, single oral dose therapeutic regimen" of azithromycin, ciprofloxacin, and metronidazole to all consenting adults in the treatment communities.[74] Two groups received a modified treatment regimen: individuals who had positive syphilis serologies were also treated with intramuscular benzathine penicillin (the standard of care for syphilis at the time) and pregnant women received an oral dose of azithromycin and cefixime instead of the standard oral treatment, supplemented with intramuscular penicillin injections where indicated.[75] In order to keep the study single-blinded (so that participants did not know whether they were in the treatment or control arm), members of the

[72] Lutalo OH.
[73] Lutalo OH.
[74] "Rakai STD Control for AIDS Prevention Project, Project Summary."
[75] "Rakai STD Control for AIDS Prevention Project, Project Summary."

control communities received oral doses of antihelminths and multivitamins. Individuals with syphilis in both the intervention and control communities received appropriate treatment.

In the beginning, recruiting and enrolling study participants was challenging. In the earliest years of the Rakai Project, people actively avoided contact with the survey team. Project staff related anecdotes about people running away from project vehicles and making sure to vacate their homes on the days the study team was due to visit.[76] Nor was suspicion the only impediment to smooth implementation. As Kiwanuka described it, "Rakai was very remote—a very remote district. You can imagine what Rakai is now—imagine in 1994. Terrible. There was no tarmac roads anywhere in the district. If you had to make a call, you had to drive to Masaka post office to make a call to Entebbe or to Kampala. There were no facilities to make a telephone call anywhere in the district. It was really remote."[77]

Despite these obstacles, by the end of May 1995, the study had enrolled 8700 individuals into the control and treatment arms of the study. In the two arms combined, a little over 17 percent of participants were HIV-positive at the time of enrollment (18% in the treatment arm and 17.4% in the control arm).[78] By August, the team had completed enrollment, sample collection and interviews for the whole cohort with 88.1% of eligible and present residents consenting to participate and receiving treatment (either the intervention treatment or the control treatment).[79] Nested within the main study, to assess the relationship between STD control and HIV incidence, were two sub-studies: a study of the effect of mass treatment on vaginal flora and a study of the effects of mass treatment on infections in pregnant women and the outcomes of their pregnancies—the maternal-infant supplementary study (MISS).[80] Overall, 95% of the pregnant

[76] Nalugoda OH; Kiwanuka OH.
[77] Kiwanuka OH.
[78] "Rakai STD Control for AIDS Prevention Project, Project Summary."
[79] Nelson Sewankambo, Fred Wabwire-Mangeni [sic], David Serwadda, and Lynn Paxton, "RAKAI – PROJECT: Columbia University, Johns Hopkins University, Makerere University and UVRI Collaborative Project," in Sylvester Sempala, *Uganda Virus Research Institute Annual Report No. 37*, 1995, 45, UVRI archive.
[80] Sewankambo, Wabwire-Mangeni, Serwadda, and Paxton, "RAKAI – PROJECT," 44.

women accepted treatment (either in treatment or control arms) and 90% of the women with syphilis agreed to be treated with penicillin.[81]

Preliminary results after the first round of testing and treatment indicated that the prevalence rates of STD and HIV were approximately as predicted and that the randomization appeared to have successfully resulted in similar treatment and control groups. 20% of the women in the study were HIV-positive and 16.4% of men were infected with HIV. Rates of syphilis were 12.2% and 12.5% for men and women respectively. In the first year of the MISS, 17% of the women enrolled in the STD control study between the ages of 15 and 49 were pregnant. 19% of those pregnant women were HIV-positive and17% were infected with syphilis. Everything seemed to be proceeding according to the plan and the team began the second round of treatment and data collection the following month.

The maps made by the study team to organize the study communities had real consequences for people living in Rakai. By the time the STD study was well underway, the Rakai Project occupied a different position in the public imagination in the district. One of the residents of the District, who has acted as a mobilizer for the Project since 1995, described the perceived benefits of participating in the research, benefits available only to those living in households "on the map":

> [T]hese people outside the map were given only treatment for fever but these people in a map were being drawn off blood, swabs were taken and even other care yet those people outside the map could not get such services, so they also wanted to access services like being blood drawn off, swabs taken and urine taken but they never had such chances—but they were only giving treatment to fever all other diseases but these services they didn't get them.[82]

People that I spoke with rarely distinguished between treatment for HIV (antiretroviral therapy), treatment for opportunistic diseases, and treatment for non-STIs. While antiretroviral treatment for HIV infection was not available in Rakai at the time of the study, the Project did operate both

[81] Sewankambo, Wabwire-Mangeni, Serwadda, and Paxton, "RAKAI – PROJECT," 47.
[82] Nsubuya Harriet OH. *"Abali wabweru wa maapu tebabajangako musaayi naye nga babajanjaba endwadde ezibaluma, kati bano abali mu maapu, twatolebwangako omusaayi, muulo, bupamba naye nga bali abatali mu maapu nabyo babyagala naye nga tebalina mukisa ogwo, naye nga webaba balwadde musujja babakolako."*

a mobile and a fixed-site clinic for trial participants and a separate clinic for staff.[83] Study participants who reported genital ulcers, whether in the treatment or control arm, were referred to the study clinic where they were examined by a physician and treated.[84] In the rest of the district, however, as in Uganda nation-wide, access to quality care for STDs was extremely limited.[85]

The design of the study as an open cohort—one in which people migrating in and out of the geographically-defined study area were added and subtracted from the cohort—meant that for some Rakai residents, periods of their life were marked in terms of whether or not they were "in the map." For example, one woman recalled, "when she was still at mum's place she was in a map, and when she was with the husband she was still [inside the boundary], when she divorced with the husband … she was still [inside the boundary]. Now like the neighbor is not in the map and they don't work on him."[86] A few years after the study, the Rakai Project attempted a formal evaluation of the perceived benefits or costs of participating in research. They surveyed research participants, people who had declined to participate in research, and individuals identified as "opinion leaders".[87] 85% of their respondents reported believing that the Rakai Project research had personally helped them "a great deal" and 88% believed that it had delivered benefits to their community.[88] The benefits they cited were improvements in the local economy, the expectation of future health benefits, improved health knowledge, and access to project-sponsored medical services.[89] By the early 2000s, shortly after the STD trial ended, being "on the map" with the Rakai Project was regarded as a positive by many Rakai residents.

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[83] Sewankambo, Wabwire-Mangeni, Serwadda, and Paxton, "RAKAI – PROJECT," 47-48.
[84] Kiwanuka OH.
[85] Bart Jacobs, James Whitwoth, Fred Kambugu, et al., "Sexually transmitted disease management in Uganda's private-for-profit formal and informal sector and compliance with treatment," *Sexually Transmitted Diseases* 31, no. 11 (2004): 650-654.
[86] Anonymous 3 OH. *"Zikyaali ze zimu kati tolaba nze gyenava eri ewa mama, e wa mwami nali natumbulira mu nsalo era nga wetuli, ewa mama wange ne ntambulira mu nsalo eyo nga nnobye mu 2000 nenzija ne nzimba wano mu poloti yange era nga tukyatambulira mu nsalo, wabula nze nali nasaba busabi nti singa oba yeyongera yo, kuba tollaba oli* neighbor *wange eba temusika yo, naye nze eba entambuza kuba nava mabega naki, nayo. Waliwo n'abakyala abalala twali tubakolako nga bapangisa eri ku bbibiro naye kati baziimba maanju wange wano, era nabo baba tebabasika yo."*
[87] Carrie Thiessen, Robert Ssekubugu, Jennifer Wagman, et al., "Personal and Community Benefits and Harms from Research: Views from Rakai, Uganda," *AIDS* 21, no. 18 (2007): 2493-2501.
[88] Thiessen, Ssekubugu, Wagman, et al, "Personal and Community Benefits," 2496-2497.
[89] Thiessen, Ssekubugu, Wagman, et al, "Personal and Community Benefits," 2497.

Results and Controversies

In 1995 the team from Mwanza published the results from their trial. Like Rakai, Mwanza's study was designed as a cluster-based community-randomized trial. Using a sampling strategy based on the Tanzanian government's ten-cell organization, similar to the LC organization in Uganda, they selected 5 pairs of matched clusters divided into three strata: urban, roadside, and rural.[90] In a *Lancet* article they reported a 40% reduction in HIV incidence among the population given access to improved STD treatment protocols as compared to the control group.[91] These results were even more significant because they came at a time when funding agencies were losing faith in the power of behavior modification and heath education to control the epidemic and discussions of "donor fatigue" predicted growing challenges for sustaining programs. In this context, the Mwanza authors concluded their study with the following prediction: "The demonstration that HIV incidence can be almost halved by a modest intervention in one of the world's most disadvantaged countries should provide a message of hope, and help stimulate renewed efforts to control this epidemic throughout the developing world."[92]

Given that Rakai expected to have a greater impact on STD rates with their mass treatment strategy than the Mwanza group had with improved syndromic management (which didn't include treatment for people without symptoms), they anticipated finding somewhere between 50% and 80% reduction in HIV incidence.[93] There was even some discussion of terminating the Rakai and Masaka trials because the size of the effect found in Mwanza was sufficient to justify program investment and it might be unethical to deny such an effective intervention to the control groups.[94] However, the different design of the trials and the

[90] Longin R. Barongo, Martien W. Borgdorff, Frank F. Mosha, et al., "The Epidemiology of HIV-1 Infection in Urban Areas, Roadside Settlements and Rural Villages in Mwanza Region, Tanzania," *AIDS* 6, no. 12 (1992): 1521-1528.
[91] Heiner Grosskurth, Frank F. Mosha, James Todd, et al., "Impact of Improved Treatment of Sexually Transmitted Diseases on HIV Infection in Rural Tanzania: Randomised Controlled Trial," *Lancet* 346, no. 8974 (1995): 530-536.
[92] Grosskurth, Mosha, Todd, et al., "Impact of improved treatment," 535.
[93] Wawer OH.
[94] Wawer OH.

populations in which they were implemented as well as the substantial resources already invested in the trials convinced NIH and MRC who were funding the two ongoing trials to let them continue.[95] In a letter responding to the article reporting Mwanza's results, three NIAID officers pointed out that the Mwanza study alone didn't permit cost-effectiveness calculations and that a mass treatment protocol might prove to be both more cost effective and effective in terms of comprehensive population coverage. As they concluded, "it is difficult to interpret conceptually and practically the sum of effects of improving the diagnosis and treatment of STDs on the incidence of HIV infection, and impossible to assess the relative efficacy without mass STD treatment trials results.[96] Other letters mentioned other concerns about the Mwanza study results such as the lack of a measurable impact on STD rates, failure to account for different baseline HIV prevalence between the control and treatment arms, and the feasibility of scaling up what appeared to be a relatively resource-intensive protocol.[97]

With such high expectations, the revelation that there was absolutely no difference in HIV incidence between Rakai's control and treatment arms, including the MISS, and that the trial would be terminated in January 1998 came as a serious blow. Wawer's description of her initial reaction was echoed by a number of team members: "[Y]ou just freeze. Your blood runs cold…you're not even disappointed. You're just beyond shock."[98] While Serwadda recalls disappointment but not doubt in the integrity of the results, the first response of many team members and external observers was the same—there must have been a mistake. The Rakai team began checking every element of their data collection, entry, and analysis; reviewed their protocols and procedures; and tried to consider every possible way that their study might have been compromised. But eventually they concluded that the study had been a success—mass

[95] Wawer OH. The decision to continue the trial was made by the study's Data Safety and Monitoring Board. Email communication from Ronald Gray, February 24, 2017.
[96] Mary A. Foulkes, Wasima N. Rida, and Rodney Hoff, "Impact of Improved Treatment of Sexually Transmitted Disease on HIV Infection," *Lancet* 426, no. 8983 (1995): 1158.
[97] Tarjei Ryngnestad, Lars Smabrekke, Lars Nesje, et al, "Impact of improved treatment of sexually transmitted disease on HIV infection," *Lancet* 426, no. 8983 (1995): 1157-1159.
[98] Wawer OH.

treatment for STDs simply didn't have any impact on HIV incidence. As Wawer put it, "[T]his wasn't a failed trial; a failed trial is if you can't get your enrollment up or your loss to follow-up is horrific—if the data are un-interpretable. That's a failed trial. But this was just a negative trial. The results were crystal clear—nothing."[99] But nothing was a disappointing finding. As Sewankambo described the experience, "When you try to test out a concept, much as you are a researcher, you also hope for the best from them. If you started out to test whether this intervention works, you are hoping that indeed, this will work."[100] And, as discussed above, the Ugandan researchers had very personal stakes in the search for an effective intervention.

For the more junior researchers, the "middle management" the results were even more devastating. Noah Kiwanuka's memories of the end of the study, almost twenty years later, are visceral:

> It was a big disappointment. Those are some of the […] things you don't want to reminisce about. At first, I didn't believe it. When the message was communicated to us as a study team, I thought something was wrong with the statisticians. That's what I thought —they had to look at these data again. And, you know, on top of what the results really mean, when they told us what the results were, the first question we asked ourselves, Was this worthy of our effort? You look at all the effort you've put in, all the sacrifices you've made over and above what you are required to do as your own paid job. Because, I can tell you, everybody did beyond what our JD [job description] was. Everybody in the field office did over and above what the program was paying for. If the program was paying us for what we were doing in the STD trial, their personnel budget would have doubled at the very least. We put in a lot and sacrificed a lot to make sure that that study is well done. Now for the results to come out and say there is no difference, it was as if we had done nothing. That was the feeling that went through me. And remember, I'm naïve in public health and research. And so I'm not even aware that results can be — can come as you expected, can come out differently. They can come out equivocal. I mean these — there are all these possibilities — positive, negative, and equivocal results. I was like, Why did I sacrifice all that much? So then I started wondering, was there something — are there any flaws that we as a study team made? There's a level of disappointment that leads you to start condemning yourself or looking into whether you are to blame for what happened. I remember the — like for four days after I'd known the results, I almost fell sick. I didn't have the energy to work anymore. I kept thinking about these things at night. I was staying with my little sister and she really noticed that I wasn't sleeping well. I started thinking of all the things that could have gone wrong. I was just like, But we resolved that. But we resolved

[99] Wawer OH.
[100] Sewankambo OH.

> that. But we had that protocol. Why? You know, you have this feeling of why? What is the explanation? Then there was the daunting task of communicating to the research team itself. Because, you know, the PIs had communicated to us, the middle management. We knew the results. We had a meeting in Entebbe and Kampala. And then we had to communicate to the team. After that, we had to also disseminate that to the communities. I started — at that time, I actually thought, Should I continue working here or not? I was like, all this effort and these things turned out the way they did.[101]

Godfrey Kigozi was less adamant about the disappointment of the results, but recalled great sadness that what appeared to be such a powerful tool in the fight against AIDS was ineffective.[102]

These negative results did not go uncontested. The WHO and other organizations doing HIV and STD work had already committed significant resources to STD treatment programs in the name of HIV prevention. The global STD/AIDS community was reluctant to rethink what had become certainty about the efficacy of STD treatment for HIV prevention and to roll back the programs that they had already implemented. As Gray recalled, while hypothetically investigators and the scientific community in general were supposed to keep an open mind, the rationale for the hypothesis that STD control would reduce HIV infection was so persuasive that they couldn't accept Rakai's results.[103] A letter published in *Lancet* in May 1999 from one of the authors on the Mwanza trial suggested that Rakai's results were probably due to reinfection between the treatments and that "The results of the Rakai study…must not be used as an excuse to cut back on the resources made available for STD control."[104] The doctors in Rakai were certainly not advocating that STD treatment, whatever the mode of delivery, shouldn't be made available—they simply hadn't found evidence that it made an impact on HIV transmission at a population level. They did not, however, contest the assertion from the Mwanza team that "Adequate public

[101] Kiwanuka OH.
[102] Kigozi OH.
[103] Serwadda OH.
[104] Angus Nicoll, Anne Johnson, Michael Adler, and Marie Laga, "Correspondence: Preventing HIV-1: Lessons from Mwanza and Rakai," *Lancet* 353, no. 9163 (1999): 1522.

health services for the diagnosis and treatment of STDs should be regarded as a basic right, and not as a 'magic bullet'."[105]

Researchers involved with the Rakai project at the time the study results were released remember it as a period of challenges from the larger HIV/STD community at the same time that it affirmed the confidence the group had in itself. The conflicting results of the two trials were reported on the continent as well as in the major professional journals.[106] The overwhelming reaction was skepticism. One Kampala-based AIDS doctor was quoted in the newspaper *The East African* saying, "It is dangerous and much too early to conclude as a result of the Rakai study that STDs do not facilitate the transmission of HIV".[107] The article reported his further claim that "no credible scientific evidence had been presented by Wawer and her colleagues to rebut the results of the Mwanza study."[108] The benefit of the doubt didn't extend to the Rakai Project until the results of the third major trial, the MRC's trial in Masaka, were released in 2003.[109] In their trial of a combination of syndromic STD treatment and behavioral interventions, the MRC also found no difference between the treatment and control arms.

Several efforts were made to reconcile the apparently contradictory results of the three trials. One explanation was the differences between the populations of the three study sites. While all three studies routinely described their study populations as rural African or rural East African, closer analysis of baseline STD rates and the epidemiology of the HIV epidemic revealed significant differences between the Ugandan sites and the Tanzanian site. For one thing, the two Ugandan sites had significantly higher baseline HIV rates than the Tanzanian site. It was proposed that Uganda's AIDS epidemic was at a different "stage" than the Tanzanian epidemic

[105] Richard Hayes, Heiner Grosskurth, and Gina ka-Gina, "Authors' reply," *Lancet* 346, no. 8983 (1995), 1160.
[106] "Uganda: Twist in Uganda HIV Study," *New Vision* January 29, 1999, http://allafrica.com/stories/199901290097.html, accessed January 2, 2017; Peter Masebu, "Africa: Confusion Over Sexually Transmitted Infections Control," *Panafrican News Agency*, http://allafrica.com/stories/199910200006.html, accessed June 5, 2014.
[107] V.A. Moss quoted in D. Kimani, "Uganda: Scientists Clash Over Uganda STD-Aids Study," *The East African* June 2, 1999, http://allafrica.com/stories/199906020116.html, accessed February 21, 2017.
[108] Kimani, "Uganda: Scientists Clash".
[109] Anatoli Kamali, Maria Quigley, Jessica Nakiyingi, et al., "Syndromic Management of Sexually-Transmitted Infections and Behaviour Change Interventions on Transmission of HIV-1 in Rural Uganda: A Community Randomized Trial," *Lancet* 361, no. 9358 (2003): 645-652.

and that STD treatment became less effective as an epidemic "matured".[110] Subsequent analyses of the combined results of all three trials and their methodologies were more sympathetic to the Rakai Project than the initial responses to their findings. Some commentators went so far as to suggest that the Rakai study, not the Mwanza one, should be the "gold standard" and that the public health community at large had been overhasty in adopting "a hastily built orthodoxy—derived from the results of the Mwanza trial".[111] As a WHO scientist and team coordinator put it, the Mwanza results, seeming to indicate that there was a "magic bullet" for HIV prevention, came "at a time when the public health community needed hope."[112] But the accumulation of evidence from Rakai, Masaka, and subsequent studies dashed that hope.[113]

Nonetheless, as Wawer said, the STD trial was not a failed study. While the primary outcome of interest didn't materialize, the study revealed a number of significant findings. For one thing, the MISS concluded that pregnant women were more at risk for HIV infection than non-pregnant women, a very significant finding in a country with fertility rates as high as Uganda's.[114] But perhaps the most high-profile finding to come out of the study, both in the long- and short-term, was the observation that viral load (the number of copies of the HIV virus in a specimen) was correlated with risk of HIV transmission. In the secondary analyses that followed the un-blinding of the study, researchers at the Rakai Project and Johns Hopkins retroactively identified 415 discordant couples, or individuals with different serostatuses who had reported one another as sexual contacts, in the study population. They used stored serum specimens to test the

[110] Heiner Grosskurth, Ronald Gray, Richard Hayes, et al., "Control of Sexually Transmitted Diseases for HIV-1 Prevention: Understanding the Implications of the Mwanza and Rakai Trials," *Lancet* 355, no. 9219 (2000): 1981-1987.
[111] Christopher P. Hudson, "Community-Based Trials of Sexually Transmitted Disease Treatment: Repercussions for Epidemiology and HIV Prevention," *Bulletin of the World Health Organization* 79, no. 1 (2001): 48-58; Kevin R. O'Reilly and Antonio Carlos Gerbase, "STI Care: One of Many Necessary Approaches for Prevention of HIV Infection," *Bulletin of the World Health Organization* 79, no. 1 (2001): 58-59.
[112] O'Reilly and Gerbase, "STI care," 58.
[113] Trials of STD treatment for HIV prevention among commercial sex workers in Nairobi and in a sample of the general population in eastern Zimbabwe also found no effect of STD treatment on HIV incidence. Rupert Kaul, Joshua Kimani, Nico J. Nagelkerke, et al., "Monthly Antibiotic Chemoprophylaxis and Incidence of Sexually Transmitted Infections and HIV-1 Infection in Kenyan Sex Workers," *Journal of the American Medical Association* 291, no. 21 (2014): 2555-2562; Simon Gregson, Saina Adamson, Spiwe Papaya, et al., "Impact and Process Evaluation of Integrated Community and Clinic-Based HIV-1 Control: A Cluster-Randomised Trial in Eastern Zimbabwe," *PLoS Medicine* 4, no. 3 (2007): 545-555.
[114] Serwadda OH.

viral loads of these individuals and found that the viral loads of positive partners in couples that became concordant over the course of the study (i.e. the negative partner became infected with HIV) were significantly higher than the viral loads of positive partners in couples that remained discordant.[115] In a nutshell, "The viral load is the chief predictor of the risk of heterosexual transmission of HIV-1, and transmission is rare among persons with levels of less than 1500 copies of HIV-1 RNA per milliliter."[116] This finding would go on to be the basis of a new strategy for managing HIV: treatment as prevention (TasP).

But the most immediate response to the viral load article, published in the *New England Journal of Medicine* (*NEJM)* in 2000, was not enthusiasm; it was a pointed critique in the form of an editorial by the journal's senior editor, Marcia Angell. Published in the same issue as the original article, Angell's editorial suggested that the Rakai Project had committed a grave breach of ethics by failing to inform HIV-negative individuals that their partners were HIV-positive and by failing to deliver ARV therapy to the infected members of their cohort.[117] No one I spoke with at the Rakai Project recalled any of those concerns being raised during the oversight of the trial or the peer review process for the article. They had not been alerted ahead of time that such a critical editorial would accompany the article and were not given an opportunity to respond to the claims Angell made before the editorial appeared. Gray called it "one of the low points of my professional life."[118] Wabwire-Mangen recalled receiving "hate mail" after the editorial appeared, accusing him and others of violating human rights and victimizing hapless African study subjects.[119] Preparing to present data from the MISS at the International AIDS Conference in

[115] Thomas Quinn, Maria Wawer, Nelson Sewankambo, et al., "Viral Load and Heterosexual Transmission of Human Immunodeficiency Virus Type 1," *New England Journal of Medicine* 342, no. 13 (2000): 921-929.
[116] Quinn, Wawer, Sewankambo, et al, "Viral Load," 921.
[117] Marcia Angell, "Investigators' Responsibilities for Human Subjects in Developing Countries," *New England Journal of Medicine* 342, no. 13 (2000): 967-973.
[118] Gray OH.
[119] Wabwire-Mangen OH.

Durban that year, Wabwire-Mangen said he was "terrified…people would throw rotten tomatoes at me."[120]

The controversy hinged on what the RP maintained was a misunderstanding of their study methodology—the identification of discordant couples. As Gray reiterated in his response to Angell's editorial, the couples were identified only retrospectively. At the time of the trial individuals were not linked to one another. Moreover, disclosing the HIV status of any person to a third party without the patient's consent, or even revealing that patient's status to him or her without consent, was explicitly prohibited by Ugandan law. Finally, like in most sub-Saharan countries at the time, there was no infrastructure or capacity to deliver ARV therapy, either privately or publicly.[121] While the project team was confident that the ethical charges made against them were unjustified, the incident raised their awareness of the level of scrutiny being applied to international research projects in Africa and the need to be proactive in managing the perception of their work and the way it was communicated.[122]

Conclusions

The process of making Rakai, and Uganda more broadly, into a place that produces valuable information about viruses is ongoing. Not unlike the West Nile Burkitt's lymphoma study, the STD trial in Rakai is one that researchers don't think could be reproduced because of the change in the mobility of people in the study area. Gray observed, "we could not do the STD trial today, because there's so much mobility."[123] The ability of the researchers to identify geographic groups of closed sexual networks with a reasonable degree of certainty ended with the improvement of roads and forms of transportation in the district. At the same time, the communities mapped at the

[120] Wabwire-Mangen OH.
[121] Ronald Gray, Thomas Quinn, David Serwadda, et al., "The Ethics of Research in Developing Countries," *New England Journal of Medicine* 343, no. 5 (2000): 361-363. It wasn't until the advent of the President's Emergency Plan for AIDS Relief (PEPFAR) in 2003 that ARVs became widely available in Uganda. The RHSP was one of the first projects selected to be a PEPFAR implementing partner in Uganda and began distribution ARVs in 2004.
[122] Gray OH, Wawer OH, Wabwire-Mangen OH.
[123] Gray OH.

beginning of the STD study still form the core of the Rakai Community Cohort Study (RCCS), which has been operating continuously since 1995.

These maps were made, maintained, edited, and interpreted by Ugandan researchers who were instrumental in shaping the cohort, and thus the results, of the STD study and subsequent cohort studies. Their approach to sampling, recruitment, data collection, and analysis, was informed by their international training, their relationship with their US partners, and their experiences of the epidemic and its impact in the community. While the role of Gray and Wawer and other US-based collaborators was integral to the work of the project, the study was far more than an American study executed in Uganda. Serwadda, Sewankambo, and Wabwire-Mangen at the level of study design, and Kiwanuka, Kigozi, Nalugoda, Lutalo and others at the point of creating study instruments, training and supervising project staff, processing data, and managing the dissemination of results played a major role in the study. The controversies surrounding the results of the STD trial challenged but ultimately strengthened the sense of professional solidarity with the Rakai team and empowered them to undertake an even more audacious study in the following decade: a randomized controlled trial of male circumcision for HIV prevention.

Figures

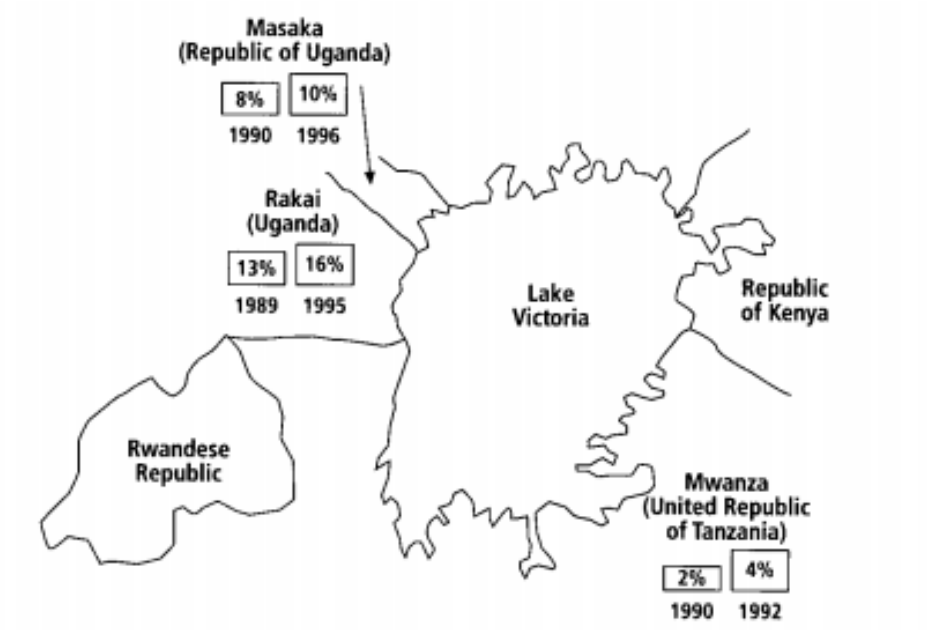

Figure 3: Map of the 3 study sites indicating their earliest documented community HIV prevalences and their HIV prevalences at the time the studies began. Reproduced from Christopher P. Hudson, "Community-based Trials of Sexually Transmitted Disease Treatment: Reperussions for Epidemiology and HIV Prevention," *Bulletin of the World Health Organization* 79, no. 1. 2001. (With permission)

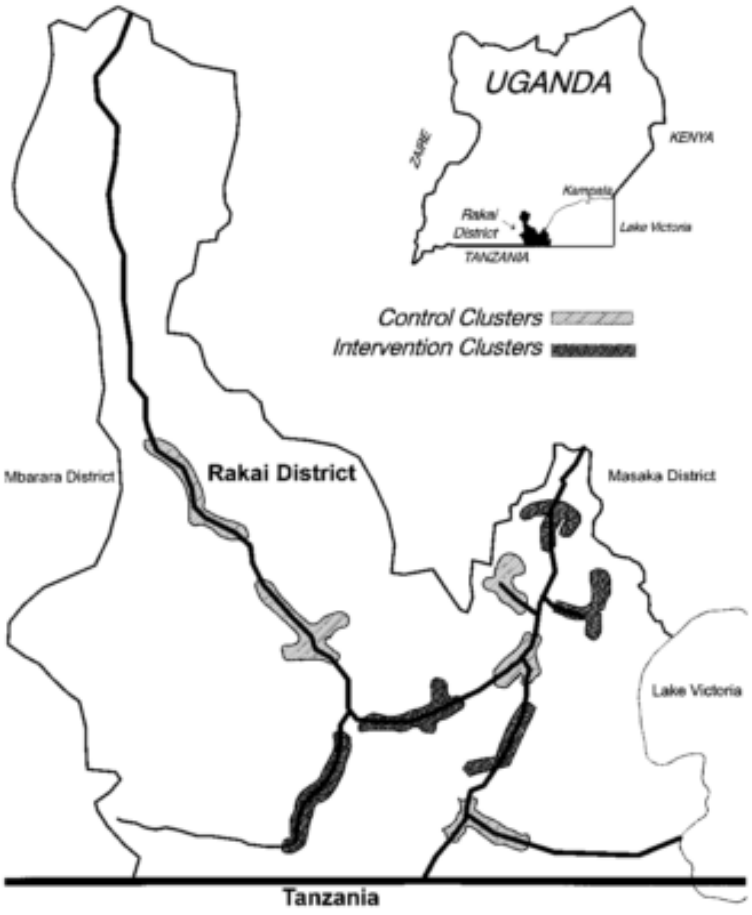

Figure 4: Map of the intervention and control clusters in the STD trial. Reproduced, with permission, from Wawer et al, "A randomized, controlled trial," 1998.

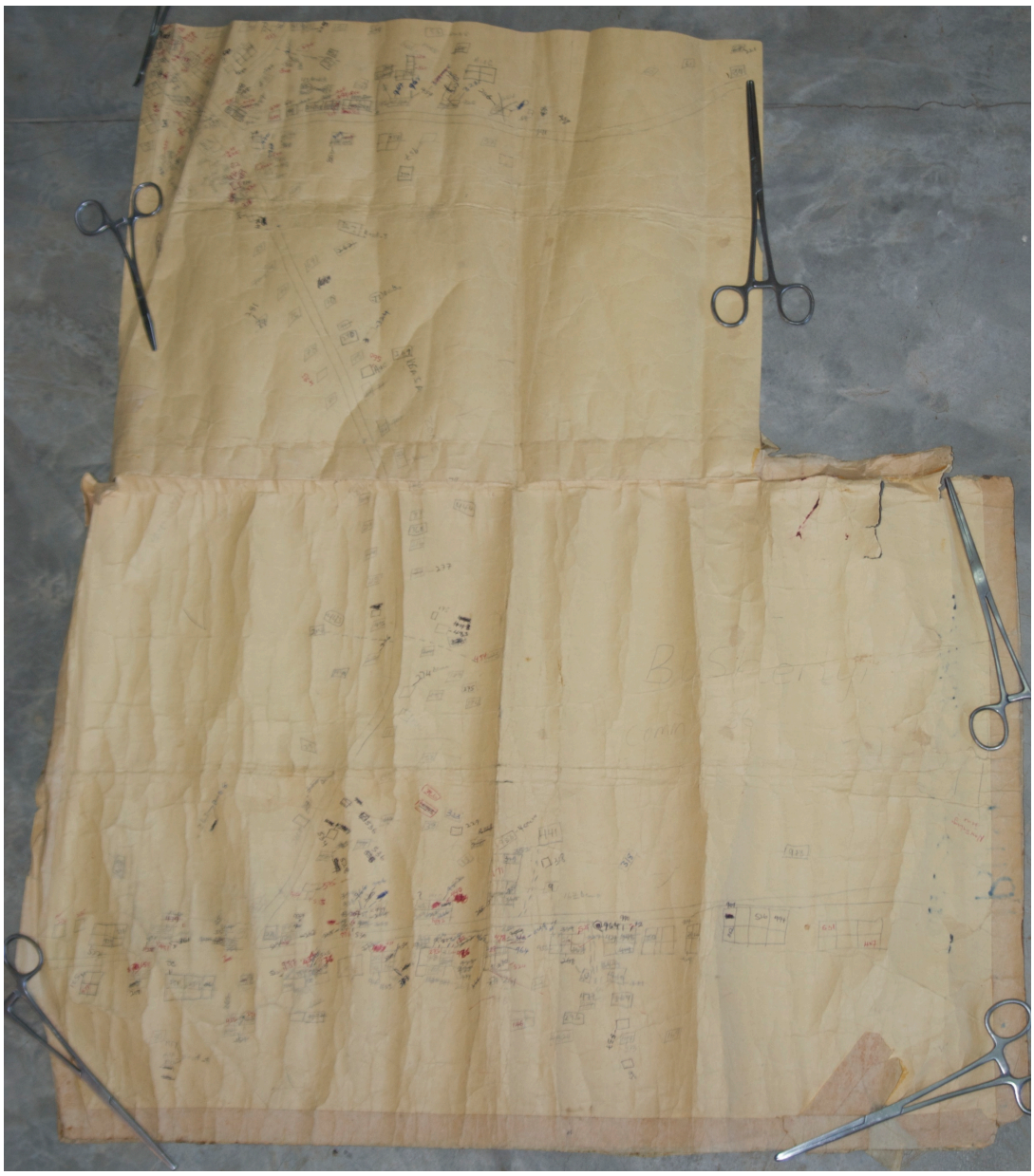

Figure 5: Map of Balanga-Bushenyi. Courtesy of the Rakai Health Scienes Program. Photograph by the author.

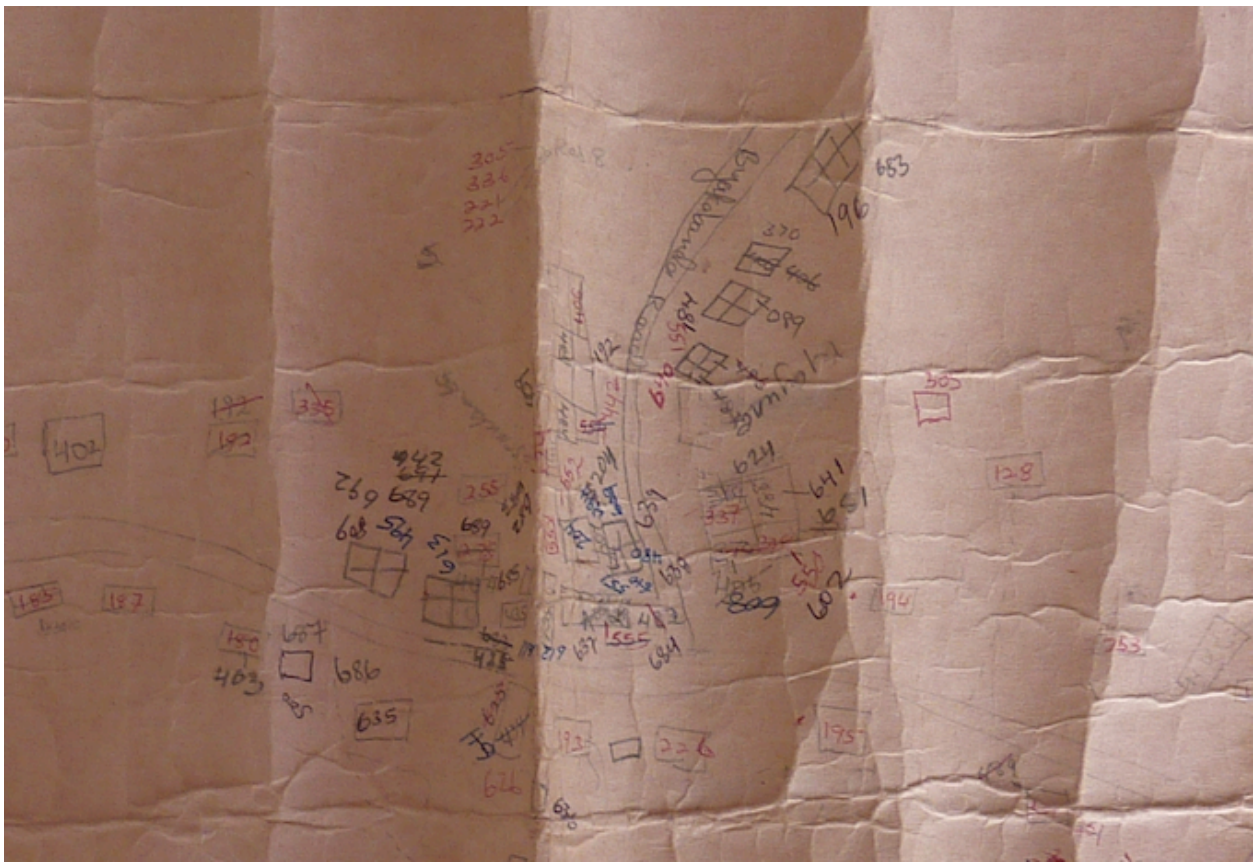

Figure 6: Detail of Kabala. Courtesy of the Rakai Health Sciences Program. Photograph by the author.

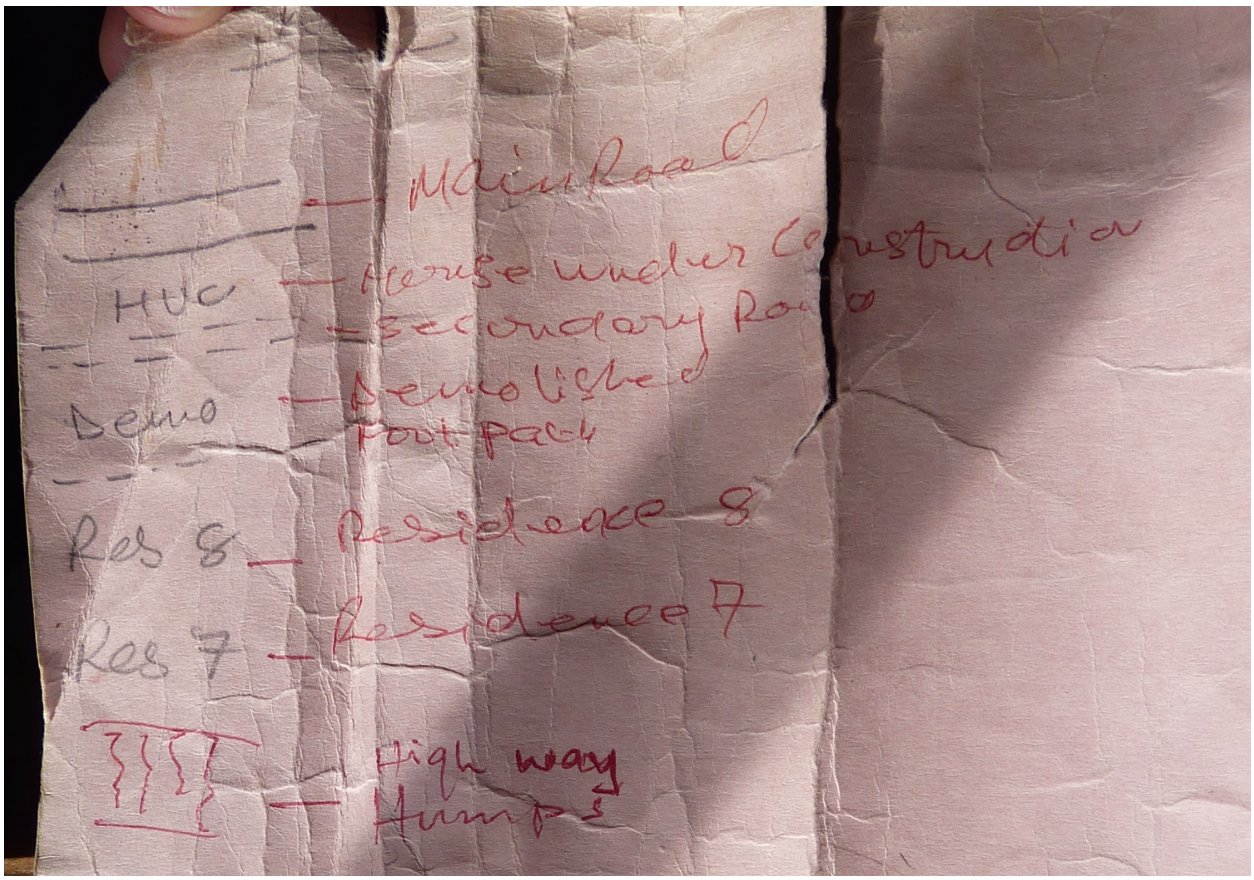

Figure 7: Map key. Courtesy of the Rakai Health Sciences Program. Photograph by the author.

Conclusion

The entrance to the Uganda Virus Research Institute, 2012. Photograph by the author.

Today, the entrance to the Uganda Virus Research Institute (UVRI) is a conspicuously secured gate in a tall fence topped with razor wire. The fence, which surrounds the hilltop compound, was part of a package of security measures required and paid for by the United States following a visit from Senator Richard G. Lugar of Indiana and a deputation of Pentagon officials in 2010.[1] Fearing that al Shabab or another terrorist group might raid the Institute's stocks of Ebola, Marburg, and other viruses that could be weaponized, the Americans characterized the Institute as a vulnerable target and prioritized the enhancement of its security. But in a *New York Times* article profiling the senator's visit, Uganda itself seems to be understood as the source of infectious disease, not just the Institute. Journalist Josh Kron, who described the Institute as a "remote Ugandan lab" in 2016, wrote, "Warm, wet and on the equator, Uganda is a biological

[1] J. Kron, "Uganda Seen as Front Line in the Bioterrorism Fight," *The New York Times* November 11, 2010, A8.

petri dish."[2] What sets Uganda apart from its equally warm and wet neighbors, however, is not a uniquely virus-friendly climate, but its unique history of virus research.

Next to the security checkpoint at the campus entrance is a large sign indicating the various national and international agencies with projects hosted at the UVRI: the Medical Research Council (MRC)/UVRI Uganda Research Unit on AIDS, the MRC/Wellcome Trust Projects, the Rakai Health Sciences Program, the International AIDS Vaccine Initiative, the Centers for Disease Control and Prevention (CDC) Uganda, the World Health Organization Expanded Program on Immunization, and the CDC/UVRI Plague Projects. This more accurately captures the significance of the UVRI than sensational headlines. The UVRI is, and has been for over 80 years, a home for virus researchers from around the world.

Making and sustaining a home for virus research at the Institute was not always easy. As I argued in chapter one, researchers in the 1930s and '40s struggled to reconcile the standards of international medical research on yellow fever with the contingencies of life and work in Uganda. Everything from the diet of experimental mice to the selection of senior scientists had to conform to the social, political, and physical climate of Entebbe. Transplanting people, animals, equipment, and techniques from New York and Rio de Janeiro to Entebbe was rarely effortless. Translating the results of the work conducted in Uganda to the board rooms of the International Health Division in New York or the *Office International d'Hygiène Publique* (OIHP), where decisions about sanitary conventions, vaccine requirements, and quarantines were debated, codified, and enforced, was equally challenging. But in this period, when virus research was relatively low-tech and had to be conducted in close proximity to sources of live virus, the Entebbe laboratory was a critical source of biomedical knowledge about yellow fever and other newly-discovered arboviruses.

Of course the laboratory in Entebbe was not the only place in Uganda where Institute scientists produced new knowledge about viruses. Alexander J. Haddow's work in the 1940s

[2] Kron, "Uganda Seen as Front Line," A8.

transformed Bwamba into a locus of knowledge production on the ecology of yellow fever transmission in the Ugandan forest. He used skilled African field assistants, techniques adopted from the laboratory, non-native monkey species, and dozens of highly detailed maps to discipline the wilderness of Bwamba for experimental purposes. As I showed in chapter two, however, the work Haddow and his colleagues did to make Bwamba a place where the natural dynamics of yellow fever could be observed also made it a very artificial place in many ways.

After the IHD turned over responsibility for the East African Virus Research Institute (EAVRI) to the British colonial government, and the Institute lost its most significant financial benefactor, scientists at the Institute had to develop new types of relationships with collaborators and donors in and beyond East Africa. The collaboration between the EAVRI and the Imperial Cancer Research Fund (ICRF), explored in chapter three, illustrates the opportunities and challenges these new relationships presented to and for the EAVRI. The possibility of a link between a yet-undiscovered arbovirus and a newly-described pediatric cancer appeared to be a way for the Institute to contribute to one of the most exciting fields in virology or cancer research—the hunt for the first human cancer virus.

When investigations at better-equipped laboratories in the United Kingdom and the United States suggested that the pathogen causing the tumor was the ubiquitous Epstein-Barr virus (EBV), the Institute shifted its approach to capitalize on its location in the heart of the "tumor belt," where most cases of the cancer were found. They found a new partner, the International Agency for Cancer Research (IARC), to invest in the infrastructure to build and maintain a large population cohort in the West Nile District that they hoped would shed light on the mechanisms that linked EBV with Burkitt's lymphoma in select parts of the world. As I argued in chapter four, the maintenance of this research project during the turbulent years of Idi Amin's regime positioned the Institute to survive the ensuing civil war and resurrect its research agenda when AIDS was discovered in the early 1980s.

Much has been written about Uganda as a site of international AIDS research in the 1990s and 2000s. Anthropologists have described the "scramble" by overseas academic institutions to establish exclusive claims to Ugandan urban and rural populations for research projects; the development of patron-client relationships between AIDS programs and the individuals who rely on them for life-saving research; and the "projectification" of the overall landscape of AIDS research, care, and treatment in Uganda.[3] These and other accounts of AIDS research in Uganda and Africa pay little attention to the historical context of biomedical research in East Africa and even less to the particular history of Ugandan virus research. One author has gone so far as to claim that at the time global health researchers developed an interest in AIDS in Africa, "research in exotic locales was in many ways a new business."[4] As I showed in chapters five and six, the Rakai Project, one of the most important studies of AIDS in rural sub-Saharan Africa, was built on the foundation laid by earlier generations of virus researchers in Uganda and was very much the product of Ugandan expertise, effort, and ingenuity. International partners have always played, and will almost certainly continue to play, an important part in the development and conduct of virus research in Uganda, but they do not operate in a vacuum.

Successful research programs beget new research opportunities, and recent developments at the UVRI and the Rakai Project, now the Rakai Health Sciences Program (RHSP), suggest that their achievements in the period described in chapters five and six have succeeded in laying the foundations for the next generation of biomedical research in Uganda. Both the UVRI and RHSP have expanded the scope of their research programs in the last several years. At UVRI, recent projects have included investigations into tuberculosis, malaria, and parasitic infections.[5]

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[3] Johanna Crane, *Scrambling for Africa: AIDS, Expertise, and the Rise of American Global Health Science* (Ithaca: Cornell University Press, 2013); Susan Reynolds Whyte, Lotte Meinert, and Jenipher Twebaze, "Clientship," in *Second Chances: Surviving AIDS in Uganda*, ed. S. R. Whyte (Durham: Duke University Press, 2014), 56-69; L. Meinert and S. R. Whyte, "Epidemic Projectification: AIDS Responses in Uganda as Event and Process," *Cambridge Anthropology* 32, 1 (2014): 77-94.

[4] Larry Krotz, *Piecing the Puzzle: The Genesis of AIDS Research in Africa* (Winnipeg: University of Manitoba Press, 2012), 53.

[5] Michael Brown, George Miiro, Peter Nkurunziza, et al., "*Schistosoma Mansoni*, Nematode Infections, and Progression to Active Tuberculosis Among HIV-1-infected Ugandans," *American Journal of Tropical Medicine and Hygiene* 74, 5 (2006): 819-825.

Similarly, the RHSP has taken advantage of its population cohort to study the relationships between HIV and intimate partner violence,[6] liver disease,[7] and non-communicable diseases.[8] While the imperatives of international funding agencies certainly have an impact on the kind of research for which Uganda-based scientists are able to attract funding, both the UVRI and the RHSP have supported researchers who wish to investigate problems or topics that they observe in the laboratory or the field and believe are significant.[9] And they are still making important contributions to global health science. In February 2017, for the first time since they began measuring HIV incidence in 1989, the RHSP reported a 41% decrease in new HIV infections—the first population-level decline in HIV incidence since the first Rakai studies in 1989.[10]

There is much more to be written about the history of the UVRI. I could not hope to do justice to the scope of its work in a single thesis. However, by selecting a few episodes in the history of the Institute, I have made an argument that the history of virus research in Uganda has had a real impact on the development of global health in Uganda and well beyond. Ugandan researchers, from the unnamed laboratory and field assistants of the YFRI to the internationally-renowned principal investigators at the Rakai Project, have shaped what we know about many viruses and their interactions with humans and other animals. At the same time, the long history of virus research in Uganda has been formative in the way Uganda, and particular places in Uganda, have become known to people around the world. Uganda may not be a "petri dish" for viruses, but it has been irrevocably constructed as a place where knowledge about viruses is made.

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[6] Jennifer Wagman, Fredinah Namatovu, Fred Nalugoda, et al., "A Public Health Approach to Intimate Partner Violence Prevention in Uganda: The SHARE Project," *Violence Against Women* 18, 12 (2012): 1390-412.
[7] Andrew D. Redd, Sarah K. Wendel, Mary K. Grabowski, et al., "Liver Stiffness is Associated with Monocyte Activation in HIV-Infected Ugandans Without Viral Hepatitis," *AIDS Research and Human Retroviruses* 29, 7 (2013): 1026-1030.
[8] Laura D. Sander, Kevin Newell, Paschal Ssebbowa, et al., "Hypertension, Cardiovascular Risk Factors and Antihypertensive Medication Utilization among HIV-Infected Individuals in Rakai, Uganda," *Tropical Medicine & International Health* 20, 3 (2015): 391-396.
[9] See, for example, Tom Lutalo, Ronald Gray, Sanyukta Mathur, et al., "Desire for Female Sterilization Among Women Wishing to Limit Births in Rural Rakai, Uganda," *Contraception* 92, 5 (2015): 482-487.
[10] Mary Kate Grabowski, Gertrude Nakigozi, Fred Nalugoda, et al., "Combination HIV Prevention and HIV Incidence in Rakai, Uganda," Conference on Retroviruses and Opportunistic Infections, Seattle, Washington, February 13-16, 2017, Seattle, Washington, Abstract 34LB.

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Biryahwaho, Benon, July 16, 2014^*

Gray, Ronald, September 12, 2014; October 22, 2014; August 14, 2015^*

Haddow, Alastair, September 30, 2015^*

Kabuye, Patrick, March 24, 2014^*

Kagaayi, Joseph, May 7, 2014^*

Kajumbula, Rose, March 8, 2015

Kalibbala, Sarah, April 2, 2014^*

Kengeya-Kayondo, Jane, March 3, 2015

Kighoma, Nehemiah, April 24, 2014^*

Kigozi, Godfrey, March 27, 2014; April 17, 2014^*

Kigozi, Grace, April 28, 2014^*

Kiwanuka, Noah, July 8, 2014^*

Lutalo, Thomas, June 6, 2014^*

Lutwama, Julius, July 16, 2014^*

Makumbi, Fred, June 2, 2014^*

Matovu, Sarah Hajat, April 17, 2014^*

Mireego, Eddie, March 3, 2015

Mugamba, Stephen, April 1, 2014^*

Mukwaya, Louis, May 27, 2014; June 5, 2014^*

Musagara, Makko, May 30, 2014^*

Musgrave, StanleyMay 13, 2014^*

Nakanwagi, Margaret, March 24, 2014^*

Nakigozi, Gertrude, April 25, 2014^*

Nakyanjo, Neema, April 15, 2014

Nalugoda, Fred, February 10, 2015; March 4, 2015^*

Nantongo, Agnes, May 22, 2014^*

Nantume, Betty, April 15, 2014^*

Ndyanabo, Anthony, May 6, 2014^*

Nkale, James, June 10, 2014^*

Olwit, George, May 7, 2015

Opendi Ochala, Pius, May 12, 2014^*

Reynolds, Steven, May 5, 2014^*

Serwadda, David M., May 14, 2014; May 26, 2014 ^*

Sewankambo, Nelson S., July 3, 2014 ^*

Ssebugenyi, IvanApril 23, 2014^*

Ssekubugu, Robert, April 3, 2014^*

Ssemanda, Robert, May 21, 2014^*

Ssembatya, Joseph, February 11, 2015^*

Wabwire-Mangen, Fred, May 26, 2014^*

Watya, Stephen, April 29, 2014^*

Wawer, Maria, September 16, 2014; October 20, 2014^*

Woodall, JohnAugust 20, 2013^*

Oral History Interviews Conducted by the Author and Charles Ssekyewa (*Transcript at the Uganda Virus Research Institute, Entebbe, Uganda. ^Transcript at the Institute of the History of Medicine, Johns Hopkins University, Baltimore, Maryland, USA)

Anonymous 1, June 25, 2014^*

Anonymous 2, June 30, 2014

Anonymous 3, June 26, 2014^*

Anonymous 4, June 25, 2014^*

Anonymous 5, June 24, 2014^

Anonymous 6, June 26, 2014^*

Kalanda, Richard Kibi, June 26, 2014^*

Kuteesa, Samuel, July 1, 2014^*

Lukyamuzi, Twaha, June 25, 2014^*

Lusiba, John, June 27, 2014^*

Mayanja, Dominic, June 30, 2014^*

Mbaziira, George, June 27, 2014^*

Nakalema, Magreti, Eva Nakyanzi, and Joseph Ssemanda, June 24, 2014^*

Nakamana Juliet, June 27, 2014^*

Nakanwagi, Gorreti, June 27, 2014^*

Nakimera Angella, June 30, 2014^*

Namanya, Mary, June 30, 2014^*

Namiiro, Annet, June 24, 2014^*

Namiiro, Jane, June 27, 2014^*

Namugyigga, Gertrude, June 30, 2014^*

Nansubuga, Milly, July 1, 2014^*

Nantume, Jane, June 26, 2014^*

Nsubuya, Harriet, June 26, 2014^*

Ssamula, Nuuh, June 25, 2014^*

Ssekamanya, Tom, June 30, 2014^*

Ssempijja, Tadeo, July 1, 2014 ^*

CURRICULUM VITAE

Julia R. Cummiskey, MPH
Department of the History of Medicine
1900 East Monument Street
Baltimore, Maryland 21205
jcummis1@jhmi.edu

Education

Doctor of Philosophy May 2017
Johns Hopkins University School of Medicine
Department of the History of Medicine
Dissertation: "Placing Global Science in Africa: International Networks, Local Places, and Virus Research in Uganda, 1936-2000." Defense: March 10, 2017.
Advisor: Professor Randall Packard
Fields in history of medicine, African history, history of international health, history of the laboratory

Masters Degree in Public Health
May 2007
Columbia University Mailman School of Public Health
Department of Sociomedical Sciences
Concentration in the History and Ethics of Public Health and Medicine

Bachelor of Arts Degree
June 2004
Carleton College
Major: History
Magna Cum Laude, Distinction in major, Distinction in senior thesis

Journal Articles

2014 **Cummiskey, J**. "Drugs, Race and Tuberculosis Control in Baltimore, 1950-1978." *Social History of Medicine* 27(4): 728-750.

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2008 **Cummiskey, J**, M. Mavinkurve, R. Paneth-Pollak, J. Borrelli, A. Kowalski, S. Blank, B. Branson. "False-Positive Oral Fluid Rapid HIV Tests – New York City, 2005-2008." *Morbidity and Mortality Weekly Report* 57: 660-665.

Book Reviews

2017 "Barbra Mann Wall, *Into Africa: A Transnational History of Catholic Medical Missions and Social Change*." *Global Public Health,* online advance publication http://www.tandfonline.com/eprint/tBrr5zgQ9FwxgqWzWr7B/full.

2016 "James C. McCann, *The Historical Ecology of Malaria in Ethiopia: Deposing the Spirits*." *Social History of Medicine* 29, 3 (2016): 662-663.

Presentations

2017 "Medicalizing and De-medicalizing HIV Prevention: The Case of Male Circumcision in Uganda." To be presented at the May 2017 Annual Meeting of the American Association of the History of Medicine, Nashville, TN.

2016 "Known Knowns, Known Unknowns, and Unknown Unknowns: Late 20th Century Archival Silences and the History of Medical Science in Uganda." Presented at the December 2016 Annual Meeting of the African Studies Association, Washington, DC.

2016 "African Research on an African Cancer: Burkitt's Lymphoma and International Medical Research Collaborations in Post-colonial Uganda." Presented at the 2016 Annual Meeting of the American Association for the History of Medicine, Minneapolis, MN.

2016 "Disparity and Decrepitude in the Archive." On a panel on "Archival Disruptions: Exploring the Temporalities of Medical and Ethnographic Archives in Africa." Presented at the 2016 University of Minnesota International African Studies Conference, Minneapolis, MN.

2015 "Foreskins, Health, and the Stylish Life in Uganda." Presented at the 2015 Annual Meeting of the African Studies Association, San Diego, CA.

2015 "Viral Landscapes: Three Generations of Medical Geography in Uganda." Presented at the 2015 International Conference of Historical Geographers, London, U.K.

2015 "Causal Cartographies: Maps and the Investigation of Yellow Fever and Burkitt's Lymphoma in Uganda, 1936-1979." Presented at the 2015 Annual Conference of the British Society for the History of Science, Swansea, U.K.

2015 "Stylish Men: Promoting Male Circumcision for HIV Prevention in Uganda." Presented at Dreaming of Health and Science in Africa: Aesthetics, Affects, Poetics, and Politics, Wellcome Trust Conference Center, Cambridgeshire, U.K.

2015 **"**Viral Landscapes: Mapping Yellow Fever and Burkitt's Lymphoma in Africa." Presented at the 2015 Annual Meeting of the American Association for the History of Medicine, New Haven, CT.

2014 "The Rakai Health Sciences Program: Post-Conflict HIV Research in Uganda." Presented at the 2014 Annual Meeting of the African Studies Association, Indianapolis, IN.

2012 "'A Special Baltimore Problem': Race and Tuberculosis in the Age of Antibiotics." Presented at the 2012 Annual Meeting of the American Association for the History of Medicine, Baltimore, MD.

2011 "'A Special Baltimore Problem': Tuberculosis and the Early Development of Directly Observed Therapy." Presented at the 2011 Joint Atlantic Seminar for the History of Medicine, New York, NY.

2007 "Increasing the Predictive Value of Clinic-Based Rapid HIV Antibody Screening Using oral Fluid and Whole-Blood Rapid Testing in 10 Publicly-funded Sexually Transmitted Disease Clinics, New York City, 2006-2007." Presented at the 2007 HIV Diagnostics Conference, Atlanta, GA.

Teaching Experience

Fall 2016 Instructor, Johns Hopkins University
Course: "Health, Medicine, Gender, and Sexuality: Gender, Sexuality, and AIDS in Africa"

Summer 2016 Teaching Assistant, Discover Hopkins Health Studies, Johns Hopkins University
Course: "History of the Hospital"

Fall 2015 Instructor, Johns Hopkins University
Course: "Jungle Doctors: Medical Missions in Africa from David Livingstone to Paul Farmer"

Fall 2014 Teaching Assistant, Johns Hopkins University
Course: "History of Medicine: Antiquity to the Scientific Revolution"

Spring 2012 Teaching Assistant, Johns Hopkins University
Spring 2013 Course: "History of Modern Medicine: Enlightenment to Present Day"

Fall 2012 Teaching Assistant, Johns Hopkins University
Course: "African History to c.1850"

Fall 2011 Teaching Assistant, Johns Hopkins University Bloomberg School of Public Health
Course: "Life and Death in Charm City: Histories of Public Health in Baltimore, 1750 to the Present"

2005-2007 School Programs Educator, New-York Historical Society
Programs: "Slavery in New York"; "Objects Tell Stories"; "Life in New Amsterdam"; "The American Revolution in New York"; "New York City and the Civil War"

Grants, Awards, and Fellowships

2016 Women and Gender Studies Teaching Fellowship, Johns Hopkins University, Baltimore, 1 semester, $8,000.

2015 Dean's Teaching Fellowship, Johns Hopkins University, Baltimore, 1 semester, $11,500.

2015 Travel Grant for the Annual Meeting in New Haven, Connecticut, American Association for the History of Medicine, $300.

2015 J.B. Harley Research Fellowship, United Kingdom, 3 weeks, £1,200.

2013 J. William Fulbright U.S. Student Program, Uganda, 9 months, $26,000.

2013 Mellon International Dissertation Research Fellowship, Social Sciences Research Council, Uganda, 12 months, $15,600.

2013 Rockefeller Archive Center Grant-in-aid, 1 month, $1,500.

Language Skills

English (native speaker), French (advanced), Swahili (intermediate), Luganda (beginner)

Other Professional Activities and Experience

2017	Panelist, Roundtable on teaching beyond the undergraduate classroom, Annual Meeting of the American Association for the History of Medicine (AAHM), May 4-7.
2016	Panelist, Roundtable on 21st-century archival reference, Mid-Atlantic Regional Archives Conference (MARAC), November 3-5.
2016	Invited Speaker, undergraduate class "The Public Health Crisis in Africa", Johns Hopkins University, Baltimore, MD, November 1.
2016	Invited Speaker, Johns Hopkins University HIV/AIDS Alternative Break, Baltimore, MD, January 16.
2015	Chair, Panel on Disease & Demography, International Conference of Historical Geographers, London, U.K.
2012-2016	Steering Committee Member, Joint Atlantic Seminar for the History of Medicine.
2009-2010	Program Manager, Montefiore Medical Center School Health Program, Bronx, NY.
2007-2009	Public Health Epidemiologist, New York City Department of Health & Mental Hygiene, New York, NY.
2004-present	Alumni Admissions Representative, Carleton College, Northfield, MN.