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 E[315] 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 Cameron, 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 *troglodytes* 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 *troglodytes* 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 *troglodytes* 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 Cyangugu, 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 Beheyt, 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 proably 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 Lépine'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 Vandamme[342] 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 straightfoward. 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 Lépine, 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 remembereed 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. Lépine'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 Mitzic, in what is now northern Gabon.[359] The doctor in charge (Dr L-J André) 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 Lépine 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 André 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 André, "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 Lépine 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 Lépine 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 Lépine'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 direector of that lab, from 1961 onwards. He told me that Pasteur polio vaccines were administered throughout AOF, in present-day Guinea, Senegal, Mali, Niger, Côte 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 Mitzic. If this detail is correct, then this would represent a major difference between the European and African polio vaccines of Pierre Lépine. (Lépine's polio vaccines were given in France from 1957 onwards, and in West Germany in 1958, and only IPVs 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 (Côte 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 Mitzic 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 Lépine, 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 Lépine.)

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 meretricious[374] 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 unanswered[385] 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 tinkerings were 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 dephlogistication[390] 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".

<u>j) Phantom science.</u>

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