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Dephlogistication,* Imperial Display, Apes, Angels, and the Return of
Monsieur Émile Zola.

New developments in the origins of AIDS controversy, including some observations
about ways in which the scientific establishment may seek to limit open debate and
flow of information on "difficult" issues.

Edward Hooper

Abstract.

Since the mid-1950s, in laboratories around the world, oral polio vaccines (OPVs) have been routinely amplified in locally-prepared primate kidney cell cultures, before being diluted and fed by mouth to local populations. During the fifties, it was possible for a virologist to prepare polio vaccines in the primate cells of his or her choice: to do this contravened neither scientific rules nor recommendations of the day. Some OPV developers (such as Albert Sabin and collaborators) reported the fact that local preparation of the vaccine had occurred in the country of use, such as the U.S.S.R.; while others (such as Hilary Koprowski and collaborators) reported

* According to the Oxford English Dictionary, this rather impressive word means a "depriving of phlogiston", or a "relieving of inflammation". It is used in both contexts in the course of this paper, but overall I prefer to think of it in a slightly wider context – that of a "reduction in hot air levels".

Additional note: The year 2000 paperback editions of *The River* contain fully rewritten postscripts, and page references from page 827 onwards are therefore different from those in the 1999 hardback editions. All references in the current paper refer to the 2000 editions, which are henceforward referenced merely as "*River*, 2000". Because the figures in the new postscript were inserted on different pages in the US and UK paperback editions, references to passages in the postscript are sometimes one page earlier or later than specified.

only that their vaccines had been diluted, with no mention of local amplification. However, Dr Koprowski has since stated that his polio vaccines *were* amplified, both in Europe and elsewhere, and evidence from several sources indicates that amplification of his Type 1 OPV, CHAT, occurred in the Belgian Congo during the 1957-60 period. In particular, one witness has reported that an oral polio vaccine was being made in the virology department of the *Laboratoire Médical de Stanleyville* (LMS) in February 1958, and that he personally helped administer this locally-made CHAT vaccine by mouth to local African populations, including the inhabitants of a military camp.

Evidence acquired from numerous sources over a period of years reveals one other unique detail about the *Laboratoire Médical de Stanleyville*. From 1956 (at the latest), the major departments within that lab were routinely using tissue cultures prepared from chimpanzee cells (often with chimpanzee sera employed as growth medium). No other type of tissue culture is mentioned by these sources until a few tubes and bottles of baboon tissue culture, which were prepared experimentally in the middle of 1958. Until now, tissue culture and polio vaccine preparation at the LMS has been a subject surrounded by secrecy, for which documentation was either missing or misleading. What this new evidence strongly indicates is that the oral polio vaccines distributed by the LMS up to at least the middle of 1958 (save for 2,000 doses brought from the US in capsule form) were given a final passage in chimpanzee cells.

The fact that the closest known relative to HIV-1 Group M is the simian immunodeficiency virus of the common chimpanzee (SIVcpz), raises the question of whether this might be the way in which SIVcpz transferred to humans to give birth to HIV-1 Group M, and thus to the AIDS pandemic. The LMS chimp tissue cultures were primitive Maitland-type cultures, made without trypsin, and were thus prepared in a fashion which (a) would have failed to inactivate any SIVs that might have been present, and (b) would have facilitated recombination between individual SIV strains. This new evidence about the local preparation of CHAT vaccine in the Belgian Congo lends significant support to the so-called “OPV hypothesis” of AIDS origin.

Several different lines of evidence, presented here, offer further support to the hypothesis. Between 1956 and 1959, some 500 common chimpanzees (*Pan troglodytes*) and 80 pygmy chimpanzees or bonobos (*Pan paniscus*) were sacrificed at a chimpanzee research camp at Lindi, an isolated spot in the bush 15 kilometres from

Stanleyville. Although it has been claimed that much of this research involved testing the safety and immunogenicity of CHAT vaccine, virtually no significant details about the research have ever been published. Three African eye-witnesses say that the man who regularly extracted organs and blood from these chimpanzees and bonobos was the head of the Stanleyville virology department. In 1958, an outbreak of fatal *Klebsiella pneumoniae*, one of the opportunistic infections of both AIDS and simian AIDS, was reported at Stanleyville hospital. A similarly fatal *Klebsiella* outbreak had previously taken place at the isolated and quarantined chimp camp. It is proposed that the common denominator was a batch of CHAT vaccine prepared locally in chimp cells, and fed to local people.

Dr Koprowski's base for the polio vaccine research was the Wistar Institute in Philadelphia, of which he formally became director in 1957, although his links with that institute appear to go back several years before that. Between 1953 and 1957, a group of scientists at the Wistar, funded mainly by the U.S. Army Chemical Corps, worked on the mass production of viruses (notably polioviruses) in different substrates, such as human amnion cells, HeLa, and other cell lines – many of which, we now know, had been taken over by HeLa. The same and similar human cells and cell lines were in use in several labs in the Belgian Congo from 1954 onwards. According to a contemporary report, CHAT vaccine was being prepared in one of these Congolese labs (a place where *only* human cells were used for cultures) from around August 1958 onwards. On the basis of this and further evidence, it is proposed that some of the CHAT vaccine that was made in chimp cells in Stanleyville may later have been further passaged in human cells (including HeLa), with potentially even more serious implications for human health. This “HeLa addendum” is more tentatively proposed than the main OPV theory. It is not fundamental to that theory, but should be viewed as an adjunct, which (if substantiated) may help to explain the uniquely dreadful impact of this particular HIV strain (HIV-1 Group M) in human populations.

There are striking coincidences of place and time between the 1950s feedings of CHAT vaccine in Africa, and the first appearances in the world of HIV-1 Group M and pandemic AIDS. The earliest cases of Group M-related AIDS in Africa through 1980 come from the DRC, Rwanda and Burundi, these being the three African countries where CHAT was field-tested in the fifties, in 27 known campaigns. In fact, 68% of the earliest recognised cases come from the specific places where CHAT was

fed. Significant corroboration is afforded by the fact that the earliest evidence of Group M infection comes from the DRC and Burundi, with 76% of all recorded African Group M infections up to and including 1981 coming from the same towns and villages where CHAT is known to have been fed. These coincidences of place and time would be remarkable by themselves; but allied to the hitherto unrevealed information about local CHAT preparation in Stanleyville, they assume a real and ominous significance.

Several scientific arguments have been put forward to “disprove” the OPV/AIDS theory of origin. Most notable are: i) that phylogenetic dating analysis allows one to trace AIDS back to the 1930s (before the time of the African OPV trials); ii) that SIV would not survive the vaccine-making process; and iii) that the immediate viral ancestor to HIV-1 Group M comes from a “different chimpanzee”. The author reviews a dozen or more of these arguments. Closer analysis of the evidence reveals that many of these alleged disproofs are untenable, while the others are much weaker than has previously been claimed. To date, there is not one compelling argument against OPV/AIDS, although a *bona fide* sample of HIV-1 Group M from before the date of the OPV trials would constitute powerful evidence.

A great deal of effort has been devoted by both senior scientists and the major scientific journals to trying to persuade the scientific community and the general public that the OPV/AIDS theory has been “destroyed” by facts and evidence. In reality, the theory is shown to stand stronger than ever. An analysis is made of why most mainstream scientists find the OPV/AIDS theory so difficult to accept, and why there has been such a virulent response to the theory in certain quarters. The present author has been accused of inventing witnesses, misrepresenting evidence and lying. Evidence (both documentary and recorded) exists to refute each of these allegations, and the author invites those who have made the charges to review this evidence with him. The article ends with the author, assisted by Monsieur Émile Zola, making a series of charges of his own about the iatrogenic (physician-related) origins of AIDS, which the relevant protagonists are invited to refute.

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1. Introduction: “experts” and expertise.

Claims of expertise have been ringing down the ages since the snake first hissed into Eve’s ear, offering her a bite from the fruit of the Tree of Knowledge.

I have been assailed by claims of superior expertise since my book *The River*¹ (which proposed that an experimental oral polio vaccine, or OPV, might have sparked the AIDS pandemic) first appeared in the autumn of 1999. This is perhaps not altogether surprising, in that I am a writer, not a scientist, and there are very many scientists who surely should know more about this issue than I do.

To begin with, the most prominent such “expert” was an individual, Dr John P. Moore, who has worked for many years in immunology and AIDS vaccine research. Moore has a reputation for being academically able, and for turning a good phrase. However, he has also cultivated another reputation: that of an activist who makes robust and forthright contributions to scientific debates, specifically those concerning AIDS. He is less universally appreciated in this latter role. I have heard him referred to variously as a man with “his own agenda”, and as “an attack dog for the science establishment”.²

Whatever, Dr Moore began writing responses to my book. He wrote to newspapers, he wrote to his fellow-scientists, he wrote a review for *Nature*, and another for the Amazon web site. And he claimed that wonderful thing – expertise.

What he wrote, in effect, was: I am a scientist; Hooper is a journalist; I say the OPV theory is wrong; he says it's right; who are you going to believe? (The fact that I had not been a journalist since 1987 did not deter him; what was important here was that I should be pigeon-holed and dismissed.) And then, in each of these letters and reviews, he offered a little jokey metaphor. He likened the theory that an oral polio vaccine might have started the AIDS pandemic to the theory that the moon was made of cream cheese, that there was a monster at the bottom of Loch Ness, that there were little green men on Mars. And that was it. That was the basis of his argument.

There was only one problem. Dr John P. Moore might legitimately be considered an expert in AIDS vaccine development. But he had absolutely no expertise concerning how AIDS might have begun. He had never had anything published about it; had never (as far as can be discovered) spent any time researching it. His extensive knowledge and experience in other areas of AIDS research should have provided him with some decent perspective. But when his detailed arguments on the origins debate were revealed, such as they were, they were not impressive.

However, because he was a scientist, with letters after his name, and because he had worked in the field of AIDS for several years, he felt he had the right to claim that he was right. And many other scientists believed him.

In March 2000, the leading scientific proponent of the OPV theory, the universally respected evolutionary biologist Bill Hamilton, died tragically after a second expedition to the Congo to try to collect data that might throw more light on the origins of AIDS debate.

Almost as soon as Bill was interred, the experts emerged in droves. They let it be known that the theory would soon be dismissed by compelling scientific arguments. They effected a postponement of the Royal Society conference on “Origins of HIV and the AIDS epidemic”, in order that “more scientific data could be collected”, and during that postponement the balance of the meeting was adjusted a little here and there, so that the expertise of certain favoured colleagues could be presented in a more favourable light. And then, soon after that meeting, they claimed that they had looked at the OPV theory freely and fairly and that, through their expertise, had discovered some fatal flaws. Later, one of their number (Professor Robin Weiss, one of the organisers), even claimed to much publicity that the theory had been “destroyed”.

How did he know? He knew because he, too, was an expert.

Dr Weiss is a highly intelligent man. However, if you care to spend a little time looking at his arguments on the origins of AIDS from a purely scientific perspective, many of them are not quite as persuasive as they initially seem. Almost invariably they are dressed up nicely, but they sometimes contain more assertion than true science. In fact, on closer examination, some of these arguments seem little better than those propounded by his former student, the aforementioned Dr John P. Moore.

I am not an expert. However, I have studied different aspects of AIDS for sixteen years now, for the last twelve of which I have looked fairly seriously into its possible beginnings. During that time I have been lucky – I have had many tutorials from wise and knowledgeable scientists. I have educated myself as best I can by interviewing many of the protagonists, and by reading everything relevant I could get my hands on, sometimes several times. Those who have read *The River* with open minds – and they are, I believe, the majority of its readers – generally recognise that I am, at the least, an honest and painstaking researcher. I believe that I am well-steeped in the facts and arguments pertaining to this debate. But I do not call myself an expert.

In fact, I have learnt to be wary of those claiming expertise. They aren't always what they seem to be. Especially if they claim that they have *proved* themselves right, when actually they have not. Especially when one of them (Professor Weiss) begins to get a little wild, and starts to imply that the other party (myself) is not only a non-expert, but that he is also deluded, or dishonest, or that he has invented witnesses. When people start to tell untruths like that, then the whole process becomes something else again. It becomes a piece of political theatre, a charade.

In this paper, I (the non-scientist, the non-expert) am proposing a controversial hypothesis, but one which I believe to be supported by good science. Many of the experts (like doctors Weiss and Moore) dispute this. And so what I have to do is not the easiest of tasks. I have to try to persuade my readers that the experts (well-intentioned though many of them may be) are not necessarily always right.

The truth is that Dr Weiss has grown used to taking on the role of the arbiter, the judge who weighs all arguments carefully and then determines which is right, or which is more likely to be right. But, like all men and women, he has his own favourites, his own prejudices, his own bias. And over the years he has grown used to wielding considerable power behind the scenes – to being both a scientist and a politician. It took me quite a while to realise it, but some of his arguments are based as much on political pragmatism (and on working for what I suspect he considers to be “the greater good”), as they are based on purely scientific reasoning. This is something to which I shall return later in this paper, but it is something which, I think, needs to be borne in mind from the start.

So I would invite all those who really care about the truth in this matter, who still believe that the origin of AIDS is an important issue (and one that can teach us much), to lay aside preconceptions about who has letters tagged on their name and who has not; about who has been published in *Nature* and who has not. And instead to listen again, as if for the first time, to the arguments that the different sides are able to put forward. And then decide – what makes sense? What can be supported? And what is based merely on an assertion of superior expertise?

2. Background: the origin of HIV-1 Group M: transfer via African cuisine, or modern medicine?

By 2002, the AIDS pandemic had caused the deaths of over 20 million people world-wide, and the infection of more than 60 million with the causative virus, HIV-1 Group M.³

In July 2002, UNAIDS issued a report which estimated that 68 million persons would die in the world's 45 most affected countries in the next twenty years. No estimate was advanced for the rest of the world, although it was noted that in many of these "lower-risk countries", HIV had now moved beyond specific risk groups, and was spreading at an accelerated rate in the general population.⁴

The syndrome now represents the gravest threat to human health in recorded history. But what started this dreadful outbreak of a disease which has swept across the planet, but which was unrecognised just 21 years ago?

a) The two main hypotheses.

Professor Bill Hamilton probably had the highest standards of probity of anyone I have ever known. He was (as is widely recognised) softly-spoken, modest and generous to a fault. But there was also another side to Bill – less familiar save to those who knew him well. He could be assertive (sometimes explosively so) when he felt that a wrong was being done, or that an untruth was being presented as a truth. I am confident, therefore, that Bill would not disapprove of the robust paper which follows.

Between 1993 and 1999, Bill and I worked together closely on the question of how AIDS might have started: I collected new information and evidence, while he provided comment, guidance and good counsel. We knew each other well.

In August 1999, after nine years of research and writing, *The River* was published, and near the start of the book I examined more than fifteen hypotheses about how the AIDS pandemic might have begun.

I proposed that the field could actually be narrowed down to two competing theories – one involving a "natural" zoonosis, and the other involving an iatrogenic (or physician-caused) event. Both theories sought to explain how humans had become infected with the simian immunodeficiency virus (SIV) of the common chimpanzee (*Pan troglodytes*), which for several years now has been widely recognised as the only really close ancestor to the major HIV-1 variant, Group M.⁵

I referred to the more widely accepted theory of origin as the "natural transfer" or "cut hunter" theory. As espoused by its leading proponents,⁶ this theory proposes that someone who hunted, skinned, ate or played with a chimp, became infected with chimpanzee SIV (SIVcpz) in or around the 1930s, somewhere in the west central African region of Cameroon, Equatorial Guinea, Gabon and Congo Brazzaville. It further proposes that the initial infectee, or someone infected by him/her, ended up not long afterwards in Leopoldville (now Kinshasa, the capital of the DRC, the Democratic Republic of Congo). There, the theory goes, transmission to other parties began, and the new human virus then began to spread up the river Congo to infect people in other parts of the DRC and, by the very end of the seventies, in other parts of Africa.⁷

The most plausible iatrogenic theory of origin, and the one which I came to favour, is called the oral polio vaccine (OPV) hypothesis. It proposes that the AIDS pandemic was sparked by the vaccination of approximately one million Africans with an experimental OPV called CHAT. The 27 known African trials⁸ of CHAT vaccine

occurred between the years of 1957 and 1960, in the central African countries now known as the DRC, Burundi and Rwanda. In the fifties, these lands comprised a colony (the Belgian Congo) and a trust territory (Ruanda-Urundi) administered by Belgium.

By the latter half of the 1950s, virtually all polio vaccines were prepared in a substrate of so-called “monkey kidney tissue culture”. Most of the major polio vaccine developers – such as Sabin, Salk, Cox, L epine and Gear – identified the species they were using in one or more of their early publications. The first three used rhesus and cynomolgus macaques from Asia; L epine mainly used Guinea baboons from West Africa, and Gear used vervets (a type of African green monkey) from South Africa. Alone of the major manufacturers, the developer of CHAT vaccine, Hilary Koprowski, never revealed the species of primate he had used to prepare CHAT in any publication of the fifties.⁹

For several reasons, I began to suspect that Koprowski and/or his collaborators might have used cells from the common chimpanzee to make some of the CHAT vaccine batches fed in Africa.

The first reason was that a short while before he developed CHAT, Hilary Koprowski had inaccurately reported the substrate in which he had prepared his previous Type 1 polio vaccine, SM N-90. In three articles published in 1956 and 1957, Koprowski reported that he had been using a tissue culture of chick embryo, when in fact he had been using one of monkey kidney.¹⁰ The reason for this misrepresentation is still not clear.

A second reason was that, at the time he developed CHAT, Koprowski – in collaboration with researchers from the Belgian Congo – had just opened a huge chimpanzee research station (*Mission Courtois Koprowski: Centre d’Experimentation*)¹¹ at a place called Lindi, about 15 kilometres outside Stanleyville (now Kisangani). There are virtually no details in the published literature about the polio work which was carried out at Lindi camp, and despite claims to the contrary,¹² that research was conducted under a veil of secrecy.¹³ However, before long I learnt that more than 400 chimpanzees had been present at the camp during the time of the polio research, in the first twenty months of its existence (June 1956 to February 1958). By the time it closed, in December 1959 or January 1960, more than 600 chimpanzees may have been utilised in the Lindi research.¹⁴ It is reported that 86 of these were pygmy chimpanzees (otherwise known as bonobos);¹⁵ the rest were common chimps.

But the most compelling reason involved the epidemiology of HIV and AIDS. Quite early in my research, I began recording the earliest cases of AIDS, both clinically plausible and serologically confirmed, which could be found in the medical literature, or else in documents such as doctoral theses. The following data have been updated to include those cases that have recently come to light.

Apart from one early cluster involving a Norwegian sailor and his family (all of whom turned out to have been infected with a minor variant of HIV-1, Group O, in the 1960s),¹⁶ the first plausible evidence of AIDS outside Africa (in the U.S., Europe

and Haiti) emerged in the year 1978.¹⁷ All these latter cases seemed likely to have been caused by the pandemic strain of HIV-1, Group M.

But within Africa, the epidemic began *at least five years earlier*. I was able to document fifteen clinically-defined African cases in which first symptoms appeared between 1973 and 1977, as well as one other case dating from 1962.¹⁸ (I shall return to the question of other possible early AIDS cases, from the sixties and fifties, later in this paper.) Interestingly, every one of the sixteen cases from 1962-1977 involved people from the DRC, Burundi and Rwanda, or else foreigners infected there.¹⁹

For the period 1978-1980, I documented 23 further African cases, mostly from the same three former Belgian colonies.²⁰ It was only in the latter two years that a few cases began emerging in adjoining countries, like Uganda, Tanzania and Zambia.²¹ About a quarter of these 39 clinically-defined AIDS cases were serologically confirmed.

It was in the middle of 1992, some time after I had carried out the greater part of the above research, that I first heard about the OPV/AIDS hypothesis, which was then being propounded by independent researchers such as Louis Pascal, Blaine Elswood and Tom Curtis.²² Some further research in Belgian archives revealed that many of the places where CHAT had been administered in Africa were, ominously, the very places where HIV-1(M) and AIDS had first appeared. **[Figure 1]** However, there were no such correlations in Europe, where more than eight million people had been fed with CHAT, in countries which had not witnessed any early cases of AIDS.

Some years later, I plotted out the vaccination sites, together with early African AIDS cases up to and including 1980, on a map of the old Belgian colonies, and discovered a remarkable fact. Thirty one of these cases could be linked to a specific city, town or village, and of these, 68% came from places where this experimental vaccine, CHAT, had been fed in the 1957-1960 period. **[Figure 2]** By contrast, 42% of the early cases came from Kinshasa, the city that features in both major theories of AIDS origin. (It was a CHAT vaccination site, but it is also viewed as a hub in the natural transfer theory.)

I then mapped the earliest serological evidence of HIV-1 Group M infection in Africa for the years up to and including 1981. Altogether there are sixteen sites in Africa for which we have evidence that HIV-1 Group M was present between 1959 and 1981,²³ and nine of these sixteen sites are places where CHAT vaccine was fed in 1957-1960. **[Figure 3]**

Through 1981, there are 70 instances of African Group M infection that can be linked to a city, town or village, of which 30% come from Kinshasa. However, there is a far stronger correlation, for 76% of these infected sera were obtained from places where CHAT vaccine was fed in the late fifties.

On the basis of such correlations, and of considerable further circumstantial evidence, I proposed in *The River* that batches of the vaccine fed in Africa *might have been prepared* in chimpanzee cells which contained SIV, and that the 27 CHAT vaccination campaigns conducted before independence in the DRC, Rwanda and

Burundi might have allowed chimpanzee SIV to infect one or several of the vaccinees.

b) The historical, scientific and political debates.

As I say, *The River* was published in August 1999, and by December of that year it was evident that the book had sparked a major controversy.

During the last two months of 1999, Bill Hamilton approached the Royal Society in London asking it to host a conference about the origins of HIV and AIDS, and the implications for modern medicine. (At around the same time, he was approached by the *Accademia dei Lincei*, and invited to explain his point of view at a similar conference.) Before he left for Kisangani in January 2000, the London conference had been approved, and two co-organisers appointed. What happened after his tragic death in March 2000 is a complicated story, and one that will be touched on later in this paper. Suffice it to say that after Bill's demise, the balance of the meeting became subtly but irrevocably tilted against the OPV theory.

When the London meeting took place in September 2000, the results of the testing of certain archival CHAT samples released by the Wistar Institute were announced: they were found to contain neither HIV, SIV nor chimpanzee DNA. In vain did I argue that the tests were relatively meaningless, because there was nothing to indicate that any of the samples which had been released, and tested, came from batches that had been prepared for use in Africa.²⁴

Most of the scientific and lay press overlooked this point, and concluded that CHAT vaccine had been vindicated. These conclusions were based to a large degree on a press conference which was staged immediately after the announcement of the Wistar test results, and on Professor Weiss's closing speech at the conference – and they were further reinforced seven months later, when *Nature* and *Science* published three brief formal reports of the CHAT vaccine testing, together with a short theoretical article about Group M phylogenetics.²⁵

“Disputed AIDS theory dies its final death”, ran the headline in *Science*.²⁶ “Polio vaccines exonerated”, was the title of Robin Weiss's commentary in *Nature*, which ended with a memorable sound-bite. “Some beautiful facts”, wrote Weiss, “have destroyed an ugly theory”.²⁷

I believe this was an irresponsible claim, for very few relevant facts (beautiful or otherwise) had been presented, and no theory had been destroyed.

There is a reason why I can write this so confidently. By a strange quirk of fate, just two weeks before Dr Weiss published his comments in *Nature*, I was visiting Kisangani, where (much to my surprise) I learned that, more than forty years earlier, CHAT vaccine had been prepared in the local medical laboratory – and almost certainly in a culture of chimpanzee cells.

By itself, this new evidence could be presented in the space of a few pages. However, so much disinformation and untruth has now been written in response to the OPV hypothesis, that the waters have become comprehensively muddied. Indeed, I believe

that a deliberate attempt has been made to obfuscate the issues, and that it is important that this obfuscation should be revealed for all to see. So, in order to answer the various claims (serious and spurious) which have been made, and to clarify some of the issues that have caused confusion, I have had to enlarge dramatically the scope of this paper. I have decided to present a great deal of new material, and I shall now be examining not just the scientific arguments, but several other aspects of the debate as well. I am grateful to the Lincei academy for giving me the opportunity to present such a detailed paper.

Unravelling the truth of what actually happened in the Belgian Congo nearly fifty years ago has not been an easy or straightforward process. Throughout the last decade, I have approached it through painstaking research, careful cross-checking and, above all, by a conscious attempt to avoid jumping to premature conclusions. (Doctors Plotkin and Koprowski claim that I have done exactly the opposite.)

Although I have looked at relevant materials from several different fields of science, it is not only science that has provided relevant information. Other important evidence has come from historical archives and testimonies, and, because the debate has become steadily more politicised in recent years, more again has come from what might be termed the political arena.

The remainder of this paper will therefore present the case for the OPV/AIDS hypothesis in three separate sections, relating respectively to the historical, scientific and political debates about how AIDS began.

3. The historical debate: was CHAT made in chimp cells?

This section will feature a large amount of newly-gathered historical evidence, much of which has only come to light since I delivered my address at Lincei in the autumn of 2001. This new evidence (much of it from Africa and North America) has allowed several key pieces of the historical jigsaw to slot into place, and has (I believe) moved the hypothesis that CHAT was made in chimp cells from the realms of the possible to those of the highly probable. The process has also revealed the first few pieces of a secondary jigsaw, one for which far fewer pieces have been located. It remains to be seen whether enough new pieces of this secondary jigsaw come to light to move the subsidiary hypothesis (that other cells may also have been involved with the preparation of the Congo vaccines) from the realms of the possible to those of the likely.

On my first visit to Kisangani, with Bill Hamilton in June 1999, I only managed to locate one significant witness to the events at Lindi camp, forty years earlier. However, on my second visit to Kisangani, in April 2001, I was present in the city for a longer period of time, and enjoyed more success in tracking down people who had participated in, or witnessed, the events that took place in the fifties.

[*A brief note on names.* In the revised postscript to *The River*, I referred to the 1999 witness from Lindi as “Antoine”, clearly indicating by the quotation marks that this was a pseudonym.²⁸ Dr Plotkin has since disingenuously reported that a search by African doctors in Kisangani failed to reveal an “Antoine” who fitted the bill, adding that this discovery “leaves open the question of where, how or whether the interview

was done”.²⁹ This false implication that I invented a witness does Plotkin no credit, although it is not atypical of the approach he has used in responding to *The River*.³⁰ The main reason why I avoided giving “Antoine’s” real name, was that I suspected that pressures might be brought to bear on him to modify his account – and indeed, this is exactly what has happened with other witnesses to the events recounted in *The River*; (for examples, see below). In an attempt to forestall this, I am continuing to refer to him, and to several of the other African witnesses, by pseudonyms or by descriptions only (e.g.: Osterrieth’s first assistant) in the text that follows. The identities of all these persons will be revealed at a later date.]

a) The Lindi camp nurse.

First I came across Joseph, the so-called “nurse” of Lindi camp, the man who had helped the Belgian doctors perform autopsies on the chimpanzees. (This came as a very pleasant surprise, because in 1999 I had been informed by “Antoine” that Joseph had died in 1964.)³¹ Joseph had been the key indigenous worker at Lindi camp, and was treated as boss by the other Africans who worked there. It was he who had sacrificed many of the chimps, opened them up, and had then carried out most of the initial gross dissections.³² He said that altogether, in the three and a half years of the camp’s existence, Lindi had housed over 600 “chimpanzees”, a term which includes both common chimps and bonobos; [see below].³³ He said that the chimpanzees were held in cages in two large hangars, and that all were eventually sacrificed, apart from the relatively small number which died of natural causes, and apart from the final 60, which were taken elsewhere when the camp closed.

Joseph said that he himself had probably sacrificed some 500 chimps. Sometimes he killed two or three in a single day. The work, he said, was secret: nobody outside the camp knew what was going on. There were some policemen based at the camp to make sure people stayed away.

For most of this time, he said, the doctor performing the autopsies had been Paul Osterrieth, the head of the new virus laboratory that had opened in Stanleyville in September/October 1957. During these autopsies, which were carried out on a table behind the cages in the second hangar, the major organs (including the kidneys) were taken away in metal containers – and were then, he thought, sent abroad, mostly to America. He said that the kidneys were always taken, as were large quantities of blood. Afterwards, he said, he had to pour poison on what remained of the corpses, so that local people would not eat them.

Many of the significant details of Joseph’s testimony were supported by other witnesses – including “Antoine”, who also added some other important details. During a further interview given in 2001, “Antoine” once again mentioned that there had been a group cage for the younger chimps. And he reiterated that organs and blood had been taken from the chimps while they were anaesthetised but before they were killed. The one documentary record that exists of the process of sacrificing chimps at Lindi to obtain kidneys also mentions that the kidneys were removed using “aseptic precautions”,³⁴ clearly to minimise the possibility of bacterial contamination.

These observations by Joseph and “Antoine” are crucial, because contemporary references confirm the importance of these procedures (the bleeding of animals, and

the taking of organs from anaesthetised animals, before sacrifice) when organs such as kidneys were being removed specifically for tissue culture work. One example features in a 1955 article from the von Magnuses, who carefully describe how they prepared the Danish polio vaccine (an IPV) that was used in 1955. “The kidneys”, they wrote, “are removed aseptically from monkeys exsanguinated under sodium pentobarbitol anesthesia.”³⁵

b) The assistants at the Stanleyville virology laboratory.

Perhaps the most important part of the new *Laboratoire Médical de Stanleyville* building which opened in Stanleyville in September 1957 was the virology department (or, as it was commonly known, the virology lab, or virus lab). Over the next few days, I spoke with two of the men who had worked in this virus lab, under Dr Paul Osterrieth, between 1958 and 1960.

The first of these assistants³⁶ had been speaking about his former boss for several minutes when he quite casually volunteered: “and he was also making the polio vaccines in the laboratory”. Hiding my astonishment, I asked how this had been done. To begin with, the assistant responded by talking about Osterrieth’s visits to Lindi camp to take blood from the chimps, and his spinning the blood down into serum when he got back to the lab. I asked him again how Osterrieth had made the polio vaccine, and he replied: “I was just sterilising the materials in the lab. What he was doing with that blood to make vaccine I don’t know.” He went on to explain how he used regularly to accompany Osterrieth to Lindi on Saturdays, when the two of them would take blood from the chimps, using different syringes for each animal. He knew nothing about autopsies or the taking of organs, saying that it was the camp workers at Lindi who should be asked about those subjects.

In another answer given later in the interview, the assistant once again linked the making of chimp serum to the making of polio vaccine. He was unable to provide further details, but the very fact that the two events were associated in his mind seemed significant.

When I asked for more details about the occasions when the vaccine was made, he said that what happened first was that an order for more vaccine would come from the “provincial director”. After that, he said, Osterrieth would start preparing the vaccine, often staying behind in his lab in the evenings, after the other workers had gone home. (This was Osterrieth’s own lab, he added, the one which was kept sterile, and which nobody else was allowed to enter. This was also where Osterrieth used to take the bloods from the chimpanzees to spin them down into sera.) Osterrieth was not making the vaccine all the time, he said. It was only after the orders came from the provincial government that he would do so. The taking of serum, by contrast, happened on a regular basis, which suggests that although the vaccine-making may have required serum, this was probably not the only reason that serum was taken.

Later, when Osterrieth had finished preparing the vaccine, the assistant would help transfer it into smaller bottles, which sounded like phials. There was a “machine” to help them do this, he said. Sometimes he (the assistant) also helped administer the vaccine that Osterrieth had made – and this was always done by mouth. Apparently

the vaccinations continued up to 1960, and he added that the other doctors in the lab all knew that Osterrieth was making the polio vaccine.

One particular vaccination the assistant recalled was that at Lukusa, the military camp of Stanleyville, on the south side of the river, where he helped feed the vaccine. This was significant because, unusually, the vaccination at this site was documented in a letter in the Belgian government archives. Over 3,000 men, women and children were vaccinated here on February 27, 1958 – this being the eighth anniversary of the date when Hilary Koprowski, the developer of CHAT, became the first scientist to feed an OPV to a non-immune subject.³⁷

The assistant had started working at the laboratory on February 12, 1958, and he emphasised that Osterrieth had already been making the polio vaccine before he arrived there. Osterrieth had been away on leave from July 1957 until about February 4th, 1958, so this indicates that he must have been preparing vaccine from around the time of his return to the lab, a week or so earlier.

The assistant also said that Dr Osterrieth used to send reports about the vaccinations overseas, and he recalled that Osterrieth made two further trips abroad before he finally left in 1960.³⁸

Shortly after my return from Africa, the proceedings of the Royal Society meeting were published,³⁹ and they included a contribution by Dr Osterrieth, entitled “Vaccine could not have been prepared in Stanleyville”.⁴⁰ In this article, and in statements quoted in articles by Stanley Plotkin,⁴¹ Dr Osterrieth made a series of definitive statements, some of which were clearly responses to different parts of *The River*.⁴²

He stated that after receiving training in tissue culture techniques in the USA between October 1957 and January 1958, he returned to Stanleyville in February 1958 with the aim of setting up a cell culture laboratory. He said that as far as he recalled, it took several months before he succeeded in producing cultures from baboon kidneys and from HeLa cells. He added that he tried to make tissue culture from the kidneys of other small monkeys, but failed, and that trypsin was uniformly used to disperse the cells. He stated that when he was not present in his laboratory, the room was locked, for fear of contaminating the tissue culture, and that nobody else had access to the virology lab. He stated that autopsies of chimps were never done inside the main medical laboratories, and went on: “I have no knowledge of polio vaccine being diluted or distributed into smaller flasks at the Stanleyville laboratories, and in any case it was never done in my laboratory.” He also stated: “There is no possibility that chimpanzee cells could have contaminated the vaccine that was produced elsewhere.”

He ended his statement to Stanley Plotkin with the following passage: “[A]t no time did I ever attempt to make cell culture from chimpanzee tissues. In addition, I wish to state categorically that no poliovaccine was ever produced or could have been produced in Stanleyville, since the facilities were totally inadequate for a production or control of poliovaccine.” (It is unclear whether the italics were contributed by Osterrieth or Plotkin.)

How are we to resolve the basic differences between these two accounts from the director of the Stanleyville virology laboratory and his assistant? Should we be more

impressed by Osterrieth's account, because it comes from a Western scientist, rather than an African lab technician?⁴³ Or should we compare the two accounts, and also, where possible, compare them with accounts from other sources? I propose to adopt the latter approach, which clearly seems fairer.

Firstly, it should be noted that there may be some confusion about terminology. According to his assistant, there was a sterile room within the virology lab which was always kept locked, and it was here that Dr Osterrieth did most of his work – like centrifuging the chimp blood to produce sera. So, when Paul Osterrieth writes about “my laboratory”, he may be referring either to this locked sterile room, or to the virology lab as a whole. It seems very probable that the sterile room is the same place as the lab which, according to Osterrieth, was kept locked to avoid contamination of the tissue culture.

Dr Osterrieth denies ever having handled polio vaccine in his laboratory. However, the assistant not only states that Osterrieth himself regularly handled polio vaccine in the lab, but he recalls his boss preparing it in the sterile room on several occasions, whenever an order came down from the provincial government.

Osterrieth says that the lab facilities were totally inadequate for the production and control of polio vaccine, but there is substantial evidence to counter this claim. For instance, a Leopoldville newspaper article from August 1958 specifies that Koprowski's polio vaccine had been both “prepared” and “controlled” in the Belgian Congo, and that the control, at least, had been carried out at the lab in Stanleyville.⁴⁴ Apart from illustrating the inaccuracy of Osterrieth's claim about controlling the vaccine, this also highlights the fact that it is not *production* that is at issue here, for the initial production step for CHAT had already been carried out in the U.S. The key issue is whether batches of CHAT could have been *prepared* locally – and it is becoming clear that this was quite a straightforward process, which could be achieved simply by placing a little of the existing vaccine into a new tissue culture.

The fact that the vaccines made by Osterrieth were fed by mouth is vitally important, because the only vaccine administered orally in the late fifties was OPV, and the only OPVs reported in the Congo at that time were the Koprowski vaccines, CHAT and Fox.⁴⁵

A document in the Belgian government archives reveals that CHAT was fed to 3,102 individuals at Stanleyville military camp on February 27th, 1958. Koprowski's type 3 vaccine, Fox, was fed to 3,131 people at the same camp on May 27th, 1958.⁴⁶ We don't know for sure whether the assistant helped with the February vaccination, or the May vaccination, or both. The important detail, however, is his evidence that Osterrieth was already making the vaccine in early February 1958, which confirms that *CHAT vaccine*, at least, was prepared and fed locally.

The account of new vaccine orders coming periodically from the “provincial director” corresponds nicely with a previous account provided by Dr Ninane, who talked of requests for vaccine coming in sporadically from local doctors – requests which clearly would have been channelled through the provincial medical directorate in Stanleyville. It also ties in with the annual reports of the Stanleyville medical laboratory. These reveal that during 1958 responsibility for “the control, storage and

distribution” of all vaccines passed from the medical laboratory to the Hygiene Department, and that its director, Dr E. Peeters, advised the *Medecin Provinciale* on such matters. This transfer clearly happened after the responses to polio epidemics in *Province Orientale* in January and February 1958, and after the Ruzizi Valley campaign in February to April, in both of which the doctors from the medical laboratory played the leading role. The Ruzizi trial actually took place outside *Province Orientale*, but Courtois helped organise the programme, while Ninane took charge of several of the feedings.⁴⁷

The testimony of the first assistant in the virology lab contains a number of precise details, which are all the more impressive because they come from someone who is not himself a specialist. Clearly he did not dream up these details, nearly all of which are corroborated by other sources. Examples include the fact that vaccine orders came in from the “provincial director”, that Osterrieth sent reports about the vaccine overseas, that vaccine was transferred into smaller bottles, that it was fed by mouth, and that it was administered at the local military camp. Throughout the course of two interviews the assistant spoke quietly, and yet confidently. I do not believe his testimony can be “explained away” as the product of a faulty (or over-imaginative) memory. Furthermore, there is no reason for his statements to have been fabricated.

By contrast, many of Dr Osterrieth’s various statements would appear to be qualified in one way or another, or else are capable of different interpretations – such as the references to “my laboratory”, or to “production and control of poliovaccine”. The main exception would seem to be the categorical and italicised denial that Dr Plotkin has reprinted.

At this point, let us turn to Lindi. I now have the detailed testimony of three African witnesses who spent time at Lindi camp, who say that Dr Osterrieth was a regular visitor to the camp, and that it was he who did most of the autopsies, and who carried out most of the extractions of organs and blood from the Lindi chimpanzees. By contrast, Dr Osterrieth has minimised the importance of his own role at Lindi camp. He has never acknowledged that the research involved the sacrifice of chimpanzees. Furthermore, he has never vouchsafed how the organs such as kidneys were obtained (for instance for Dr Deinhardt’s hepatitis work). In short, Dr Osterrieth has consistently avoided answering specific questions about the work conducted at Lindi.

There are further question marks, also. One of Plotkin’s articles quotes Osterrieth as stating that: “I never tried to dilute the polio vaccine that was received”. The article then reveals that Dr Ninane had apparently made the same statement, word for word, just six days earlier: “I never tried to dilute the polio vaccine that was received”.⁴⁸

On one level, this raises questions about the ways in which these statements were obtained. But it also begs a question – that of whether Osterrieth (and/or Ninane) perhaps diluted a *different* variety of polio vaccine – not the one “that was received” from abroad, but one which had been prepared locally by further passage in tissue culture. (As discussed more fully below, this would essentially conform to the normal process which took place in labs that received polio vaccine from overseas in the fifties.)

Two other interviews need to be mentioned. The first was with another of Osterrieth's lab assistants, who joined the lab in December 1958. Like the first assistant, this second man recalled that Dr Osterrieth had made vaccine, but said he could not recall which one it was. I wanted to avoid any possibility of prompting him, and so changed the subject for a while. Earlier, he had mentioned that he had recently had some news from Dr Osterrieth, so I asked him about this. He explained that a few months before, he had received a letter from Dr Osterrieth, the first communication for some 40 years. Why had his former boss written, I asked? Apparently Osterrieth had wanted to know which of his former African assistants at Lindi camp, and at the Stanleyville virology laboratory, were alive – and which were dead.

Later, I asked the second assistant if he had helped with the polio vaccinations that were taking place at around this time, and he became perceptibly nervous. Eventually, he answered: “yes, but I don't know well. I have forgotten. But I worked, I worked”. Later, he added that he had helped vaccinate children in Kisangani town during 1959, but could add no further information. 1959 was when the whole population of Stanleyville, some 15,000 people, was vaccinated with CHAT.

A further interview involved a technician who had worked under Ghislain Courtois in the microbiology department at the old *Laboratoire Medical de Stanleyville* from April 1956 onwards. The key revelation came when the assistant revealed that they had indeed been making tissue culture in the microbiology lab, and that he himself had prepared culture media..

When asked from which animals these tissue cultures had been made, he answered “*surtout des chimpanzés*”, or “mainly chimpanzees”. He gave this answer confidently and immediately. Later, he said that they were also making tissue cultures in Dr Ninane's histopathology department.

When asked how they were making these cultures, he said that it would be better to ask Joseph (the Lindi camp nurse), because they were doing autopsies of the chimpanzees at Lindi camp. Later, he added that they would take the lungs, the heart and other organs from the chimps, and put them into flasks, and then work with them, but he wasn't sure of the precise details. I decided not to press him with further questions on this issue because it seemed clear that he had given what he had to give – and I did not wish real memories (which these clearly were) to get confused by well-intentioned “attempts to remember”.

Courtois' assistant said that Dr Osterrieth had worked for a time in the microbiology lab, and had then left to become head of the virology lab (where, as we know, he took over after his leave, in February 1958). He said he did not know what they were doing in the virology lab, although he stated that the work on the poliovirus had involved chimpanzee blood and sera.

The information supplied by Courtois' assistant is highly significant, in that it is the first unqualified confirmation from someone working in the Stanleyville medical laboratory that (a) tissue cultures were being made locally in that lab, and (b) that they were being prepared from chimpanzee organs.

The fact that he did not know the precise details of tissue culture manufacture is frustrating, but not entirely surprising, because (as is discussed elsewhere), the Belgians did not routinely hand on knowledge, or train their African workers in different lab techniques. Indeed, some of the Africans I spoke with felt that, at least in some instances, they were deliberately left in the dark about what was going on.

However, we do have one brief, but extremely telling sentence from his old boss, Dr Courtois, which goes a long way towards confirming his account. At a conference about the use of primates in the laboratory which was held in Lyon, France, in December 1967, two French doctors delivered a speech entitled “Monkey cell cultures in virology”, in which they stated that some twenty primate species had been studied for the purpose of making tissue culture, adding that, in addition: “some laboratories may have used or regularly use species without disclosing the fact in publications”.

In the discussion that followed, someone commented: “Some laboratories, instead of killing monkeys themselves, obtain refrigerated kidneys from another laboratory. Is this satisfactory or wasteful?” Ghislain Courtois responded: “More than 10 years ago we sent kidneys from the Congo to Europe and they were quite satisfactory.”⁴⁹

More than ten years ago (note the careful phrasing) means before December 1957, and the response by Courtois’ assistant, that the tissue culture in Courtois’ lab was made mainly from chimps, strongly suggests that Courtois was dispatching *chimpanzee* kidneys for tissue culture work. So by 1957 (at the latest) Ghislain Courtois was apparently sending chimpanzee kidneys to Europe (which most likely means to Belgium). It seems likely that the making of chimp cultures in the microbiology lab which he headed would have been going on at the same time.

The new, concrete, custom-built Stanleyville medical labs opened officially in September 1957, but Lindi camp opened more than a year before that, in June 1956. So a further possibility that needs to be considered is that the making of chimp cultures may have started even earlier, back in the old redbrick medical laboratory in 1956. (Indeed, it may be that Courtois’ “more than ten years ago” comment should be read less conservatively – as “*at least eleven years ago*”, which would indeed put the date for his handling chimp kidneys back to at least 1956.)

At this point, we have to confront the problem of which versions of events are right: those of the two Belgian doctors, Ninane and Osterrieth, or those of five different African witnesses.⁵⁰

The testimonies of the African witnesses sometimes conflicted on minor details, but they corresponded convincingly on the major points. By contrast, Osterrieth and Ninane contradicted each other on what was, in effect, the key point. Each of them, at different times, claimed that he was the only one in the Stanleyville medical laboratory who had ever tried to make tissue culture.⁵¹

What is even more telling, however, is the fact that Dr Osterrieth’s own accounts given at different occasions over a period of seven years have featured gaps, anomalies and internal contradictions. Indeed, on the question of whether chimp kidneys were ever sent abroad (and if so, to where), Dr Osterrieth has now given three fundamentally different versions of events.⁵²

Taking all this into account (and having listened again to the original tape recordings of my interviews with him in 1993 and 1994), I now believe that Dr Osterrieth's memories cannot be relied upon, at least with regard to the key issues such as making tissue culture and vaccine, and the Lindi research. Neither, I believe, can the memories of Dr Ninane be relied upon on these key issues.

c) Making tissue culture in Stanleyville.

So, let us assume for a moment that Dr Osterrieth's first assistant is correct in his confident account, and that oral polio vaccine *was* being prepared in Stanleyville. Do we have any indications at all about how it might have been made? Paul Osterrieth believes that it was only several months after February 1958 that he "could attempt tissue cultures using trypsinisation". Fair enough. But what about cell cultures that did not require trypsin?

By good fortune, we happen to have a precise description of how a non-trypsinised tissue culture was prepared in Stanleyville. This features in an internal report published by the U.S. Armed Forces Epidemiology Board (AFEB) in 1959.⁵³ The AFEB report concerns the research into hepatitis in chimpanzees carried out by a Philadelphia virologist, Fritz Deinhardt, who arrived in Stanleyville on February 1st,⁵⁴ 1958, and stayed until the end of April.⁵⁵ He apparently collaborated with both Ghislain Courtois and Paul Osterrieth (in the microbiology and virology departments of the new laboratories), and he spent a lot of time at Lindi camp.

In order to continue his hepatitis research *in vitro* after he had returned to the US, Dr Deinhardt decided to transport chimpanzee cells back to Philadelphia. This was facilitated by the fact that, as explained in the AFEB report, there was already an existing sacrifice programme which he was able to plug into.

The passage in question reads: "*Several chimpanzees used for poliomyelitis studies at the Lindi camp had to be sacrificed at intervals. The doomed animals were bled one week prior to sacrifice, and the serum separated. The kidneys were removed under aseptic precautions, and transported to the laboratory in containers filled with Hanks' solution. They were then minced and to the washed pieces was added 5% isologous serum in Hanks' solution. These preparations were shipped in an insulated box without refrigeration to Philadelphia, where they arrived within 3 to 5 days. In spite of the long sojourn the tissue was viable and 3 of 4 specimens yielded, after trypsinisation, excellent cultures.... None of the cultures revealed evidence of foamy agents or other ECCO viruses.*"

This account has been confirmed and broadened by other testimonies (including those of Paul Osterrieth, during our first interview), and by a paper which appeared in the early sixties, which enlarged the number of chimp kidney shipments related to the hepatitis research from four to six.⁵⁶

In his article published in the Royal Society proceedings, Dr Osterrieth refers to this episode as follows: "It is true that six minced chimpanzee kidneys were sent to the Wistar Institute at the request of Fritz Deinhardt, who came to Stanleyville to

experiment with hepatitis infection of chimpanzees. Although I do not remember exactly when that was done, no cultures were retained in Stanleyville.”⁵⁷

This statement is highly revealing in two respects. Firstly, it contradicts Dr Osterrieth’s several earlier statements that chimp kidneys had been sent only to the Children’s Hospital Of Philadelphia (CHOP), and not to the nearby Wistar Institute.⁵⁸ The last of these statements by Osterrieth (“I also want to state very clearly that I never sent chimp kidneys to the Wistar Institute”) was apparently made in February 2000, and is quoted by Stanley Plotkin in his first Royal Society paper. However, by the time of his own speech to the conference, made seven months later, Dr Osterrieth is admitting that someone had indeed been sending chimp kidneys to the Wistar. The clarification is welcome. The key point is that although the six kidney shipments were intended for Deinhardt at CHOP, *they were sent via the Wistar Institute*. This does not, of course, reveal whether *other* chimp kidney shipments were also sent to the Wistar.

However, Dr Plotkin, in his various publications, reports that none of six persons (including himself) who used to work at the Wistar as students or technicians during this period ever saw or heard of chimpanzee cells at the Institute, and adds: “there is absolutely no evidence that chimpanzee kidneys...found their way to the Wistar”.⁵⁹ Though forthright, this stops short of an absolute denial. And now Paul Osterrieth has provided the evidence that Plotkin claimed to be lacking: that minced chimp kidneys *were* sent to the Wistar Institute. In fact, a similar account had already been given by the widow of Hilary Koprowski’s chief lab assistant, Tom Norton, who recalled her husband returning from Stanleyville to the USA in March 1957 with various chimpanzee materials (including several kidneys), and handing them to the Wistar Institute driver, who delivered them to the Wistar.⁶⁰ This was several weeks *before* Koprowski and Norton officially moved to the Wistar from Lederle in May 1957 (an apparent discrepancy that will be explored later in this manuscript). Mrs Norton’s account had already been published in *The River*, so I am surprised that Dr Plotkin stated that there was “absolutely no evidence that chimpanzee kidneys...found their way to the Wistar”.

All this begs the question of what chimp kidneys might have been used for at the Wistar Institute in 1957-1958? Does this not suggest that they might, after all, have been under investigation as an experimental vaccine substrate? This seems not unreasonable, in that it is hard to imagine Dr Osterrieth taking the decision to try out chimp kidneys as a substrate for CHAT in Stanleyville, unless he had already got the explicit approval of the scientist who developed CHAT – Hilary Koprowski.

The second revealing aspect of Dr Osterrieth’s Royal Society statement is the key detail that “cultures” were prepared from chimpanzee kidneys in Stanleyville (even if he goes on to say that no cultures were retained there).

Osterrieth’s description of the minced chimp kidney preparations as “cultures” is highly significant, for it reveals that he is referring to Maitland-type cell cultures, rather than trypsinised monolayer cultures. Maitland-type cultures were simple to make, even in the fifties: the process required mixing together some minced-up kidneys, some serum and some growth medium, and then adding a few drops of antibiotics. With Osterrieth’s new testimony, it becomes clear that Maitland-type

cultures were initially made from the chimp kidney cells, chimp sera and Hanks' solution in Stanleyville, and that later at least six shipments of these cultures were forwarded to Philadelphia, and were treated on arrival with trypsin, to produce "excellent" trypsinised monolayer cultures for Deinhardt's hepatitis work.⁶¹

It should be noted that both types of culture – trypsinised and Maitland-type – were approved substrates for making oral polio vaccines throughout the 1950s.⁶²

However, Maitland-type cultures were already considered a bit primitive, the "poor country cousins". In the abstract to his paper, Osterrieth apparently confirms this by stating: "In Stanleyville, at the time of vaccination campaigns, tissue cultures were primitive, experimental and used solely for diagnostic purposes."

With that statement, Osterrieth confirms that "primitive", or Maitland-type, cultures *were* being prepared in his lab, at the same time as the polio vaccinations. On this issue at least, we appear to have consensus.

Where we do *not* have consensus is where Dr Osterrieth states that: (a) the primitive tissue cultures (from whichever species they were made) were used only for diagnostic purposes, (b) that he neither handled nor manipulated the polio vaccines in his laboratory, and (c) that he himself never tried to make cell culture from chimp tissues.

d) The who and the how....

To try to unravel these issues, one needs to reexamine the limited archival material that does exist about the Stanleyville medical laboratory, and about Lindi – and then to ask some further questions.

In February 2000, Dr Osterrieth apparently gave a categorical assurance to Dr Plotkin that "at no time did I ever attempt to make cell cultures from chimpanzee tissues".⁶³ Yet by the time he delivered his own speech in September 2000, Osterrieth was acknowledging that chimp cultures *were* made in Stanleyville. He does not mention who might have done this.

Instead of diving in to that question straight away, let us take Osterrieth's statements at face value, and move to another question. If Osterrieth did not make *chimp* cultures, then which cultures did he make?

When I first asked Osterrieth about making tissue culture in 1993, he was initially unable to remember which species he might have used, but he did emphasise that it had not been the chimpanzee. Later in that interview, after I told him that Lépine had used baboons, he said that he too was likely to have done so. (This is interesting, because there were certainly no baboons at Lindi, and there were none to be found in the nearby rain forest, except for a few which might have been scavenging along the main roads through the forest. The nearest baboon habitat was the savanna grassland, 400 kilometres to the north. Baboons, in short, were not a very practical choice for a locally-available primate.)

However, there is a single paragraph in the 1958 medical lab annual report which, at first glance, provides a substantial level of support for Dr Osterrieth's statement. This states that, in 1958, tissue culture had been "exclusively" made from baboon kidneys, and that 200 tubes and 10 bottles had been prepared. It goes on to state that 36 of the tubes were used for viral analyses, while the remainder of the tubes and bottles were used to prepare adenovirus antigen for complement fixation. However, the adenovirus did not survive being transported without ice, and so the latter tissue culture was apparently wasted.

But this raises more issues than it settles. For one thing, this quantity of tubes and bottles represents the product of a small number of baboon kidneys – probably from two to four, unless the receptacles were unusually large.⁶⁴ For another, why emphasise that cell culture had been made "exclusively" from baboons? For a third, why go to all that trouble to train Osterrieth in making tissue culture, and then report that just a few tubes and bottles had been produced, carefully adding what every tube and every bottle had been used for – including the fact that most of them had gone to waste?

This odd emphasis on the baboon tissue culture seems all the more remarkable when one considers that the annual reports of the Stanleyville medical lab for 1956, 1957 and 1958 contain a total of eight brief sentences about the polio research then being conducted on 400 Lindi chimpanzees, which fail to provide any substantive information about the work taking place, which, as we now know, included the removal of kidneys and other organs.⁶⁵

The 1956 entry explains that experiments have started, and that 60 chimps have already been "used". Those for 1957 and 1958 each find reasons for not reporting anything about the research. The 1958 reference states that the OPV work on the chimps is almost complete, and is the subject of a report that is in the process of being edited. This promised paper is referenced in two other published works also. But it was never publicly released, even though one suspects that copies were produced and circulated, and almost certainly still exist somewhere in the USA and/or Belgium. So this is all that is revealed about a programme that involved a historically unprecedented mass-sacrifice of apes.

To me, this only underlines the fact that the work conducted on the chimps at Lindi, at least for the first two years, was highly secret (something which contemporary visitors to Stanleyville and Lindi camp, both African and non-African, have almost universally confirmed). Furthermore, the careful reporting of the *failure* to make any significant quantity of tissue culture, and the claim that *only baboons* had been used for this purpose, suggests to me that even back in 1958 a need may have been felt to provide a cover story.

Dr Osterrieth was a virologist, and had expressly been trained in how to make tissue culture. He admits making tissue cultures from baboons (which are not very common near Stanleyville),⁶⁶ yet he had a camp full of chimpanzees (which apparently "had to be sacrificed at intervals") just fifteen kilometres away. So when Dr Osterrieth denies making cultures from chimps, this begs a question. Why on earth not?

Was it not his job, his duty even, to at least *attempt* to do this?

According to that strangely precise entry in the 1958 lab report, no chimp tissue culture was made. In addition, the report inexplicably fails to mention that polio vaccine was being prepared – even though (under normal circumstances) this would have been a significant feather in the cap of the new lab, and one which would surely have been proudly announced. The deliberate misreporting of one detail, and the non-reporting of the other, are the first strong indications that something strange was going on here. Something so strange that it was felt necessary for the lab scientists to get their plausible denial enshrined in the public record, even if (at that stage) nobody was asking awkward questions.

Let us leave that for a moment, and return to the original question. If not Osterreith, then was there anyone else at Stanleyville who might have made Maitland-type cultures from chimpanzees? There are in fact quite a few potential candidates.

Between February and April 1955 the director of the Stanleyville lab, Dr Ghislain Courtois, went on a study tour to the Rockefeller Foundation labs in New York and Trinidad, and then on to the Osvaldo Cruz Institute, a Rockefeller-funded lab in Rio de Janeiro. The notebooks from that tour make it clear that he received a training course in virology, focussing on tissue culture, virus isolation and cultivation, and vaccine-making. There are notes on four different approaches to making Maitland-type cultures from monkey kidney, and on the growing of poliovirus in those cultures. There is also a page on trypsinisation.⁶⁷ Courtois and Koprowski apparently first met later in 1955, when plans were made to set up the chimpanzee camp, which opened in June 1956. So Courtois certainly had the knowledge and wherewithal to have produced tissue cultures in Stanleyville from an early stage. This, of course, is further confirmed by his assistant's recollection that tissue culture *was* being made in the lab, and that it was produced “mainly from chimpanzees”.

Then there is the histopathologist, Dr Gaston Ninane. During my first interview with him in 1992, Dr Ninane told me three times in the space of a few minutes that CHAT vaccine had been made in chimpanzee kidneys. When I pointed out that the literature of the day had mentioned only *monkey* kidneys, he suddenly retracted, saying this had been a slip of the tongue, and that this is what he had meant to say – that some sort of “monkey” (such as a chimp or a baboon, he added) had been used, but that he was not sure which.⁶⁸ It seemed to me that there had been genuine confusion (and he *was* speaking English for my benefit), and I accepted that he had made a mistake. Nowadays, I am much less sure that this was the real reason for his “slip”.

When I asked Ninane later in that interview whether polio vaccine could have been manufactured in Stanleyville itself, he denied it, saying that vaccine-making would have been far too technical an operation for a lab like theirs, and that anyway, no freezers had been present in the lab until 1960.⁶⁹

This is certainly incorrect, in that there is documentary evidence that chimp sera were being stored in a freezer at the Stanleyville lab by June 1959,⁷⁰ and one certainly would have expected the new medical laboratory that opened in September 1957 to have boasted at least one freezer. (Elsewhere in the Belgian colonies, walk-in refrigerators with a freezing capacity were installed in smaller labs such as Butare and Usumbura during the course of 1957.) Besides, freezers are apparently not essential to

the process of preparing, or amplifying, polio vaccine. Apparently a refrigerator would suffice for keeping the various materials such as organs, sera and growth medium below 4 degrees centigrade.

However, might there be a kernel of truth in Ninane's statement? If there was no freezer in the Stanleyville virology lab until, say, the end of 1958 or early 1959, might this help to explain why Dr Osterrieth had to make new batches of vaccine at regular intervals? (Polio vaccine that was merely refrigerated would have progressively lost titre over time, so that further passage would have been necessary not just to amplify the quantity, but also to boost the concentration of the attenuated poliovirus.)

However, if a virologist like Osterrieth had to keep making fresh batches because he lacked a freezer, is it not likely that for a number of reasons (such as minimising contamination risks, and maintaining titre) he would have made each vaccine batch from the last batch, in series? From the perspective of the polio vaccine, this would be a lot safer than continually opening, closing and pipetting from the original vaccine bottle, but equally it would mean that the twentieth batch of vaccine would potentially contain primate cells from all previous batches (1 to 19), rather than just from two sources: batch 1 and the tissue culture used to make batch 20. Since it is likely that different chimps provided cells for different batches, this would have substantially increased the risk of multiple (yet unrecognised) SIV contamination, and thus of recombination between different SIV strains.

Later, during my third interview with him in 1994, Dr Ninane talked for several minutes about the fact that he had "tried to make tissue culture in Stanleyville" in 1957, using the methods he had learnt at the laboratory of Professor Chevremont in the early fifties. At one point he mentioned that Chevremont had "described the possibility of [using] macrophages" for making cultures, but he did not elaborate. (This is intriguing, because macrophages, which were apparently rather "unfashionable" cells in the fifties, are now known as the natural target cells for SIVs and HIVs. I am informed that a few people were indeed growing viruses in macrophage cultures back in the fifties, but it is hard to know whether or not this has relevance for what was happening in Stanleyville.)

Ninane went on to repeat that in Stanleyville in 1957 he had tried to make tissue culture on his own, over a period of four or five months. Apparently they did not then have trypsin available in the lab, "whereas in the United States at that time, already they used trypsinised extract of organs". When I asked if it was chimpanzee tissue culture he tried to make, he answered: "not only chimpanzee but human". He reemphasised that this had involved using "the old system", and when I proposed that this would have involved making suspended cell cultures (in other words, Maitland-type cultures), he did not object.⁷¹

Another person who could undoubtedly have made Maitland-type cultures in Stanleyville was Dr Fritz Deinhardt. He was, in fact, a tissue culture expert, with eleven of his first thirteen published articles, from 1954 to 1958, featuring "cell culture" or "tissue culture" in the title, even if this previous work had concerned other types of culture (mainly human cells, including HeLa), rather than culture from primate kidneys.⁷² However, that was hardly an obstacle, and Deinhardt clearly had

the expertise to prepare Maitland-type cultures from chimp kidneys (or to help Osterrieth do this).⁷³

Another person who undoubtedly had sufficient experience was Dr Jean Vandepitte, who took over from Courtois as head of the Stanleyville lab during the latter's leave, from March to September 1958. However, according to Dr Osterrieth, he and Vandepitte did not get along, partly because Vandepitte wanted to do everything in a different way from Courtois. Osterrieth told me that he regularly used to disobey Vandepitte's instructions to, for instance, go and take blood from the local population, preferring to stay behind and do "the interesting work" in the lab.⁷⁴ On the one hand, this perhaps tells us something about how devoted the other doctors were to Courtois. However, it also illustrates how easy it might have been for someone to have followed a line of unofficial research on their own initiative, feeling that they were justified because "this is what the boss would have wanted". It is worth noting that it was Dr Vandepitte, together with the *agent sanitaire* Pierre Doupagne, who organised the reunion of the Stanleyville doctors in 1994 – a reunion also attended by Dr Osterrieth, Ghislain Courtois' son André, and one or two other Belgian doctors (though not, apparently, by Gaston Ninane). It is believed that my own investigations into Lindi and Stanleyville were probably the catalyst that prompted this reunion, and certainly information-gathering thereafter became more difficult.

However, what really matters here is not the identities of the doctors who made the kidney cultures in Stanleyville, but *the fact that they were made*. And since this is not a question of apportioning blame, but rather one of assessing opportunity, it is enough to note that there was no shortage of candidates.

Now let us turn to the more important question of timing. Dr Osterrieth says he is unsure *when* those chimp kidney cultures were prepared and sent to Philadelphia. But here I can provide some help. In 1993 Dr Deinhardt's former boss, Professor Gertrude Henle, told me unequivocally that Fritz Deinhardt had himself sent "chimpanzee tissue cultures" to Philadelphia during his time in Africa. In fact, it seems that four shipments of this material were sent by Deinhardt during his three-month stay, and a further two shipments were sent later, presumably by Osterrieth.⁷⁵

Immediately after this, I asked Dr Henle: "Had chimp tissue cultures been sent from there [Stanleyville] before, do you think?". There was a pause, and then she said: "Well of course, they had tissue cultures from the monkey kidney for making polio vaccine". At the time her comment seemed something of a *non sequitur*. When I asked her which monkeys, there was another pause, and then she said she had no idea. However, from the ensuing conversation it became apparent that she had been referring to primate kidneys which had been available in Stanleyville before Deinhardt's arrival in February 1958, and which had been used (somewhere) to make polio vaccine. At the time, I thought she meant that these kidneys were being sent overseas. Now it seems that she was referring to local production of vaccine in Stanleyville. As for the species, there is no evidence of any primate kidneys other than chimp kidneys being available in Stanleyville before 1958.

Dr Henle then asked me why I was so interested, and I realised that she had not been aware that chimpanzee SIV was the ancestor of HIV-1. I explained about the relationship between the two, and about the OPV/AIDS theory. Then, perhaps twenty

minutes or so later, I asked her again whether chimp kidneys had been used to make the polio vaccine. She declined to answer, but it was noteworthy that she did not withdraw what she had said earlier. Finally she said: “I don’t say you are wrong, but it might be futile [to try to follow this up]...Something has happened, yes, but what can you do about it?”

It is only now, in the light of the testimony of the various African witnesses, that the sequence of question and answer makes sense. It seems that Dr Henle had presumed that I already knew that they were using chimps to make polio vaccine *in Stanleyville*.⁷⁶ It was only when I pressed her on the question of the species that she declined to be more specific. However, she neither backtracked in the way that Gaston Ninane had done a year earlier, nor did she withdraw her statement. She merely pointed out that it might be difficult to prove.

Around this point, I realised that a pattern was beginning to establish itself, whereby the first responses of some of those scientists who were directly or indirectly involved with the Lindi research were rather telling, but were often followed by something more non-committal, or by retraction. By contrast, those without any agenda, like the African assistants, tended to be much more forthright.

This still leaves us with an important question – that of which tissues were used to make CHAT vaccine in Stanleyville. Given the evidence about chimp sacrifice and organ extraction, chimp tissues would seem to be a plausible substrate, but can anything be proved?

I believe it can. At this point, we need to look at all those statements which have been made by persons who were directly involved with the work at Lindi and with the Congo vaccine trials, and which confirm different details about tissue cultures, and whether or not polio vaccine was locally prepared.

First of all there is Gaston Ninane. When he was visited in hospital in February 2000 by doctors Koprowski and Prinzie, at a time when (according to his sister) he was recovering from a fall caused by Parkinson’s (and possibly Alzheimer’s) disease,⁷⁷ he apparently specifically denied just one aspect of all the many pages which are devoted to his testimony in *The River*. This related to whether he had ever tried to make cell cultures, and, more specifically, chimpanzee tissue cultures, in Stanleyville. Dr Ninane apparently signed a document which stated: “The statements which are attributed to me on this subject are false and are lies”.⁷⁸

However, I have the cassette tapes to prove that Dr Ninane said both these things. The signed statement he apparently gave to Dr Koprowski, and which is quoted by Plotkin, is incorrect, and I am hereby offering to play the relevant passages of these tapes to Dr Koprowski, Dr Plotkin, or to both of those gentlemen, to prove that fact. In return, I would like to see the original of Dr Ninane’s signed statement.

In any case, this point has since been confirmed by an impeccable source. In January 2001 (before my second trip to Kisangani), I spoke with Dr Maurice Kivits, the former assistant Inspector-General of Hygiene for the Belgian Congo. Between 1956 and 1960, it was Dr Kivits (rather than his elderly boss, Paul de Brauwere) who was effectively responsible for public health, including vaccinations, in Belgium’s African

colonies, and in 1959 he had visited Stanleyville and Lindi camp as part of a three month African tour of duty in which he travelled to all the medical laboratories, and assessed the impact of the various vaccine field-trials.

Even in his late eighties, Dr Kivits is still a very precise man. When asked what the Lindi chimps had been used for, he replied that it had been for the “preparation of the vaccine.” Shortly afterwards, Dr Kivits added: “the vaccines were tried in the chimps” – an apparent reference to testing the immunogenicity and/or safety of the vaccines. But his initial answer involved polio vaccine *preparation*, not testing.

When one doctor (Ninane) says that chimp kidneys were used to make the vaccine, but then insists that this was a slip of the tongue, then it is not unreasonable to accept his word. (I was not looking for a conspiracy theory, merely for the truth.) When a second doctor, the man with overall responsibility for the vaccinations, says the same thing, but seems disinclined to elaborate, then one ought to sit up and take notice. But even at this late stage, I still believed that the Koprowski vaccines had only been made in the United States and Europe. It was only with the further confirmation by the African assistants in Kisangani, three months later, that I finally realised the full significance of Dr Kivits’ remark.

To sum up: Osterrieth’s first lab assistant has reported that polio vaccines were being prepared in Stanleyville in early 1958, at the latest – and this appears to have been confirmed by Fritz Deinhardt’s boss (Gertrude Henle).

Dr Kivits and Dr Ninane, who were both direct witnesses to events, have both stated that chimpanzees (or chimp cultures) were used to make the polio vaccine – even if the latter subsequently modified that statement, and the former did not supply further details.⁷⁹

However, the crucial detail that chimpanzee tissue cultures were being prepared in at least two departments at the LMS is confirmed by Courtois’ assistant.

It is a shame that nobody has yet felt able to make one clear, unequivocal statement which sums up the entire chimpanzee/polio vaccine programme, but this is perhaps not surprising, given that these activities were shrouded in secrecy in the fifties, and continue to be so to this day. Indeed, it appears to have been policy among the Belgian doctors to keep the Africans in the dark about many of their activities – including, it now seems, the vaccine-making process. (Osterrieth’s first assistant explained: “The whites didn’t show us blacks what they were doing. That’s why, when they went back to Europe, nobody else could follow on that work.”)⁸⁰

None the less, if we put all this evidence together, then the only reasonable conclusion is that none of the European and American doctors involved with the Stanleyville medical lab, Lindi camp and the CHAT trials, has yet told the whole truth about what was happening.

I believe it is now clear beyond any reasonable doubt that tissue culture was being prepared in at least two different departments at the Stanleyville medical lab (virology and microbiology), starting in 1957 or before, and that most, if not all of the tissues were derived from chimpanzees. I also believe it is now clear beyond reasonable

doubt that Koprowski’s polio vaccines were being locally amplified, and that one of the persons doing this was the man who was gathering most of the organs and bloods from the Lindi chimpanzees – Dr Paul Osterrieth.

Scientists apparently like to talk in terms of parsimony – and the most parsimonious explanation, notwithstanding the protestations of those involved, is that CHAT vaccine was being amplified in the Stanleyville medical laboratory in the second half of the fifties, in the cells of chimpanzees. [See **Figure 4**, and the section on “Local amplification”, below.]

Figure 4: Was CHAT made in chimpanzee cells in the *Laboratoire Medical de Stanleyville* (LMS) in the 1950s? Testimonial evidence for (top section) and against (bottom section).

NAME	TITLE	TESTIMONY
		EVIDENCE FOR...
Paul Osterrieth	Microbiology lab, LMS, 1956-1957; head of virology lab, LMS, 1958-1960	Concedes that a few chimp kidney cultures were prepared in Stanleyville, and sent to the Wistar Institute. [However, also see other testimony, below]
Gaston Ninane	Head of histopathology lab, LMS, 1955-1960.	Originally says that chimp kidneys were used to make CHAT; later modifies this. Originally says that he himself tried unsuccessfully to make tissue culture (including human and chimpanzee tissue culture) in his lab; later retracts.
Joseph	“Nurse” and head of the African team at Lindi camp, 1956-1959.	Confirms that the work at Lindi was secret. Says that the sacrificing of chimpanzees was routine, and that most of the autopsies were done by Osterrieth (though others were done by Courtois and Ninane). Says that kidneys were always taken, and placed in liquid in metallic canisters. Thinks that most of them were sent overseas, including to the USA.
Louis Bugyaki	Head of Stanleyville veterinary lab, 1956-1959.	During first three interviews, explains that both Osterrieth and Ninane had told him that they had been taking kidneys from the Lindi chimps and sending many of them to America; begins vacillating after recent approaches by Belgian doctors.
First assistant to Osterrieth	Helped Osterrieth in LMS virology lab from February 1958 on.	Recalls Osterrieth frequently making polio vaccines, in response to requests from the provincial government, and says this began before his own arrival in February 1958. Associates the vaccine-making with the preparation of chimpanzee serum. Fed this same polio vaccine orally in several places including Stanleyville military camp, a known CHAT vaccination site.
Second assistant to Osterrieth	Helped Osterrieth in LMS virology lab from December 1958 on.	Recalls that Osterrieth prepared a vaccine; cannot recall which. Has recently received a letter from Osterrieth, asking him which of the African workers from the LMS and Lindi camp are still alive and which are dead.
Courtois’ assistant.	Helped Courtois in the microbiology lab of LMS from April 1956 on.	States that tissue culture was made in Courtois’ microbiology lab, and that this was “mainly from chimpanzees”. Says the process was linked to the autopsies conducted at Lindi camp. Says tissue culture was also made in Ninane’s histopathology lab, but he doesn’t know about the virology lab.
Hilary Koprowski	Director, Wistar Institute, Philadelphia from May 1957 on.	States that his polio vaccines were routinely amplified in labs all over the world, and that this may have included the Congo [but also see other testimony, below]
Priscilla	Wife of Tom	Says that her late husband brought kidneys and other materials

Norton	Norton, Koprowski's chief lab assistant from 1946-1957.	from the Lindi chimps back to the US in March 1957, and that these were delivered to the Wistar Institute
Gertrude Henle	Joint head of virology lab, CHOP, 1950s.	Says that the "monkey kidney culture" in Stanleyville was being used to make polio vaccine. Later makes it clear that the "monkeys" in question were in fact chimpanzees.
		EVIDENCE AGAINST...
Hilary Koprowski	Director, Wistar Institute, Philadelphia from May 1957 on.	Denies everything
Gaston Ninane	Head of histopathology lab, LMS, 1955-1960.	Signs a statement for Koprowski and Plotkin shortly before his death denying that he ever said that he tried to make tissue culture, or chimpanzee tissue culture, in his lab.
Stanley Plotkin	CDC Epidemiology Intelligence Service based at Wistar, August 1957 on; later associate director at Wistar.	Indignantly denies all charges, calling <i>The River</i> "a house of cards built on a swamp of conspiracy theory, unsubstantiated allegations, and character assassination". He and his team have persuaded some witnesses to modify their stories; however, at least one such witness was sent a prepared letter to sign which did not reflect his own views, but rather those favoured by Dr Plotkin. Much of the information that he has presented in response to <i>The River</i> is inaccurate, or misleading.
Paulette Dherte	Nurse and pharmacist, LMS, 1955-1960.	"Laughed uproariously and said it was completely impossible", according to Stanley Plotkin.
Paul Osterrieth	Microbiology lab, LMS, 1956-1957; head of virology lab, LMS, 1958-1960.	Denies that he had significant dealings with the chimps at Lindi, or that he conducted autopsies on chimps. Denies that he was able to make tissue culture in the lab before mid-1958, and insists that even then it was only prepared from baboon kidneys. Denies ever making polio vaccine in his lab. However, has changed his story on several occasions, and refuses to answer certain of the key questions.

e) The when.

The major question that remains to be answered about the African CHAT trials is: when did the making of vaccine in Stanleyville begin?

Osterrieth's first assistant began work on February 12th 1958,⁸¹ and says that his boss had been making polio vaccine before his arrival. According to Dr Osterrieth, he and his wife returned to Stanleyville "a very short time" (his wife says "just a few days") after Deinhardt's arrival in that city.⁸² Since the latter is documented as having occurred on February 1st; it seems that Paul Osterrieth must have returned from leave on or around February 4th. So had he been making polio vaccine for just a week prior

to the hiring of the first assistant? Or was he – or someone else – making polio vaccine even before that?

Here, for perspective, it may be helpful to return to Dr Ninane's statements. As I wrote earlier, Dr Ninane told me that he had tried, on his own, to make tissue cultures from both chimpanzee and human cells for a period of four or five months during 1957, using the old-fashioned methods of Professor Chevremont. Later, he told me that he eventually stopped these attempts, because "it was impossible to make tissue culture with the material we had." He added that Courtois had laughed at him, saying he was an old man who could not do anything right.

Ninane also told me, on several occasions, that he had been trained in tissue culture techniques in Dr Lise Thiry's lab in Brussels for ten to twelve weeks in the summer of 1957, and fortunately we have a paper written by Lise Thiry in 1958, which gives a sense of what these techniques might have involved. The paper reports that she had the Koprowski vaccines CHAT and Fox in her lab by July 1957 (when Ninane is likely to have been present), and that they grew CHAT and Fox (and several other viruses) on "several [trypsinised] batches of monkey kidney cultures" from (it is hinted) more than one species.⁸³ Experiments were also conducted with many other cell cultures, and we shall return to these later.

It seems reasonable to propose that the four or five months when Gaston Ninane tried to make tissue culture alone were from the time of his return from leave in September 1957, up to January 1958. (However, if he was really using techniques taught him in the early fifties, then it may have been that he was referring to the period before he went on leave – for instance from October 1956 to February 1957).

But in any case, there is no doubt that *somebody* at the Stanleyville medical lab was successfully making tissue culture during the late 1957/early 1958 period, because between January 8th and February 1st, 1958, at least 22,000 people were fed CHAT vaccine in response to epidemic outbreaks of polio in different towns in the surrounding province, *Province Oriental*. Dr Plotkin tells us that the vaccine used was pool 8 or 9 of CHAT (rather than pool 10A-11, which arrived in February 1958) – and it is clear that, like the other polio vaccine used in the Congo, this also must have been amplified locally. Since neither Osterrieth nor Deinhardt was present in the Congo during January 1958, this means that the preparatory work, the making of the cell culture, had to have been done by Courtois, Doupagne or Ninane – and, given the various testimonies, I would favour one of the first two. It is recorded that Ninane was the one who fed the vaccine in the epidemic outbreaks.

A document in the Belgian government archives⁸⁴ reveals that the supply of CHAT ran out half-way through the final "anti-epidemic vaccination", in Bambesa, on February 1st – and that contrary to what is reported by Koprowski,⁸⁵ only half the village was vaccinated. [See later for further discussion.] In other words, the four outbreaks used up all the vaccine which had been prepared from pool 8 (and/or 9) just as the new pool, 10A-11, was arriving in Stanleyville – almost certainly with Fritz Deinhardt – on that same day, February 1st.

But it turns out that Koprowski's polio vaccines may have been amplified in Stanleyville even before January 1958. First there is the testimony from Courtois's

assistant that tissue culture made “mainly from chimpanzees” was being prepared in the microbiology lab, and his allusion to both Courtois and Osterrieth working there, which makes it clear that here he is referring to the microbiology lab in the old building. This suggests that the making of tissue culture may have been going on before Osterrieth moved labs – which would also mean before July 1957, when Osterrieth set off on leave. Then there is Courtois’s claim that he was sending kidneys for tissue culture work from Stanleyville to Europe before December 1957 – kidneys which, as outlined above, must surely have been from chimpanzees. And then there is the evidence that sixty of the Lindi chimps had already been used in the polio vaccine research by the end of 1956.

Finally, there is the statement by one of the former Stanleyville vets, Joseph Mortelmans, that Courtois had already been conducting medical research on a number of chimps in his laboratory even before he (Mortelmans) arrived in Stanleyville. Mortelmans worked there only between December 1955 and June 1956, so this suggests that Courtois may already have been using chimps for tissue culture by late 1955. During this same interview, Mortelmans told me he thought that the chimp kidneys could have been used for the final passage of the polio vaccine. He has since told Plotkin that he was only expressing “a hypothetical possibility”.⁸⁶ None the less, for someone who was a friend of Courtois, and who was familiar with the latter’s chimpanzee research at this early stage, it was a telling comment.⁸⁷

As it happens, Dr Courtois was not the first man in the Congo to produce oral polio vaccines locally. Starting in 1953, Mortelmans’ fellow-vet, Alexandre Jezierski, was making polio vaccines (both killed and live) in a wide range of local primate tissues at his small veterinary lab at Gabu, some 500 kilometres east of Stanleyville. By 1954, he had already tried out chimpanzee tissues, and found that they were “very good” for growing poliovirus.⁸⁸ This will be discussed in more detail below.

f) Vaccine shortfall during the Ruzizi campaign.

But let us now return to the crucial period of February to April, 1958, the period for which we have first-hand testimony that CHAT vaccine was being prepared locally. This is also the period when Fritz Deinhardt was visiting Stanleyville, and when the new CHAT vaccine pool, identified as 10A-11, came onto the scene. We have some quite precise details about 10A-11, because there is a single page of paper from the Wistar which refers to this pool, and which can only have been written between January 23rd and January 27th, 1958.⁸⁹ This means that a sample of the new vaccine pool was almost certainly carried out to Stanleyville by Fritz Deinhardt, who flew out from the USA on January 30th, arriving on February 1st, with an insulated box for his hepatitis-infected stool specimens.⁹⁰ It is not hard to imagine that Deinhardt’s trip, which was paid for by the US army,⁹¹ would have been seen as an ideal opportunity to deliver a 100 c.c. bottle, or half-litre flask, of the new CHAT pool to Stanleyville, and it seems that as soon as Osterrieth arrived a few days later, he began amplifying the vaccine, just as his assistant reports he was doing when he himself started work on February 12th. Since Deinhardt was a tissue culture expert, it would have been convenient to have had him available, to offer advice or help if needed.

The new vaccine, CHAT pool 10A-11, was then fed by mouth to over 3,000 soldiers and their families in Stanleyville military camp, on the south bank of the Congo river

on February 27th.⁹² I have been told by a former Belgian colonial resident that *the soldiers based at this camp would, in all likelihood, have come from all over the Belgian Congo, and would therefore have dispersed to their various home regions at the end of their periods of service.*

10A-11 was also fed in the huge Ruzizi Valley trial which was staged between Bugarama (now in Rwanda), Kamanyola, Kabunambo and Uvira (DRC)⁹³, Kihanga and what is now Bujumbura (Burundi). The dates given in Koprowski's brief article on the CHAT vaccinations are from February 24 and April 10, 1958.⁹⁴ However, Dr Ninane recalled returning to the Stanleyville lab after the main Ruzizi campaign and picking up more vaccine which he then fed along the eastern shore of Lake Tanganyika, from Bujumbura down to Nyanza Lac, almost on the frontier with what is now Tanzania. Altogether, in the two campaigns in the Ruzizi Valley and along Lake Tanganyika, some 215,500 persons were vaccinated.

It seems likely that the mass-vaccination of the town of Lisala (in Equatoria province) by Dr Ninane, an event to which he frequently referred (and which clearly took place during the early round of field-trials, when he himself was directly involved with the feedings), may have happened during the same time period.

It is worth noting that despite the quite specific details about the first African CHAT feedings which are provided in the brief, but key, article which appeared in the July 26th 1958 edition of the *British Medical Journal*, there is no mention at all of three of the above-mentioned vaccinations: those at Stanleyville military camp (February 27th), along the Lake Tanganyika shoreline (April 1958), and at Lisala (date unknown). According to Ninane, it was Koprowski (not the lead author, Courtois) who wrote this article, and who merely sent it to Stanleyville for checking. But the failure to mention these three field-trials (at least two of which Koprowski would surely have known about) is intriguing. Is it possible that they were somehow "more experimental" than the others?

Two American doctors were present for the Ruzizi Valley mass-trial of CHAT, one of whom was Koprowski's long-time collaborator George Jervis, who headed the laboratory at Letchworth Village (a huge facility for developmentally disabled children in upstate New York), and who had previously prepared experimental Koprowski vaccines there in monkey kidney tissue culture.⁹⁵ The other was Agnes Flack, the medical director of Clinton State Farms, the women's penitentiary in New Jersey where, since late 1955, Koprowski had been testing his vaccines on infants born to the prisoners. It is her diary which provides additional insights about how the Ruzizi campaign was organised.

According to Dr Plotkin, on March 4th 1958, eight days after the start of the mass-trial, Dr Ninane telegraphed Dr Koprowski, asking him urgently for more vaccine capsules, and more liquid vaccine.⁹⁶ Koprowski apparently refers to this telegram in a letter which he wrote to George Jervis in the Congo that same day (which is quoted by Plotkin), in which he tells Jervis that he has advised Ninane "to request from you more of liquid Type 1 which will be sent to Usumbura end of March".⁹⁷

The suggestion that Ninane should request more vaccine *from Jervis* is strange, not least because Jervis was due to leave the Congo a fortnight later, on March 17th.⁹⁸

Unless, that is, Koprowski knew that Jervis was preparing fresh batches of the vaccine at the medical laboratory in Bukavu, where he appears to have been based for at least part of his four week stay in Africa.⁹⁹ As the lab man on the trip (a last minute replacement for Tom Norton, who had had a heart attack over Christmas 1957), it would be natural for Dr Jervis to have taken responsibility for both control of the vaccine, and for local amplification. It appears, therefore, that Koprowski was expecting Jervis to fill the gap until more vaccine could be sent out at the end of the month.

The Bukavu laboratory had opened seven months before the lab in Stanleyville, at the end of February 1957.¹⁰⁰ The official opening, on February 25th, was followed by a week-long conference on the standardisation of lab techniques, attended by the directors of all the medical labs in the Congo.¹⁰¹ This included discussion about the essential tasks of the labs, which (it was stated) included the production of vaccines. There was a special showing of a film from the Eli Lilly corporation about making polio vaccines. (Although Eli Lilly made IPV, the film would almost certainly have included details about how to make “monkey kidney tissue culture”.) The same laboratory directors met again at the virus symposium that coincided with the opening of the Stanleyville lab in September 1957.

The Bukavu lab was an impressive building of three storeys, and according to Agnes Flack it was “beautifully equipped”, and had “modern instruments”. Recent research has revealed that attached to these labs was a large animal house where several primates, including chimpanzees, were caged.¹⁰²

We do not know where the vaccine for the Ruzizi trial came from. George Jervis’s widow, Ruth, believes he may have carried the vaccine with him, but this has never been confirmed, and Koprowski refused to answer questions on the subject. But even if Jervis did carry the vaccine, there may still have been a problem. This is because Jervis and Flack’s plane was delayed by bad weather in New York for 24 hours, so that the journey to Africa took three days instead of two. If they *were* carrying vaccine with them in an insulated box, the 24-hour delay may well have been crucial, meaning that the vaccine was no longer viable on arrival.¹⁰³

However, this should not have been so serious, because Paul Osterrieth was at that time amplifying the new vaccine pool in Stanleyville. Agnes Flack’s diary reveals that she and Jervis stopped off in that city for three hours on February 20th, en route to the Ruzizi Valley, and that during this time they visited the medical lab. In other words, Osterrieth may have been able to give them enough vaccine to tide them over for the start of the Ruzizi vaccination. We do know from the diary that on the first eight days of the mass trial, from February 24th up to and including March 3rd (the day before Ninane sent his telegram), only 15,000 people were fed. By the time Jervis left, on March 17th, the rate of vaccination had more than doubled, and the total had risen to 72,500, so it seems likely that some new vaccine had kicked in from somewhere, whether from Stanleyville, Bukavu or elsewhere. After March 17th, the supply problem seems to have been solved, because they began vaccinating upwards of 10,000 persons a day, and Flack wrote about “production lines...competing with Ford and General Motors”.

And which cells might have been used to amplify the vaccine locally? Each batch would have needed fresh cells from a primate or primates: the so-called “primary monkey kidney tissue culture”. So Jervis might have sacrificed one of the primates in the Bukavu animal house – or he might have used some of the chimp cells that we know were then available in Stanleyville. If the latter, then he could have carried the fresh chimp tissue culture with him to Bukavu, and then inoculated pool 10A-11 into different bottles of that culture, to produce further batches of vaccine.

The secrecy that surrounds this episode continues to this day, for Dr Koprowski has not responded to requests to release copies of Dr Ninane’s telegram, or his own response to Dr Jervis. All that we know, therefore, is that the Ruzizi Valley vaccine may have been amplified in Stanleyville, in Bukavu, or both. My own hunch is that Osterrieth supplied vaccine for the first few days, and that Jervis amplified some of this vaccine as soon as he arrived in Bukavu, and this latter vaccine began to be used during the first days of March, with more becoming available by the middle of the month.

The hypothesis that one of Dr Jervis’s major roles in the Congo would have been to prepare batches of vaccine locally would seem to be tenable, for all the necessary “ingredients” were present. The fact that Jervis was called in to replace Tom Norton after his heart attack indicates that in addition to an experienced vaccinator (Flack), a lab man was felt to be necessary at this crucial trial. And Dr Plotkin has admitted that it was their habit at the Wistar to produce vaccine from vaccine, rather than from seed virus,¹⁰⁴ which demonstrates that this same “easy” method could have been used in the Congo.

I have checked the details of this process with three eminent virologists who worked with poliovirus in the fifties (either in vaccine houses or labs), and these men have confirmed that to make a new batch of vaccine from scratch (making a Maitland-type cell culture, inoculating that with a small quantity of poliovirus or polio vaccine, and checking the titre of the new vaccine batch) would have taken about two weeks. All of them made it clear that this would have been a simple task, a kitchen sink operation, even back in the fifties. No special materials were needed, and it could have been carried out in virtually any lab.

Thus, Dr Paul Osterrieth’s statement that “vaccine could not have been prepared in Stanleyville” in 1958 simply doesn’t stand up.

This is the simple truth that any virologist worth his salt knows – but that I did not know when doing my interviews for The River. During the fifties it was eminently possible to make batches of polio vaccine locally, using cells from whichever primates happened to be available.

g) Local amplification of polio vaccines in Europe and Africa.

Was amplification of CHAT only done in the Congo? The answer provides further important perspective on the “hidden issue” of local production of live polio vaccines.

After my trip to Kisangani in April 2001, I undertook a further review of the papers pertaining to the world-wide CHAT field-trials which took place from 1957 onwards.

These papers (combined with certain comments by Dr Koprowski) revealed the surprising and important information that during this period it was *common practice* for live polio vaccines to be passaged again in the country which hosted the trials.¹⁰⁵

I looked, for instance, at the papers describing Koprowski's major field-trial, which involved feeding CHAT to more than seven million children in Poland, starting in June 1959. Papers written by Koprowski's Polish collaborators made no mention of local amplification of the vaccine. However, they revealed that CHAT *must have been passaged again in a locally-prepared tissue culture* before it was administered.¹⁰⁶ This practice both amplified the quantity of vaccine, and potentially boosted its titre, which might otherwise have fallen during the long sea voyage to Poland, for even frozen vaccine loses strength quite quickly after it leaves the original lab.¹⁰⁷ Subsequent amplification in the recipient lab meant that less vaccine had to be sent overseas, but that – after dilution of the amplified vaccine to a suitably immunogenic level – more persons could be vaccinated. Local amplification thus made a lot of sense.

It is not known which substrate was used for amplification in Poland, but the kidneys of rhesus or cynomolgus macaques from Asia seem very probable. The cells of these two species were then being used to make polio vaccines throughout Europe, and the Polish authors report that these same two species were used for safety testing the Koprowski vaccines in Poland.

It is known that CHAT vaccine was also amplified in Sweden in 1960-1962, using cynomolgus cells as a substrate, because a 1966 article reported this fact.¹⁰⁸ And the evidence strongly suggests that the same thing happened to the CHAT that was fed to approximately 1.7 million children in Switzerland and Croatia during the same period.¹⁰⁹

This local amplification of the vaccine virus is not mentioned in any of the early articles about the Koprowski field-trials. In fact, the first literature reference I have managed to find to local preparation of a Koprowski vaccine comes from a Yugoslav journal published in 1964.¹¹⁰

By contrast, the several articles which describe the large-scale CHAT trials of the late fifties allude only to “dilution” of the vaccine virus – or else hide behind an imprecise use of language. (The possible reasons for this apparent coyness on the part of Koprowski and his collaborators will be discussed below.)

The paper which reveals the truth about the Polish trials is interesting. In the initial references to the CHAT vaccine that was “supplied by Dr H. Koprowski”, the titre is noted as log 7.0 TCID₅₀¹¹¹ (50% Tissue Culture Infectious Doses).¹¹² Later in the paper, however, the process of sending out the CHAT vaccine from the central lab in Warsaw to the provincial laboratories is described, together with the recommendation that these labs should dilute by a factor of 500:1, to end up with 200,000 (or log 5.3) TCID₅₀ per vaccinee. This reveals that the vaccine which those labs were diluting had a titre of log 8.0, and was therefore ten times stronger than the Koprowski original. This can only mean that the original vaccine as supplied by Koprowski had been amplified in Warsaw. (Indeed, a later paper by the same team reports on the analysis of several different batches of CHAT at titres ranging from log 6.8 to log 8.3;

these batches have clearly been made locally from the log 7.0 original supplied by Koprowski.)¹¹³

When I went back to the transcripts of my second interview with Hilary Koprowski in December 1993, I found that despite his caginess about matters pertaining to Lindi and the African trials, *he quite openly acknowledged that other labs around the world had produced their own versions of CHAT. He said sometimes this was done through local passage of the vaccine itself, and sometimes by cloning (or making a new pool from a seed lot).*¹¹⁴

During the course of the interview, he stated that as far as he knew local amplification had occurred in Poland and Croatia, and that it might also have occurred in other places that had received his vaccine strains, such as Switzerland, South Africa¹¹⁵ and the Congo.

At the time of the interview, I failed to realise the full significance of Dr Koprowski's statements. But in retrospect it seems that he wished to place it on the record that anyone, once they had a sample of a live polio vaccine like CHAT (whether it be the seed virus or the vaccine itself), could have produced further vaccine locally, simply by onward passage through another cell culture.¹¹⁶

So, what of the articles about the vaccinations in the Congo? Do any of them refer to, or hint at, local passage of the virus? They do not. Just like the articles about the Polish and Swiss vaccinations, they refer only to "dilution". In Koprowski's one brief article about the early Congolese vaccinations,¹¹⁷ he merely states that the approximate minimum dose given to vaccinees in all the early trials (from Stanleyville in February 1957 to the Ruzizi trial ending in April 1958) was 5.3 log doses, or 200,000 TCID₅₀ – the same titre as in Poland.

However, there is also an article by Ghislain Courtois that describes the Ruzizi campaign from the Belgian perspective.¹¹⁸ He writes: "The vaccine used was pool 10A-11 of the CHAT strain. Since the titre of the mother-solution was 7.2 cytopathogenic units per cubic centimetre, it was decided in accord with Dr Jervis to dilute the mother-solution in such a fashion that each vaccinee received the equivalent of more than 250,000 cytopathogenic units." [Author translation.] According to this account, the "mother-solution" (which was just under 16 million units per c.c.) was diluted to produce a final vaccine which was about 63 times weaker.

What is interesting is the single page of paper relevant to CHAT pool 10A-11 which the Wistar Institute released to the Wistar's AIDS/poliovirus advisory committee in 1992, and which was later passed on to me by David Ho.¹¹⁹ This reveals that pool 10A-11, "which is to be used in the 1958 Congo trials", had been tested for immunogenicity by vaccinating infants born at Dr Flack's prison (Clinton State Farms) with serial ten-fold dilutions of the vaccine virus. This paper, which must have been written between the 23rd and 27th January, 1958,¹²⁰ revealed that the original titre of CHAT pool 10A-11, as measured at the Wistar, was 6.7 log doses,¹²¹ or 5 million units of virus.¹²²

So the Ruzizi trial vaccine, CHAT pool 10A-11, as measured by Dr Jervis in the eastern Congo, was 7.2 log doses (nearly 16 million doses of virus), and was thus over

three times more concentrated than the 6.7 log dose vaccine that had left the USA. I am told that this difference of 0.5 log doses might fall *just within* the margin of error to be expected when titrations are being performed in different labs.

But there is another factor here. In practice, polio vaccine starts to *lose* titre as soon as it leaves the laboratory where it was made. In fact, the titre of Type 1 polio vaccine apparently falls within days of preparation, even when it has been frozen at minus 20 degrees centigrade. The only contemporary paper I have been able to find concerning the loss of vaccine titre over time relates to a titration study of frozen Type 1 drageé-candies manufactured from Sabin virus in the former Soviet Union, which reports a fall in titre of 0.3 log doses within three days and of 0.6 log doses within fifteen.¹²³ Another paper by Roderick Murray and colleagues from the National Institutes of Health (NIH) which examines polio vaccine Type 1, 2 and 3 strains from Koprowski, Sabin and Cox, finds that all Type 1 strains have lower titres when tested at the NIH in Bethesda, than when tested at the source laboratory. CHAT pool 13 has a titre of 0.3 log doses lower at the NIH, while the Lederle Type 1 has fallen by 0.4 log doses, and Sabin's Type 1 has fallen by 0.6 to 0.9 log doses.¹²⁴

Since the CHAT pool 10A-11 used in Ruzizi is clearly the same CHAT 10A-11 which was tested at Clinton in January, the pool which was intended for the "1958 Congo trials", then there are only two possibilities.

One is that the vaccine was brought out on the plane by Flack and Jervis, and had therefore spent three days in a cool-box surrounded by ice. This, of course, is very different from being frozen at minus 20 degrees, but none the less, let us assume that the fall in titre was merely 0.3 log doses, like the Soviet candy-drops. This would mean that the vaccine which arrived in the eastern Congo would have had a titre of *no more than 6.4 log doses*, more than six times weaker than the 7.2 log doses of the mother-solution alluded to by Courtois. I believe that this difference of 0.8 log doses falls outside the boundaries of testing error.

The second possibility is that Jervis picked up more locally-produced vaccine from Osterrieth when he passed through Stanleyville on February 20th, at a time when Dr Osterrieth's assistant says he was already "making polio vaccine".

Whichever is the correct scenario, it is apparent that *the Ruzizi vaccine must have been locally amplified*. It is also clear that when Courtois writes that he and Jervis decided "to dilute the mother-solution in such a fashion that each vaccinee received the equivalent of more than 250,000 cytopathogenic units", he is falling in line with the other early Koprowski collaborators by mentioning the dilution, but not the prior amplification.

Further supporting evidence comes from what is known about other OPVs that were being used in Africa at this time. For instance, in 1957 an oral polio vaccine was being administered in parts of French Equatorial Africa (such as present-day Congo Brazzaville and in rural Gabon), as Simon Wain-Hobson discovered when he interviewed former workers from the Pasteur Institute satellite in Brazzaville in 1999. One of these ex-workers told Wain-Hobson that (in the latter's words) he "grew polio on local monkey kidney cultures". This man was not sure which species was, or were, involved, but said it probably included the moustached monkey, *Cercopithecus*

cephus cephus, which was and is the most common primate in the area.¹²⁵ It is not clear whether Simon Wain-Hobson specifically asked whether this man had grown polio vaccines in local primate kidney cultures, but given the fact that (apart from very minor trials) virtually all OPVs seem to have been amplified locally to boost titre, this “last step” would seem to be extremely likely. In any case, as Wain-Hobson has since put it: “the principle is established. It would be hard for anyone to deny this.”¹²⁶

In any case, there is documentary evidence that OPVs were being amplified locally in the Union of South Africa, where scientists at the Poliomyelitis Research Institute, under James Gear, “commenced cultivating and reaping the 3 strains of Sabin polio [vaccine] virus during 1957”.¹²⁷ Gear’s team was using the kidney cells of the locally abundant vervet monkey, *Cercopithecus aethiops pygerythrus*,¹²⁸ and the vaccines so produced were apparently first fed in Kenya, Uganda and Mauritius in 1959.¹²⁹ The date when South African local OPV production began is significant, because Gear and Koprowski were very close throughout this period.¹³⁰ It was Koprowski’s intention to travel to South Africa to discuss OPV field trials with Gear after his visit to the Belgian Congo in February 1957,¹³¹ though I have been unable to get definite confirmation that he made the trip.

James Gear was very much in the thick of polio vaccine research. He was on the July 1957 WHO Expert Committee on Poliomyelitis which recommended that OPV field trials could go ahead in places like Africa under certain conditions. He had been producing Salk’s IPV in the kidneys of vervet monkeys since 1954, and in an article published in June 1956, he revealed that attempts to *develop* oral polio vaccine strains were being made in his Johannesburg lab, just as in those of Koprowski and Sabin.¹³²

The most dramatic example of local, small-scale African production of OPVs, however, relates to Dr Alexandre Jezierski, the Polish vet who had, since 1953, been producing both inactivated polio vaccine (IPV) and OPV in the cells of local African primates at his small laboratory at Gabu in the eastern Congo. The reason why a Congo-based vet working for INEAC¹³³ (an agronomic research institute) had been cleared by his superiors to concentrate on experimental polio vaccine production in the heart of Africa over a four-year period is not immediately apparent, but it is known that he had close links with, and paid frequent visits to, both the Pasteur Institute in Paris, and James Gear’s labs in South Africa. After his death, one of his relatives told me that Jezierski had been unexpectedly wealthy, and that he used to move gold bars around Europe, from bank to bank, in his ancient Citroën. However, it is not known whether this is in any way related to his earlier activities at Gabu!¹³⁴

Whatever, Jezierski’s published papers reveal that he experimented with the kidneys of fifteen different African primates, including chimpanzees, and found that all of them produced “very good” cultures;¹³⁵ and that he grew his polio vaccine virus in these chimp cell cultures when he conducted comparative titrations on OPVs which he had prepared in cells from three different colobus species: *Colobus abyssinicus*, *C. badius* and *C. angolensis*. These vaccines were later fed experimentally to chimpanzees and to human volunteers.¹³⁶

At some point during the early part of February 1957, Hilary Koprowski and his assistant Tom Norton spent “about three days” with Dr Jezierski – apparently at the

small village of Epulu, where there was an animal collection centre run by Jean de Medina, which seems to have supplied Jezierski with his chimpanzees.¹³⁷ Chimps from Epulu were later supplied to Lindi camp as well.¹³⁸ Later in 1957, Jezierski was forced to terminate his polio vaccine research, and to leave his job with INEAC – for reasons unknown.

What all this underlines is that by 1957, whether it was reported in the medical literature or not, oral polio vaccines such as CHAT were being amplified in locally-prepared cell cultures, derived from locally-available primates. This was not done on an occasional basis, but routinely. This was the normal way in which OPVs were prepared for trials around the world.

Local amplification of the vaccine meant that much smaller quantities of liquid seed virus or vaccine needed to be transported on those long plane journeys, making temperature control that much easier. All that was needed was to prepare a half-litre flask or a 100c.c. bottle of vaccine, pack it around with ice, place it in an insulated box, and then have it taken to the airport.

h) Primate availability in the Congo.

In Stanleyville, between 1956 and 1958, as has already been demonstrated, the available primate was quite clearly the “chimpanzee”, though this term appears to have been used to describe both the common chimp (*Pan troglodytes*) and the pygmy chimp/ bonobo (*Pan paniscus*). The various reports of harvesting tissues and blood at Lindi make it clear that these species were locally cheap and “available”. Furthermore, since safety testing was being performed in these species, it was both logical and consistent to also use them for local vaccine production.

But what if Jervis was the one to amplify vaccines for the Ruzizi trials? What primate cells might he have used? It is reported that the mother-solution of CHAT 10A-11 was stored in the freezer at Usumbura (nowadays Bujumbura in Burundi) in early 1958.¹³⁹ I have already published an eye-witness account from Juma Jamnabas, a former microscopist at the Bujumbura medical laboratory, of chimps and other primates being held in cages behind the lab in the period up to 1957 or 1958, and their kidneys being removed one at a time.¹⁴⁰ This has been strenuously denied by Dr Plotkin in his “Postscript”, who quotes three Belgians who used to work at the lab at that time, all of whom deny that any primates were ever present there. I too have looked into this further, and can add two further denials to those mentioned by Dr Plotkin, both of which come from other Belgian health workers based at the Bujumbura lab. However, I also have two further “positive sightings”, the more compelling of which is from a Belgian who used to visit the animals regularly during the period up to and including 1958, when he was an eight-year-old boy living in Usumbura. He recalls there being monkeys and at least one chimpanzee, and believes that the primates were being used for virus research.¹⁴¹

Although this particular debate is not settled either way, it seems to me that it is far easier to explain the “negative sightings” than the positive sightings. To my mind, these three positive sightings call into question the testimonies of the various Belgian doctors and health workers who deny that primates were ever held at the Bujumbura lab.

In addition, there was from 1956 onwards a small medical laboratory which focussed mainly on malarial and bilharzial studies, at the *Mission Medicale de Ruzizi* (MMR), sited just outside the village of Kabunambo, which served as the health headquarters for the Congolese side of the Ruzizi Valley. This was where Jervis and Flack were officially based during their first four weeks in the Ruzizi Valley. The aforementioned Juma recalled driving here with one of the Belgian doctors, and says that both chimps and other monkeys were caged here as well. Again, there is partial confirmation, in that three other Africans recall monkeys (of unspecified species) being held here. Others, however, recall no monkeys at all. As with the Bujumbura lab, we have contradictory evidence from different quarters, but once again it is difficult to explain away the positive sightings from different sources. My personal belief is that monkeys and chimps were probably held in cages at both Usumbura and at Kabunambo, but only for a brief and finite period – which could well have been around the time of the polio vaccinations.

In any case, although these recollections from Usumbura and Kabunambo are intriguing, they are not crucial to what happened at the Ruzizi Valley vaccinations. If Dr Jervis (rather than Osterrieth) was indeed the one to amplify the vaccines used in Ruzizi, then the likeliest place for him to have done so was at the lab in Bukavu, where it seems he spent much of his time during his African visit. In 2001 I visited Bukavu for the first time, and made repeated attempts to locate the archives of the medical laboratory. I eventually gained access to the director's room in that building, which had been locked and nailed shut, and where, sadly, all that remained were some papers stuffed haphazardly into a filing cabinet.¹⁴² However, in another room I located the blueprints of the lab, and for the large *animalier* which had been built alongside it, many of the brick walls of which are still standing today. The plans showed that there was a section in the animal house devoted to “monkeys”, and the presence of both monkeys and chimpanzees has been confirmed by two ex-workers at the lab.

What this boils down to is that if Jervis amplified the vaccines for Ruzizi, he would have had no shortage of available material. He could, for instance, have obtained chimp kidneys and sera from Osterrieth in Stanleyville, or he could have used chimpanzees or other primates from Bukavu (or possibly from Usumbura or Kabunambo). It is my belief that if Jervis used local primates to produce primary tissue culture to amplify the vaccine, he most likely would have used chimpanzees, for by that stage this was the African primate species which was best known and characterised for the Koprowski collaborators.

i) Commercial concerns.

The fact that local vaccine amplification was not spelt out in contemporary articles by Koprowski and his collaborators would seem to have largely been prompted by commercial concerns, and the fear that competitors might misappropriate one's carefully-attenuated strains – for instance by obtaining a phial of vaccine, or even a stool from a vaccinee.

As Hilary Koprowski once put it to me about his live vaccine strains: “There was no proprietor. As far as I know there was no patent.” In 1960, Albert Sabin (who had distributed his strains quite widely, to places such as the USSR, Singapore and South

Africa), wrote to the WHO, stating: “I would like to say that nobody who has received [vaccine strains] from me is authorised to give them to anyone else without my permission.”¹⁴³ Although the basic passage histories of the Sabin strains had been published in the fifties, it was only in the mid-seventies, long after they had been adopted on a global basis, that Sabin and a colleague wrote a paper detailing the full histories of the strains.¹⁴⁴ This paper described the OPVs which had been prepared in different labs and different countries around the world in terms such as “SO+3” or “SO+4”, to indicate how many further passages had taken place from the “Sabin original” seed strains.

But what is apparent is that the 1950s race to develop effective oral polio vaccines was not only a heroic attempt to control, and even eradicate, a much-feared disease. It was also quite clearly a commercial race, which is why pharmaceutical companies like Lederle (part of American Cyanamid) were involved, and why scientists like Pierre L epine were trying to patent their polio vaccines in the U.S.¹⁴⁵ Not unreasonably, the scientists who had spent years developing their OPV strains wanted to try to protect their considerable investments.

This, I believe, is one of the reasons why no overt reference was made in Koprowski’s papers published in the late 1950s to the onward passage of the vaccine virus in locally prepared tissue culture. From a commercial perspective, he didn’t want to draw attention to the fact that a single phial of vaccine would have been enough to produce enough batches of fresh vaccine to immunise millions.

However, by the mid-sixties, it was no longer necessary to try to keep this a secret, which is why articles about local production of CHAT begin appearing in 1964. By the time I interviewed Koprowski in 1993 there was even less need for secrecy, which is why he was quite open about the fact that local passage of his vaccines had occurred in other labs. Unfortunately, I wasn’t quick enough to pick up on the importance of what he was telling me.

But was this practice of local passage also safe? Until the sensational announcement about the discovery of SV40 in rhesus and cynomolgus cultures in 1960,¹⁴⁶ none of the other 39 adventitious viruses which had been found in monkey kidney tissue culture appeared to be pathogenic for humans. The more far-seeing of virologists (like Sweden’s Sven Gard) expressed concerns about several safety aspects of live vaccines like OPV,¹⁴⁷ but most of those developing live polio vaccines continued working on the basis that the risk was worth it. The question of the safety of the vaccine substrate was all but ignored.

The only time that Hilary Koprowski formally mentioned the question of vaccine substrate during the 1950s, he made it clear that he considered it an issue of little importance. This is what he wrote in a paper published in 1956, just as Lindi camp was opening: “The source of material used for virus cultivation cannot be disregarded altogether. It should be represented by tissue that is least apt to harbor human pathogens – although the dilution factor which can be applied to a poliomyelitis virus suspension may be beneficial for the elimination of other ‘passenger’ viruses.”¹⁴⁸

This paper by Koprowski was originally delivered as a speech at a symposium on “Newer knowledge of viral and rickettsial diseases” held in November 1955, which

focussed on the problems and benefits of different tissue cultures. A remarkably prescient paper delivered at that conference by John Enders' former collaborator, Tom Weller, examined the potential pitfalls of using tissue culture as they "might affect the operations of a hypothetical team of virologists...in Uganda".¹⁴⁹ He pointed out that as an alternative to bringing in tissues from abroad, "the [Uganda] group might equally rely on tissues, either of monkey or human origin, locally available". Weller emphasised the problem of hidden contaminating viruses which might be lurking in the monkey tissues, and which might cause visible cytopathogenic effect (CPE) in tissue culture only after a lengthy period, and he concluded that although such problems could be minimised, they "cannot be entirely circumvented".¹⁵⁰

One wonders what Koprowski thought of Weller's speech. His own approach to such potential problems with substrate seemed to be one of circumvention rather than cure, for this was one of three papers he wrote in 1956 and 1957 in which he claimed to be growing his polio strains in chick embryo tissue culture, when in reality he was already using some variety of primate kidney tissue culture. The reason for Koprowski's misreporting his polio vaccine substrate is not known.

j) The true purpose of Lindi camp.

In recent years, Stanley Plotkin and Hilary Koprowski have frequently repeated that the original purpose of Lindi camp was to test the safety and immunogenicity of Koprowski's polio vaccines in chimpanzees. No persuasive evidence has ever been presented to support this claim, and it now appears an inadequate explanation for the establishment of such a complicated operation. Back in 1994, Stanley Plotkin admitted to me that "I don't think a lot of real importance came out of those studies".¹⁵¹ And indeed, from the account provided by Koprowski, it would appear – anachronistically – that CHAT was fed to humans in Africa before it was tested on chimps.

It is documented that the first open trials of CHAT began in Stanleyville in February 1957,¹⁵² and the fact that CHAT vaccination was occurring at the time of Koprowski and Norton's visit to that city at the start of that month is proved by a contemporary photo which was published in *Time*, which shows Koprowski and Osterreith at Lindi camp, watching an African woman down vaccine from a tablespoon.¹⁵³ Since Koprowski stayed in Stanleyville for less than a week, which is not long enough for results to be obtained from vaccination and challenge or from intraspinal safety testing, it follows that (as with his first vaccine, TN, in 1950)¹⁵⁴ human subjects were actually fed the vaccine *before* its safety and immunogenicity had been assessed in chimpanzees. This suggests that testing the safety and immunogenicity of CHAT and Fox were not the *raison d'être* of Lindi camp.

All of which begs the question. What did the CHAT-related "experiments in chimpanzees", which had to be conducted at Lindi camp, actually comprise? Or, to put it another way: why would Koprowski go to such expense and trouble to set up a chimpanzee camp in the middle of the rain forest if the vaccines which it was allegedly vital to test there had already been publicly declared fit for human use, and were already being tried out in human "volunteers"?

What the scant historical records reveal is that polio-related experiments were carried out on a total of 83 Lindi chimps up to February 1958 (this representing 20% of the 416 chimps “used” in those twenty months). This work might have required the sacrifice of 48 of the 83 animals. Furthermore, it is apparent that most of the work (just as Plotkin hinted) failed to produce any significant scientific information. This was not least because chimps are a far less accurate barometer of vaccine safety than the lower monkeys – a fact which Koprowski already knew before he started.¹⁵⁵

However, the local press reported that while they were in Stanleyville Dr Koprowski, assisted by Tom Norton, “initiated Dr Courtois, as well as his assistants, doctors Ninane and Osterrieth, into his methods of work”.¹⁵⁶

So what was the work into which they were initiated? Was it intraspinal inoculation? This work is highly skilled, and it seems possible that most, if not all, was carried out by Koprowski’s experienced lab man, Tom Norton, during his six week stay in February and March 1957. As for vaccination and challenge (using whichever strains), this required little in the way of training.

I believe it likely that the principal technique which Tom Norton was teaching the three Belgians in February and March 1957 was how to make tissue cultures from chimp kidney cells and sera, in order to amplify the vaccines. At this stage, in early 1957, the brand new medical lab had not yet been opened, but the old brick-built laboratory was fully operational, and certainly Maitland-type cell cultures could have been prepared there.¹⁵⁷ It may also be that the early attempts at tuition were not completely successful, because when Ninane departed on his triannual leave in late March 1957, and Osterrieth in July 1957,¹⁵⁸ they both received additional training in tissue culture preparation: Ninane at Lise Thiry’s lab in Brussels, where he apparently spent about “ten to twelve weeks”; Osterrieth at the Wistar Institute in Philadelphia. During those periods, CHAT vaccine was present in both labs.¹⁵⁹

However, is it possible that tissue culture preparation may have started even earlier? The 1956 annual report of the medical laboratory has just this to say about Lindi camp: “Poliomyelitis. In collaboration with Doctor H. Koprowski, Vice-President of the New York Academy of Sciences, trials are in progress at the camp of experimentation at Lindi. More than 60 chimpanzees (meant for the study of an oral attenuated virus-vaccine) have already been used in the first trials.”¹⁶⁰ Since CHAT was only brought out to Stanleyville in February 1957, it would seem that these “first trials” in 1956 must have involved a different vaccine. Now, we know that pool 14 of Koprowski’s previous Type 1 vaccine, SM N-90, was present at Lindi, and was still being used in research as late as August 1957. SM N-90 (pool 14) would have been in existence by 1955 or (at latest) early 1956, so this Lederle-made Type 1 vaccine was almost certainly the one utilised for the 1956 trials, with 60 chimps being “used”, perhaps as a dry run for the CHAT vaccine that would arrive with Koprowski the following year.¹⁶¹

Whether chimp tissues and sera were used to amplify SM N-90 pool 14 back in 1956 is, of course, not known. However, one thing is certain. The camp at Lindi was not a small, or easy, or inexpensive operation to set up in the middle of the Congolese rain forest – and yet the scientific benefits which could be gained from its *alleged*

programme of testing the safety and efficacy of OPVs were relatively small. Clearly something is wrong here.

The true purpose of the chimp colony has never been revealed, but certain clues [see below] begin to suggest that the real *raison d'être* for Lindi may have been to test the safety of Koprowski's vaccine *substrates*, rather than that of his vaccines. The latter had, in any case, already been tested quite extensively on monkeys and humans (eg the infants born at Clinton State Farms, New Jersey) in the U.S.

There is also another possibility. Given the very detailed accounts by Joseph, the camp nurse, and by "Antoine", of chimpanzee organs being put into canisters (either in formalin or in a watery liquid, which sounds like Hanks' solution), and then packed off to the USA (and possibly Belgium), accounts that were in large part confirmed by the Stanleyville vet, Louis Bugyaki, it seems that there were possibly other research programmes which linked in to Lindi camp, from the time of its June 1956 opening. If this hypothesis is correct, then the polio research conducted at Stanleyville may not have been the sole reason for the establishment of Lindi. According to this scenario, it may be that chimp kidneys were used for local vaccine production mainly because they were available, as a by-product of the sacrifice of hundreds of chimps, and scores of bonobos for other purposes.

k) The possible use of other tissue cultures.

Since 1994, a number of investigators (such as Billi Goldberg, Blaine Elswood and Raphael Stricker) have proposed that the polio vaccine used in the Congo might have contained a simian immunodeficiency virus (such as SIVcpz) which became human-adapted after being further passaged either in human diploid cell strains (such as the varieties from WI-1 to WI-38, which were developed at the Wistar by Leonard Hayflick),¹⁶² or else in human cell lines (such as WISH, also developed at the Wistar, and HeLa).¹⁶³ The use of human cells, they proposed, might not only have helped an SIV or SIVs to adapt to humans, but might also have allowed different SIVs to recombine *in vitro*, before crossing to humans *in vivo*.¹⁶⁴

For many years I have rejected this idea, mainly because the timings did not seem to fit. Firstly, there was evidence that WISH was not produced until the end of 1958, and did not transform into a cell line until the last day of that year. Secondly, it appeared that Hayflick's human diploid cell strain series, WI-1 to WI-38, did not begin to be produced until 1959, which was the year when Professor Sven Gard was on sabbatical at the Wistar, and when he appeared to have arranged a supply of the embryos on which Hayflick's work was based, obtained from abortions conducted in Sweden. If correct, this meant that amplification of polio vaccines in such substrates could not have started until 1959 or 1960 – which appeared to be too late to have sparked the Group M outbreak.¹⁶⁵

There was also another reason, for it appeared that HeLa, by itself, was unable to grow HIV or SIV, but only in the form of a genetically engineered cell line like HeLa T4+.¹⁶⁶ Such a modified cell line, I was informed, could not have existed before the 1980s.

Furthermore, although HeLa grows poliovirus to a high titre, it was widely accepted (even in the fifties) that this cell line could only be used for research and diagnostic purposes, because of the potential risks of preparing a human vaccine in a culture derived from a particularly vigorous human tumour. For instance in 1954 the Armed Forces Epidemiological Board, the members of which were not renowned for being especially faint-hearted, ruled out HeLa for the production of an adenovirus vaccine for soldiers.¹⁶⁷ So although HeLa had been present in the Congo from 1954 onwards, there was no evidence to suggest that anyone had actually used it as a polio vaccine substrate.

However, in the course of writing the present paper, I was reminded of certain important details, such as Ninane's claim that he was trying to make human cell cultures in Stanleyville, and the fact that Fritz Deinhardt had extensive experience of working with this type of culture. And so I decided to look once again into the possibility that human cells might have been used for polio vaccine production in the Congo.

Whilst I would wish to emphasise that I still differ from the "Goldberg/Elswood school" on several issues, I do now believe that their long-standing hypothesis (that the additional preparation of CHAT vaccine in human cells contributed to the birth of AIDS) may perhaps have merit.

Gaston Ninane told me several times that he had been working with human cells in his lab – but which cells might these have been? Firstly, because he spoke in terms of "trying to make cultures" from human cells, it appears that he was not talking about HeLa, a line which grows continuously in culture, and which is notoriously robust. So what other possibilities are there?

At Lise Thiry's lab in Brussels, where Dr Ninane trained in the summer of 1957, they had been growing CHAT and Fox in different batches of "monkey kidney", and in several different human cells. These included human cell lines (such as Eagle's KB cells, Chang's liver cells, and T1 kidney cells) and human amnion cell lines (including two, "N" and "LoFi", which were reported as being susceptible to poliovirus). They had also grown CHAT and Fox in other cultures too, including SCH, a cynomolgus heart cell line obtained from Jonas Salk, and even a line of "transformed embryo rabbit kidney", ERK-1, which grew poliovirus to a surprisingly high titre.¹⁶⁸

Interestingly, it seems that others who were directly involved in this story were also doing research along parallel lines. Fritz Deinhardt and the Henles were working with several different human amnion cell lines in their virus lab at CHOP in Philadelphia, and by 1955 or early 1956 these included intestine 407, liver 407, MAF-E, and another, "Lung TO", which they subsequently supplied to Lise Thiry's lab.¹⁶⁹ By February 1958, they were reporting work on three other human amnion cell lines (Line T, 103 and 185) which they had apparently obtained from Leonard Hayflick.¹⁷⁰

Meanwhile, at the Wistar itself, between 1953 and 1957, a virology research unit was set up under Geoffrey Rake and William McLimans, which experimented with several different types of tissue culture, and engaged in the semi-industrial production (in 5-litre spinner cultures, and 20-litre stainless steel fermentors) of viruses such as the

polioviruses in substrates such as HeLa, Chang's conjunctival cells, FL (a human amnion cell line), and the embryonic rabbit kidney cell line, ERK-1.

Leonard Hayflick, who worked at the Wistar for most of the fifties, told me that this period, just after John Enders proved that you could grow poliovirus in human cells (and ones which, in contrast to the previous accepted wisdom, did not derive from nervous tissue), was "the Golden Age of Virology".¹⁷¹ Between 1952 and 1956, Hayflick did his doctoral dissertation at the Wistar on the growth of mycoplasmas in tissue culture, and then he did two years of post-doctoral work, again concentrating on cell culture, at Charles Pomerat's lab in Galveston, Texas. (Pomerat, known to his friends as "Pom-Pom", had headed the U.S. Navy's Subtropical Marine Laboratory at Woods Hole during the second world war.) Hayflick returned to the Wistar at the end of 1957, after Koprowski had taken over as director, "to organise a cell culture lab and provide cells of different types to the investigators".¹⁷²

There was in fact a small coterie of virologists (including Koprowski, McLimans, Rake, Pomerat, the Henles, Deinhardt and Thiry) who were at the forefront of research into human cell cultures in the second half of the fifties, and there were close links between different members of the group, which extended to the young scientists (like Hayflick, Osterrieth and Ninane) whom they trained. Pomerat oversaw Hayflick's post-doctoral work, and Hayflick then moved back to the Wistar (together with two others from Galveston, Moorhead and Fernandes). Hayflick thanked Thiry for her help at the end of his WISH article, and he provided human amnion cell lines to the Henles and Deinhardt. They, in turn, provided one of their cell lines to Thiry. Thiry trained Ninane. The Wistar trained Osterrieth, and while in Philadelphia Osterrieth became close friends with Deinhardt.

The one figure linking all these people together was Hilary Koprowski.

One particular preoccupation of these scientists during the "golden age of virology" was the question of which cells would grow which viruses, and – in particular – which cells would grow the benchmark viruses of the era, the polioviruses. In short, there was a lot of experimentation with different substrates, which tended to be reported separately from what some apparently viewed as the pure virus research, on subjects such as attenuation.

And there was one particular substrate that looked especially promising in 1957 and 1958. At that key polio conference in Geneva in July 1957, the one which was attended by Koprowski, McLimans, Thiry and Ninane, and the one at which the WHO Expert Committee gave the go-ahead for open trials of OPV "in the face of epidemics", William McLimans from the Wistar spent ten minutes or more singing the praises of the embryonic rabbit cell line, ERK-1. He reported that in the Wistar labs all three types of poliovirus grew to very high titres in ERK-1, with 7.5 log doses or better being obtained within 40 hours. He stressed that, because of the fear of malignant agents, stable cell lines of human or monkey origin should not be used for producing polio vaccines – at least not IPVs, which were inoculated into the bloodstream. However, he continued, the use of a stable cell line from a species far removed from man (such as the rabbit) would "obliterate the fear...of malignancy". He ended up by posing a question: "can we take the monkey business out of polio vaccine production?"¹⁷³

In fact, in several respects ERK-1 seemed a miracle – the first non-primate cell line to be discovered that grew poliovirus, and – furthermore, which did so to titres that were just as high as those of HeLa.¹⁷⁴

Should anyone have seen the warning signs? Although misgivings were officially expressed in the early sixties,¹⁷⁵ it was not until 1966 that a geneticist, Stan Gartler, pointed out that many of these cell lines appeared to be not just similar, but the same – and they all looked like HeLa. Apparently the cell biologists in the audience received the news with extreme hostility.¹⁷⁶ So it was not until 1973 that the painstaking work of Walter Nelson-Rees *proved* that many of the world’s commonly-used cell lines had in fact been taken over by HeLa cells. The HeLa cell line was so vigorous that if someone opened a stoppered flask of it, or pipetted a sample, a little carelessly, and sent a few micro-droplets drifting into the air to land on another cell culture, the latter would be outgrown and replaced within days. Suddenly it became apparent why, back in the fifties especially, so many normal cells (like human amnion cells) had suddenly miraculously “transformed” into cell lines. In reality, their nests had been taken over by HeLa cuckoos.

Among the cells which Nelson-Rees “outed” as HeLa cultures in his two famous articles in *Science*, in 1976 and 1981,¹⁷⁷ were most of those really productive cell lines of the fifties.

These included seven of those mentioned above: KB, T-1, FL, Chang’s liver cells,¹⁷⁸ Salk’s cynomolgus heart, Henle’s intestine 407, and Hayflick’s WISH.¹⁷⁹ All seven cultures had been present in the labs of Koprowski, the Henles and Fritz Deinhardt, and Lise Thiry, in the period 1955-1960, and all seven had been colonised by HeLa.

The Nelson-Rees announcement was one to which many members of the medical establishment did not respond either wisely or graciously, and shortly afterwards, he was rewarded with the sack. Several of his detractors seemed determined to ignore the bad news, and to go on as if nothing had changed. For instance, when Jonas Salk rather courageously admitted, at the Lake Placid conference in 1978, that some of the injectable polio vaccine he had produced in the fifties must have been mistakenly prepared in a culture of HeLa, the organisers apparently persuaded him to omit this detail from the published proceedings.¹⁸⁰ Even today, many cell lines are wrongly described, and, as a contemporary article expresses it: “Chaos reigns and fraud – unwitting or deliberate – is condoned”.¹⁸¹

Nowadays it is known that polioviruses grow only in primate cells, so whatever the ERK-1 cell line was, it was certainly not embryonic rabbit kidney. Walter Nelson-Rees never reported in the literature on this particular cell line, but written on one of the copies of his own 1981 paper is a contemporary note, in his hand, which indicates that “ERK-1 (rabbit) of Westbrook, 1957, was also shown to be HeLa.”¹⁸²

It is clear that even the 1981 list from Nelson-Rees was not exhaustive, for he was not invited to test every cell culture that was then in use. An example of another possible omission is Chang’s conjunctival cell line. However, Nelson-Rees has recently sent me a page of detailed documentation on this subject, which concludes: “I am 99.99% certain that Chang’s conjunctiva [cells], like his liver [cells], are HeLa”.¹⁸³ Nowadays,

he says that many of the other cell lines of the era are also likely to have been HeLa, from the moment that they miraculously “transformed” from normal cells. And of course, it is precisely because they had transformed and become more robust that virologists valued the presence of such cells in their laboratories.

It is worth reiterating that four of the five major cell lines being researched at the Wistar in the mid-fifties (HeLa, FL, Chang’s conjunctival cells and ERK-1) were actually different forms of HeLa.

The repeated failures by doctors Koprowski, Plotkin, Ninane, Osterrieth and others to give a proper account of what they were doing in the Congo, the increasingly unbelievable retrospective denials that polio vaccines were, or could have been, prepared there (denials which are quoted extensively by Dr Plotkin, as if he and Dr Koprowski were unaware that local vaccine amplification was taking place), the ongoing attempts to persuade witnesses around the world to modify their stories – all these things lead one to suspect that it may be that more than just chimpanzee cells were being field-tested in the Belgian Congo in the late fifties.

Meanwhile, there is some rather significant evidence “from the horse’s mouth”. In their various polio articles in the mainstream medical literature in the years up to and including 1960, Doctor Koprowski and his various collaborators hardly, if ever, mentioned the possibility of using human cells for making oral polio vaccines. However, in a lecture which he delivered in Kenya and South Africa in July and August 1955, and which was later reprinted in the *South African Medical Journal*, Koprowski presented two tables which encapsulated “the author’s views on the acceptability of attenuated viruses as vaccines”. One of these tables (sub-titled “Personal Credo of the Author”) listed various potential OPV tissue cultures, and made it clear that both “monkey epithelial” and “human epithelial” cultures would be considered acceptable as substrates.¹⁸⁴

The next reference by Koprowski to the use of human cells as OPV substrates that I have been able to find came five years later, in November 1960, when he and Plotkin co-wrote a letter to the WHO, entitled “Notes on acceptance criteria and requirements for live poliovirus vaccines”.¹⁸⁵

In a section entitled “Viruses other than polio”, they analysed the new-found (post-SV40) fear of adventitious simian viruses which might cause human cancers. “Any tissue that is obtained from a normal animal”, they wrote, “may be parasitized by viruses probably harmless to the host most of the time. When such an organ is removed from the host and the cells allowed to multiply outside the control of the whole organism, as, for instance, in tissue culture, the virus ‘infected’ cells seem to multiply...and the virus which parasitized them is released.” Their response to this, however, was a pragmatic one, for they suggest that “vaccine be allowed to contain these agents if the titre is so low that each human subject will receive only a small dose of these [adventitious simian] viruses.”¹⁸⁶

Then, in a section titled “The phantom of cancer virus”, Koprowski and Plotkin begin to get more controversial. “One may consider that the chance or the presence of a cancer virus in a cell-free preparation obtained from monkey kidney tissue culture is

as small or as great as in a cell-free product obtained from cultures of HeLa cells, even though the latter originated from a malignant tumour of man.”

And then comes the punch-line. “Even if HeLa cultures”, they write, “will not be considered as suitable menstrea for growth of poliovirus strains, there are at present in existence several tissue culture lines which have been originally isolated from normal human embryo, and which grow only for a limited number of passages (30-40) *in vitro*. These culture lines which are susceptible to poliovirus growth have morphological and chromosomal characteristics of a *normal human cell*.”

This is clearly one of the first public references to Hayflick’s human diploid cell strains (WI-1 to WI-38), which would later feature in an article by Hayflick that was published in 1961 (but which was apparently first submitted, and rejected, in 1960).¹⁸⁷ At around the same time, Hayflick had also been examining the potential of another culture, WISH (Wistar Institute Susan Hayflick), based on amniotic cells obtained at the birth of his own daughter in November 1958, which transformed into a cell line on the last day of that year.¹⁸⁸ Like the WI-1 to WI-38 series, WISH was good at growing Koprowski’s polio vaccines, CHAT and Fox. Walter Nelson-Rees later identified WISH as yet another front for HeLa, although Leonard Hayflick has apparently never accepted this.¹⁸⁹

The question that has to be asked is: given Koprowski’s ambivalent position, between 1955 and 1960, about the use of human cells for making OPVs, and the fact that human cell substrates of various types were being prepared at the Wistar Institute between those same years, 1955-1960, just what is the possibility that Koprowski and/or his collaborators might have tried out vaccines prepared in human cells on “volunteers” in central Africa during this period? This category would include vaccines prepared in human amnion cells, or HeLa cultures, or human cell lines that were really HeLa, or other cell lines (like ERK-1) that were really HeLa, or Hayflick’s new “semi-stable human cells” (such as the diploid strains, the WI-1 to WI-38 series).

Two points need to be appended here. Firstly, there is substantial evidence that other hitherto untested, and potentially unsafe medical, pharmaceutical and chemical preparations were administered experimentally to populations in central Africa during this period, especially during the late fifties.¹⁹⁰ Some of these trials were fully reported in official documents, but for others one has to read between the lines, or follow up on stray references and other, similar clues. Secondly, some of these trials took place without even a nod towards what is nowadays referred to as “informed consent” – and it is clear that as independence approached, the time for conducting such trials on convenient “volunteers” in Africa was growing short.¹⁹¹

I wrote earlier that I have long been sceptical that polio vaccines made in human cells could have been tested in Africa, and could have been connected to the birth of AIDS. However (and not for the first time), Bill Hamilton was more far-seeing than I was. In a hand-written note he sent me about a draft of one of Blaine Elswood’s early papers on this subject, he commented as follows: “I haven’t had time to read his piece and size up his references with full care, but at the moment I don’t see any major snags with his ideas, and don’t see them as exclusive to most of yours about the Congo events. K. [Koprowski] may have been experimenting with several culture techniques,

and [viruses in] kidneys brought back from the Congo could have infected HeLa lines in his labs. Maybe these lines were used in the Congo, maybe Pan paniscus kidney cultures...I had forgotten (if I knew) that HeLa was long known as an excellent medium for poliovirus, but now reminded, I can see that for a man as determined and uncaredful as K., any cell lines that reared the virus well would have been tempting ...¹⁹²

There appears to have been a considerable degree of interest in the African CHAT trials by scientists who were at the forefront of research into human cell cultures. Apart from the frequent visits by members of the Koprowski group at the Wistar, there was Fritz Deinhardt's three month visit to the Congo in early 1958. The fact that Deinhardt is reported to have been present at some of the CHAT trials (apparently those in Stanleyville itself)¹⁹³ suggests that his visit may not have been prompted solely by the hepatitis research, as does the fact that for some reason his bosses, the Henles, were apparently unenthusiastic about his visiting Stanleyville.¹⁹⁴ Also interesting is the visit by several Belgian virologists (such as Lise Thiry, Piet De Somer and E. Nihoul) to the Stanleyville lab for the virus symposium in September/October 1957, a visit which some of them combined with longer stays in the country. De Somer, who later (in 1959, it seems) produced CHAT vaccine for use in Burundi at the Belgian pharmaceutical house, RIT,¹⁹⁵ stayed on in the Congo for two more months, and is said to have become a keen supporter of OPV during that visit.

Then there are other intriguing links. Lise Thiry met Agnes Flack at Brussels airport on her way out to the Ruzizi trials, and later spent two days with her when she passed through Brussels on her way back home, which suggests that she may have had some particular interest in the progress of those trials, or in certain sections thereof.¹⁹⁶ Again, there is the fact the CDC was apparently interested, shortly after independence in the Congo, in testing pre-vaccination and post-vaccination blood samples from one of the final CHAT trials, that at Coquilhatville.¹⁹⁷ Even though this mooted collaboration apparently never materialised, is it possible that there was something especially interesting about that particular trial?

But let us leave conjecture, and return to documented facts. Precisely which tissue cultures were available, or being used, in Belgian colonial laboratories during the 1950s? I can find no records mentioning ERK-1 cells, although this is not to say they were not present. What is documented is as follows. The first lab to make tissue culture in the Congo was the virology lab that opened in 1954 as part of the *Laboratoire Médical d'Elisabethville*, in the heart of the Copper Belt, in Katanga province in the south of the Congo. This lab used HeLa cells from late 1954 onwards,¹⁹⁸ and human amnion cells from an unspecified date between 1954 and 1957. (The virology lab was conveniently situated near to a maternity department.)¹⁹⁹ With regard to the capital, Leopoldville, Dr Michel Vandeputte told me that he joined the central laboratory there in July or August 1956, soon after which he was asked to set up a virus lab. He wrote to Stefan Pattyn, who then headed the virus lab in Elisabethville, and they exchanged sera, viral strains and tissue cultures, namely HeLa cells and amniotic cells, which, Vandeputte told me, "we used more or less for experimental purposes". The Leopoldville virology lab opened in October 1957, and subsequent papers show that Dr Vandeputte was using both HeLa and human amnion cells by that month at latest.²⁰⁰ Vandeputte told me that the Leopoldville lab had mice,

rats, guinea pigs, rabbits and monkeys (which in this case would appear to mean “primates of unknown species”, which may or may not have included chimpanzees).

Two other large and impressive purpose-built medical laboratories also opened in the Belgian Congo in 1957, at Stanleyville and Bukavu. Both had virology sections, and large animal houses, and we know that both either had (or had ready access to) chimpanzees. We do not know which tissue cultures were available at Bukavu, but (as explained above) it appears that Maitland-type tissue cultures based on chimpanzee cells and sera were present at the Stanleyville virology lab by February 1958 at the latest. However, the fact that there was a mass sacrifice programme of the Lindi chimps, beginning in the second half of 1956, and that Courtois was doing research with chimps in Stanleyville even before Lindi camp opened, suggests that these types of cultures may have been prepared at the *old* medical laboratory from as early as 1955. According to Dr Osterrieth, it was not until “several months” after February 1958 that he began attempting to make tissue cultures using trypsin, and he only succeeded in making a few trypsinised cultures during 1958, these being from baboons. He states that he first obtained HeLa cells in 1959. Gaston Ninane, meanwhile, spoke to me about trying to make human cell cultures during 1957, and it seems likely that, like Pattyn in Elisabethville, he could have been using amniotic cells obtained from the local maternity ward.

If we collate the documentary and testimonial evidence, then HeLa cells were available in the Belgian Congo from 1954 onwards, human amnion cells (and perhaps cell lines) from some time between 1954 and 1957, chimpanzee cultures from some time between 1955 and February 1958, and trypsinised monkey kidney tissue cultures (for instance from baboons) from around the middle of 1958. The relevant articles refer only to “amniotic cells” and “human amnion cell tissue culture”; no attempt is made to clarify which of these, if any, had “transformed” into human amnion cell *lines*. However, it is worth repeating that in many other labs where human amnion cells and HeLa were both present during the 50s, it was only a matter of weeks, or months, before the amnion cells were overtaken and colonised. One article from Gertrude Henle and Fritz Deinhardt describes five such “transformations” in the CHOP virology lab in the space of a few months in 1955-6.²⁰¹

It is likely that different variants of HeLa (*both recognised and unrecognised*) were present in Congolese laboratories from an early stage. Whether or not any of these cells were ever used as a polio vaccine substrate is not known. However, it certainly seems possible that the very availability of these cells, especially those which were officially “not HeLa cells”, might have led to their being assessed in one or more of the vaccine field-trials.

1) The potential significance of HeLa contamination.

The fact that chimpanzee cells (which may well have been SIV-contaminated) appear to have been used to make human vaccine in Stanleyville in the period up to April 1958 is, in itself, alarming. But if such vaccines, in turn, were subsequently contaminated with, or passaged in, or diluted by, HeLa cells, then this could potentially have been far more serious in terms of the impact on human health.

Although much work has been done on the natural history of HIV in the human body (and of SIV in the bodies of primates), many details, such as the precise mode of viral entry, remain uncertain. What is clear, however, is that both lymphocytes and macrophages are prime target cells for HIV and SIV. However, there is a difference, for SIV-infected lymphocytes have one furious burst of virus production *in vitro*, and then die off. By contrast, SIV-infected macrophages continue throughout the life of a culture to spew SIV out into the supernatant.²⁰² Macrophages do not die off and, as Robin Weiss has observed, they make up approximately 1% of all epithelial cell cultures.²⁰³ They are sometimes referred to as cellular vacuum cleaners.

So let us imagine a chimp cell culture of which one part in a hundred is made up of SIV-contaminated macrophages, which is then combined with cells that are described as KB, or FL, or ERK-1, but which are in fact HeLa. Is it conceivable that SIVcpz could grow in HeLa? When I first asked this question of microbiologists and virologists back in the late nineties, most of them said that this could only happen in a genetically engineered cell line, such as HeLaT4+, which was not created until the 1980s. But is that really the only way?

What follows in the next two paragraphs is sheer speculation, for I can find nothing in the literature about whether or not SIVcpz will grow in a HeLa culture – or, indeed, about what impact HeLa might have on the chimp virus, if it did support its growth.

Some microbiologists I have spoken with believe that because macrophages are fusogenic, the act of transferring chimp cells containing macrophages into HeLa, that most turbocharged of tissue cultures, could in itself generate a hybrid cell line which would combine chimp CD4 cells with the immortality of HeLa. They say that such a hybrid cell line would, from that point on, represent an excellent substrate for growing SIVcpz (for instance if SIVcpz-contaminated tissue culture was introduced into the HeLa/chimp hybrid). Furthermore, they say, the necessary process might be even simpler, and require just a single step – that of putting primate cells which contained already SIV-infected macrophages into HeLa. However, in this instance the outcome is less readily forecastable, in that the hybrid cells might spew out SIV, or might be killed off by the SIV.

If such a hybrid HeLa/chimp cell line was accidentally created in the course of the CHAT vaccine research that was taking place in the Congo during the fifties, then the potential implications could have been “a recipe for disaster”, in the words of one respected microbiologist. Such a cell line could introduce a crucial amplification step to the basic OPV theory, by allowing the mooted chimp SIV contamination of CHAT to become human-adapted. Furthermore, if two or more SIVs were present in a HeLa/chimp hybrid culture, they would be likely to recombine even more rapidly than in a chimp cell substrate. [For the potential implications of such recombinations, see “Dating the epidemic”, below.]

I must repeat that such analysis clearly remains in the realm of the hypothetical – at least until such time as someone stages an appropriate *in vitro* experiment.

The basic OPV theory, involving a contaminated chimpanzee tissue culture used to make the polio vaccines fed in the Congo, stands up on its own. However, if HeLa contamination of those cultures also occurred, this just might represent the “extra

ingredient”, the amplification step, that could help to explain why this particular zoonotic transfer was some orders of magnitude more serious, in terms of human disease, than those other SIV transfers which resulted in the human outbreaks of HIV-2, and HIV-1 Groups O and N. These latter three outbreaks may have resulted from contact with bushmeat, or from iatrogenic (possibly polio vaccine-related) episodes [see below] in west Africa and west central Africa, but they have probably resulted in fewer than 20,000 fatalities in total.²⁰⁴ By comparison, by 2002 the Group M-related AIDS pandemic seems to be some three orders of magnitude greater, having caused an estimated 20 million deaths.

At this point, two historical details which may be relevant to the hypothetical CHAT/HeLa scenario need to be mentioned. Firstly, an article from a Leopoldville newspaper in August 1958, published a week before the start of the vaccinations of all the children aged five and under in the capital, reported that the new polio vaccine of Dr Koprowski “has been prepared at Elisabethville by the Wistar Institute, and is controlled from the point of view of efficacy and safety by the Stanleyville laboratory”.²⁰⁵ To date, nobody has been able to explain what this sentence actually means. **[Figure 5]**

The opening paragraph of this article reveals that the new polio vaccine in question had previously been “perfected [*mis au point*] by Koprowski at Stanleyville, in collaboration with the medical services of the Congo”. The phrase *mis au point* also embraces the idea of something which is being “fine-tuned”, or to which someone is “putting the finishing touches”. The concept fits nicely with that of amplification in a new substrate.

This link between Elisabethville and the CHAT vaccine that had been perfected in Stanleyville is intriguing. Dr Stefan Pattyn, who headed the virus lab at Elisabethville in the fifties, has since stated to Stanley Plotkin that “certainly poliovaccine was never produced” in Elisabethville between 1955 and 1960.²⁰⁶ This, as Plotkin points out, is a clear denial, but it leaves unexplained why such a specific statement should have appeared in the local press in 1958, in an article which was apparently based on an interview with one of the Belgian doctors involved, and which was, in all other respects, extremely well-informed. It is worth noting that Pattyn appears to have been familiar with the work of Koprowski, Plotkin, Hayflick and Gelfand. An article he wrote in the early sixties on “Anti-poliomyelitic vaccination in tropical countries” focuses on the Koprowski strains, and on attempts that Koprowski and Hayflick had made to get away from the potential dangers of contaminated primate tissue cultures by adopting “a technique for culturing fibroblasts from human embryos”.²⁰⁷

Let us suppose for one moment that Dr Pattyn was not fully informed about all that happened in Elisabethville in the second half of the fifties, and that one of the many labs in that city *was* producing the Koprowski vaccines as reported in the article.²⁰⁸ In that case, which locally-available substrate would have been used?

During my various interviews with Belgian doctors who used to work in the Congo, the fact that the Elisabethville lab had used human cells rather than monkey cells for its virus research was mentioned quite often. This conclusion is supported by a paper which the two leading scientists from the Elisabethville lab, the director (Jean Delville) and the head of virology (Stefan Pattyn) delivered to the Stanleyville virus

symposium in September 1957, which explained that in their virus research in Elisabethville they had worked since 1954 with HeLa cells, which had considerable advantages over “monkey kidney cell cultures”.²⁰⁹ This account is expanded in a review by Pattyn of his own enterovirus research in the Congo, which was published in 1962. Here, Pattyn explains that he decided to concentrate on poliomyelitis research after he witnessed a polio epidemic in the savanna region of Upper Katanga. He continues: “It was my good fortune to work in a well-equipped laboratory where a centre for tissue culture was established. The HeLa cell line was used as a source of continuously proliferating cells, whereas human amnion cells were used as a source of freshly trypsinised cells, with broader susceptibility to polioviruses. Amnion cell tissue cultures could be produced on a large scale as our laboratory was near the maternity department where an average of 24 deliveries were carried out in 24 hours.”²¹⁰

The descriptions of the research makes mention only of virus isolation and diagnostic work, but if CHAT was indeed prepared by Wistar Institute scientists in that city in 1958, is it not possible, indeed likely, that they would also have used one of these human cultures as a vaccine substrate?

Pattyn’s various articles make it clear that he was actively engaged in polio research in the Congo throughout the fifties. For instance, it was he who coordinated the polio antibody studies in Leopoldville, Elisabethville and Bukavu – and who may have done the same in Astrida (now Butare, Rwanda). If the newspaper article is correct, then many will feel that it is Pattyn’s virology lab, where they used only human cells, that is the likeliest venue for CHAT vaccine (which had previously been perfected in Stanleyville) to be amplified for the Leopoldville trials.

It is my impression that Dr Pattyn may know more than he says about the various polio vaccines that were prepared and tested in the Belgian Congo. After all, it was he, in the mid-nineties, who once advised me that if I wanted to know more about the chimp research at Lindi, I ought to “ask Osterrieth. He was implicated in this whole thing.”²¹¹ Having said that, however, he declined to elaborate further. When I interviewed Dr Pattyn again in 2000, I was surprised by the change in this previously helpful man. On this occasion, he was hostile and defensive, and referred to me with heavy sarcasm as “the man who wants to be famous”. He had not read *The River*, but told me he thought that the OPV theory was “foolish”. When asked why, he said because there was a “lot of evidence” that AIDS had existed before 1959, but was unable to support this claim.

The second historically relevant point is that in July or August 1958, Dr Henry Gelfand, an American epidemiologist who was based at Tulane University in New Orleans, hand-carried the latest Koprowski strain (CHAT pool 13) from Brussels²¹² to Leopoldville. He also visited Stanleyville, Bukavu and Elisabethville, in order, as he put it, “to acquaint regional authorities about the vaccine, and its proposed use”. However, the precise purpose of his journey outside the capital has recently become a bone of contention, for Dr Gelfand’s accounts of these events (which in 1996 included his declaration that he “must have carried” polio vaccine to labs in the three other cities) have changed as the years have passed. In a letter which he wrote to Stanley Plotkin in 2000, Dr Gelfand claimed that it was “extremely unlikely” that he had

taken polio vaccine to the other three cities. He also claimed that this is what he had told me, which is untrue.²¹³

In a letter which he wrote me in 1996, Dr Gelfand closed as follows: “P.S. I forgot to mention that I went to B.C. [Belgian Congo] only as a consultant to Koprowski and the Wistar Institute”.²¹⁴

This postscript is interesting in the light of the 1958 newspaper report which records that the new Koprowski polio vaccine, which had previously been perfected in Stanleyville, “has been prepared at Elisabethville by the Wistar Institute”.

Let us suppose for a moment that, despite what Dr Pattyn says, a visiting scientist or consultant representing the Wistar Institute did prepare the CHAT vaccine that was used in the Leopoldville trials at one of the several Elisabethville labs. The scientist in question could possibly have been Dr Gelfand (who, despite officially being an epidemiologist, was also an acknowledged expert on polioviruses).²¹⁵ Or that scientist could have been someone else representing the Wistar, someone who had visited Elisabethville in the preceding weeks or months.

Because of Dr Gelfand’s ambivalent answers about what he actually did with the CHAT pool 13 vaccine, we are left to speculate about how the vaccine used in Leo might have been prepared in Elisabethville. Did Dr Gelfand, despite his recent protestations to the contrary, carry a bottle of pool 13 to Elisabethville, and amplify it there – perhaps in the virus lab which was the oldest, and almost certainly the best-equipped, in the Congo? Or had another doctor already brought some locally-prepared CHAT from Stanleyville to Elisabethville, in order to amplify it further? Or did Gelfand (and/or another Wistar representative) amplify both pools in Elisabethville – the newly-arrived pool 13, presumably made at the Wistar, and the older pool 10A-11, which had already been amplified in Stanleyville? Both the second and the third scenarios would theoretically allow a batch of CHAT vaccine made in chimp cells to be further passaged in human cells, which might (for instance) have been human amnion cells that had been overtaken by HeLa.

Henry Gelfand’s clarification of his official status in the letter he wrote me is also intriguing for another reason. If he was in the Congo “only as a consultant to Koprowski and the Wistar Institute”, then he was clearly representing neither Tulane University (which is the home of tropical disease research in the US), nor the CDC, where he moved soon after the Leopoldville trials to take over the enterovirus unit. This is further underlined in the paper which Gelfand, Plotkin, Koprowski, Courtois and two other Belgian doctors subsequently wrote about the Leopoldville trials, for a note on the title page states that: “the participation of Dr Gelfand does not necessarily imply endorsement of the studies by the Public Health Service.”²¹⁶

Another note on this paper makes a similar claim about Stanley Plotkin’s participation. And the next paper in the series, on which Plotkin was lead author, claims that “This work was done when Dr Plotkin visited Leopoldville in May 1959 while on leave from the Public Health Service, and the opinions expressed are those of the authors only.”²¹⁷ It is for the reader to decide whether these are *pro forma* disclaimers, or whether one is meant to take them seriously.

m) Sources of funding.

Some have questioned why it was that so much money was poured into medical research in the Belgian Congo in and around 1957, when it was already clear that independence was approaching fast. Large and impressive new medical laboratories were erected at Stanleyville, Bukavu, and Bunya, all of which featured virology departments. Furthermore, a dedicated virus lab was opened in Leopoldville. Since the colony was officially self-financing, and was famously short of cash, this has suggested to some observers that foreign funds might have been involved.

Whether or not the United States Public Health Service (PHS) endorsed Koprowski's research programme and vaccine trials in the Belgian Congo, what is certain is that that Service was supporting and at least partially bank-rolling the venture.²¹⁸ In *The River*, I detail several documented links. There was a PHS research grant [E-1799] which was cited in all the papers about Koprowski's polio research in Africa; an internal paper by Ghislain Courtois which clearly stated that the PHS was supporting the research, and was paying for Koprowski and members of his staff to visit Stanleyville annually for the next five years;²¹⁹ a published comment by Agnes Flack indicating that the Congo vaccinations, including the Ruzizi trial, represented a joint undertaking of the Belgian and American public health services; and the fact that Stanley Plotkin, who did much of the organisation and preparation not only for sections of the Congo trials, but also for those in Poland, Croatia, and the US, was apparently one of a team of "top epidemiologists" who were working for "the USPHS field post that is located in the Wistar".²²⁰ Indeed, despite the claim that Plotkin was on leave from the PHS while he was working in the Belgian Congo, a letter that he wrote to Fritz Deinhardt from the Congo in May, 1959 was typed on Public Health Service headed notepaper.²²¹ There are other examples as well, but these make the point.

And then there is the role of Karl Friedrich Meyer.

Here, a little additional history is needed. There were plans for a second stage of the Lindi project, which would have involved the United States and Belgium establishing a chimpanzee colony dedicated to medical research in the Congo, one which was intended to survive beyond independence. The venture had the backing of the Belgian king, and of the US Public Health Service.²²² Karl Meyer, the long-time director of the George Williams Hooper Foundation (an institute of medical research in San Francisco, financed by a bequest from a leading industrialist) led a team of four American scientists to the Congo in May 1960. They discussed possible locations for the new chimp camp, with an island near Lindi and the IRSAC²²³ establishment near Bukavu being the leading candidates. Koprowski was meant to be a member of the group, but finally did not attend; the Belgian representatives were Ghislain Courtois and A. Lafontaine, and there were two chimp specialists from IRSAC. In the end plans for the research centre fell through because of the collapse of the political situation so soon after independence.

However, there is another interesting clue from Lindi itself. In 1999, the local villagers unearthed what appeared to be a foundation stone at the camp, which was inscribed on all four sides. There was 1956 (the year of opening), N (for North), IGCL (unknown), and KF/003. Initially I thought that the latter might be a burst of egoism –

Koprowski Foundation Number 3, or something of that sort. Only much later on did I remember that Karl Friedrich Meyer of the George Williams Hooper Foundation was universally known as “KF” by his contemporaries.²²⁴ The nickname is even referred to in his obituaries.

In his Alvarenga Prize Lecture in 1959, Hilary Koprowski recounts the history of his ten year association with polio vaccines, and identifies a key moment. In January 1952, he had what he calls a “fateful meeting” in New York with K.F. Meyer, and with Joseph Smadel, whom he identifies as an associate director of the US Public Health Service. Apparently Koprowski sought advice from these two great men, and Smadel suggested that he and Meyer should establish a cooperative study. “This led to prolonged and fruitful collaboration” which lasted several years, explains Koprowski, and “the results...of the investigations were very gratifying”. In 1952, and again in 1955, Meyer helped Koprowski set up trials of his OPVs in Sonoma, a facility for the developmentally disabled just north of San Francisco. Thus the setting up of Lindi camp in 1956 appears to have been the third aspect of the Meyer/Koprowski collaboration, KF/003.

But there may be a little more to that “fateful meeting” than meets the eye. In 1952, Jo Smadel was not yet with the PHS. In those days, he was head of the department of viral and rickettsial diseases at Walter Reed Army Medical Service Graduate School (later known as the Armed Forces Institute of Pathology) in Washington DC, and a renowned (and sometimes feared) organiser and power-broker in the worlds of medicine and military medicine. He played a key role in several commissions of the Armed Forces Epidemiological Board (AFEB), and was one of the scientists who helped coordinate America’s biological warfare programme. In the latter role, he helped to organise investigations into the potential dangers from, and uses of, different pathogens, notably rickettsial diseases and arboviruses (arthropod-borne viruses).

Karl Meyer meanwhile, was a hero of public health, having developed several different human vaccines, as well as a technique for eliminating botulism which opened the way for the canning of food. Some of the diseases which these vaccines protected against (such as psittacosis, pneumonic plague and brucellosis) were rather rare in the United States, but they were described as “diseases of military importance”, in that they were viewed as constituting a potential threat to the armed forces of the United States. Much of Karl Meyer’s work, therefore, involved developing vaccines for the troops, vaccines which would protect them against new or little-known diseases when travelling into tropical areas, or alternatively when they were exposed (from whichever quarter) to biowarfare agents. There was, in short, an ongoing collaboration between Smadel and Meyer: the military medic, and the civilian researcher who developed vaccines against some of the same diseases.

A good example of this type of collaboration relates to 1954, and gives some sense of the eminent American scientists who were at least knowledgeable about (if not indirectly involved with) biowarfare (BW) research during the Cold War years. In that year a Dr Devignat, who was director of the “Ecole A.M.I.”²²⁵ at Elisabethville in the Congo, wrote to Joshua Lederberg, offering to dispatch three strains of pneumonic plague – one highly virulent, one which had been attenuated in the lab and which could perhaps serve as a vaccine, and one intermediary strain, which possibly showed

evidence of recombination. Lederberg immediately contacted Dr Ellis Englesberg, one of Karl Meyer's plague experts at the George Williams Hooper Foundation, and Dr Werner Braun, a significant figure at what was then called Camp Detrick, and which later became Fort Detrick [see below]. Professor Lederberg wrote back to Dr Devignat to say that Englesberg was otherwise engaged, but that Dr Braun (not to be confused with rocket scientist Wernher Von Braun) "would be interested in the question of genetic recombination in this species", and would like to collaborate.²²⁶

So, doctors Smadel and Meyer were the men from whom Koprowski sought counsel in 1952, and with whom he established a long and fruitful collaboration. And those two initials, KF, sat quietly on the foundation stone beneath Camp Lindi, the experimentation centre of the *Mission Courtois/Koprowski*, from 1956 until they were uncovered again, forty-three years later.

n) Events at the Wistar Institute.

At this point, we need to turn back a few years, and examine what was happening at the Wistar Institute before Hilary Koprowski was appointed director in 1957. The Institute had officially been without a director for the previous nineteen years.²²⁷ However, it had not been asleep.

In 1952, it came under the wing of the new vice-president for medical affairs of the University of Pennsylvania, Professor Norman Topping. An ex-navy man, and an expert on rickettsial diseases, Topping came from the milieu of military medicine which had taken control of American public health during the years of the second world war, and which had, among other things, responded to the threat posed by the Japanese biological warfare programme. Topping had also helped oversee the process whereby crucial fields such as viral and rickettsial research were officially returned to the civilian fold after the end of that war. Before arriving in Philadelphia, Topping had been the first associate director of the National Institutes of Health, serving from 1948 to 1952. Like Smadel and Meyer, he was a man of substantial influence and power, and one who tended to dispense it quietly, behind the scenes.

Topping knew Lederle Laboratories well, for under Herald Cox, the viral and rickettsial division had prepared vaccines against several of the rickettsial diseases which were felt to pose threats to American troops. These included Topping's own speciality, Rocky Mountain Spotted Fever. And so it was that Topping got to know Cox's deputy, Hilary Koprowski, who had spent the years from 1944 to 1948 conducting research into a wide variety of arboviruses, and had then shifted his attention to two of the great neurotropic viruses: rabies and poliomyelitis. By this stage, Koprowski was probably known as a determined and ambitious man, and also as one who was adept at modifying viruses by growing them in different cell cultures.

In his autobiographical memoir, Topping explains that when he took over the running of medical affairs at the University of Pennsylvania in Philadelphia in 1952, his first job was to reinvigorate the Wistar Institute, which, like the G.W. Hooper Foundation at UCSF, was an independently-funded biomedical research organisation situated at the heart of the campus. In order to do this, he recruited Hilary Koprowski. According to the account given by Dr Topping, it seems that Koprowski must have been brought on board between 1952 and 1954.²²⁸

This may seem strange to those who know that Koprowski was not formally appointed director of the Wistar until May 1957. However, it appears that although he continued to work at Lederle in the intervening years, he may have been wearing two hats. There are at least four separate instances in which witnesses have apparently seen Koprowski at the Wistar, or have had contact with him via the Wistar, in the years before 1957. One example involves Joshua Lederberg, who Koprowski tried to sign up in October 1956, presumably once he had been given the green light to recruit his own team.²²⁹ But there are others from long before that. The doctor who was in day-to-day charge of Koprowski's second polio trial at Sonoma, between April and July 1955, has repeatedly stated that his cheques were paid not by Lederle, but by the Wistar. And Dr Andrew Hunt, who played a similar role at the Koprowski vaccine trials in Clinton, New Jersey, from October 1955 onwards, recalls that Koprowski's links with the University of Pennsylvania (and in particular with Joseph Stokes, who also worked at CHOP) began in 1953 or 1954. He also, strangely, recalls a meeting with Koprowski in 1955 which took place at a business school adjoining the Wistar.

It may well be that Koprowski played only a behind-the-scenes role at the Wistar Institute in the years before 1957. But there are indications that, whether organised by Topping, Koprowski or both, substantial funding started rolling in during this period. The Institute was now publishing the proceedings of the quasi-annual symposia held by the Division of Biology and Medicine of the Atomic Energy Commission (headed by Shields Warren), and by the Biology Division of the Oak Ridge National Laboratory. Several of these AEC meetings dealt with cutting-edge subjects such as "The effects of radiation and other deleterious agents on embryonic development", and "Genetic recombination", and some were co-sponsored by the Biology Council of the National Academy of Sciences-National Research Council. In addition, there are indications that the AEC may have provided further funding, perhaps *sub rosa*, to the Wistar during the fifties. It is worth noting that before she became medical director of Clinton prison in 1953, Agnes Flack had apparently worked for eight years at Union Carbide, the main contractor at Oak Ridge, where she was engaged in "medical research at the...atomic center".²³⁰

Soon after he brought Koprowski on board, Norman Topping recruited two other luminaries for the Institute. "Each was recruited without a committee", writes Topping; "I've never believed in committees." The first was Geoffrey Rake, a famous microbiologist from the Rockefeller Institute and Squibb Institute, and the second William McLimans, who had helped build up the virus and rickettsial lab at the Navy Research Institute in Bethesda.

In 1953 or 1954, Rake and McLimans started assembling a team to concentrate on the semi-industrial production of viruses and rickettsia, most notably the polioviruses, at the Wistar. The young Leonard Hayflick was probably a member of this team. They grew the viruses in sealed containers of between five and twenty litres capacity, and the work was conducted as part of a programme called "Microbiology in Medicine", set up jointly between the Wistar Institute and the University of Pennsylvania's School of Veterinary Medicine, which had just opened an annexe at the New Bolton Center. (This is the same NBC where, two decades later, researchers would report inducing leukemia and *Pneumocystis carinii* pneumonia, PCP, in two chimpanzees by feeding them milk from cows infected with a retrovirus, Bovine Leukemia Virus.)²³¹

Almost all of the Microbiology in Medicine studies of mass production of viruses in different substrates were funded by the US Army Chemical Corps at Fort Detrick, Frederick, Maryland. Since the Second World War, Fort Detrick had been America's major centre of biological warfare research, both defensive and offensive.

One such Fort Detrick/Wistar "Microbiology in Medicine" collaboration was a study of anthrax in industrial settings, of which Dr Philip Brachman was one of the co-authors.²³² As an apparent follow-up to this study, in May 1957, just as Dr Koprowski formally took over as director, a Fort Detrick anthrax vaccine was tried out at one of the industrial plants in question: a wool mill in Manchester, New Hampshire. Three months later, there was a sudden outbreak of inhalation anthrax at the mill, the first and only such epidemic outbreak in the USA in the twentieth century – although nowadays, sadly, we are more familiar with such events. Four of the mill workers died, and a two-man team from the PHS's Epidemic Intelligence Service field post at the Wistar was sent in to investigate. Its members were Philip Brachman and the young Stanley Plotkin.²³³

There appeared to be two remarkable coincidences. The first was that an unprecedented outbreak of inhalation anthrax had followed so soon after a vaccination against that very disease. The second was that a new commercial detergent of a type which was known to increase the virulence of inhaled anthrax spores by an order of magnitude²³⁴ had been introduced to certain departments at the mill (to replace the soap and soda ash which had previously been used to clean the goat hair) on the same morning that the first patient fell sick. Despite this, the Wistar investigators concluded that there was no provable link between the two events (because the first patient had not worked in one of those departments where the detergent was being used), and then declared the anthrax vaccine a success, with an "effectiveness of...92.5 per cent".²³⁵ (Others have since disputed their analysis.)²³⁶

A few months later, in June 1958, doctors Brachman and Plotkin attended a meeting of a medical advisory committee at the Fort Detrick Biological Warfare Laboratories (BWL), and Brachman reported in detail about the anthrax vaccine. There were 56 scientists present, of whom only seven appear to have been civilians.²³⁷ Certain information relevant to the Manchester tragedy appears to have been withheld from the minutes of the meeting, such as (it would seem) certain details concerning sampling by Fort Detrick scientists which took place at the mill six months after the outbreak, in February 1958. During the discussion that followed, it was revealed that the unusually virulent pathogen which had caused the Manchester anthrax outbreak was "about as virulent" as the highly virulent anthrax strain then being used at the Fort Detrick BWL. (This whole episode strikes an eerie historical echo, in the light of the recent revelations that the anthrax sent though the US mails in 2001, with such devastating consequences for several persons, apparently had an "identical" genetic sequence to a strain that was developed at a laboratory at Ames, Iowa, and weaponised at Fort Detrick.)²³⁸

Immediately after this discussion of the Manchester incident, the assembled doctors (with Plotkin, at least, still in attendance) began discussing the stability, mass production, stockpiling and aerosolisation of "Agent N". This is the military term for weaponised anthrax. Some might feel that the presence of the two Wistar doctors at

such a meeting at the Biological Warfare Laboratories raises issues about potential conflicts of interest.

Virtually the same anthrax vaccine is still in use in the US today. Apparently even now, in semi-retirement, Philip Brachman and Stanley Plotkin continue to be regular participants at committee meetings at which the safety, efficacy and production of the U.S. anthrax vaccine are discussed and assessed.

This gives some idea of the type of work which the Wistar Institute was undertaking in the 1950s, both during the early years when Koprowski was only informally involved with that institute, and after May 1957, when he officially took over as director.

But let us return to the other Microbiology in Medicine studies published by Wistar scientists. The first study, involving poliovirus production in HeLa cells and published in 1956, was funded by the National Foundation for Infantile Paralysis.²³⁹ But the later studies, which included investigations of virus growth in L cells (a cell line derived from a mouse), FL (human amnion cells), Chang's conjunctival cells (a human cell line), and ERK-1 cells (the cell line derived from rabbit embryos), were funded by Fort Detrick.²⁴⁰

It is not certainly known why Fort Detrick was involved with such investigations, but one of the final Microbiology in Medicine papers explains that this research "permits one to contemplate the production of viral vaccines, hormones and other physiological agents by methods analogous to techniques employed in microbiological fermentations".²⁴¹

It has been suggested that the Fort Detrick and Wistar scientists may have been interested in the potential for the rapid production of vaccines against specific diseases during a time of national emergency, as, for instance, a BW attack. But of course the viruses mass-produced as "discrete units" (in other words, in sealed containers) did not necessarily have to be attenuated ones. Among the other viruses being studied were human adenovirus 4 (in FL cells), herpes simplex virus, and Venezuelan equine encephalitis virus (VEEV) – the latter two being grown in L cells, the mouse cell line.

The presence of VEEV on this list is significant, because this is not a virus which one would normally expect to encounter outside the laboratory, or unless one was wandering through central or south America. It is, however, considered "a disease of importance to the military". The history of scientific research into VEEV is rather interesting. This was the virus with which Koprowski first made his name in 1943, while he was working at a Rockefeller Foundation-funded lab in Rio de Janeiro, Brazil. While engaged on a contact experiment with VEEV-infected mice, he discovered in the most vivid fashion that the virus was capable of infecting humans. In the course of the experiment, he and several other workers in the lab became infected, and were incapacitated for several days with blinding headaches and fevers.²⁴² By the fifties, the U.S. army had prepared VEEV strains of varying strengths, and developed a vaccine against the virus, which promptly became one of the favoured weapons in America's biological warfare arsenal.²⁴³

The first Microbiology in Medicine study, involving the production of poliovirus in HeLa cells, was based on MEF-1, a strain which several virologists (including Koprowski and colleagues at Lederle) had modified to create their Type 2 polio vaccines. Of course, the use of HeLa cells for human vaccine production was already acknowledged to represent an unacceptable risk. But presumably nobody realised the extent to which HeLa had already taken over the world's virus labs. According to Walter Nelson-Rees, ERK-1, FL, and Chang's conjunctival cells had almost certainly been taken over by HeLa by this stage. Even though there are some indications that, as early as 1956, some scientists were beginning to twig that HeLa contamination of other cell lines might be occurring,²⁴⁴ there is no reason to believe that anyone at the Wistar had been entertaining such suspicions.

In July 1957, just after Koprowski's formal taking over as Wistar director, he and William McLimans attended the Fourth International Poliomyelitis Conference in Geneva. Here, Koprowski made his first public announcement about his new polio vaccines, CHAT and Fox.²⁴⁵

Some of his listeners may have been surprised that Koprowski was speaking about vaccines which must have been largely developed at Lederle, where he had been working until ten weeks previously, and that Lederle had apparently accepted the departure of those vaccines without comment – or at least without legal intervention. However, the *sub rosa* relationship he enjoyed with figures such as Topping and Smadel might explain a great deal. One source from Lederle has told me that Koprowski acted as a law unto himself during his final two years there, taking virus strains from the lab without permission, and engaging in “under-the-table dealings”.²⁴⁶

Despite this, it seems possible that Koprowski's departure may not have come as a complete surprise to the Lederle management. The suggestion that Lederle may have retained an interest in Koprowski's polio vaccine research even after May 1957 is also supported by further clues, such as the fact that it was apparently a Lederle car which took George Jervis to Idlewild airport when he set off for the Ruzizi Valley trials in February 1958.²⁴⁷ This is despite the fact that Dr Jervis worked at the Letchworth Village facility for developmentally disabled kids, and had no formal links with Lederle.

There is no doubt that Lederle was where the CHAT and Fox vaccine strains were developed,²⁴⁸ and this is where Koprowski had been misreporting the substrate he had been using to grow his polio vaccines during 1956 and 1957.²⁴⁹ Meanwhile, however, he had a clandestine relationship with the Wistar, where research work was clearly focussing on the different tissue cultures and cell lines in which vaccines could be grown.

In other words, the vaccine research on CHAT and Fox was done at Lederle, but it seems that the vaccine substrate research may have been carried out at the Wistar.

But back to the Geneva conference. On the afternoon of the day that Hilary Koprowski made his first announcement about CHAT and Fox, William McLimans got up to sing the praises of ERK-1, the embryonic rabbit kidney cell line, and made his comment about taking the monkey business out of vaccine production.

He may have been a little over-eager. In the paper which described the original development of ERK-1, written by J.C.N. Westwood from the Microbiological Research Establishment at Porton Down (Britain's equivalent of Fort Detrick) and published in early 1957, the author emphasised caution.²⁵⁰ "In our experience the transformation [of ERK-1 and other cell lines] has occurred with sufficient frequency to suggest that it may be expected in a large proportion of cell lines derived from normal tissues and, in view of the growing importance of serial subcultivation, it is imperative that a fuller understanding of its true nature be obtained", he wrote. Westwood went on to note the similarity of ERK-1 (and five other cell lines developed at Porton) to HeLa, and began the discussion section as follows: "*The possibility of using cancer cells for the production of virus vaccines for human use raises issues as much of a political as of a medical nature, and the political issues are less susceptible to the results of experimental investigation...*" [My italics]

However, in the very next article in the same journal, two scientists from the Bacteriology department at University College Hospital Medical School in London describe the ease with which ERK-1 can produce polioviruses.²⁵¹ Again they emphasised that further investigations were needed before virus grown in ERK-1 could be used for immunising humans, but now at Geneva, just a few months later, McLimans from the Wistar was actively promoting the making of human polio vaccines in this substrate.

This seems a good example of the way in which the science from one article may be inoculated into a second, leaving only the sensible caution behind. Sometimes, however, one suspects that both groups of scientists may have been party to the process, as an effective way of moving a debate forward, especially one that occupies politically sensitive areas.

It is not known, of course, if CHAT and Fox were ever grown in ERK-1 (or any other HeLa-contaminated substrate) in the Congo. It is worth noting, however, that the necessary materials for serial propagation of such lines were available, since all the major Congolese labs had rabbits in their *animaliers*, and nearby maternity wards where amniotic cells were in good supply. Given this availability, combined with the background history, it does not seem absurd to propose that Koprowski's vaccines may have been tried out in some of these substrates.

This is the first time in the course of this ten year investigation that I personally have believed that there is substantial evidence to support the scenario that human cells (as well as chimpanzee cells) may have been used to grow some of the polio vaccines that were being field-tested in Belgium's African colonies in the 1956-60 period.

But it must be emphasised that there is a difference in the quality of evidence for the two substrates. Whereas there is documentary evidence that chimpanzee cells were present in Osterrieth's lab in February to April 1958, at the time when we have a convincing first-hand account of his "making polio vaccine", the links between HeLa and CHAT vaccine are more tenuous. They depend on an August 1958 newspaper article which states that CHAT vaccine had been prepared in Elisabethville, which was written at a time when both HeLa and human amnion cells (some of which may have been in the form of HeLa-transformed cell lines) were present in that city, but

when primate cells were apparently not. (Both the principal doctors from the Elisabethville medical lab during the fifties, Jean Delville and Stefan Pattyn, used to stress that they found HeLa and similar stable cell lines far more reliable than simian cultures.)²⁵²

It is important to stress that there is no direct evidence that vaccine made in chimp cells was later amplified in HeLa, or cell lines taken over by HeLa. However, according to Hayflick's detailed accounts, this would represent a precise parallel to what happened when he first tested his human amnion cell line, WISH, and his first human diploid cell strain, WI-1, for susceptibility to polioviruses. In both instances, Hayflick describes taking CHAT and Fox vaccines which had previously been prepared in "monkey kidney tissue culture" and growing them up in the new human substrates.²⁵³ If similar experimentation had taken place in the Congo, then vaccines made in chimp kidney tissue culture would have been grown up in substrates such as WISH and WI-1 (both of which appear to have been developed after August 1958), or in other cell lines like FL, ERK-1 and "Fernandes" (which appear to have been developed in 1956 and 1957). FL and ERK-1 were being investigated by the Rake/MacLimans team at the Wistar by 1956, while "Fernandes" (which was originally called a "cell strain", though it bore close similarities to both HeLa and WISH) was developed by Mario Fernandes, a Portuguese scientist trained in Lisbon who collaborated with Hayflick while he was at Galveston, Texas in 1956-7, and who later, together with his cell line namesake, followed him to the Wistar.²⁵⁴

Given what generally happened when polio vaccine ran out, it does not seem implausible to propose that CHAT vaccine from one lab (such as Stanleyville) might have been sent to another (such as Elisabethville), and then amplified in a different cell substrate. This might have happened occasionally, to take care of a shortfall, or it might have happened several times. But because, as Plotkin has revealed, CHAT vaccine was routinely prepared from previous batches of vaccine (rather than from seed virus), such an event only needed to happen the once, from the perspective of viral contamination, for the entire output of the second lab to be compromised. And because of the nature of the virus, it is unlikely that, back in the fifties, even careful observation of the cultures would have led to the recognition of SIV contamination.

If the scenario of further CHAT passage in human cells has substance, then this would introduce an important new element to the OPV theory. I believe that this historically-supported possibility demands further investigation.

o) The notebooks.

But is it ever going to be possible to know for sure what work was being conducted by Dr Koprowski and his colleagues in Stanleyville and elsewhere? Well, as it happens, there is one way of gathering more information. It appears that Hilary Koprowski's and Tom Norton's lab notebooks from 1950 to 1957 are still held at the facility that used to be Lederle Laboratories, on microfiche. I have a list of the numbered notebooks, and copies of certain pages from them. These reveal, for instance, that Koprowski was already plaquing out the Charlton strain in July 1956, the month after Lindi camp opened. (Charlton was the original name for CHAT, which was based on Charlton plaque 20, which we know was tested intraspinally on five chimps – presumably from Lindi.)

In January, 2000, I wrote to Professor Patrick Gage, who was heading the research and development arm for Lederle's then-owners, and I requested that I should be allowed to view the microfiches in the company of a Lederle scientist. A detailed letter of support (probably the last professional letter he ever wrote) had been prepared by Bill Hamilton, and I enclosed that with mine.

Two months later, Dr Gage wrote back to refuse my request, explaining that the notebooks contained "highly confidential and proprietary information", and that it was the company's policy to "protect against disclosure of confidential proprietary information and the potential loss of valuable intellectual property rights".²⁵⁵ He added that he had "assigned a team of scientists experienced in virology and vaccine development" to review the relevant notebooks, and explained to me that they did "not contain any evidence that Lederle used chimpanzee kidney tissue as a substrate for the development of any oral polio vaccine".

Given what is now known about the making of chimpanzee kidney cultures *in Stanleyville*, that may well be the case. However, the notebooks do make it clear that among the substrates being used in the lab were "Detroit 6" (a human cell line, later revealed to have been taken over by HeLa), together with "human lung" and "human kidney". They also reveal that embryos were arriving in the lab, presumably for making tissue culture.

Later in 2000, I again wrote to Dr Gage, asking him to reconsider. Once again he declined.²⁵⁶ I am unable to see what commercial secrets might require protection fifty years on. Furthermore, I am reliably informed that the review of the microfiches was in fact carried out by a team of lawyers.

I believe that one of the more obvious reasons for my request being refused might be that the notebooks contain information which would lend support to the OPV/AIDS theory of origin. However, if the vaccine strains were developed at Lederle, and the vaccine substrates at the Wistar – as now appears to be the case, then there should be nothing in the notebooks which needs to be concealed.

I am therefore calling publicly on Wyeth, the new owners of Lederle Laboratories, to reverse the previous decision by Dr Gage, and to invite me (together with one of their own scientists, and perhaps one other party, who might serve as a "neutral observer") to view these microfiches, together with the report submitted by the team of lawyers. A continued refusal to grant access to these materials might well lead people to the unfortunate conclusion that Wyeth has something to hide about this period of research.

p) "Weisswash".

One last point: after the Lincei meeting, Dr Weiss told me that he thought it was not fair for me to "accuse" Dr Osterrieth when he was not present. Dr Osterrieth has now had six separate opportunities, and more than eight years, in which to clarify what happened at Stanleyville lab, and at Lindi camp.²⁵⁷ Despite this, Dr Osterrieth's accounts still contain major gaps and contradictions.

I suspect that what Dr Weiss really means is something rather different. When he himself suggests, on an entirely theoretical basis, that local OPV production in chimpanzee cells might have happened in Africa, as he has now done on several occasions, then this, apparently, is acceptable.²⁵⁸ But when I go out to Kisangani (without, I should stress, a “prior agenda”, not least because I didn’t really believe that Weiss was right about local vaccine production), and I return with information indicating that this is precisely what did happen, then Dr Weiss changes his tune.

According to him, I am now being unfair. Why? Because “Dr Osterrieth has categorically stated...that chimp tissues were not used.” What this argument seems to boil down to is that one should not embarrass those of whom Dr Weiss approves. And he certainly does approve of Dr Osterrieth, having referred to his Royal Society speech as both “elegant” and “resonant”.²⁵⁹ (It may well have been both, of course, without being accurate.)

The bottom line, it would appear, for Dr Weiss is that whereas a theoretical and non-specific argument is acceptable, one which spells out a detailed scenario and which “names names” is not.

Whatever Dr Weiss may think or say, this isn’t a witch-hunt. This is, and always has been, an attempt to get to the bottom of a hugely important issue.

Scientists, like those from most other walks of life, tend to shy away from the whiff of scandal – and there is always, of course, a natural tendency for the establishment to try to defend its own. However, there is now substantial evidence (some of which will be presented later in this paper) that several eminent members of this profession have participated in an attempted cover-up.

Indeed, Professor Weiss himself does have something of a reputation for defending establishment views (at least in public) over the years. Back in 1985, when the first story questioning the role of Robert Gallo in the “discovery of the AIDS virus” debacle broke in *New Scientist*, Dr Weiss wrote in with a letter stating that the article “was the nastiest piece of writing I have seen in twenty years of studying retroviruses”. Later, Abraham Karpas commented that the story in question might have revealed a “‘Gallogate’, in spite of a *Weisswash*”.²⁶⁰

This is most certainly not the right time for a *Weisswash*, for it is no longer possible to brush the less comfortable aspects of this debate under the carpet. If these issues are dismissed unfairly and unscientifically by a scientific establishment which has merely gone through the motions of proper investigation, then they will keep returning (not least because there is more of this story which remains to be told). If that does happen, then those who have initiated, or contributed to, the process will bear a heavy moral responsibility.

For many reasons it is imperative, I believe, that what began as an open debate should continue to be so, and should be played out to its natural conclusion, however enormous the potential implications may be in terms of finance, ethics and – indeed – the future conduct of science.

4. The scientific debate: could a chimp-based vaccine have sparked AIDS?

In this section I shall review the latest developments in the scientific debate about how AIDS started, and in the process will respond, one by one, to the various scientific arguments which have been put forward over the last three years, and which, it is claimed, have “seriously weakened”, “disproved”, or “destroyed” the OPV theory.

The first two responses below relate to scientific evidence about whether or not CHAT vaccine batches were made in chimpanzee cells. The remaining responses relate to those scientific arguments which come into play if CHAT, in any shape or form, *did* incorporate chimpanzee cells.

a) The testing of the Wistar vaccines.

In the year 2000, samples of CHAT vaccine which had been released by the Wistar Institute were tested, and found to contain the DNA of macaques from Asia, but not that of chimpanzees. They were also found not to contain either HIV or SIV. The results of such testing, it has been claimed, have prompted the OPV theory to “die its final death”.

In fact, they have done nothing of the sort. As far as is known, none of the vaccine samples in question were prepared for use in Africa, and it is now becoming increasingly evident that most, if not all, of the CHAT vaccine that was sent abroad was passaged again in locally-made tissue culture before being fed to humans.

Some have complained that I was among the most vociferous of those calling for the testing of the samples, and yet now it is done I am still not satisfied. Certainly I called for the samples to be tested – the same samples which the Wistar initially offered (but failed) to have tested back in 1992. And I welcomed the fact that some testing was finally carried out, albeit eight years later. However, I never suggested that such tests would be definitive.²⁶¹

Neither, I thought, did anyone else who knew anything about the background. For instance in February 2000, Simon Wain-Hobson told me that no serious scientist was going to believe that the testing of the Wistar samples would provide proof of the theory either way. Yet apparently he was wrong. Just seven months later, after the Royal Society meeting, Robin Weiss was saying this with regard to the Wistar testing: “I think it was worth doing...I’m slightly surprised Hooper pooh-poohs it now.”²⁶²

At the London meeting, both before and after the announcement of the Wistar results, I continued to stress what I had already been stressing in *The River*: that the CHAT *pool* numbers were not of any intrinsic significance, in that it was now clear that different *batches* of these pools had been prepared in different labs and in different substrates.²⁶³ (A “pool” of vaccine virus indicates all the material produced at a specific passage level, while a “batch” indicates a specific production run of material prepared from a vaccine pool.) CHAT pools 10A-11 and 13 had indeed both been used in Africa, but what really mattered was where and how the specific batches of those pools that were fed in Africa were made.²⁶⁴

It seemed to me that this point had been made clearly enough. And yet, in Robin Weiss's dismissal of the OPV/AIDS theory in his *Nature* commentary in April 2001,²⁶⁵ he referred to 10A-11 and 13 as "batches", and by so doing, sowed confusion around the central issue pertaining to the legitimacy of the CHAT testing. Professor Weiss is not a careless man, and so it was surprising that he had made such a careless mistake.

In the light of the new evidence which indicates that in the late fifties batches of CHAT were made locally in Stanleyville (and perhaps elsewhere in the Belgian Congo too), it is clear that the samples which need to be tested are those which were prepared in the Congo (if any still exist), rather than those from the Wistar.²⁶⁶ I thank the Wistar Institute for arranging for the testing of some of their vaccine samples, but in terms of proving whether or not CHAT vaccine was produced in chimp cells, this testing has (it is now revealed) been of rather limited relevance.

As I write this, in mid-2002, Simon Wain-Hobson has just had yet another paper on the testing of the Wistar vaccines published, this time in the prestigious *Proceedings of the National Academy of Sciences*.²⁶⁷ Papers which appear in the *Proceedings* have to be "communicated" by a member of the NAS, and Wain-Hobson's paper was communicated by Hilary Koprowski. Wain-Hobson claimed that his finding that the CHAT samples released by the Wistar had contained only macaque DNA had "effectively scotch[ed] the [OPV] hypothesis". In a statement quoted by *USA Today*, he amplified his conclusion. "This issue is resolved", he said. "The vaccine lots were made using macaque kidney samples, not chimp, and we know that macaques are not infected by any virus of this ilk."²⁶⁸

We also know (as here the "we" includes Simon, for he sent me a rather brisk e-mail about it)²⁶⁹ that the vaccines that are likely to have been SIV-infected are those that were made in the Congo, not at the Wistar. His testing of the Wistar vaccines has therefore not resolved any issue, and his public claim that it has done raises issues of its own – such as whether he and Robin Weiss are involved in a genuine scientific debate, or in an attempt to persuade the world of a certain version of events.

b) "A totally absurd substrate"?

In a 1994 letter to me, Stanley Plotkin wrote that chimp cells "would have been a totally absurd substrate for a vaccine, considering the difficulty, the expense and the rarity of the species".²⁷⁰

This is simply untrue. In fact, most of the evidence suggests the opposite.

To demonstrate this, I shall examine the rather wide range of arguments which has been advanced by Dr Plotkin and others, as to why chimp cells might have been a poor, an inappropriate, or an "absurd" substrate for an oral polio vaccine, in order to see which, if any, are persuasive.

- If Plotkin is suggesting that chimpanzees were too expensive to use, then he is wrong. According to the chief government vet in Stanleyville at the time that Lindi camp opened, Joseph Mortelmans, chimps could be obtained from African

sources for between one and ten U.S. dollars a time during the 1956-1960 period.²⁷¹

- If Plotkin is suggesting that chimps were physically too difficult to handle, then again he is wrong, for the history of Lindi camp has tragically proved how very easy it was to experiment upon, and sacrifice, many hundreds of young chimpanzees and bonobos.
- If he is suggesting they were too rare, then I am surprised. In 1959, Plotkin's Belgian collaborator, Ghislain Courtois, stated publicly that 2,000 litres of OPV would be enough to immunise the whole world against polio,²⁷² which (given the fifties rule-of-thumb that two litres of vaccine could be produced from one primate) would have required the sacrifice of some 1,000 primates. The fact that some 600 chimpanzees and bonobos were sacrificed at Lindi camp in three-and-a-half years gives a fairly clear idea of how Plotkin and his colleagues would have viewed the rarity issue back in the fifties.
- If Plotkin is suggesting that substrates other than rhesus macaque kidneys would not have been considered acceptable, then he is wrong. According to the recommendations of the second and third WHO expert committees on poliomyelitis, which sat in 1957 and 1960, any suitable species of primate could provide cells for a polio vaccine substrate, whether that vaccine be oral or inactivated.²⁷³
- If Plotkin means that Koprowski would never have used an unfamiliar, or poorly characterised, substrate, then this would appear to be a somewhat stronger argument. However, the Koprowski team *was* familiar with chimpanzees. Koprowski and Tom Norton, together with George Jervis (who ran his own lab at Letchworth Village in upstate New York, where he had almost complete freedom), had been doing polio research with chimpanzees since 1949, utilising at least 16 chimps during the period up to 1952,²⁷⁴ and others again in the period up to 1956, when Lindi camp opened in the Congo. It seems probable that most (if not all) of these chimps were eventually sacrificed, and it is not unreasonable to imagine that after they had been killed, the potential uses of all available organs (such as kidneys) would have been investigated. I recently spoke with the respected virologist Robert Hull (who apart from producing inactivated polio vaccine for Eli Lilly during the fifties, was also perhaps the foremost world authority on the identification of adventitious viruses in simian tissue culture during that decade). In the course of the conversation he quite casually mentioned that his lab had also had chimps, and that at one point (perhaps, he said later, around the time that India restricted the export of rhesus monkeys – which was in 1955), they had prepared some experimental tissue cultures from their kidneys, simply because they were available. The cultures were found to be good for growing poliovirus, but the work was not considered important enough to be reported in the scientific literature. These cultures were apparently not used for polio vaccine preparation, but the point was that they *could have been*. It is not unreasonable to propose that a similar process may have occurred with Koprowski's team. Dr Hull, by the way, was shocked to learn that the latter team had been amplifying vaccine direct from vaccine (as has been acknowledged by Dr Plotkin), rather than culturing it from virus seed. "It doesn't make sense to me", he commented. "It would increase your chance of getting an adventitious virus."
- With regard to the previous point, we know from scattered comments in the medical literature (mainly from discussion sessions at different conferences) that

sera from the Lindi chimps had, at least by 1958, been tested for major pathogens like simian B, tuberculosis, Coxsackie B, measles, mumps and influenza by Koprowski, Courtois, Deinhardt and Werner Henle.²⁷⁵ Later, tests for different arboviruses were made by Dr Osterrieth,²⁷⁶ and it is certainly possible that tests were made for other pathogens as well. All this suggests that chimpanzees may in fact have represented a well-characterised substrate to Dr Koprowski and his collaborators. Of course, it was not possible in the fifties to test for then-unknown pathogens, like SIV.

- During my second interview with Gaston Ninane in 1993, he briefly suggested that Dr Koprowski might have been using chimps to make some of his vaccines in the U.S. Thereafter, he declined to elaborate, so there is no way of knowing if he was speaking of a matter which he knew something about, or was merely hypothesising. However, the comment is interesting, given what is now known about the making of chimpanzee kidney tissue culture (CKTC) in Stanleyville. It may of course be that, before that initiative was taken, some experimental batches of CHAT in CKTC were prepared at the Wistar, to check its various qualities. To quote one of Koprowski's former colleagues, who wished to remain anonymous, on the subject of the testing of CHAT samples from the Wistar Institute freezers: "I think that Hilary would have been most eager to have the testing done.... I can see any number of reasons why you would not detect SIV...Let's say that they used 20 kidneys from [primates] that would not produce [an HIV-1-like] SIV, and two kidneys that would produce SIV...." He left the rest of the sentence to my imagination.
- Alternatively, if Plotkin is suggesting that chimpanzees were too close to humans for their cells to be considered safe as a substrate for OPV, then this concept is hardly borne out by the historical evidence. Throughout the fifties, the vaccine developers were rejecting substrates like chick embryo, which were distant from human cells, and which grew poliovirus very poorly (if at all), and were turning instead to primate kidney cells. The best substrate of all, it seems, would have been human cells, but human cell lines such as HeLa were of course deemed too dangerous. Hayflick appears to have developed the first human diploid cell strains in about 1959,²⁷⁷ but in the three or four years preceding, cells from Man's closest relatives (chimps and bonobos) might well have looked like the ideal alternative. In several contemporary articles, bonobos are referred to by members of Koprowski's team as the "closest blood relatives to man".²⁷⁸
- In support of the foregoing analysis is a volume entitled "Experimental medicine and surgery in primates", which was published in 1969 by the society of which Dr Koprowski used to be president, the *New York Academy of Sciences*. The opening paper, entitled "The use of primates in biomedical studies; a review of suitable species" was written by the assistant director of the Yerkes Regional Primate Center, Dr W.C. Osman Hill. He commented: "Naturally, experimental results most likely to be applicable in human medicine...can be expected only from the use of those species having the closest blood-relationship (i.e. phylogenetic proximity) to man. These are the great anthropoid apes of the species *Pan* (chimpanzee), *Gorilla* and *Pongo* (orangutan)...[T]he chimpanzee, [which is] less rare and hardier than the others, is certainly the best choice, providing that financial and housing considerations do not preclude their [sic] use. They are, however, likely to remain the prerogative of relatively few and specialized institutions (primate centers and others), and cannot be recommended for general use."²⁷⁹

- One final point needs to be made. It was common practice in the fifties to use the same species for vaccine substrate that had already been used to test the safety of the polio vaccine strains. (It is said by some scientists that the *same individual animals* were sometimes used, provided they had suffered no ill reactions from the safety testing.²⁸⁰ This, of course, would have reduced the number of sacrifices required.) The only place that CHAT was safety tested exclusively in chimpanzees was the Belgian Congo. This is just one more argument to add to those already cited, and which indicates that *in the Congo, uniquely, chimpanzee cells may have been considered the ideal substrate for preparing oral polio vaccine.*

c) Presence of HIV and SIV in kidney cells.

It has frequently been claimed that HIV-1 cannot survive, or replicate, in kidney cultures. These claims are incorrect, as was demonstrated a couple of months after the Royal Society meeting, by an article entitled: “Renal epithelium is a previously unrecognised site of HIV-1 infection”.²⁸¹ An amusingly-titled editorial (“Much at stake with kidneys?”) co-written by doctors John P. Moore and R.W Doms,²⁸² devoted a lengthy passage to explaining why some persons, “mainly journalists and some laymen on the fringes of AIDS activism...will no doubt cite this article as being supportive of their argument [about how AIDS began]. And so it is, but only to a certain extent.” Why only to a certain extent? Because “OPV was prepared using monkey kidneys, not chimpanzee kidneys, and it is chimpanzees, not monkeys, that harbored the HIV-1 precursor virus”.

I thank Moore and Doms for highlighting the fact that if they are not correct, and chimp kidneys *were* on certain occasions used to make OPV, then this would represent a real opportunity for the crucial zoonosis.

d) Trypsinisation.

Paul Osterrieth states that trypsin was not available in Stanleyville until “several months” after his return from leave at the start of February 1958, and that it took time to set up a tissue culture lab. Thus, even as he denies that polio vaccine ever was made in Stanleyville, he seems keen to establish that it *could not possibly have been made* during the early months of 1958. This, of course, is exactly the time when it seems CHAT vaccine *was* being prepared in his lab, e.g. for the Ruzizi trial.

The claim that trypsin was not available until later in 1958 is very possibly true. On this point, Dr Osterrieth’s claim is supported by the recollection of Dr Ninane, who also stated that, at least in 1957, trypsin was not available at the Stanleyville lab.²⁸³ But it now seems that trypsin is probably irrelevant, because in the fifties it was not mandatory to make polio vaccine with trypsinised cultures. Maitland-type cultures were equally acceptable throughout the fifties, and such cultures could have been produced in Stanleyville from the early fifties onwards – and certainly from the time that Lindi camp opened in mid-1956.

e) Survival of SIVs in polio vaccine preparations.

It has been claimed that the chances of a hypothetical SIV surviving through the various stages of vaccine production to the final vaccine are many trillions to one against.

These well-intentioned but ultimately misleading back-of-an-envelope calculations by professors John Beale and the late Florian Horaud²⁸⁴ are no longer applicable – if, indeed, they ever were. This is because they have been rendered irrelevant by the new evidence indicating that batches of CHAT were prepared locally in Stanleyville – and in all likelihood in primitive cultures made without trypsin, and using cells and sera from locally available primates.

If a proper protocol for the Stanleyville vaccine is ever released, then perhaps some appropriate calculations – or experiments – can be done. However, this seems unlikely, since to date the only CHAT protocol which has come to light (albeit in incomplete form) relates to pool 23, the last pool made in primate kidney culture, this being in 1960 or 1961.²⁸⁵

After speaking with several virologists and a microbiologist, the impression I get is that a live polio vaccine prepared from Maitland-type culture which incorporated SIV-infected cells and sera would be quite likely to retain a significant amount of viable SIV at the end of the vaccine-making process. This is not least because, even when using a good centrifuge and taking great care, it is practically impossible to remove all lymphocytes and – especially – macrophages (those preferred target cells for HIV and SIV) from serum. It may be that some of those chimps which were sacrificed at Lindi were newly-infected (having acquired SIV through the co-caging and group-caging procedures) but had not yet developed antibodies, in which case they would be expected to have had particularly high levels of SIV in their macrophages and lymphocytes at the time of death.²⁸⁶ But in any case, a recent article makes the interesting point that “SIVcpz-infected chimpanzees...produce high infectious virus titres in their peripheral blood”.²⁸⁷

It is worth reemphasising that the “golden age of virology” really had very little in the way of rules and regulations. The tissue culture “bible” of the second half of the fifties was a lengthy chapter by Joseph Melnick entitled “Tissue culture methods for the cultivation of poliomyelitis and other viruses”, which appeared in a book published by the American Public Health Association in 1956.²⁸⁸ It underlines that both (Maitland-type) suspended cell cultures, and trypsinised cultures were considered suitable polio vaccine substrates, and goes on to state that suspended cell cultures can be maintained “for periods varying from 2 weeks to 1 month” by frequent changes of medium, even if “cell growth does not take place in this system”.

This ties in well with the recollection of Osterrieth’s first assistant that polio vaccine orders would come down from the provincial medical director at intervals (presumably in response to requests from doctors in different towns), and that each time Dr Osterrieth would make new (ie a fresh batch of) vaccine.

f) The simian ancestor of HIV-1 Group M.

It has been claimed by some supporters of a west central African origin of AIDS that the wrong species or subspecies of chimpanzee was present at Lindi camp for there to be any chance of the Group M epidemic having emanated from there.

This is unproven. Beatrice Hahn and Paul Sharp quite strenuously argue that the true ancestor of HIV-1 Group M is the SIV of the *Pan troglodytes troglodytes* chimpanzee from west central Africa (Cameroon, Equatorial Guinea, Gabon and Congo Brazzaville), and not that of the *Pan troglodytes schweinfurthii* chimpanzee from central Africa (the DRC, Uganda, Rwanda, Burundi and Tanzania) . However, so few SIV-infected chimps have thus far been sampled that this seems little more than a hunch. [Figure 6]

As I understand it, this is how the debate stands at present. The only known close relatives to the human virus, HIV-1, are the SIVs from common chimpanzees. Of the two SIV-positive *schweinfurthii* chimps which have been reported to date, one comes from Jane Goodall's Gombe Stream camp in north-western Tanzania, while the other (Noah) hails from an unknown location in the DRC (which may or may not be close to the Tanzanian border, and Gombe). Phylogenetic analysis reveals that both of the *schweinfurthii* SIVs are rather less closely related to the HIV-1(M) branch than are the *troglodytes* SIVs from west central Africa.²⁸⁹

However, we still know very little about levels and types of SIV infection in chimps coming from the rest of the *schweinfurthii* range, such as the vast body of the rainforest in the DRC, to the north of the Congo river. Chimps from around Kisangani (Stanleyville), or Ango in the far north, or Gemena further west, may carry very different strains of SIV. Further sampling and testing – of the type which has recently been done so successfully in Cameroon and Gabon – is clearly needed.

So far, the sampling of chimps and bonobos from the central African rain forest has been very limited, and the reporting of tests on those samples has been even more so. In other words, there is insufficient data for theories about which chimp populations hosted the precursor virus to Group M to be advanced on anything more than a tentative basis.²⁹⁰

Of course, the discovery of even one SIV sequence from the DRC which sits closer to the M group than do the present SIVcpz sequences from Cameroon and Gabon would transform the picture. This would be even more dramatic if the SIV in question branched off from *within* Group M.

Probably the best riposte to the assumption that the *P. t. troglodytes* SIV is the “only true ancestor” of Group M comes from the primatologists, and in particular the paper presented by Pascal Gagneux and colleagues at the Royal Society meeting.²⁹¹ They emphasise that recent genetic studies of chimp populations have revealed just how much remains to be discovered. Such studies have resulted in the recognition of a new subspecies (*Pan troglodytes vellerosus*, from Cameroon and Nigeria), but have “called into question the long-accepted genetic distinction between eastern chimpanzees (*Pan troglodytes schweinfurthii*) and western equatorial chimpanzees (*Pan troglodytes troglodytes*)”. The paper features a phylogenetic tree that demonstrates a striking interweaving of mitochondrial DNA sequences from the two latter subspecies, and bears the legend “There is no support for monophyly of [either]

subspecies.” In other words, it seems possible that *troglydytes* and *schweinfurthii* may in future be redefined as a single subspecies.

The paper explains that chimpanzee gene flow in equatorial Africa is still little understood, and that “it seems reasonable to speculate that a chimpanzee population or populations may exist which both harbor the putative HIV-1 ancestor, and which have remained reproductively isolated from other chimpanzee populations over the time-scale relevant to the evolution of the SIVcpz/HIV-1 complex of viruses”. In other words, the ancestral host to today’s pandemic AIDS viruses may exist, or may have existed, in an isolated pocket somewhere in central Africa, and may either not have been sampled yet, or may have died out in recent times.

To highlight the fact that atypical chimp populations exist, Gagneux points to a group of chimps from Mambasa, in the eastern Congo, which appeared to be unlike any other known group in terms of the pattern of blood group antigens. The 1961 paper on these blood group studies²⁹² is written by, among others, Osterrieth and Ninane, and records that 21 of these Mambasa chimps were among those housed at Lindi. (Its lead author, Dr André, told me in 1994 that he believed that none of these chimp bloods are still in existence.) Other chimps from Mambasa *territoire* were supplied to Alexandre Jezierski for his OPV and IPV research.

Even leaving aside the question of whether *troglydytes* and *schweinfurthii* should be defined as one, or two, subspecies, there is a far more basic question to ask with respect to the OPV theory. Can we be certain which apes were present at Lindi?

Despite Ghislain Courtois’ summarising article from 1967, which states that only *Pan troglydytes schweinfurthii* and *Pan paniscus* were present at the camp, the truth is that we simply don’t know (and for that matter, neither would Courtois have known for sure).²⁹³ We do have some clues, however, which suggest that *Pan troglydytes troglydytes* may also have been present.

Fritz Deinhardt’s hepatitis databook from 1959 mentions 54 apes, of which 41 are identified by species, but not subspecies – all 41 were common chimps, *Pan troglydytes*. (That thirteen apes were not identified is probably due to the fact that species was apparently not documented for the earliest arrivals at Lindi, from June 1956 to early 1957.) Two of these 54 apes (one common chimp; one unidentified) came from zoos (with the previous history unrecorded), and one unidentified ape, Ikela Marie, came from the district of Coquilhatville (now Mbandaka), about 1,000 kilometres downstream from Lindi and Stanleyville. However Ikela Marie came to be at Lindi, it seems likely that the River Congo would have played some part in her journey there. She may have been brought upriver by a trader travelling on one of the huge Congo steamers, in which case her precise origin, other than “Coquilhatville”, would probably have been unknown.

Although Ikela Marie was not identified by species, it seems probable that she was a common chimp.²⁹⁴ Several doctors recall that the bonobos were only present at the camp during the first year or so of its existence – ie up to 1957, or, at latest, early 1958 – and a newspaper article written in March 1959 describes the bonobos as having been already “used” for the Lindi “experiments”.²⁹⁵ Ikela Marie arrived at

Lindi in April 1957, and was still alive in April 1959, so it seems unlikely that she was a bonobo.

But which type of common chimp was she? The former district of Coquilhatville (now Mbandaka) was situated on the south bank of the River Congo, directly opposite the natural territories of both *troglydytes* and *schweinfurthii* chimpanzees. The very real possibility that Ikela Marie was not just a common chimpanzee, but a *Pan troglodytes troglodytes* chimpanzee, has significant implications because of the co-caging that was routine at Lindi.

Dr Deinhardt's databook documented rather less than 10% of all the chimps that were "guests" at Lindi camp, so it may well be that not just one, but several *Pan troglodytes troglodytes* were incarcerated there.

So even if Beatrice Hahn is right with her hunch about the precursor virus coming from *P. t. troglodytes*, the real possibility that one or more representatives of this subspecies were present at Lindi is enough to dispose of the argument that the Group M precursor could not have existed at the camp.²⁹⁶

One of the most exciting developments in AIDS research in the last two or three years has been the acceleration of investigations into African SIVs. Recently there have been some particularly interesting discoveries.

In April 2001, Sentob Saragosti's team reported the sequencing of an SIV from Wolf's monkey, *Cercopithecis mona wolffi*, and the fact that this SIV contained a *vpu* gene which is characteristic of the SIVcpz/HIV-1 lineage, and not found in other lineages (such as SIVsm, SIVagm, SIVmnd and SIVsyk). This important finding, which to date is only available as a conference abstract,²⁹⁷ is made all the more significant because Wolf's monkey is found on the southern side of the Congo river, where it shares the greater part of its range with the bonobo, *Pan paniscus*.²⁹⁸

The recent helpful contribution to the debate by Anne-Mieke Vandamme's group, reports on the SIV testing of 26 *Pan paniscus* (14 wild-caught, and 12 born in captivity), and the finding of no positive samples.²⁹⁹ However, the paper goes on to state that bonobos "are known as a very social and peaceful species, and hunting or aggressive interactions with sympatric primates have not been observed. Therefore, the chance of SIV interspecies transmissions to bonobos seems very low." This conclusion is then used to cast doubt upon the OPV theory.

I am surprised by this line of reasoning. *Pan paniscus* is omnivorous, and there can be little doubt that (despite their passivity) bonobos do on occasions eat, or come into contact with, other monkeys, as when they come across individuals that have been wounded or recently killed by other predators. And whether or not it has been observed, it is certainly possible that there may be occasional fights between bonobos and other primates (just as we know there were fights, admittedly under artificial conditions, when chimps and bonobos were caged together at Lindi). The very fact that a *vpu* gene has been found in a monkey that shares its range with *Pan paniscus* should only encourage further interest in sampling the bonobo for SIV.

One looks forward to further reports, since it is known that at least three other teams have conducted similar surveys of *Pan paniscus* in recent years.

Bill Hamilton, in particular, always stated that his best hunch for the immediate ancestor to HIV-1 Group M would be an SIV in *Pan paniscus*. He also pointed out that even if such an SIV had existed among the *paniscus* populations which supplied Lindi camp in the fifties, it might be that such populations were now extinct. Sadly, his attempt during his final expedition to arrange for the collecting of faecal samples from these places proved to be unsuccessful, because the samples that finally arrived in the U.K. were revealed to be from *Pan troglodytes*, and not *Pan paniscus*.³⁰⁰ However, it is hoped that another, more recent expedition may have enjoyed more success.

With regard to the faecal and urine samples from *Pan troglodytes schweinfurthii* which were collected by Bill Hamilton in July 1999 and January 2000, portions of which were then delivered to Simon Wain-Hobson, it is regrettable that we still do not have any formal results. This is despite the fact that one of the published reasons for the postponement of the Royal Society meeting from May to September 2000 was to allow time for a report to be prepared on these samples. All we have is Wain-Hobson's verbal answer to a question posed at that meeting by Stanley Plotkin, in which he stated that he had found no evidence of SIV in the Hamilton chimp samples, but gave few further details.³⁰¹

However, a final point. I have over the past year been informed from several different sources of instances in which, allegedly, SIV-like results have been obtained from samples derived from the two anthropoid apes (*Pan troglodytes schweinfurthii* and *Pan paniscus*) which made up the bulk of the experimental population at Lindi camp. (These reports do not refer to the two SIV-positive *schweinfurthii* chimps which have already been identified, but to other apes.) I do not know what credence to place in these reports. I fully accept that they may be incorrect, and that even if they are real, that the alleged findings may be inaccurate. None the less, this does emphasise the crucial importance of the SIV testing of the anthropoid apes, and the desirability of its being conducted in an open and transparent manner. I believe that all the results of such "sensitive" testing should be fully reported – whether they be positive, indeterminate, or negative.

g) The question of HIV-1(M) diversity.

In the recent past, it has been proposed that not only is the greatest diversity of SIVs seen in Gabon and Cameroon, but that these countries also contain the greatest HIV-1 Group M diversity, meaning that they represent the hearth of the AIDS pandemic.

The first contention (regarding SIV) is unproven; the second (regarding Group M) is now generally accepted as wrong.

The range of SIVs identified in west central Africa during the last few years is wide, culminating in the recent impressive report by Peeters et al, who detected SIV in 13 of 16 primate species tested, and in 16.6% of all primates tested.³⁰² The research was especially valuable in that blood samples were taken both from pets and from

bushmeat found in local markets, revealing that seroprevalence in the wild was rather higher (18.4% versus 11.6%).

However, this comprehensive study was possible largely because Cameroon and Gabon are stable and viable places in which to conduct research, and because there are supportive organisations and institutions functioning in the region, such as PRESICA (*Projet Prevention du SIDA au Cameroun*) in Yaounde, Cameroon, and CIRMF (*Centre International de Recherche Medicale de Franceville*), with its large primate facility, in Franceville, eastern Gabon. There are currently no comparable bases in the DRC, which has been riven by civil war in recent years, with the result that relatively little sampling of primate populations has taken place. Had there been a similar level of sampling, one suspects that a similar level and range of SIV infection might have been detected.

It may be for similar reasons that the two minor groups of HIV-1, Group O and Group N (which are commonly considered to represent separate transfers from chimpanzees to humans), have mainly been encountered in the same two countries, Cameroon and Gabon.

As for the perceived “centre” of HIV-1 diversity, this has changed over the years. In 1996, a team of French and Belgian researchers from the Institute for Research and Development, Montpellier, France (led again by Martine Peeters, and by her husband, Eric Delaporte) proposed that there was a relatively low seroprevalence, but high diversity, of M group subtypes in Gabon, with five subtypes detected there, and that this diversity, together with the presence of HIV-1 Group O, in Gabon, Cameroon and the Central African Republic, could indicate that the epidemic in this region was older, and that “the HIV viruses [might] somehow originate from this part of Africa”.³⁰³ Even as late as 2000, Beatrice Hahn, Paul Sharp and Kevin De Cock were still claiming that it is “within west equatorial Africa that the greatest diversity of HIV-1 Group M viruses has been found”, citing studies in which seven of the M clades had been detected in Gabon and Cameroon.³⁰⁴ Of course, this tied in with their belief that all three groups of HIV-1 (M, N and O) had transferred from different SIVs found in *Pan troglodytes troglodytes*.

It was actually the same Peeters/Delaporte team that proved the flimsiness of this line of reasoning, when Martine Peeters announced at the Royal Society meeting that 247 HIV-1 sequences obtained in 1997 from three cities in the DRC (Kinshasa, Mbuji-Mayi and Mbandaka),³⁰⁵ had demonstrated an “unprecedented diversity”.³⁰⁶ Many of these sequences appeared to be deep-rooted in the phylogenetic tree, and another article by the same team acknowledged in its title that the AIDS pandemic seemed to have originated in “central Africa”.³⁰⁷ This subtle change from “west central Africa” indicated that the DRC, rather than the former French colonies to the north (Cameroon, Gabon and Congo Brazzaville), was now becoming accepted as the likeliest hearth of the Group M epidemic. Further analysis of the Peeters dataset is eagerly awaited.

Other recent studies of HIV-1 sequences from the DRC have been equally remarkable, including one which reported the presence of seven Group M subtypes in a single small town, Kimpese, situated some 200 kilometres west of Kinshasa.³⁰⁸ (It is

worth noting that the nearest known CHAT vaccination site to Kimpese was just 50 kilometres away, at Mbanza-Ngungu, formerly Thysville.)³⁰⁹

The most dramatic evidence, however, has been the recent detection of *all ten recognised Group M subtypes* in a group of 70 HIV-1-positive samples which were originally obtained from Kinshasa women at the start of the recognised African epidemic, in 1983-1985.³¹⁰ Just six of the 70 Kinshasa sequences did not cluster with any of the known subtypes; and three of these six clustered together as what is referred to as a “recombinant form”. In February 2002, one of the lead authors, Tom Folks, chief of the HIV and retrovirology branch at the CDC, informed me that the sequences of the ten subtypes and the recombinant form were “present at nearly the same numbers [ie frequencies] as now”.

The fact that one of these samples was a HIV-1(M) subtype B, the so-called “Euro-American strain”, is especially interesting, for the only evidence of African subtype B infection prior to this report related to white gay South African males in the eighties, and a scattering of isolated African cases in the nineties – all of which appeared likely to be the result of “reimported” infections from the West. Many had begun to suspect that indigenous African clade B might have died out, and that this subtype existed only in Western countries. This Kinshasa sequence may possibly represent the first evidence of indigenous subtype B infection in the African continent, but it still remains to be seen how closely the Kinshasa sequence resembles typical Euro-American sequences from the same time frame.

In his helpful communication, Professor Folks also provided some indication about how his team was interpreting these remarkable findings: “We are stunned by the high diversity and low prevalence situation that we continue to encounter in that region. Obviously I don’t know the answer, but we now hypothesise that multiple events had to occur for HIV to adapt and evolve into a transmissible agent, as well as one that is pathogenic. Whether this is because multiple viral infections were followed by recombinations or whether infections were followed by behavioural events is unclear, but we think that a single spark probably did not happen.”

Tom Folks also wrote that he did not think this supported an iatrogenic theory of origin, but rather one in which only a certain sort of SIV isolate (those resembling HIV-1 Group M) had managed to transfer successfully to humans.

However, having discussed this with others (a geneticist and a molecular biologist), I believe that a far more parsimonious explanation might apply. It seems possible that the microcosm of the Group M epidemic which is represented by the 1983-1985 data from Kinshasa may indicate that a number of individual chimp-to-human transfers occurred in Leopoldville/Kinshasa and its hinterland. These might, for instance, have occurred through the immunisation campaigns in which some 75,000 Leopoldville children (the entire population of under-fives between August 1958 and April 1960) were vaccinated with CHAT, or they might have occurred through the campaigns in which nearly a million others were vaccinated elsewhere in the Belgian colonies.

Urban drift to Leo/Kinshasa was pronounced, for the population seems to have increased almost four-fold in six years – from 350,000 in 1958, to 1.3 million in 1964.³¹¹ The early 1980s population was apparently over three million. So not only

vaccinees from Leo/Kinshasa, but also those from other towns in the Congo, Rwanda and Burundi, may have introduced a variety of different SIVcpz/HIV-1(M) strains to the city that is the only really substantial conurbation in central Africa. It is worth repeating that many of those vaccinated in the military camps, such as that in Stanleyville, would have originated from Leopoldville, and would have returned there at the end of their military service.

This hypothesis would fit rather well with the “punctuated event” origin theory, as initially proposed by Tom Burr, Mac Hyman and Gerry Myers at the Royal Society meeting.³¹² Gerry Myers and his group found an unusual degree of symmetry in the phylogenetic tree of Group M, with remarkable uniformity in the distances between subtypes, leading them to conclude that a punctuated event had occurred, involving multiple, near-simultaneous transfers of SIVcpz to humans. “The natural transfer theory for the origin of AIDS cannot easily be reconciled with these findings”, they concluded.

Following the London meeting, the multiple event hypothesis was challenged by a brief communication by Rambaut et al.,³¹³ from Eddie Holmes’ group at Oxford. They based their conclusions on the assumption that the 1930s date for the most recent common ancestor (MRCA) of Group M was correct, and concluded “our results give us no reason to doubt that the last common ancestor of HIV-1 Group M was present in a human host”. This analysis was summed up for me by Eddie Holmes, shortly after the London conference, as follows: “Tom Burr is assuming there are subtypes. But the DRC [dataset from Martine Peeters] suggests there aren’t....If you look at the global tree, there [appear to be] subtypes. In the DRC tree, it isn’t [like that].”³¹⁴ Holmes was apparently claiming that the DRC shows a “cloud” of variants that have evolved from the MRCA of Group M, and that the subtypes of M that are recognised today are the result of several founder effect episodes, as individual strains were exported and became successfully established in different locales (eg subtype B in the US; subtype E³¹⁵ in Thailand).³¹⁶

However, the 1983-5 sequences from Kinshasa reported by the Folks group at the CDC suggest a very different scenario, for they find that it was the same recognised subtypes which dominated the picture in the DRC at an early stage of the global epidemic, and that the pattern in Kinshasa in the early eighties looks almost identical to the global subtype pattern seen today.

The CDC dataset from 1983-5 suggests to several observers (myself included) that a multiple origin of Group M may, after all, be possible. Further information about the 1983-5 sequences is promised soon, and may shed further light on these issues.

All that can be said at present is that the debate has not been resolved. Many scientists now believe that given the level of recombination that is seen in HIV-1(M), any analysis that depends on phylogenetic *dating* theory is controversial [see below]. It remains to be seen which analysis of the history of the AIDS pandemic will prove to be more correct.

h) The epidemiology of HIV-1 Group M and AIDS.

It has been proposed by Kevin De Cock from the CDC, who was the only epidemiologist invited to deliver a full-length address (as a speaker, not a discussant) at the Royal Society meeting, that “the OPV hypothesis is not supported by data, and the ecological association proposed between OPV use and early HIV/AIDS cases is unconvincing”.³¹⁷

I would argue strongly against his conclusion, which was clearly based on the assumption that Beatrice Hahn’s version of the natural transfer theory is correct. Indeed, this was unsurprising, given that De Cock was one of the co-authors on the paper in which Hahn expounded her position.³¹⁸ But more significantly, De Cock’s conclusion was based on his analysis of the DRC data presented in my book, but not on the data from Rwanda and Burundi, which he ignored. No reason was given for this selectivity, either in the paper, or when I asked the author about this after his speech.

Dr De Cock dismissed the OPV theory by applying strict epidemiological criteria, yet he made a number of unsupported assumptions elsewhere in his piece. One example was his claim that: “the hypothesis that children could have become subclinically infected and survive for many years to go on and spread HIV-1 when adult is improbable.” Another example: “The first indication of epidemic AIDS in the Congo was a report of increased cases of cryptococcal meningitis in 1979, illustrating how HIV disease essentially went unrecognised for decades...” Here he juxtaposes a valuable comment about one of the first recognitions of multiple cases caused by one of the classic opportunistic infections of AIDS with an uncritical assumption that phylogenetic dating theory is correct when it proposes a 1930s MRCA.

To my mind, the early epidemiological clues actually reveal the Achilles heel of the Hahn/Sharp/Korber/De Cock theory of natural transfer origin. The aforementioned doctors all believe that west central Africa is the area where the crucial SIV transferred from a *Pan troglodytes troglodytes* chimp to a human, to spark the pandemic. They believe that the original chimp-to-human transfer may have happened at their MRCA date (in the 1930s), or else some time before that.³¹⁹ In the year 2000, they were still aligning themselves with theories like that advanced by anthropologist Jim Moore (no relation, genetically or spiritually, to Dr John P. Moore), who proposed that poorly conducted vaccinations and injections conducted in their “preferred area”, French Equatorial Africa, in the first half of the twentieth century, may have kick-started the epidemic.³²⁰

More recently, however, with the reports from the Peeters group of multiple variants of Group M being found in three different towns in the DRC, they have had to acknowledge that it is the DRC, rather than Cameroon, Gabon or Congo Brazzaville, that represents the likeliest hearth of the human pandemic.

They seek to explain the apparent dichotomy between simian source and human epicentre by proposing that the capital of the Belgian Congo/DRC, Leopoldville/Kinshasa, may have served as the hub of the human epidemic, a place where the virus could have arrived in the early years after the transfer to humans from *P. t. troglodytes*, and where it could have both spread and diversified. But there are problems with this scenario as well.

Firstly, to get the *troglydytes* virus down to Leo is not a trivial thing. Back in the early decades of the twentieth century, which is the time-frame that Hahn, Sharp and Korber favour, the nearest *troglydytes* chimps to Leo would have been found 100 kilometres or more away, across the river Congo, in a different country, which in turn was under a different European ruler. In those dark, colonial days, when Africans faced so many restrictions on basic freedoms (such as the freedom to travel, to live in another town, or to do business), this was not a small distance for an African human, or a newly-acquired human virus, to travel.

Secondly, they need to have rapid viral diversification within Leo/Kinshasa between 1931 and 1985. However, even then there are problems, for I believe they have no ready explanation for why ten distinct subtypes of M appear to have emerged. That seems a very large number to have evolved in a single city from their one original index case. One detail that would seem to conflict with their hypothesis is that two of the very earliest cases of AIDS seem to have come to Leo-Kinshasa from *outside* the city (in one case from a thousand kilometres away), in order to seek treatment [see below]. It is not clear how they would explain this boomerang effect, whereby viruses apparently escape the capital, but then come winging back again.

Thirdly, they have to explain why the epidemiological pattern in the 60s and 70s suggests that infection spread only to other towns in the Congo, Rwanda and Burundi, so many of which (such as Lisala, Stanleyville, Uvira and Bujumbura) were CHAT vaccination sites. There were fewer travel restrictions after decolonisation, so why did we not see the virus crop up during the same period in the countries to the north, such as Congo-Brazzaville, or to the south, such as Angola?³²¹ Even those pockets of early Group M infection which are not known to coincide with CHAT vaccination sites are either close to them (like Yambuku) or else are cities such as Likasi and Lubumbashi in the mining region of the Copper Belt, where the work-force was comprised of young men (with an attendant population of young women) from virtually all over the Congo, Rwanda and Burundi.

Fourthly, they cannot afford to have any productive infectees left behind in the area of their mooted original chimp-to-human transfer – wherever in west central Africa that might be. If any had been, then the genie would have been out of the bottle – and the first emergence almost certainly would have been in Cameroon (as it was for HIV-1 Group O) or Gabon or Congo Brazzaville, and not in the DRC. Instead, the earliest detection of HIV-1 Group M in the former French colonies relates to the town of Brazzaville, across the river from Kinshasa, where a Soviet man received an HIV-1-infected blood transfusion in 1981. The HIV-infected child of this man was subsequently admitted to a hospital in Elista, Georgia, where the reuse of unsterilised needles led to one of the world's worst nosocomial outbreaks of HIV infection, with another 57 infants infected by the end of the eighties.³²² The children were apparently infected by a Group M variant with a typical subtype G envelope, which strongly suggests that this outbreak was a descendant of the one which occurred earlier in the DRC, and not its ancestor.³²³ This lack of early cases in the countries which comprise the range of *P. t. troglodytes* also makes Jim Moore's theory of improperly sterilised needles spreading early HIV-1(M) infections in French Equatorial Africa look far less plausible.

Taking all these arguments together reveals that the *troglydites* source version of the natural transfer theory has to become increasingly contorted if its adherents really want to fit it to what is known about the early epidemiology of Group M. (Indeed, this is probably why they tend to keep their theory of spread as vague as possible, apart from the insistence that Kinshasa was a hub.)

Finally, let me once again point out the available early HIV-1(M) and AIDS data with respect to the two theories: OPV and natural transfer. 42% of African AIDS cases through 1980, and 45% of African HIV-1(M) infections through 1980, come from Kinshasa.³²⁴ By contrast, through 1980, 68% of all Africa's clinically plausible or serologically confirmed AIDS cases, and 85% of Africa's proven HIV-1(M) infections, came from CHAT-vaccinated places.³²⁵ (Note that in this instance, I am using 1980 as the cut-off year for both disease and infection; whereas the data presented earlier in this paper for HIV-1 infection included 1981.)

This works out as 60% more AIDS cases, and 90% more HIV infections, coming from CHAT vaccination sites than from Kinshasa alone. It is only when one looks at the whole picture that the weakness of the Kinshasa hub theory becomes apparent, for it is quite unable to explain why so many other places where HIV and AIDS first emerged were also places where Koprowski's vaccines were used.

Even the fact that the first cases *appear to* emerge from Leopoldville/Kinshasa may be a red herring. It should be borne in mind that, because of political instability in the Congo (then called Zaire), Rwanda and Burundi in the sixties and seventies, relatively few Western doctors stayed on to work in those countries – and the vast majority of those who did were based in the most Westernised centre, Kinshasa. It may well be that occasional AIDS cases were cropping up in hospitals in places like Kisangani, Lisala, Uvira, Rumonge and Bujumbura, but were either not recognised, or not recorded for posterity. (Indeed, it might be illuminating for an experienced African clinician to conduct a survey of physicians who worked in those places during the 60s and 70s – most of whom, of course, would be Africans.) In short, there is no evidence that the Group M virus spread *upstream* from Leopoldville/Kinshasa to infect the rest of the Congo (as the Hahn/De Cock group would tend to propose). It may have been that, in reality, infectees came downstream to Leo to seek treatment, or (most likely) that the flow was multidirectional, but with most *recognitions* of AIDS occurring in the capital.

The best example of this tendency is afforded by the earliest mooted case of AIDS [see below], a woman from Lisala who came down to Leo/Kinshasa for treatment in 1962, and who died shortly afterwards. If she had stayed in Lisala, we would almost certainly know nothing of her fate. It was only the fact that, when already gravely ill, she was brought to the department of internal medicine in Leopoldville hospital run by doctors Sonnet and Michaux that led to her condition being documented for posterity.

To sum up: although I agree with Dr De Cock that the appearance of early AIDS cases and instances of HIV infection in CHAT-vaccinated towns and villages does not demonstrate causation, it most certainly does suggest a possibly significant association.

One of the most telling statements in De Cock's piece was the final sentence: "epidemiology cannot provide data about events that perhaps happened long ago, and is a discipline that avoids speculation". De Cock is saying that the science of epidemiology has no tools with which to interpret what it considers circumstantial evidence – which allows him to dismiss the OPV theory fairly readily. But I wonder why he did not use his skills to analyse the considerably more contorted epidemiological scenario which is required by natural transfer proponents.

Dr De Cock's paper was identified at the end as "US government work". It is worth reiterating that the US Public Health Service was supporting and at least partly bank-rolling Dr Koprowski's researches in the Congo,³²⁶ which raises questions about whether epidemiological analysis coming from the same source can be relied upon to be absolutely impartial.

i) The earliest AIDS cases.

Some have objected that, although the polio vaccinations occurred in the late fifties, the first AIDS cases did not crop up until the seventies, nearly two decades later.

In all likelihood, of course, this was not the case. In addition to the 38 plausible AIDS cases from the seventies (and one from 1962) alluded to earlier, there is also sporadic evidence suggestive of further potential cases from the 60s. Partly by dint of the passage of time, such evidence tends to be more anecdotal, and the chance of corroboration through blood or tissue samples is reduced. However, it is interesting to note that almost all of these anecdotal cases (just like the 39 clinically-defined cases) relate either to the former Belgian Congo, or to persons originating from Rwanda and Burundi (although in the latter case, we do not always know when they left their countries of origin).³²⁷

Furthermore, there are several reports of AIDS-like fatalities emerging from vaccinated areas in the period following the CHAT vaccinations. In 1962 in eastern Congo, 16 of 21 Rwandese refugee children treated for malnutrition were found to be also suffering from tuberculosis. They and their parents had fled from Cyanguu, a town that had apparently been vaccinated with CHAT in 1959. And in Kampala between 1962 and 1967, five fatal cases of generalised *Herpes simplex* were recorded, again in apparently malnourished children. The three most AIDS-like of these cases (with ancillary symptoms that included TB, chicken pox and bronchopneumonia) involved children whose parents came from Rwanda and Burundi. Another instance would be the three adult AIDS-like cases that Bill Hamilton found in the pathology archives at Mulago Medical School, Kampala, when searching through autopsy records for the early sixties. In two of three cases, the ethnic group indicated that the patient had originated from Ruanda-Urundi; in one case the tribe was not identified. The three cases involved, respectively: pneumonia caused by a "heavy pure growth of Klebsiella" and wasting; B-cell lymphoma and Kaposi's sarcoma (KS) with unusual distribution, including the lymph nodes; and interstitial pneumonia, massive TB, lymphadenopathy, fever, oral sores, and generalised skin rash in a 2-year-old.³²⁸

These examples are not *compelling* as cases of "early AIDS". In children and infants, especially, such immune collapse could have been caused by several other factors (such as starvation and stress during refugee flight). The conditions of some of the

adults may have been caused by cancers, whether diagnosed or not. But they are worth noting, as is the ethnic distribution.

Regarding individual cases, I have recently interviewed, or reinterviewed, some of the doctors who served in the Belgian Congo in the years after the second world war, asking them if they recalled any early cases suggestive of AIDS. The results here are more persuasive, because they come as personal testimonies from Africa-based clinicians with substantial experience.

Dr Jean-Louis Michaux, who served under the late Dr Jean Sonnet in the *Hôpital des Noirs* (later the *Hôpital Generale de Leo-Est*, and then the *Hôpital Mama Yemo*) in Leopoldville/Kinshasa from 1958 to 1967, recalled two cases which, in retrospect, he thought might well have been AIDS. The first case (already cited as the “1962 case”, above) involved a 50-year-old woman from Lisala who died in Léopoldville in early 1962, from generalised Kaposi’s sarcoma, pneumonia, fever, and bacterial infections of mouth and jaw.³²⁹ (Lisala is a town about 1,000 kilometres north-east of Léo, where the entire population was vaccinated with CHAT, possibly at an early stage, though the precise date is not known.) The second potential case involved a 26-year-old male student who apparently came from outside Léopoldville, and who was under Dr Michaux’s care in April 1964. He had TB pneumonia (“an extraordinary tuberculosis evolving in the lung”), haemolytic anemia, and a malignant B-cell lymphoma of the spleen, and he died within a few days. It should be noted that both these plausible early cases of AIDS came in to Leopoldville for treatment, but originated from outside the city.

But the doctor with perhaps the widest overview is Dr Paul Beheydt, who served as a clinician in the same Kinshasa hospital between 1946 and 1981. He recalled that when he was chief of Internal Medicine between 1968 and 1976, he saw a lot of cases which might, in retrospect, have been AIDS, including some which involved atypical Kaposi’s sarcoma, diarrhoea and weight loss. He particularly recalled one young woman who he believed he had seen between 1968 and 1970, who had been suffering from an atypical tuberculosis-like disease (perhaps caused by a rare mycobacterium?) and generalised Kaposi’s sarcoma. This woman, he told me confidently, had represented his first encounter with AIDS.

It is perhaps worth adding that the second and third earliest samples of HIV-1(M)-positive blood from anywhere in the world, which were both obtained in Kinshasa in 1970, were obtained in Lemba, “a new middle-class suburb of single-storey concrete dwellings which had been built near the university between 1967 and 1970”.³³⁰ I believe this raises at least a possibility that they too may have originated from outside Kinshasa.

Of the eight or nine Belgian doctors whom I have interviewed who served in Leopoldville, Stanleyville, Usumbura, Elisabethville or Katana during the 1950s or earlier, not one volunteered, or could recall, an AIDS-like case from *before* the 1960s. The only potential cases I know of which might precede these would be the Stanleyville *Klebsiella* cases reported in 1958. This is not, of course, to say that there were no AIDS cases before 1958.

These mooted early cases of AIDS potentially provide random snap-shots of a slowly brewing epidemic. They do not constitute proof of any kind – but neither should they simply be dismissed out-of-hand. I think of them as messages on old post-cards found at a jumble sale, and I believe that they may well provide serendipitous and useful clues about now-forgotten events from the 50s and 60s.

j) The strange case of the *Klebsiella* outbreaks.

In 1958, Paul Osterrieth reported 142 strains of *Klebsiella* from Stanleyville, most of which came from patients at Stanleyville Hospital (almost certainly the “*Hôpital des Noirs*”) “presenting with urinary infections or fatal pneumonias”.³³¹ This almost casual reference to an outbreak of fatal *Klebsiella pneumoniae* cases in Stanleyville is intriguing, as is the fact that no further information about the sources of the strains was provided. We do not know the exact number of fatal human cases because Dr Osterrieth did not record that detail in his paper; when asked again in the nineties, he said he did not remember. We only know that “cases” were referred to, in the plural, and that they had apparently happened within the previous two years, since Dr Osterrieth only started working at the Stanleyville medical laboratory on August 1st, 1956. However, fatal cases of *Klebsiella pneumoniae* are extremely rare. Dr Jack Davies, in neighbouring Uganda, apparently saw only one fatal case during nearly two decades of pathology work in the major city, Kampala.

What might have caused this outbreak? What may well be a significant clue lies in the fact that a few of the *Klebsiella* strains isolated by Osterrieth had apparently been obtained from chimpanzees. Several of the former members of the medical lab at Stanleyville have mentioned that the *Klebsiella* saprophyte had been killing chimps and/or bonobos at Lindi in the early months of the camp’s existence. Unfortunately, Osterrieth’s report refers only to “chimpanzees”, a catch-all phrase which tended to be used as shorthand for both of the species at Lindi camp (common chimps and pygmy chimps/bonobos). It is therefore not known whether only one ape species was affected, or both. However, Osterrieth does record that: “There were no significant biochemical differences between the strains isolated from chimpanzee and man”.

Although Dr Plotkin, in a recent paper, attempted to play down the significance of this episode,³³² it needs to be reemphasised that *Klebsiella pneumoniae* is one of the opportunistic infections that typifies both human and simian AIDS.

As an opportunistic infection, it only causes disease and death when there is prior immunocompromise, which means that both humans and apes seem to have been infected with both *Klebsiella* and another underlying infection, probably viral in origin, something that was recognised by the Belgian doctors back in the fifties. One candidate pathogen would be an immunodeficiency virus that was new to both species, such as a bonobo SIV which had transferred to both common chimps and humans – or a common chimp SIV which had transferred to humans and bonobos.

It is certainly remarkable that two simultaneous and fatal outbreaks of *Klebsiella* were taking place, among Africans in Stanleyville and among anthropoid apes at Lindi camp (which was fifteen kilometres away in the rain forest, and where – as was repeatedly stressed at the time – the primates were quarantined from the outside world). The most plausible explanation is that there was some common denominator.

One possible common denominator would be a live polio vaccine made in “chimpanzee” cells, and administered locally.

An aside: a newspaper report from March 1959, entitled “Congo may lead world in the fight against polio”, explained that a series of large-scale tests of the Koprowski strains was “being completed” in the Belgian Congo, and that the colonial authorities had decided to vaccinate the whole of the Congo’s child population. It then provided a little historical background. Doctors Ghislain Courtois and Hilary Koprowski had supervised the tests, and for the preliminary field-trials of the vaccine, they had used “between 70 and 80...of a rare, thin-limbed species called *Pan paniscus*....considered by scientists as ‘blood relatives of man’”. To begin with, “a score or two of them died” because they could not adapt to captivity, but then Dr Courtois gave them antibiotics, and housed them with “other, less shy, monkeys” (clearly the common chimps). The remaining bonobos were apparently used in experiments designed “to discover the minimum attenuation of the strains required for complete protection against the disease”, and sections of the brain and spinal cord of animals that died were sent to universities and research centres for investigation. The report ended: “Scientists pronounce the field-trials at the Lindi station fully successful.”³³³

The description of the work conducted on the bonobos would appear to constitute a less than accurate melding together of safety tests and immunogenicity research – and may have stemmed from an interview with a scientist (presumably Courtois) who didn’t want to get too specific. None the less, the emphasis on the crucial role played by the bonobos in this research is intriguing, and suggests that it may have been bonobos that provided tissue and serum for the very first vaccine substrates.

The *Klebsiella* outbreaks are alluded to only in passing in the paper on *Klebsiella* strains which Dr Osterrieth submitted to Belgium’s main journal of tropical medicine in July 1958.³³⁴ Since it now appears that experimental human immunisations with Koprowski’s vaccines may have started around the time that Lindi camp opened in June 1956, this would mean that if these human *Klebsiella* cases were instances of AIDS, and were related to the use of materials derived from the chimpanzees, then in each case death would probably have occurred in less than two years.

I would propose that a simultaneous introduction of SIV and *Klebsiella* into humans could have led to rapid human fatalities, for there is no innate reason why SIVcpz, on entering a new (human) host, would necessarily react in the same slow-acting manner as HIV-1 in that host. An example of dramatically altered SIV pathogenicity in a new primate host is afforded by the strain of sooty mangabey SIV, PBJ14, which – after being introduced experimentally into pig-tailed macaques – caused a crash-and-burn disease which led to death in around 10 days.³³⁵

It may even be that an early version of the chimp-based vaccine was specifically tested on patients in the “Hospital for Blacks”. As mentioned earlier, there is evidence that other unrecorded, experimental (and possibly dangerous) vaccinations and medical interventions were staged in Belgium’s African colonies during this period. In a situation like this, in which so little transparency has been shown about the events surrounding Lindi, it seems to me that such uncomfortable possibilities do have to be confronted.

k) Opportunities for recombination.

It has been claimed that even if vaccine had been made in chimp kidney cells and sera at a lab such as that in Stanleyville, materials from only a single animal would have been required for each new batch (thus, at least theoretically, removing the potential risk of viral recombination *in vitro*).

This claim is correct, but in this instance it seems probable that it does not apply. The Stanleyville chimp cultures described in the AFEB report were produced by combining chimp kidney cells and “isologous sera”, and there is no mention of any attempt to utilise matched kidneys and serum from one animal at a time.

The account by Joseph of chimp autopsies at Lindi included the detail that large amounts of blood were often collected during the sacrifice process, and the account by Osterrieth’s first assistant indicates that blood was also routinely collected from the chimps on Saturdays, often on a weekly basis. Both he and Courtois’ assistant said that the chimp blood was centrifuged to create serum, and this seems to have been done in the sterile room in the virology lab, where tissue culture was prepared. The key question would seem to be whether the growth medium that was required to sustain the polio vaccine virus incorporated *pooled* sera from different chimps.³³⁶

It seems that it could have. The frank recollections of American, British and French virologists and primatologists who worked in the fifties suggest that there was then a low level of awareness of the potential dangers of pooling tissues and fluids obtained from different animals. For instance, the 1956 Melnick chapter cited above advises combining the material from up to eight monkey kidneys at a time to make trypsinised culture.³³⁷ It also recommends that poliovirus prepared in HeLa cells should incorporate growth medium prepared from “serum pools obtained from two to five human donors”.³³⁸ Both these preparation methods involved trypsinisation. But the point, surely, is that if one were devising a method to grow polio vaccine virus in untrypsinised chimp cells and chimp sera (or even, for argument’s sake, in untrypsinised *macaque* cells and chimp sera), then one might well decide to adopt a similar approach. One might decide to pool the cells, the sera, or both.

Furthermore, as outlined earlier in this paper, it may be that batches of vaccine were prepared in series – either because the Stanleyville lab lacked a freezer in the early days (meaning that it was difficult to maintain the titre of stored vaccine), or simply because the easiest way to produce good quality vaccine for a new campaign was to prepare a fresh batch. Plotkin has already explained that with CHAT it was routine to prepare vaccine from vaccine, and serial preparation would almost automatically have meant that cells from different primates were included in each individual batch.

It is also well-known that in the fifties gang-caging (that other potential “mixing agent”) was still routine in many primate centres around the world, including those which held monkeys destined for polio vaccine production. For instance in 1997, I visited Pastoria, near Kindia in present-day Guinea Conakry, which served as a primate holding centre for the Pasteur Institute from the 1920s until the early 1960s (long after independence). Here there was a single large cage where the baboons bound for export to France had been housed, together with other “small monkeys”, including sooty mangabeys.

Similar situations existed in Europe and America, though in many instances changes seem to have been made in the course of the fifties. For instance, John O'Hare Tobin, who worked on polio vaccine quality control at the Biological Standards Control Laboratory in Hampstead, north London, between 1955 and 1960, told me that when he arrived there, just one big cage existed for the vaccine-production rhesus macaques, but that he soon made sure that the monkeys were split up into smaller cages, two monkeys to a cage.³³⁹ Apparently this did not apply everywhere in Europe, however. A former vaccine-maker from the Pasteur Institute apparently told Simon Wain-Hobson that until 1965 there had been a large monkey house in Paris, where baboons (and later other species such as patas monkeys) for producing Pierre L epine's polio vaccine had been held. By this stage, we would hope that the species were kept separate, but the animals were still apparently gang-caged in groups of up to twenty.³⁴⁰

At Lindi, chimpanzees and bonobos were regularly placed two to a cage, and up to ten at a time were placed in the communal play-cage. There are no reliable data on SIV-prevalence in wild chimps, and, as pointed out by several authors, it might vary radically from troop to troop. But a working figure of 2% in juvenile chimps has been used by some authors, on the basis of those chimps which have been tested for SIV so far, most of which were juveniles. Stanley Plotkin, by contrast, argues that chimps get SIV infection by the sexual route, and that because the Lindi chimps were juveniles, they were unlikely to have been infected. In fact, there are no available data on the means of transmission of SIV between chimps. But the fact that the first four SIV infections detected in chimps were all in juveniles (or animals which must have been infected while juveniles) suggests that the parenteral and perinatal routes of infection may be significant.

Even adopting this potentially conservative infection level of 2% would suggest that some eight of the young Lindi chimps used for the polio research would have been SIV-infected on arrival at Lindi, and that the co-caging practices would have allowed further chimp-to-chimp SIV transmission to have occurred thereafter.

So if SIVs were circulating among the chimps, then recombination either *in vivo* (in the Lindi cages) or *in vitro* (in the tissue culture lab) would have been eminently possible. A 1997 paper by Wooley and Desrosiers was probably the first to demonstrate that recombination was possible in both systems. The authors commented: "Recombination may be an important mechanism for increasing variation in retroviral populations."³⁴¹

l) Dating the origin of Group M.

It has been proposed that the most recent common ancestor (MRCA) of today's Group M viruses can be traced back to a time before the start of the oral polio vaccination campaigns.

The phylogenetic dating analysis of professors Sharp, Korber and Vandamme³⁴² suggests that the most recent common ancestor of today's Group M viruses existed in the 1930s, with 95% confidence intervals that extend approximately ten to fifteen years, plus or minus. And so these geneticists conclude that the last common ancestor

of Group M must have existed *at least some few years* before the beginning of the CHAT polio vaccine trials in 1957. They and their supporters maintain that this disproves the oral polio vaccine theory – or renders it very unlikely.

This is not a fact (as it has apparently been accepted by many scientists and journalists), but a theoretical calculation. Nowadays, the dating argument is widely presented as the cornerstone of the alleged “proof” that the OPV theory is wrong. And yet there are several inherent flaws in the theoretical model that has been employed, as more and more geneticists (and other scientists) are coming to recognise.

The date when chimp SIV might have crossed to humans lies at the crux of the argument between the natural transfer and OPV theories. Phylogenetic analysis, and its construction of family trees, is relatively straightforward. It is when one attempts to calculate the *rate of change*, and to date the branches, that things become more problematical. This is because HIVs have a pronounced tendency towards recombination, and phylogenetic dating analysis cannot really cope with recombination.

More and more scientists in the fields of genetics, molecular biology and virology are beginning to acknowledge that phylogenetic dating analysis is essentially an inappropriate tool for calculating the age of a retrovirus like HIV. They suspect that the phylogeneticists may all be making similar assumptions in support of their calculations, and that some of these assumptions may be wrong. Many sceptics now believe that these attempts to make allowances for recombination are, in reality, little more than “educated guesses”, which, in the words of one, means that such analysis becomes “as much art as science”.³⁴³

I am very pleased that Professor Mikkel Schierup, a geneticist who is not afraid to express an interpretation that is different to the Hahn/Sharp/Korber group, has presented a paper at this meeting.³⁴⁴ In the past, he has reported that “very small levels of recombination invalidate the likelihood ratio test of the molecular clock”.³⁴⁵

In his present paper, Dr Schierup finds evidence for extensive recombination in Group M, and points out that “recombination events occurring early in the evolution are very difficult, if not impossible, to detect”. He proposes that failing to make proper allowances for recombination may lead phylogeneticists to either under-estimate *or over-estimate* the time to the most recent common ancestor, and proposes that the error bars need to be set considerably further apart.

Perhaps most intriguingly, *he proposes that if two divergent chimp SIV sequences which differed by 5% or more transferred to humans and recombined, this alone could have created the range of Group M variants seen today.*

This last concept potentially aligns rather well with the OPV theory. This is because recombination between two divergent chimp SIVs could be exactly what happened, either in a vaccine tissue culture made from chimp cells and chimp sera, or else in humans living in a vaccinated town, soon after two different chimp SIV sequences were transferred to different vaccinees. The former explanation would seem to be more parsimonious.

Taking all this into account, it seems to me that phylogenetic dating analysis, which has been represented by many eminent scientists as the “disproof” of the OPV theory, is not a disproof at all.

One additional point: at a discussion session at this conference, Professor Paul Sharp referred to the work done on the impact of recombination on phylogenetic dating by Michael Worobey,³⁴⁶ and implied that Dr Worobey might have significantly changed his mind about his findings, following recent discussions with Professor Bette Korber.

I am not alone in being surprised by Professor Sharp’s implication. My feeling is that it might be better for us to wait for Dr Worobey to let us know whether his position has changed. Otherwise, if we all started making similar implications, then I might decide to imply that Dr Sharp had just changed his mind, and now believed that the origin of Group M stemmed from 1957 – or that Dr Koprowski had just recalled that he did, after all, approve the amplification of CHAT in chimpanzee cells in Africa!

m) The background to the ZR59 sample.

A review of recent papers which rely on phylogenetic dating theory reveals that great weight is placed on the alleged “confirmation” provided by the phylogenetic tree position of the most ancient HIV-1 isolate, ZR59. What this fragmentary sequence actually comprises is about 600 base pairs (about 7% of the HIV-1 genome) derived from a blood sample apparently collected in Leopoldville in 1959.

For instance, Yusim et al., who estimated the MRCA of the M Group as 1931, with 95% confidence intervals of 1915-1941, then used the fragmentary sequence of ZR59 as a control, to test the accuracy of their dating method. They concluded that the time of sampling of ZR59 would be 1957, with 95% confidence intervals of 1934-1962, which, they claimed, confirmed the accuracy of their analysis.³⁴⁷

In the original draft of the paper which reported the ZR59 sequence, the phylogenetic analysis, which was done by doctors Paul Sharp and Bette Korber, was summarised as follows: “It seems reasonable to speculate that the ancestor of the dominant form of HIV-1 was introduced into humans in the early part in the 1950s”. That draft was prepared in August 1997. The final paper, as published in February 1998, postulated that the ancestral virus to Group M was transferred to humans “in the 1940s or the early part of the fifties”.³⁴⁸ Yet some two years later, the same authors concluded that the most recent common ancestor existed in around 1931 (Korber) or before 1940 (Sharp), and that the initial transfer from chimp to human may have been even earlier. I am not disputing the right of doctors Korber and Sharp to adapt their thinking, but I do wish to highlight the fact that such thinking can change fairly radically in a short period of time.

I had some personal involvement with the arrangements for the ZR59 testing, in that it was I who initially approached Professor Andre Nahmias, the Atlanta researcher who then held the last tiny portion of HIV-positive serum, to ask if he would be willing to release part of the sample for PCR analysis. Later, I submitted over a page of text to the lead author on the investigation, David Ho, which related mainly to the provenance of the sample. This detailed the apparent date when Motulsky and Vandepitte had collected the “Leo” series of blood samples (including L70, the

sample which later produced the ZR59 sequence), and the fact that there were several unknown factors about the Leo series. In the final version of the text, this was boiled down to: “This positive plasma sample was obtained in early 1959 from an adult Bantu male, with a sickle-cell trait and a glucose-6-phosphate-dehydrogenase deficiency, living in Leopoldville, Belgian Congo”.

I have to confess that five years after contributing this background information, and after extensive further study of the literature relating to this research, I am no longer confident that all these published details about the L70 donor were correct.

The main reasons for my concern are as follows:

- The blood samples in question were originally taken for a series of four genetics papers which were published seven years later, in 1966; the principal authors were the American, Arno Motulsky, and the Belgian, Jean Vandepitte.³⁴⁹ A review of these and related papers of the era reveals that of the 12 different series of blood samples described in these papers, the “Leo” series (which included the donor of the ZR59 sequence) is the least well characterised in terms of where, when and by whom the samples were obtained.
- We know only that the Leo series was obtained from “mixed Bantus from Leopoldville (a few being hospital patients)”. The series contained 99 blood samples, these coming from 78 males and 21 females. The ZR59 isolate came from a male of unknown age with the sickling trait and G6PD-deficiency. Although an age-analysis is available for a subset of the group, involving 66 samples (from 47 males and 19 females), no age analysis is provided for the group as a whole. It is unclear why this is.
- Rather surprisingly, neither of the two main authors has been able to locate any additional papers or raw data, and neither can recall further salient details about the “Leo” specimens.
- There is no single unambiguous statement in the four 1966 papers indicating that all the blood samples were taken in 1959. Some of those statements relating to the date of the samples seem strangely incomplete, and even the sentence that opens the first article: “In 1959, blood specimens from 1,860 individuals originating from the Congo were collected” could be interpreted in different ways. This total of 1,860 would appear to include all the 12 major series described in the papers, including the Leo series. However, the statement is clearly incorrect in one respect, because the 1,860 samples described in the papers included nearly 400 which did not originate from the Congo, but from Ruanda-Urundi. By the same token, it seems possible that although 1,860 Congolese blood specimens were undoubtedly collected in 1959, *not all the 1,860 specimens described in the Motulsky genetics papers* were necessarily obtained that year.
- In 1996, Arno Motulsky admitted to me that the Leo series could have been sent to Seattle after his departure from the Belgian Congo in March or April 1959, and could have been collected by a much-respected Belgian doctor called Jan Stijns, who obtained thousands of blood specimens for laboratory work. In fact, as I have indicated in the past, there is evidence which suggests that the Leo series may have been obtained a substantial period of time after Motulsky’s visit to the Congo and Ruanda-Urundi – by which I mean some years afterwards. I am still researching certain aspects of this question, but it is my intention to publish something about it in the foreseeable future.

- However, there is also another (even more important) uncertainty about the ZR59 isolate. It is possible that ZR59 did not, after all, come from an adult. There are some clues available, but they are tantalisingly incomplete. The entire “Leo” series is described as “adult” in a figure in one of the 1966 papers about these samples.³⁵⁰ In the same figure, the “Stan” and “Ya” series are also described as “adults”. However, it is revealed in another paper that the Stan series included a child of eight years, and that the Ya series included a child (or children) as young as three. (All three series – Leo, Stan and Ya – comprised or included hospital patients, so it seems possible that these children were accompanying parents in adult hospital wards at the time they were tested.) A subset of 66 of the 99 members of the Leo series reveals that it included males as young as 17, and females as young as 10; (the latter, remember, had been described as “adult”). Unfortunately, there is no available information on the remaining 33 Leo blood donors, which may or may not have included the ZR59 donor, and may or may not have included some young male children.
- If ZR59 was obtained from a child, there are important implications. All children in Leopoldville aged five and under were reportedly vaccinated with CHAT between August 1958 and April 1960,³⁵¹ so the L70 donor (especially if his blood was taken after April 1960) may well have been a CHAT vaccinee.

n) The other types of HIV.

One other argument that has quite widely been used against the OPV theory is that the minor outbreaks of HIV infection (caused by HIV-1 Group O and Group N, and by HIV-2) appear, it is claimed, to have been caused by “natural transfer”.

This may be the case, for with HIV-1 Group O and Group N, there is indeed a geographical coincidence between the range of the ancestral host primates, and the location of the initial outbreaks. However, for HIV-2, which has an apparent hearth in Guinea-Bissau [see below], the correlation is less clear, (just as it is with HIV-1 Group M, if one subscribes to the Hahn/Sharp/Korber version of events).

But as with Group M, there are other possible explanations for the minor outbreaks of HIV. As pointed out in the postscript to the 2000 paperback edition of *The River*,³⁵² in the late 1950s experimental polio vaccines were administered in French West Africa (AEF) and French Equatorial Africa (AOF), the areas which embrace the hearths of the three minor HIV outbreaks.

Between September and December 1999, Simon Wain-Hobson, the head of retrovirology at the Pasteur Institute, was industrious in his attempts to find out more about the polio research that had taken place in those former French colonies of Africa. Like me, he searched the Pasteur archives, and discovered that the key annual reports from the Pasteur satellite at Brazzaville (in French Equatorial Africa, AEF) were missing for the years 1955 to 1960 inclusive, and that for Pastoria (in French West Africa, AOF)³⁵³ no annual reports existed for the years after 1956.

At this stage, he began arranging to meet some of the doctors and technicians who had worked in those two establishments (and at the Pasteur in Paris) during the late 1950s and early 1960s.³⁵⁴ Two of the most fascinating interviews were with a doctor who had worked at the Pasteur satellite in Brazzaville (now in Congo Brazzaville) between

1955 and 1961, who told Wain-Hobson that he had administered both injected and oral polio vaccines made by Pierre L epine, head of virology at the Pasteur in Paris, in AEF from 1957 onwards. (In those days, Brazzaville was responsible for public health, including vaccinations, not only for AEF – the present-day Gabon, Congo Brazzaville and Central African Republic – but also for the adjoining trust territory of the French Cameroons, which make up the greater part, including most of the south and east, of the present-day country of Cameroon. In practice, that responsibility also extended across the border into the neighbouring colony of Spanish Guinea – now Equatorial Guinea.)

The Brazzaville doctor said that he had fed OPV in a rural area just inland of what is now Port Gentil, Gabon, in both 1957 and 1959. He also said that he had administered both IPV and OPV in the city of Brazzaville in the same two years, and remembered using a syringe to squirt polio vaccine into the mouths of children lined up in a Brazzaville school-yard.³⁵⁵

It is now apparent that the key question is whether these oral vaccines had been prepared locally. In the first interview, the doctor in question apparently told Wain-Hobson that he “grew polio on local monkey kidney cultures” (as well as in HeLa and KB human cell line cultures) in the Brazzaville lab, but it seems that he was referring to tests for polio antibodies. During a second interview, he again spoke about growing poliovirus in local monkey cultures (including, he thought, those from the most common primate in the region, the moustached monkey, *Cercopithecus cephus cephus*), and this time it appears to have been said within the context of a discussion of the polio vaccinations.³⁵⁶

Another scientist whom Wain-Hobson interviewed assured him that local African monkey kidneys would undoubtedly have been used, since that way you could produce more vaccine. “It was [all] a question of production”, he told him.³⁵⁷

Even if these testimonies do not constitute absolute proof that polio vaccines were prepared in the AEF, it would now take a brave (or foolish) person to insist that they were *not* locally amplified. Indeed, Wain-Hobson recently wrote to me about the subject of local amplification, and stated: “you can find references to people culturing polio in central Africa at the time, so the principle is established. It would be hard for anyone to deny this.”³⁵⁸

In fact, it would be more foolish than hard, because there is documentary evidence that scientists were preparing polio vaccines in local primate tissues in other African countries during this period. L epine’s collaborator, Alexandre Jezierski, was making polio vaccines (both IPV and OPV) in African primate tissues at Gabu in the Belgian Congo from 1953 onwards, as was James Gear in Johannesburg (starting in 1955 for IPV and 1957 for OPV). And exactly the same thing was then happening with CHAT in Stanleyville, according to the multiple strands of evidence presented in the present paper.

Another AEF polio vaccination which both Wain-Hobson and I looked into took place in three stages between November 1957 and January 1958, in response to a polio epidemic around the town of Mitziac, in what is now northern Gabon.³⁵⁹ The doctor in charge (Dr L-J Andr e) explained that the vaccine had originally been intended for use

elsewhere, but that, given the gravity of the situation, it had instead been diverted to Libreville, and thence to him in Mitzic. He gave three shots of the vaccine to the scholars in a local school, and to villagers in the rural areas around Mitzic, which apparently included some across the border in what is now Equatorial Guinea. He believed it to be a Pasteur-made inactivated vaccine – but since there appear to be no precise records, this does leave open some room for doubt. One of the several ideas which Pierre L epine had frequently floated in speeches and articles between 1955 and 1958 was that of giving a mixture of killed and live polio vaccines, and the regime which he particularly seemed to favour involved two shots of IPV to confer initial protection, followed by a shot, or an oral dose, of live vaccine. In June 1958, at a conference in France, he commented: “We have conducted experiments along these lines, and we continue them, but we can only do so with great prudence and much deliberation.”³⁶⁰

Interestingly, one of the few other contributions made to the medical literature by Dr L-J Andr e was a brief paper written in 1987, which proposed that AIDS was an ancient disease, which might have originated from “Hispaniola” (Haiti and the Dominican Republic) as early as the fifteenth century.³⁶¹

Many of the French colonial doctors of this era were, like Dr Andr e, “captains of medicine” who were based in AEF and AOF as part of their military service; (AEF apparently had the undeserved reputation of being the worst possible colonial posting). It seems likely that some of the other “prudent experiments” staged by Dr L epine may have been effected through their good offices, though with or without their knowledge is less clear. When Wain-Hobson visited the French military archives in southern France, he apparently found them in a state of conspicuous disarray. He said they contained almost no relevant details about colonial polio vaccinations in the fifties, though other sources assured him that extensive vaccinations had been carried out in places such as Cameroon, Gabon and Congo Brazzaville, but without being recorded.

Wain-Hobson also discovered that the large primate centre at Franceville, Gabon (nowadays called the CIRMF, and renowned as probably the leading centre of African SIV research) had actually started up as a bit of private enterprise by French military vets in the late fifties. Apparently they wanted to ensure a good supply of local primates – notably chimpanzees and mandrills. It is worth adding that a paper by L epine and some Pasteur Institute colleagues in 1955 analysed the presence of microfilaria in a tissue culture made from the kidneys of mandrills, pointing out that this could lead to contamination of cultures used for the preparation of “non-inactivated” (ie live) vaccines – which in this context clearly meant polio vaccines.³⁶² Mandrills are found only within the former AEF, and as we now know (partly due to the efforts of scientists from the CIRMF), they carry their own, unique SIVs, albeit ones that are not closely related to either HIV-1 or HIV-2.

But it was not only in AEF that L epine’s vaccination experiments were staged. The central laboratory in French West Africa (AOF) during this period was at Pastoria, in present-day Guinea Conakry, and in 1997 I interviewed Dr Kecoura Camara, who had been the first African director of that lab, from 1961 onwards. He told me that Pasteur polio vaccines were administered throughout AOF, in present-day Guinea, Senegal, Mali, Niger, C ote d’Ivoire and Burkina Faso, and said that this probably

began around Pastoria itself in 1956. He did not know whether these vaccines would also have been given in contiguous territories, like Portuguese Guinea (later Guinea-Bissau), though it is recorded that Pasteur-made rabies and yellow fever vaccines *were* given in the Portuguese territory.

He did not mention anything about oral polio vaccines, and I did not ask him about *local* vaccine production. But Dr Camara told me something far more dramatic. He said that some time after 1956, the vaccination regime changed to two injections of IPV, followed by one of attenuated vaccine – the same regime which I already suspected might have been used at Mitzi. If this detail is correct, then this would represent a major difference between the European and African polio vaccines of Pierre L epine. (L epine’s polio vaccines were given in France from 1957 onwards, and in West Germany in 1958, and only IPV’s were used.)³⁶³

Such a difference would be especially significant if any batches of the injected live component allegedly given in Africa had been prepared from the cells of baboons which had been gang-caged (as mentioned above) with SIV-infected sooty mangabeys at Pastoria.

HIV-2 and sooty mangabey SIV (SIVsm) are nowadays often described as “the same virus in different hosts”. The baboon seems to be the only known African primate apart from the sooty which can be experimentally infected with HIV-2 without causing disease,³⁶⁴ and the co-caging of the two species at Pastoria means that there is a clear potential chain of SIV transmission from sooty mangabey to human. The Pasteur IPV was inactivated with beta-propiolactone, but a live component would not, of course, have been inactivated at all – and injection would have been an extremely effective method for introducing an adventitious virus like SIVsm to humans.

Guinea-Bissau appears to be the natural hearth of the HIV-2 epidemic,³⁶⁵ though before HIV-1 moved into West Africa at the start of the 1990s and “took over” from the less infectious virus, it was apparent that all the former countries of the former AOF had a low, but none the less significant, level of HIV-2 infection.³⁶⁶

It should be added that although sooty mangabeys are still abundant in several countries in the HIV-2 belt (C ote d’Ivoire, Liberia, Sierra Leone and Guinea Conakry), and are found occasionally in southern Senegal, no sooty has been seen in Guinea-Bissau since the 1940s. However, it is reported that mass-vaccination campaigns with different vaccines were staged by military doctors in Guinea-Bissau throughout the 1960s, and that some campaigns may have started in the previous decade. (Because of a dearth of records in Lisbon and Bissau, it is not known if polio vaccines were among those given. However, it would have been entirely surprising if polio vaccines of some variety had *not* been given.)

With regard to AEF, it appears that other polio vaccine field-trials like those at Mitzi may have been staged in Gabon, Cameroon and Congo-Brazzaville in the late fifties. Given the primates which appear to have been in demand at that time, it seems possible that some of these trials may have involved vaccine prepared in tissues from primates such as moustached monkeys, mandrills and chimpanzees. Although all three species carry their own SIVs, it is only SIVcpz, as far as is known, that can be transmitted to humans.

HIV-1 Group O appears to have a hearth in southern Cameroon and northern Gabon, though it has since spread to other countries, notably Nigeria. HIV-1 Group N appears to have a hearth in Cameroon, though at one stage all of the former French provinces of that country featured cases of infection, whereas neither of the two former British provinces did so.

It is important to point out that during the 1950s, there were close links between many of the scientists from different nations who played major roles in the experimental polio vaccine trials in Africa. What follows is but a summary from the sparse records that are available.

Alexandre Jezierski worked for several months with Lépine in Paris in 1954-5, during which time he continued his researches on IPVs and OPVs made in substrates from fifteen different African primates; he met up with Koprowski for three days in the Congo in 1957, and visited Gear in South Africa in 1957 (and on other occasions, too). Ghislain Courtois also worked on tissue cultures at the Pasteur in Paris in 1954, and in the same year he visited the Pasteur Institute in Brazzaville. Pierre Lépine, who was a good friend of Koprowski throughout the fifties, spent over two weeks with him at the Muguga conference in Kenya in 1955, after which he intended to visit Jezierski in Gabu, and then the Pasteur in Brazzaville (which he had already visited in 1954). Other frequent visitors to Brazzaville, Stanleyville and Johannesburg were senior staff (including Dr Carvalho de Sousa, and Dr Fraga de Azavedo) from the Institute for Tropical Medicine in Lisbon, and these same doctors hosted a huge tropical medicine conference in Lisbon in 1958, that was attended by Courtois, Lépine, Albert Sabin and many others. During the key 1955-1957 period, Hilary Koprowski twice visited Courtois in Stanleyville and Gear in South Africa. Koprowski, Courtois, Gear, the director of the Brazzaville Pasteur, and the inspector-general of the overseas branches of the Pasteur, were among those who attended the Stanleyville virus symposium when the new labs were opened in September 1957.

It is more than likely that during these meetings, ideas about (and approaches to) polio vaccination were frequently exchanged and discussed.

To sum up, there appear to be close parallels between what Koprowski and the Belgians were doing with OPVs in the Belgian Congo, and what the French scientists were doing with different types of experimental polio vaccines in French Equatorial Africa and French West Africa. These three regions embrace the hearths of all four known outbreaks of AIDS, and no cases of any of the four types of HIV have been identified from before the time of the polio vaccine trials in the 1950s.³⁶⁷

I have not done anything like the same degree of research and cross-checking on the subject of the minor outbreaks of HIV (HIV-1 Groups O and N, and HIV-2) that I have done for the HIV-1(M) story that centres on Stanleyville/Kisangani, and the sad cessation of collegiate relations with Simon Wain-Hobson has meant that one formerly valued source of information has dried up.

I believe that further research into the minor outbreaks of HIV should be carried out, and that it would be best conducted by a native French (and/or Portuguese) speaker.

Before closing this section, it is both necessary and relevant to provide some background details about how certain of the scientists who developed the experimental African polio vaccines (or their successors) have responded to the various polio vaccine theories of origin of AIDS in recent times.

In Paris, in April 1992, a meeting took place between Stanley Plotkin and Luc Montagnier, who was then head of virology at the Pasteur Institute. We know about this meeting only because a few days after this, Leonard Hayflick mentioned it to Chuck Cyberski, a Californian television journalist who was himself suffering from AIDS. Hayflick also revealed that during the previous few weeks, he had been involved in discussions with Plotkin and Koprowski about Tom Curtis's article about CHAT and AIDS that had appeared in February 1992 in *Rolling Stone*.

Hayflick told Cyberski that although everyone was now pointing the finger at Koprowski's vaccines, nobody had yet realised that Pierre L epine, from the Pasteur Institute, had also made injected and oral polio vaccines from the tissues of baboons, and had field-tested these vaccines in French Equatorial Africa.³⁶⁸ As Hayflick expressed it to Cyberski: "not only they tested [the vaccines] there, but the baboons came from that area". (It was this intriguing clue, first passed on by Blaine Elswood in 1992, which started my own investigations into the French-made vaccines. Also intriguing was the fact that in an interview with me just one year later, Dr Hayflick frankly stated that "the final substrate [for polio vaccine] was constantly contaminated monkey kidney" which could have included "dangerous viruses, maybe even HIV-1, who knows?")³⁶⁹

The intriguing thing about all this is the timing. The Paris meeting between Plotkin and Montagnier took place two months after Dr Montagnier, who was then also editor of the Pasteur-published journal *Research in Virology*, had forwarded Dr Koprowski a copy of an article entitled "Polio vaccines and the origins of AIDS", which had been submitted to that journal by Blaine Elswood and Raphael Stricker.³⁷⁰ In a covering letter, Montagnier informed Koprowski that he was going to publish the article as a "medical trend paper", and invited his comments, which he said could be published at the same time.³⁷¹ Koprowski then began a correspondence with Albert Sabin about how best to respond to the paper.³⁷² In the end, he accepted Sabin's advice that it was better to ignore it, and not to submit a formal response.

(As an aside, Albert Sabin had his own interest in the issue, for his live polio vaccines had also been prepared in different substrates around the world. His strains had been amplified in rhesus macaque tissues in the Soviet Union in 1957, and in vervet monkey tissues in the South African vaccine prepared by James Gear and colleagues in 1957-1958. Both of these local preparations of the Sabin vaccine were fully reported in the literature of the fifties.³⁷³ The South African version of the vaccine was field-tested on millions in Kenya, Uganda and Mauritius in 1959, and in South Africa itself from 1961 onwards. The vervet monkey carries its own SIV, but fortunately it appears that it is not transmissible to humans. So, leaving aside such issues as the clandestine or open testing of polio vaccines and of polio vaccine substrates, it may be that Sabin and Gear were simply more fortunate than Koprowski and L epine.)

But I digress. A few weeks after the Plotkin/Montagnier meeting at the Pasteur, the editorial board of *Research in Virology* wrote to Elswood and Stricker, asking them to scale their article down to a brief letter, which was eventually published in January 1993, twelve months after their original paper had been submitted. When I spoke with Luc Montagnier about this in 1997, he agreed that the article had been among the matters discussed with Plotkin in April 1992, but implied that their discussion had not been linked to the subsequent decision to ask Elswood and Stricker to downsize their article. Although I consider Professor Montagnier an honourable man, I find it hard (given the background as provided by Dr Hayflick) to dismiss the possibility that there may have been some linkage.

Seven years later, just weeks after the publication of *The River*, Stanley Plotkin apparently made a similar approach to a senior official at the Pasteur. I am reliably informed that in September or October 1999, Dr Plotkin wrote to the then director of the Pasteur Institute, Professor Maxime Schwartz, mentioning my book, and its references to the use of Pierre Lépine's polio vaccines in Africa. Apparently, Plotkin proposed that the Pasteur could not remain silent, or idle, about what I had written. Plotkin's letter was written on the headed notepaper of the Pasteur Merieux vaccine house, of which he was then managing director. Although it is not known how Professor Schwartz responded, it seems that he was unimpressed by Plotkin's approach.

Meanwhile, Professor Schwartz (who was due to retire at the end of 1999) told Simon Wain-Hobson to continue his investigations into the ancient Pasteur vaccines, while keeping him discreetly informed. It is not known whether or not Dr Schwartz's successor as Pasteur director adopted a similar policy towards the investigation, but Simon Wain-Hobson's apparent change of heart about these issues early in 2000 should perhaps be viewed within this historical context.

5. The political debate: even if it did happen, do we really want to know about it?

"Is man an ape or an angel? Now I am on the side of the angels." (Benjamin Disraeli, 1864)

In this section, I shall concentrate on some of the behind-the-scenes activities which have been going on in response to the OPV theory, which mean that this is now as much a political controversy as a scientific one.

a) Good doctors and spin doctors.

In an article responding to *The River* in 2000, Stanley Plotkin ended with the following passage: "*The River* is a house of cards built on a swamp of conspiracy theory, unsubstantiated insinuations and character assassination. It is fundamentally meretricious³⁷⁴ and does not withstand critical analysis."³⁷⁵

I am not entirely surprised that Dr Plotkin has elected to adopt the position of the injured party. However, I do find it intriguing when other, supposedly fair-minded, scientific commentators on the origins-of-AIDS debate also begin to abandon the time-honoured approach of first examining and testing, and then providing balanced and informed analysis.

I believe that the aforesaid professors and doctors, aided and abetted by some of the world's leading scientific journals, have abandoned good scientific practice in order to argue their position in the manner of spin doctors and public relations consultants. It has been both illuminating and chastening to watch this process over the last two years.

The noble oath sworn by doctors the world over begins: "First, do no harm". By contrast, the oath sworn by spin doctors begins: "First, get one's point-of-view across, and then, if there is time, attend to the patient".

Even back in 1998, which is when Bill Hamilton wrote his foreword to *The River*, he had little doubt that background manoeuvrings were taking place in response to the OPV hypothesis. When I reread this foreword now, I am simply stunned by Hamilton's prescience. He saw clearly that Truth had already become the patient, the party in urgent need of care and attention. And he responded by levelling a remarkable accusation at the powers-that-be.

"Every time", he wrote, "two people put their heads together, Truth suffers; when many put their heads together, she suffers more. A major point of this book is that when the heads are great ones and have owners with much to lose (employed perhaps in giant companies or government departments), Truth can be made so ill that we should all shiver."

He continued: "Once there is acceptance [of evasion and untruth] by an 'Establishment', there is often no need to whisper about it any more: in those who have jointly suffered to win, say, the Queen's Commission in the British armed forces, or the privilege of saying the Hippocratic Oath, a solidarity springs up automatically, and with it a deep conviction that the purpose of the discipline, whatever it be, must be good."

Bill ended by suggesting that everyone should "think hard" about the implications of the OPV theory, "...all this before Truth, more white and sick even than with AIDS, quietly rejoins us through another door".³⁷⁶

With the historical and scientific information presented above, there is now compelling evidence to indicate that CHAT vaccine was amplified in the Stanleyville laboratories in the late fifties, and that this was done in a substrate of chimpanzee cells. Even the most hardened sceptic, I believe, should at least be willing to accept that this *might* have happened.

Whether human cells (which may or may not have been HeLa cuckoos) were also subsequently used to amplify the vaccine fed to African colonial subjects is unproven, and is in any case not central to the argument.

And whether these events sparked the AIDS pandemic is unproven also. However, it is worth noting that, to date, those scientific arguments which have been put forward to counter the OPV theory are either readily disprovable, or else are far less persuasive than their proponents sometimes like to claim. Furthermore, there is a

discernible tendency among many of those who have become involved in this debate to fall back on the argument that “we must be right, because we’re scientists”.

There is another issue here, however. The way that certain scientists have responded to these allegations has been deeply disturbing. Even some of those who began as open-minded investigators have since become compromised. A major question-mark has been raised about the ability of certain individuals to cope with, and respond openly to, the possibility that they or their professional colleagues may have blundered.

The lack of transparency shown by most of those who were involved with the CHAT trials in Africa has meant that this debate has dragged on for years longer than it ought to have done. However, in some ways, this protracted process has been beneficial, because some persons have, with the course of time, gradually begun to reveal their true positions. It can now be clearly seen that certain people have told lies, and that others have tampered with evidence. This needs to be said.

I believe that certain members of the scientific establishment realise that they are losing the scientific arguments about how CHAT was made, and have instead resorted to fabrication and spin. Instead of trying to get to the truth of the matter, they have instead invested rather a lot of time and money attempting to construct a position that they believe can be defended. The priority of these people, it seems, is to win the battle of public opinion.

Such a web of disinformation has been woven around these issues that I have finally decided, albeit reluctantly, that it is now time for me to abandon “the higher ground”, and to expose some of the sad things that have been taking place in the good name of Science.

b) Beautiful things, ugly things.

At the end of the Lincei meeting, as those who attended it will know, Professor Weiss and I did not see eye to eye. By this stage, I was already convinced that he had played a less than noble role in this debate, and for that reason, I was moved to walk out during his closing speech, calling it a “disgrace”. Let me now amend that. The speech itself was not a disgrace, for much of it was wise and clever and helpful. What was highly regrettable was the portion that addressed the origin of AIDS, where Professor Weiss continued to show frank bias.

After the meeting, at the back of the hall, Professor Weiss and I had words. Some bitter and unguarded things were said (by both parties). Presently, I asked Robin to explain the statement he had made in *Nature*, that “some beautiful facts ha[d] destroyed an ugly theory”. To begin with, he was unable to respond. At the second time of asking, he told me: “It’s a well-known phrase that we sometimes use in lab meetings”.³⁷⁷

That may well be the case. However, it does not answer the question. And it doesn’t justify Professor Weiss’s public (and much-quoted) statement that the OPV theory had been “destroyed”. That claim is untrue.

Later, I ended up joining the large restaurant table where Professor Weiss and several of the other speakers and Lincei professors were taking their lunch, and something of a truce was declared. As I got up to leave, I offered Robin my hand, and said that his speech had been “magnificent, except for the parts about the origin of AIDS”. We shook hands.

However, a couple of months later, I read an article in an American magazine for HIV-positive people, *POZ*. The journalist had asked Robin for his thoughts about the new information I had presented in my speech at Rome, and he responded: “I am not aware of any new information recently reported by Hooper, only speculation that seems to grow wilder by the month”.³⁷⁸

Since the Rome meeting, Professor Weiss has also made the following on-the-record comments to a journalist (and I quote):

- “Osterrieth has categorically stated at the Royal Society conference last year (and in its printed proceedings published in June 2001) that chimp tissues were not used by him or anyone else at Camp Lindi or Stanleyville/Kisangani to make polio; in fact, the techniques were not available there, so I was wrong to suggest so in my book review. Either Osterrieth is lying through his teeth or Ed has got it wrong (his African ‘witness’, it appears, wasn’t born until 1960, 2 years after the alleged event)”.
- “Read [Hooper’s] press release with care, eg: when stating that 5/16 places where HIV (or AIDS) is known to have been present before 1981 were places where CHAT vaccine was given, that means that 11/16 early AIDS sites are unrelated to CHAT”.

Disturbingly, both these comments contain untruths and inaccuracies.

In the first, he states that I have quoted someone as a witness who was not even born at the time of the events in question. He is, in effect, implying that I have (either knowingly or extremely carelessly) invented a witness. This claim is false. Where Dr Weiss got it from, I don’t know, unless he is misquoting the similar false claim previously made by Plotkin.

In the second, it is he who perhaps should be “read[ing] the press release with care”. I have actually stated that nine (not five) out of sixteen places where HIV is documented as having been present in Africa by 1981 were CHAT vaccination sites. I also wrote that five of those nine were places where CHAT had been fed *between February and April 1958*, the specific time for which there is evidence that CHAT was being made in Stanleyville, apparently in chimp cells.

Professor Weiss had a copy of my speech (which I had personally handed him at the Lincei meeting), so there was really no excuse for misrepresenting me in these on-the-record statements to a journalist.

Dr Weiss’ casual assumption that polio vaccine was not, and could not have been, prepared in Stanleyville *because Dr Osterrieth says so* goes against the most basic principles of the society to which he had so long aspired to be a fellow – a wish that was realised in the late nineties. “*Nullius in verba*” reads the motto of the Royal

Society: “take nobody’s word for it.” (As an aside, this intriguing detail about the Society’s motto featured in an editorial entitled “Protest is an ally of science”, in which Prime Minister Tony Blair’s keynote speech about “speak[ing] up for science” was criticised on the grounds that it seemed to be more concerned with spin than with true science.³⁷⁹ Although Dr Weiss has apparently, in 2002, been appointed a scientific advisor to the government on the BSE epidemic, he is not known to have contributed to this particular speech.)

But I digress. I believe that Dr Weiss’ comments to the journalist reveal two things. Firstly, that he is capable of making careless mistakes. Secondly (and highly significantly) that he is now quite determined that the OPV case must be presented as “destroyed”, and myself as “wrong”.

As it happens, Professor Weiss features quite prominently in a new book about the Gallo/Montagnier debacle, which reveals in passing that he has made some quite significant scientific mistakes in the past. In the *Dramatis Personae* section of the book, he is described as: “Scientific Director, Chester Beatty Laboratories, London, who isolated HL-23V, putative first human cancer virus which proved to be a monkey virus contaminant; later isolated AIDS virus CBL-1, which proved to be contaminant of Pasteur’s LAV.”³⁸⁰ In other words, Robin Weiss’ lab, just like that of Robert Gallo, had cross-contamination of its own cultures from AIDS patients with Montagnier’s LAV cultures, and then tried to claim the Pasteur LAV (HIV-1) isolate as its own.

I have recently discovered some more about the background to Professor Weiss’ involvement in the origins of AIDS debate. It goes back a long way – at least to the second half of the 1980s. Apparently, he first came across literature discussing the idea of a possible iatrogenic origin when he read a report put out by the (British) National Anti-Vivisectionist Society (NAVS) in 1987.³⁸¹ Later, during an interview which I conducted with him in 1990, reference was made to a letter which had been sent to one of his colleagues at the Chester Beatty Labs, which claimed (almost certainly wrongly) that contaminated inactivated polio vaccine (IPV) might have started the epidemic.³⁸² And then in 1992, he read Tom Curtis’s article linking CHAT and AIDS in *Rolling Stone*.³⁸³

By good fortune, there is some documentary evidence indicating where Dr Weiss stood in the origins debate in 1994. I have a copy of a page of referee’s comments about a letter which Bill Hamilton submitted to *Science* in that year, in which Bill argued that the OPV theory should be taken more seriously.³⁸⁴ Various details indicate quite clearly that the referee in question was none other than Robin Weiss. The content is very interesting.

Although Weiss ends his referee’s comments with: “I do not consider polio vaccine to be one of the more likely theories of origin”, he begins in different vein. “One cannot state with any certainty yet”, he writes, “that the oral polio vaccine was not the source of HIV-1 introduction into humans. Anyone who has looked at a monkey kidney monolayer culture, especially by time-lapse cinematography, will have seen numerous macrophages moving across the epithelial cells like vacuum cleaners. By secondary passage they have disappeared, but I would consider them much more likely to bear HIV than the very few lymphocytes present”.

He continues: “Like Hamilton, and unlike Haseltine as reported in Curtis’s article, I believe the origin of human HIV infection is important, as a lesson to prevent further modern, possibly iatrogenic epidemics. Actually, I think the lesson is already made explicit, and that testing the stored vaccine seed samples at the Wistar will not provide an answer. If they are PCR-positive, it will provide a further law-suit, but no compelling evidence that it is the source of the pandemic; if they are PCR-negative, it will leave unanswered³⁸⁵ the possibility of local contamination by chimpanzee tissue in central Africa.”

This is fascinating, not least because it reveals something of the gulf between the public and the non-public person. Professor Weiss is intelligent enough to know that the OPV theory is plausible (not least because chimp tissues may have been used locally to passage the vaccine – and I now realise that he was actually seven years, not two years, ahead of me on this!) He also realises that the testing of the Wistar Institute CHAT samples is likely to reveal very little. (Mind you, what a difference from his comments after the Royal Society meeting, when he expressed “surprise” that I was so dismissive of the Wistar test results, and then described the OPV theory as “contrived” and “fatally weakened”).³⁸⁶ But he is mainly interested in the theory because of the lesson it can teach about preventing possible future man-made epidemics. And that question, he says, has already been answered.

Responsible scientists don’t need to be hit over the head with this, he seems to be saying; we already know that if we’re careless, we can spark iatrogenic disasters. The important thing is that we should learn the lesson for the next time around. And yes, says Weiss, we have indeed learnt it: “the lesson is already made explicit.”

To which I have one word of response. Nonsense!

What Robin the referee has actually done is to move the goalposts, in order to come up with a comfy rationalisation. He starts by saying that to discover the origins of HIV is important, and ends by saying that it will serve little purpose to test the OPV theory, to investigate it further. To my mind, what this really means is: no need to pay the butcher’s bill; let’s just find a new butcher and start with a clean slate.

Robin the arbiter, the judge, seems to have already decided that the theory must be wrong – or, to be more accurate, not worth pursuing. I think he lets Science off far too easily. Most hard lessons in life are *not* acquired through an intellectual process; they are taught through bitter experience. Besides, there is quite a lot of self-interest involved with a stance that neatly absolves his profession, and his scientific colleagues, of responsibility.

What the referee’s letter reveals is that Robin Weiss’ response to this debate has long been influenced by the demands of political expediency.

We may assume that Weiss voted against the publication of Hamilton’s letter. Whatever, it was rejected by *Science*, even after Hamilton wrote a second (and this time, personal) letter to the editor, Daniel Koshland, pleading that the theory deserved a fair and public hearing. A sub-editor replied a few weeks later, acknowledging that Hamilton was “superbly qualified” to comment on this issue, but still declining to publish the letter.³⁸⁷

Later in 1994, Hamilton submitted a slightly stronger version of the original letter to *Nature*, which journal appointed a single referee, and finally rejected it on the grounds that “it doesn’t contain any substantially new revelations”.

I recently discovered, from two different sources (one of whom might be best described as being close to the editorial staff at *Nature*), that Robin Weiss has exercised enormous control over AIDS coverage in that magazine for the better part of twenty years. For most of that time, there has been what amounts to a set response at *Nature* with regard to the more important letters or articles about AIDS. It seems that such submissions are routinely sent to Professor Weiss for refereeing, and that for other submissions he frequently offers advice about who the referee(s) should be.

To my knowledge, at least seven (and probably quite a few more) separate submissions to *Nature* about the OPV theory made from the late eighties to the present have all been rejected, including Bill Hamilton’s 1994 letter. Now it seems that the inherent bias of that journal against OPV/AIDS may actually reflect the inherent bias of Professor Robin Weiss.

Furthermore, it may be that it was Weiss who rejected Hamilton’s letter on behalf of both *Nature* and *Science*.

Robin Weiss is a most powerful and influential man in the field of virology and in the field of AIDS, and I now believe that over a lengthy period of time he has done his best to minimise “ugly” discussion about the OPV theory. However, when open discussion of the theory became inevitable, he was not slow to put himself in a position where he could exercise considerable control.

As the senior co-chair of the Royal Society meeting, Robin was able to alter the guest list and speaking order, to make significant interventions at the press conference, and, most crucially, to deliver the concluding remarks which, he knew, would have such an influence on how neutrals, and members of the press (and, through them, those who did not attend the meeting), came to view the proceedings.

Other, more subtle tinkering was also possible. For instance, in June 2000, Robin wrote to Brian Martin, the only other full speaker on the programme who was widely recognised as being sympathetic to the OPV hypothesis, to say that he was concerned that the discussion meeting would “fall into 2 camps...who will yell at each other but not listen”, and that he hoped Brian would provide a social scientist’s perspective on the debate, “rather than espousing or rejecting the OPV theory”.³⁸⁸ It appears that Robin was not averse to my being the only full speaker openly espousing the OPV theory at the meeting, even if there were many speakers who were known to virulently oppose it.

Increasingly it seems to me that the thing that Robin craves, above all, is the ability to influence, or even better to control, certain of the big debates and high-profile events in science. In this, one sees definite similarities with the behaviour of two other famous scientists: Weiss’s friend and associate, Robert Gallo, and Gallo’s friend and mentor, Hilary Koprowski. Koprowski, Gallo and Weiss are not the only power-

brokers, or politician-scientists, in the history of science. But they do seem to represent a clear lineage.

On the basis of the foregoing information (which has only become known to me in the last few months) I have to say that in retrospect, the chances of a free-and-fair discussion of the origin of AIDS hypothesis were considerably reduced once Robin Weiss had been asked to join the team of organisers. Especially when Bill Hamilton died, a few months later.

c) All about phlogiston.

Here some background is needed. Bill Hamilton quite openly acknowledged that he hated the more political aspects of science, such as the organising of meetings, and so he was keen that someone who was an able organiser (and a member of the Royal Society) should be on the team. He therefore happily accepted Simon Wain-Hobson's suggestion that Robin Weiss should be invited to join them, and left for the Congo in January 2000, secure in the knowledge that by the time he got back, much of the nitty-gritty organisational work would be over and done with.

It turns out that, although they have apparently crossed swords in the distant past (over the Gallo/Montagnier controversy), Simon Wain-Hobson and Robin Weiss have also collaborated in a number of areas. Robin may even see Simon as his natural successor, as both an eminent scientist and as a power-broker.

An interesting example of a Weiss/Wain-Hobson collaboration is a letter that appeared in *Nature* just a couple of weeks after the Royal Society conference. Entitled "If free speech costs lives that's a high price to pay", it was predominantly a riposte to Peter Duesberg's contention that HIV does not cause AIDS.³⁸⁹ It could, however, have been read as a wry commentary on other debates as well.

It was an amusing letter, and featured the following: "We are staunch believers in the right to free speech, but is *Nature* the appropriate place to militate in favour of the pre-Copernican model of the universe, or the existence of phlogiston?"

I am certainly no fan of the Duesberg theory. However, in the light of the Royal Society debacle and its aftermath, this particular pronouncement by the two surviving organisers of the Royal Society meeting now has a rather different ring to my ears. It strikes me that a little dephlogistication³⁹⁰ might be in order – or at the very least a reduction in hot air.

It gives me no pleasure to have to write about Simon Wain-Hobson's role in this debate, not least because when it comes to apes and angels, Disraeli sided with the angels, and Darwin with the apes, but Simon appears to have sided with both. By which I mean that he started out very much as an open-minded, free-thinking independent but that, when it came to the crunch, he was (perhaps understandably) loath to cut his links with colleagues in the scientific mainstream. For some time thereafter, he tried to please both camps. Perhaps it wasn't a very satisfactory compromise, for in this debate at least, it'd hard to please everyone. So he ended up back with the people he knows best – his scientific peers.

Back in September 1999, after publication of *The River*, Simon had written a powerful review in *Nature Medicine* in which he called on fellow scientists (in the words of Oliver Cromwell) to “think it possible you may be mistaken” about the OPV theory.³⁹¹ Over the next five or six months, he played what I consider a heroic role in investigating the theory. It was he who first suggested to Bill Hamilton that he should ask the Royal Society to convene a conference to look into how AIDS might have started. And it was he who made a search of the Pasteur Institute archives, and who interviewed a dozen or more of the former doctors and technicians who used to work for the Pasteur satellites in places like Brazzaville and Libreville in French Equatorial Africa, discovering (among other things) that doctors in those places had “grown polio” in the tissues of local primates in the 1950s. Later, in December 1999, Simon flew to England at his own expense, and shared his interview notes with me, generously adding that I could include these details in the new postscript which I was then about to write for the paperback version of *The River*.³⁹²

Simon had also followed up in other archives, and he told me about how military doctors had approached public health care in the French colonies – how they would load a doctor, a couple of nurses and some Africans into a jeep, and then go out and “vaccinate everything...cows, people, everything.” “It’s absolutely outrageous” he went on; “I’m tempted to say it’s the tip of the iceberg”. He told me that at the Royal Society meeting, in addition to reporting on the Hamilton chimp samples, he would have to present this new information about French polio vaccine research in Africa. “I have no choice”, he said. It was a courageous and honourable decision, and I was impressed.

In early 2000, the CHAT vaccine samples from the Wistar were being prepared for independent testing, and Wain-Hobon’s lab was invited to be one of those which participated. The fact that he personally would be involved with the testing process reassured me greatly, for I had been concerned that the testing process might turn out to be simply a public relations exercise for the Wistar. (It was obvious that, if they wanted to, the Wistar scientists had had both the time and opportunity to check the samples themselves, and I felt that they only had to send out samples which had been prepared in a non-chimpanzee substrate, and then offer this up as an easy “disproof” of the OPV theory.) Simon argued that it had to be assumed that everyone was acting in good faith, and that it was important to do the tests. However, he also reassured me that no “serious scientist” was going to believe that a negative result disproved the theory.

By March 2000, the proposed conference was causing a furore in AIDS circles, and Simon was coming under increasing pressure from fellow-scientists. Some had become noticeably cool towards him, others had snubbed him, while one (Dr John P. Moore again) had written him vitriolic and abusive letters. Meanwhile, members of the National Academy of Sciences (including one Nobelist) had apparently written to the Royal Society complaining that the London conference would cause untold damage; Beatrice Hahn and Bette Korber had simultaneously withdrawn, claiming that the mooted list of speakers was not balanced; and Stanley Plotkin was hinting that he might do the same. Then, on March 7th, Bill Hamilton, who had been in a coma for five weeks after his return from the Congo, quietly died.

The next day I had to be in Paris, and I met up with Simon. He assured me that he and Robin now bore “a heavy responsibility” to honour Bill’s memory, and to ensure that a free-and-fair debate took place.

However, at around this point, things started changing. I sent Simon a copy of the passage about his work in my new postscript, for comment. He made a couple of small suggestions, and gave it his approval, but I sensed he was becoming nervous. A few more days passed, and then Simon did something I had always feared he might do. He began to give in to the pressure.

Suddenly the London meeting was postponed from May to September, nominally to allow different labs time to test the CHAT samples released by the Wistar, but also to allow Simon time to test Bill Hamilton’s chimp samples from the Congo. I and a few others were opposed to the postponement, which we feared was part of a creeping takeover of the conference by those who were profoundly opposed to the OPV theory. Simon assured me he had not been involved in the decision to postpone, but almost immediately I was informed otherwise by two different sources at the Royal Society.

The next time I phoned him, he said that he and Robin were busy organising a letter of opposition to Peter Duesberg, which was to be signed by a group of eminent scientists, and published in *Nature*.³⁹³ As sympathetic as I was to that particular crusade, I was also concerned that Wain-Hobson seemed to be becoming rather less open-minded about OPV, and that he and Robin Weiss appeared to be suddenly getting very cosy together. From that point on, communications between Simon and myself slowed, and then ground to a halt.

Contrary to his previous promise, he made no direct scientific contribution to the Royal Society meeting, although he did provide one brief answer from the floor to a question from Stanley Plotkin about the Hamilton chimp samples, saying that there didn’t appear to be any evidence of SIV infection therein. He said nothing to me at the meeting itself, and when I phoned him a few days afterwards to ask how he thought things had gone, he replied that he now found the OPV theory less plausible. He told me that he still found the phylogenetic dating arguments of Sharp and Korber unconvincing, but that Plotkin had seemed persuasive, while (from a scientific point-of-view) he had been impressed by the Martine Peeters dataset from the DRC. He said that Peeters had demonstrated a large number of Group M variants in the DRC, and claimed that some of them were so deep-rooted that it would require not just half a dozen transfers from chimps to humans, but perhaps ten times that number, for the Gerry Myers version of the OPV theory (involving multiple and near-simultaneous transfers from ape to human) to work.

It was an interesting response, and over the next few months, I kept an open mind about it, and about the significance of the Peeters dataset. I now believe, however, that Wain-Hobson’s analysis is based on false premises. The Peeters DRC sequences ably demonstrate that the Group M hearth is situated (as I have long insisted) in the DRC, but the “cloud of variants” could actually have been produced by just two chimp SIVs which recombined early in the epidemic, as demonstrated by Mikkel Schierup.

My feeling now is that Simon Wain-Hobson was uncomfortable about his changing stance in the origins debate, and his failure to make the contributions he had promised

to make at the Royal Society meeting. And I feel that he needed to some extent to rationalise his new position. In the last twenty months, I have sent him five formal e-mail requests, asking him for details about the testing of the Congo chimp samples collected by Bill Hamilton in July 1999 and January 2000 (on the first of which studies I was a collaborator; and to the second of which I provided some assistance). Although he has replied to the e-mails briefly (and often cryptically), he has never given any response about the testing process, or what it has revealed.

The importance of these samples, especially those which were obtained from chimps in the wild, hardly needs to be emphasised. Wain-Hobson's refusal to provide any further information about the samples (even on a confidential basis), which reneges on a clear verbal agreement with Bill Hamilton and myself, is all the more worrying in that, in his last statement to me on this subject in mid-2000, he revealed that his team had found some interesting non-SIV viruses therein.

Meanwhile, Simon has begun suggesting to others that they distance themselves from me. He wrote to Walter Nelson-Rees, the man who revealed that many of the world's tissue cultures were in fact HeLa contaminations, advising him: "Don't nail or couple your story to Hooper's. You are very different people."³⁹⁴ Nelson-Rees sent a copy straight to me, proclaiming the letter "impertinent and foolish".

d) Monkeying around at the Royal Society.

But back to the Royal Society meeting. Perhaps I was naïve to hope for a free and fair hearing. Certainly I hadn't at all thought through what I would do if the whole business started to get dirty.

Without doubt an effort had been made to provide some balance, not least because of Simon Wain-Hobson's efforts in the early days of the organising. There were speakers like Tom Burr (from Gerry Myers' lab) and Pascal Gagneux who contributed information and analysis which lent real support to the OPV reading of events, as well as one speaker (Brian Martin) who was overtly sympathetic.³⁹⁵ But in the key sessions (the first afternoon session which discussed theories of origin, and the closing session, when Robin Weiss gave his summarising speech) matters were arranged very efficiently so that the OPV theory could be (or would appear to be) neatly "disposed of".

Brian Martin has published his analysis of what happened there,³⁹⁶ and I have yet to decide when and where to publish my own account. But for now, let me just note a few of the more disturbing occurrences:

- Contrary to Dr Weiss's account of events, two additional speakers *were* invited to the rescheduled September meeting, both of whom (Hilary Koprowski and Paul Sharp) were virulently opposed to the OPV argument. One of these, Paul Sharp, had apparently been invited because his long-time colleague, Beatrice Hahn, had insisted that he had very different material to present from herself. Yet in the end, they submitted a joint paper to the Proceedings of the meeting. The net effect of having all three of the leading natural transfer proponents – Beatrice Hahn, Paul Sharp and Bette Korber – as speakers was to weigh the meeting inexorably in

favour of their theories about a west central African hearth, and an epidemic which could be “sourced” phylogenetically to the 1930s.

- Despite the death of Bill Hamilton, the OPV camp was not allowed any further speakers. (Bill had been tentatively scheduled to open the conference, but not to deliver a formal address after that. However, there is little doubt that, despite his natural shyness at meetings, he would have made a significant contribution on behalf of a theory about which he was “95% persuaded”).³⁹⁷ In particular, I repeatedly asked Robin Weiss for another epidemiologist to be invited to speak about the coincidence between the vaccination sites and the first appearances of HIV, to balance what I suspected might be a one-sided epidemiological presentation from Kevin De Cock. He refused.³⁹⁸
- The Monday afternoon session, which was apparently rejigged at the last minute, was set up in such a way that my own speech was followed in short order by a series of “denials” from doctors Plotkin and Koprowski, Claudio Basilico with the Wistar test results, then by the press conference, and after that with further denials by doctors John Beale and Paul Osterrieth.
- At the press conference, where each speaker had been allotted three minutes to present his or her case, Dr Weiss twice interrupted me to tell me what I could and could not say. On the second occasion he was shouted down by a reporter, who told him to let me speak.
- Dr Weiss’s closing speech, which bore little resemblance to the carefully-crafted version which later appeared in the Proceedings, was blatantly prejudiced against the OPV hypothesis, praising each of the “scientific” speakers, but gently and persistently denigrating my arguments. At one point, Weiss admitted that his speech was just his “personal biased view....plain, personal prejudice”, but this passage did not appear in the written version. Many people contacted me after the conference to express their disquiet (or in some instances disgust) at the way I had been treated, and in particular at the performance by Robin Weiss. When I mentioned this in a newspaper interview, Weiss wrote to me asking for their names, so that he could send them a copy of his speech. I declined.
- Although a video camera filmed the proceedings to relay them to an overflow hall, I was later informed that (contrary to previous information) no video copies had been made. Later, when I asked for copies of the audio tapes of the meeting, I was told that the tapes were the “intellectual property of the Royal Society”. Apparently the Royal Society was willing to host a conference on origins, but wanted the precise details of what had been said at that conference to remain confidential.

For me, the most disturbing aspect of the meeting related to the presentations by Stanley Plotkin and Hilary Koprowski. Their support team apparently included Dr John P. Moore,³⁹⁹ a Dutch researcher (Dirk Teuwen) who had taken six months off from Plotkin’s lab to contact many of the witnesses whom I had interviewed, and several Belgian doctors from the colonial era, some of whom had been directly involved with the CHAT research. In addition, Plotkin’s party let it be known that “lawyers” were present, one of whom, according to a report that later appeared in the American press, left saying that he would shortly have work to do. The team had clearly decided that above all they needed to win the public relations battle, and that their best defence was attack.

At the press conference, they issued three press releases (one each from Koprowski and Plotkin, and a “backgrounder” from Plotkin). Each of these contained untruths, and was littered with examples of misrepresentation, error and spin. The intention, it seemed, was to give the impression of authority to the gathered press, and in this the Plotkin group largely succeeded. I have already made a point-by-point response to these highly inaccurate press releases on Brian Martin’s web-site.⁴⁰⁰

The speech by Dr Plotkin, by contrast, was a carefully crafted piece of spin. This was a much more professional presentation, but once again it relied on misrepresentation, inaccuracy, and untruth. Plotkin had been unable to find more than one or two errors – or possible errors – in the whole of *The River*, but he focussed heavily on these, claiming they were “key points”.⁴⁰¹

He stated that the purpose of Lindi camp was “not at all mysterious”. However, he still failed to provide any but the vaguest of details of the research conducted there, these details being copied from the sources already quoted in my book.

In this, and in his subsequent postscript, Plotkin employed classic disinformation techniques.

There was the harping on trivial points, while failing to address many of the key issues raised in the book (some of which I have raised again in this paper).

There was very little in the way of new and substantive information, and much of what there was was sourced to private papers or signed statements, which were not made available for public viewing.⁴⁰² The dubious methods used to obtain some of these statements are discussed in more detail below.

Most notably of all, there was the complete failure to explain what had really happened in Stanleyville and at Lindi. In particular, there was no reference to the fact that Koprowski’s vaccines had been amplified in locally-available tissue cultures.

e) Information and disinformation.

Apart from this, Dr Plotkin largely concentrated on attempts to discredit the theory and myself. I have referred above to his reliance on signed statements, and there are various indications that some, if not all, of these were obtained by sending out prepared letters to witnesses and inviting them to sign at the bottom. (One example has already been cited, where both Osterrieth and Ninane are said to have stated exactly the same words: “I never tried to dilute the polio vaccine that was received.”)

There is evidence to indicate that at least some of the supporting statements used by Plotkin in his speeches were obtained by questionable means. Because this is informative about the way that Plotkin’s team have prepared their case, I shall cite three examples in detail.

In or around February, 2000, Dr Gaston Ninane was visited by Dr Koprowski, Dr Prinzie, and one other doctor (who may possibly have been Dr Plotkin). Although doctors Koprowski and Plotkin have recently referred to Dr Ninane as a friend and colleague, I am told that until this approach, neither of them had been in contact with

him for the previous forty years. According to his sister, Dr Ninane had at the time of the doctors' visit been in hospital recovering from a serious fall caused by Parkinson's disease, and was just a few weeks away from a second fall that would prove to be fatal. These, apparently, were the circumstances under which Dr Ninane signed a statement for the doctors in which, inter alia, he claimed that the statements attributed to him in *The River* about his having tried (and failed) to make tissue cultures in Stanleyville "are false and are lies".

Dr Ninane's alleged claims on this point are incorrect: I have checked the relevant tape and transcript, and I have also checked my various notebooks. I can confirm that Dr Ninane said exactly what I stated in the book, and that he talked about his attempts to make tissue culture on three separate occasions, in the course of two interviews. On one of these occasions he stated that he had tried to make tissue culture from chimpanzees. The first, lengthy interview (conducted in 1993) was recorded, the second (a brief phone interview conducted in 1997) was not. As stated earlier, I am willing to play the two relevant sections of the tape recording to doctors Plotkin and Koprowski, in order to prove that Dr Ninane was correctly quoted. If they do decide to take me up on this offer, then I believe that the honourable thing for them to do would be to issue a public retraction thereafter.

The second example involves the former sanitary agent from Ruanda-Urundi, Hubert Caubergh. Early in 2000 he was apparently twice approached by Dr Abel Prinzie, a man who had formerly spoken quite frankly with me, but who had now become one of the most dedicated members of Dr Plotkin's support team. On each occasion, Prinzie enclosed a prepared letter that included claims that statements attributed to Caubergh in *The River* were false. At the bottom of each letter Caubergh's name had been pencilled in, showing where he was expected to sign. He was being invited, in effect, to falsify his evidence. Caubergh was half-indignant, half-amused, and refused to cooperate. Later, he confirmed to me that I had quoted him correctly in the book, and said that Prinzie's approaches had constituted a "dishonourable proposition". Since that time, Mr Caubergh says he keeps hearing from Plotkin's researcher, Dirk Teuwen, who sends him clippings and friendly messages in the post, presumably in an effort to keep open the lines of communication.

The third example is more complicated, and involves the Hungarian, Louis Bugyaki, who headed the veterinary lab in Stanleyville (and helped out at Lindi) in the late fifties. I had already interviewed Dr Bugyaki twice, in 1994 and 1996, and in August 2000 I once again interviewed him at his home in Brussels. He was as charming as ever, and once again repeated on tape his recollections of Lindi camp, and the fact that he had been told by doctors Ninane and Osterrieth that kidneys had been extracted from the chimps and sent to America. (The only difference was that earlier he had said that Dr Courtois was also involved.) However, this time he made an additional comment – that perhaps the use of chimp kidneys had been a commercial secret which Dr Koprowski wanted to keep from competitors, like Sabin and Salk. Apart from these minor details, Dr Bugyaki's testimony matched those he had given me four and six years earlier in all its significant points.

That evening, I decided that if Dr Plotkin's team was getting signed statements (as I had just discovered that they had done from Dr Ninane, shortly before his death), then perhaps I should do the same. I transcribed the tape, compiled a statement based on

Bugyaki's latest testimony, and the following day took a French and an English version to show him. We read them through together, and he was happy to sign both statements.

I presented Dr Bugyaki's statement as part of my speech at the London conference, and was surprised to learn that Dr Plotkin's team had apparently obtained a conflicting statement from Dr Bugyaki in February 2000, six months before my third interview. Soon after the Royal Society conference, Dr Bugyaki was telephoned, and asked if he could clarify the situation. He explained that at some point he had been visited at his apartment by five or six persons, probably doctors. Later, he was apparently called in to one of the institutes in Brussels, where a senior official told him that he was not happy with Dr Bugyaki's statements on this issue.

At this point in the phone conversation, Dr Bugyaki became upset. Now, for the first time in six years and four interviews, he suddenly gave a different version of events. Now he said that the person he had heard about all this from was not Osterrieth or Ninane (as he had clearly told me on three separate occasions), but Jean Brakel, a sanitary agent who was now dead. This is virtually the same version of events which he had apparently signed for Plotkin's team in February 2000, *but about which – tellingly – he had made no mention when I visited him six months later, in August*. Apparently he had since been reminded of it (presumably by the group of five or six visitors, or by the senior doctor at the institute).

It seems that a few days after this phone conversation, in November 2000, Dr Plotkin's team obtained a further statement from Dr Bugyaki which, they claim, was instigated at his request. Plotkin also claims that Dr Bugyaki complained that he had been misquoted by me. However, I have the tapes to prove that this allegation, like many of Dr Plotkin's other allegations, is false – and, as stated earlier, I am willing to play the relevant passages of these tapes to Dr Plotkin to demonstrate that fact. I have quoted Dr Bugyaki accurately throughout, just as I have quoted others accurately.

Dr Bugyaki gave me very clear statements on three occasions. I believe that he is a kindly old man who tried his best to help the investigation, but who has now been pressurised into adapting his account by a number of medical colleagues, including at least one senior figure in the Belgian medical establishment.

These three accounts of approaches made by members of the Plotkin team (plus their various support groups) suggest something of a pattern in the way that they have attempted to refute the evidence presented in *The River*. Visits have been made (sometimes by quite large teams) to elderly doctors who have previously given interviews to me. I believe that at some, at least, of these meetings, subtle pressures were brought to bear, and that under these circumstances, some witnesses were willing (even relieved, I suspect) to sign the prepared letter that followed in the post. I am fortunate that one man, at least, Hubert Caubergh, was not prepared to bow to such pressures.

The tactic, at least in these three instances, seems to have been to try at all costs to discredit the evidence that I have gathered, and (if possible) to discredit me also. If that failed, then the secondary tactic was to obfuscate the issues. Such an approach is

not especially original. I am told these are classic disinformation techniques practised by different intelligence agencies around the world.

f) A rocky road.

At the end of his concluding remarks to the Royal Society conference, Robin Weiss made much of how difficult it had been for him and Simon to organise the conference. “It wasn’t always easy”, he said; it had been a “pretty rocky road”. He thanked Simon “for helping me to carry on”.⁴⁰³

At that moment, I didn’t have very good perspective on what was happening, but I was just beginning to get the sense that the meeting had gone through the motions of having a free-and-fair debate, but that those in control had apparently made up their minds beforehand about who was to win, and who was to get their comeuppance. It was only later, when I began to get feedback from others, that the evidence for this began to accumulate.

However, I was brought back to basics by a question that a reporter asked me soon after the closing session. “Do you think it would have gone the same way if Bill Hamilton had been alive?”, she asked. I hadn’t thought about it until then, but the answer was obvious. No, it wouldn’t.

Seven months later, in April 2001, *Nature* and *Science* got together to present a collectively stony face against the OPV theory. Three scientific teams (including one led by Wain-Hobson) reported no evidence of finding HIV, SIV or chimp DNA in samples of CHAT. I had no problem with these undoubtedly accurate reports of the testing of the samples that the Wistar Institute had chosen to release. However, in the accompanying commentary which Robin Weiss wrote for *Nature*, he claimed that CHAT 10A-11 and 13 were “batches”, not pools (thus obfuscating the key issue about exactly what had been tested), and then ended with his famous statement about facts and theories, beauty and ugliness. Misleadingly, the article was titled “Polio vaccines exonerated”, as if I had been questioning the safety of *all* polio vaccines.

Other articles about the testing on *Nature*’s web-site were headed “Origins of HIV: polio vaccine cleared”, and “Polio researcher innocent of HIV pandemic”. Meanwhile in *Science*, Jon Cohen gave his (very similar) views, in an article apparently inspired by the Munchkins, and entitled “Disputed AIDS Theory Dies its Final Death”.⁴⁰⁴

But perhaps the most significant event occurred a couple of months later, when the proceedings of the Royal Society meeting, edited by doctors Weiss and Wain-Hobson, were published. Despite the point-by-point refutation of the press releases by Plotkin and Koprowski which I had posted on Brian Martin’s web-site, and despite the clear statements I had made at the Royal Society meeting (and its press conference) about the questionable approaches made by members of Dr Plotkin’s team, I now found that he had been afforded an additional five-page “Postscript” to reply to “new allegations made by Edward Hooper at the Royal Society conference”.

Many people, including myself, felt that the editors’ decision to provide a further platform for Dr Plotkin’s version of events provided more information about the editors than about the origins debate.⁴⁰⁵

A detailed response to both of Plotkin's Royal Society articles, and perhaps to other related matters, will be posted in due course on Brian Martin's web-site.⁴⁰⁶

g) Who polices the police?

As Bill Hamilton stated in his powerful foreword to *The River*, "When eminent rivals in an ancient profession are seen to be uniting to crush an outside critique [the OPV theory], and when the best-funded branch of science, to which the rivals belong, draws almost all its practitioners into line behind them...then it is time for the rest of us to wake up....."

"In the same vein and equally unsettling, we have seen the best known and seemingly most independent science and medical journals join forces on the side of the countercritique, without publishing details of the original issue. Again, it is time for us to wake up and consider what is happening to freedom of discussion and to the spirit of science."⁴⁰⁷

Despite my enormous admiration for Bill, I have on occasions in the past accused him of political naïveté. With this foreword, however, he was clearly well ahead of me in appreciating how the scientific community was responding (and would continue to respond) to the OPV hypothesis. Indeed, the events he described were to happen all over again within months of his death.

I am not alone in believing that in many ways Bill Hamilton's foreword to *The River* constituted his scientific epitaph, a timely farewell warning to his fellow-scientists. Robin Weiss, by contrast, apparently "didn't like Bill's preface to *The River* one bit". In fact, the expression he used to describe it to me (and, it seems, to others) was both pungent and dismissive.

The fact that the origins-of-AIDS debate has become so politicised is not solely because scientists and governments fear that a proven theory of iatrogenic origin might engender damage claims and law-suits. Neither is it solely because some believe the theory might have "detrimental effects on vaccination programmes in general", as Kevin De Cock puts it.

The natural transfer theory is innately more acceptable to the scientific community than the OPV theory for many other reasons. One that has often been asserted in recent articles about new SIV discoveries, is that if SIV infection can be readily acquired from handling wild primates, or from the eating of bush-meat, then we may see further AIDS epidemics caused by an HIV-3 or an HIV-4. The only protection, imply scientists like Beatrice Hahn, and science writers like Jon Cohen, is for scientists to be on the ground in a state of alertness, ready to tackle the next potential epidemic before it gets out of hand.

I am sceptical about such claims. Alertness on the ground can be mightily effective when it comes to responding to highly virulent and infectious organisms like Ebola virus. But there is still no proof that casual exposures to primate SIV through keeping monkeys as pets, or through bush-meat butchery or consumption, actually lead to pathogenic infections, to human AIDS.

It should be borne in mind that the natural transfer argument ties in rather neatly with the new and fashionable agenda of “emerging infectious diseases”, which paints a lurid picture of pathogens lurking out there in the rain forest, waiting to get you, unless Western scientists can save the day. This is an agenda that has been popularised by writers such as Laurie Garrett and Richard Preston, and it is one that tends to go down well with virologists and microbiologists, for fairly obvious reasons.

Certainly, as world travel increases and as boundaries shrink, pathogens are invading new niches, and can cause new virgin soil epidemics. Nobody is denying this. But let us also not forget that there are other potential agendas here as well. Emerging infectious diseases are preoccupations not only of hygiene specialists and public health officials with the most genuine of concerns for human health, but also of military scientists, some of whom like nothing better than having a few new pathogens to play with.

New pathogens can of course be modified by decreasing their pathogenicity (allowing the development of attenuated vaccines) or by increasing infectivity and pathogenicity (useful if one’s business is the development of biological weapons).

I wholeheartedly agree that research in tropical environments (into viruses such as the SIVs) is important, and that it has increasing relevance for modern, global public health programmes. However, let us retain some balance, and remember that such research can also be put to wrongful and devious ends (as, indeed, it has all too often in the past). Over the last sixty or so years, the problem has not simply been one of rural Africans and Western backpackers eating “the wrong foods”, or entering the wrong caves. It has also sometimes been one of Western (and Soviet) scientists carrying out irresponsible and immoral research.

So as the British prime minister calls for more trust to be placed in scientists,⁴⁰⁸ I believe that it is actually a different clarion call that should be going out on the airwaves. To my mind, the way that the origins-of-AIDS debate has been conducted has raised serious concerns about the judgment and impartiality of certain scientists, and about the way that some respected scientific institutions conduct their debates.

The interventions which science and biotechnology are capable of making grow ever more impressive, and ever more worrying. Many observers believe that the ethical and moral checks and balances that are currently in place are unable to keep pace with technical advances – and that existing organisations like the WHO do not, in real terms, have the capacity to take a strong and independent stance on such issues.

I believe that a new global organisation needs to be established, one that has the power and authority to oversee and, if necessary, modify, the way that scientific research is conducted, the better to ensure that that noble Hippocratic oath of “First, do no harm” is properly observed.

The nuts and bolts of how such a body might be established would clearly be a subject for debate, but I believe that representatives not only from the fields of science and medicine, but also from fields as diverse as the sociology of science, philosophy, history, and the media, should all be considered for inclusion.

Should scientists be left to “police” themselves? If nothing else, the origins-of-AIDS debate has illustrated that this question can no longer be confidently answered in the affirmative.

h) Legal moves.

One of the less agreeable repercussions of the Royal Society meeting became apparent about two months later, when I received a three page letter from Professor Hilary Koprowski. In this, he claimed that the OPV theory had been refuted by “overwhelming evidence” that had been presented at the London conference, and invited me to withdraw *The River* from bookshops. I wrote back saying it was my belief that nothing had been refuted, and providing yet further scientific arguments to counter his claim. A week or so later, I received a letter from his London lawyers, claiming that I was now asserting the OPV theory to be a fact, not a theory, and threatening me with legal action.

It will be remembered that in the past, some of those who have questioned Dr Koprowski’s actions have elected to withdraw, or to issue “clarifications”, after Koprowski has initiated legal action against them. This was the third time I personally had been sent threatening letters by lawyers representing Koprowski; on one of which occasions the lawyer has also been acting for Dr Plotkin. These lawyers have also approached my American publishers, who rejected their demands to see all text relating to Dr Koprowski prior to publication of *The River*.

In their letter to me, Koprowski’s lawyers stated that although their client “could sue, and indeed that may be his only option”, he instead offered me an alternative. He proposed that “[my] OPV/AIDS claim be investigated by a panel chaired by a lawyer and flanked by scientists. The investigation would have the character of a judicial enquiry, and would be followed by an adjudication”.

This was new to me and to my UK publishers: we had never heard of a quasi-judicial panel sitting in judgement on a scientific hypothesis before. None the less, a robust letter of reply was sent, asking for more details. Who would decide who sat on the panel? Who would pay for it? What rules of evidence would apply? That was a year and a half ago.⁴⁰⁹ We have heard nothing more since.

None the less, the questions we asked remain valid. When it comes to judging science and the work of scientists, who gets to sit on “the panel”? Who pays for the process? And underlying all that, who polices the police?

The preceding information has never been revealed before, largely because the other party deemed it to be “strictly private and confidential”. (I have to say that I find it unacceptable to receive a threat, or an implied threat, and then to be told that I must keep quiet about it.)

But the question raised by Koprowski is an interesting one. Two years ago, I happened to meet John Maddox, the former editor of *Nature*, and had the chance to speak with him for a few minutes about *The River*. Rather to my surprise, he told me he thought I had “proposed a plausible hypothesis. It would take 30 million over three

years to investigate it properly.”⁴¹⁰ Now, perhaps Dr Maddox was talking *yen, or lire*, and having a little joke. Or perhaps he was just making small talk. But I got the feeling that he was being sincere, even if he didn’t say who should cough up the cash.

Meanwhile, that proposition of Koprowski’s has got me thinking. A quasi-judicial medico-legal panel, eh? An interesting idea. I’ll have to get back to that one.

i) Knowing “when to call it a day”.

In a phone conversation some weeks before the Royal Society meeting, Robin Weiss warned me that I’d better be on my “best behaviour” at the conference – a statement that to my mind, revealed something about the role that Robin had already assumed (at least in his own mind) in terms of “policing” the debate.

A few months before that, Simon Wain-Hobson had given different advice. I had been contacted by a leading member of an AIDS activist organisation who had read *The River*, and who wanted to make a splash when I visited America, with demonstrations and the like. Again, this activist was a strong-willed person, and someone who felt that I needed to be channelled along certain lines. I asked Simon what he thought, and he strongly advised against getting involved. He thought there would be very little chance of Science treating the origins issue seriously, and conducting a free-and-fair debate, if it had been on the news bulletins the night before, with people in masks chanting slogans.

In different ways, I suppose that I followed both of these pieces of advice. For the record, I think that I did behave rather well in London (although not everyone followed suit). And what happened? What we got was not the free-and-fair assessment that I’d been promised, but rather a manipulated process, and a biased “verdict” which, none the less, I was apparently expected to accept with good grace.

Although I had a sense of unfairness at the time, it took me many months before enough evidence was in for me to be convinced that the cards had been stacked from the outset. And as more months go by, I’m ever more certain that the official scientific investigation into how AIDS might have started has been inherently tainted.

The preoccupation of the organisers seems to have been to protect the scientific *status quo* at all costs. Perhaps they felt that they were acting “for the greater good” by protecting the reputation of vaccination programmes. Or perhaps they had other motivations.

But their positions continue to be entrenched. At one point during the Lincei conference, Robin Weiss told me that I should be proud, for there are not many scientists who have prompted two scientific meetings by something which they’ve written. On one level, of course, this is a charming compliment. But I believe there is also an unspoken subtext. What he is really saying, I believe, is “Look, Ed, you’ve been invited to conferences not once, but twice, and you’ve had the chance to present your ideas. It’s not your fault that they’ve been dismissed by the scientific community. At least you have been heard. Let nobody say that we aren’t willing to listen to dissenting views.”

This reading was confirmed a few weeks after Lincei, when I read Robin's latest article, "Reflecting on the origin of human immunodeficiency viruses", which he posted on Brian Martin's web-site.⁴¹¹ The paper ends with the following sentence: "So we can thank investigative writers like [Tom] Curtis and Hooper for shaking the medical establishment's complacency, but they should recognise when to call it a day."

Let me be frank. I find Dr Weiss' analysis both condescending and partisan, for it was he, in particular, who helped *deny* the OPV theory the chance of a free-and-fair debate at the Royal Society. In all its major and most controversial aspects, the London meeting was carefully controlled, so that in the end a version of events that was acceptable to the scientific mainstream (and to its "accused" representatives) was presented to the audience and the press, and later enshrined in the medical literature.

And in public statements since the meeting, Weiss has used the fact that the meeting was staged, and his own role as organiser, to "legitimise" a series of unscientific claims that the OPV theory has been disproved and discredited.

This is exactly the sort of inversion of true science that Bill Hamilton warned about in his foreword to *The River*.

It's now sixteen years since I started working on AIDS. At the end of the Royal Society meeting, I was fully prepared to thank the scientific community for its honest engagement with a difficult problem, and to announce that I was now withdrawing from the debate, and would make available the materials I had collected to interested parties. Unfortunately, I never had the chance to make that announcement.

Instead, I am still involved. And until Science gets its house in order, and stops the attempted cover-up on this issue, then I'm afraid that I shall have to disappoint Dr Weiss. For until that happens, I won't be "call[ing] it a day".

j) Phantom science.

Unravelling what really happened in the past, especially in an area as controversial as this one, is a painstaking process, one that involves careful interviewing of the protagonists, examining of the published evidence, and the trawling of archives for forgotten details.

Over the last three years, many claims have been made about *The River*, some positive and some negative, and some of which have been simply untrue. The criticism which I have found most galling (or amusing, depending on mood) is the one which has been made by doctors Plotkin and Koprowski, among others – that I have looked only at the evidence which supports OPV, and ignored the other side. (This was even proposed by John Maynard Smith, in what was clearly a planned "last question from the floor" to close the Royal Society meeting. Interestingly, I heard from elsewhere that Professor Smith had not actually read *The River* at that stage.)

I believe that anyone who has read the book with an open mind should know that that claim is untrue. In fact, precisely because I am a non-scientist, a non-expert, an amateur, I have worked hard to avoid jumping to premature conclusions. Whenever a

new piece of apparently supportive evidence has come in, I have first of all acted as devil's advocate. I have contacted scientists from appropriate fields, to sample their views. And unless I have been certain that something is sound, I have always erred on the side of caution. My American and British publishers, too, have played an important role here, with the very careful scientific and legal readings which the book was given before publication. Not surprisingly, they wanted to be certain that I was able to justify everything I'd written.

Perhaps the best evidence that I have not bent the facts to the theory, or rushed to premature judgement, would be the thrice-given testimony given by Gaston Ninane in 1992, that the Koprowski vaccines had been made in "chimpanzee kidney cells". After I told Dr Ninane that the scientific literature at the time had mentioned only "monkey kidney cells" he suddenly retracted, saying that this was what he had intended to say – and I accepted that he had made a mistake. It would have been far easier, had I been intent on forcing the issue, simply to quote the words that he had said, and to ignore the subsequent denial. In the end, it took nine years of further research before I was finally convinced that what he had originally told me *was* the truth.

But what of "untruths" told by the other side? There are quite a few examples of these to be found in *The River*, and others that can be discerned in the rivulets that run through the endnotes, or between the lines. One of the more obvious examples involved Koprowski's repeated claims in the literature that he was making his OPVs in chick embryo, and his thrice denying the use of primate kidney tissue culture.⁴¹²

Others examples are still coming to light. One relates to the "vaccinations in response to epidemics" which were staged in *Province Oriental* in January and February 1958. I have recently reviewed three different accounts of these outbreaks (one by Koprowski,⁴¹³ one by Courtois,⁴¹⁴ and the third by Wilfrid Bervoets, a Congo-based government inspector of hygiene),⁴¹⁵ and realised that they offer comprehensively conflicting versions of (a) the number of persons who tested positive for Type 1 polio antibodies before it was decided to initiate the "anti-epidemic" vaccinations, and (b) the number of polio cases that occurred among both vaccinees and non-vaccinees after the vaccinations with CHAT.

The evidence strongly suggests that these vaccinations were not primarily staged in response to epidemic outbreaks of polio, as claimed, but rather because a new batch or batches of CHAT pools 8 and 9 vaccine had been prepared, and needed to be field-tested. In reality, what may have been a serious polio outbreak (that at Bambesa) was ignored for a month, while the vaccine was instead transported to two large military camps, where the majority of the vaccinations were carried out. (Over the years, military camps have frequently been viewed as good testing-grounds for new biomedical materials.) When the vaccine finally did arrive in Bambesa, there was not enough to go around, and only a part of the village was vaccinated.

Koprowski, however, reported that in Bambesa as elsewhere, "every inhabitant received the vaccine". He also reported that "after vaccination, no more cases of paralysis were reported in the four localities involved in the outbreaks". In fact, the hygiene inspector's letter reveals that more than 17 cases were reported in these locales in the following months, including at least one in each of the four villages.

Bervoets reveals that 1,500 further people were vaccinated in the mining township of Kilo at the same time as the Watsa vaccination – and that Kilo experienced twenty further polio cases between April and July. (None of this is mentioned by Koprowski.) The likeliest explanation is that the vaccine was reverting to virulence, which seems to have been a regular problem with Koprowski vaccines over the years.⁴¹⁶

It is, I believe, inconceivable that neither Koprowski nor Courtois were informed about any of the 17 post-vaccination polio cases before they wrote the reports which announced to the world that in the first large-scale field-trials of oral polio vaccine anywhere on the planet, CHAT vaccine had been an unqualified success.⁴¹⁷ [For further details, see Figure 7]

Figure 7: Different accounts of the “vaccinations in response to epidemics” in Province Oriental of the Belgian Congo, January – February, 1958, and of the post-vaccination polio cases seen in the same towns and villages.

Town or village where polio outbreak occurred	Numbers of suspected polio cases pre-vaccination (B)	Dates of epidemic outbreak	Dates when CHAT vaccine given	Numbers vaccinated with CHAT (Koprowski) (D)	Polio cases more than 4 days after vaccination (Bervoets)	Polio cases more than 4 days after vaccination (Koprowski) (G)
BANALIA	8	29/11/57 to 4/1/58	8/1/58 to 12/1/58	4,182	1	0
GOMBARI (A)	12	Late January 1958	27/1/58	3,482	1 (plus more from March onwards)	0
WATSA (A)	2	Not recorded	29/1/58 to 31/1/58	12,789	8 (F)	0
BAMBESA	7	Early January 1958	1/2/58 (C)	2,433 (E)	7	0

NOTES:

- (A) Both Watsa and Gombari boasted large military camps.
- (B) According to Koprowski, just 3 of the 29 persons suspected of having polio in the four towns and villages were confirmed serologically as having Type 1 poliovirus – all in Banalia. According to Courtois, only one case was confirmed as Type 1 in Banalia, and one other in Gombari. Bervoets, however, reports that eight cases were confirmed as Type 1 in Banalia, and six in Bambesa. Nobody reports any confirmed Type 1 cases in Watsa, the town where the bulk of the vaccinations occurred.
- (C) The four week gap between the report of the epidemic in Bambesa (which was the most significant in terms of cases per head of population) and the (partial) vaccination of that village with CHAT has not been explained.
- (D) These are the numbers vaccinated according to both Koprowski and Courtois. There are some small discrepancies with the vaccination figures provided by Bervoets for the last three towns. He records 2,925 vaccinees in Gombari; 13,069 in Watsa, and 2,350 in Bambesa.
- (E) According to Bervoets, 2,350 persons were vaccinated in Bambesa, and this was “only a part of the population”. The fact that there were subsequently seven cases of polio in Bambesa may well be related to the fact that part of the village was not vaccinated, and suggests that the vaccine may

have reverted to virulence. Koprowski, by contrast, says that “every inhabitant” of Bambesa was vaccinated, and reports no post-vaccination polio cases.

- (F) Bervoets mentions that in addition to the 13,069 vaccinees at Watsa, 1,500 people were vaccinated at the same time in the nearby mining town of Kilo, and that Kilo subsequently had 20 polio cases between April and July. The 1,500 vaccinees are believed to represent about a quarter of the population of Kilo, for subsequent arrangements were made for 5,000 more doses of CHAT to be given here. With regard to the original vaccination there appears, as in Bambesa, to have been reversion to virulence.
- (G) It is noticeable that details which conflict with Koprowski’s argument that CHAT vaccine is safe and effective do not appear in his *BMJ* paper. (Although Koprowski is officially recorded only as last author on this *BMJ* paper, I was told by at least two of the Belgian doctors from Stanleyville that Koprowski wrote it, and sent it to Courtois only for checking.) Koprowski claims that “after vaccination no more cases of paralysis were reported in the four localities involved in the outbreaks”, yet Bervoets makes it clear that polio cases occurred in each one of the four outbreak villages and towns, beginning five days after the vaccinations. (In his paper, published a few weeks after that of Koprowski, Courtois says that there were six post-vaccination polio cases, but does not say where.) Koprowski’s article was published on July 26, 1958, and Courtois’ paper appeared in August, so only in Kilo (which neither Koprowski nor Courtois mention in any case), could a persuasive case be made that the post-vaccination cases might have occurred after the papers had been written. Other potentially awkward details, such as the partial vaccinations at Kilo and Bambesa (both of which were followed by polio outbreaks) also go unreported by Koprowski and Courtois. Interestingly, in Koprowski’s paper there is no mention of the vaccinations at Stanleyville military camp, or along the eastern shore of Lake Tanganyika.

Sources: “Koprowski”: Article by G. Courtois, H. Koprowski et al.; *Brit. Med. J.*; 1958; (ii); 187-190.

“Courtois”: Article by G. Courtois; *Ann. Soc. Belge Med. Trop.*; 1958; 38; 805-816.

“Bervoets”: Official government letter from W. Bervoets to P. De Brauwere, 17/9/58.

What the CHAT vaccine researchers seem to have been concerned with are impressive statements which could be broadcast publicly, and enshrined in the medical literature, rather than with the reality of what was happening on the ground.⁴¹⁸

And 44 years later, in an uncanny echo of these events, we have the publicly-broadcast statements that the OPV theory has been destroyed, when the reality is so very different.

This is not Science. It is phantom science.

I know that some who have followed the origins debate closely suspect that the main reason why there has been such a premature rush to “bury” the OPV theory is that certain scientists may be trying to buy time, so that they can continue with the search for some real (or perhaps not so real) evidence that would genuinely damage the theory – such as a sample of HIV-positive blood from before the start of the OPV trials.

If scientists are (even belatedly) staging an honest search for such materials, then that is commendable. However, there are indications that at least some of these searches for ancient HIV may not be entirely above board. Simon Wain-Hobson (when he still believed in the merits of the OPV theory) told me that late in 1999, a few months after *The River* was published, he spoke with a CDC scientist at a meeting in the U.S. This scientist claimed that some of his colleagues were working with a sample of HIV from 1952, from which they had already obtained a sequence. Simon asked him where the sequence sat in the phylogenetic tree. The scientist held out the fingers of

one hand, to represent the Group M star-burst, and pointed to near the end of one of the fingers. Simon swiftly pointed out that if the 1952 virus was positioned near the end of a branch, whichever branch, it must surely be a contamination with a modern virus. The other scientist quickly changed the subject.⁴¹⁹ Interestingly, nothing more has since been heard of this “ancient HIV sample”.

And it seems that something similar may be going on even today. Repeated attempts (some of them clandestine) have been made by scientists representing KUL (the Catholic University of Leuven),⁴²⁰ among other institutions, to obtain a set of pathology slides which was recently located in the basement of the former medical laboratory in Stanleyville. These apparently include materials obtained between 1955 and 1958. Because this may represent the period both before and after the beginning of the OPV trials, I believe it is of paramount importance that these slides, and the accompanying data, should be investigated by “neutral” institutions, rather than by one that was itself directly involved in the original research programme.⁴²¹

Because a *genuine* sample of HIV-1 Group M from before the time of the OPV trials would constitute a powerful piece of evidence against the OPV hypothesis, it is all the more important that the provenance of any such sample (if it exists) should be above suspicion.

k) The emperor’s new clothes.

Everyone knows the fairy story by Hans Christian Andersen. The emperor parades through town, showing off his new clothes. The courtiers and townspeople gasp and cheer, and applaud the finery. But then one day a young boy stands up at the back and says the unsayable....that the emperor’s clothes aren’t clothes at all. That he is naked. And the people stop bowing, and look up, and see that it is so.

I believe that, in the course of this debate, certain members of the scientific community have been acting with imperial and empirical disdain for the most basic tenets and ethics of science. And I think it’s time that someone stood up at the back, and pointed out just what the emperor is wearing today.

Until a moment ago, it seemed that perhaps I was going to have to be the little boy. But fortunately someone else has just stood up over there, behind the pages. I’m not sure who he is, but he’s rather small, with a beard and glasses, and he appears to be getting quite red in the face.

In fact, he really does seem quite upset....inflamed, even. By the looks of things, he could use a bit of dephlogistication.⁴²²

Now he’s shouting something. What’s that he’s saying? “*Desolé, mais il faut le dire.*” “I’m sorry, but this has got to be said.” Good grief, I think that must be Monsieur Émile Zola. I thought he’d died years ago. Anyway, by the look of things, he’s about to read out a series of accusations. Let’s listen.

- *J’accuse.*

- I accuse various scientists of having participated in research which resulted in a disastrous error of judgement perpetrated in Africa by an international scientific team nearly half a century ago.
- I accuse Dr Paul Osterrieth of having participated in a large-scale chimpanzee sacrifice programme in the Congo in the 1950s, in order to gather both organs and blood for purposes that are still largely unknown. I further accuse him of having prepared polio vaccine in his lab in Stanleyville in the 1950s, in a primitive Maitland-type tissue culture based on chimpanzee cells and chimp sera – and of having given incomplete and misleading answers about this episode over a period of several years.⁴²³
- I accuse Dr Hilary Koprowski of having instigated the programme under which Dr Osterrieth prepared the polio vaccine, and under which various scientists (including Koprowski himself, Ghislain Courtois, Gaston Ninane, Paul Osterrieth, Agnes Flack and George Jervis) conducted and oversaw the human field-trials of that vaccine in the Belgian Congo and Ruanda-Urundi. I also accuse him of having instigated the research programme at Lindi camp, and of consistently failing to provide meaningful and adequate answers about the work that was conducted there. I believe that Dr Koprowski was well aware of the true nature of the chimpanzee-related work that was going on in Stanleyville and at Lindi camp, from first to last.
- I accuse Dr Stanley Plotkin of having been involved with the Stanleyville/Lindi research programme, and of having coordinated an attempted cover-up in response to the hypothesis proposed in *The River*. I also accuse him of saying nasty and untrue things about my friend, Monsieur Hooper.
- I accuse doctors Beatrice Hahn, Paul Sharp, Bette Korber and Kevin De Cock, among others, of having demonstrated a bunker mentality in promoting a version of events which is increasingly far-fetched. It is apparent that neither their epidemiological scenario nor their phylogenetic dating argument stands up to close scrutiny, yet they continue to present both as if they were proven facts, not hypotheses. The evidence about the focal role that recombination has played in HIV-1 Group M makes it increasingly clear (at least to me and Madame Zola) that there is no reliable phylogenetic basis for making estimates about when the Group M epidemic began. We feel that the fact that such estimates are made at all is prompted (knowingly or unknowingly) by the perceived necessity of “disproving” the OPV hypothesis.
- I accuse Dr Simon Wain-Hobson of having failed to honour his agreement with Bill Hamilton, and having failed to properly investigate and report on the chimp samples which Bill and his colleagues brought back from the Congo. There is little enough primary data available on this issue, and many people consider that Professor Hamilton effectively sacrificed his life in order to procure these materials. The least he deserves from his former collaborator is a full and properly detailed scientific investigation and report. Furthermore, I accuse Dr Wain-Hobson of having done a *volte-face* on the origins issue, a flouncy flip-flop that has been prompted primarily by pragmatism.
- I accuse Dr Robin Weiss of having presided over a *Weisswash* in the origins of AIDS debate. I accuse him of having used his power (and he is indeed very powerful and influential within the world of science) to spin the arguments against the OPV theory, and to attempt to persuade both scientific and lay observers that the theory has been disproved. I further accuse him of having told a blatant untruth by stating that the OPV theory has been “destroyed”. He has failed to

provide any supporting evidence for that assertion, and yet he has also failed to withdraw it. In reality, Professor Weiss has based his arguments on nothing more or less than his own “plain, personal prejudice” – to quote his own (unpublished) words from the Royal Society meeting. I am informed that Professor Weiss has recently written to a journalist that “it strikes me that HIV researchers are to a man and a woman weary of [Hooper’s] mutating hypothesis”, an interesting comment, in that it suggests he thinks that hypotheses should not adapt to the arrival of new evidence. It also suggests that he and his colleagues are now too bored to think further about whether the world’s most disastrous human epidemic may have been started by Man himself. However, the word on the *vigne* is that, despite Dr Weiss’ protestations of *ennui*, he himself is now either editing, or organising, a new book of invited essays about “how AIDS began”. If the rumours are correct, then we hope that the book will be more balanced than Dr Weiss’ previous contributions on the subject. But we are not holding our breath.

- I also accuse my friend Mr Edward Hooper of having failed to investigate the provenance of the L70 sample (which produced the ZR59 sequence of HIV-1) with sufficient care in 1997, when he was submitting information about that sample to be published as part of an article in *Nature*. This only goes to show that we can all make errors of judgement, and this suggests also that Mr Hooper is sometimes *un petit peu* too willing to ascribe evil intent to others when they too make mistakes.
- I accuse the little boy of going home early without permission. He should be kept in for a week, and lose pocket money.
- And I accuse the emperor of being naked. Look up quickly, people of the town, if you want to see.

Gosh, that Monsieur Zola – once he starts accusing, he does get carried away, doesn’t he?

6. Conclusion: the importance of the level playing-field.

The new evidence about the local production of CHAT in Stanleyville, and the strong probability that it was prepared in chimpanzee cells and sera, are vitally important.

This new information does not, however, prove that CHAT vaccine started the AIDS pandemic.

On the other hand, the scale of the attempted cover-up (both at the time, and in the last few years) suggests that others besides myself suspect that it may have done so.

Some things which have happened in the course of this controversy are deeply regrettable, and have reflected no credit on those responsible. Having said that, I do realise that many of those who have been so determined to prove the OPV theory wrong may have acted in good faith. When one sincerely believes that this simply has to be just another conspiracy theory, and that the African CHAT campaigns could not possibly have been the source of AIDS, then one may also come to believe that almost any action one might take is permissible provided it has the right outcome, and persuades others also that the theory is misguided.

It is hoped that this paper will persuade at least some of those who were previously convinced of the inherent wrongness of the OPV hypothesis that it might be moot to reconsider.

Because of the role he has played in this controversy, I am not alone in regretting that Professor Robin Weiss was invited to deliver the closing summary at this conference. In all other ways, the events here at Lincei have demonstrated an even-handedness that was sadly lacking from sections of the Royal Society meeting.

I would like to thank the Lincei academy for inviting me to speak (and write) in the place of the late, lamented Bill Hamilton (as if any could take his place!). I have not written the paper that Bill would have written, but I hope that what I have contributed, though sometimes blunt, has done honour to his legacy. The decision about how to write this paper has not been made lightly.

By honouring their promise to Bill, and by allowing this conference to go ahead on a level playing-field, the *Accademia Nazionale dei Lincei* has done a great service both to Science, and to ethical conduct in Science.

And by their conduct during and since the conference, the officers and members of the academy have acknowledged that truth does not automatically, or exclusively, come from the pens and the mouths of the “experts”. I thank them for that.

Let me close by adapting Benjamin Disraeli’s question to this dreadful and unprecedented epidemic.

Was AIDS caused by an ape, by an angel (if so, surely a dark one) – or by Man himself, in all his bungling and clumsy ambition and human frailty?

We still don’t know the answer. But my vote would go to the last of the three.

EH July 2, 2002

- *I would like to thank more than a dozen scientists from different fields, and a number of non-scientists who are interested in the debate, for their help with, and input to, this paper. Because this remains such a highly controversial subject, and because I do not wish to cause anyone any embarrassment, I shall refrain from citing them by name, at least for now.*
- *I would also like to thank my partner, Ms P. Griffin, for providing love, ongoing support facilities – and the hand-drawn maps.*
- *As I was completing this manuscript, I was informed of the sad death, on June 7, 2002, of Dr Kamil Kucera, the brilliant Czech parasitologist. Between 1991 and 1993, Professor Kucera prepared several hundred pages of detailed hand-written notes and tables about his researches into *Pneumocystis carinii*, which causes PCP, the pneumonia that is the classic opportunistic infection of “Euro-American AIDS”. He wrote these notes in meticulous copperplate on large sheets of ancient air mail paper, and he sent them to me in batches, inside home-made envelopes, stuck down with glue.*

- *Like Bill Hamilton, Kamil Kucera was a man whose scientific commitment and integrity shone through his work, and through his whole life, and I would like to dedicate this paper to both of these admirable men.*

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7. Afterword: the Culture of Secrecy.

Just as this paper was about to be submitted, I was informed that on November 20th, 2001, Dr Paul Osterrieth had posted on the Web an official response to the speech which I delivered at Lincei on September 28th, 2001 (which contained the bare bones of the evidence presented in the current paper).

Entitled “The truth is what happened and not what one wishes that had happened”, Osterrieth’s four page disclaimer initially comes across as an impressive riposte – one that is calm, measured, generally dignified, and seemingly authoritative.⁴²⁴

However, his belated decision to provide some additional details about his work in Stanleyville actually raises more questions than it answers. Firstly, much of the key information he provides is demonstrably inaccurate, or else contradicts material in his past statements. Secondly, Osterrieth’s latest declaration only highlights his unwillingness to make any clear statement about the most important issues – such as what he and his fellow scientists were doing with the Lindi chimps, and what happened to the CHAT polio vaccine once it had arrived at the Stanleyville medical lab.

Several of Dr Osterrieth’s claims have already been covered in the main text, but I shall examine his new claims point by point below:

- Osterrieth now states that he returned to Stanleyville on February 28th, 1958, and he sources this detail to the annual report of the LMS. What that report actually records is that he returned to work at the lab on February 23rd, but even that detail is inaccurate. We know this because in a tape-recorded interview in 1993, Dr Osterrieth told me that he arrived back in Stanleyville “a very short time” after Dr Deinhardt’s arrival (which is independently documented as having been on February 1st, 1958). His wife added that their arrival in Stan was “just a few days” after Deinhardt’s, and I therefore believe that his return must have been on or around February 4th. Support for this is provided by Osterrieth’s “first assistant”, who began work on February 12th, 1958 (I have seen his official stamped work records, which confirm this), and who recalls that Osterrieth was already making polio vaccine when he, the assistant, started work. Dr Osterrieth’s sudden insistence that he was not present in the lab until February 28th, 1958, may not be unrelated to the fact that the vaccination at Stanleyville military camp took place on February 27th.
- Osterrieth states that he “did not carry out any autopsy [on a chimpanzee], since that was the job of Dr Ninane the pathologist”. He adds that “when an autopsy was carried out, limited pieces of different organs were taken to carry out various analys[es]”. These statements are directly contradicted by the recollections of two witnesses from Lindi camp: Joseph the camp nurse (who carried out most of the

sacrifices and gross dissections, and who was present at most of the autopsies), and “Antoine”, who watched some of them “from the wings”. Both say that it was Dr Osterrieth who conducted most of the autopsies on the chimps, and who regularly used to remove entire organs and put them into metal canisters. On the basis of multiple pieces of evidence, it appears probable that Ninane conducted the autopsies on dead or diseased chimps, but that Osterrieth dissected, and removed organs from, those which were sacrificed for other reasons.

- Osterrieth states “I was not the one who took blood from the chimpanzees”. This is directly contradicted by the testimony of his first lab assistant, who recalls that he and Dr Osterrieth regularly visited Lindi camp on Saturdays in order to obtain bloods from the chimpanzees, which Osterrieth later spun down into serum in the sterile (tissue culture) lab in the virology department. The assistant says that this sometimes happened at weekly intervals, and sometimes about once a month. Furthermore, although the assistant does not know the details of how the polio vaccine was made, he associates the taking of chimp blood with the making of the vaccine.
- Osterrieth asserts: “clearly my primary task was neither to prepare cell culture nor to prepare vaccine” at the Stanleyville lab. Yet in my first interview with him in 1993, he told me the exact opposite with regard to making tissue culture. At the beginning of the interview, he explained that his work in the virus lab “was essentially to develop tissue cultures, to expand the virus work”. When I asked him what sort of cultures, he answered “certainly monkey kidney”, but was unable to remember the species, apart from assuring me that it was not the chimpanzee. (Only later did he decide it must have been the baboon.) Later, he stated that his four month visit to the US in October 1957 to January 1958 had been mainly to get training in cell culture and virology, and that he had spent two weeks [or a month, according to his latest statement] at the Wistar Institute, where he “was working only in the lab of tissue culture, and looking how to do things”. At another point in the 1993 interview, Osterrieth said that Koprowski had demanded that he come to the Wistar “because of the work on the polio” which, Osterrieth explained, related to the two vaccine strains, CHAT and Fox.
- Osterrieth states that “no Maitland type cultures of any animal were produced in the lab, and no vaccine was produced”. The first part of this statement is contradicted by the testimony of Courtois’ assistant, who says that chimpanzee tissue cultures were being produced in the microbiology lab where Osterrieth worked before he took over the virology lab. Furthermore, an AFEB report documents that the materials sent to the US in early 1958 were minced chimpanzee kidneys in isologous (chimpanzee) serum and Hanks’ solution, which effectively constitute Maitland-type cultures. Osterrieth’s second claim, that no vaccine was produced, is contradicted by both of the assistants in his virology lab. Indeed, the first assistant states that Osterrieth was already making polio vaccines before he (the assistant) started working in the lab in February 1958, and that he continued to make polio vaccines, on request from the provincial government, during the next two years.
- Osterrieth states that: “the allegation that chimpanzee serum was prepared to be used in tissue culture medium for chimpanzee cells in culture is sheer nonsense, since at that time one used calf serum or sometimes foetal calf serum to enrich the culture medium, since this type of serum was available in sufficient amounts.” Despite Dr Osterrieth’s protestations, he is incorrect. He ignores the fact that foetal calf serum was expensive to purchase, and that local production of calf

serum was unlikely, given that cows did not survive well in the rain forest environment of Stanleyville; (certainly in 1999, there were no cows living in or around Kisangani). By contrast, chimp serum was freely available, and it was a good idea to use precisely because it was isologous, and came from the same species as the kidneys. And crucially, we know from the AFEB report that in fact chimp serum *was used* to nourish the chimp kidney cells.

- Osterrieth states: “we could not even check reliably the titre of [polio] vaccine lots”. This is interesting on two counts. Firstly, that they proceeded with local vaccinations despite being unsure of the titre of the vaccine. And secondly, that Dr Osterrieth uses the collective “we” with respect to attempts to titrate the vaccine, which is the first time he has ever directly acknowledged that he himself was one of those who handled the vaccine in the lab. Despite his alleged desire to provide clarification, and despite having made seven oral or written statements about these matters over a nine year period, Dr Osterrieth has never revealed any significant details about the polio vaccine, or what was done with it.
- Osterrieth states that the chimps “were kept alone in individual cages and not together in a common one, with the possible exception of infants that could be handled easily.” This is incorrect. According to several contemporary accounts (including a review article prepared by his boss, Ghislain Courtois, in 1966), the chimps were often kept two to a cage, common chimp and bonobo together. Courtois also writes of a group-cage, where up to ten chimps could play at a time.
- Osterrieth writes: “Mr Hooper states that in the year 2000 I wrote to natives in Stanleyville to ask them to say nothing about what was carried out in my lab in the years of vaccination. This is simply not true. I never wrote such a letter, and if such a document exists Mr Hooper should produce it for examination.” Dr Osterrieth’s claim is interesting, because what I actually wrote in a footnote to my Lincei speech was that in late 2000, one of his ex-assistants “received a letter from Osterrieth, this being the first time he had heard from him for some forty years. What his old boss wanted to know was which of his former assistants at the virus lab and Lindi camp were still alive, and which were dead”. In any case, Osterrieth’s demand that I should produce the letter is inappropriate: since the letter was not written to me, I clearly do not have it to produce. What is available, however, is clear evidence to refute Dr Osterrieth’s denial, which will be produced in good time. Meanwhile, one is left to wonder why he should suddenly have been seeking such information.
- Osterrieth states that Dr Ninane’s family denies that he had either Parkinson’s disease or Alzheimer’s disease, and he remonstrates with me for making claims about Dr Ninane’s health in order “to cast doubt on the value of his testimony”. This is both misleading and untrue. When interviewed in August 2000, two weeks after her brother’s death, Gaston Ninane’s sister (with whom he had lived for the last ten or more years of his life) told me that he had suffered from Parkinson’s disease, which led to his falls, and to his being hospitalised; she added that doctors Koprowski and Prinzie interviewed him after his first fall, while he was in hospital. I believe that Alzheimer’s was also mentioned during that conversation (though I can find no mention of it on the tape), but in any case Dr Ninane had often told me that he feared he was suffering the first symptoms of this condition. The key point, however, is that (whatever his state of health) the statement which Dr Ninane apparently signed in February 2000, stating that claims that he had tried to make tissue culture, and chimpanzee tissue culture, in Stanleyville were “lies”, is itself untrue. I have the tape recordings of our interviews, which

demonstrate that Dr Ninane was correctly quoted, and that he did indeed say these things.

- Finally, Osterrieth states: “It remains astonishing that people who recognise that they had no access to these facilities [the sterile room in his virology lab] would know precisely what kind of work I did carry [out] in this lab, what type of tissue culture was performed, what kind of virus was inoculated, what kind of serum was used”. This is a strange comment, for Osterrieth’s assistant did not make any claims to knowing these precise details. What he did know, however, was that Osterrieth was making a polio vaccine which he (the assistant) then fed to the people living at the local military camp. And fortunately, another African technician who had worked in another department of the Stanleyville lab since 1956 knew more precise details, including the key fact that chimpanzee tissue culture was being made in different departments at the Stanleyville laboratory.
- I believe that, taken as a whole, Dr Osterrieth’s comments are highly revealing of the culture of secrecy within which he worked – and with which he worked.

The title of Dr Osterrieth’s disclaimer, “The truth is what happened and not what one wishes that had happened”, would seem to be appropriate, though perhaps not in the way intended.⁴²⁵

In comments which he made to a journalist in October 2001, Robin Weiss said: “Either Osterrieth is lying through his teeth or [Hooper] has got it wrong”. I would put it a little differently. I have made clear statements about what can and cannot be proved, and I have the evidence to support those statements. By contrast, I believe that Dr Osterrieth’s often contradictory statements on this matter are not supported by evidence, and cannot be relied upon.

8. POSTSCRIPT: January 1, 2003.

Since this paper went to press, there have been a number of significant developments with regard to the OPV hypothesis of the origin of AIDS.

In July 2002, a brief communication by Andreas Meyerhans and colleagues, published in *Nature*, revealed unprecedented evidence of rampant recombination occurring within the individual HIV-infected cells of AIDS patients.⁴²⁶ It was left to Jon Cohen, writing in *Science* about what was referred to as a “beautiful study” by the Meyerhans group, to sum up its significance. Cohen commented that their work “raises serious questions about phylogeny trees that attempt to date the origin of HIV, all of which intentionally discard suspected recombinants to make the data interpretable”.⁴²⁷

So the cat is finally out of the bag. At long last, a consensus is emerging that phylogenetic dating is quite simply an inappropriate technique to apply to a virus as mutable and capable of recombination as HIV. The main scientific “disproof” of the OPV theory is revealed as an illusion.

Meanwhile, additional interviews conducted by this author in Europe have confirmed and substantially enlarged the central revelations of the present paper, concerning the events that took place in the 1950s at Lindi camp, and at what even Hilary Koprowski referred to, in 2001, as “the vaccine laboratory” in Stanleyville. It is neither practical

nor appropriate to try to incorporate this new information into the present paper, but details have been deposited with various parties, and the full story will be reported in due course, both in the scientific literature and elsewhere.

The questions that now have to be asked are these...

- For how long can mainstream Science continue to insist that CHAT was not made in chimp cells?
- How long will it be before independent laboratories conduct the tests that now clearly need to be conducted, to see if this vaccine was linked to the genesis of AIDS? (For the record, virological testing of chimpanzee and bonobo populations in the DRC is finally underway. An appropriate next step, I believe, would be an epidemiological and virological survey of CHAT vaccinees and non-vaccinees in the DRC and Burundi.)
- What does this story (including the attempted cover-up) have to tell us about the way that Science is conducted in the modern world?
- And to what extent will a proper understanding of how AIDS began advance the search for a vaccine or therapy?

¹ Hooper, E., *The River: A Journey Back to the Source of HIV and AIDS*. (London: Allen Lane – Penguin Press; 2000). This edition was completed in March 2000. For a version that is updated to October 2000, with additional material about the Royal Society conference on pages 874-877, see: Hooper, E., *The River: A Journey to the Source of HIV and AIDS*. (Boston: Little, Brown; 2000).

² For those who are interested, an article about Dr John P. Moore's role in this debate (and some of the things that he has written about it) will be appearing in due course on Brian Martin's web-site.

³ UNAIDS epidemic update – December 2001.

⁴ Anon., "New UNAIDS report warns AIDS epidemic still in early phase and not levelling off in worst affected countries"; UNAIDS press release; July 2, 2002.

⁵ Huet T., Wain-Hobson S., "Genetic Organization of a Chimpanzee Lentivirus Related to HIV-1"; *Nature*; 1990; 345; 356-359.

⁶ Notably Beatrice Hahn, Paul Sharp and Bette Korber (but also enthusiastically propounded by others, such as Stanley Plotkin, Hilary Koprowski and Paul Osterrieth).

⁷ For the cut hunter school, the recency of the epidemic is explained by new developments in the mid twentieth century, such as urbanisation and more liberal sexual mores, or else the emergence (and ill-advised reuse) of disposable needles, allowing the chimpanzee virus, once transferred, the new opportunity of being passed parenterally from human to human. At its most basic, the natural transfer theory of a casual chimp-to-human zoonosis seems quite reasonable, even if it is impossible to prove or disprove. On the other hand, the theory also has certain innate logistical problems, some of which are discussed later in this paper.

⁸ In several instances in this paper, I have updated the epidemiological data published in *The River*. In particular, I have omitted one of the 28 CHAT trials that feature in the book (trial #22, of 64,000 persons at Lubudi, or Kabare-Lubudi), because I agree with Stanley Plotkin that there is no concrete evidence that it took place. Dr Plotkin, however, should not get carried away, for despite his efforts, I believe this is the only error (or potential error) of any significance that he has managed to identify in the book. In my own defence, I should point out that "Kabare-Lubudi" was apparently proposed as a forthcoming trial at a 1959 press conference at which Plotkin himself was one of the three major speakers. Furthermore, accepting that the trial is not proved to have occurred is not the same as accepting that it did not occur. (Kabare and Lubudi *territoires* are indeed hundreds of kilometres apart, as Plotkin observes, but they were also, in 1959, the sites of two of the Belgian Congo's three major cement factories. I still suspect that the trial may have taken place, perhaps at the behest of the chief medic of the cement company.) But for now, I agree that without proof, this trial should be omitted from the list.

⁹ *River*, 2000, pp 387-389.

¹⁰ *River*, 2000, p. 385. Dr Koprowski is in many ways a ground-breaking scientist, one not afraid to step where others fear to tread, but there are also several examples in *The River* of occasions when he appears to have displayed recklessness. One such was his involvement in the staging of a field trial of an early genetically modified rabies vaccine in Argentina in the mid-eighties, without the permission or approval of the Argentine government. The London *Times*, in a leader, described this as a “reckless and illegal release”. [*River*, 2000, p. 449]

¹¹ Above this legend on the sign outside Lindi camp was the single word “Polio”, which presumably helped to deter the curious. To this day, the camp is referred to by local villagers as “Camp Polio”.

¹² Plotkin S.A. “Untruths and Consequences: the false hypothesis linking CHAT type 1 polio vaccination to the origin of human immunodeficiency virus”. *Phil. Trans. R. Soc. Lond. B*, 2001; 356, 815-823. “The purpose of the camp was not at all mysterious”, writes Plotkin (p. 819), but his non-mysterious account substantially conflicts with the testimony of the Lindi workers, as reported in the present article, and it omits all details pertaining to sacrifice and removal of organs. This is despite the fact that both African and Western witnesses report that all the Lindi chimps (save for those which died natural deaths, and around 60 which were removed by lorry when the camp closed around the end of 1959) were sacrificed for experiments. [For instance, see *River*, 2000, page 725.]

¹³ With the sole exception of a retrospective article published in 1967 by the former director of the *Laboratoire Médicale de Stanleyville*, Ghislain Courtois, there are only a few sentences published about the work carried out at Lindi camp, and these include next-to-no information about the polio work, and nothing at all about the sacrifice programme. [G. Courtois, “Sur la réalisation d’une singerie de chimpanzés au Congo”; *Symposium international sur l’avenir des animaux de laboratoire*, Lyon, September 18-20, 1966 (Lyon, Institut Merieux, 1967, pp. 235-244).] The annual reports of the *Laboratoire Médical de Stanleyville* reveal that there was vaccination, vaccination and challenge, and intraspinal safety testing of the polio vaccines at Lindi, but provide no further details during the first three years (1956-1958), which were the period of the so-called “polio research”. The lack of information about the research conducted at Lindi is very striking, and even some of the Belgians who used to work in the Stanleyville lab commented upon it. A promised 1958 article about the polio research at Lindi, which was thrice referred to as “in preparation”, or “being edited”, was never released (at least to the public). During the period the camp was open, visitors to Lindi were discouraged, and apart from the Belgian and American doctors and their African assistants, nobody was allowed inside the second hangar, which housed the chimps that were scheduled for experimentation. Despite this secrecy, certain visitors managed to discover interesting details. For instance, early in 1960 a visiting Dutch primatologist, Adriaan Kortlandt, was told that 86 pygmy chimps had died in 3 weeks in the course of the polio research, and that he should not ask too many questions on the subject, because otherwise he “might cut [his] fingers”. It appears that all biomedical samples pertaining to this research have now been lost or discarded. Furthermore, all Koprowski’s records about Lindi camp, and the research on his vaccines conducted there, have apparently been “lost in a move”, while the relevant archives in the polio correspondence file in the Belgian Ministry of Foreign Affairs archives section for the key period (November 1956 to June 1958) are missing. See *River*, pp 708-723.

¹⁴ The round figure of 400 chimps (in the first 20 months, the time of polio research) has been provided by many sources, and is now no longer in question. The figure of “over 600” for the entire three-and-a-half-year period that Lindi camp was open has been given by two of the Africans who worked there, and seems consistent with other available evidence.

¹⁵ A. Kortlandt, “Statements on pygmy chimpanzees” [letter]; *Lab. Primate News.*; 1976; 15(1); 15.

¹⁶ T.O. Jonassen et al., “Sequence analysis of HIV-1 Group O from Norwegian patients infected in the 1960s”; *Virology*; 1997; 231; 43-47. Also see *The River*, 2000, pp. 772-774, for the story of the Norwegian merchant seaman who was probably infected with HIV-1 Group O in Cameroon in 1961-2, who developed first symptoms of AIDS in 1966, and who died in 1976.

¹⁷ Those very few European and American cases from before 1978 all appear to have been infected in Africa – and, more specifically, in the DRC, the former Belgian Congo. There are two possible exceptions, only one of which is confirmed by serology – this being a girl born in New Jersey in 1973 or 1974, who was clearly immunocompromised soon after birth, and who died of AIDS in 1979. The girl’s mother was a drug-injecting 16-year-old with multiple partners, who must have been born between late 1956 and 1958. The mother had thrombocytopenia at the time of the girl’s birth, suggesting that she might have passed HIV infection vertically to her daughter. In *The River*, I proposed that the mother might have been one of those infants who was born at Clinton prison, the major long-stay penitentiary for female prisoners in New Jersey, where almost every baby born

between late 1956 and 1958 was vaccinated with one or more of Koprowski's OPVs, most of which were experimental. In his speech to the Royal Society, Dr Plotkin rejected this hypothesis, explaining that he had approached the pediatrician who had tended to the young girl, and discovered that the mother's name did not match that of any of the Clinton infants from the 50s. That appeared to have settled the matter. However, in June 2001 I was contacted by a man who knew that he had been born at Clinton prison, and suspected that he might have been one of the OPV vaccinees. From certain data he provided, I was able to confirm that he had indeed been one of the vaccinees. However, this man had been adopted at the age of two, and had been given the family name of his adopted parents. At the time he approached me, he did not know his original family name. Other vaccinated Clinton infants may similarly have had their names changed following adoption, and may not even know this fact. For this reason, Plotkin's "refutation" of a link with CHAT in this instance may not be a refutation at all.

¹⁸ These comprise the 16 cases listed on page 746 of *The River*, less case 2 (which was caused by HIV-1 Group O), but including an additional 1976 case from Burundi, which would be entered into the table on page 746 as: "1976; Congolese; 40; M; Bujumbura; 'Slim', chronic diarrhoea, generalised KS, dry cough; 0 (same site)".

¹⁹ Later in this article, two other potential AIDS cases from the 1960s are detailed, dating from 1964 and 1968-70. Both come from Leopoldville/Kinshasa, DRC, though the first apparently originated from "outside Leopoldville". Both cases were volunteered by doctors with considerable clinical experience of Africa. Other less specific potential AIDS cases from 1958 onwards are also detailed later in this paper. Because of various considerations, these two additional cases are not included in the maps and AIDS data in this paper.

²⁰ These comprise the 22 cases listed as numbers 17-38 on pages 746-7 of *The River*, plus one additional 1980 case from Burundi, which would be entered into the table on page 747 as: "1980; Burundian; 42; M; Muramvya; chronic diarrhoea, interstitial pneumonia, atypical KS; 0 (same site)".

²¹ For the old list, see: *River*, 2000; pages 746-747. As for the new list, all 39 of these early African cases had been documented retrospectively as likely cases of AIDS by the doctors involved, either in medical journals, in books or dissertations, or in unpublished articles. For 9 of the 39, retrospective serology had confirmed HIV-1 infection. As explained above, the new list includes two additional Burundian cases of AIDS from 1976 and 1980, and omits the former case 2, since that relates to Group O infection. In addition, it seems that details of one AIDS case (#32) were somehow omitted from this table during the final production stages of the book. The relevant details should read: "1980; Congolese; 21; F; Congo; herpes, candidiasis".

²² See *River*, 2000; pp 187-192 and 227-235, together with relevant endnotes.

²³ Twelve of the sixteen sites are from the DRC, Burundi and Rwanda. The remaining four sites, in Tanzania, Kenya, Congo Brazzaville and Senegal, begin to show evidence of Group M only in 1980 and 1981, when the trans-African spread of the virus first became detectable.

²⁴ Although two (later three) of the samples tested came from CHAT *pools* (10A-11 and 13) which had been used in Africa, it became clear that different *batches* of such pools of CHAT had been prepared in different laboratories, and in the kidney cells of different species. It was where and how the individual batches had been prepared which was significant.

²⁵ The word was that three major scientific journals had been competing to publish the results of the Wistar testing, and in the end *Nature* and *Science* shared the honours. See the three articles in *Nature*; 2001; 410; 1045-1048, plus one article in *Science*; 2001; 292; 743-744.

²⁶ J. Cohen, "Disputed AIDS Theory Dies Its Final Death"; *Science*; 2001; 292; 615.

²⁷ R.A. Weiss, "Polio vaccines exonerated"; *Nature*; 2001; 1035-1036.

²⁸ *River*, 2000, pp 843-846. "Antoine" was clearly indicated as a pseudonym, for the first time the name appeared, on page 843, it was in inverted commas.

²⁹ S.A. Plotkin et al, "Postscript relating to new allegations made by Edward Hooper at The Royal Society discussion meeting on 11 September 2000", p. 829. This is a good example of the way that, at least in this debate, Professor Plotkin has repeatedly set up straw men, or false issues, in order to shoot them down. By the time this updated version of the *River* postscript was being written, in January to March 2000, it was already apparent that opponents of the theory were resorting to a number of questionable tactics, which is why I decided to protect the witness by using a pseudonym.

³⁰ In this paper, to reduce the risk of further such mischievous claims, I shall place quote marks around "Antoine" every time he is mentioned.

³¹ On my last day in Kisangani in 1999, I asked "Antoine" to see if he could locate Joseph. He managed to track down some people from Joseph's home town, who appeared to know the correct man: they stated that Joseph had died in 1964, having got "thinner and thinner" at the end of his life. It is now

apparent that this was not the right Joseph, but I am satisfied that what “Antoine” reported to me (which I in turn included in the new postscript) was reported in good faith.

³² Joseph had previous experience from working in the pathology lab at the Stanleyville hospital for Africans. The Lindi work was performed on a table in the second hangar – the one which nobody else apart from the Stanleyville doctors and the Lindi camp workers was allowed to enter. For many (though not all) of these operations, Joseph the nurse was the only African officially present, though it seems that the other camp workers sometimes watched from the wings.

³³ This ties in with my own figure of 416 chimps housed at Lindi during the first twenty, very hectic months after the camp opened (June 1956 – February 1958). It was, however, the first time that I had heard such a high overall total.

³⁴ W. Henle, G. Henle and F. Deinhardt, “Studies on Hepatitis”, Annual Report to the Commission on Viral Infections of the Armed Forces Epidemiology Board, March 1958 – February 1959, p. 5. [See extract later in this paper.]

³⁵ P. von Magnus, H. von Magnus et al., “Tissue cultures of trypsinised kidney cells from different monkey species”; *Dan. Med. Bull.*; 1955; 2(8); 236-240.

³⁶ In light of the dubious approaches made to some other witnesses, I am not prepared at the moment to identify this man, and certain others who feature in the text. They will be fully identified at an appropriate time in the future.

³⁷ Letter from W. Bervoets to P. De Brauwere, September 17th, 1958; file H4484/1058 at the Belgian Ministry of Foreign Affairs archives.

³⁸ This is intriguing, for the latter trips are not revealed by the annual lab reports for 1958 and 1959. (The 1960 report was never published.)

³⁹ R.A. Weiss and S. Wain-Hobson (editors), “Origins of HIV and the AIDS Epidemic”; *Phil. Trans. R. Soc. Lond. B*; 2001; 256; 771-977.

⁴⁰ P.M. Osterrieth, “Vaccine could not have been prepared in Stanleyville”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 839.

⁴¹ S.A. Plotkin, “CHAT oral polio vaccine was not the source of human immunodeficiency virus type 1 Group M for humans”; *Clin. Inf. Dis.*, 2001, 32, 1068-84. S.A. Plotkin, “Untruths and consequences: the false hypothesis linking CHAT type 1 polio vaccination to the origin of human immunodeficiency virus”. *Phil. Trans. R. Soc. Lond. B*, 2001; 356, 815-823. S.A. Plotkin, D.E. Teuwen, A. Prinzie, and J. Desmyter, “Postscript relating to new allegations made by Edward Hooper at the Royal Society discussion meeting on 11 September 2000”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 825-829. The last of these articles states at the end that “letters cited in this paper will be deposited” at one of two libraries, in Philadelphia and Leuven. However, I would also be interested to see the source documents that Plotkin quotes in “Untruths and consequences”, especially the passage chart, and the March 4, 1958 letter from Koprowski to Jarvis, which refers to Ninane’s telegram. Requests to Dr Koprowski to view the latter document have thus far been ignored.

⁴² For what it is worth, I had neither heard Osterrieth’s presentation at the Royal Society, nor did I know of its content, before I left for Africa at the start of March 2001. It was only when I returned to England, that I was reminded of the statements by Osterrieth quoted by Stanley Plotkin in his Royal Society speech (an early version of which had been distributed at the time of the London meeting). Even then I intended to approach Dr Osterrieth once more, to give him an opportunity to respond to the testimonies from Kisangani. But then came the publication of the Royal Society article, and I realised that there was little point, for Osterrieth had already made his definitive statement.

⁴³ Dr Koprowski, at the Royal Society meeting, dismissed one of my other African witnesses as a “low technician”, a comment which prompted some booing from the audience. My own experience is that African witnesses frequently have more accurate memories of events than Westerners – which may be partly due to African traditions of oral history.

⁴⁴ Anon., “Application au Congo du nouveau (antipolyo) du Dr. Koprowski”; *L’Avenir* (Leopoldville); August 9th/10th, 1958. The article states that the new polio vaccine of Dr Koprowski “has been prepared at Elisabethville by the Wistar Institute, and is controlled from the point of view of efficacy and safety by the Stanleyville laboratory”.

⁴⁵ The records suggest that CHAT was fed widely – to well over 800,000 people in 27 campaigns I have been able to document, and probably in other places too. By contrast, it seems that Fox was fed only on a small scale, to a few thousand individuals in three places: Aketi (December 1957), Stanleyville (May 1958), and Leopoldville (starting in September 1959). In the latter place, it seems likely that only Europeans were vaccinated. By that stage, the experimental trials were over, and September 1959 was the month when both CHAT and Fox were first fed to the bulk of the European population. By contrast, it was apparently felt not to be necessary to vaccinate the African population

against Type 3 polio. This again provides a clue about the real purpose of the Congo vaccinations, which appear to have been staged primarily in order to experiment with new varieties of vaccine, rather than in order to protect the local population (as has often been claimed since).

⁴⁶ Letter from W. Bervoets to P. De Brauwere, September 17th, 1958; file H4484/1058 at the Belgian Ministry of Foreign Affairs archives.

⁴⁷ The 1958 annual report of the medical service of *Province Oriental* reveals that “almost 50,000 individuals” had been vaccinated with the Koprowski strains in the province during 1958. Since the vaccinations in Aketi, Stanleyville and the epidemic areas totalled only about 30,000 people, it would seem that the hygiene department vaccinated a further 20,000 or so. I have records of 4,000 doses being given at Rungu in June, and 5,000 at Kilo (probably in July), so some 10,000 further vaccinations apparently took place at places unknown.

⁴⁸ S.A. Plotkin, “CHAT oral polio vaccine was not the source of human immunodeficiency virus type 1 group M for humans”; *Clin. Inf. Dis.*, 2001, 32, 1068-84.

⁴⁹ R. Sohier and O.G. Gaudin, “Monkey cell cultures in virology”; *Primates in Medicine*; 1969; 3; 80-92; see Courtois in “Discussion” on page 91.

⁵⁰ I also interviewed several other African witnesses in 1999 and 2001, but they were either less directly involved, or their recall of events was far less impressive or precise. The five referred to here are the two assistants from Osterrieth’s lab, the assistant from Courtois’ lab, together with Joseph and “Antoine” from Lindi.

⁵¹ *River*, 2000, p. 569.

⁵² Since 1993, Dr Osterrieth has claimed variously: (a) [between 1993 and February 2000] that chimp kidneys were *only* sent to the Children’s Hospital of Philadelphia, and not to the Wistar Institute; (b) [in 1994] that no chimp kidneys were ever sent abroad from Stanleyville; (c), in his latest [September 2000] account before the Royal Society, that chimp kidneys *were* after all sent to the Wistar Institute.

⁵³ W. Henle, G. Henle and F. Deinhardt, “Studies on Hepatitis”, *Annual Report to the Commission on Viral Infections of the Armed Forces Epidemiology Board*, March 1958 – February 1959, see p. 5.

⁵⁴ The accuracy of this date of arrival for Deinhardt is underlined by the fact that it featured in a report which was completed just nine days later. W. Henle, G. Henle and F. Deinhardt, “Studies in Viral Hepatitis”; *Annual report to the commission on viral infections of the armed forces epidemiological board*; March 1, 1957 to February 10, 1958.

⁵⁵ Anon, “Monkey Business”; *Thermometer* (published by the Children’s Hospital of Philadelphia); 1958; 9(2); 3 and 6.

⁵⁶ F. Deinhardt, G. Courtois, P. Dherte, P. Osterrieth, G. Ninane, G. Henle and W. Henle, “Studies of liver function tests in chimpanzees after inoculation with human infectious hepatitis virus”; *Am. J. Hyg.*; 1962; 75; 311-321.

⁵⁷ P.M. Osterrieth, “Vaccine could not have been prepared in Stanleyville”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 839.

⁵⁸ *River*, 2000; pages 352-355 and 595.

⁵⁹ S.A. Plotkin, “CHAT oral polio vaccine was not the source of human immunodeficiency virus type 1 Group M for humans”; *Clin. Inf. Dis.*, 2001, 32, 1068-84; see page 1071.

⁶⁰ *River*, 2000; page 688.

⁶¹ This tissue culture of chimpanzee kidney cells and chimpanzee serum was of course different from the classic tissue culture of the fifties, which typically employed monkey kidneys (usually from rhesus or cynomolgus macaques), and foetal calf serum. The latter was expensive to buy, and would also have been impractical to make in Stanleyville, not least because of the lack of cows in this “island in the rain forest”. Besides, if serum from the same species which had provided the cells was available, it would have made sound scientific sense to use it. [*River*, 2000, pages 847-848.]

⁶² Anon., “Expert committee on poliomyelitis. Third report.”; *W.H.O. Tech. Rep. Ser.*; 1960; 203; 1-53.

⁶³ Osterrieth quoted on page 818 of S.A. Plotkin, “Untruths and consequences: the false hypothesis linking CHAT type 1 polio vaccination to the origin of human immunodeficiency virus”; *Phil. Trans. R. Soc. Lond. B*, 2001; 356, 815-823.

⁶⁴ I was originally working on the assumption that the tubes would have been of 10c.c., and the bottles of 500 c.c., (which would have meant a total output of up to 7,000 c.c., or seven litres, of tissue culture), but I recently learnt that the bottles used in most labs of the fifties were of 4 oz, or 100 c.c., capacity. If correct, this would reduce the total to just 3,000 c.c., which could almost certainly be produced from two kidneys, coming from a single baboon.

⁶⁵ By contrast, there were eleven sentences and a lot of detail in the 1958 annual report about the much smaller hepatitis research programme involving the chimps.

⁶⁶ See: J. Kingdon, *The Kingdon Field Guide to African Mammals*, (San Diego: Academic Press, 1997), pages 32-35, which reveals that the olive baboon, *Papio anubis*, is found in savanna, light woodland and forest mosaics (ie transitional forest). Kingdon adds that: “here and there olive baboon populations range deep into the rain forest”, and Kisangani/Stanleyville is shown as being included in its range on the tiny map on page 33. None the less, baboon numbers near to Stanleyville would probably have been quite small, not least because of the long distance from its natural habitat.

⁶⁷ Ghislain Courtois’ notebook, marked “Mission USA. Trinidad. IOC Rio. 1955”, was kindly made available by his son, Dr André Courtois. “IOC” apparently stands for the *Institut Oswaldo Cruz* in Rio.

⁶⁸ *River*, 2000, pages 276-277.

⁶⁹ *River*, 2000, chapter 20, especially pages 277-279.

⁷⁰ Letter from Paulette Dherte to Fritz Deinhardt, June 20, 1959, made available by Jean Deinhardt.

⁷¹ An hour later in the interview, when we returned to this subject of making tissue culture, Gaston Ninane told me that after a while he had stopped trying to make it, “because after three or four or five months, it was impossible to make tissue culture with the material we had.” However, this time he only mentioned using human cells. When I reminded him that earlier he had told me that he had used both human cells and chimpanzee cells, he replied: “Oh, it’s possible. I don’t remember.” By this time, I was familiar with this particular phrasing from Dr Ninane, one that indicated that he was unwilling to answer any further questions on a topic. In *The River*, I wrote that Ninane “tried, but failed” to make tissue culture from chimp and human cells in Stanleyville, and that he later reiterated that he had never managed to make any successful cultures [page 569]. In fact, on reviewing the tape and tape transcripts, I find that he *never specifically stated* that he had failed to make successful cultures, although he repeatedly implied this. I did make one final phone contact with Dr Ninane in September 1997, partly in order to check this point, and he told me: “Yes, I tried [to make tissue culture], but I surely don’t have success, because it was impossible in the lab, where nothing make success in tissue culture.” This was the only time that he claimed explicitly that he had never managed to make any tissue culture in Stanleyville. Despite this, I feel that his repeated avoidance of answering the question directly (and the tautological nature of his final answer) raise issues about the reliability of that answer.

⁷² In her unpublished scientific memoir, Gertrude Henle relates how (presumably in around 1954) she and Dr Deinhardt sought the help of one of the great acknowledged experts on tissue cultures, Wilton Earle of the NIH, only to be told that “three years of intensive training in his laboratory would be required. When [Dr Henle] pointed out that we were virologists who wanted to use the cultures merely as a tool...he threw up his hands in disgust. Yet we learned enough during the visit to start out on our own, an ineptly as it was initially.” It seems clear that by 1958 Deinhardt possessed the background know-how to produce successful cultures from chimpanzee cells, especially if these were Maitland-type cultures, which were relatively straightforward to make, involving the cutting up of some kidneys with scissors, and then mixing them with some serum, growth medium and adding a few drops of antibiotics. Deinhardt did of course work with chimp kidney cultures, of the trypsinised variety, as soon as he returned to Philadelphia at the end of April 1958.

⁷³ Others who theoretically could have helped with the making of tissue culture in Stanleyville during this period include Dr Courtois’s deputy Dr Mangen (about whom little is known, other than the fact that he and Courtois did not get along), the nurse/pharmacist Paulette Dherte, and Professor Welsch, a bacteriologist from Liege, who appears to have visited both Stanleyville and the Ruzizi Valley in the course of a lengthy African tour in early 1958.

⁷⁴ P. Osterrieth, personal communication, 1993. Osterrieth told me: “He [Vandepitte] wanted us to take the samples, and the technicians to do the work. And I thought that was completely silly. To stay the whole morning in a place waiting for people to come, just to take blood, instead of being in the lab and doing the interesting work. So he told me: ‘tomorrow it’s your turn’. I said: ‘yes’. I didn’t go. And then he said: ‘You were not there’. I said: ‘No, I forget’. Looking him straight in the eyes, [I said] ‘I forgot’. It was finished.”

⁷⁵ G. Henle, personal communication, 1993. The Armed Forces Epidemiology Board reports for 1957-8 and 1958-9 make it clear that four shipments of chimpanzee kidneys took place during 1958, which are presumably the ones dispatched by Deinhardt between February and April of that year. Two further chimp kidney shipments were sent later, presumably by Osterrieth. [See: Deinhardt F., Courtois G., Dherte P., Osterrieth P., Ninane G., Henle G. and Henle W.; “Studies of liver function tests in chimpanzees after inoculation with human infectious hepatitis virus”; *Am. J. Hyg.*; 1962; 75; 311-321.] Dr Henle added that Dr Koprowski had helped set up Deinhardt’s visit. This further supports the possibility that Deinhardt would have been happy to deliver a bottle of the new CHAT vaccine pool (10A-11) to Stanleyville, as a *quid pro quo* to Koprowski.

⁷⁶ My problem here was that on the basis of what I had been told by doctors Ninane and Osterrieth, I was still locked into believing that they had *not* been able to make polio vaccine in Stanleyville – and that the vaccine must have been made either in Philadelphia or Belgium.

⁷⁷ It is my belief that Alzheimer's disease was mentioned by Dr Ninane's sister as well as Parkinson's, though it is not referred to in my notes, or on the tape recording. Certainly in our last two interviews Dr Ninane had often told me that he feared that he was suffering the first symptoms of Alzheimer's, but this may have been in a bid to explain lapses in memory, rather than an accurate self-diagnosis.

⁷⁸ S.A. Plotkin, "CHAT Oral Polio Vaccine Was Not the Source of Human Immunodeficiency Virus Type 1 Group M for Humans"; *Clin. Inf. Dis.*, 2001, 32, 1068-84; see page 1074.

⁷⁹ In addition, there is further confirmation of other aspects of this question by other indirect witnesses. For various reasons these are being held in reserve for now, but they will be reported at a later date.

⁸⁰ According to his former assistant, Georges Lambelin, Alexandre Jezierski (the Polish vet who spent more than four years experimenting with locally-prepared OPVs and IPVs at his lab in Gabu, eastern Congo) was equally secretive about the process, which ceased upon his departure in late 1957. *River*; 2000, page 611.

⁸¹ Source: the assistant's official work documents from the colonial era, viewed by the author.

⁸² Personal communications: Paul and Odette Osterrieth, 1993.

⁸³ L. Quersin-Thiry, "Action of anti-cellular sera on virus infections. II. Influence on heterologous tissue cultures"; *J. Imm.*; 1959; 82; 542-552. See also the first part of this article, sub-titled: "Influence on homologous tissue cultures infected with various viruses"; *J. Imm.*; 1958; 81; 253-260.

⁸⁴ Letter from W. Bervoets to P. De Brauwere, September 17th, 1958; file H4484/1058 at the Belgian Ministry of Foreign Affairs archives.

⁸⁵ G. Courtois, H. Koprowski et al., "Preliminary report of mass vaccination of man with live poliomyelitis virus in the Belgian Congo and Ruanda-Urundi"; *Brit. Med. J.*; 1958; 2(i); 187-190.

⁸⁶ S.A. Plotkin, "Untruths and consequences: the false hypothesis linking CHAT type 1 polio vaccination to the origin of human immunodeficiency virus"; see page 820.

⁸⁷ *River*, 2000, pages 572-3.

⁸⁸ G. Barski, A. Jezierski and P. Lépine, "Sensibilité au virus de la poliomyélite *in vitro* des tissus de différentes espèces des singes d'Afrique centrale. Non réceptivité des tissus des certains mammifères", *Ann. Inst. Past.*; 1954; 86; 243-247. A. Jezierski, "Action cytopathogène des trois prototypes de virus de la poliomyélite *in vitro* sur les tissus de différentes espèces des singes d'Afrique centrale. Non réceptivité des tissus des certains animaux"; *Ann. Inst. Past.*; 1955; 89; 78-82.

⁸⁹ The paper in question reveals that CHAT pool 10A-11 had by that stage been fed to 25 Clinton infants. By comparing with the dates on which these infants were fed [see table in *River*, 2000, pages 695-698], and cross-checking with the titres fed to each infant, it was possible to calculate between which dates the paper must have been written [see: *River*, 2000, page 701]. The Moorestown trial (in a New Jersey dormitory suburb of Philadelphia) also began on January 27th, and in the book, I hypothesise that it might have involved another CHAT pool, 4B-5. With the additional information now available from Dr Plotkin, it seems that I was probably wrong, and that the Moorestown feeding also probably involved pool 10A-11. However, it seems that it involved a different substrate in New Jersey to that used in Stanleyville and Ruzizi.

⁹⁰ Dr Deinhardt's widow, Jean, has reported that he booked two seats on the plane, one for himself and one for the "box of shit", this being a cool-box containing faecal specimens from the children of Willowbrook state school, who had been experimentally infected with hepatitis. (The stools were later used to infect some of the Lindi chimps.)

⁹¹ Anon, "Monkey Business"; *Thermometer*; 1958; 9(2); 3 and 6.

⁹² It cannot be stated with certainty whether Osterrieth's assistant helped with the CHAT feeding on February 27th, with the Fox feeding on May 27th, or with both, but it makes little difference to the overall argument. Fresh vaccine had to be produced locally for every new vaccination, the assistant knows that this locally-made vaccine was fed by mouth at the army camp, and chimp tissues were available in the lab, not only during the February to April 1958 period, but after that as well (for two further shipments were sent to Deinhardt in Philadelphia, almost certainly by Osterrieth, after Deinhardt had left in April 1958). Thus both the army camp feedings are likely to have involved vaccine prepared locally in chimp cells.

⁹³ Uvira is actually four miles south of the tarmac road through the Ruzizi Valley along which the teams vaccinated, and so whether or not they vaccinated in the town itself, it seems likely that the population of the Uvira (believed to be 3,000 to 4,000 in 1958) would, just like the other local populations, have answered the call of the drums. In one interview in 1993, Dr Ninane told me that he thought he had vaccinated in Uvira, but he did not confirm this the next time I spoke with him.

⁹⁴ G. Courtois, G. Jervis. A. Flack, H. Koprowski and G. Ninane, "Preliminary report of mass vaccination of man with live poliomyelitis virus in the Belgian Congo and Ruanda-Urundi"; *Brit. Med. J.*; 1958; 2(i); 187-190. I can confirm the February 24th start date for Ruzizi from Dr Flack's diary, but not the end date. There is a line in the diary that reads "Will probably finish polio April 10???", but Dr Flack herself left Usumbura on March 30th, by which time the Ruzizi Valley part of the trial seems to have been completed. Dr Ninane told me that he had to go back to Stanleyville for more vaccine for the final (Lake Tanganyika) leg of the vaccination [*River*, 2000, page 358], so it seems reasonable to propose that he and Hector Meyus completed this leg in early April, ending on the 10th. Interestingly, the grand total of 215,504 vaccinees recorded in the *BMJ* article is correct for the entire vaccination campaign – Ruzizi Valley plus Lake Tanganyika – so Koprowski clearly *was* briefed on the lakeside feedings before he wrote that article. This once again raises the question of why he failed to mention them in the article.

⁹⁵ G.A. Jervis, "Comparative susceptibility of tissue culture cells in experimental animals and man"; *Ann. N.Y. Acad. Sci.*; 1955; 61; 848-851.

⁹⁶ Plotkin does not reveal whether Ninane telegraphed from Stanleyville, or from somewhere close to the Ruzizi Valley, like Bukavu.

⁹⁷ S.A. Plotkin, "Untruths and consequences: the false hypothesis linking CHAT type 1 polio vaccination to the origin of human immunodeficiency virus". *Phil. Trans. R. Soc. Lond. B*, 2001; 356, 815-823, see p. 818.

⁹⁸ Source: Agnes Flack's diary. According to Dr Jervis's widow, Ruth Jervis, her husband had other demands on his time, and it was always intended that he would leave Africa early, and before Dr Flack.

⁹⁹ In Agnes Flack's diary, there is no mention of Dr Jervis after the first couple of days of vaccinating, on February 24th and 25th, which suggests that, after this period, he may have been based at the Bukavu laboratory, rather than with Flack and the vaccinating teams at the Kabunambo field centre. An article dated March 7th places him in Bukavu at the start of that week (March 3rd), and gives the clear impression that, though he is working with Dr Courtois, Jervis is in overall charge of the campaign. Anon., "Debut d'une grande campagne anti-polio dans la vallee de la Ruzizi"; *Courrier d'Afrique*; March 7, 1958, page 4.

¹⁰⁰ It seems likely that Koprowski and Norton would have called in at the Bukavu lab when they drove by from Usumbura, en route for Epulu and Stanleyville, between February 2nd and 5th, 1957. [*River*, 2000, p. 614.]

¹⁰¹ Anon., "Compte-rendu de la première conférence des médecins-directeurs de laboratoire du Congo Belge et du Ruanda-Urundi (Bukavu, 25 février – 2 mars, 1957)"; unpublished document available at the library of the Institute of Tropical Medicine, Antwerp. This also reveals that the meeting of medical directors had originally been scheduled to coincide with the opening of the *Stanleyville* lab in February 1957, but that this opening had been postponed because of construction delays. This may explain why Koprowski came out first in February, 1957 and then again in September.

¹⁰² Personal research in Bukavu, March 2001. It has always struck me as surprising that so much money was being poured into building new medical laboratories in the Congo in 1957 and 1958, when independence was clearly not far away. This is all the more surprising because Belgium was still a relatively poor country, and development money for the Congo traditionally had to be engendered from within the colony itself. Perhaps it was different on this occasion.

¹⁰³ The plane made long stops in the heat of Athens and Khartoum en route to Stanleyville, making meltdown all the more likely.

¹⁰⁴ S.A. Plotkin, "Untruths and consequences", page 816, confirms that "no seed system was used" for the early pools of CHAT.

¹⁰⁵ Passaging again both amplified the vaccine, and (even more importantly) boosted its titre. (Presumably the scientist would inoculate vaccine into several culture bottles, let the virus grow, and then choose the culture with the highest titre for further amplification.) Of course, the better the titre, the more people could be immunised with the vaccine, and the more impressive were the figures that one could publish. This was the period when Koprowski, Sabin, Cox and Lépine were battling to win the race to develop an OPV that would be adopted the world over.

¹⁰⁶ F. Przesmycki et al., "Vaccination against poliomyelitis in Poland with Koprowski's live attenuated strains", in "Second international conference on live poliomyelitis vaccines: papers presented and discussions held (Washington, D.C., June 6-10, 1960)", *Pan-American Sanitary Bureau*, 1960, Scientific Publications No. 50, pp 522-531. See original vaccine titre on page 522, and compare with dilution details on page 526. The ten-fold disparity between the original titre, and that at the Warsaw lab prior to dilution, indicates that local amplification must have occurred. Other clues suggest that local passage of the vaccine virus may have happened routinely in Poland, for the article mentions that

in the previous small-scale trials (staged in Wyszkiw, Poland in 1958-9) the Polish doctors had employed “live attenuated vaccine, prepared from Koprowski’s type 1 CHAT strain”.

¹⁰⁷ In Africa, the problem was rather that of keeping the vaccine temperature below 4 degrees centigrade. Above this temperature, live poliovirus rapidly becomes inactivated. This was always the major headache involved in moving live vaccines, like CHAT, long distances around the world, and then out into the field. In reality, quite a lot of the live polio vaccine fed in places like South America, and the Congo, may have been useless, having fallen to a non-immunogenic titre because it got too warm.

¹⁰⁸ M. Bottiger et al., “Vaccination with Attenuated Type 1 Poliovirus, the CHAT strain. III. Antibody response and spread of virus in schoolchildren”. *Acta. Paed. Scand.*; 1966; 55; 422-431. Various doctors who used to work for the Stockholm labs during this period have told me that cynomolgus monkeys were exclusively used for vaccine production from about 1957 onwards.

¹⁰⁹ S.A. Plotkin, “Recent results of mass immunization against poliomyelitis with Koprowski strains of attenuated live poliovirus”, *Am. J. Pub. Health*; 1962; 52(6); 946-960.

¹¹⁰ Anon., “Requirements for production of attenuated vaccine for poliomyelitis (Koprowski strain)” [English translation of title and article made by Croatian]; *Immunolski. Zavod. Radovi*; 1964; 2; 124-125. This brief article clearly described work from the 60s, not the 50s, for the article states that no more than two passages of the primary virus from the Wistar Institute can take place, in either African green monkey kidney cells, or in WI-38.

¹¹¹ TCID₅₀ (or 50% Tissue Culture Infectious Doses), this being the titre at the point when 50% of the inoculated tissue cultures are infected by the virus.

¹¹² TCID₅₀ was the standard measure of titre of the day, though PFU (plaque-forming units), a method in which individual plaques were supposedly counted, was also sometimes used. In practice, the two methods gave comparable titres, though work by Roderick Murray and others revealed minor discrepancies.

¹¹³ F. Przesmycki et al., “Vaccination against Poliomyelitis in Poland with Types 1 and 3 Attenuated Viruses of Koprowski”; *Bull. W.H.O.*; 1962; 26; 733-743.

¹¹⁴ A personal letter written in April 1962 by Drago Ikic to Koprowski’s chief lab technician and right-hand-man, Tom Norton, reveals that Croatia was indeed supplied with CHAT seed-lots; Ikic requested that a half litre of each new strain of seed virus, or at least “as much as possible”, should be sent to him “as usual”. But the evidence suggests that the other European countries which “made their own vaccines”, such as Poland, Switzerland and Sweden, may have done so by onward passage of the CHAT vaccine strains, not the seed-lots. At another point in the interview I asked Dr Koprowski about the adverse results reported by George Dick and David Dane, who had fed his previous polio vaccines, SM N-90 and TN, to 14 and 190 people (respectively) in Belfast and Oxford (U.K.) in 1956, and found that they became more virulent (in the case of TN, much more virulent) after passage through the human gut. Koprowski replied that the British doctors had passaged the vaccine virus again in their own lab, implying that any shortcomings in the vaccines were their fault, not his. When I put this to doctors Dick and Dane, they both vigorously denied it. The vaccines they fed were exactly what Koprowski had sent them, they insisted. But the significant thing was that Koprowski’s immediate instinct had been to claim that the vaccine had been amplified locally in Belfast.

¹¹⁵ No use of Koprowski’s vaccines in South Africa is reported in the literature, and it is stated that Sabin’s OPVs were eventually preferred to Koprowski’s in that country. Koprowski, however, did mention that a vaccine trial had been planned in the town of East London, though he later acknowledged that it might not have occurred. [River, 2000, p. 471.] My suspicion is that he may have supplied vaccines for such a trial but, as in Kenya, they were never used.

¹¹⁶ H. Koprowski, personal communication, December 1993. Even though Dr Koprowski was quite open about the theory of local amplification, he was less helpful about the practical aspects. I continually asked him about the quantities of vaccine which had to be sent abroad, which gave him an opportunity to explain the mechanics of the process, and the fact that only small quantities needed to be dispatched. But in response, he merely said that he couldn’t remember any details. And whenever I pressed him for specific details about the operation of the Congo trials, he either couldn’t remember, or gave what I found to be confusing information. An example came when I understood him to have confirmed that doctors Flack and Jervis had flown out from America with the vaccine for the Ruzizi trial in February 1958. Immediately he snapped back: “I said *possibly*. This is all invention, hypothesis and fantasy.” At another point, he told me that the first vaccine used in the Congo had been made by the Wistar, whereas later vaccines had been made, he thought, by the Wistar and by the Belgian firm, RIT. (River, 2000, pages 467-468.) At this stage of the interview, he avoided mentioning anything about local preparation of the vaccine in the Congo.

¹¹⁷ G. Courtois, G. Jervis, A. Flack, H. Koprowski and G. Ninane, "Preliminary report of mass vaccination of man with live poliomyelitis virus in the Belgian Congo and Ruanda-Urundi"; *Brit. Med. J.*; 1958; 2(i); 187-190. Gaston Ninane told me that despite Courtois' being lead author and he, Ninane, last author, this article had actually been prepared by Koprowski (and perhaps Norton) at the Wistar, and merely sent to the Belgians for checking.

¹¹⁸ G. Courtois, "Vaccination antipoliomyelitique par virus vivant au Congo Belge"; *Ann. Soc. Belge Med. Trop.*; 1958; 38; 805-816.

¹¹⁹ David Ho was one of the members of the Wistar-appointed "AIDS/Poliovirus Advisory Committee", which sat three times in 1992 to respond to the controversy caused by the Tom Curtis article in *Rolling Stone*. Some years after he had given me a copy of the documents furnished by the Wistar Institute to the Advisory Committee, Dr Ho told me that he "got into trouble" from other unnamed scientists for doing this.

¹²⁰ Anon., "History of the use of CHAT strain 'Type 1' attenuated polio virus in humans", undated, but clearly written between January 23rd and January 27th, 1958; (single-page document provided by the Wistar Institute to the AIDS/Poliovirus Advisory Committee in 1992). My calculations on the date of origin for the paper tie in well with other historical evidence.

¹²¹ This is not explicitly stated in the one-page paper. However, immunogenicity trials are traditionally conducted by feeding aliquots of the vaccine, and ten-fold serial dilutions of same – and the Clinton infants were fed 6.7, 5.7, 4.7 and 3.7 log doses of CHAT 10A-11. Moreover, the titre details on that single page (both for pool 10A-11 and for the first pool of CHAT, which may have been pool 8) match those given in the published paper on the Clinton trials: [S.A. Plotkin, H. Koprowski and J. Stokes Jr., "Clinical trials in infants of orally administered attenuated poliomyelitis virus"; *Pediatrics*; 1959; 23; 1041-1062.], confirming that the titre of pool 10A-11 at the Wistar Institute was log 6.7.

¹²² The "units" in this particular instance are unspecified. The Wistar scientist (probably Tom Norton) may have meant TCID₅₀, PFU (plaque forming units), or the "cytopathogenic units" referred to by Courtois. I am informed that they were taken by most virologists of the era to have equivalent values. In a study conducted in 1959 by Dr Roderick Murray and colleagues from the Division of Biologics Standards at the National Institutes of Health on different OPV strains (three each from Koprowski, Sabin and Cox), it was found that both TCID₅₀ values and PFU values for the Type 1 vaccines (as measured at the NIH) were usually less than the TCID₅₀ values as measured at the source laboratory. In the case of CHAT (pool 13), PFU titre as measured at the NIH was 0.1 of a log dose *higher* than TCID₅₀ as measured by the Wistar, but this minor difference falls well within the range of testing error. [R. Murray et al., "Comparative virulence for rhesus monkeys of poliovirus strains used for oral administration"; in *First international conference on live poliovirus vaccines: papers presented and discussions held* (Washington D.C., June 22-26, 1959), Pan American Sanitary Bureau, 1959, Scientific publication No. 44, pp. 39-64. See table 2 on page 42.]

¹²³ M.P. Chumakov et al., "On the course of mass immunization of the populations in the Soviet Union with the live poliovirus vaccine from Albert B. Sabin's strains", in "Second international conference on live poliomyelitis vaccines: papers presented and discussions held (Washington, D.C., June 6-10, 1960)", *Pan-American Sanitary Bureau*, 1960, Scientific Publications No. 50, pages 413-428; see figure 1 on page 418.

¹²⁴ Murray et al., "Comparative virulence for rhesus monkeys of poliovirus strains used for oral administration"; table 2 on page 42.

¹²⁵ S. Wain-Hobson, personal communications, 1999 and 2000. *River*, 2000, pages 852-858.

¹²⁶ S. Wain-Hobson, personal communication, October 2001.

¹²⁷ E.D. Cooper and W.J. Roberston, "Problems relating from the use of live attenuated poliomyelitis virus type 1 in a mass campaign in a large urban area", *S. Af. Med. J.*; 1961; 35; 232-235. Other papers on this subject cite 1958, rather than 1957, as the year when local vaccine preparation began in South Africa, but the fact that in this article the start date of 1957 is sourced to a "personal communication" (probably either from Gear or from Hubert Malherbe, who trained under Albert Sabin in the US in 1957, and who is known to have returned to South Africa with the three Sabin strains) suggests that this version is probably correct.

¹²⁸ James Gear, personal communication, 1993.

¹²⁹ *River*, 2000, pp 520-521.

¹³⁰ It is not known whether Koprowski went ahead with the visit, but the South African literature reveals that at around this time it was decided to use Sabin's strains in preference to Koprowski's.

¹³¹ Anon., "City polio proposal. Talks held on trial of new vaccine in Kenya"; *East African Standard* (Nairobi); February 1, 1957.

¹³² J. Gear, “The South African poliomyelitis vaccine”; *S. Af. Med. J.*; 1956; 30; 587-594. The British, also, were interested in this field. An article in the same journal in September 1955 reveals that a Medical Research Council expedition to Fajara, Gambia, earlier that year, to examine the viability of using African primate kidneys for polio vaccine production [*River*, 2000, p. 388] was related to OPV, rather than IPV, research. [R. Turner, “Active immunisation against poliomyelitis”; *S. Af. Med. J.*; 1955; 29; 833-844.] The minutes of the committee meeting following this expedition are (unusually) missing from MRC files, and may have been effectively “censored”, according to the MRC official with whom I spoke. In 1997, I visited the Gambia, and the current director of the MRC labs at Fajara, Dr Hilton Whittle, ended up failing to give a promised interview under rather extraordinary circumstances. The same Dr Whittle chaired the final session of the Royal Society conference in 2000, a session which featured Dr Weiss’s closing summary, and which was felt by some parties to be biased in terms of selection of speakers from the floor. Bill Hamilton’s partner, Luisa Bozzi, was passed over in favour of an apparently preplanned anti-OPV “closing line” from John Maynard Smith, and Walter Nelson-Rees was so disgusted at what he considered Dr Whittle’s deliberate ignoring of him that he eventually walked out.

¹³³ INEAC: *L’Institut National pour l’Etude Agronomique du Congo Belge*, based in Yangambi.

¹³⁴ *River*, 2000, page 611.

¹³⁵ G. Barski, A. Jezierski and P. Lépine, “Sensibilite au virus de la poliomyélite *in vitro* des tissus de différentes espèces des singes d’Afrique centrale. Non receptivite des tissus des certains mammiferes”, *Ann. Inst. Past.*; 1954; 86; 243-247. A. Jezierski, “Action cytopathogene des trois prototypes de virus de la poliomyélite *in vitro* sur les tissus de différentes espèces des singes d’Afrique centrale. Non receptivite des tissus des certains animaux”; *Ann. Inst. Past.*; 1955; 89; 78-82.

¹³⁶ A. Jezierski, “Atténuation des trois types des virus de la poliomyélite sur les tissus de singes de l’espèce colobus. Virus vivant modifiés et leur application par différentes voies sur les singes”; *Ann. Soc. Belge Med. Trop.*; 1959; 39; 69-96. A. Jezierski, “Destruction of poliovirus in the digestive tract of chimpanzees by means of specific high-titre gamma-globulin”; *Ann. Soc. Belge Med. Trop.*; 1960; 40; 169-171, plus related discussion on 172-181.

¹³⁷ *River*, 2000, p. 610.

¹³⁸ A. André, G. Courtois, G. Lennes, G. Ninane and P.M. Osterrieth, “Mise en évidence d’antigènes de groupes sanguins A, B, O et Rh chez les singes chimpanzés”; *Ann. Inst. Past.*; 1961; 101; 82-95; see Mambasa group on page 87, and note the interesting tautology in the title: “chimpanzee monkeys”.

¹³⁹ G. Courtois, “Vaccination antipoliomyélique par virus vivant au Congo Belge”; *Ann. Soc. Belge Med. Trop.*; 1958; 38; 805-816.

¹⁴⁰ E. Hooper, “Experimental oral polio vaccines and acquired immune deficiency syndrome”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 803-814.

¹⁴¹ The names of these witnesses will be published at an appropriate time.

¹⁴² Although I asked several officials, none could furnish an explanation for when the other papers had been removed, and what had happened to them afterwards.

¹⁴³ A.B. Sabin, “Notes on international requirements for live, oral poliomyelitis vaccine”, [letter dated 29 October 1960 received from Dr Albert B. Sabin], WHO internal document WHO/BS/IR/87.

¹⁴⁴ A.B. Sabin and L.R. Boulger, “History of Sabin attenuated poliovirus oral live vaccine strains”; *J. Biol. Stand.*; 1976; 1; 115-118.

¹⁴⁵ Anon., “Le vaccin du professeur Lépine sera vendu par une firme pharmaceutique americaine”; *L’Echo du Stan* (Stanleyville); June 24th, 1957. This brief report from AFP makes it clear that this is referring to a live vaccine from Lépine, not his more well-known IPV.

¹⁴⁶ B.H. Sweet and M.R. Hilleman, “Detection of a ‘non-detectable’ simian virus (vacuolating agent) present in rhesus and cynomolgus monkey-kidney cell culture material. A preliminary report”, in “Second international conference on live poliomyelitis vaccines: papers presented and discussions held (Washington, D.C., June 6-10, 1960)”, *Pan-American Sanitary Bureau*, 1960, Scientific Publications No. 50, pages 79-85.

¹⁴⁷ For a good article about the risks of antigenic drift (reversion to virulence), written soon after a sabbatical at the Wistar during which he had worked with CHAT, see: S. Gard, “Immunological strain specificity within Type 1 poliovirus”; *Bull. W.H.O.*; 1960; 23; 235-242.

¹⁴⁸ H. Koprowski; “Immunization against poliomyelitis with living attenuated virus”; *Am. J. Trop. Med.*, 1956; 5; 440-452.

¹⁴⁹ Although Weller described his team of Uganda-based virologists as one that might be “preparing to investigate febrile entities of unknown etiology”, he made it clear in the same paragraph that tissue culture techniques applied as much to “the propagation of [viral] agents in quantity” as to methods of “viral isolation”.

¹⁵⁰ T.H. Weller, "Problems revealed by the expanding use of tissue culture procedures in studies of infectious agents"; *Am. J. Trop. Med.*, 1956; 5; 422-429.

¹⁵¹ *River*, 2000, page 549.

¹⁵² G. Courtois, H. Koprowski et al., "Preliminary report of mass vaccination of man with live poliomyelitis virus in the Belgian Congo and Ruanda-Urundi"; *Brit. Med. J.*; 1958; 2(i); 187-190. In fact, it seems that CHAT feeding in Africa may have begun in January 1957. When Hilary Koprowski and Tom Norton appeared in Nairobi (Kenya) at the end of that month, Koprowski announced to fellow-scientists and the local press that "we feel we have now reached the point where large-scale clinical trials [of OPV] should be attempted". "We are anxious", he went on, "to have it [his OPV, which apparently had "no name"] tried out in pilot schemes in any country in the world where it will be under the supervision of responsible medical people". He went on to invite participation by the medical communities in both Kenya and South Africa, which he said he would visit later in his trip, and his speech prompted a front page article and an editorial in one of Nairobi's two leading dailies. [Anon., "City polio proposal. Talks held on trial of new vaccine in Kenya"; *East African Standard* (Nairobi); February 1, 1957.] The article also stated that he and Norton, who were "carrying supplies of their vaccines" were leaving that very day for Stanleyville in the Belgian Congo, where they would "carry out experiments in chimpanzees". However, interviews have revealed that Koprowski also distributed some of his polio vaccines in Kenya. He gave Type 1 vaccine capsules (which may have contained either CHAT or his previous Type 1 strain, SM N-90) to both the man in charge of vaccine procurement for British East Africa, Dr Geoffrey Timms, and to Dr Jimmy Harries, who was at risk because he worked with polio patients in Mombasa. The former were apparently destroyed by the Kenyan health authorities, following the furore about the Northern Ireland trials that erupted a few days after this, when Timms' brother-in-law, George Dick, and his colleague, David Dane, reported that Koprowski's Type 2 polio vaccine strain, TN, reverted to virulence after human passage, and was therefore too risky to be tested further in human subjects; the two British doctors also raised questions about the suitability of Koprowski's Type 1 strain, SM N-90. [*River*, 2000, Chapter 15, especially pages 223-224.] However, by this stage Dr Harries had already given the capsules to his family to eat with their Sunday lunch. [*River*, 2000, page 618.] Since Koprowski's 1958 *BMJ* article reveals that CHAT vaccination began in Stanleyville in February 1957, this strongly suggests that it was the same CHAT vaccine that Koprowski handed out in Kenya, and this is further supported by the fact that CHAT capsules were fed in the Congolese town of Aketi in May 1957.

¹⁵³ Anon., "Live virus in the jungle"; *Time*; August 11, 1958, p. 30. The accompanying photo could only have been taken during February 1957, for Osterrieth was not present during Koprowski's second visit to Stanleyville in September and October of that year.

¹⁵⁴ *River*, 2000, page 383.

¹⁵⁵ From published reports in the formal medical literature we know just one detail of the polio work that was conducted on nearly 400 Lindi chimps between June 1956 and February 1958. This is that CHAT vaccine and its Type 3 cousin, Fox, were each tested intraspinally on five chimpanzees, without apparent ill effect. This hardly suggests that the safety testing of CHAT and Fox was the reason why Lindi camp had been created. Later, in 1959, Koprowski claimed at a polio conference that he and Courtois had tested an additional 29 chimps intraspinally with different vaccine strains. However, he failed to provide any significant details apart from the fact that the Type 1 predecessor to CHAT, SM, was among the vaccines tested. [Discussion by Koprowski in: in "First international conference on live poliomyelitis vaccines: papers presented and discussions held (Washington, D.C., June 22-26, 1959)", *Pan-American Sanitary Bureau*, 1959, Scientific Publications No. 44, p. 201.] This mass testing of vaccine safety in chimps is interesting, in that Albert Sabin had already reported in January 1957 (at a conference which Koprowski attended) that chimps were far less sensitive to polioviruses injected into the central nervous system than were lower monkeys, like macaques. [*River*, 2000, pp 528-529. A.B. Sabin, "Properties of attenuated polioviruses, and their behaviour in human beings"; in T.M. Rivers (ed.), "Cellular Biology, Nucleic Acids and Viruses", *Special Publications of the New York Academy of Sciences*; 1957; 5; 113-127.] Chimpanzees did not, in short, provide a good barometer of vaccine safety. At another discussion session at the 1959 polio vaccine conference, Dr Courtois revealed that 30 of the Lindi chimps had been fed with large amounts of virulent Type 1 and Type 2 polioviruses, Mexican and YSK, to check whether these viruses were capable of paralysing chimpanzees; he reported that nine animals became paralysed. As for the testing of immunogenicity, there is a record from Dr Fritz Deinhardt's hepatitis databook, which dates from 1959, of just one such experiment having been carried out. It was not, however, conventional vaccination and challenge, because it involved an assessment of Koprowski's previous Type 1 vaccine, SM N-90, which was fed to 14 chimps that were subsequently challenged with virulent Type 2 poliovirus. (It had long been one of

Koprowski's pet theories that Type 1 vaccine might protect against both Types 1 and 2 poliomyelitis. The hypothesis, as we now know, is incorrect.) Because of the dearth of documentation about the Lindi polio work, there is no evidence that conventional vaccination and challenge work (such as vaccinating with Type 1 and then challenging with virulent Type 1) was ever carried out.

¹⁵⁶ Anon., "Guerre ⋄ la polio dans la brousse stanleyvilloise"; *Le Stanleyvillois*; February 11, 1957, pp 1 and 4.

¹⁵⁷ It was originally intended that the new lab at Stanleyville would have been opened in February 1957 (which may well explain the timing of Koprowski's first visit), but apparently there was a hold-up with the building work, which delayed the formal opening ceremony by seven months. However, Tom Norton's photos from February and March 1957 reveal that the building itself was completed, but that it apparently lacked windows. In any case, I am told that some of the individual rooms in the new lab were open and functioning well before September, 1957.

¹⁵⁸ Based on the annual reports of the *Laboratoire Médical de Stanleyville* for 1957 and 1958. Even though the official dates recorded in these reports for triannual leaves are not absolutely precise (for instance, Dr Osterrieth is recorded as having returned to Stanleyville on Feb 23rd, 1958, even though we know he returned some three weeks earlier), they do give a good general idea of when individual doctors were present at, or absent from, the lab.

¹⁵⁹ *River*, 2000, pp. 720-721.

¹⁶⁰ This figure of 60 is nicely supported by the Lindi camp records, which reveal that by far the greatest influx of chimpanzees was during the first seven months of the camp's operation, starting in June 1956. Over 200 had been admitted by mid-January 1957, of which only 60 were still alive at the start of February. If we assume that there was a death rate from natural causes of 25%, this means that over 100 chimps were sacrificed in seven months – before Koprowski and Norton had even arrived for their first visit. Even if the death rate was higher to begin with – say 50% - this still means that 70 chimps were sacrificed in seven months. Either figure is consistent with 60 chimps being "used" by the end of 1956.

¹⁶¹ The Lederle lab notebooks [see later] reveal that the faecal virus from patient "Charlton" (which was the origin of the CHAT strain) was being plaqued out in July 1956, so the earliest known version of CHAT cannot have been given to the great Italian virologist, Renato Dulbecco, to test for neuropathogenicity until the final months of 1956. Dulbecco discussed Koprowski's new OPV strains at the polio conference in New York City in January 1957 [R. Dulbecco, "Discussion", in T.M. Rivers (ed.), "Cellular Biology, Nucleic Acids and Viruses", *Special Publications of the New York Academy of Sciences*; 1957; 5; 138-139], and later identified the versions he had been given as "Charlton, plaque 20" and "SM N-90, pool 21". [R. Dulbecco, "Mutants of poliomyelitis viruses with reduced efficiency of plating in acid medium and reduced neuropathogenicity"; *Virology*; 1957; 4; 141-155.] The timings strongly suggest that CHAT research in the Congo did not begin before February 1957, when the new vaccine arrived with Koprowski and Norton – and was tested in Lindi chimps, and local humans. It therefore seems probable that SM N-90, pool 14, was used in the Stanleyville and Lindi research staged before February 1957. But when did pool 14 arrive in Stanleyville? Ghislain Courtois was in central and south America on a yellow fever tour in 1956, and would probably have passed through the U.S., so perhaps he met Koprowski then, and brought pool 14 back to Stanleyville with him. However, in an article published in April 1958, Courtois claimed he first met Koprowski in New York in 1955. [Anon. "Ruzizi. Campagne de vaccination massive contre le poliomyélite", *Temps Nouveau d'Afrique*, April 13, 1958.] This version of events, published in a local African newspaper three months before Koprowski's "official" account [*Brit. Med. J.*; 1958; 2(i); 187-190] was published, is intriguing. Koprowski's version is that he was initially put in touch with Courtois by the Stanleyville vet, Tad Wiktor, whom he met at the Muguga rabies conference in Kenya in July 1955. However, Courtois was back in Stanleyville from his 1955 foreign tour by the time that Muguga started, and Koprowski only returned to the U.S. in late August. Unless Courtois flew back to America later in the year (which would have been extremely unusual, so soon after his previous visit), or unless he was wrong about the date, we are left to conclude that he must have met Koprowski in New York in spring 1955, before Koprowski went to Muguga. The latter scenario raises the possibility that whatever role Wiktor played in the Stanleyville OPV research, it was not the alleged one – that of introducing the two men.

¹⁶² L. Hayflick, "The establishment of a line (WISH) of human amnion cells in continuous cultivation"; *Exp. Cell Res.*; 1961; 23; 14-19. L. Hayflick, S.A. Plotkin, T.W. Norton and H. Koprowski, "Preparation of poliovirus vaccines in a human fetal diploid cell strain"; *Am. J. Hyg.*; 1962; 75; 240-258.

¹⁶³ According to the definitions of Leonard Hayflick, cell strains consist of cells which have divided several times, up to a maximum of about fifty, *in vitro*, and which retain the characteristics of normal

cells; by contrast cell lines have become immortalised, have changed karyology, and reproduce ad infinitum.

¹⁶⁴ B.F. Elsworth and R.B. Stricker, "Polio vaccines and the origin of AIDS"; *Med. Hypoth.*; 1994; 42; 347-354. B. Goldberg and R.B. Stricker, "Bridging the gap: human diploid cell strains and the origin of AIDS"; *J. Theoret. Biol.*; 2000; 32; 1068-1084.

¹⁶⁵ I believe that there are also some other, less important shortcomings in the Elsworth/Goldberg/Stricker hypothesis, about which I wrote to one of them in September 1999, but without receiving a proper reply. The only really serious flaw, however, appeared to be that relating to the timing. I believe that if one adapts their hypothesis so that it applies to cell lines (such as human amnion lines) which had been overtaken by HeLa, rather than to cell lines like WISH or human diploid cell strains, then the timing difficulties disappear. I would like to add that Billi Goldberg, in particular, is deserving of considerable praise for keeping open lines of communication about these topics (mainly through distributing relevant articles by e-mail to supporters of both major theories of origin).

¹⁶⁶ B. Forghani et al., "Advantages of a human immunodeficiency virus type 1 (HIV-1) persistently infected HeLa T4+ cell line for HIV-1 indirect immunofluorescence assay"; *J. Clin. Microbiol.*; 1991; 29(10); 2266-2272.

¹⁶⁷ L. Hayflick, "History of cell substrates used for human biologicals"; *Develop. Biol. Standard.*, 1989; 70; 11-26; see page 14.

¹⁶⁸ L. Quersin-Thiry, "Action of anti-cellular sera on virus infections. I. Influence on homologous tissue cultures infected with various viruses"; *J. Imm.*; 1958; 81; 253-260. L. Quersin-Thiry, "Action of anti-cellular sera on virus infections. II. Influence on heterologous tissue cultures"; *J. Imm.*; 1959; 82; 542-552.

¹⁶⁹ G. Henle and F. Deinhardt, "The establishment of strains of human cells in tissue culture"; *J. Imm.*; 1957; 79; 54-59.

¹⁷⁰ W. Henle, G. Henle and F. Deinhardt, "Studies in Viral Hepatitis"; *Annual report to the commission on viral infections of the armed forces epidemiological board*; March 1, 1957 to February 10, 1958; p. 2.

¹⁷¹ J.F. Enders, T.H. Weller and F.C. Robbins, "Cultivation of the Lansing strain of poliomyelitis virus in cultures of various human embryonic tissues"; *Science*; 1949; 109; 85-87. This brief, but oft-cited article was published in 1949, and by the first half of the fifties, the use of tissue culture had revolutionised virology research.

¹⁷² L. Hayflick, personal communication, 1993.

¹⁷³ W. McLimans, "Discussion", in *Papers and discussions presented at the Fourth International Poliomyelitis Conference* (Philadelphia: Lippincott, 1958), pages 284-287.

¹⁷⁴ F.W. Sheffield and G.M. Churcher, "The serial propagation of poliomyelitis viruses in cells derived from rabbit embryo kidney"; *Brit. J. Exp. Path.*; 1957; 38; 154-159.

¹⁷⁵ Anon., *Registry of animal cell lines certified by the cell culture collection committee* (Washington, D.C.: US Department of Health Education and Welfare, Public Health Service, 1964), page 1.

¹⁷⁶ M. Gold, *A Conspiracy of cells. One woman's immortal legacy and the medical scandal it caused*. (New York: SUNY Press, 1986), pp 27-31.

¹⁷⁷ W.A. Nelson-Rees and R.R. Flandemeyer, "HeLa cultures defined"; *Science*; 1976; 191; 96-98. W.A. Nelson-Rees, D.W. Daniels and R.R. Flandemeyer, "Cross-contamination of cells in culture"; *Science*; 1981; 212; 446-452.

¹⁷⁸ Nelson-Rees tells me he strongly suspects that Chang's conjunctival cells were also a HeLa contaminant.

¹⁷⁹ WISH began life as a human amnion cell culture obtained at the birth of Leonard Hayflick's daughter (hence the name: Wistar Institute Susan Hayflick) and it transformed into a cell line 35 days later, on December 31st, 1958. Years later it was revealed by Walter Nelson-Rees to have been immortalised because of HeLa contamination.

¹⁸⁰ W. A. Nelson-Rees, "Responsibility for truth in research"; *Phil. Trans. Roy. Soc. Lond. B*; 2001; 356; 849-851.

¹⁸¹ J.R. Masters, "HeLa cells 50 years on: the good, the bad and the ugly"; *Nature Reviews Cancer*; 2002; 2; 315-319.

¹⁸² J.C.N. Westbrook et al., "Transformation of normal cells in tissue culture: its significance relative to malignancy and virus vaccine production", *Brit. J. Exp. Path.*; 1957; 38; 138-154.

¹⁸³ W. Nelson-Rees, personal communication, June 2002.

¹⁸⁴ H. Koprowski, "Living attenuated poliomyelitis virus as an immunizing agent of man"; *S. Af. Med. J.*; 1955; 29; 1134-1142; see table 14 on page 1141.

¹⁸⁵ H. Koprowski and S.A. Plotkin, *Notes on acceptance criteria and requirements for live poliovirus vaccines*; WHO internal document WHO/BS/IR/85; November 1, 1960; see especially pages 7-10.

¹⁸⁶ Just prior to this passage, Koprowski and Plotkin had written: "Since there is no evidence from the epidemiological surveys of a vaccinated population indicating widespread harmful effects of the non-polio viruses present in the vaccine, and since elimination of these viruses from each lot of vaccine prepared from monkey kidney may present insurmountable obstacles for successful launching of the manufacture of the vaccine, the following requirements are proposed, based on a realistic rather than an idealistic solution to the problem." The casual reference to a lack of *widespread* harmful effects in vaccinated populations is telling, as is the emphasis on commercial viability.

¹⁸⁷ L. Hayflick and P.S. Moorhead, "The serial cultivation of human diploid cell strains"; *Exp. Cell Res.*; 1961; 25; 585-621. In a communication in 1997, Hayflick stated that this same article (presumably bar certain updates) had been submitted to, and rejected by, the *Journal of Experimental Medicine* in 1960. See: L.A. Hayflick, "Mortality and immortality at the cellular level. A review", submitted August 1, 1997, and available on . My thanks to Billi Goldberg for alerting me to this unpublished article.

¹⁸⁸ L. Hayflick, "The establishment of a human cell line (WISH) of human amnion cells in continuous culture", *Exp. Cell Res.*; 1961; 23; 14-20.

¹⁸⁹ W. Nelson-Rees, personal communications, April and June 2002.

¹⁹⁰ There are countless examples, of which I shall cite just one. Several trials of DDT and the organochlorine pesticide, Lindane, were staged in the Ruzizi Valley in the fifties. In one such trial, so much Lindane was put into a tributary of the Ruzizi river, that the fish were killed as far as 50 kilometres downstream. It is reported that Africans flocked to the river to partake of the easy catch. It is even possible that the immunosuppressive effects of such chemicals may have rendered local populations more vulnerable to the effects of a primordial HIV.

¹⁹¹ For comments on "volunteers" in the African CHAT trials, see: *River*; 2000; pages 733-734, 736.

¹⁹² W.D. Hamilton, undated, part of hand-written note written in margins of draft of (unsent) letter from author to B. Elswood.

¹⁹³ Joan Phillips, personal communication, 1995.

¹⁹⁴ *River*, 2000, page 483.

¹⁹⁵ S.A. Plotkin, "Untruths and consequences: the false hypothesis linking CHAT type 1 polio vaccination to the origin of human immunodeficiency virus". *Phil. Trans. R. Soc. Lond. B*, 2001; 356, 815-823, see p. 818. Here, Dr Plotkin states that he has found a note in his files to the effect that CHAT lot 101 from RIT had been used in Ruanda-Urundi in late 1959 or early 1960. It now appears that on this detail, at least, he may well be correct, and this may indeed have been the first time that Belgian-made vaccine was used in Africa (It now seems that previously the vaccinators had used Wistar-made vaccine, albeit locally amplified in the Congo.)

¹⁹⁶ In 1997, Dr Thiry's last-minute refusal of an interview about these events was accompanied by dramatic accusations that I was misleading my research assistant (who, according to Dr Thiry, was apparently too young to know her own mind), and that I was "damaging vaccinations and reputations". *River*, 2000, pp 781-783. Dr Thiry, I have since learnt, has a mixed reputation among her contemporaries. According to one fellow-virologist, she is "Koprowski's humble servant", and has "skin as thick as cowhide".

¹⁹⁷ G. Courtois, letter to Jan Stijns in Leopoldville, February 17, 1961.

¹⁹⁸ J.P. Delville and S.R. Pattyn, "Epidémiologie de la poliomyélite au Congo Belge et au Ruanda-Urundi. Etat actuel de nos connaissances"; *Ann. Soc. Belge Med. Trop.*; 1958; 38; 283-292.

¹⁹⁹ S. Pattyn, "A review of enteroviruses in the Belgian Congo"; *Trop. Geog. Med.*; 1962; 14; 71-79.

²⁰⁰ A. Lebrun, J. Cerf, H.M. Gelfand, G. Courtois, S.A. Plotkin and H. Koprowski, "Vaccination with the CHAT strain of Type 1 attenuated poliomyelitis virus in Léopoldville, Belgian Congo. I. Description of the city, its history of poliomyelitis, and the plan of the vaccination campaign"; *Bull. W.H.O.*; 1960; 22; 202-213. S.A. Plotkin, A. Lebrun and H. Koprowski, "Vaccination with the CHAT strain of Type 1 attenuated poliomyelitis virus in Léopoldville, Belgian Congo. II. Studies of the safety and efficacy of vaccination"; *Bull. W.H.O.*; 1960; 22; 215-234. M. Vandeputte, "Endemicity of the enteroviruses in Leopoldville"; *Bull. W.H.O.*; 1960; 22; 313-318.

²⁰¹ G. Henle and F. Deinhardt, "The establishment of human cells in tissue culture"; *J. Imm.*; 1957; 79; 54-59.

²⁰² The same happens with HIV-infected macrophages, as illustrated, for instance, in: V. Maréchal et al., "Human immunodeficiency virus type 1 entry into macrophages mediated by macropinocytosis"; *J. Virol.*; 2001; 75(22); 11166-11177.

²⁰³ *River*, 2000, p. 661.

²⁰⁴ I do not recall seeing any figures (or even estimates) for AIDS deaths from HIV-2, HIV-1 Group O, or HIV-1 Group N. The combined figure of 20,000 is my own very approximate estimate, as of June 2002.

²⁰⁵ Anon., “Application au Congo du nouveau (antipolyo) du Dr. Koprowski”; *L’Avenir* (Leopoldville); August 9th/10th, 1958.

²⁰⁶ S.A. Plotkin, D.E. Teuwen, A. Prinzie, and J. Desmyter, “Postscript relating to new allegations made by Edward Hooper at the Royal Society discussion meeting on 11 September 2000”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 825-829; see page 827.

²⁰⁷ S.R. Pattyn, “Anti-poliomyelitic vaccination in tropical countries”; *Trop. Geog. Med.*; 1964; 16; 4-9. In this article, Pattyn also points out that for an oral polio vaccine, “shipping and conservation are difficult”, but that it can be “transported in concentrated form, to be diluted immediately before use”. Once again, the reference is only to dilution, and not to local amplification. And yet it is quite clear that these vaccines were routinely amplified in locally-available tissue culture, in the Congo as elsewhere.

²⁰⁸ There were apparently four quite impressive labs sited close together in Elisabethville – medical, veterinary and hygiene labs, together with the local branch of IRSAC (*Institut pour la Recherche Scientifique en Afrique Centrale*). There also appears to have been a lab at the local headquarters of FOREAMI [see main text], and there may have been others.

²⁰⁹ J.P. Delville and S.R. Pattyn, “Epidémiologie de la poliomyélite au Congo Belge et au Ruanda-Urundi. Etat actuel de nos connaissances”; *Ann. Soc. Belge Med, Trop.*; 1958; 38; 283-292.

²¹⁰ S. R. Pattyn, “A review of enteroviruses in the Congo”; *Trop. Geog. Med.*; 1962; 14; 71-79.

²¹¹ *River*, 2000, pages 567-568.

²¹² Whether pool 13 of CHAT was initially manufactured in the U.S. and then forwarded to the lab in Brussels (as Gelfand now proposes), or initially manufactured in Belgium is uncertain. It is also no longer of importance, because it is now clear that the vaccine was later amplified in locally-available substrates in the Belgian Congo. Despite my frequently asking, Dr Gelfand was unable to give me any idea about the quantity of vaccine which he had carried to Leo. I believe that it was probably just a small bottle or two, and that this original vaccine was then amplified locally. Once again, it is the local amplification that is the step which, even now, those involved with the Congo CHAT research wish to keep a secret.

²¹³ In two phone interviews in 1996 and 2000, Dr Gelfand gave me six different answers on this question, ranging from: “I had the vaccine, I believe, for use throughout the Congo”, and “I feel with a moral certainty that I must have carried the vaccine around [to the other cities]”, to “My preference for recollection is that I did not carry the vaccine [to the other cities]. I’m not certain either way”. Since more of the views he expressed were positive than negative, and since the positive views were more forthrightly stated, I reported in my Royal Society speech that “Dr Gelfand is unsure, but believes that he also carried the vaccine onwards to Stanleyville, Bukavu and Elisabethville”. In retrospect, I accept that perhaps it would have been better had I written “believes he *may also have* carried the vaccine onwards...” However, Dr Gelfand has now reported to Dr Plotkin (as quoted in Plotkin’s “Postscript” article) that what he actually told me was: “I am unsure, but it is very *unlikely* that I carried vaccine from Leo to other cities in the Congo”. This is untrue. At no point in our two phone conversations, or in his several letters, did Dr Gelfand ever say, or intimate, that this was very unlikely.

²¹⁴ H. Gelfand, personal communication, December 1996.

²¹⁵ See for instance, Gelfand’s previous paper about poliomyelitis in Liberia (another African nation with close links to the US), in which he concluded that it was not then justifiable to vaccinate the indigenous population against polio, but that foreign visitors should consider vaccination. [H. Gelfand and M. J. Miller, “Poliomyelitis in Liberia”; *Am. J. Trop. Med. Hyg.*; 1956; 5; 791-796.] (Unfortunately, the journal details are not marked on my copy, but I believe that this is probably the correct reference. The page numbers are correct, and the article was almost certainly published in 1956.)

²¹⁶ A. Lebrun et al., “Vaccination with the CHAT strain of Type 1 attenuated poliomyelitis virus in Léopoldville, Belgian Congo. I.”; *Bull. W.H.O.*; 1960; 22; 202-213.

²¹⁷ S.A. Plotkin, A. Lebrun and H. Koprowski, “Vaccination with the CHAT strain of Type 1 attenuated poliomyelitis virus in Léopoldville, Belgian Congo. II. Studies of the safety and efficacy of vaccination”; *Bull. W.H.O.*; 1960; 22; 215-234.

²¹⁸ *River*; 2000; pages 737-739.

²¹⁹ *River*; 2000; p. 529.

²²⁰ Anon., “Wistar Institute is both monument and prototype of modern research”; *Scope Weekly*; May 21, 1958, pp. 6-7.

²²¹ S. Plotkin, letter to F. Deinhardt, May 28, 1959 (made available by Jean Deinhardt).

²²² *River*; 2000; pp 738.

²²³ IRSAC: *L'Institut de Recherche Scientifique en Afrique Centrale*, based in Lwiro.

²²⁴ I have found countless references to this nickname. It was mentioned in almost every review article that has been written about Meyer and his work (including obituaries), and in several other documents, such as personal letters from the era, and a memoir of the first Sonoma vaccination by Tom Norton.

²²⁵ Probably part of FOREAMI: *Fond médical Reine Elisabeth pour l'Assistance Médicale aux Indigènes*, another royal-backed institute of scientific research. It is not immediately clear how this type of research would have benefited the indigenous population.

²²⁶ Correspondence can be found on the excellent "Profiles in science: Joshua Lederberg" web-site at: <<http://profiles.nlm.nih.gov/BB/>>

²²⁷ Anon., "Wistar Institute names chief", *New York Times*, March 27, 1957, page 6.

²²⁸ N. Topping, with G. Cohn, *Recollections* (Los Angeles: USC Press, 1990), see pages 140-141. The book was apparently finished in 1989, and Topping writes that he recruited Koprowski to the Wistar first of all, and that this was "more than 35 years ago", ie 1954 or earlier. He also writes in his foreword, that "places, dates and sometimes names may escape me or become muddled". However, his recollection that Koprowski was involved with the Wistar from long before his formal arrival in 1957 is confirmed by multiple sources [see text], and I had previously discussed it in *The River*, 2000 (page 976, note 15; and page 707), long before I discovered Topping's book. Another relevant detail about the timing is that Geoffrey Rake is known to have been recruited in 1953, and Topping says he enlisted Koprowski before Rake.

²²⁹ Available in the "letters" section in the "Profiles in science: Joshua Lederberg" web-site at: <<http://profiles.nlm.nih.gov/BB/>>.

²³⁰ Anon., "Dr. Agnes Nelson Flack, 92, pioneer of polio research"; [U.S. local paper, title unknown]; December 19, 1989. See also: Anon., "Call to duty", *Wilkes-Barre Record* [Pennsylvania]; January 7, 1958.

²³¹ H.M. McClure et al., "Erythroleukemia in two infant chimpanzees fed milk from cows naturally infected with the bovine C-type virus"; *Cancer Research*; 1974; 34; 2745-2757. The C-type virus of the title was later named bovine leukemia virus. The abstract noted that this was the first time either leukemia or PCP had been reported in chimpanzees. The NBC researchers appear to have established a herd infected with bovine leukemia at least a decade before this report was published, for National Cancer Institute funding can be traced back to 1965, for research that included BLV transmission experiments to different animals and cell lines.

²³² P.S. Brachman and F.R. Fekety, "Industrial Anthrax"; *Ann. N.Y. Ac. Sci.*; 1958; 70; 574-584.

²³³ The two main reports are: S.A. Plotkin, P.S Brachman et al., "An epidemic of inhalation anthrax; the first in the twentieth century. I. Clinical features"; *Am. J. Med.*; 1960; 29; 992-1001; and P.S Brachman, S.A. Plotkin, et al., "An epidemic of inhalation anthrax; the first in the twentieth century. II. Epidemiology"; *Am. J. Hyg.*; 1960; 72; 6-23. In the second article, there is a single, bland sentence which reads: "The potential civil defence problem posed by anthrax aerosols is also emphasized". This is the only overt clue that the study may in any way relate to biowarfare research.

²³⁴ J.M. Barnes, "The development of anthrax following the administration of spores by inhalation"; *Brit. J. Exp. Path.*; 1947; 28; 385-394. Dr Barnes, who worked for Britain's BW research centre at Porton Down, devoted most of his paper to an analysis of how anthrax spores sprayed from solutions of a commercial detergent, *Tergitol*, increased death rates in guinea-pigs by a factor of ten. Brachman and Plotkin acknowledged that this was "an effect which also was demonstrated with [the] particular detergent" that had been used in the Manchester mill (which is not described by brand name, but only as a "non-ionic detergent"), before insisting "it seems impossible for patient no. 1 to have come into contact with detergent-scoured hair before the onset of his illness". Since the patient in question apparently died before he could be interviewed about his activities in the mill in the hours before he fell sick, I find this a surprising and unwarranted conclusion.

²³⁵ P.S. Brachman, S.A. Plotkin et al., "Field evaluation of a human anthrax vaccine"; *Am. J. Pub. Health*; 1962; 52(4); 632-645.

²³⁶ Meryl Nass, "Talk to anthrax vaccine committee, Institute of Medicine, October 3, 2000"; <<http://www.anthraxvaccine.org/IOMtalk.htm>>.

²³⁷ Meeting of the medical committee, U.S. Army Chemical Corps Advisory Council; 11 June 1958, Biological Warfare Laboratories, Fort Detrick, Maryland.

²³⁸ D. MacKenzie, "Anthrax attack bug 'identical' to army strain"; *New Scientist.com news service*; May 9, 2002.

²³⁹ C.V. Harding, D. Harding. W.F. McLimans and G. Rake, "Cytological changes accompanying the growth of poliomyelitis virus in cells of human origin (strain HeLa)"; *Virology*; 1956; 2; 109-125.

²⁴⁰ I can find no documentary evidence indicating that collaboration between the Wistar Institute and the US Army Chemical Corps extended beyond 1958. Geoffrey Rake died in March of that year, and soon after that the Fort Detrick team discussed bringing in William McLimans from the Wistar to head its virus research programme. It is not known whether a formal offer was made, or if he accepted it, but by the early sixties he was working at the National Institutes of Health.

²⁴¹ D.W. Ziegler et al., "The propagation of mammalian cells in a 20-liter stainless steel fermentor"; *App. Microbiol.*; 1958; 6; 306-310.

²⁴² E.H. Lennette and H. Koprowski, "Human infection with Venezuelan equine encephalomyelitis virus. A report of eight cases of infection acquired in the laboratory"; *J. Am. Med. Assoc.*; 1943; 123; 1088-1095.

²⁴³ *River*, 2000, page 399; see also page 970, notes 3 and 4.

²⁴⁴ See: J.C.N. Westwood et al., "Transformation of normal cells in tissue culture: its significance relative to malignancy and virus vaccine production"; *Brit. J. Exp. Path.*; 1957; 38; 138-153.

²⁴⁵ It is now clear that Koprowski did not speak about CHAT and Fox at the *New York Academy of Sciences* meeting in January 1957, although a six-page paper about them did appear in the proceedings that were published that December. Koprowski was at that time President-elect of the Academy. [H. Koprowski, "Discussion"; *Spec. Pub. N.Y. Acad. Sci.*; 1957; 5; 128-133. See also: *River*; 2000; page 969, note 29.]

²⁴⁶ *River*, 2000, pages 689-690.

²⁴⁷ Ruth Jervis, personal communication, 1995.

²⁴⁸ Anon., "'Live' polio vaccine aids in Congo epidemic area"; *Science News Letter*; August 9, 1958, page 94.

²⁴⁹ *River*, 2000, pages 384-387.

²⁵⁰ J.C.N. Westwood et al., "Transformation of normal cells in tissue culture: its significance relative to malignancy and virus vaccine production"; *Brit. J. Exp. Path.*; 1957; 38; 138-153.

²⁵¹ F.W. Sheffield and G.M. Churcher, "The serial propagation of poliomyelitis viruses in cells derived from rabbit embryo kidney"; *Brit. J. Exp. Path.*; 1957; 38; 154-159.

²⁵² They repeated this, for instance, at the Stanleyville virus symposium in September 1957. [J.P. Delville and S.R. Pattyn, "Epidémiologie de la poliomyélite au Congo Belge et au Ruanda-Urundi. Etat actuel de nos connaissances"; *Ann. Soc. Belge Med. Trop.*; 1958; 38; 283-292.]

²⁵³ L. Hayflick, "The establishment of a human cell line (WISH) of human amnion cells in continuous culture", *Exp. Cell Res.*; 1961; 23; 14-20. L. Hayflick and P.S. Moorhead, "The serial cultivation of human diploid cell strains"; *Exp. Cell Res.*; 1961; 25; 585-621. Lise Thiry is thanked at the end of the first article for "valuable technical assistance", and Stanley Plotkin at the end of the second as one of four Wistar scientists who "participated in several of the virus studies".

²⁵⁴ M.V. Fernandes, "The development of a human amnion strain of cells", *Tex. Rep. Biol. Med.*; 1958; 16; 48-58. Intriguingly, both "Fernandes" and WISH transformed into what we can now identify as cell lines on the 35th day after being put into culture. Further research into "Fernandes" (now renamed "Amnion Ep. L") was conducted at the Wistar in the years up to 1960, and revealed it to be almost identical to HeLa. [V. Defendi et al., "Immunological and karyological criteria for identification of cell lines"; *J. Nat. Canc. Inst.*; 1960; 25(2); 359-379.]

²⁵⁵ P. Gage, personal communication, March 2000.

²⁵⁶ P. Gage, personal communication, September 2000.

²⁵⁷ These opportunities comprised two interviews between Dr Osterreith and myself in 1993 and 1994, a letter he wrote me in 1994, two statements he made to Stanley Plotkin in 2000, and his own brief paper delivered in September 2000, and published by the Royal Society in 2001.

²⁵⁸ The following is an example, this one coming from Robin Weiss's review of *The River* in 1999: "To me, the possible use of small batches of experimental OPV made locally seems a more plausible source of contamination than the Wistar preparations". [R.A. Weiss, "Is AIDS man-made?"; *Science*; 1999; 286; 1305-1306.]

²⁵⁹ There is one point I would like to add, however. In *The River*, I wrote about the death of Dr Osterreith's son from kidney failure in the 1960s, and speculated that he (like the rest of the family) might have been fed CHAT. In retrospect, this was an error of judgement on my part, and I apologise to Dr Osterreith for it.

²⁶⁰ John Crewdson, *Science Fictions* (New York: Little, Brown, 2002), p. 184. See also: O. Sattaur, "How Gallo got credit for AIDS discovery"; *New Scientist*, February 7, 1985, pages 3-4; and R. Weiss, "AIDS rivalry" [letter]; *New Scientist*, February 28, 1985, pages 45-46.

²⁶¹ There were several other problems highlighted by the testing process, not least the status of the supposedly independent "AIDS/Poliovirus Advisory Committee" which was set up (and presumably

funded) by the Wistar Institute. It was chaired by a scientist (Dr Claudio Basilico) whose performance at the London meeting was felt by many observers to be anything but detached and independent – in that he acted throughout as if he was representing, and defending, the Wistar. Furthermore, Dr Basilico failed to provide certain basic information, such as the selection criteria used for those CHAT samples that were released.

²⁶² J. Cohen, “Forensic epidemiology. Vaccine theory of AIDS origins disputed at Royal Society”; *Science*; 2000; 289; 1850-1851.

²⁶³ *River*, 2000, p. 600.

²⁶⁴ At that point in time, the choice seemed to be between two labs in the US (those of George Jervis at Letchworth Village, and of Koprowski at the Wistar Institute), and three in Belgium (those of Lise Thiry in Brussels, RIT in Genval, and the Rega Institute in Leuven). Almost the last addition that I made to the original (1999) version of *The River* was to insert two pages [*River*, 2000, pp. 789-791] in which I discussed the theoretical possibility that the vaccine could have been made in Stanleyville, even though I did not, at that stage, know of any *persuasive* evidence to support such a scenario.

²⁶⁵ R.A. Weiss, “Polio vaccines exonerated”; *Nature*; 2001; 1035-1036.

²⁶⁶ Whether any samples of this vaccine still remain (perhaps in a freezer somewhere in the U.S. or Belgium), or (if they do) whether they would ever be released for independent analysis, are moot points.

²⁶⁷ J.-P. Vartanian and S. Wain-Hobson, “Analysis of a library of macaque nuclear mitochondrial sequences confirms macaque origin of divergent sequences from old oral polio vaccine samples”; *Proc. Nat Acad. Sci.*; 2002; in press.

²⁶⁸ S. Sternberg, “Polio vaccine AIDS issue ‘is resolved’”; *USA Today*; May 14, 2002, page 8D.

²⁶⁹ S. Wain-Hobson, e-mail to author, October 4th, 2001. In this e-mail, Wain-Hobson accuses me of “changing the goal-posts – now the focus is on local amplification”. However, he continues, “this is frequent in science. There is no problem here per se.” Despite the latter comment, seven months later Wain-Hobson was declaring that the theory has been “scotched”, following his testing of CHAT vaccine samples that were prepared, not locally in Africa, but in the USA.

²⁷⁰ Letter from Dr Stanley Plotkin to author, June 30, 1994.

²⁷¹ *River*, 2000, pages 572 and 718.

²⁷² Anon., “Belgian scientists have high hopes of new vaccine”, *Iraq Times*; March 15, 1959. Courtois’ estimate may have been based on a previous claim, made by Koprowski in 1957, that he could vaccinate a million people with a litre of vaccine. [Anon., “City polio proposal. Talks held on trial of new vaccine in Kenya”; *East African Standard* (Nairobi); February 1, 1957.] Since the typical vaccine dose was a millilitre, Koprowski was apparently building in an amplification/dilution factor of 1,000:1, and Courtois of 2,000:1 (based on a 1959 world population was 4 billion).

²⁷³ Anon., “Expert committee on poliomyelitis. Second report.”; *W.H.O. Tech. Rep. Ser.*; 1958; 145; see especially pages 23-27. Anon., “Expert committee on poliomyelitis. Third report.”; *W.H.O. Tech. Rep. Ser.*; 1960; 203; 1-53; see especially pages 35-38. (Both of these expert committees stated that any suitable monkey species could be used to produce OPVs, employing either trypsinised or Maitland-type cultures.) The second committee sat in July 1957, and lit the blue touch paper for trials in places like Africa, when it declared: “the Committee strongly recommends that controlled field trials be carried out for the purpose of testing further the value of these agents [OPVs]”. It went on to make certain recommendations about conditions under which such field-trials could reasonably be staged. Later, it became clear that several of these recommendations may not have been adhered to by those, like Koprowski, who staged the early trials. (For brief analysis, see *River*, 2000, page 737.) Also see: Anon., “Requirements for Biological Substances. 7. Requirements for poliomyelitis vaccine (oral)”; *W.H.O. Tech. Rep. Ser.*; 1962; 237; 1-29. On pages 13-15 of the latter report it is stated, for the first time, that only monkeys which have not been used for experimental purposes of significance to the safety of the vaccine could be used for vaccine production, and that monkey kidney cultures should not be propagated in series. Monkeys of any suitable species could still be used, but this is the first time that detailed requirements for OPV production were laid down in writing. The WHO study group that produced the latter report sat in November, 1960, so all OPVs produced before that month were, to some extent, experimental.

²⁷⁴ H. Koprowski, G.A. Jervis, and T.W. Norton, “Oral administration of poliomyelitis virus to man and ape – a comparative study”; *Arch. Ges. Virusforschung*; 1954; 5; 413-424.

²⁷⁵ *River*, 2000, p. 718.

²⁷⁶ There are a number of papers from the Stanleyville scientists which report on the occurrence of different arboviruses in both humans and chimps. In one such paper, Dr Osterrieth and colleagues report on the occurrence of Chikungunya in 141 humans and 79 chimpanzees, with roughly half of

each group coming from the rain forest, and half from the savanna regions to the north. P. Osterrieth et al., “Recherche sur le virus Chikungunya au Congo belge. II. Enquête sérologique”; *Ann. Soc. Belge Med. Trop.*; 1960; 40; 205-213.

²⁷⁷ L. Hayflick, S.A. Plotkin, T.W. Norton and H. Koprowski, “Preparation of poliovirus vaccines in a human fetal diploid cell strain”; *Am. J. Hyg.*; 1962; 26; 733-743.

²⁷⁸ For instance: Anon., “7 David’s Island children first test new polio cure”; *Boothbay Register* (Bar Harbor, Maine); July 31, 1958. See also the *Iraq Times* article cited above.

²⁷⁹ W.C. Osman Hill, “The use of primates in biomedical studies; a review of suitable species”; *Ann. N.Y. Ac. Sci.*; 1969; 162(1); 7-14.

²⁸⁰ Indeed, this may be what is implied by the WHO technical report of 1962, which says that in future, only monkeys which have not been used for experimental purposes of significance to the safety of the vaccine could be used for vaccine production. [Anon., “Requirements for Biological Substances. 7. Requirements for poliomyelitis vaccine (oral)”; *W.H.O. Tech. Rep. Ser.*; 1962; 237; 1-29; see page 13.]

²⁸¹ L.A. Bruggeman, M.D. Ross et al., “Renal epithelium is a previously unrecognised site of HIV-1 infection”; *J. Am. Soc. Nephrol.*, 2000; 11, 2079-2087.

²⁸² J.P. Moore and R.W. Doms, “Much at stake with kidneys?”; *J. Am. Soc. Nephrol.*, 2000; 11, 2138-2140.

²⁸³ *River*, 2000, p. 569.

²⁸⁴ J. Beale and F. Horaud, “Polio vaccine and retroviruses”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 841-843.

²⁸⁵ For details of the CHAT pool 23 protocol, see: *River*, 2000, 702-703.

²⁸⁶ Cecil Fox, personal communication, June 2002.

²⁸⁷ P. Ondoa, G. van der Groen, L. Kestens et al., “In vitro replication of SIVcpz is suppressed by beta-chemokines and CD8+ T cells but not by natural killer cells of chimpanzees”; *AIDS Res. Hum. Retroviruses*; 2002; 18(5); 373-382.

²⁸⁸ J.L. Melnick, “Tissue culture methods for the cultivation of poliomyelitis and other viruses”; in *Diagnostic Procedures for Virus and Rickettsial Diseases*; (NYC: American Public Health Association; 1956); 97-151. The title of Melnick’s chapter is significant, and the content deals with the growth of polioviruses and polio vaccines, rather than relating to “diagnostic procedures”, as per the title of the book. In one place, Melnick discusses which tissues are best for cultivating polioviruses. Having listed a number of human and primate tissues derived from different organs, he comments: “It is difficult to recommend one of these tissues in preference to another, and final choice must depend upon their local availability”.

²⁸⁹ M.L. Santiago et al., “SIVcpz in wild chimpanzees”, *Science*; 2002; 295; 465.

²⁹⁰ This is despite the valiant efforts of Bill Hamilton, some of whose chimpanzee stool samples are now, I hear, being reexamined by Dr Hahn with, I believe, interesting results. See also: P.M. Sharp, B.H. Hahn et al., “The origins of acquired immune deficiency viruses: where and when?”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 867-876.

²⁹¹ P. Gagneux, M.K. Gonder, T.L. Goldberg and P.A. Morin, “Gene flow in wild chimpanzee populations: what genetic data tell us about chimpanzee movement over space and time”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 889-897.

²⁹² A. André et al., “Mise en évidence d’antigènes de groupes sanguins A, B, O et Rh chez les singes chimpanzés”; *Ann. Inst. Past.*; 1961; 101; 82-95. See also: *River*, 2000, pp 566-567 about the apparent fate of these chimp blood samples from Lindi.

²⁹³ G. Courtois, “Sur la réalisation d’une singerie des chimpanzés au Congo”; *Symposium international sur l’avenir des animaux de laboratoire*; (Lyon: Institut Pasteur, 1967), pp 235-244. The fact that at least one ape (probably a *Pan troglodytes*, and possibly a *Pan troglodytes troglodytes*) came from Coquilhatville is, I believe, more significant than Ghislain Courtois’ retrospective claim that at Lindi they worked only with *Pan troglodytes schweinfurthii* and *Pan paniscus*, for it is very unlikely that Courtois and colleagues would have been able to distinguish between the two common chimp subspecies, *troglodytes* and *schweinfurthii*, on the basis of physical differences alone.

²⁹⁴ According to several sources, it was decided at an early stage of the Lindi experiments that because the bonobo population was not hardy (with about half having died “naturally” from disease or stress), the remainder would be “used up” in experiments, and further bonobos would not be procured. When primatologist Adriaan Kortlandt visited Stanleyville in February 1960, Lindi camp had closed a month or two earlier, but had been superseded by a holding facility in a hangar sited behind the medical laboratory. Apparently there were no bonobos among the 60 apes housed there, but Kortlandt was told that a total of 86 bonobos had been procured for the former polio research programme at Lindi, and that all had “died within three weeks”. [*River*, 2000, page 717 and 1026, n. 51.] Although the details given

to Kortlandt would appear to have been sanitised, his account does give a sense of the speed with which the surviving bonobos may have been utilised. Kortlandt also spoke of an atmosphere of secrecy surrounding both the Stanleyville lab and the chimp research (a detail which has been confirmed by several other visitors).

²⁹⁵ Anon., “Belgian scientists have high hopes of new vaccine”, *Iraq Times*, March 15, 1959.

²⁹⁶ There is also another reason. As Bill Hamilton suggested, it may still emerge that an SIV closely related to HIV-1 Group M exists in *Pan paniscus*. To date, very few *Pan paniscus* have been sampled for SIV – and sadly, Bill’s own efforts to obtain such samples appear to have been unsuccessful.

²⁹⁷ S. Saragosti et al., “Molecular characterisation of primate lentiviruses from a *Cercopithecus wolffi* and a *Cercopithecus ascanius*”; 8th International discussion meeting on HIV dynamics and evolution, (Paris, April 2001, abstract p. 19).

²⁹⁸ J. Kingdon, *The Kingdon field guide to African mammals* (San Diego: Academic Press, 1997), pp 70-75

²⁹⁹ S. Van Dooren, A-M Vandamme et al., “Lack of evidence for infection with simian immunodeficiency virus in bonobos”; *AIDS Res. Hum. Retro.*, 2002, 18(3), 213-216.

³⁰⁰ M. Worobey, personal communication, 2001.

³⁰¹ This answer does not feature in the published proceedings of the meeting, which do not include the discussion sessions.

³⁰² M. Peeters, B.H. Hahn, E. Delaporte et al., “Risk to human health from a plethora of simian immunodeficiency viruses in primate bushmeat”; *Emerging Infectious Diseases*; 2002; 8(5); page numbers unknown.

³⁰³ E. Delaporte, M. Peeters et al., “Epidemiological and molecular characteristics of HIV infection in Gabon, 1986-1994”; *AIDS*; 1996; 10; 903-910. See also: J.N. Nkengasong et al., “Genotypic subtypes of HIV-1 in Cameroon”; *AIDS*; 1994; 8; 1405-1412. Although Delaporte referred to “central Africa” as the potential hearth, his supporting references all referred to the west central African countries of the former French Equatorial Africa (plus the related state of Cameroon). For further commentary, see: *River*, 2000, pp. 1064-1065, note 25.

³⁰⁴ B.H. Hahn, G.M. Shaw, K.M. De Cock, and P.M. Sharp, “AIDS as a zoonosis: scientific and public health implications”; *Science*; 2000; 287; 607-614.

³⁰⁵ I have been unable to locate Bwamada on the DRC map, though it apparently lies in Equatoria province. It is possible that it is a new name for Mbandaka.

³⁰⁶ K. Yusim, M. Peeters, E. Delaporte, B. Korber et al., “Using human immunodeficiency virus type 1 sequences to infer historical features of the acquired immune deficiency syndrome epidemic and human immunodeficiency virus evolution”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 855-866.

³⁰⁷ N. Vidal, M. Peeters, E. Delaporte et al., “Unprecedented degree of HIV-1 Group M genetic diversity in the Democratic Republic of Congo suggests that the HIV-1 pandemic originated in central Africa”; *J. Virol.*; 2000; 74; 10498-10507.

³⁰⁸ J.L K. Mokili et al.; “Genetic heterogeneity of HIV-1 type 1 subtypes in Kimpese, rural Democratic Republic of Congo”; *AIDS Res. Hum. Retro.*; 1999; 15(7); 655-664.

³⁰⁹ Furthermore, it is not impossible that CHAT immunisation took place at Kimpese itself. It has a Baptist-run hospital which, in the fifties, was the major polio rehabilitation centre for the entire Belgian Congo. Elsewhere in the Congo, doctors from the same group of Baptist hospitals conducted their own CHAT vaccination campaigns (as, for instance, around Yakusu, near Stanleyville).

³¹⁰ M.L. Kalish, T.M. Folks et al., “Evidence for the presence of all known HIV-1 Group M subtypes and some unclassified strains in Kinshasa, Zaire, in the early to mid-1980s”; 8th Conference on Retroviruses and Opportunistic Infections (Chicago, February 2001), abstract 268.

³¹¹ Stanley Plotkin and his team of CHAT vaccinators reported that the 1958 population was 350,000, while Dr Julian De Moor reported that this had risen to 550,000 by 1960, 850,000 by 1962, and would rise to an estimated 1.3 million in 1964. See *River*, 2000, pages 735 and 1034, note 73.

³¹² T. Burr, J.M. Hyman, and G. Myers, “The origin of acquired immune deficiency syndrome: Darwinian or Lamarckian?”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 877-888.

³¹³ A. Rambaut, D.L. Robertson, O.G. Pybus, M. Peeters and E.C. Holmes, “Human immunodeficiency virus: phylogeny and the origin of HIV-1”; *Nature*; 2001; 410; 1047-1048.

³¹⁴ E. Holmes, personal communication, October 2000.

³¹⁵ The former Group M subtype E has now been reclassified a “circulating recombinant form”, CRF01-AE.

³¹⁶ Also based on Andrew Rambaut’s responses to a series of questions in July 2001.

³¹⁷ K.M. De Cock, “Epidemiology and the emergence of human immunodeficiency virus and acquired immune deficiency syndrome”, *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 795-798. Of course, as Kevin

De Cock emphasised, I have only been able to provide a random selection of early cases and infections (generally those which can be traced through the literature, or which have been located through serendipity), and my data only provide a snapshot from the 1950-1980 era. None the less, the data do give some useful indications about where HIV-1(M) was present (and where absent) in these early years. And there has certainly been no conscious “observer bias”, though I suspect he would argue that such bias is usually unconscious. For HIV-1, the data pertain both to those whose blood was sampled in the years up to 1981, and who were only retrospectively checked for HIV-1 infection, and to those who tested HIV-1-positive later than 1981, but for whom it could be proved that infection must have begun by 1981 or before.

³¹⁸ B.H. Hahn, G.M. Shaw, K.M. De Cock and P.M. Sharp, “AIDS as a zoonosis: scientific and public health implications”; *Science*; 2000; 287; 607-614.

³¹⁹ D.M. Hillis, “AIDS. Origins of HIV”; *Science*; 2000; 288; 1757-1759.

³²⁰ A. Chitnis, D. Rawls and J. Moore, “Origin of HIV type 1 in colonial French Equatorial Africa?”; *AIDS Res. Hum. Retro.*; 2000; 16(1); 5-8. See also the helpful debate about this issue on Jim Moore’s web-site.

³²¹ D. Low-Beer, “The distribution of early acquired immunodeficiency syndrome cases and conditions for the establishment of new epidemics”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 927-931.

³²² M. Smallman-Raynor, A. Cliff and P. Haggett, *Atlas of AIDS* (London: Blackwell, 1992), pages 332-334.

³²³ A. Bobkov, R. Cheingsong-Popov et al., “Identification of an *env* G subtype and heterogeneity of HIV-1 strains in the Russian Federation and Belarus”; *AIDS*; 1994; 8(12); 1649-1655.

³²⁴ Data obtained (for period up to and including 1980) by expressing (a) AIDS cases from Kinshasa, and (b) HIV-1(M) infections from Kinshasa, as a proportion of the total number of AIDS cases/HIV-1(M) infections which can be associated with a specific town, this being 13 of 31 and 21 of 47, respectively.

³²⁵ Data obtained (for period up to and including 1980) by expressing (a) AIDS cases from CHAT vaccination sites, and (b) HIV-1(M) infections from CHAT vaccination sites, as a proportion of the total number of AIDS cases/HIV-1(M) infections which can be associated with a specific town, this being 21 of 31 and 40 of 47, respectively. It is worth adding that every instance of proven Group M infection in Africa up to the end of 1980 comes from a venue that is “within range” (within 225 kilometres, to be precise) of a CHAT vaccination site.

³²⁶ *River*, 2000, pages 529 and 737-739.

³²⁷ Their departures could have been as refugees following the ethnic unrest that began in 1959, or as economic migrants (some of whom had left only a few months before, and some of whom may have been settled outside Ruanda-Urundi for decades).

³²⁸ For the cases in this paragraph, see: *River*; 2000; pages 764-765. In 2000, I too reviewed the Mulago pathology records, and found Bill Hamilton’s “flags” still marking specific cases. Like Bill, I was unable to find the case of *Pneumocystis carinii* pneumonia (PCP) from 1960, which Dr Jack Davies had recalled as Uganda’s “first case of AIDS”. I did, however, find the case in which death was ascribed to pneumonia caused by a “heavy pure growth of *Klebsiella*”, and suspect that this was probably, in reality, the AIDS-like pneumonia which Dr Davies had remembered as the “first case”. The occurrence of this case so soon after the fatal *Klebsiella* cases in Stanleyville is intriguing. I found two small discrepancies from the scribbled notes brought back by Bill in 1995: the 1960 *Klebsiella* case was *not* identified by tribe, and the third case was from 1965, not 1960-1961 as previously reported. (However, the case is still a potential instance of HIV infection via CHAT, for the boy in question may have been infected perinatally by a previously vaccinated parent.)

³²⁹ J. Sonnet et al., “Early AIDS cases originating from Zaire and Burundi (1962-1976)”; *Scand. J. Inf. Dis.*; 1987; 19; 511-517. Also see: *River*; 2000, pp. 259-261.

³³⁰ *River*; 2000, page 288.

³³¹ An article in a local Stanleyville paper from August 1957 refers to the hospital for blacks as being in “the sad state which everyone knows about – it is a collection of buildings which was suitable thirty years ago”, and goes on to claim that the hospital for Europeans is “falling into ruin”. Anon., “Toujours l’hôpital”; *Le Stanleyvillois*; August 19, 1957, page 1.

³³² S.A. Plotkin, “CHAT oral polio vaccine was not the source of human immunodeficiency virus type 1 group M for humans”; *Clin. Inf. Dis.*, 2001, 32, 1068-84; see p. 1076.

³³³ Anon., “Belgian scientists have high hopes of new vaccine”, *Iraq Times*, March 15, 1959. An identical version of the article, entitled “Congo may lead world in fight against polio” was published in the *Uganda Argus* (Kampala), at around the same date, and a briefer version (“New polio vaccine in Congo”) appeared in the *East African Standard* (Nairobi).

- ³³⁴ P. Osterrieth, "Propriétés biochimiques des Klebsiella", *Ann. Soc. Belge Med. Trop.*; 1958; 38; 721-730.
- ³³⁵ P.N. Fultz et al., "Identification and biological characterization of an acutely lethal variant of simian immunodeficiency virus from sooty mangabeys (SIV/SMM)"; *AIDS Res. Hum. Retro.*; 1989; 5(4); 397-409. See also: U.S. Army Medical Research Institute of Infectious Diseases [USAMRIID], Fort Detrick, Frederick MD, Annual Report FY 1989, pp 189-193.
- ³³⁶ The fact that Osterrieth apparently sometimes used different syringes for different chimps indicates that keeping sera separate was seen to be important, at least on occasions, but it is also possible that those particular blood extractions were for diagnostic work rather than for tissue culture preparation.
- ³³⁷ Melnick, "Tissue culture methods for the cultivation of poliomyelitis and other viruses"; page 117.
- ³³⁸ Melnick's description of making poliovirus pools in HeLa cells specifies that for the growth medium, "serum pools obtained from 2 to 5 human donors are made, to avoid the use of serum deficient in antitryptic effect". See: Melnick, "Tissue culture methods for the cultivation of poliomyelitis and other viruses"; page 132.
- ³³⁹ One other example: in 1999, Florian Horaud told Simon Wain-Hobson of how in the fifties, in Romania, 150 rhesus monkeys destined for polio vaccine production in Romania were gang-caged together, and added how horrified he felt, looking back, at the risks that he had once taken. As for official recommendations, it was not until 1962 that the WHO proposed that monkeys used for the production of polio vaccine should be kept in cages, two to a cage, and that "cage-mates [should] not be interchanged". See: Anon., "Requirements for Biological Substances. 7. Requirements for Poliomyelitis Vaccine (Oral)"; *WHO Tech. Rep. Ser.*, 1962, 237, 1-29, see page 14.
- ³⁴⁰ S. Wain-Hobson, personal communication, December 1999.
- ³⁴¹ D.P. Wooley, R.A. Smith, S. Czajak and R.C. Desrosiers, "Direct demonstration of retroviral recombination in a rhesus monkey"; *J. Virol.*; 1997; 71(12), 9650-9653. The article refers to retroviral recombination in tissue culture systems (generating doubly infected cells), and reported on recombination in a rhesus monkey that had been artificially infected with two different SIV strains.
- ³⁴² Sharp et al., "The origins of acquired immune deficiency viruses: where and when?"; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 867-876. K. Yusim et al., "Using human immunodeficiency virus type 1 sequences to infer historical features of the acquired immune deficiency syndrome epidemic and human immunodeficiency virus evolution"; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 855-866. M. Salemi, K. Strimmer, W.W. Hall, M. Duffy, E. Delaporte, S. Mboup, M. Peeters and A.-M. Vandamme, "Dating the common ancestor of SIVcpz and HIV-1 Group M and the origin of HIV-1 subtypes by using a new method to uncover clock-like molecular evolution"; *FASEB J.*; 2000. Meanwhile, the phylogenetic dating argument continues to be lauded in the mainstream press. See, for instance, Gina Kolata, "The Genesis of an Epidemic: Humans, Chimps and a Virus", *New York Times*, September 4, 2001, which claims that these doctors have "unravel[led] the mystery of the origin of the epidemic".
- ³⁴³ In his Royal Society article, Paul Sharp wrote that my interpretation of the impact that recombination might have on dating was "strange", because "it is clear that recombination would make the date of the common ancestor seem more recent". [Sharp et al., "The origins of acquired immune deficiency viruses: where and when?"; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 867-876.] But what is Sharp's evidence for this claim? An increasing number of articles are being published which conclude that he is wrong in this assumption, and that ignoring recombination can lead to either under-estimation or over-estimation of the MRCA. See, for instance, recent articles by Mikkel Schierup and Michael Worobey, cited immediately below. One sceptical geneticist states that all the work done on dating the Group M epidemic up to now has been a "dog's breakfast".
- ³⁴⁴ M.H. Schierup and R. Forsberg, "Recombination and phylogenetic analysis of HIV-1"; in *Proceedings of the conference: origin of HIV and emerging persistent viruses*; (Rome: 2003; 231-245).
- ³⁴⁵ M.H. Schierup and J. Hein, "Recombination and the molecular clock" [letter]; *Mol. Biol. Evol.*; 2000; 17(10); 1578-1579. See also: M.H. Schierup and J. Hein, "Consequences of recombination on traditional phylogenetic analysis"; *Genetics*; 2000; 156; 879-891.
- ³⁴⁶ M. Worobey, "A novel approach to detecting and measuring recombination: new insights into evolution in viruses, bacteria and mitochondria"; *Mol. Biol. Evol.*; 2001; 18(8); 1425-1434.
- ³⁴⁷ K. Yusim et al., "Using human immunodeficiency virus type 1 sequences to infer historical features of the acquired immune deficiency syndrome epidemic and human immunodeficiency virus evolution"; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 855-866.
- ³⁴⁸ T. Zhu, B.T. Korber, A.J. Nahmias, E. Hooper, P.M. Sharp and D.D. Ho, "An African HIV-1 sequence from 1959 and implications for the origin of the epidemic"; *Nature*; 1998; 391; 594-597. [See also: *River*; 2000; 775-777.] The short ZR59 sequence is used by some phylogeneticists as a check on

the accuracy of their dating calculations. However, I hear from “inside sources” that the scientist at the Aaron Diamond Center who analysed ZR59 “was having a most difficult time with the sequence”.

³⁴⁹ A.G. Motulsky, J. Vandepitte and G.R. Fraser, “Population genetic studies in the Congo. I. Glucose-6-phosphate dehydrogenase deficiency, hemoglobin S, and malaria”; *Am. J. Hum. Gen.*; 1966; 18(6); 514-537. See also following papers in same volume, on pp. 538-545, 546-552, and 553-558.

³⁵⁰ E.R. Giblett, A.G. Motulsky and G.R. Fraser, “Population genetic studies in the Congo. IV. Haptoglobin and transferrin serum groups in the Congo and in other African populations”; *Am. J. Hum. Gen.*; 1966; 18(6); 553-558.

³⁵¹ S.A. Plotkin, A. Lebrun, G. Courtois and H. Koprowski, “Vaccination with the CHAT strain of type 1 attenuated poliomyelitis virus in Leopoldville, Congo”; *Bull. W.H.O.*; 1962; 24; 785-792.

³⁵² *River*, 2000, pages 827-836.

³⁵³ The abbreviations AEF and AOF refer to the more commonly-used French acronyms for *Afrique Équatoriale Française* (French Equatorial Africa) and *Afrique Occidentale Française* (French West Africa), respectively.

³⁵⁴ *River*; 2000; pages 853-861.

³⁵⁵ However, in a second interview with Wain-Hobson (given after he had spoken with some of the other former Pasteur workers, including the late Florian Horaud, who was apparently a friend of Stanley Plotkin), this man apparently retracted on the latter piece of information – saying that he had used only IPV in Brazzaville. What is not disputed, however, is that he administered both OPV and IPV in French Equatorial Africa between 1957 and 1959.

³⁵⁶ S. Wain-Hobson, personal communications, October to December 1999. Dr Wain-Hobson was kind enough to give me copies of his notes from these (and other) interviews with former Pasteur Institute employees, and I would like once again to thank him for sharing this information. What is not clear, however, from these notes is whether he specifically asked this doctor whether he had grown *polio vaccine* in locally-prepared cultures. When I review my own notes of previous conversations with Wain-Hobson in September and October 1999, it is clear that he suspected that the French vaccine had been locally prepared, so it seems strange that when he came to interview one of the key doctors involved, he merely recorded that he “grew polio on local monkey kidney cultures”, without specifying whether or not this meant vaccine amplification. (I was also remiss for not pursuing this more rigorously with him.) I now wonder whether this rather ambiguous phrase in the written notes which he gave me in December 1999 indicates that he was already becoming uneasy about some of the implications of his findings, and was not wishing to put anything too damaging on the record.

³⁵⁷ *River*, 2000, pp. 852-858.

³⁵⁸ S. Wain-Hobson, personal communication, October 2001.

³⁵⁹ L-J André and E. André-Gadras, “16 cas de poliomyélite observés dans un district de brousse de Gabon” [sic]; *Med. Trop.*; 1958; 18; 638-641. The vaccination is mentioned only briefly at the end of the article.

³⁶⁰ P. Lépine, “Discussion”; *Rev. Lyonnaise de Med.*; 1959; 8; 46. (See also the preceding speech by Hilary Koprowski about the Congo vaccinations, on pages 39-40.)

³⁶¹ L.J. André, “Le S.I.D.A., a-t-il déjà existé?”; *Med. Trop.*; 1987; 47(3); 229-230.

³⁶² P. Lépine et al., “Présence apparemment insolite et conservation de microfiliaries du singe dans les cultures de tissus”, *Bull. Soc. Path. Exot.*; 1955; 48; 838-843.

³⁶³ *River*, 2000, pages 827-835.

³⁶⁴ B.A. Castro et al., “Persistent infection of baboons and rhesus monkeys with different strains of HIV-2”; *Virology*; 1991; 184; 219-226.

³⁶⁵ *River*; 2000; pages 338-346.

³⁶⁶ See figure in *River*; 2000; page 625.

³⁶⁷ As commentators like Tom Schulz have acknowledged, the fact that the major pandemic may have had an iatrogenic origin would not *require* that the minor outbreaks also had an iatrogenic origin. My personal position at present is that either theory of origin (natural or iatrogenic) is tenable for HIV-2, and for HIV-1 Groups O and N, not least because it is possible that the minor outbreaks were only recognised by the medical community because of the research that had already taken place in response to the Group M-related pandemic.

³⁶⁸ Transcript of relevant passage from Chuck Cyberski’s interview with Leonard Hayflick, recorded May 7th, 1992 at UCSF in San Francisco, and made available by Blaine Elswood.

³⁶⁹ *River*, 2000, page 487. See also page 488, where Hayflick admits that Koprowski had done “things behind my back which I didn’t appreciate”, which included “something which I thought was underhand”. One wonders what these “things” might have been, and whether they included the early testing in Africa of vaccines made in experimental substrates, such as human cells.

³⁷⁰ It was Elswood who, back in 1991, had initially alerted Tom Curtis to the potential importance of the CHAT vaccine trials in Africa.

³⁷¹ Letter from L. Montagnier to H. Koprowski, February 12th, 1992, made available by the Albert B. Sabin archives at the University of Cincinnati.

³⁷² *River*, 2000, pages 802-804.

³⁷³ For the local preparation of Sabin's strains in the Soviet Union, see: A.A. Smorodintsev et al., "Experimental and epidemiological data on the effectiveness of live poliomyelitis vaccine. Part 1. Experience in the production, biological control, and use of live poliomyelitis vaccine made from the Sabin strains", in *First international conference on live poliovirus vaccines: papers presented and discussions held* (Washington D.C., June 22-26, 1959), Pan American Sanitary Bureau, 1959, Scientific publication No. 44; pages 305-312. This reveals that the first batches of Sabin's vaccines were further attenuated and then produced in rhesus macaque tissues in the USSR in 1957, and that further batches were prepared in the tissues of other Asian monkeys in 1958-9. The local preparation of polio vaccines in South Africa was being reported in the literature by 1956 (for Salk's IPV) and 1959 (for Sabin's OPV). [J. Gear, "The South African poliomyelitis vaccine"; *S. Af. Med. J.*; 1956; 30; 587-594. Anon., The South African Institute for Medical Research, annual report, 1959, pages 134-135.] For a summary of the South African trials, see: Anon., "Immunization against poliomyelitis"; *S. Af. Med. J.*; 1963; 37; 497-498, and the equally important articles on the following pages: 499-518.

³⁷⁴ "Meretricious" is defined in the Cassell Popular English Dictionary as: "pertaining to or befitting a prostitute, alluring by false or empty show; unreal, tawdry."

³⁷⁵ S.A. Plotkin, "CHAT oral polio vaccine was not the source of human immunodeficiency virus type 1 Group M for humans"; *Clin. Inf. Dis.*, 2001, 32, 1068-84.

³⁷⁶ W.D. Hamilton, "Foreword"; *The River*, 2000, pages xxvii-xxxiii.

³⁷⁷ Dr Weiss added that this was an inversion of the original phrase, which involved "ugly facts" destroying a "beautiful theory". I have since been told that the original phrase may have first been coined by Thomas Henry Huxley, but have been unable to confirm that.

³⁷⁸ B. Lederer, "Chimp and see"; *POZ*; December 2001, p. 17.

³⁷⁹ Anon., "Protest is an ally of science" [editorial]; *Independent on Sunday* (London); May 26, 2002.

³⁸⁰ J. Crewdson, *Science fictions. A scientific mystery, a massive cover-up, and the dark legacy of Robert Gallo* (Boston: Little, Brown, 2002), p. 627.

³⁸¹ J. Creamer (ed.) *Biohazard: the silent threat from biomedical research and the creation of AIDS* (London: National Anti-Vivisection Society, 1987).

³⁸² The man who sent the letter was a Dr Herbert Ratner, from Oak Park, Illinois, and he included various articles; [such as: H. Ratner, "Monkey viruses, AIDS and the Salk vaccine, Parts I and II"; *Child & Family*; 1988; 20; 134-138; see also *River*; 2000; pp. 236-237, and notes 1 and 2 on page 951.] This was the same Herbert Ratner who, in 1955, had reservations about the safety of the Salk vaccine, and refused to inject it into his patients. His caution seemed well justified a few days later, when the Cutter incident showed that some batches of that vaccine had been incompletely inactivated. Ratner's samples of the Salk IPV stayed in his freezer for more than 40 years, but just before his death he gave them to Michele Carbone. Dr Carbone (who had been unable to procure such samples from the FDA, which had apparently destroyed all its ancient stocks in the early 1990s, officially as a cost-cutting measure; see: *River*, 2000, page 866) tested the Ratner samples, and found them positive for SV-40. So despite being wrong about the origins of AIDS, Dr Ratner has none the less played a heroic role in protecting the public health.

³⁸³ T. Curtis, "The origin of AIDS: a startling new theory attempts to answer the question: 'Was it an act of God, or an act of man?'" *Rolling Stone*; March 19, 1992; issue 626; pp. 54-60, 106 and 108.

³⁸⁴ J. Cribb, *The White Death* (Sydney: Angus and Robertson, 1996); see pp 254-257 for the Hamilton letter to *Science*, and 182-184 for Cribb's analysis.

³⁸⁵ Extract from referee's comments on Hamilton's letter, forwarded by editorial staff at *Science* to W.D. Hamilton on April 28th, 1994. The typed text at this point reads "answered", but an unknown hand has written "un?" beside the word, and it seems clear from the context that the writer [i.e. R. Weiss] meant "unanswered".

³⁸⁶ *River*, 2000, page 877.

³⁸⁷ For background to this episode, see *River*, 2000, pp. 508-511.

³⁸⁸ E-mail from R.A. Weiss to B. Martin, June 8, 2000.

³⁸⁹ S. Wain-Hobson and R.A. Weiss, "If free speech costs lives that's a high price to pay" [letter], *Nature*, 2000, 407, 834.

³⁹⁰ Dephlogistication: a "depriving of phlogiston", here in the figurative sense.

³⁹¹ S. Wain-Hobson, *The River – a journey back to the source of HIV and AIDS* (review); *Nature Medicine*; 1999; 5(10); 1117-1118.

³⁹² *River*, 2000, pp. 852-858.

³⁹³ A brief search in *Nature* has not revealed the letter in question. Possibly it ended up as the “If free speech costs lives...” letter cited above, and was finally signed not by fifty scientists, but by just the two of them.

³⁹⁴ Letter from S. Wain-Hobson to W. Nelson-Rees, October 19, 2001. This rather crude attempt to separate Nelson-Rees and myself is especially surprising, given that Nelson-Rees had ended his Royal Society talk by frankly stating that there was “no logical reason” why chimp cells would not have been used (either at the Wistar or elsewhere) to grow the CHAT vaccine used in Africa, and that the prevailing custom in the fifties was to use cells “about which little or nothing was known, except that they could optimally support the growth of a given virus”. The fact that two letters have been written by the Royal Society organisers seeking to persuade others to modify their positions on (or public statements about) the OPV debate (one to Brian Martin from Robin Weiss, and one to Walter Nelson-Rees from Simon Wain-Hobson) suggests that other, more successful approaches may have been made to other scientists.

³⁹⁵ B. Martin, “The burden of proof and the origin of the acquired immune deficiency syndrome”; *Phil. Trans. R. Soc. Lond B*; 2001; 356; 939-946.

³⁹⁶ B. Martin, “The politics of a scientific meeting: the origin-of-AIDS debate at the Royal Society”; *Politics and Life Sciences*; 2003; in press. See also: B. Martin, “Investigating the origins of AIDS: some ethical dimensions”; *J. Med. Ethics*; forthcoming.

³⁹⁷ Bill Hamilton apparently wrote this in a letter to Professor Patrick Bateson, secretary of the Biology section of the Royal Society, in around June 1999.

³⁹⁸ The geographer, Daniel Low-Beer, did make a significant and more open-minded contribution. However, since he was only a “discussant”, not a full speaker, he was allotted only seven or eight minutes on the second day. None the less, he had time to make some interesting points – notably the very short average distances from CHAT vaccination sites to sites of AIDS cases, especially at the start of the epidemic. He also pointed out that if Kinshasa really was a hub, it was hard to explain why HIV-1 spread had only taken place eastwards from there, and not to the north or south. D. Low-Beer, “The distribution of early acquired immunodeficiency syndrome cases and conditions for the establishment of new epidemics”; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 927-931.

³⁹⁹ The association between Plotkin and Moore had been rumoured for several months before Plotkin thanked Moore, at the end of his article in *Clinical Infectious Diseases*, for “lending me some of his courage to face defamatory accusations”. Apart from the instances already detailed in the first section of this paper, Moore’s “courage” has apparently involved approaching several of those who had seen merit in the OPV theory, sometimes sending them abusive letters, and sometimes inviting them to friendly meetings, because he happened to be about to visit their part of the country. According to several sources, Moore was also actively engaged trying to persuade people not to attend the Royal Society meeting. He publicly declared that he himself would not be attending, having “more important things to do” (but neglecting to add that he had only been invited as discussant, not as a speaker). Later, however, when he realised that most key figures in the debate were going, he after all found the time to attend. I myself received two unsolicited e-mails from Moore after he read my description of his *Nature* review of *The River* as “scurrilous” [*River*, 2000, pp. 851-852]. They opened by accusing me of being a “tenth-rate journalist”, and went on from there. One of these e-mails was copied to doctors Weiss and Wain-Hobson, and Robin Weiss subsequently offered to tell Moore (a former pupil of his) to “cool it”. I thanked Weiss, but told him that I would prefer him not to intervene. Since then, however, I have not heard further from Moore, (although I am told that he did ask me a question from the floor during the Royal Society meeting). I believe that it is important that people are able to see how Dr Moore operates, and for this reason his two letters to me will be posted on Brian Martin’s web-site. All in all, Moore seems to have acted from the outset as a spin doctor for Plotkin and Koprowski and the natural transfer school, and one wonders what his motivation, or incentive, may have been.

⁴⁰⁰ Brian Martin’s web-site, at <<http://www.uow.edu.au/arts/sds/bmartin/dissent/documents/AIDS/>>.

⁴⁰¹ At one point, Plotkin even claimed that I had wrongly included two cases of AIDS from Kikwit in my analysis of early cases, and produced a map to support this claim; unfortunately, the two cases did not appear in the map in my book, but only in his version of it!

⁴⁰² Of particular interest is the letter from Dr Koprowski to George Jervis in the Congo, dated March 4, 1958, of which only the postscript is quoted. Plotkin states that there is “no reference to local production in the Congo”, but for obvious reasons, it would be valuable to be able to see the rest of the letter.

⁴⁰³ These remarks were spoken by Dr Weiss in his closing address at the Royal Society meeting, but do not feature in the published version of his speech.

⁴⁰⁴ J. Cohen, "Disputed AIDS Theory Dies its Final Death", *Science*, 2001; 292; 615. I had had dealings with Jon Cohen in the summer of 2000, when I spent (I estimate) a total of five or six hours on the phone answering his questions while he was preparing two big feature articles for *Science* and *The Atlantic Monthly*. Rather to his chagrin, I sensed, he was unable to catch me out on any awful blunders, or devious motives. (At one point, he even told me that some scientists feared that I might be able to out-argue them at the London meeting, because I had spent so much time devoted to the subject – as if this, somehow, gave me an unfair advantage.) The article that Cohen wrote for *Science* was largely fair. [J. Cohen, "Forensic epidemiology. Vaccine theory of AIDS origins disputed at Royal Society"; *Science*; 2000; 289; 1850-1851.] Later, however, came the *Atlantic* piece, which rather cravenly reported the words of OPV sceptics like Beatrice Hahn, and which performed a gentle but none the less determined assassination job on me. [J. Cohen, "The hunt for the origin of AIDS"; *Atlantic Monthly*; October 2000.] After that, I decided to cross Cohen off my greetings card list.

⁴⁰⁵ Another person who was less than happy with the published proceedings of the Royal Society was Walter Nelson-Rees. Certain changes were made to his copy, allegedly for legal reasons. The fact that one scientist had repeatedly lied about the origin of his cells was omitted, even though this information had already appeared in a 1981 article in *Nature*. [D. Dickson, "Contaminated cell lines"; *Nature*; 1981; 289; 227-228.] And an extra sentence was inserted before one of Nelson-Rees's paragraphs which quoted John Maddox, the former editor of *Nature*, who ended a 1981 editorial on the topic as follows: "There is no reason to suppose that the few cases of dishonesty that have come to light are in any sense the tip of an iceberg....It would be tragic if civilised habits [of honesty in research] were to be corrupted by the activities of self-appointed vigilantes." [J. Maddox, "Responsibility for trust in research", (editorial); *Nature*; 1981; 289; 212-213.] This appeared to be a fairly frank closing condemnation of whistle-blowers like Nelson-Rees. The sentence that was inserted at the start of Nelson-Rees' paragraph in the Royal Society proceedings read as follows: "Editors of scientific journals do have to strike a balance between undue criticism of people's ethics, and uncritical acceptance of their results." Nelson-Rees wrote to the editorial office of the Royal Society, protesting the changes which had been made, and observing that "the points I am making are precisely the sorts of items I had intended to talk (write) about under the title of 'Responsibility for Truth in Research'." He eventually accepted the changes, but only "reluctantly". [W. Nelson-Rees, "Responsibility for Truth in Research"; *Phil. Trans. R. Soc. Lond. B*; 2001; 356; 849-851.] Nelson-Rees points out that Sir John Maddox is on the editorial board of the Royal Society journal which published the proceedings, "Philosophical Transactions B". [W. Nelson-Rees, personal communications, May 2002.]

⁴⁰⁶ Found at <<http://www.uow.edu.au/arts/sds/bmartin/dissent/documents/AIDS/>>.

⁴⁰⁷ W.D. Hamilton, "Foreword"; *The River*, 2000, pages xxvii-xxxiii.

⁴⁰⁸ T. Radford, "Britain losing its scientific grip, says PM", *Guardian* (U.K.); May 24, 2002, page 13.

⁴⁰⁹ This sentence was updated to July 2002.

⁴¹⁰ This is my recollection of the figure that Dr Maddox suggested, though I see that in an account of this episode penned only a couple of weeks after it took place, I wrote "100 million". It hardly matters, for whichever it was, it was a significant number of millions.

⁴¹¹ R. Weiss, "Reflecting on the origin of human immunodeficiency viruses"; *AIDS and Hepatitis Digest*, January 2002, page numbers unknown, but available on Brian Martin's web-site..

⁴¹² *River*, 2000, pp. 384-387. The rest of Chapter 28 contains other similar examples.

⁴¹³ G. Courtois, H. Koprowski et al., "Preliminary report of mass vaccination of man with live poliomyelitis virus in the Belgian Congo and Ruanda-Urundi"; *Brit. Med. J.*; 1958; 2(i); 187-190. This is the paper which was, in reality, written by Koprowski, and merely sent to Stanleyville for checking.

⁴¹⁴ G. Courtois, "Vaccination antipoliomyelitique par virus vivant au Congo Belge"; *Ann. Soc. Belge Med. Trop.*; 1958; 38; 805-816.

⁴¹⁵ Letter from W. Bervoets to P. De Brauwere, September 17th, 1958; file H4484/1058 at the Belgian Ministry of Foreign Affairs archives.

⁴¹⁶ *River*, 2000, pages 221-222, 416 and 734-736.

⁴¹⁷ An additional clue that Koprowski was aware of at least some of the data that Bervoets reported to De Brauwere in his letter of September 17th is that although Koprowski records 2,433 vaccinations at "Bambesa and Kule-Ponge", he mentions "2,350 inhabitants" for Bambesa in the text. 2,350, according to Bervoets, is the number who were actually vaccinated at Bambesa, although it was not the full population of the village.

⁴¹⁸ This lack of candour may once again be in evidence in Koprowski's vague claim in his *BMJ* article that, in Stanleyville, "a large group of schoolchildren, mostly European in origin, were vaccinated with

living virus every week during...the 12 months preceding the mass trial at Ruzizi Valley”. None of the Belgians I spoke to could recall such an early and ongoing vaccination of Europeans in Stanleyville, and the Bervoets letter states simply that overall, the vaccinations had included “a certain number of European volunteers”. I believe that Koprowski’s version of events represents, at the least, a substantial exaggeration.

⁴¹⁹ *River*, 2000, pp 864-865, and see note 81 on page 1066.

⁴²⁰ The university at Leuven, and the affiliated Rega Institute, had been directly involved with the original CHAT research through the virologist Dr Pieter de Somer.

⁴²¹ I have a couple of suggestions to make of “neutral” institutions, where scientists are known to be interested in participating in testing the Kisangani samples.

⁴²² Dephlogistication: the relieving of inflammation.

⁴²³ Despite the comments made in this paper, I find it difficult not to feel some real sympathy for the position of Dr Paul Osterrieth, not least because it is clear that whatever was done in Stanleyville was very likely done under instructions from “superiors”. However, I do believe that it is now time for him to give a full and candid account of what happened at Lindi and in Stanleyville. This might well stir up a hornet’s nest, but I believe that the courage and integrity that such a baring of the soul would require would also engender a good deal of respect, understanding and support from his peers.

⁴²⁴ P. Osterrieth, “The truth is what happened and not what one wishes that had happened”, November 20, 2001. Available at: <http://www.aegis.com/news/misc/2001/PO011101.html>

⁴²⁵ The epigram to Dr Osterrieth’s disclaimer seems rather a strange choice. It reads: “The point is that creationists and social critics who decry science as dogmatic obedience to authority and old-boys networks of closed-minded fogies are simply mistaken”, and it comes from an article by Michael Shermer in the October 2001 edition of *Scientific American*, which was entitled “I was wrong”.

⁴²⁶ A. Jung, A. Meyerhans, et al., “Multiply infected spleen cells in HIV patients”; *Nature*; 2002; 418; 144.

⁴²⁷ J. Cohen, “Tough challenges ahead on political and scientific fronts”; *Science*; 2002; 297; 312-313.