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02 WHERE DID AIDS COME FROM?

INTRODUCTION

For all intents and purposes, the issue of HIV/AIDS's origins has been resolved. The cut hunter, or natural transfer theory, based in phylogenetic mapping, concludes that the contemporary epidemic started when simian viruses spread from primates to humans in the early twentieth century (Worobey et al. 2003, 2016; Gilbert et al. 2007; Sharp and Hahn 2011).[1] A series of coincidental, unspecified accidents, such as monkey bites or the eating of undercooked meat, conjoined with the circumstances by which the virus could take hold and spread. African truck drivers and gay men in America took center stage in this AIDS-origin narrative. These men were aided by social structures, such as prostitutes and bath houses, and medical interventions, such as needle sticks and blood transfusions. Virtually anyone, if they know anything about it at all, will recite some version of this viral modeling combined with light social history. The press and scientific literature ubiquitously present the natural transfer theory as demonstrable fact, despite the impossibility of independent verification and many unanswered questions.

I have been curious about the lack of debate over the natural transfer theory as the origin of AIDS. Even a cursory nod toward twentieth-century bioscience, chockablock with cross-species blood and tissue experimentation, often between apes and humans, reveals multiple possible routes by which viral transfers could have—and indeed did—occur. The mystery of AIDS's origins combined with the severity of the disease would, one might expect, raise some serious, painstaking investigation into those cross-species transfers. And yet, one finds the opposite: not only have biomedical practices involving interspecies fluid transfers virtually *not* been

studied for potential side effects (such as viral transfer), but those few studies that have been done have been dismissed with a nearly casual disregard. Somehow the question of how HIV might be linked to medical experimentation with animal blood and tissue is not only unanswerable, it has been unthinkable. The recent attention to the lab leak hypothesis for COVID-19 indicates a shift in attention toward the possibility of accidents and side effects. In this chapter I revisit the AIDS origins debate and suggest that it has been prematurely resolved.

An intriguing path dependency can be tracked in light of early explanations for HIV and their continued impact on later assumptions. The first explanations of its quick and wide spread play on stereotypes of oversexed gay men and Central Africans. Surely there was a lot of sex in these communities, but more evidence would be needed to prove it as the sole route of transmission. Later discoveries about the virus, such as its long latency period, did not lead to a reinvestigation of early findings that were based on an assumption that latency was a matter of months. Even the collapse of the Patient 0 myth in which an airline pilot was blamed for spreading the illness has not led to a rigorous revisiting of those early explanations.

It's relatively easy to see why this enormous task has not been broached. One would need to revisit the difficulties and controversies in identifying the virus through the 1980s, including the impact of variously efficacious testing methods on how the earliest cases were identified. These diagnostic confusions still muddy the waters, specifically relating to the earliest cases, the "Manchester Sailor" and Robert Rayford, both of whom are now considered not to have been AIDS cases, and yet whose early positive testing laid the framework for the acceptance of certain explanations for the epidemic's etiology. Since HIV presents through a patient's infections with more common diseases such as pneumonia and Kaposi's sarcoma, the record has been pockmarked with much confusion over the verifiable cases and their relevance.

Add to this the sheer complexity of the task: the amount of information to be parsed, from human mobility (laborers, traders, tourists, aid workers), to the global animal trade and export business (probably millions of primates in global circulation for research and to make the tissue cultures for vaccine preparation), and a global market in human blood, including imports to the United States from Africa and the Caribbean. Much of that information, undocumented anyway, is simply not available at the granular level required to track the mobility of a virus. If such complexities

account for why AIDS origins have not been thoroughly investigated, they also seem to counter the rather vicious dismissal of another origin theory, one that quite reasonably suggests that the cross-over event resulting in HIV was the result of a polio vaccine trial in the 1950s.

The oral polio vaccine (OPV) theory of the origins of HIV remains worth considering for the fascinating details of the theory and the light it sheds on biomedical attitudes and practices of the mid-twentieth century. The short-lived debate it spurred in the late 1990s, and its "resolution" in favor of the cut hunter theory, also reveals much about how scientists adjudicate questions of the past and our own mistaken trust in such forms of scientific resolution.

In 1999, British journalist Edward Hooper described the OPV hypothesis (Hooper 2000c). Over the course of nearly one thousand spellbinding pages, Hooper unfurls an account of an OPV trial undertaken by American scientists in the Belgian Congo and Ruanda-Urundi between 1956 and 1960 (Hooper 2001; Courtois et al. 1958; Plotkin et al. 1961). He finds a stunning correlation between the geography of the earliest cases of HIV and the OPV trials, presents a detailed reconstruction of the chimpanzee lab in Stanleyville (the base location of the trials), and details a history of the development of the vaccine by Hilary Koprowski at the Wistar Institute in Pennsylvania and its testing in several American states, Europe, and the Congo. Given that scientists from the Wistar Institute sprayed or spooned live polio vaccine grown with animal tissue cultures into the mouths of about a million Congolese, a simian immunodeficiency virus (SIV) could, in theory, by this route cross over into humans through oral cuts or abrasions. This OPV theory, Hooper argues in detail, makes more sense than natural transfer theories, and it works from the same data beginning from the first known case of HIV-1 in Kinshasa (Léopoldville) in 1959.[2]

The River immediately received laudatory reviews in major press outlets (Cimons 1999; Altman 1999; Trivers 2000; B. Martin 2000). Praise, however, came to a swift end after a conference at the Royal Society in London (held September 11–12, 2000), which was convened to discuss the OPV theory. The precipitous and, I believe, premature closing of the debate with a widely reported press conference led to the near-universal labeling and dismissal of the OPV hypothesis as a "conspiracy theory" (rather than, say, as a plausible counterfactual hypothesis). Despite, or perhaps because of, the unusual way in which a conference came to be the arbiter of the OPV proposal, Hooper and his remaining supporters were excluded from subsequent discussions in the scientific press. In light of

that, Hooper continues to publish his and other's doubts and rejoinders on a website, AIDS Origins (http://www.aidsorigins.com).

In what follows, I will not argue that the vaccine trials launched the acquired immunodeficiency syndrome (AIDS) epidemic, nor will I recite Hooper's account. Rather, I analyze how the genealogy of the dismissal of the OPV hypothesis demonstrates that the closure of the debate precluded discussion, fact-finding, and uptake of the key, and very much needed, contributions of Hooper's research. Specifically, *The River* offers one of the very few analyses of the massive global infrastructure of post–World War II vaccinology, one that includes highly mobile geographies of human experimentation involving interspecies and viral fluid exchanges on a scale nearly unimaginable to a lay reader. This infrastructure relied on the importation and sacrifice of millions of primates and other animals, particularly monkeys from India, Africa, and the Philippines (Kalter and Heberling 1971); local animal trade and care networks; Cold War and colonial politics; technologies of refrigeration, preservation, and shipping; exchange networks for biomaterials among Europe, the United States, and Africa; and high-stakes, fragile, competitive, and collegial power struggles among scientists committed to controlling how debates were framed and what information was documented and shared. By literally opening vectors for the transmission of pathogens among human and nonhuman bodies, this biomedical and technological infrastructure, which elsewhere I have called the "The Wetnet," choreographed a zone that fundamentally altered potential and real viral dynamics, spillovers, and exchanges.[3] Inter- and intraspecies viral transfers became possible in entirely new and unpredictable ways. Along with this infrastructure arose logics—such as the promise of vaccines—by which new risks were made to seem normal and justifiable; these rhetorical means became the foil and norm against which other possibilities have been judged. I argue that the OPV hypothesis can be understood in this context not exclusively for its truth or provability but as a plausible counterfactual that reveals much about how belief structures underpin what comes to count as truth.

To make this argument, I consider questions of historical reconstruction in conditions of uncertainty. Catherine Gallagher (2018) theorizes "what if" and "but for" scenarios as counterfactual histories. Such modeling, when applied to possible vectors of disease, can identify the architecture of trust relied upon: *If* an iatrogenic spillover event had occurred, how *would* we know? What kinds of information, not included in scientific reports and publications, would be necessary to reconstruct such possibilities? If

a counterfactual method offers another way forward, enabling consideration of the multiple possibilities that may have resulted from complex biological exchanges in the context of uncertainty and naivete among scientists, it also offers a way to practically understand how allied practices, such as record keeping and shared archives, impact how the origins of emerging diseases can be reconstructed. Given how little research there is in this enormously complex and crucially important area of vaccinology, and given the burgeoning interest in medical anthropology on zoonosis (Keck and Lynteris 2018), I believe the OPV-HIV story provides insights that increase awareness of and languages for describing the complex global bioformations constituted by midcentury vaccinology.

I base this historical ethnography on the recording of the meetings archived at the Royal Society Library in London; interviews with two spectators (Elizabeth Tilly and Vinh-Kim Nguyen); interviews with participants Edward Hooper, Stanley Plotkin, and Robin Weiss; a comprehensive analysis of *The River* and the papers from the conference published in a special issue of the *Philosophical Transactions of the Royal Society of London* (Hamilton, Weiss, and Hobson 2001); a review of the scientific literature on the hypothesis published before and after the controversy's closure; and study of primary and secondary literature in vaccine history.

HOOPER'S HYPOTHESIS

The River parses an astonishing array of primary and secondary documents; Hooper's materials range from flight schedules to chimp behavior to dozens of interviews with scientists and others who were involved in, or adjacent to, the vaccine trials. The hypothesis sets forth two distinct components. First, Hooper provides arguments and evidence about why routes of HIV transmission based on human mobility proposed by other scholars lack credibility. He also documents the uncanny geographic correlations between the vaccine testing and the earliest cases of what would become known as AIDS, whereby "all 46 documented instances of HIV-1 infection from Africa through 1980 come from within 140 miles of CHAT [the OPV vaccine] vaccination sites" and "70% of these earliest AIDS cases come from a town or village where CHAT had been vaccinated" (Hooper 2001, 806). This and other data provide circumstantial evidence for the vaccines as a plausible source of the initial spillover events. Second, the tissue cultures on which the polio virus was grown offer a plausible

explanation for the *mechanics* of a spillover. For instance, the seed lots of vaccine made at Wistar could have been, at the lab's base in Stanleyville, either attenuated (further developed) with chimpanzee kidney tissue cultures or, alternatively, contaminated with fluids from chimpanzee dissections.

In the United States and Europe in the 1950s and 1960s, the renal tissues of various monkey species were used for a range of medical and virological purposes, requiring the sacrifice of vast numbers of animals (Ahuja 2013; Bookchin and Schumacher 2004). Hooper interviews several experts who verify that animal kidney tissue cultures would contain lymph and other fluids that could harbor viruses. Chimpanzees and other apes generally did not contribute organs for tissue cultures in the United States; this was due not to any biological barrier but rather because the animals were expensive and dangerous. However, in Congo, chimpanzees were in plentiful supply, and the Stanleyville lab housed between four hundred and six hundred chimpanzees (in 1956–58), many of which were sacrificed without explanation (Hooper 2001). Hooper has identified these chimps, tracked where they might have been captured, and interviewed a local African lab technician who had worked in the lab and claimed that they had been making OPV with chimpanzee tissues.[4] Additionally, Hooper located a Belgian scientist, Alexandre Jezerski, who was at the time growing tissue cultures from the kidney cells of various primates (including chimpanzee) at a rudimentary lab nearby, and with whom Koprowski had met during one of his visits to Congo.

If a chimpanzee virus *had* contaminated the vaccine and instigated a crossover event, contemporary circumstances would have militated against recognition of it. For one thing, as Koprowski himself readily admitted, follow-up with trial participants was lax. Koprowski had selected rural, medically underserved areas for testing a vaccine containing strains of live polio virus whose key danger was the risk of spreading polio, yet he had no formal plans for keeping records. Tracking side effects of the vaccine was, in any case, curtailed by Congo's unexpected independence in 1960, which resulted in the expulsion of most Belgians and other Westerners—although, to be sure, the United States maintained covert operations in the country (perhaps including the Stanleyville lab) for political and economic strategic reasons (van Reybrouk 2014).[5] In addition, researchers at the time would not have linked a vaccine to early AIDS cases if, as would have been the case, AIDS-related illnesses had presented as familiar pneumonia or TB. If the virus had to be transmitted one or more times before

it became virulent to humans, recognition of any causal link between the vaccine and virus would have been beyond the ken of any clinician or researcher.

The discipline-wide, broad-based intellectual framework necessary to have recognized the possibility that a virus could have contaminated tissue cultures and then have been spread through vaccines and have gained virulence after spreading almost certainly simply would not have existed—even if the trial had taken place in the United States under tighter regulations. The SV-40 case explained below details some of the resistance in the scientific community to acknowledging the dangers of animal viruses in tissue cultures. And as examples such as the synthetic estrogen diethylstilbestrol (DES) and Thalidomide have shown, scientific methods and interests tend not to be oriented toward understanding multigenerational and long-term effects of medical and industrial interventions.

While Hooper relays conversations with a number of the scientists he interviewed who found his theory plausible, he gained only one strong ally willing to speak out for the possibility of the OPV hypothesis during the course of his research. Bill Hamilton, a well-respected professor of evolutionary biology at Oxford University, became a proponent of the OPV theory and proposed the Royal Society conference. He never made it to the event that he initiated: he died in March 2000 from an illness contracted in the Congo while conducting research on the OPV question. One can speculate that his death had ramifications for the direction that the Royal Society conference took, as it left Hooper with no one inside the establishment with an interest in the theory. While this point speaks to science and technology studies' (STS) debates about controversy resolution, the existential overtone hints at the potentially significant ramifications of coincidental events in the course of history.[6]

The complexity, detail, and novelty of Hooper's theory cannot be overstated. While arguably the length of the book may deter casually interested readers, it would have had to have been hundreds of pages longer than any of the scientific reports related to the oral polio trials for it to have effectively tracked and explained to a nonspecialist audience the history of the vaccine and the various ways in which the trial, the virus, and the cross-species contamination might have played out. Indeed, as I argue below, the controversy highlights how conflicting demands for and requirements of evidence and burdens of proof measure against assumptions about normative and reasonable behaviors and expectations in the construction of historical truths.

THE CONFERENCE

The Royal Society meeting participants fell into three main groups: (1) four of the scientists involved in the Congo Trials (Paul Osterreith, Jan Desmyter, Hilary Koprowski, and Stanley Plotkin) and allies, including a group of phylogeneticists; (2) Hooper and allies; and (3) a varied group of speakers addressing zoonosis generally and epidemics broadly related to HIV. This last group added to the notion and milieu of a "conference," but it did not address or contribute to the debate at hand.

The agenda was skewed from the get-go. No one but Hooper could bolster the OPV hypothesis with additional facts or evidence. Since he had the same time allotment as every other speaker, he simply could not address the many dimensions of the theory. His main allies consisted of the Australian sociologist of science Brian Martin (2001), who gave a paper on the notion of proof in science, and Walter Nelson-Rees (2001), a well-known scientist active in publicizing cell-line contamination, who gave rather damning testimony on the believability of the Wistar scientists.

Hooper's paper, dense with detail, tracks among other things the numbers of chimpanzees at different research sites; it documents interviews with the scientists and lab technicians working in Central Africa in the 1950s; and it offers circumstantial evidence suggesting both that chimpanzee kidneys were being extracted and sent to the Wistar Institute and that batches of the polio vaccine were being made in Africa.[7] His paper addresses further issues related to chimpanzee subspecies, the geography and timelines of the OPV theory versus phylogenetic modeling, and other possible arguments against the theory.

Stanley Plotkin, who would become a giant of twentieth-century vaccinology, had in the 1950s just launched his career at Wistar as a junior researcher and had traveled to Africa for the trials. His paper refutes the OPV theory not with independent records of how the vaccine was made, but with the flat denial that any chimp tissues had been sent to Wistar. He writes: "I was in the laboratory from August 1957 to June 1961, and never saw or heard of chimpanzee cells" (Plotkin 2001, 816). He concludes, "*The River* has been praised for its precise detail and wealth of footnotes, but one can be precise without being accurate" (Plotkin 2001, 822). By contrast, Belgian scientist Paul Osterrieth worked at the lab in Stanleyville where the Wistar scientists did efficacy and other testing on chimpanzees. He claims in his paper: "It is true that six minced chimpanzee kidneys were sent to the Wistar Institute" (Osterrieth 2001, 839). Such discrepancies in

personal recollections stand in lieu of records, with no wider or structured attempt at rebuttal or reconciliation. As a result, the reader has no way to judge the veracity or likelihood of the different narratives.

The means by which the Royal Society meeting hammered the first nail into the OPV theory's coffin has received a rigorous STS work-up by Brian Martin (2010), the STS scholar who also presented at the conference. He portrays the form of the rather stunningly rancorous proceedings, and he suggests that the Royal Society meeting and subsequent events demonstrate the ways in which "supporters of orthodoxy have a tactical advantage over challengers" (B. Martin 2010, 215). He compares the Royal Society meeting's tactics, one for one, to other dominant political movements such as those of the Indonesians' justification for violently quashing protestors in East Timor. Martin's observations about the mechanics of justification asks his readers to see the violence behind, and enabled by, the epistemological and aesthetic front of the conference—one behind which all kinds of reasonable and normative people and assumptions can scurry.

The most crucial point made at the conference all but sneaked out of the building via a fire escape; certainly, it was not reported in the press. At the meeting's conclusion, long after the reporters had left, the chair and convener of the meeting, Robin Weiss, an expert in retroviruses and cross-species viral transmission, stated that experimental vaccines *could* credibly have been the cause of the zoonosis that resulted in HIV. He later wrote: "To reduce the argument over the origins of HIV to the OPV hypothesis versus the cut hunter hypothesis is an over simplistic and false antithesis. Both natural and iatrogenic transmission of many retroviruses, including HIV, have been thoroughly documented and are not mutually exclusive" (R. Weiss 2001a). *Surely Hooper's challenge is worth truly understanding*, we can hear Weiss intimating. And yet, closure on the OPV-HIV debate had already been achieved—not based on the evidence (which was inconclusive) but because the politics of certainty in science demanded it. Certainty in this case came down to the insistence of the scientists in the room.

A close reading of the Royal Society meeting reveals an event mired in the confusing intentions of the organizers who at once claimed to want to investigate the OPV hypothesis while making that structurally impossible. Many of Hooper's key points were not taken up or addressed at all by the speakers and the resulting collection of essays. No other formal structures for investigation—such as through law or a third party—were or are available to address questions of this kind or scale, and no independent

researchers emerged to take on the considerable effort and risk of continuing, or verifying, Hooper's research.[8]

Even a cursory reading of the Royal Society's conclusions, which are presented in an essay by conference convener Robin Weiss, renders problematic any ready acceptance of the notion that universally emerged from it: that the OPV hypothesis is debunked. Weiss's paper can be read as a clear warning about the possibilities of zoonosis, and his prevarications relay an ambivalent conclusion. Indeed, Weiss explicitly echoes journalist Tom Curtis, who had originally introduced the OPV hypothesis in a 1992 article: "If the Congo vaccine turns out not to be the way AIDS got started in people, it will be because medicine was lucky, not because it was infallible" (T. Curtis 1992, 108). It is telling, and certainly a result of Weiss's rhetorical approach, that while Koprowski sued Curtis for libel, Weiss's finding flew under the radar (Hooper 2000a; Plotkin and Koprowski 1999; R. Weiss 1999).

One final epitaph to the OPV hypothesis bears noting. Hoping to confirm his hypothesis, Hooper had advocated for any extant vaccine to be tested by a neutral third party. After the conference, samples provided by Wistar tested negative for chimp DNA and SIV/HIV. The Wistar scientists claimed absolution, and the press once again declared the case closed. For his part, Hooper pointed out flaws in the testing, most specifically, "There is no evidence that any of the CHAT samples produced at the Wistar Institute and Wyeth Laboratories . . . have any relevance to the vaccinations conducted in Africa." He added: "It is now apparent that the vaccine used in Ruzizi and along Lake Tanganyika did not comprise one homogeneous preparation of CHAT pool IOA-11 [the pool that was tested], but rather several different CHAT preparations, made at different times and originating from different laboratories" (Hooper 2001, 807). While even Koprowski had claimed that samples of the vaccine used in the trials no longer existed (Vaughan 2000), this testing was the final nail in the coffin.

If this strategy of consensus science worked, it was because scientists have a great deal of cultural and economic capital that they used to guide the debate, and journalists and historians have generally fallen into line. It remains true, however, that the free and open debate of the OPV theory would have required institutions, record-keeping practices, independent peer review, and modes of interrogation that simply did not and do not exist. Despite good reasons to critique legal reasoning and practice, the legal system does offer a structure for determining the likelihood that events occurred in particular ways based on evidence and testimony.

Clearly if capital-S science, or capital-M medicine *had* wanted to develop a means of self-regulation, ample opportunities have been presented over the decades. With no formalized way to handle a narrative such as Hooper's, however, personal responses and judgments took on an outsized role, and major slippages stood uncontested.

THE FINAL REPORT: AMBIVALENT INTENTIONS

Academic conferences typically gather independent researchers to present work on overlapping interests, and as such they are not intended to resolve controversies in any structured or rigorous way. Thus, a conference offers a curious format in which to tackle a subject of such complexity, and Robin Weiss's published paper assessing and summarizing the proceedings similarly offers a problematic finale, one that provides neither the evidence nor the logic to adequately conclude the debate, despite its presentation as such.[9] In his essay and in person, Weiss represents the two-day Royal Society meeting as an open and rigorous debate whose aim was to "lay open all the arguments and counterarguments."[10] One can only guess at the reasons for this rush to closure in a mere two days. He had already reviewed *The River* for *Science*, where he described it as "a towering achievement; right or wrong in its main conclusion, there is much to learn from Hooper's exposition" (R. Weiss 1999). As such, Weiss's focus was on a second tier of "important lessons to be learned from Hooper's analysis," which he lists as "our complacency over 44 years' use of primary monkey kidney cells as a substrate for live viral vaccines" and the use of litigation to shut down debate, as Koprowski had done in suing Tom Curtis.

Weiss's conclusion to the proceedings uses an intriguing rhetorical method to leave the door ajar for future consideration of the OPV theory while still appearing to reject it outright (Weiss 2001a). After each point he makes in favor of the cut hunter theory, he curiously loops back to note that none of his points actually disprove the OPV hypothesis. Such rhetorical skill, I would argue, was a crucial factor in the closure of the debate over OPV as a source of HIV, and it suggests that subsequent commentators did not closely read the document. His argument consists of a series of subjective assessments: his trust in the scientists' testimony; his view that the OPV theory seems "contrived"; and his belief that the burden of proof lies with Hooper. Weiss finds no motive or evidence for a cover-up on

the part of the scientists: he finds them believable and reasonable, falling squarely into a kind of old-school notion of reasonableness as described by Steven Shapin and Simon Schaffer in their classic work on experimental science (Shapin and Schaffer 2017). Weiss also discusses what he considers to be the unassailable reputation of the pharmaceutical industry. Both of these points are irrelevant to the OPV theory, unless one believes they preclude the need for further confirmation of events.

Notably, given the stakes of the argument, crafting objectivity was a personal and rhetorical accomplishment. As a result, the entire edifice of the conference depended on the believability and characterization of the OPV scientists as disinterested bystanders, genuinely wanting to engage a debate that put them at the center of a poorly run trial on medically underserved colonized people, and may have been the cause of the HIV epidemic that had killed tens of millions of people.

Weiss clarified to me in an interview his reasons for believing the scientists. In the mid-1950s, he explained, it would have been completely acceptable for the scientists to have used chimpanzee tissues for vaccine manufacture.[11] This ironic twist of reasoning (they are honest because it was standard practice to do the very thing that is purported to be a root cause of the HIV cross-over event) enables him to both embrace the possibility of OPV transmission and retain the credibility of the scientists involved in these trials. Weiss offered another confusing premise equally unproblematically. He writes, "Neither does the polio vaccine industry have a particularly bad record of cover-up" (R. Weiss 2001a, 952). Leave aside that no unitary "polio vaccine industry" existed at the time: What industry there was had virtually nothing to do with Koprowski's trials. Still, Weiss gives two questionable examples of the "success" of the industry. He cites the Cutter incident, in which an improperly made vaccine was found to have given some forty thousand people polio, resulting in five deaths and fifty-one cases of permanent paralysis, and which was aggressively defended by Cutter Labs in subsequent personal injury cases.[12] Then he mentions SV-40, a monkey virus that contaminated Salk's polio vaccine and that was spread to millions of Americans. Weiss praises the "quick response" to SV-40 by describing the replacement of kidney cell substrates derived from rhesus macaques with that of African Greens in polio vaccine manufacture. To describe these incidents as successes is simply bad faith.

The take-away from Weiss's points is emphatically not that there were no cover-ups, but that the whole infrastructure of vaccine development,

testing, and administration was highly experimental in the 1950s and early 1960s, with an adventitious simian virus, SV-40, being spread to large populations; lax manufacturing protocols; unethical experimentation; little regulatory oversight; and, ultimately, the likelihood of SIVs "on rare occasions" slipping into vaccines.

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Questions relating to subtypes and recombination lie beyond this chapter's purview. Amid complexity and speculation, Weiss turned to Occam's Razor. This problem-solving principle asserts that the simplest explanation is generally the correct one, and Weiss used it to argue that the OPV theory is "unnecessarily complicated" (2001a, 949). Specifically, the diversification date of the virus according to phylogeneticists would have been the date that it entered the human species, whereas for Hooper, it would have diversified in chimps and then been transferred to humans.

Turning to medieval philosophy to adjudicate an issue of this magnitude offers an intriguing strategy. Surely, the "simplest" explanation depends on one's basic disposition or knowledge base. For many Black Africans and colonial subjects, the simplest explanation would be that white people have hated and murdered Black people for centuries. Here again Weiss prevaricates and allows the possibility of multiple routes of cross-species transmission. In other words, despite going through the motions of describing Occam's razor and finding Hooper's more complex, he admits that both theories of the crossover could be true.

Ultimately, Weiss's essay (both brilliantly and disappointingly) offers a conclusion that implies that the conference had properly adjudicated and dismissed the OPV theory. Only by engaging the text does one see what little evidence this conclusion rests on. Barely discussing Hooper's findings, he relies instead on a strong belief in the good of science and its spokespeople. The writing may well be in bad faith, as Martin's (2010) broader reading of the conference suggests. Hedging also offers an effective form of manipulation. Weiss might have been eager to close the debate for good reasons superseding the implications of the debate: fears of an anti-vaxx movement, the challenge of an accomplished journalist-historian "outsider" who was unpopular with Weiss's powerful (and, not incidentally, senior) scientific colleagues, and the consequences of acknowledging the magnitude of the possible events. Difficult as it is to know what to make of this document, it offers an intriguing method of

closing a controversy, one that surely evinces a missed opportunity to do exactly what he seems to want to do: that is, open debate on the risky practices of the era.

In the aftermath of the conference, scholars have gone some way to reinforce the idea of the debate's closure. For example, in *The Origins of AIDS* (Pepin 2011), a book that has emerged as the model for the explanation for AIDS, physician and historian Jacques Pepin devotes three pages to *The River.* Pepin wrongly bases his dismissal of Hooper on Plotkin's argument and accused Hooper of a rookie mistake in confusing local dilution of concentrated vaccine stock with local production or amplification (Pepin 2011, 52; Gellin, Modlin, and Plotkin 2001). This caricature of the OPV hypothesis belittles both the hypothesis and Hooper's research, making him an easily dismissed strawman.

Like the post–Royal Society conference press, Pepin relies solely on the word of the scientists who ran the trial. But instead of addressing this question about evidence and objectivity head-on, Pepin accuses anyone who would doubt his reliance on the defendant scientist's account of conspiracy thinking. In considering the vaccine that tested negative for chimpanzee DNA, he writes, for example: "Conspiracy theorists could argue that [Wistar] had a vested interest in supplying vials which they already knew were not contaminated" (Pepin 2011, 52). He resorts to an anti-intellectual ad hominem attack rather than engaging Hooper's hypothesis raising the question of why Pepin himself is so dependent on, and ready to accept, the scientists' word.

I am not claiming that the OPV theory is correct. But it is notable that a book that serves as the go-to resource for the origins of the epidemic resorts to mischaracterization and name-calling, and it is equally noteworthy that this tactic flies under the radar of reviewers. A discussion of Pepin's article by physician and science historian Howard Markel (2011), patronizingly titled "It's the Science, Stupid," illustrates the latter point. Markel briefly parodies Hooper's book as "insisting" on a "fanciful thesis." He then poses Pepin's breakthrough based on "meticulous scientific analysis," that "a viral strain called SIVcpz, which infects large numbers of . . . chimpanzees living in central Africa, was the central source of HIV-1." This point is definitively not a breakthrough, and it is actually one that both Hooper and Pepin agree on. They differ in their hypotheses of *how* the species jump took place. But despite Markel's assertions to the contrary, no evidence marks Pepin's account as specifically more "convincing" or

"brilliant" than Hooper's, aside from Markel's own ability to be convinced. Both accounts require the reader to fill in details and gaps with what they consider to be reasonable.

WHAT CANNOT BE ASKED

It is obvious why Koprowski and Plotkin would want to kneecap the theory and the messenger. However, it is not as clear why others have not taken an interest in the deeper story-behind-the-story of the OPV hypothesis either as a legitimate possibility for zoonotic events or as a fascinating story of the complex intertwining of human, animal, and viral interspecies transmissions quite apart from HIV.

The late scholar of historiography Hayden White makes the point that a discipline is constituted by what it forbids its practitioners from doing. He writes that "the so-called 'historical method'" consists of little more than the injunction to "get the story straight" (without any notion of what the relation of "story" to "fact" might be) and to avoid both conceptual overdetermination and imaginative excess at any price (1978, 126). This useful insight reflects on the OPV debate, since "the science" relied on in its resolution consists not of provably true facts, but rather, as I have outlined, finds its truth in a historical narrative based on what commentators assume as plausible, sensical events. Pepin, Markel, and others (Nattrass 2012) who dismiss the OPV hypothesis out-of-hand indicate precisely what is "forbidden" in historical scholarship about science: historians cannot disagree with "the science" *as constituted by scientists*. It does not help that Hooper's account is organized not as a lucid explication of his results but as a narrative of his decade of interviews, discoveries, and hypotheses; few casual readers would put in the time it takes to get through *The River.* But the same could be said for numerous historical and academic texts and archives that historians manage to closely parse and analyze.

Looking back at vaccine production in the 1950s and 1960s certainly gives the sense that if there was no species jump it was pure luck. In fact, the focus on what *actually* happened has left a major gap in the history of science, STS, and medical anthropology. Namely, biomedical infrastructures, such as tissue cultures, vaccines, and blood products, created the new routes for zoonotic and intraspecies viral transmissions that need to be better understood.

One example of an iatrogenic zoonotic transfer of a monkey virus occurred in Jonas Salk's killed polio vaccine in the 1950s. Revisiting that story opens some larger questions about the history of vaccinology infrastructures, risk, and regulatory infrastructures.

Salk completed his 1954 field trial of a vaccine consisting of killed polio virus on 1.8 million American children. The vaccine, subsequently used in a mass effort to eradicate polio, was made by growing polio virus on the kidney tissues of rhesus macaques that were imported from India by the tens of thousands per year. The polio virus was killed with formaldehyde, with the assumption among vaccinologists that any extant monkey viruses would thereby also be killed. It was further surmised that monkey viruses would not cross the species barrier, and therefore, that potential cross-over events need not be seriously studied (Bookchin and Schumacher 2004, 79).

A complicated and relevant story ensued. A brief version is as follows. Bernice Eddy was a scientist, working at the Laboratory of Biologics Control (LBC) since 1936, who had completed award-winning work devising potency and safety tests for gamma globulin and developing influenza and polio tissue cultures. With Sarah Stewart, a National Institutes of Health (NIH) scientist, Eddy received international recognition and founded the field of viral oncology with her codiscovery of the SE-polyoma virus (Eddy and Stewart 1959). Having shown that a mouse virus could cause cancer in small mammals, she began to wonder whether a monkey virus could cause cancer in other primates, including humans. While the occasional virologist had raised misgivings about the possibility of vaccines as a possible vector of zoonosis (Hull, Minner, and Mascoli 1958), no one raised the possibility that simian viruses could cause cancer. Not finding anyone at the LBC willing to collaborate on what was considered politically sensitive and possibly career-hijacking work, Eddy began research on this question, and soon found that 109 of 154 hamsters injected with a rhesus kidney cell extract developed tumors and eventually died. She suspected the tumor-causing "substance" was hardy and virulent, had a long latency period, and maintained oncogenicity over time and through passage from animal to animal. And it originated in the monkey tissues.

At this point Eddy presented the results to her boss, the head of vaccine safety testing at the Division of Biologics Standards (DBS), Joe Smadel. Smadel discouraged Eddy's work, eventually forbidding her to publish without his permission (which he rarely gave) and moving her

into a tiny lab, "stripping her of all her vaccine responsibilities" (Bookchin and Schumacher 2004, 67). While Eddy did ultimately publish her work, Debbie Bookchin and Jim Schumacher (2004), in their detailed history of SV-40, explicitly labeled Smadel's response a "cover-up," a point to which I return below.

Smadel eventually admitted to Eddy's discovery of SV-40 when Ben Sweet and Maurice Hilleman (Hilleman 1998) disclosed their simultaneous detection of the same agent contaminating rhesus and cynomolgus monkey tissues.[13] The debates that followed among virologists over what to do about SV-40 were confounded by a morass of competing interests: the USSR was winning the "polio gap" with a more effective, cheaper, and painless oral polio vaccine developed by Albert Sabin (Bookchin and Schumacher 2004, 70); there was competition between Hilleman and Sabin for their respective killed and live polio vaccines; the manufacturers had questions regarding liability; there were real concerns about SV-40's dangers; and fear about losing public trust in a vaccine already widely distributed and celebrated.

Koprowski himself thought it best not to exaggerate the significance of viral contamination: "If an adequate number of persons exposed to these agents have been shown to develop specific antibodies without any clinical disease, the evidence should be regarded as overwhelmingly in favor of the harmlessness of these agents" (Koprowski 1960, 975). Once Koprowki's lab developed a human diploid vaccine strain made of fetal tissue, his opinion changed, and he subsequently advocated against the use of monkey tissues (Wadman 2017); this later advocacy was foregrounded in the Royal Society meeting.

My point is that the SV-40 scare could have led to a reconsideration of the fundamentals of the vaccine program: the conditions of monkey importation, including gang caging, sacrifice, and sterilization; the pooling of tissues; and the testing of tissue cultures for contaminants. It did not. While vaccine companies were allowed to use up their stocks of SV-40-contaminated vaccine, no plan was made for long-term testing of the ten to thirty million Americans who now carried SV-40; and the press did not cover the virus. The scientific literature since then has generally accepted that SV-40 was benign to humans, or at least that no immediate and noticeable effects were evident. Significantly, those who have carefully tracked the studies on SV-40's potential impact on humans find that the research done was insufficient to rule out rare or chronic illnesses, or those that present later or in future generations (Lewis 1973).

CONCLUSION

Eve Sedgwick began her famous article on paranoid thinking by asking what we would know differently if we knew the origin of AIDS—if we knew, say, that AIDS was a result of medical or military experimentation. Sedgwick quotes the noted historian Cindy Patton, who argues: "Even suppose we were sure of every element of a conspiracy: that the lives of Africans and African Americans are worthless in the eyes of the United States; that gay men and drug users are held cheap where they aren't actually hated; that the military deliberately researched ways to kill noncombatants whom it sees as enemies. . . . Supposing we were ever so sure of all those things—what would we know then that we don't already know?" (Patton, as quoted in Sedgwick 2003a, 123).

From the perspective of Patton and Sedgwick, there is nothing surprising about the general contour of the events tracked here, from the conditions of the vaccine trial itself to the virtual, and multi-sited cover-up of even the possibility that a viral transfer could have, in theory, taken place. In that view, racism and homophobia are so intractably part of the way that the events and their entry into the historical record took place that even to uncover the truth of those biases cannot change the narrative of the science history. Based on my reading of the events, this analysis is plausible. It's hard to find another explanation for why the research has not been undertaken to more thoroughly investigate the origins of AIDS, albeit in academic systems that reward short turn-around times and at best semi-controversial findings.

Still, the labeling of the OPV hypothesis as "conspiracy theory" has resulted in a missed opportunity to read *The River* as a detailed account of the conditions of possibility underlying the vaccine project writ large, and the immense social, political, technological, and interspecies infrastructure on which the vaccine project relied in its reorganization and intercalation of animals, humans, and viruses. At the very least, Hooper's magnificent research gives us a starting point from which to attempt to trace the complex fragility and the enormous risks that were undertaken in twentieth-century vaccinology. In the late 1990s, potential failures seemingly had to remain invisible. This is no longer the case. And so, while the origin of the HIV epidemic is not particularly controversial, perhaps it should be.

NOTES

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1 The explanation relies on an enormous coincidence—that crossovers happened around the same time as at least five main cases of HIV, with at least two types of primates and in different areas of the continent—despite thousands of years of butchering and eating primate meat during which such crossover did not occur—and that HIV then lay dormant or unnoticed for decades.

2 I use cut hunter and natural transfer theories here interchangeably as the main hypothesis of phylogeneticists.

3 Lochlann Jain, "The WetNet: What the Oral Polio Vaccine Hypothesis Exposes about Globalized Interspecies Fluid Exchange," *Medical Anthropology Quarterly* 34, no. 4 (December 2020): 504–24.

4 Edward Hooper, "The Origin of HIV-1 Group M: The CHAT Polio Vaccine Theory," presentation at the Origin of HIV and Emerging Persistent Viruses conference, Accademia Nazionale dei Lincei, September 28, 2001, https://pages.ucsd.edu/~jjmoore/publications/hivhooper2001b.html. Hooper explains why Paul Osterreith's claim that the lab was not sophisticated enough to make tissue cultures is inconsistent with other evidence.

5 American interests and activism in the Congo remained heightened both because of Cold War strategic reasons and Russian presence in the region, and because of the mineral-rich geography. Neil Ahuja suggests that the involvement of the chimpanzee lab in Stanleyville may have been a Cold War pawn in the early 1960s (Ahuja 2013).

6 Koprowski, on the other hand, lived to be ninety-six and vigorously and litigiously shut down debate on the OPV hypothesis.

7 Hooper discusses his method of triangulating information sources. For example, he quotes an interview with a worker from one of the research labs who said he vaccinated locals in Butare with Wistar's vaccine in 1957. Hooper corroborates this with interviews of community members in eight villages around Butare, finding "two old men [who] independently told us that they recalled oral vaccinations against *mbasa*, or polio." These data are then linked to the epidemic: in 1984, 88 percent of prostitutes in Butare were HIV positive, "an extraordinary percentage for so early in the AIDS epidemic" (Hooper 2001, 806).

8 My own efforts to gain funding for such a project were unsuccessful.

9 "In 2001, I jumped off the fence on the polio vaccine hypothesis in favour of 'disproved.' . . . But I am open to persuasion that my conclusion was premature" (Robin Weiss, email to the author, November 12, 2017).

10 Robin Weiss, interview by the author, December 13, 2017.

11 Robin Weiss, interview by the author, December 13, 2017

12 Gottsdanker v. Cutter Laboratories [Civ. No. 18413 and 18414. First Dist., Div. Two. July 12, 1960.] 182 Cal. App. 2d 602 (Cal. Ct. App. 1960). https://casetext.com/case/gottsdanker-v-cutter-laboratories.

13 The monkeys, imported from India, were gang caged in transportation, thus enabling the SV-40 virus to spread among the monkeys. Vaccine companies used different techniques to make the vaccines. Vaccines made with one kidney had a 20 percent contamination rate; those made with kidneys from two to three animals had a 70 percent contamination rate; and when ten or more animals' kidneys were used, the resulting vaccines had a 100 percent contamination rate. "Studies estimate that the vaccine infected between 10–30 million adults" (in itself a tellingly vague estimate), and that "potentially contaminated vaccine had been administered to almost 90% of individuals under 20" (Shah and Nathanson 1976, 5).