

1959 MANCHESTER CASE OF SYNDROME RESEMBLING AIDS[†]

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Bailey and Corbitt's letter to *The Lancet* about the 25-year-old man who died in Manchester Royal Infirmary, UK, in August, 1959, with a clinical syndrome resembling AIDS¹ is welcome but it leaves several points unresolved, including some raised by a science journalist in March, 1995.²

A particular puzzle is that the original polymerase chain reaction (PCR) study³ was claimed to be of a randomised double-blind design. Properly applied, such a design makes it difficult for an interpretative bias to generate a false positive or negative result, and impossible for random contamination to do so. On application of Fisher's exact test to the results of 1990, the probability that random contamination of the test and control samples would produce four positive results in six test samples and none in six controls is 1 in 33. Occurring in 1990, before the dangers were fully appreciated,⁴ accidental contamination in the first PCR study of a potential early case of AIDS would be understandable. However, the subsequent failure to address the statistical anomaly above and the neglect of other anomalies is not. We wish to highlight not only the mystery of how random contamination could have led to the results but also five more questions. For the third and fifth and partly for the fourth we suggest possible answers; the other two remain open. The questions are:

- (1) How did the original tissue samples from the patient come to be found HIV-1 positive by PCR when these results cannot now be repeated?
- (2) How have archival human tissues, which were apparently well enough preserved in 1990 to allow human and viral genetic analysis after 30 years in storage, apparently ceased to be so in the past five years?
- (3) Accepting contamination,¹ what is its likely source?
- (4) How have four (and possibly five) different human genotypes been reported for HLA-DQ α in tissue samples claimed to be from one cadaver?
- (5) What was the patient's fatal disease?

[†] *The Lancet* 348, 1363–1365 (1996).

Contrary to speculation mainly, but not wholly, in the non-medical press, investigations by EH have shown no evidence to suggest that ‘the Manchester sailor’ (MS) was either homosexual or bisexual, or that he ever visited Africa. In early 1957 his ship did dock in Gibraltar for a fortnight. A day trip (well recalled by members of the ship’s company) was made by about a dozen sailors to Tangier in Morocco, but a member of that party has no recollection that MS was present. Even if he was, or there were other day trips to Tangier, and even if (as has been hypothesised) he had sex in a brothel during such a visit, this can hardly be characterised as a high-risk episode. HIV prevalence varies widely across Africa and the seroepidemiological evidence suggests that Morocco has always been among the least affected countries. The earliest evidence of HIV infection in the country pertains to 1984–87, when seven of 8161 individuals (0.086%) tested positive, all from Casablanca. Six were in high-risk groups (gay men, male prisoners, and female prostitutes), the seventh was one of 3577 blood donors. None of 283 blood donors and pregnant women tested in Tangier in 1991 proved to be HIV-1 positive.⁵

Questioning MS’s fiancée, family, friends, colleagues, and doctors suggests that he was neither sexually adventurous nor very experienced, and that he was not an intravenous drug user and had received no blood transfusions. Clearly one sexual encounter could have been enough, but everyone who knew him rates him as an improbable candidate for HIV infection. Those closest to him were saddened, indignant, and (rightly as it now appears) near to incredulous at the suggestion that he might have died of AIDS.

That incredulity is now borne out by Bailey and Corbitt,¹ who have joined Zhu and Ho⁶ in concluding that the posthumous AIDS diagnosis was unsound, and that certain of the archival tissues made available to them may have been or have become contaminated with a modern (subtype B or ‘Euro-American’) strain of HIV-1. They suggest contamination ‘sometime from sectioning onwards’, and that the most likely source ‘would be from within our own laboratory’.

The following scenario might go some way towards explaining the facts. The positive control used during the PCR work on MS was a CEM cell line infected with CBL-1.¹ In 1991, Weiss reported that CBL-1 had 98.0% identity with LAV-1 BRU (or, as it is now referred to, LAI) and 97.8% identity with HTLV-IIIB in *env*, *tat*, and *nef*.⁷ An accompanying commentary on this ‘remarkable similarity’ cited laboratory contamination as the possible cause,⁴ and reported that Gerry Myers of the HIV Sequence Database in Los Alamos considered that up to 3% divergence in *env* usually indicated different isolates from the same person, whereas, at the other extreme, genuinely unlinked isolates usually diverged by more than 10% in the envelope gene.

The earliest versions of LAI are the French patent application sequences bearing the Genbank/EMBL acquisition numbers A04321 and A07867, and Fergal Hill, of the MRC Laboratory of Molecular Biology in Cambridge, has characterised A04321 as ‘apparently the most similar sequence to the Manchester isolate sequence currently known—at approximately 90% identity

over large tracts, including the envelope gene'. Hill concludes that 'this high degree of sequence similarity, and the fact that CEM/CBL-1 was grown in Manchester, *strongly* [his emphasis] suggest that the Manchester isolate is...derived from LAI via its derivative CBL-1'. Clearly Hill believes that repeated passaging of CBL-1 (for instance in Corbitt's laboratory) could explain the 10% divergence between this positive control and the MS isolate. Myers is less convinced, considering that 'the contaminant may have been a lab strain, or...another patient sample'.

We have already mentioned that only MS's tissues came to be contaminated in spite of their random interspersions with the controls. Thus conventional significance points either to earlier contamination, before the coding and dispatch of the samples to Corbitt's laboratory (in which case considerations of the last paragraph suggest that the CEM cell line might also have been present in the source laboratory) or to error during the breaking of the codes. Sections were cut 'with separate knives for case and control and with careful cleaning, with alcohol soaked swabs, of knives between blocks'.³ If we accept that the procedure was as stated, the best scenario at this point would seem to be that a knife cleaned neither before nor between section cutting happened to be contaminated with modern HIV-1-infected tissue and thus passed not only HIV-1 DNA but also appreciable human cell material to the first four sections, which happened to be from MS. By the fifth and subsequent cuttings the knife supposedly had wiped itself clean. As discussed below, however, there are still many problems.

The hypothesis of prior contamination might be clarified by a detailed description of the storage and location of the two sets of tissues, and of how and where sectioning was undertaken. EH learned from one of the doctors involved that for at least a part of the period of the PCR investigation the blocks were being stored in Williams' home, and Williams later confirmed this.

Both Corbitt and Williams told EH that the code had been broken during a telephone call, in which Corbitt read through the list of numbered samples, indicating for each whether or not the presence of HIV had been demonstrated, and Williams then broke the codes, indicating which samples had come from MS and which from the control patient. Corbitt states that nobody else was in the room at the time; Bailey was waiting outside. A more appropriate method might have been an exchange of sealed envelopes and the presence of witnesses when the envelopes were opened.

Further examination of the original MS tissues and of the PCR products from Corbitt's laboratory is needed. In the past, Williams has stressed that there was little tissue available and that he had been keeping a judicious eye on what remained to ensure that not all was used up.⁸ But he acknowledges that about 40 blocks were taken at necropsy. These originated from a wide variety of skin lesions, together with bone marrow, heart, lung, and central nervous system, and abdominal viscera (including liver, kidneys, pancreas, and spleen), and even

if most of the tissues are not ideal for finding lymphotropic virus, some DNA from an overwhelming virus infection should be detectable. Extraction of human DNA should be feasible from any of the samples. Perhaps the Central Manchester Health Care Trust could reveal exactly what tissue remains and perhaps some of the blocks could be examined by another laboratory. One laboratory, experienced in PCR and in sequencing lentiviruses, made a written offer to test tissues from the patient in March, 1995, in response to Williams' statement² that he would 'be quite happy to supply tissue to anyone who would take it on'. This offer was apparently forwarded to the Trust but was neither acknowledged nor accepted.

Five human genotypes for MS have been mentioned.^{1,6} Zhu and Ho found that three HLA-DQ α genotypes had been sent to them, with traces of a fourth. In material from Corbitt they found type 1.2,4 'with traces of 2,3' in kidney and 1.2,3 in bone marrow. In material from Williams, on the other hand, they found 3,4 in thyroid, liver and kidney. Bailey and Corbitt now report that, working on samples received from Williams in 1989 (those from 1995 having been found unusable), they detected 2,4 in liver and brain. They also found human type 2,4 in the CEM line that was their HIV-positive control in 1990. The frequency of 2,4 in Britain is likely to be well below 5%.⁹

If Zhu and Ho's interpretation of their bands was at all equivocal and '2,4 with a trace of 1.2,3' for kidney and bone marrow is a possible alternative to their stated '1.2,4 with traces of 2,3' the inconsistency of the New York and Manchester accounts would be greatly lessened: 2,4 could then be due to the contaminating CEM cells, and Zhu and Ho's technique, perhaps more sensitive than that of Bailey and Corbitt, could be revealing the underlying tissue type 1.2,3, exactly as found by Zhu and Ho in bone marrow which had seemingly escaped contamination.³ MS would then have a puzzle of only two genotypes; a third would be due to the CEM cells.

Perhaps both DNA and proteins of the wax block material were so degraded that they provided weaker and sometimes undetectable signals relative to those provided by a recent cell contaminant, when present. This is further suggested by the partial and wholly negative results obtained, respectively, by Bailey and Corbitt and by the UK Forensic Science Service.¹ However, the idea that contaminant CEM cells explain all the genotyping and viral results since 1989 still involves many difficulties, whether that contamination arose in the laboratory where sectioning took place or in Corbitt and Bailey's laboratory.

Turning to the nature of the patient's disease, Bailey and Corbitt express themselves puzzled and reiterate that the symptoms were, retrospectively, very suggestive of AIDS. We believe, however, that the diagnosis has become the least of the problems of the case. It would be flippant to suggest that a patient with five HLA genotypes—more diploid combinations, it may be noted, than are known for any chimera apart from a few Panamanian strangler fig trees¹⁰—would of necessity be a simmering cauldron of autoimmunity and

immunocompromise. Let us propose two plausible alternatives. MS may after all have had Wegener's granulomatosis. This was the working diagnosis for the final two months of his life and for more than seven weeks after his death the gross post-mortem findings were being described as 'consistent with [this] diagnosis'. Only when the microscopic findings revealed cytomegalovirus and *Pneumocystis carinii* was this diagnosis abandoned.

A second possibility is CD4+T-lymphocytopenia (CTL). This condition was christened 'AIDS without HIV' when its existence was first announced at the Eighth International Conference on AIDS in 1992.¹¹ Other publications quickly followed (e.g. Laurence *et al.* in 1992¹²). Rezza *et al.*¹³ mention a 39-year-old man without HIV infection who died as a result of a wasting syndrome, *P. carinii* pneumonia, disseminated cytomegalovirus infection, and neurotoxoplasmosis. Apart from the *Toxoplasma* infection, the clinical profile matches that of MS. Dr T B Stretton, one of the MS physicians in 1959, now leans towards this retrospective diagnosis.

If MS did die from AIDS it is vital to our understanding of the early history of primate immunodeficiency viruses that an authentic sample of HIV DNA from such an archival case be made available for sequencing and phylogenetic analysis. Besides the controversial postmortem tissues, biopsy specimens were taken from sternal marrow, scalene region (including a lymph node), and ulcers and skin lesions. Perhaps these are still available at the Manchester Royal Infirmary.

If, however, as we believe, this patient did not have AIDS, and if there was either substantial contamination with modern HIV DNA or tissue samples from other patients came to be included in the PCR investigations, then this man's family and fiancée are owed an apology for the distress which this episode has caused them.

Unsources information in this article is based on tape-recordings and notes of interviews between EH and the various scientists mentioned, personal letters from some of these scientists, and medical records of the patient, viewed with permission of his next-of-kin.

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FOREWORD TO E. HOOPER, THE RIVER[†]

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Every time 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.

Evasion and untruth have long been known to be beneficial at many levels and useful to people in many ways. They can be presented as virtues—the little bads that add to a greater good, with a proviso, of course, that the good is of a kind that the colluders believe only they know how to attain. ‘Don’t we have faith in ourselves?—let’s keep it simple for their—for all our sakes.’ Even for God’s sake: this version has been abundantly illustrated by religious leaders ever since Christianity became official in the Roman Empire, with disastrous effects upon other faiths—and a fiery impact upon a myriad of free-thinking ‘witches,’ as well as the occasional literary loner like Giordano Bruno. Once there is acceptance by an ‘establishment,’ there is often no need to whisper about it anymore: 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. And yet, knowing the untruths that emotions arouse, especially in groups, Plato amazingly denied roles even for poetry and music in his ideal Republic.

Most of the daily untruths communicated need not be taken too seriously: we have become accustomed to them and in a sense self-vaccinate. However, when eminent rivals in an ancient profession are seen to be uniting to crush an outside critique, and when the best-funded branch of science, to which the rivals belong, draws almost all its practitioners into line behind them (as Louis Pascal and then Tom Curtis in the case treated in this book had already experienced, even

[†]In E. Hooper (ed), *The River: A Journey Back to the Source of HIV and AIDS*, pp. xxvii–xxxiii (Harmondsworth: Allen Lane, The Penguin Press. Boston: Little, Brown, 1999).

before Hooper), and when an expectant and immensely wealthy international industry is also seen marching in step with the profession in question, it is time for the rest of us to wake up.

The thesis of *The River* is that the closing of ranks against inquiry may, in this case, be preventing proper discussion of an accident that is bidding to prove itself more expensive in lives than all the human attritions put in motion by Hitler, Stalin, and Pol Pot. Furthermore, essentially unwarned by what we have recently done, we may be moving rapidly toward further and perhaps even worse disasters of the same kind. Some aspects of genetic engineering may indeed be dangerous, but a situation in which the general public has greater concerns about mystical subversion of the chemicals in soy sauce than about the risk of viruses in live animal products that are already administered, almost compulsorily, to our bodies, is near to absurd. In parallel to this, our doctors' Hippocratic Oath warns them of various temptations and dangers, but it says nothing of how they need to guard themselves, and their profession, against the effects of the millions of profit that dangle before the nascent industry proposing to transplant organs into humans from other species.

These are the foreground dangers emphasized by Hooper in this book. Its background has another danger, which is still more insidious. Litigation has been used to suppress the publication of discussions about a hypothesis; litigation is again being used as a threat to Hooper. 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, while generally avoiding 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.

It is the foreground, the potential repercussions in the next thirty or so years, which will probably most arouse the reader of this book. Perhaps something is being tardily seen by the establishment. A few months ago, the British Medical Association announced revisions to the Hippocratic Oath British doctors must take; then just a week ago, as I write, the Association's organ, the *British Medical Journal*, published for the first time an admission of a likelihood that Simian Virus 40, established as an infection in millions of humans by the Salk polio vaccine, is causing human cancers. 'Salk,' it may be remembered, is the 'dead' and therefore safer polio vaccine—safe supposedly not only from reversions to virulence but from the possibility of 'extraneous agents.' It is quite different from the type focused upon in this book—the type we now all receive. On another front, committees in recent months have enjoined slowness and caution with xenotransplants, but not before the first baboon liver transplant into a human was attempted—an operation that perhaps fortunately failed. Meanwhile heart valve implants from pigs, a species known to harbor retroviruses that can live in human tissue cultures, are in trial and application.

All this is why the world still very much needs lone researchers like Edward Hooper. They reach truth faster than committees. Shortly after I first knew him, I introduced him to someone as a journalist, knowing he had formerly been

one in Africa. Later he asked me, pained, 'Why journalist? Couldn't you call me a *writer*?' I did so from then on but stayed puzzled. Weren't journalists supposed to be the guardians of our free world, the para-predators ranging our savannah and making even the most lordly lions take care of their actions? Weren't they (the best at least) even cousins to us scientists, ferrets setting themselves to bolt the most willfully concealed and elusive truths of history where we scientists deign only to chase the immobile targets, such as atoms and missing links? Why should one not want to be a journalist? After reflection and listening to the talk of 'paparazzi' and the like that came after Princess Diana's death, I think I see better now the perspectives that journalists dread—but just as hyenas do less scavenging and far more primary predation than was once thought, so also do the best journalists.

Whatever, this book, with its almost 2,500 footnotes, demonstrates how Hooper has finished up. Not only is he the kind of predator that all in Big Science should fear, but he is a writer and historian as well. Even that is not all. He has self-taught his way to 'honorary' status in several branches of science—to be almost virologist, almost geneticist, almost evolutionist. To most of us, however, these achievements just provide the reassurance that he is writing sense in his diverse fields; in contrast it is the writing itself and the history—dare I say even the first-class journalism?—that will keep us bent over the pages that follow. What scoops, what personalities, what landscapes, what far places! Above all what enigmas, what awful inexorable tragedy (tragedy at its deepest, gnawing within millions of homes—a scale perhaps grander than any ever before described) stand there behind!

In 1995, in Africa for another purpose, I tried to help Ed by looking for some of the Ugandan friends who had helped, nearly a decade earlier, with the research for his first book, which described the AIDS disaster in that focal area close to the shores of Lake Victoria. There were two men in particular whom he wished to contact and to thank. As I discovered after some questioning, both had died. I was led to the father of one, and he in turn took me to a neat private graveyard in his *matoke* plantation and showed me the newly heaped mounds, six in all. They were for his wife and all his children. One mound, with a stone slab, was for the son Hooper knew, a local government official (who had been, perhaps, a little more important locally than the others). The old man sat on a corner of the slab and read the letter Ed had sent, while two grandchildren, come into his care after the last death, watched from nearby. The children were lively and healthy but very quiet, and I hoped the infection was going to miss them. Such graveyards, I found, were everywhere in the district, though they are not much seen from the roads. Orphans, too, were everywhere: a generation had been scythed out from between those who were too young and too old to be readily infected. I saw children in groups ranging from teens to tots seemingly loose and self-foraging in the countryside, which included as it happened trying to forage from me, the passing foreigner. Presumably these were the children

not lucky enough to have grandfathers and grandmothers who were still alive. Both in the robust elderly and in these youthful gangs I felt I was seeing how Africa would survive, if only after a period of great suffering. Yet it may end up less changed, it seemed to me, than will the continents of the First World, in spite of our lower expected mortalities.

After that brief experience in southern Uganda—a few days only—I understood better what had been driving Hooper to follow up on the lighter and more emotional book he had already written about the epidemic in Africa. I suspect he had no idea, at the start, of the magnitude of what he was undertaking, nor of the nine-year odyssey of research and travel it would require. Even before he read Louis Pascal's extraordinary paper 'What Happens When Science Goes Bad . . .' and had realized the full tragic possibility about the origin that it raised, he had been aroused by personal indignation to far more energy over the epidemic than had most of the rest of us. In the late eighties in Nairobi and Kampala, he had seen friends sicken and die around him. Despite this, in the nineties he was still finding Westerners who claimed it was all untrue, and that there was no epidemic. Instead, false trails and absurdities were glibly promoted; hypotheses were floated that seemed aimed, even from the first, to lead into impenetrable bush. At the same time, as he found later, much better hypotheses about the epidemic were studiously ignored and had needed tortuous paths to achieve any public notice at all. The ideas and research of New York-based Louis Pascal, for example, had to be published in Australia, and the investigations of science journalist Tom Curtis went perforce to an outlet in a popular magazine, *Rolling Stone*. Neither piece was much followed up.

Without question it is science that will shape the human world of the Third Millennium. Even if science can only direct us back to a dark age it will still be our cause and our guide. But it could be made to do better or worse. There is a risk that science is going to lose its fertility and change radically away from that spirit of free inquiry and exchange that first inspired the Greek and then later the Renaissance experimenters and philosophers. Indeed, this process seems to be starting already; patenting and secrecy about gene sequences are perhaps one symptom. Science may bring on us not so much a dark age in the old sense, via some spectacular collapse, but rather a super-technological state whose monstrous futures—if they could be shown to us dearly through the present smoke of excitement about more and ever more technology—would only arouse our dread. While still working its miracles on the outskirts, science may already, at its center, like a great city, be slowly dying of its very success. Dictators and businessmen everywhere want to use all the technical products of science and, if possible, to control the rights and the how-tos for creating more. They would also like to be free to hide the results of their unsuccessful or disastrous experiments.

After reading Pascal's paper, it was a great shock to me that when I passed out copies to others whom I thought would be interested, including a journalist

who had written on AIDS for a major popular science magazine, I met with exactly the wall of silence Pascal had described. From being at first impressed mainly by his theme about the origin of AIDS, I thus began to believe his arguments about scientific integrity as well—arguments that at initial reading had seemed to me just overreactions generated in a sensitive, frustrated man. Only one person (from the medical fraternity, surprisingly) replied to my mailing with any sign of taking the paper seriously. Even my old mother, a doctor, told me, 'You are going to be very unpopular if you pursue that one—polio of all things, that one is sacred! Anyway, if it's true, it's all happened and what could you do?' Well, personally I didn't pursue anything very far; after several tries with the editors of both *Science* and *Nature*, I lapsed back again into the general silence. Overall I have left it to Pascal, Curtis, Julian Cribb, and now Hooper. I have simply watched from the sidelines as each in turn has held aloft his blazing but strangely unregarded torch. However, I have become, with each new revelation, and particularly with the discoveries of Hooper, which you can now read about for the first time, more and more a convert to the underlying theme. The new facts in the case still tend to be widely separated and none by itself amounts to a proof; however, taken together the steady trend and accumulation has become very impressive. At the very least the OPV theory of the origin of AIDS now merits our acute attention.

I have pondered very much about what sorts of people should be encouraged to try which sorts of tests: Hooper also in the book gives his list. There are some that could be decisive. However, the factual case was already quite strong after Pascal, and the present situation adds up to reiterating that Pascal was also right in his other theme, and that very major questions need to be asked about why supposedly 'free' science has been so slow to listen to what should have been taken very seriously from the first. If the topic had somehow been far from Big Science and had lacked any implications touching on issues like politics and professional pride, I have little doubt that its questions would have been much more discussed and investigated by now. I very much hope this book will cause the questions to be asked and the tests to be undertaken, and that it will also stimulate a lot more of the kind of sociology and science critique which Brian Martin in Australia promoted during (and supportative to) the building of the present story. How much more useful his effort is than so much that is done under the name of the sociology of science!

Forensic high-tech analysis has been enthusiastically applied to the hair of a historic corpse, Napoleon, in order to try to separate the natural events, accidents, and malfeasance that might have played a part in his death. He was a great man by any standard and also, looked at a bit more sourly, was instrumental in causing hundreds of thousands of deaths. Most would agree that these attributes of Napoleon justify the considerable interest historians have in how he died. But this level of interest makes it all the more remarkable that another historical issue with already far more deaths to its tally, and its Waterloo not

even in sight, receives currently only a single historian's effort. Vaccine vials, which are surely much more accessible than samples of Napoleon's hair, stay untested in the Wistar Institute freezers. Through turning a blind eye to the OPV/AIDS hypothesis, our establishment actively avoids testing and hearing about the plentiful though scattered evidence that the AIDS epidemic may have had a medical accident at its origin—an accident possibly compounded, more recently, by a desire by certain protagonists to conceal the evidence.

In getting together the materials for his book, Hooper has worked harder and for much longer than any of his forerunners. Several times he has countered my plea for a start on the writing by saying there just had to be this further trip to Belgium or that one to the United States. His work has amounted to more than six hundred interviews in all, he tells me, and this says nothing of the library research. I believe no one, not even a person 'speaking as a scientist,' is going to call this book 'the wildest of lay speculation'—the criticism that was leveled, even then unfairly, at Tom Curtis's much briefer accounts in *Rolling Stone*. If the OPV theory of AIDS origin comes to be proved, I think the new standards of *evolutionary* caution in medicine that their publications will eventually engender (especially regarding all treatments that use live products from other animals on humans) should merit for Hooper and Pascal jointly a Nobel Prize. As a species we ought to have known somehow in our culture, or even genes, that intimate invasions of live animal products, especially those coming from closely related species, are inherently dangerous. I have conjectured elsewhere that these dangers may be the main reason why separate species exist generally. That notion and what happens next in the present case are all in the lap of the gods. There are as stated, however, tests which can prove convincingly whether or not AIDS was our medical mistake. Meanwhile, Hooper deserves great praise for having so tenaciously carried through his investigation and for bringing to light so many more facts affecting the main question—facts that are almost all further challenges to the null hypothesis of 'coincidence only.' Even if the OPV theory is eventually rejected or remains permanently in limbo, he has done a great service in putting so many details of the early spread of AIDS on record. He has in fact given us the best history of the epidemic.

I have seen the cost the task has had for him manifested in many stages of tiredness, illness, and despair, which however he has always managed to overcome. Truly it has been like watching an explorer—Burton or Livingstone—making his halting progress toward some center of mystery that is far inland from the obvious coastal hills which we have all been seeing. Most strangely, as it may seem at first, his story wends toward exactly the same center of Africa as those Victorian explorers sought. This comes to seem a little less strange, however, once we reflect on our evolutionary origins. What dramas on all scales have been played out in the human population in the same geographic region, around the spine of Africa and in those places where the savannah and the forest meet. Almost all of these things were happening long,

long before there was anyone who could write or even speak about them. Upright we became . . . trying for new social structures, for tools, for speech, for fire . . . Finally out of Africa, our home, there came this new disease and on its heels, in this case, a *written* drama of *how* it came. Both themes are gravid with our future, and the written one is like Sherlock Holmes, Professor Challenger, Augustus Caesar, and Mark Antony all rolled into one.

Everyone should read this book, both for its story and in order to think hard on all that it implies—all this before Truth, more white and sick even than with AIDS, quietly rejoins us through another door.