

**University of Wollongong
Science and Technology Analysis Research Programme**

**What Happens When Science Goes Bad.
The Corruption of Science and the Origin of AIDS:
A Study in Spontaneous Generation**

Louis Pascal

with an introduction by Brian Martin

Working Paper No.9

December 1991

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ISBN 0 86418 199 X

Introduction to Louis Pascal's article

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In June 1990, I received a bundle of material from Richard Sylvan, a colleague at the Australian National University who is a leading philosopher and social critic. The bundle contained an article about the origin of AIDS by Louis Pascal, plus copies of correspondence concerning this article with various individuals and journals. I wrote to Pascal and so began a correspondence which has led to the publication of this Science and Technology Analysis Working Paper.

In my studies of the suppression of dissent,¹ I have come across many cases similar to Pascal's, in which an unorthodox idea is prevented from being heard, especially if it is threatening to a powerful interest group.² Pascal argues that AIDS originated from contaminated live polio vaccines used in Africa in the 1950s, an idea very threatening to immunologists and to the medical profession generally. There are a number of theories about AIDS that challenge orthodoxy.³ Of these, I find Pascal's case particularly well documented and persuasive. To my knowledge, his arguments have not been refuted.

Pascal presents his case very clearly and with many references, and he deals with a topic of the greatest social significance. But there is more than this to recommend it to those engaged in the social analysis of science.

Pascal embeds his arguments about AIDS within an argument about science. His argument about the responsibilities of scientists and editors is impassioned. Some readers may prefer a more sociologically nuanced account of "rejected knowledge,"⁴ but it should not be difficult to make use of Pascal's account for that purpose.

It is also worth reading Pascal's account of AIDS as an active agent, enlisting the support of unwary scientists and editors to pursue its deadly path. Unlike Michel Callon's description of scallops⁵ or Bruno Latour's description of door-closers,⁶ Pascal's account provides an insight that may prove to have pragmatic (rather than just theoretical) social value.

Most of all, I believe Pascal's ideas deserve a wider hearing because a free society needs a much freer dissemination and discussion of controversial ideas than present social mechanisms allow. Paul Feyerabend's ideas to this effect⁷ have had little impact on the "scientific communication system".

Perhaps it is time for social analysts of science, rather than just studying the way things are, to contribute to a changed communication pattern. Since social analysts routinely make judgements about interests associated with a position or about whether a particular perspective is worth studying — and hence make de facto judgements about claims about scientific knowledge — it is a short step to the open promotion of the consideration of particular scientific ideas.

Another way to justify this working paper is to argue that interventions into the scientific communication system provide a fruitful way to study the system. For this reason, I look forward to your response to this publication.

The text of Pascal's article is available on Macintosh computer disk on request from me.

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 - 1 Brian Martin, "The scientific straightjacket", *Ecologist*, vol. 11, no. 1, January-February 1981, pp. 33-43; Brian Martin, C. M. Ann Baker, Clyde Manwell and Cedric Pugh (eds.), *Intellectual Suppression* (Sydney: Angus & Robertson, 1986).
 - 2 Brian Martin, "Science policy: dissent and its difficulties", *Philosophy and Social Action*, vol. 12, no. 1, January-March 1986, pp. 5-23.
 - 3 For an overview of some such theories, see Robert Lederer, "Origin and spread of AIDS: is the West responsible?", *Covert Action Information Bulletin*, No. 28, Summer 1987, pp. 43-54 and No. 29, Winter 1988, pp. 52-65.
 - 4 Roy Wallis (ed.), *On the Margins of Science: The Social Construction of Rejected Knowledge* (Keele: University of Keele, 1979).
 - 5 Michel Callon, "Some elements of a sociology of translation: domestication of the scallops and the fishermen of St Brieuc Bay," in John Law (ed.), *Power, Action and Belief: A New Sociology of Knowledge?* (London: Routledge and Kegan Paul, 1986), pp. 196-233.
 - 6 Bruno Latour, "Mixing humans and nonhumans together: the sociology of a door-closer," *Social Problems*, vol. 35, no. 1, June 1988, pp. 298-310.
 - 7 Paul Feyerabend, *Science in a Free Society* (London: New Left Books, 1978).

What Happens When Science Goes Bad. The Corruption of Science and the Origin of AIDS: A Study in Spontaneous Generation

Louis Pascal*

Abstract

In this day and age, with science tackling problems of vast import such as AIDS, and building unknown life forms through recombinant DNA research, and even in the physical arena making basic changes to the world itself, such as the artificial compound freon's destruction of the ozone layer, such qualities among the scientific establishment as integrity, good judgment, competence, keeping errors to a minimum, and prompt correction of errors once made are matters of life and death, not only for millions of people but quite possibly for our modern civilization itself. Science has come up short in all these regards. Some important examples are discussed in detail.

Key Words: Science — ethical aspects; Infectious diseases — new; AIDS — origin; AIDS — transmissibility; Vaccines — contamination; Tissue culture — contamination; Africa — AIDS; Colonialism; Human nature.

Part I

In 1986 the State University of New York Press published a book by Michael Gold called *A Conspiracy of Cells: One Woman's Immortal Legacy and the Medical Scandal It Caused*.¹ It is a short (154 pages plus references and index), highly readable book which any intelligent layperson can understand. It has gotten very little attention, yet in my view it is one of the most important works of our time, a book that should be required reading for every policy-maker, every journal editor, every scientist, every person in any way concerned with judging or evaluating science, its claims, or its trustworthiness. And unless one is prepared to argue that scientists are particularly corrupt — entirely unrepresentative of the rest of society — it also has much to say about

the general trustworthiness of those human beings and institutions in whose integrity we ordinarily place a large measure of confidence.

Here is a brief synopsis: In 1951 the first human cells were grown in long-term tissue culture. These were cervical cancer cells from a woman named Henrietta Lacks, who died of the disease, but whose name, in shortened form, lives on in the designation of the culture: HeLa. HeLa cells proved to be amazingly robust. Whenever any slight slip-up in laboratory procedures allowed one or two HeLa cells to contaminate some other tissue culture, then within a matter of weeks the new cells would outgrow and completely displace the old. Since most tissue cultures look much alike, these contaminations and takeovers often passed unnoticed. This fact had large ramifications: Scientists who thought they were studying cultures of benign human prostate cells, or human breast cancer cells, or monkey heart cells, etc., etc., were in fact studying HeLa, often spending years of wasted effort. Moreover, because their cultures were growing so well, these researchers were eager to share their particularly hardy strain of "breast cancer" with their colleagues.

Within a few years the problem had reached unbelievable proportions. In 1966 the geneticist Stanley Gartler compared 17 cultures of various human cell types, obtained from a number of different laboratories, against a known HeLa strain. He found that all 17 were HeLa cells. In 1968 the American Type Culture Collection, the premiere cell bank in the United States, set up specifically to maintain pedigreed cell lines of unquestioned authenticity and to supply such to researchers all over the world, tested all its lines of human cells. Of these 34 cell lines, 24 proved to be HeLa. In 1972, in an important scientific exchange program connected with Nixon's "War on Cancer," Russian scientists supplied American scientists with six tissue cultures taken from six cancer patients from six different locations in the Soviet Union. All six turned out to be HeLa.

Tissue cultures are used as a major part of cancer research and many other areas of biology. Not only was a significant part of the world's scientific research using tissue cultures suddenly rendered invalid, but also much other work that made use of these invalid papers, or that made use of *those* invalid papers. . . . Moreover, a vast amount of further work that *may* be legitimate must be considered suspect until it is proven that neither the authors nor their important references ran afoul of HeLa.

The problem was exposed largely due to the crusading efforts of a single individual, Walter Nelson-Rees, head of a cell bank at the University of

California, and for a time vice-president of the professional organization for scientists in his field, the Tissue Culture Association. Nelson-Rees worked tirelessly to uncover and publicize cases of contamination. The reason the problem grew so large despite all his efforts is the same reason so few have ever heard of it: instead of joining forces with Nelson-Rees to rid science of this great adversary, scientists and journal editors joined forces to cover it up, thereby becoming HeLa's greatest allies. Scientists unwilling to throw away years of work refused to admit their cultures were contaminated and continued to supply samples to other researchers without a word of warning and to publish papers with no mention of the possibility of contamination. Often researchers deceived the journals. But in at least one major case, when a researcher wrote an important paper published in *Science* without warning the journal that three years earlier he had been told by the American Type Culture Collection that his cultures were probably contaminated, he was able to deflect a portion of the criticism by proving that previously, when he *had* included warnings, two separate journals had asked him to delete all mention of the possibility of contamination.² Other journals refused to publish Nelson-Rees' lists of contaminated cultures or took unconscionably long, while researchers unknowingly using the cultures wasted valuable time and HeLa continued to spread. Meanwhile, the *Journal of the National Cancer Institute* published a cooked-up case by workers previously discredited by Nelson-Rees arguing through shoddy logic buttressed by illegitimate composite photographs of chromosomes that Nelson-Rees' charges of contamination were not valid, since, they erroneously claimed, they had found a non-HeLa culture that according to Nelson-Rees' tests might have been mistakenly classed as HeLa. Nelson-Rees and the chromosome expert at the American Type Culture Collection both agreed that this culture was not in any way mistakable for HeLa. The journal was warned of the serious defects of the paper but published it anyway, and without any mention of the fact that the acknowledged world expert on HeLa identification had told them it was blatant nonsense.³ At least one major biological supplier, Microbiological Associates (which later changed its name to M.A. Bioproducts) continued to sell a HeLa-contaminated culture for 13 years after first being told by Stanley Gartler and for 7 years after half a dozen other scientists had confirmed Gartler's charge. What they described in their catalogue as "prostate, benign, human adult" was instead cancerous cervical tissue. The culture, MA160, was a best-seller.⁴

In the end, Nelson-Rees made so many enemies with his disclosures that he was effectively forced to retire in 1981 at the age of 52. Shortly thereafter, the National Cancer Institute halted funding for his laboratory, and the most

scrupulously careful cell culture facility in the country ceased to exist. During his years of work, Nelson-Rees and his laboratory had uncovered 90 contaminated cell lines, 22 of them in his last two years. According to the head of the American Type Culture Collection, this represented "about a third of the more popular cell lines used in cancer and related research".⁵

The HeLa affair was an extraordinary, worldwide scientific debacle with much greater impact on human welfare than, for instance, such well-known scientific achievements as putting a man on the moon. It is one of the major episodes of twentieth-century science, yet it has been hushed up so thoroughly that few even know of its existence. The fight against cancer, and much other scientific research, was greatly compromised, and an unknown, but surely large, number of lives will be lost as a result.

In this day and age, with biological scientists creating novel life forms, studying exotic and deadly viruses, and engaging in new and untested technologies all over the world, any tiny slip-up resulting in the escape of one of these organisms, might spark off a deadly epidemic of a new disease against which science was helpless. This could quite literally be a worldwide disaster. If scientists of the sort Gold describes had made such a slip-up, what are the chances they would immediately come forward to explain what went wrong and to warn the world so that a maximum effort could be made to contain the disaster and so that similar errors could be avoided in the future? How much greater are the chances they would fail to see the error, would ignore or attack anyone daring to point it out, would enlist the journal editors on their side, and would bury their mistake as deeply and thoroughly as they possibly could? That is what they did this time.

Part II

There were two objections raised to live oral polio vaccine when it was first developed in the 1950s:

- 1) The special strains of living polio virus that had been carefully bred to infect without producing symptoms might revert to their former virulence as they circulated from person to person once they had been introduced.⁶
- 2) The manufacturing procedure almost guaranteed contamination with foreign viruses, and these could not be killed without also killing the polio virus and ruining the vaccine.⁷

No country volunteered to be the first to test it.

There was also the problem that the Salk vaccine had already been widely used for several years, and this would make it difficult to gauge the effectiveness of the new vaccine. To get around this last, the U.N. suggested it first be tested in an underdeveloped country which had not yet begun using Salk vaccine.⁸ Such a suggestion could probably not even be made today, but at that time, in the late 1950s, the U.N. was much more heavily dominated by the developed world than at present. Africa in particular was underrepresented: as late as December 1955, the entire continent had only four U.N. members, one of these being South Africa. By the end of 1957 five more countries had been admitted, but the region of Central Africa still had no members, because not a single Central African country had yet attained its independence.⁹ Thus when Belgium volunteered its Central African territories of Ruanda-Urundi and the Belgian Congo for the first test, everyone was happy. I suspect even the Africans were happy, since I suspect they knew nothing of these reservations.

In any event, in late 1957 in the eastern part of the Belgian Congo, and especially in early 1958 in Ruanda-Urundi, the world's first mass immunization campaign using live polio vaccine was carried out.¹⁰ A few months later the very same batch of vaccine was used again in Leopoldville, capital of the Belgian Congo; 900 miles west of the first campaign.¹¹ These are today the independent nations of Rwanda, Burundi, and Zaire; and Leopoldville is now known as Kinshasa.

The problem about reversion to virulence never materialized, but almost immediately contaminating viruses started emerging. Shortly after it was used in these two campaigns, Albert Sabin found an unidentified virus contaminating this first batch of vaccine.¹² In the early 1960s, the virus SV-40 was found to have contaminated a great many vaccine batches and to have infected millions of people.¹³ And there were several others also. Hilary Koprowski, the man who had made the batch of vaccine used in these two African campaigns, wrote that "If, indeed, somebody were to poke his nose into the live virus vaccine, he might find a non-polio virus in all the preparations currently available." He argued that this presented no real problem, however, because people were orally exposed to many viruses every day in their food.¹⁴

Koprowski's response was inadequate on three counts:

1) The vaccine as manufactured *could not* be made free of contaminating viruses. Therefore he either had to abandon his vaccine and renounce years of labor or else pronounce these contaminants harmless. He was hardly a disinterested party.

2) The expected contaminating viruses were monkey viruses, from the monkey kidneys used to grow the special strains of polio virus that constituted the vaccine. And humans are *not* daily exposed to monkey viruses. Even in those countries where monkeys are eaten, they are (I presume) cooked first. And *never* would raw monkey meat be fed to infants less than 30 days old, as occurred with this first batch of vaccine, while other batches were fed even to 48-hour-old infants.¹⁵ It is often difficult to transfer a virus from one species to another, and when scientists attempt to do so in the laboratory they employ a number of means to help the virus survive in its new host, such as using drugs or radiation to suppress the animal's immune system. One of the most important of these means is the use of infant animals, because their immature immune systems already present much less of an obstacle to a foreign virus. Even so, the first try, or the first several tries, often does not work, so that many animals are used in hopes one will prove susceptible. Once this first infection is accomplished the virus rapidly adapts to its new environment, and subsequent baby animals can be infected much more easily. Indeed, it may then be possible to infect adult animals with the adapted virus.¹⁶ If one were *attempting* to start a human epidemic of an animal disease, one could scarcely do any better than feeding multiple unknown viruses from our closest biological relatives to many millions of infants around the world.

3) New viruses starting in "virgin" populations never before exposed to them are often particularly virulent and may also be particularly contagious. Simian hemorrhagic fever infects one of the monkeys used in making polio vaccine without causing any illness. But when it is accidentally transferred into a single member of a rhesus monkey colony, it quickly infects and kills every monkey in the colony.¹⁷ Fortunately, humans seem not to be susceptible to this particular virus. But a disease much less deadly and much less contagious than this one could still easily kill hundreds of millions of human beings. Thus the biggest objection to Koprowski's defense of his vaccine was that it did not include the statement, "I realize that if I am wrong, hundreds of millions may die as a result of my error, but I have taken this into account and I still believe the risk is too slight to justify abandonment of the vaccine." He did not take the potential consequences of his being wrong into account. On arguments that a mere schoolchild could see were no more than wishful

thinking he risked hundreds of millions of lives, and was never even aware of it.

But his arguments *were* wrong. It was completely predictable that monkey viruses would get started in a new species never exposed to them before. And it was almost completely predictable that not all of them would be harmless. And now this almost completely predictable disaster has occurred. In fact it occurred right off the bat. This very first batch of vaccine gave us AIDS.

###

In 1985 the first of the simian immunodeficiency viruses was discovered in rhesus monkeys, one of the three main species used to make the polio vaccine. Since then, SIVs have been found in the other two species — cynomolgus and African green monkeys — also.¹⁸ All of the SIVs are closely related to each other and are the closest known relatives of AIDS. In fact the conclusion was immediately drawn that transfer from a monkey to a human of one of these viruses had caused AIDS. And how was it transferred? Why through a monkey bite, of course!¹⁹

This ignores the precedent of SV-40 and other monkey viruses having been transferred from monkeys into humans through polio vaccine. It ignores the fact that the manufacturing process (discussed later) would clearly have resulted in SIV contamination of the vaccine. It ignores the fact that Rwanda, Burundi, and Zaire — the site of the first campaign — have three of the world's highest incidences of AIDS, and all three are in Central Africa where epidemiologists had concluded AIDS had most probably begun.²⁰ It ignores the fact that Kinshasa, 900 miles west of the area of the first campaign and given the same batch of vaccine contaminated with an unidentified virus, is today one of the world's worst-struck cities. It ignores the fact that the earliest-known definitely HIV-positive blood sample yet found anywhere in the world was taken in Kinshasa. The Kinshasa vaccination campaign began in August 1958. The blood sample was taken in 1959.²¹ At that time Kinshasa was only a medium-sized city of 340,000 — just over one hundredth of one percent of the world population in 1958 — and the coincidence in time and place is, to say the least, extraordinary. It also ignores the fact that those opting for a monkey bite are hardly disinterested parties. And most important of all, it ignores the fact that if they are wrong, and monkey diseases such as AIDS are indeed getting into the human population through contaminated vaccines, then other new diseases are likely to get started in the same way in the future,

and hundreds of millions of additional lives will be risked. Nowhere do they say, "I realize that if I am wrong, hundreds of millions of people may die as a result of my error." Nowhere do they show any inkling of grasping the importance of this question.

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On 31 May 1987 Eva Lee Snead, a San Antonio physician, announced the oral polio vaccine contamination theory, a theory she had formulated on her own. She made this announcement on "Natural Living with Gary Null," a (now defunct) radio program broadcast on WABC in New York City featuring far-out ideas, some of which were very good but others very bad.²² And indeed, Dr. Snead's own views belonged somewhat in this mixed category, since she correctly stated that African green monkeys were heavily infected with SIV; that oral polio vaccine had been made from them; and that the monkey virus SV-40 had contaminated many batches of this vaccine. She then unaccountably went on to argue that not SIV but SV-40 was the most likely ancestor of AIDS. *Perhaps* this lapse can excuse the medical establishment's ignoring of her views. But I, who happened to be listening to the broadcast, thought her claims about African green monkeys having been used to make the vaccine, and about other monkey viruses having contaminated numerous vaccine batches, were straightforward factual claims which I could prove or disprove easily enough, and that they were extremely important if true.

It did not take long to verify these claims. The SV-40 incident is well known.²³ And the principal early article tying SIV to green monkeys, published in *Science* on 22 November 1985, contains this line: "Much of the oral poliovaccine (OPV) used throughout the world is produced on primary cultures of kidney cells from this species [African green monkey]".²⁴ If Dr. Snead's presentation was too flawed or her forum too suspect, then why did not this bald statement of fact by prominent researchers in America's most prominent scientific journal prompt the entire world in November 1985 to raise the questions she alone raised in May 1987? And if the scientific world was too self-interested or self-complacent, then why did not the reporters raise these questions?

###

My own chief contribution to this research came when I looked into the history of early polio vaccination. It was I who discovered the completely unexpected location of the first campaign, the contamination of that batch, and the fact that the same batch had been used in Kinshasa in the year before the earliest

AIDS-positive blood sample was taken there.²⁵ And indeed I found much else. But that was the smoking gun, the evidence that was too much to be ignored, too striking to permit any further stonewalling. Or so I thought.

I wrote a paper carefully laying out all the evidence so far presented here, and more, and in greater detail, all documented with references to the medical journals, not one of which was in any sense a "fringe publication." This piece was completed at the end of November 1987. The first copies went out during the first week in December. I wrote to six biologists, seven AIDS researchers, and several miscellaneous others. In the first two years I received only one reply from these sources, that being a perfunctory acknowledgement written on behalf of Luc Montagnier by a colleague.

I submitted it to three scientific publications, who rejected it, and then to two multidisciplinary publications, who advised me that it belonged in a scientific journal. One of these forwarded it to Oslo University's Center for Medical Ethics, who replied, ten months later, that they were a new institution and too busy with start-up duties, but perhaps in the future, if they became interested in such questions. Of the three scientific publications, *Lancet* gave no reason for its rejection. *Nature* replied cryptically that while the theory "cannot be ruled out, it does not seem readily to fit the epidemiology of AIDS." *New Scientist* did not reply to my submission of 8 August 1988, nor to follow-up letters of 25 October 1988 and 17 May 1990, both of which contained another copy of the original submission. *New Scientist* did, however, in its 30 June 1990 issue dismiss the polio vaccine contamination theory with these words: "There are no grounds, epidemiological or biological, for believing that this has happened".²⁶

I got a better reception when I wrote to philosophers whose work had appeared together with mine in an anthology.²⁷ Most of them responded, and all who did offered suggestions and encouragement. R.M. Hare sent my work to the *Journal of Medical Ethics* in August 1988. Its editor replied that my material was inappropriate for his journal; however he would be interested in a 3500-word treatment outlining my polio arguments but concentrating on the ethics of the editors' rejections. An unfortunate postal mix-up prevented my learning of this interest for 18 months, and when I finally did learn, in April 1990, I found myself unable to write the piece the editor had requested. I spent enormous effort composing attempt after attempt. I finally gave up and produced the distinctly different current version — the one you are reading now — completing it 17 April 1991. (It has been updated through 18 November 1991, but the only significant change, beyond this paragraph and

the next four, is the addition to the appendix of the objection about epicenters.) I got together a large packet of documentation, correspondence, rejection letters, etc. and submitted it all on 3 May 1991, asking the editor to consider it to be an independent submission if it differed too greatly from the piece requested. It had required eleven months of full-time work, while the world waited and AIDS continued to spread.

On 27 May 1991 the editor, Raanan Gillon, rejected it. He wrote: "You have a potentially very important thesis, the truth of which I simply am not knowledgeable enough to assess but certainly *prima facie* it seems a highly plausible thesis (though somewhat marred by the 1959 case [of a British sailor allegedly dying of AIDS; see appendix]; but there again you have counter-arguments concerning such cases too). . . . There is just no way that I can publish a 19,000 word paper (even if I thought that it was going to save *millions* of lives as you suggest (and I have to say that I remain unconvinced by this speculation) [emphasis his]."

My piece would have required 20 pages of his publication. The original piece sent to him in 1988 would have required ten pages. He has withheld extraordinarily vital information from the world for three years now, waiting for a version more to his liking. In March 1990 he had published a 14-page article on AIDS. Though he refused to read what I had sent him (he said he had "skimmed through" it) or to have it refereed, he asked me to write yet a third version. I repeated that I was unable to produce a piece meeting his specifications and said that I would submit the current piece elsewhere.

Meanwhile, in November 1989 the writer Alice Walker had sent my work to *African Commentary*, a promising new black periodical having among its columnists Pulitzer Prize winners Toni Morrison and Gloria Naylor, as well as South African writer Nadine Gordimer, recently awarded a Nobel Prize. In March 1990 *African Commentary* asked me to write a shorter, simpler version for them. I submitted the result of that effort 15 May 1990. On 28 August 1990 *African Commentary* accepted the piece, but then ceased publication before it could be brought out.

Alice Walker has recently sent my work to another black publication.

The bottom line is that today, 18 November 1991, it is six years since those quintessentially suggestive words appeared in *Science*, four and a half years since Eva Lee Snead's forthright claim of polio vaccine's causality, and four years since my 20 November 1987 paper proving her claim. Yet still these facts lie buried. And the argument is as simple as one could possibly want.

And the evidence is abundant and unequivocal. And no one has yet pointed out an error in the case. And the question is one of life-and-death importance for untold numbers of people.

#

How big are these untold numbers? I keep throwing out figures like "hundreds of millions." Can I support this claim? In fact it is easy to make an estimate of the numbers involved which, while very rough, is still more than sufficient to determine the probable *scale* of the disaster ahead.

In order to estimate the destruction of a small but growing fire, when there is nothing at hand to put it out, one looks not at the size of the fire but at the amount of the fuel. If 3 percent of the world's male population are actively homosexual, quite a conservative estimate, then there are roughly 75 million currently alive in this risk group. At most one-third of them are already past the age at which AIDS is likely to be acquired. That leaves at least 50 million at high risk. In America and Europe, it is unlikely the numbers dying will be much below half of this risk group, but my knowledge of homosexual lifestyles in the rest of the world is too meager for me to make a realistic estimate. However, I doubt it will be less than 10 percent overall, especially when I have not added in the smaller proportion of the larger numbers of bisexual or occasionally homosexual men. Tentatively I conclude a minimum of 5 million homosexual men currently alive will die of AIDS. Since there are already a substantial fraction of a million infected gays in the U.S. alone, I suspect this figure is extremely conservative. In any case, it does not matter, because homosexual deaths will be insignificant compared to heterosexual cases, certainly in the Third World, and quite probably in the developed world also.

Uganda, which borders both Rwanda and eastern Zaire, has an extremely high rate of HIV infection. According to the *New York Times*, as of 1988 there were 790,522 Ugandans who had tested positive, representing about one in eight adults.²⁸ But what is not explained is that this is a "snapshot" of the entire adult population, many of whom are still too young to have undergone more than a small part of their risk exposure, many of whom had already passed through the prime risk ages before AIDS emerged, and all of whom, no matter how old, will continue to experience some risk in the future. We are not interested in a cross-section of the entire population, nor of the entire adult population, nor even the incidence in the single highest-risk age group. We want the cumulative risk across an entire lifetime, and that is a figure bigger

than any of these. Less than 3¹/₂ percent of the adult population of the U.S. *currently* has had cancer, yet we know that 30 percent will get it before they die.²⁹ Consequently, the fraction of today's newborn Ugandans who will eventually become infected is much higher than one-eighth. I think one-third is reasonably conservative, especially since there is no indication (so far as I have seen) that the AIDS incidence in any given age group has yet stopped increasing.

A recent article in *New Scientist* shows that a number of other Central African countries have infection rates comparable to Uganda's, and even Zimbabwe, to the south, is rapidly becoming infected, with 15 percent of adult blood donors now testing positive, at least in urban areas. Indeed, the infection rate has already reached one-third in several major African cities, such as Zambia's capital of Lusaka, where 32 percent of adults are currently infected.³⁰ And the *Washington Post* reported 25 percent of citizens — they do not even specify adults — of Rwanda's capital of Kigali were already infected by the end of 1986.³¹ The total population, estimated to mid-1991, of ten African countries where AIDS is now striking most heavily (Burundi, Kenya, Malawi, Mozambique, Rwanda, Tanzania, Uganda, Zaire, Zambia, and Zimbabwe) is 166 million (and rapidly growing), and one-third of this comes to roughly 55 million lives. *But these countries all border one another.* If we assume this is the only reason it is striking here so heavily — i.e., that this is where it began, that this is the current border of an advancing fire — then we must add in much larger numbers. The total African population is 677 million, but Northern Africa is largely Muslim and the disease will affect it differently. Even entirely eliminating Northern Africa still leaves over half a billion Africans where AIDS may be expected to strike heavily. I expect 100 million or more deaths from those currently alive in Africa alone.

And AIDS is rapidly spreading in Brazil and other South American countries and is beginning to invade Asia. AIDS may well claim several hundred million lives among those in the Third World. I expect it to wreak very considerable havoc among the developed countries also. But since it is not yet clear how heavily AIDS will infect the heterosexual population in the developed countries (I expect very heavily, and can give reasons, but they are still somewhat tentative), I shall not attempt to estimate these numbers. Since the underdeveloped world is already nearly four times the size of the developed world, these additional deaths will not greatly influence the overall total, at least not directly.³²

Thus with very simple arguments, I have already reached several hundred million expected deaths. And I have not added in the additional deaths to be expected in coming generations, which could multiply the total many times over.

It is true that I have not taken account of efforts people will make to avoid infection. But I have also not taken account of the fact that AIDS will become more infectious as time passes and it becomes better adapted to its new environment, the human race. (I will briefly discuss this later.) It is my view that the latter point is likely to significantly outweigh the former. Nor have I taken account of the possibility of a successful vaccine or cure. But just as AIDS is becoming more transmissible, so also is it becoming less amenable to vaccine or cure, as the thousands of individual strains diverge ever further apart.³³ I seriously doubt a dozen vaccines would suffice to cover even the diversity that exists today, and in ten or twenty years we might well need hundreds. Since no lentivirus (such as AIDS is) existing in animals has ever had a successful vaccine or treatment (and in the case of sheep and horse viruses these efforts go back several decades and were not attended by the very major problems of testing in humans), and since many medical practices are serving not to reduce cases but increase them,³⁴ I am discounting any contribution of medicine to this estimate. I surely believe the search for a vaccine or cure is worth spending many times more on than we are currently doing, but this is not because I think positive results are likely, but because the situation is so desperate that it is worth grasping at straws for, even when those straws are very expensive ones.

It is useful to compare this estimate of several hundred million deaths, perhaps continuing generation after generation, with the death toll of World War II. Counting all deaths, military and civilian, on both sides, and including such indirect causes as starvation and disease, about 50 million people lost their lives in World War II.³⁵ Unless there is a very dramatic medical breakthrough very soon, AIDS will surpass this total many times over. Unless there is a very dramatic medical breakthrough very soon, AIDS will be *by far* the worst single blunder yet made by the human race. It is questionable whether even a nuclear war would kill as many.

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There are many different varieties of SIV infecting the various monkey species, but only three of these monkeys have regularly been used in vaccine production (though a number of other species have been used on a limited or

experimental basis). However, it is entirely possible for many different SIVs to exist within a single monkey species. Within our own species, there are three separate varieties of polio, requiring three separate vaccines, and dozens of other known viruses in the same genus as polio, plus an unknown number of still-undiscovered close relations. Our methods of detection of SIVs are very crude. They would have missed all or nearly all the rare varieties, and would probably have missed even very common varieties if those were antigenically distinct enough not to react to the tests for the known members of the family.³⁶

HIV-2 is an entirely separate form of AIDS started by an entirely different SIV. It was very probably started by a similar contamination incident, but I have not attempted to pin it down. Using the rate of divergence of various strains of HIV-1 and of HIV-2, and projecting backwards, Sharp and Li estimated HIV-1 began in Central Africa a little before 1960 and HIV-2 in West Africa about the same time.³⁷ Their estimate for HIV-1 was stunningly accurate. If they were similarly accurate for HIV-2, then it means both major forms of AIDS began in the first few years of oral polio vaccine use and both began in Sub-Saharan Africa. Those are very ominous findings if correct.

Judging by AIDS' rate and manner of spread, Sub-Saharan Africa is one of the most fertile soils anywhere in the world for starting an epidemic of AIDS. Even so, it required from 1957-8 until 1981 before HIV-1 had infected enough victims to be noticed. HIV-2 was not discovered until 1985. It would be expected that those SIV varieties starting just as early but in significantly less fertile soil, or in any soils at all after about 1975, would still not have spread widely enough to be detected. We have seen only those forms of AIDS that began at or near the earliest possible date and in or close to the best possible soil. This is apt to be only a small fraction of the total number of SIV varieties that have already been transferred.

While obviously very rough, it is nevertheless my best guess that new varieties of AIDS have been entering the race at a rate of one every year or two or three. Each of these varieties will almost surely require a separate vaccine; and even if science manages to develop a routinized procedure which works in every case, by the time the virus has spread widely enough to be detected, and the vaccine produced and distributed, it is likely the numbers infected will exceed one million. This is the most optimistic scenario. I believe a significantly more likely scenario is that no vaccine will ever be developed against any variety of AIDS.

Eventually the SIV varieties will run out — and we may *hope* they ran out long ago³⁸ — but I believe there is at least a 50 percent chance that one or more new AIDS varieties have arrived during the four-year delay caused by the various editors' rejections of my work and the various AIDS researchers' ignoring of it. The resulting suffering and death would likely never have occurred if any single one of them had recognized the importance of the information I sent them and had acted to make it known. In addition, I have come across several other serious diseases unrelated to AIDS that are good candidates for having been started through contaminated vaccines.³⁹ And there are an unknown number of others that I may have overlooked, or that have not been around long enough to have infected detectable numbers of people.

Part III

The circumstances of its origin are not the only important facts about AIDS that have been withheld by researchers and editors. In 1986 I wrote an account of how AIDS' transmissibility would increase as it became better adapted to its new environment, the human race. I shall not repeat the arguments here, but the central point is so obvious that it can be stated in a single sentence: of the thousands of slightly different strains of AIDS that now exist, and of the tens of thousands that will exist in the future, those that are most transmissible will be the very ones that spread most rapidly, while the least transmissible strains will be the very ones that die out, and both these factors independently will act to increase the transmissibility of the average strain as time passes. After further arguments, I concluded that AIDS' transmissibility would *certainly* increase, that this increase would almost certainly be significant, and that it would most probably be highly significant or dramatic. Although I specified no time scale, I think some manifestations should be detectable already.

Nature and *Lancet* both rejected a 1300-word version of this piece in 1986, and *New Scientist* followed in early 1987. Additionally, *Nature* rejected a greatly lengthened and more intricately argued version in mid-1987. There is not any way that my claims for a transmissibility increase could be wrong (though the magnitude is open to dispute). Nor did any of these publications offer any arguments to suggest that I was wrong. Nor did they offer any arguments as to why this was unimportant news if it was correct. They did not justify their decision to withhold this information from the world; they simply withheld it. And so it has been withheld for more than five years now. If I am

correct about a significant increase in AIDS' transmissibility, then this is a fact entirely comparable in its importance to the facts of AIDS' origin. It means AIDS is a very much worse disease than we have been led to believe. If this is true, and the fact were acknowledged, we would pull out all stops in combatting AIDS. We have not even begun to do this yet. Many millions of lives could have been saved that are now no longer saveable.

Part IV

It is important to try to grasp the magnitude of the failure that has occurred. This is no easy thing. First of all, the question of AIDS' transfer through medical practices should have been vigorously pursued as soon as it became apparent that AIDS' probable ancestor had existed in the monkeys that were used to produce vaccines. If there is an animal disease that has the potential to become a human disease (as it is only too clear that AIDS' ancestor had!), and this disease occurs in a species used to produce vaccines given to many hundreds of millions of people around the world for many years, then even if scientists had been well aware of this disease and its danger, and had had an excellent test available, and had all along been taking the greatest possible precautions to prevent its entry into our species, even so it is clear that as time passes and the vaccine batches mount into the thousands and tens of thousands, almost certainly someone is eventually going to get careless, or a test is going to give a false negative result, and a contaminated batch is going to get through. No system is perfect; and a system that requires perfection in order to avoid disaster is in fact little more than a recipe for disaster. Regardless of the precautions scientists might have been taking, it simply goes without saying that when, two decades after embarking on such a procedure, the monkey disease *is* found to have crossed over into our species, the first place one should then look is to the procedure, and not to a monkey bite. If under these circumstances six years had been allowed to pass after discovery of this worldwide disaster before finally turning to the obvious, the failure would have been monumental.

But in fact, of course, the vaccine researchers had *no* knowledge of SIV and *no* means of testing for it until four years after AIDS had been discovered and 28 years after beginning mass vaccination using these monkeys. *Unbelievable as it may seem, the oral polio vaccine consisted of nothing more than the culture fluid from the polio-inoculated monkey kidney cell cultures after passage through a filter small enough to remove bacteria but large*

enough to permit passage of the polio virus, and whatever other viruses may have been infecting the monkeys before they were killed. This filtered culture fluid constituted the finished vaccine fed to hundreds of millions of people around the world.⁴⁰ There were *no* methods used to prevent those viruses already present in the monkey kidneys from contaminating the vaccine and *no* methods used to kill the viruses after contamination. The scientists' only hope of avoiding contamination lay in eliminating all the sick monkeys they could find beforehand and all the contaminated batches they could find afterwards. The former approach would have missed all those monkeys still in the incubation period (not even to mention the fact that most SIVs do not seem to cause any illness at all in those species they infect naturally, while being fatal to species which have no previous experience with them).

The latter method was even less useful. Retroviruses are very difficult to detect, especially using the crude tests available in the earlier years of vaccine use, before reverse transcriptase was even known to exist. It is hardly surprising that no variety of SIV was among the more than 75 previously-unknown monkey viruses that were found through polio vaccine testing, despite the fact that substantial proportions of African green monkeys were infected.⁴¹ And with these methods of manufacture it is clear that contamination would be common. Indeed, the most likely outcome is for contamination to occur every time, or virtually every time, an SIV-infected monkey is used.⁴² The researchers' tests for contamination did not catch 100 percent of SIV-contaminated batches. They did not catch a grossly-unacceptable 99.9 percent. They caught zero percent.

Practically every AIDS researcher around the world who was remotely interested in AIDS' origin would have read the 22 November 1985 *Science* article tying AIDS to SIV, tying SIV to green monkeys, and tying green monkeys to polio vaccination.²⁴ Why did the article's authors not pursue this obvious important lead? Why did no single one of the thousands of AIDS researchers who would have read the article pursue it? Everyone in any way connected with or knowledgeable about vaccine research was additionally aware of the huge potential for contamination, and of the actual contamination of many batches of several different vaccines with the monkey virus SV-40, and several other viruses. This group managed to produce only a single letter-to-the-editor, four years later, suggesting the role of oral polio vaccine in the origin of HIV should be investigated.⁴³ Finally, those who worked on the early live polio vaccine knew in addition that its original test site was not in a world center of medical technology, as anyone would have expected, but in the middle of Africa. And I dare say that there is not one among the many

individuals who knew that fact who did not also know this was the location of AIDS' greatest ravages and the location many had suggested for its origin. By late 1985 this was common knowledge even among the general public. Yet no single polio vaccine researcher pursued the question either.

The monumental failures so far described apply to the entire fields of AIDS research and vaccine research. In addition, lesser degrees of failure attach to all related fields in diminishing proportion to their distance. The reflection on all the life sciences is very grave. How could they have allowed their colleagues to be so irresponsible about such important matters for all these years? Indeed, the reflection reaches much further. These were not abstruse matters only a specialist could understand. Not only scientists from all fields but reporters and the general public have followed the AIDS issue with intense interest. Why did no physicist, no geologist, no newspaper or television reporter who read the *Science* article go to an AIDS researcher and ask the simple question: "How do you know AIDS did not come from the monkeys through your vaccines?" There is no possible remotely satisfactory answer to this question.

But of course it is much worse even than this. Obvious as the lead may have been, and straightforward as the research required, there is still a vast difference amounting to several orders of magnitude between researching a topic oneself and merely understanding the results of someone else's finished and clearly presented research. I spent weeks sifting through hundreds of articles and distilling them down to a picture so clear no one could have failed to understand it. I carefully cited every reference, listing in addition to the usual information, the particular *relevant* page or pages of each article so as to further simplify the process of verification. The research the scientists should have done themselves a long time ago, I did for them. I put it all together and handed it to them on a silver platter. They had nothing to do but check it out, using the references I had supplied, references from their own medical journals. Even this was beyond them. And it is not a matter of a single editor or scientist being particularly stupid or particularly irresponsible. It happened over and over. Unless one is prepared to argue that those journals and researchers I sent my work to were a few rotten apples entirely unrepresentative of science as a whole, one must reach the conclusion that people of this calibre typify science. That is certainly my conclusion. And anyone taking the other side will have a very hard time explaining how all the good and responsible scientists who never had the benefit of seeing my work managed to miss finding so obvious and important a point themselves for all these years.

We take the kidneys from great numbers of SIV-infected monkeys, add a little polio virus, grow whatever will grow for several days, filter the solution, and feed it to hundreds of millions of children around the world. Then, a quarter century later, when we discover SIV now infects humans too, we say, "What could have happened? It must have been a monkey bite."

Part V

I will leave it to others to discuss the ethics of using a subject people as guinea pigs when testing a vaccine whose safety is in question, and what should and could be done to redress an error now made that will almost certainly destroy a number of countries and kill more Africans than died in 300 years of Western slavery. I will leave it to others to discuss the ethics of the early vaccine researchers in ignoring commonsense safety precautions and dismissing obvious objections in their zeal to combat polio and earn their place in history. It is hard to know, looking back on it now, and in full knowledge of what did happen, how harshly they should be judged. But a great error was made, and if these people are even in part excused on the ground that they could not have been expected to see with the clarity of hindsight, then blame must instead attach to the obscurity and unforeseeableness of the dangers of twentieth-century science itself, and the advisability of any further travel along a road so irredeemably hazardous should be seriously questioned. I will leave it to others to discuss the, in my view, completely inexcusable actions of these same polio researchers in not coming forward six years ago when they first began to worry — they would have *had* to begin to worry — that their vaccine might have been responsible for AIDS. It would have been an easy enough thing for any one of them to prove what had happened, but every one of them failed to investigate the question. What possible reason could there be for not looking into so important a matter so close to their central concerns, except that they were afraid of what they would find? I will leave it to others to discuss the ethics of the various AIDS researchers and other scientists who ignored the information I sent them and risked millions of lives on their unexamined opinion that I was wrong, when I was not. Any one of them could have brought this information to public attention long ago. Every one of them failed to do so. I will concentrate here specifically on the ethics of the various editors who used their positions to withhold this information from the world.

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The editors of the world's learned journals are the gatekeepers of knowledge. Their decisions determine what becomes known and what remains unknown. Indeed, their decisions determine even what can be debated. Society is dependent on the efficient performance of their jobs for one of its most basic and vital commodities: information. In a world such as the modern one, where decisions made by political leaders or political bodies determine basic facts of existence for countless millions now alive and still to be born, and where scientific errors have the potential, already partially realized, to bring about worldwide holocaust, then incorrect knowledge presents a threat of enormous magnitude. The editors of the world's learned journals are at the interface between knowledge and society. Their power is enormous. Their responsibility is enormous. How can things have gone so terribly wrong?

It is my strong view that these editors are entirely culpable. There is nothing comparable to the hindsight excuse of the vaccine workers. These people *had* the benefit of hindsight, but it did them no good. There is much ancillary blame that can be placed on the system itself, which exerts a tremendously powerful force for conformity. The system crushes those like Nelson-Rees who dare to speak unpopular truths. But the system exerts its force and achieves its censorship of dissident views through the actions of the individual human beings who make it up. Each individual editor was faced with the physical fact of a manuscript making claims that were clearly matters of life-and-death urgency for vast numbers of people *if they were true*. It was each editor's job to decide this question, making completely certain the claims were false before rejecting the manuscript. On matters of such grave importance, one does not have a right to be mistaken. One proves one's case beyond a shadow of a doubt, aware that a 10 percent or a 1 percent chance of error means a 10 percent or a 1 percent chance of the loss of millions of lives. If one tries one's best and cannot prove it wrong, then one has no choice but to print it anyway, perhaps with an editorial comment to the effect that the publication does not stand behind the author's claims but that they are far too important to be dismissed until they have been decisively refuted. Surely a 10 percent or a 1 percent chance that AIDS had come through vaccines via a process that would almost certainly lead to still other new diseases demands the most prompt and careful investigation. It demands precisely the opposite of being buried as deeply and thoroughly as possible.

And yet I find it inconceivable that even my harshest critics, yelling as loudly as they may care to that my case is still unproven, will be able to examine the evidence I have provided and show the likelihood to be even so low as 90

percent. In my own opinion the case is proven far beyond 99 percent, far beyond such commonly-accepted facts about AIDS as its non-transmissibility through casual contact (a claim that may well be true, but for which glaring holes in the evidence exist that are easily sufficient to reduce its likelihood below 90 percent⁴⁴). AIDS' origin is in fact better proven than almost any other important claim about the disease, except for the identity of its cause, HIV.

Against this mass of evidence, the editors did not raise a single concrete objection. They did not question a single point of fact or of reasoning. Yet they rejected it anyway, thereby sending who-knows-how-many present and future people to a horrible and pointless death. It was clear this would be the result, and to make certain there was no mistake they were clearly told this would be the result. Yet they did it anyway. They did it anyway despite being unable to point to a single error. I can see no conceivable excuse that can be made for them. If there is anything that they can say in their own defense, I would like to hear it.

Editors seem to be under the impression that they have an absolute right to reject anything they like regardless of the consequences. When those consequences include millions of deaths, and to individuals in many countries beyond the editor's own, and continuing indefinitely into the future, then I should think society would have a thing or two to say about that.

I have some specific recommendations for the minimal form these societal interventions should take. I will discuss them at length elsewhere. To broach this topic now would take us too far afield, and the recommendations will be given much greater weight if I wait until my claims have been investigated and confirmed.

There are some ferociously dangerous microbes infecting the world's fauna, and society has an overwhelming interest in preventing their transfer into our species. The various barriers society has erected to keep them out have proved themselves woefully inadequate in the face of the various bridges science is constructing that conduct them in.⁴⁵ Much more must be done. There are in the world a great many scoundrels, fools, and incompetents, and society has an overwhelming interest in keeping them out of positions of vast power where their errors could kill millions. Current barriers have again proved woefully inadequate in the face of a system of science that is promoting precisely these people into precisely these positions. Much more

must be done. Solving the latter problem would go far towards solving the former.

Part VI

No one should have been surprised by the response of the scientific community to the information that it had started AIDS. When large mistakes are made in any field, they are almost always covered up. It is entirely predictable. Indeed, it is very much like a prediction I did make, in 1986, in another unpublished paper on the maladaptedness of the world system, with AIDS as a particular case study: "How many of the cigarette companies have admitted their product causes lung cancer? At least in the U.S. the answer is zero. Is there any reason to believe that cigarette companies are atypically evil? Isn't this how we should expect *any* company to react in similar circumstances? If a few years down the line it should turn out that microwave ovens, say, are an even deadlier cause of cancer, what are the odds the manufacturers will meekly say 'We didn't know' and remove their product from the market? How much more likely is it that they will mount a fierce public relations campaign disputing the evidence, however indisputable?" This was written a year before I knew anything about the manner of AIDS' origin.

It is a sad and sobering fact to realize that not only the average human being but nearly every human being, if faced with a choice between risking their job and risking millions of lives, will unerringly choose the latter. This has been proven over and over again in AIDS research. I am not saying they will do this consciously. They will find a way to rationalize their decision. Or they will find a way to avoid perceiving that this is the decision that they have made. Or they will decide the risk to the millions is too slight to be taken into consideration, and will avoid looking at the question too closely lest they be forced to change their minds. But however they manage to do it, the bottom line is that the jobs will be protected and the lives will be risked.

Human beings have a positively astounding ability to rationalize whatever it is they strongly want to do, in order to make it appear to be entirely moral and just. And humans have a similarly astounding ability to rationalize whatever it is they strongly want to believe, in order to make it appear entirely reasonable and logical. This is as true of the intellectual elite as it is of the average person. Indeed, it requires great cleverness to manage to avoid the more obvious facts, and often the most gifted among us are those who lead the way.

Science has failed abysmally to take adequate account of this human weakness, a weakness that caused vaccine researchers to ignore clear signals of the catastrophic dangers of their procedure, that caused their failure to acknowledge the catastrophe after it had occurred, that caused the failure of editors and others to believe my claims about the increase of AIDS' transmissibility, and that caused their failure even cursorily to investigate the evidence of AIDS' origin. A science which ignores all evidence in order to believe what it prefers to believe is a science not worthy of the name. And now we see the results of this science in name only: a number of deaths very likely comparable to or greater than that to be expected from a full-scale nuclear war.

The philosopher Robert Nozick writes: "I do not recall any philosopher reporting in distress that on some fundamental question he is forced to conclude that the truth is awful, worse than the third best way he would want it. . . . We may wonder whether a philosophy with a foregone conclusion can have any value at all".⁴⁶ In the case of modern biology, with the power to unleash unimaginable destructiveness, the value of a science with a foregone conclusion can be a great deal less than nothing at all.

This tendency to believe whatever is most comfortable overshadows all of AIDS research and colors the final product in many ways. Investigative avenues that threaten to lead to dire conclusions are simply not pursued, however basic and important they may be. This biased selection of topics is then biased further as each researcher unconsciously emphasizes the optimistic. Editors then select among these results. Papers reaching hopeful conclusions are printed despite awesome errors; papers reaching the most pessimistic conclusions are rejected despite overwhelming evidence. This first stage of published bias is then sent back through the system and magnified: those who read these papers ignore or manage to find fault with or simply forget their darker aspects while accepting and remembering the more hopeful parts. When they write papers of their own, based on this heavily biased sampling, they bias the results even further towards the optimistic. Those with the clearest view are forced to be their own censors in order to get their work published. The end result bears little resemblance to reality. The reality is much worse.

The tendency to believe whatever is most comfortable is of course not limited to AIDS research or to science. There are many other examples from many different fields of endeavor. And by the very nature of the problem, they

disproportionately involve matters of grave concern. I do not have time to give examples here. Again, by the very nature of the problem, extensive arguments are required in order to *force* people to accept such unpleasant conclusions as this one against their will. I will merely quote two more brief passages, from letters to two correspondents, written not long after I began my AIDS research. From a 15 February 1987 letter to Peter Singer: "There are great obvious holes in many disciplines where all the terrible things have been dumped together and ignored." From a 24 May 1987 letter to Robert M. May referring to the same paper from which I took the cigarette quotation: "My long unpublished paper took the position that societal failure was more important in making this such a terrible disease than the admittedly frightening properties of the disease itself. The new information I have . . . makes the disease even worse than I had realized, so I am not sure I would still hold to that position. On the other hand, society appears to be failing even more thoroughly than I predicted there."

I suspect the recipients of these letters thought the views were too extreme. However, the first comment was made three months, and the second, seven days, before I heard Eva Lee Snead's broadcast of 31 May 1987, and learned of the greatest, most obvious dumphole and largest societal failure of them all.

Part VII

Over the last four years I have tried a great many different approaches to getting this information made public, including several less conventional ones I have not listed here. No matter how clever the effort I launched, there was always somebody there ready to bat it down. For four years they intercepted every attempt. In even the most modestly well-constructed society information of life and death importance to many millions is *easy* to deliver. One but speaks it to any public official, or any person of influence, or just whispers it in the streets, and soon it reaches the proper ears. What we have constructed is a society where precisely the opposite is so, an Alice-in-Wonderland society where the officials most directly responsible for disseminating this information have been most directly blocking its dissemination, a Madison Avenue society where all is hype, where the truth does not count so long as everyone buys the product, a Yuppie society full of Yuppie scientists, pursuing not science, nor the public welfare, but their Yuppie careers.⁴⁷ It should not have been difficult to bring this story out; it should have been impossible to keep it quiet.

One can view AIDS as a disease which is exploiting not only weaknesses in the human immune system but also in the human mind, the human character, and in the structure of society. And just as HeLa found its allies, so also AIDS has found allies of its own (and among some of the same sources). And these allies, ably deflecting every threat, can be thought of as AIDS' immune system, protecting it against the assaults of a hostile world. Unlike our own immune system, AIDS' immune system has been strikingly effective.

I have already discussed a number of the weaknesses AIDS is exploiting. There are a great many more I will not have time to pursue. In the specific area of scientific publication, I have already listed the intellectual/character weakness of editors refusing to believe what they don't want to hear, and the weakness of the social structure which has given editors the power to make decisions of immense worldwide, and indeed immense historic, significance with no accountability and at their whim. It is a bad combination, and AIDS has been a clever enough adversary to have found and exploited both these weaknesses to great advantage.

I will briefly mention four other weaknesses of the information distribution network that AIDS has been exploiting. I will not have time for the lengthy discussion each deserves. There are many more examples I am not even listing.

Reality is a seamless whole where virtually everything affects virtually everything else. There are, however, various concentrations of interaction or causation, and we have somewhat artificially divided these up into "disciplines." There is a certain amount of overlapping at the edges of many closely-related disciplines, and this is good. There is a certain amount of bridging that is done even between more distantly-related disciplines, and this is also good. But there is much about the structure of *reality* that is missed by this artificial classification. There are important connections between information fitted into separate disciplines that are being badly overlooked. These weaken our man-made structure. And there are important gaps in the seams between adjacent disciplines. We have a leaky structure where information that we need to encompass is leaking out. Another way of putting it is that AIDS has found these cracks in our defense and is getting in. And these *inherent* weaknesses in our way of dealing with reality by dividing it into self-contained, graspable chunks, become magnified by social interactions within each discipline that tend to draw it into itself and thereby widen the gaps: the tendency of disciplines to develop a jargon and often a dogma and

to some extent a clique, all of which make it more difficult to bridge the gaps. This is further magnified by a tendency for research not to push out the borders of the discipline at the edges, nor to establish connections to other disciplines, but rather to superspecialize and plunge ever deeper into the minutiae of the subject until it is impossible to be an expert except by expending all one's effort in the field, with little or nothing left over to become even cursorily familiar with other disciplines. Superimposed on all this is a greater or lesser degree of frank territoriality. Finally the publication process steps in, with its journals rigidly demarcated along disciplinary lines, and bars the gate to anyone foolish enough to attempt to bridge the gaps or describe the connections the information structure is leaving out. Thus the cracks become gaping holes, and it is no great compliment to AIDS that it has been able to find them.

In the current situation, widespread ignorance among vaccine researchers of the seriousness of epidemics of new diseases was a prerequisite for the original experiments that started AIDS. Widespread ignorance about techniques of vaccine production and about the recent history of their own discipline are prerequisites for medicine's continuing to deny responsibility for AIDS' origin. Widespread ignorance and misunderstanding about the most basic mechanisms of natural selection in large part account for how it is possible for AIDS researchers to remain unaware of AIDS' increasing transmissibility. There are many, many other examples of gross ignorance masquerading as common knowledge in the field of AIDS research.

The second weakness in the structure of the information network is its too great emphasis on form over content. Even within a single discipline, if information doesn't fit into the ordinary length requirements, for example, it is virtually unpublishable. There are important pieces of information being overlooked either because they inherently require a longer or otherwise different format from what normally appears in the journals, or because their authors are unable to make them conform. Whosever fault it is, important information is escaping the information structure through this route. When information is important, then somehow means must be found to accommodate it. A single piece of information of great importance is far more valuable to society than any journal, or any hundred journals. No matter what hundred journals you pick, the world would not change in any material way if they suddenly ceased to exist. The single preventable error of AIDS has already changed the world in a far profounder way, and in my view it is likely that only a fraction of one percent of AIDS' ultimate death toll has so far been realized.

The third weakness in the information structure is an unconscionable tolerance for errors and a very great disinterest in getting to the bottom of things. While this is more true of AIDS research than any other field I have encountered, it is in fact a very widespread phenomenon. On matters of the utmost importance, due care is not taken. Glaring errors that should never have been made in the first place abound, and then no one steps in to correct them. All attention is directed toward endless strengthening of the points that are already strong; the weak points, at least the important ones, are ignored for fear of the consequences (both to the field and to the researcher) should they be found to be in error. This piece has given important examples from at least the fields of AIDS research, vaccine research, and editorial practices. Michael Gold's book¹ certainly presents a striking example in the field of tissue culture. However, this is a large topic and these examples barely scratch the surface.

The fourth weakness is a lack of perspective on what is important and what is trivial. Material of fundamental significance is ignored even as tremendous resources are being poured into the nonessential. Again this is a large topic, again examples abound, and again Gold's book is a particularly good one. This is not only because it presents such a frightening picture of a field which had no sense of perspective, but even more because the book itself is a most striking example of the phenomenon. I don't know a lot about tissue culture, and for this reason I read the book twice and spent more than a week beyond that in checking out Gold's claims before including him in this piece.⁴⁸ I am convinced that the story he tells is accurate. And if it is accurate, then his book is indeed among the most important of our time. It was favorably reviewed in the *New York Times*. Articles appeared in *Science Digest* and *Reader's Digest*.⁴⁹ No one has refuted his claims. Yet in the five years since its publication, it has been cited only four times in the 3200 professional scientific journals listed in the *Science Citation Index*. Here is one of the most important books of our time, with enormous implications for all of scientific research, and much beyond. For a brief moment the information structure held it in its grasp. But it too slipped through the gaps.

The fundamentally important question of SIV's manner of spread in wild monkeys has received almost no attention. Clearly it is not spread through needles, and almost as clearly not in the main through anal intercourse. Whatever the method, it will almost certainly become a significant source of human spread once the virus has adapted more fully to our species (unless, of course, it is spread through biting or some other means that does not have an

important human counterpart). This is one of the first things that should have been investigated, particularly since the very similar maedi-visna virus of sheep has been known since the 1950s to be spread through airborne droplets.⁵⁰

There are many, many more examples. One should also note how, as in this last case, the various weaknesses interact to strengthen one another.

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I cannot conclude without pointing out the obvious. The origin of AIDS was a low-technology error, easy to see and — one would have thought and hoped — foresee. Nevertheless it was done not by a single inept scientist but by a number of the world's foremost researchers, was done in public, not in an obscure or minor way but in some of the most heavily publicized experiments of the twentieth century, all well documented in the scientific literature. Yet the mistake — not some insignificant mistake but the greatest yet made by the human race — once made was not caught even by the largest medical effort ever mounted against a disease, despite evidence that should have been unmistakable, and was not acknowledged even four years after it had been explicitly called to their attention, and done so multiple times (by someone from outside their field and finally published in a periodical from outside their field). Do you understand how *easy* all this *should* have been? Do you understand how *utterly* the system failed?

We are now entering a much different era. Many times more researchers, 99 percent of them less competent than those who gave us AIDS, almost all of them more focused on their own specialty and more ignorant of everything else, are engaged in countless high-tech experiments whose untoward consequences may *legitimately* be hard to foresee, may indeed be *impossible* to foresee, by anyone and particularly by an outsider, are doing many of these behind the closed doors of corporate research facilities where secrecy is the watchword and profit the bottom line, are doing them all over the world, largely unsupervised. Before proceeding further with such a hazardous enterprise, one should be able to come up with at least a half-way plausible argument that the likely benefits of such science outweigh the likely costs. This is another *fundamental* piece that is missing from the structure. I think there will be more such disasters and that they will outweigh any conceivable benefits many times over. At the very least this will be true under the system of science as currently practiced.⁵¹

It does seem to me that this is a conclusion that follows so immediately and obviously from all that I have said that the only way around it is to show that my scientific allegations about AIDS' origin and transmissibility are very badly wrong. Much that I have said in this piece is of an extreme nature, and extremely uncomplimentary to scientists, editors, and indeed the whole human race. But again, these conclusions follow so immediately from the extremity of the facts, if they are as I allege, that the best if not only way around them is to disprove the allegations. You are quite welcome to try. I think you will find there is a good deal more evidence for my position than I have given here. And I do not think you will find a single thing (that is both valid and of any significance) that can be said on the other side. I think that in fact I have understated things, in places quite considerably.

Part VIII

When an organism suddenly finds itself in a new ecological niche, if it is capable of reproducing within that niche, then little by little over the generations (and it will happen rather rapidly) it will adapt itself better and better to this new niche, shaping itself to the mold it has fallen into. Thus AIDS came to us able to exploit important weaknesses of the human immune system, weaknesses we did not even suspect were there. It is likely that other, related weaknesses exist which AIDS cannot yet exploit but which, as its roots grow more and more into every chink and cranny of its new niche, we shall first become aware of through AIDS' revealing them to us in its complementary impression of its surroundings. And along with exploitable weaknesses of the human immune system, AIDS has found other exploitable weaknesses in the human mind, character, and society. Again these are weaknesses we scarcely knew were there, and again AIDS' talent for exploiting these weaknesses will increase with time. And it will ferret out other weaknesses still unsuspected and reveal these weaknesses to us through a much starker image than any social critic could possibly sculpt. There is a live thing growing within us. Whether we will survive depends on the extent of weaknesses we can not find but through AIDS' help and can not fight until AIDS has already established its beachhead. I cannot guess the outcome. Anyone who can, and claims we will win, has nothing remotely resembling a grasp of our adversary. These people, those who have no inkling of what they are facing or what they are doing or what *awful* consequences could accrue if they slip up, are the very sort we hire to build our new organisms and to fight

against AIDS. This is a *profound* societal weakness. AIDS has revealed it to us. Can we correct it or will it prove fatal? And what of all the others?⁵²

Appendix

When a theory is met with counterarguments or with new facts that at first appear to cause a problem, then one knows that *if* the theory is correct, then these arguments *must* be invalid, or these alleged facts *must* be in error, or else the allegation of conflict *must* be mistaken. And if one looks into these arguments or alleged facts and finds this to be so, then the theory has not only survived the challenge but has been very materially strengthened thereby. A prediction based solely on the theory has been proved true, despite the fact that original appearances were to the contrary.

So far, in the four years since its formulation, and despite a welter of new research, I have seen only three facts that at first seemed to present problems. The first to come to light was convincing evidence that the 22 November 1985 *Science* paper,²⁴ which had formed an important part of my original argument, contained a major error. The virus supposedly isolated from African green monkeys turned out itself to be a contaminant that had come from a completely different monkey species whose virus was being experimented with in the same laboratory.⁵³ A little research showed: a) While the SIV actually isolated had come from a different species, African green monkeys were indeed infected with an SIV of their own, and in the proportions the researchers had claimed.⁵³ Thus my theory was saved. b) The monkeys the isolated SIV had actually come from were rhesus monkeys,⁵³ which in the early days were even more heavily used in polio vaccination than African green monkeys. My theory was doubly saved. c) The rhesus monkeys (and also cynomolgus monkeys) themselves had almost surely caught their SIV from yet another monkey species, the sooty mangabey, through other scientific experiments.⁵⁴ Scientists have inadvertently transferred SIV not only into humans but into two other species as well. And they acknowledge the likelihood of these latter two transfers. The new information not only fails to conflict with my theory, thus fulfilling the prediction, but independently adds strong support. d) Prominent researchers at a prominent institution (Harvard) equipped with much superior isolation methods nevertheless contaminated tissue cultures two separate times (their cultures from Senegalese prostitutes were also contaminated), and with virtually the same virus as contaminated the polio vaccine.⁵³ My theory is doubly strengthened by this new information.⁵⁵

The second potential problem was controversial (but growing) evidence that HIV-1 may have come from chimpanzees.⁵⁶ Though I was never able to find what primate species Koprowski had used, I think it is very unlikely to have been chimpanzees. However, most of the polio researchers used chimpanzees for *testing* their vaccines. Taking a cue from the Harvard contamination above, we might speculate that SIV from one of these test chimpanzees contaminated the vaccine cultures. This would require a modification of my theory in the case of HIV-1, but surely it is a minor modification.

A little research shows that not only did Koprowski use chimpanzees in his testing but that his usage was extraordinarily heavy. He started a chimpanzee breeding colony near Stanleyville, Belgian Congo. The vaccine used in the two African campaigns had been tested in ten chimpanzees, who were killed and their brains and spinal cords examined for signs of polio damage. Is it unreasonable to suspect that an implement used in removing a monkey's kidneys for this new batch of vaccine might not earlier have been used in dissecting a chimpanzee during testing of one of his previous experimental batches?⁵⁷ Is it not more than a little surprising to look into the possibility Koprowski tested in chimpanzees and find such *heavy* involvement? How many single papers in all scientific literature can there be that indicate a greater potential for a chimpanzee virus contamination than this one, the paper describing the first oral polio vaccination campaign, mentioning both a breeding colony and ten chimpanzees killed in a single experiment?⁵⁸ The evidence that HIV-1 in fact came from chimpanzees is comparatively weak. Perhaps it didn't even happen. But the fact that my investigation showed such unexpectedly large potential for such a source means either the chimpanzee theory is correct or else we have an impressively large coincidence here. Koprowski's heavy involvement with chimpanzees significantly strengthens the case for a chimpanzee origin. And the coming together of these two theories also strengthens my own case for AIDS' origin through Koprowski's vaccine, at least a little bit.

The final piece of evidence potentially troublesome for my theory is the case of a British sailor allegedly dying of AIDS in 1959.⁵⁹ The "proof" of AIDS in this case was a positive test for HIV-1 in the sailor's preserved tissue specimens. There are two ways this result could be compatible with my theory. First of all, the test may have been a false positive. The test they used (the polymerase chain reaction) is so sensitive that it regularly produces positive results when even a single DNA molecule is present.⁶⁰ *Extreme* measures must be taken to

prevent contamination, since one AIDS virus particle is tens of thousands of times smaller a quantity of material (and the complete DNA of one AIDS virus is several million times smaller) than the minimum of one entire, intact, *living* HeLa cell necessary to produce contamination. Page 64 of Gold's book¹ describes the rigorous procedures at the U.S. National Cancer Institute's Emergency Virus Isolation Facility (no windows, controlled air flow throughout the building), procedures which proved insufficient to prevent at least two instances of HeLa contamination at that facility. Pages 53-56 detail the even more rigorous procedures at Nelson-Rees' facility. This is in marked contrast to the measures described by the British sailor's researchers. They say, "Sections were cut with separate knives for case and control and with careful cleaning, with alcohol-soaked swabs, of knives between blocks." If this is all they did, it is certainly one and probably two orders of magnitude short of what would have been necessary to guarantee uncontaminated samples. There are a number of AIDS-like cases from the pre-AIDS era. It may well be that samples from several have now been examined by the PCR test. The first of these to be contaminated (and the control not contaminated) would be the one that makes the newspapers as the earliest case of AIDS. If this turns out to be yet another instance of contamination, then it not only fails to conflict with my theory but provides even more evidence of how rife this error is and how little prepared researchers are to recognize and combat it. (Note that the 1959 Kinshasa sample was found HIV-positive by ELISA, Western blot, indirect immunofluorescence, "and this plasma was also tested in three other laboratories by different techniques".²¹)

If the sailor did indeed have AIDS, and yet my theory is correct, then we can surmise there might have been earlier medical experiments that could have given someone AIDS. Looking into this question, we find there were *many* possibilities from the early-to-middle 1950s when polio researchers and adenovirus researchers were developing their vaccines from monkey kidneys and testing in small numbers of people. (The original African campaign was the first *mass* usage of oral vaccine — counting it and the Leopoldville campaign together, 1/3 million Africans were fed the suspect batch.) There were other experiments where live monkey cells were injected directly into humans.⁶¹ And there were important possibilities from much earlier. In 1935 Kolmer polio vaccine was used on 11,000 people in 36 U.S. states and Canada.⁶² It consisted of the spinal cords of polio-infected rhesus monkeys suspended (Kolmer specifically says not to filter the solution⁶³) in liquid. The vaccine was injected rather than being given orally. Each injectee received roughly 75 mg of spinal cord. Kolmer had treated the vaccine with a 1 percent

solution of a soap made from castor oil, which he thought would weaken the polio virus, and with 50 percent glycerine, which he thought would add slightly to the weakening. Later he concluded his treatment had affected the polio virus very little if at all.⁶⁴ A number of people developed paralytic polio, and five died as a result of Kolmer's vaccine. Most of the February 1936 issue of *American Journal of Public Health* is devoted to this vaccine and another early polio vaccine (dangerous, but much less so) given to more than 9000 people in 1935. I did not look back any further, but I have reason to suspect there were other experiments even earlier, and experiments in other parts of the world (such as the endemic centers of HTLV-1 in Japan and the Caribbean — STLV-1 is found in both rhesus and African green monkeys and is 90-95 percent identical in its nucleotide sequence to HTLV-1⁶⁵). One might note that this single 11,000-person vaccine trial of 1935 almost certainly injected more monkey material into humans than has occurred from all the monkey bites of the last hundred years.

There is an objection based on misunderstanding that keeps coming up over and over again: If contamination occurred in as many batches as I claim, then we would see AIDS epicenters all over the world rather than an epidemic spreading out from a single epicenter in Central Africa.

In a letter dated 6 April 1990, which was mailed to a number of correspondents, I refuted this objection no less than five independent and individually sufficient ways. I included four of these in my piece for *African Commentary*. The objection ignores a great many important points, such as the degree of contamination of the average batch and the variability about that average, the average level of susceptibility among humans and variation about that level, the average level of infectiousness and variation about that level for each of the different SIVs, the number of batches expected to be infected with each SIV, the effect of the reduced transmissibility of the early cases, the effect of large variations among cities and regions of the world in regard to the ease with which the viruses spread, the effect of exponential growth in magnifying size differences produced by growth rate differentials over time, the effect of differences in starting times on an epidemic growing exponentially.

I need not take the time here to formulate this list of shortcomings into separate refutations, because the objection cannot even be made if my claim is properly understood. My claim is that all or nearly all current HIV-1 cases arose via person-to-person spread from a relatively small number of individuals infected by Koprowski's contaminated vaccine given in Central Africa, and that with the

possible exception of Koprowski's other batches, it is likely no further batches were contaminated with the ancestor of HIV-1. If contamination with that particular SIV had occurred very many times at all, we would have been able to find HIV-1's ancestor by now. HIV-1's ancestor is either a *rare* SIV infecting the monkeys used in vaccine manufacture, or else an SIV of another primate species not ordinarily used in vaccine manufacture but used by Koprowski, or else an SIV of another primate species that entered the vaccine through a separate contamination event, similar to the rhesus SIV contamination of Kanki's and Essex's African green monkey and human prostitute cultures.⁵³ This being my claim, no one can say that my claim predicts HIV-1 epicenters all over the world. It predicts an epidemic spreading out from a single epicenter in Central Africa.

In the case of HIV-2, my claim is that a single still-unidentified batch given out in West Africa is the most likely origin of all current cases. HIV-2's ancestor *has* been identified. It is an SIV common in sooty mangabeys, not ordinarily used in vaccine manufacture, which has infected a very few rhesus and cynomolgus monkeys. It is easy to imagine that only one batch of vaccine ever became contaminated with the ancestor of HIV-2.

Finally, my claim is that many hundreds of batches were undoubtedly contaminated with the common SIV of African green monkeys. This is the *only* known SIV that could have contaminated more than a very few batches. But HIV-1, even when *injected* in very *large* quantities, fails to infect African green monkeys. Would it be surprising to find that SIV_{agm}, even if *injected* in very *large* quantities, failed to infect humans? Of course not. It would not be surprising if it did, and it would not be surprising if it did not. Each SIV differs in the range of species it is able to infect.

When my claims are properly understood, the objection cannot even be made. When my claims are not properly understood, as for instance if it were believed I had claimed many batches were contaminated with the ancestors of HIV-1 and/or HIV-2, there are still at least five different ways to refute it.

There are two studies that have purported to investigate whether oral polio vaccine could have been contaminated with SIV; however both are clear attempts at whitewash in my view. The more incriminating of these dates all the way back to a 15-16 July 1985 meeting of a panel of experts,⁶⁶ thus showing that the medical establishment has been well aware of the problem since even before the 22 November 1985 *Science* article appeared. They concluded SIV did not present a danger because: a) 250 vaccine recipients

tested did not show evidence of infection; b) long-term follow-up of recipients has not revealed a problem; c) kidney cultures contain "few, if any" T-lymphocytes; d) during the 1970s, kidney cultures were tested for retroviruses and none were found; e) tests of 20 current vaccine batches revealed no SIV.

I would reply: a) It is certainly to be expected that SIV infection via the oral route is a rare event. If 250 people were fed an equivalent amount of semen from known HIV carriers, the most likely number of resulting infections would be zero. This test is virtually meaningless. b) Since probably thousands of times more people have been infected by human-to-human spread than directly from contaminated vaccine, follow-up of those vaccinated would not be expected to produce any detectable difference. This is also meaningless. c) Surely it is clear there is a world of difference between a few T-lymphocytes and none. Which is it? In fact, the other study, discussed below, admits there are a few. This is more than plenty. Indeed, how can some portion of these few possibly not have been infected? To say there are only "a few" T-lymphocytes is in effect to say there is only "a little" SIV contamination of the vaccine.⁶⁷ Cells other than T-lymphocytes can be infected in any event. d) Both during the 1970s and today perhaps half or more of all kidney cultures were contaminated with simian foamy virus — a retrovirus.⁶⁸ Obviously the tests of the 1970s, which found no retroviruses, were not any good at all. e) At least as late as 1985 no one had *ever* been able to find HIV in hemophiliac factor VIII, despite the fact it was known to be there because more than half of U.S. hemophiliacs were already infected.⁶⁹ The tests of 1985 were not much good either. But the most mind-boggling deficiency of this piece is that they seem to think it is sufficient to test only 20 batches. Even if as many as 10 percent of batches were so heavily contaminated as to be detectable with their poor tests, that would still leave a chance of .9 to the 20th power — *or 12 percent* — of finding no contaminations! On such slipshod work have millions of lives been risked.

The other study is a letter to the editor of the journal *AIDS*.⁷⁰ They used very poor tests⁷¹ to try to find SIV in kidney cultures from known SIV-infected monkeys. They tested a grand total of *two* cultures with these very poor tests and concluded they were uninfected despite the fact that one showed a 40 percent increase in reverse transcriptase activity over the 4 weeks of the study. They then applied SIV to a grand total of *two* kidney cultures from uninfected monkeys and concluded kidney cultures were not susceptible to infection. They drew this conclusion despite the fact that their graph shows almost constant levels of reverse transcriptase during weeks 1-3 in both cultures followed by exactly parallel 2^{1/2}-fold increases in both cultures between weeks

3 and 4. It seems clear to me that both cultures were in all probability infected. Why in the world did they not continue this test, and their previous one, for longer periods? Since they themselves stress the fact there were only a few T-lymphocytes, and since in both experiments virus was having to adapt to new conditions, and since in the first case the virus could have been in a latent state besides, testing for 4 weeks is just absurd. In experiments with simian foamy retrovirus, even though 58 percent of 81 viable African green monkey kidney cultures were found naturally infected, this was seldom detected before 30-39 days in culture, with some cultures not showing contamination until after more than 100 days.⁷² On the basis of two questionable experiments on two cultures each, these researchers felt entitled to conclude: "From these results, poliomyelitis vaccines may be considered not to be contaminated with SIV_{agm}, even though they are prepared in primary kidney-cell cultures from SIV_{agm}-infected AGM." On such slipshod work have millions of lives been risked.

No criticism of either study has appeared in the literature. Both have, however, been cited by several other groups of researchers (including a group of six at the FDA) in support of their contention that polio vaccine was not contaminated.⁷³ The only piece to take the other side was unaware of the African campaigns and reached the relatively mild conclusion: "While it would be simplistic to assume and even more difficult to prove that polio vaccine is the source of HIV infection in man, it would be equally naive to ignore the possibility".⁷⁴ For their heresy, their letter was described as "scientifically, factually and conceptually incorrect, and in view of national and international efforts to control poliomyelitis reprehensively irresponsible misinformation".⁷⁵

Earlier I stated, "Papers reaching hopeful conclusions are printed despite awesome errors; papers reaching the most pessimistic conclusions are rejected despite overwhelming evidence." I claim my five previous rejected pieces on AIDS as examples of the latter. And I have now presented two stunning examples of the former. These pieces do not discuss some minor, technical point. They discuss a point which may well turn out to be the most important question science has ever investigated. "On matters of the utmost importance, due care is not taken. Glaring errors that should never have been made in the first place abound, and then no one steps in to correct them."

I could list many more examples.

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- * Louis Pascal, 51 MacDougal Street, #146, New York NY 10012, USA.
- 1 Gold M. *A conspiracy of cells: one woman's immortal legacy and the medical scandal it caused*. Albany: State University of New York Press, 1986.
 - 2 See reference 1: 136-138 and Broad WJ. The case of the unmentioned malignancy. *Science* 1980 Dec 12; 210: 1229-1230.
 - 3 See reference 1: 107-124 and Pathak S, Siciliano MJ, Cailleau R. A human breast adenocarcinoma with chromosome and isoenzyme markers similar to those of the HeLa line. *Journal of the National Cancer Institute* 1979 Feb; 62: 263-271.
 - 4 See reference 1: 98, 148.
 - 5 See reference 1: 147-148. This is a direct quote of Gold's indirect quote of ATCC-head Robert Stevenson.
 - 6 a. Dick GWA. Discussion. In: Alfrey VG, Bell JA, Bodian D, et al. *Cellular biology, nucleic acids and viruses (Special publications of the New York Academy of Sciences, vol 5)*. New York: New York Academy of Sciences, 1957: 134-137. b. Horstmann DM, Niederman JC, Paul JR. Attenuated type 1 poliovirus vaccine: its capacity to infect and to spread from "vaccinees" within an institutional population. *Journal of the American Medical Association* 1959 May 2; 170: 1-8.
 - 7 Koprowski H. The tin anniversary of the development of live poliovirus vaccine. In: *Second international conference on live poliovirus vaccines*. Washington, DC: Pan American Health Organization, 1960: 5-11. (Pan American Health Organization Scientific Publication No. 50) See also reference 40.
 - 8 Sabin AB. Recent studies and field tests with live attenuated poliovirus vaccine. In: *First international conference on live poliovirus vaccines*. Washington, DC: Pan American Sanitary Bureau, 1959: 14-33. (Pan American Health Organization Scientific Publication No. 44)
 - 9 Dates of U.N. admission are given in United Nations. *Everyone's United Nations*. 10th ed. New York: United Nations, 1986: 11-13. Dates of independence are given in Hoffman MS, ed. *World almanac and book of facts 1989*. New York: Pharos Books, 1989.
 - 10 Courtois G, Flack A, Jervis GA, Koprowski H, Ninane G. Preliminary report on mass vaccination of man with live attenuated poliomyelitis virus in the Belgian Congo and Ruanda-Urundi. *British medical journal* 1958 Jul 26; 2: 187-190.
 - 11 a. Lebrun A, Cerf J, Gelfand HM, Courtois G, Koprowski H. Preliminary report on mass vaccination with live attenuated poliomyelitis virus in Leopoldville, Belgian Congo. In: Reference 8: 410-418. b. Lebrun A, Cerf J, Gelfand HM, Courtois G, Plotkin SA, Koprowski H. Vaccination with the CHAT strain of type 1 attenuated poliomyelitis virus in Leopoldville, Belgian Congo. *Bulletin of the World Health Organization* 1960; 22: 203-213. For evidence that the same batch was used in both campaigns, see reference 25, below.
 - 12 a. Sabin AB. Present position of immunization against poliomyelitis with live virus vaccines. *British medical journal* 1959 Mar 14; 1: 663-680. b. Sabin AB. Discussion. In: Reference 8: 577-579. See also reference 25, below.
 - 13 Geissler E, Scherneck S, Waehlte H, et al. Further studies on the relationship of SV40-like viruses to human tumors. In: Essex M, Todaro G, zur Hausen H, eds. *Viruses in naturally occurring cancers*. Cold Spring Harbor, New York: Cold Spring Harbor Laboratory, 1980: 343-355.

- 14 See reference 7: 7-8.
- 15 Reference 11a: 415 says infants under 30 days were given 10 times the normal dose (because they make antibodies less readily). However p. 417 says they received 15 times as much, and the latter figure is confirmed in reference 11b: 204. For vaccination of 48-hour-old infants, see pp. 463-464 of Anonymous. Poliomyelitis prevention. *WHO chronicle* 1960 Dec; 14: 462-468.
- 16 See, for example: a. Sabin AB, Schlesinger RW. Production of immunity to dengue with virus modified by propagation in mice. *Science* 1945 Jun 22; 101: 640-642. b. Maruyama K, Dmochowski L. Cross-species transmission of mammalian RNA tumor viruses. *Texas medicine* 1973 Aug; 69: 65-75. c. Kuwata T. Studies on adaptation of Rous sarcoma virus to ducks. *Cancer research* 1964 Jul; 24: 947-953. d. Burnet FM. Men or molecules? A tilt at molecular biology. *Lancet* 1966 Jan 1; 1: 37-39.
- 17 London WT. Epizootiology, transmission and approach to prevention of fatal simian haemorrhagic fever in rhesus monkeys. *Nature* 1977 Jul 28; 268: 344-345.
- 18 Kestler HW III, Li Y, Naidu YM. Comparison of simian immunodeficiency virus isolates. *Nature* 1988 Feb 18; 331: 619-621.
- 19 I am using the term "monkey bite hypothesis" to include all similar suggestions of a natural, accidental, one-time transfer of a monkey virus into a single human, who then started the epidemic of AIDS; e.g., a person with bleeding gums eating undercooked monkey meat.
- 20 Altman LK. AIDS in Africa: a pattern of misery. *New York Times* 1985 Nov 8: A1.
- 21 Nahmias AJ, Weiss J, Yao X, et al. Evidence for human infection with an HTLV-III/LAV-like virus in central Africa, 1959. *Lancet* 1986 May 31; 1: 1279-1280. (See Appendix for an extended discussion of another alleged early case.)
- 22 Null currently has a similar program on WBAI-FM in New York.
- 23 See reference 13.
- 24 Kanki PJ, Alroy J, Essex M. Isolation of T-lymphotropic retrovirus related to HTLV-III/LAV from wild-caught African Green monkeys. *Science* 1985 Nov 22; 230: 951-954. (I have credited Snead with the contamination theory, rather than these authors, because any suggestion that contaminated vaccine might account for the origin of AIDS is glaringly absent from their work.)
- 25 The same batch of vaccine contaminated with an unidentified virus was used in the Ruanda-Urundi/Belgian Congo campaign, the Leopoldville campaign (1/3 million vaccinated in the two together), and in several small trials, such as a 3000-person Polish campaign of October 1958. See Przesmycki F, Dobrowolska H, Olakowski T, Stanczyk R, Naruszewicz D. Report on field trials with live attenuated poliomyelitis vaccine in Poland. In: Reference 8: 497-507. Their table 1 (497) gives the batch as Pool 13 of CHAT Type 1 vaccine. Reference 11a: 416 confirms that the Leopoldville campaign also used Pool 13. Additionally, Koprowski H. Discussion. In: Reference 8: 508, says the same batch was used in Poland and Leopoldville. In reference 12b: 577 Sabin says that the batch used in Poland was the one in which he had found a contaminating virus. And in reference 12a: 678, discussing this same contaminating virus, Sabin says the batch had been used in the Ruanda-Urundi/Belgian Congo campaign. Thus, the Ruanda-Urundi/Belgian Congo and the Leopoldville campaigns both used the same contaminated batch, Pool 13.
- 26 McClure M. Where did the AIDS virus come from? *New scientist* 1990 Jun 30; 126(1723): 54-57. I wrote *New scientist* again 11 Aug 1990 and on 15 Oct finally received a reply (dated 7 Sep). Phyllida Brown said she had recently taken over as Medical Science Reporter and had sent the material to a referee. There was no explanation for the previous unanswered letters. When I had heard nothing further by 18 May 1991, I inquired yet again. As of 18 Nov 1991 no response had been received.

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- 27 Singer P, ed. *Applied ethics*. Oxford: Oxford University Press, 1986.
- 28 Perlez J. In AIDS-stricken Uganda area, the orphans struggle to survive. *New York Times* 1990 Jun 10: A1.
- 29 American Cancer Society. *Cancer facts and figures*. 1990 ed. Atlanta: American Cancer Society, 1990. This says 30 percent will get cancer and 6,000,000 (nearly all adults) have had it. I estimated 70 percent of the U.S. population was 21 or older. This gave 3.4 percent of adults.
- 30 Hooper E. AIDS epidemic moves south through Africa. *New scientist* 1990 Jul 7; 127(1724): 22. However, I stress again that I am *not* predicting that the incidence will reach one-third of a living population. An incidence of 8 percent or so, as in Kinshasa, if maintained unchanged, will result in this 8 percent dying over the next few years, then another 8 percent in the years thereafter, etc., until, I am suggesting, a cumulative total of one-third or more of the people born will catch AIDS and die.
- 31 Harden B. AIDS seen as threat to Africa's future. *Washington Post* 1987 May 31: A1.
- 32 There are very, very few estimates of such an important quantity as the ultimate toll AIDS may be expected to take. All those I am aware of reach conclusions at least as dire as mine. The principal study is Anderson RM, May RM, McLean AR. Possible demographic consequences of AIDS in developing countries. *Nature* 1988 Mar 17; 332: 228-234. They conclude (233): "For plausible ranges of parameter values the disease AIDS is predicted to be capable of significantly reducing population growth rates, and even depressing them to negative values. . . . Similar analyses of the impact of directly transmitted infections such as smallpox and bubonic plague, that were of great historical significance as causes of human morbidity and mortality, suggest that AIDS has greater potential to depress significantly human population growth rates." They show graphs indicating AIDS may be able to push even populations growing at 4 percent per year into negative rates. As of mid-1991, there was only one country in the world growing this fast (Jordan). The 10 AIDS countries I listed have a combined growth rate of 3.4 percent, for a doubling time of 20 years. One-third developing AIDS would not be sufficient to cause negative growth in a population otherwise doubling once a generation (AIDS would have to stop more than half the population from reproducing at all or an even bigger fraction from completing their reproduction). Anderson's study contains serious (and acknowledged) oversimplifications, and its heavily mathematical nature perhaps lends it an undue air of authority. I think my own simple estimate is readily graspable and less subject to large errors. According to Brown P. Africa's growing AIDS crisis. *New scientist* 1990 Nov 17; 128(1743): 38-41, "The WHO. . . predicts that the population of some African countries will actually decline because of AIDS." I am not saying I think such figures are too high. I am saying they are higher than my own estimate, which, however, is meant to be conservative. See also the major front-page series on AIDS in Africa by Eckholm E and/or Tierney J in the *New York Times* 1990 Sep 16, 17, 18, 19, and De Cock KM, Barrere B, Diaby L, et al. AIDS — the leading cause of adult death in the West African city of Abidjan, Ivory Coast. *Science* 1990 Aug 17; 249: 793-796.
- 33 a. Rensberger B. AIDS virus a clever enemy, study shows. *Washington Post* 1987 Sep 6: A1. b. Joyce C. Viral mutation rate alarms AIDS researchers. *New scientist* 1987 Jun 4; 114(1563): 28-29.
- 34 E.g., all practices that increase the duration of infectiousness without reducing the degree of infectiousness at least proportionately must necessarily increase cases. With the unproved but possible important exception of AZT, this characterizes the majority of current medical treatments in nearly all countries. And there are other sorts of examples as well.
- 35 I arbitrarily chose 50 million as a round figure roughly in the middle of estimates for World War II deaths. E.g., *Encyclopaedia Britannica* (1984), at the end of its long article "World Wars," estimated 35-60 million.

- 36 The 3 polio vaccines are usually given mixed together as one "trivalent" vaccine. Fifty human viruses in the same genus as the three polio viruses are given in Matthews REF. *Classification and nomenclature of viruses*. New York: S Karger, 1979: 104. For the haphazard way the SIVs are detected, see Hayami M, Ohta Y, Hattori T, et al. Detection of antibodies to human T-lymphotropic virus type III in various non-human primates. *Japanese journal of experimental medicine* 1985 Dec; 55: 251-255, and Ohta Y, Masuda T, Tsujimoto H, et al. Isolation of simian immunodeficiency virus from African green monkeys and seroepidemiologic survey of the virus in various non-human primates. *International journal of cancer* 1988 Jan 15; 41: 115-122. These papers (they have 3 authors in common) tested 182 African green monkeys and found only one that reacted positively to their tests. They then took the virus they isolated from this animal and used it to make a more specific test. They used this to test 343 African green monkeys and found 90 of them, or 26 percent, reacted positively. They almost missed detecting even this extremely common SIV. What about all the rare ones? What about all the common ones that are just a *little* more different than this one? And how many more of these green monkeys may have been infected but failed to react to the test made from this *aberrant* virus?
- 37 a. Sharp PM, Li W-H. Understanding the origins of AIDS viruses. *Nature* 1988 Nov 24; 336: 315. b. Li W-H, Tanimura M, Sharp PM. Rates and dates of divergence between AIDS virus nucleotide sequences. *Molecular biology and evolution* 1988 Jul; 5: 313-330. Several groups have employed backward projection of divergence to estimate the date of AIDS' origin. Every one I have seen is compatible with my own date. This group came closest, but that is not the only reason I have cited them. By considering only changes to nucleotides that do not result in changes to amino acid sequence, and are therefore nearly neutral with respect to selection, they avoid two important sources of error. I do not know of any other study that did this.
- 38 But there are important reasons, which I will not take time to go into here, why a second introduction of, say, HIV-1 now would be a very, very bad thing, quite possibly costing many millions of lives.
- 39 One of the most likely new disease candidates, HTLV-1, is mentioned in passing in the appendix. John Seale independently suggested a vaccinal origin for HTLV-1 in another extremely important unpublished manuscript submitted to *Nature* in Nov 1989.
- 40 a. Smorodintsev AA, Drobyshevskaya AI, Ilyenko VI, Klyuchareva TE, Chalkina OM. Experience in the production, biological control, and use of live poliomyelitis vaccine made from Sabin strains. In: Reference 8: 305-312. b. Potash L. Methods in human virus vaccine preparation. In: Maramorosch K, Koprowski H, eds. *Methods in virology*. Vol IV. New York: Academic Press, 1968: 371-464.
- 41 For 75 viruses, see Kalter SS. Primate viruses — their significance. In: Kalter SS, ed. *Viral and immunological diseases in nonhuman primates*. New York: Alan R Liss, 1983: 67-89. See p. 68. More than 40 of these viruses were known by 1959: see Murray R. Discussion. In: Reference 8: 577-578. The first SIV was not discovered until 1985, during the course of research on AIDS. The common SIV of green monkeys asymptotically infects a quarter or more of them. Several thousand infected monkeys were unknowingly used in making vaccine. Neither HIV-1 nor HIV-2 is closely enough related to this SIV to be its likely descendant— apparently this SIV infects humans not at all or only with great difficulty. The possibility remains quite high that rare variants can infect humans and that if the exposure is continued for long enough, we will eventually find such a variant (see reference 71). The possibility also remains that the ancestor of HIV-1 is a different and undiscovered SIV infecting green monkeys. But see Appendix for what I now regard as the most likely ancestor of HIV-1.
- 42 Hsiung GD. Latent virus infections in primate tissues. *Bacteriological reviews* 1968 Sep; 32: 185-205. From p. 201: "Latent virus infections in primary cell cultures are often not recognized. Virus stocks derived from such latently infected cultures or virus vaccines produced from these infected cells would undoubtedly be contaminated with indigenous viral agents." Yes, of course they would; but you have to be trying to find them rather than trying not to find them. See Appendix.

- 43 Lecatsas G, Alexander JJ. Safe testing of poliovirus vaccine and the origin of HIV infection in man. *South African medical journal* 1989 Oct 21; 76: 451. Also see Appendix and reference 75. Since writing this note, another letter of Lecatsas has appeared: Lecatsas G. Origin of AIDS. *Nature* 1991 May 16; 351: 179. Moreover, Professor Lecatsas has informed me (in letters dated 4 Sep and 14 Oct 1991) that his contribution to the controversy is not limited to the above. Rather, he has tried over the last three years to publish results in *Nature*, *British medical journal*, and to comment in *New Scientist*. "All were rejected with negative and often nonsensical comments."
- 44 One example of such a hole is given later in this paper. There are several others.
- 45 Beyond the vaccine bridge, there are several others that have been and are being built, including one which is rapidly building itself and which has the potential, in my view, to grow even larger than the vaccine bridge. I do not have time to pursue these here: like the vaccine bridge, they involve stepping on toes.
- 46 Nozick R. *Philosophical explanations*. Cambridge, Mass.: Harvard, 1981: 3.
- 47 For a good example of Yuppie science see Kwitny J. Science follies: at CDC's AIDS lab: egos, power, politics and lost experiments. *Wall Street Journal* 1986 Dec 12: 1.
- 48 The most important part of this research was making certain the 1/3 contaminated cultures did not consist mainly of one or a few contaminated samples among thousands of valid samples of the various contaminated lines. Gold's examples of Gartler's 17 out of 17, the Russians' 6 out of 6, and 24 out of 34 even from the ATCC itself are strong evidence that this was not the case. My further reading added to this conviction. See, for example, Stulberg CS, Coriell LL, Kniazeff AJ, Shannon JE. The animal cell culture collection. *In vitro* 1970 Jul; 5: 1-16, and Peterson WD Jr, Stulberg CS, Simpson WF. A permanent heteroploid human cell line with type B glucose-6-phosphate dehydrogenase. *Proceedings of the Society for Experimental Biology and Medicine* 1971 Apr; 136: 1187-1191. The latter's authors announce the development of the *first* permanent human cell culture having heteroploid, epithelial-like cells — i.e., resembling HeLa closely enough to be a candidate for contamination — that was not in fact HeLa. In every other case all samples tested, including the earliest available, were HeLa contaminants. This is because nearly all attempts to establish permanent human cell lines failed. The cells died out after a certain number of divisions. Until these authors developed their culture, in every case where the cells lived on (and were heteroploid and epithelial-like, as was nearly always the case for permanent lines) it was because they were already HeLa. There were *no* valid samples of these cultures because they had been HeLa contaminants from the very beginning. See also Gilbert DA, Reid YA, Gail MH, et al. Application of DNA fingerprints for cell-line individualization. *American journal of human genetics* 1990 Sep; 47: 499-514. Three of these authors are at the ATCC and two at NCI, and their description of contamination (499-500) matches Gold's very nicely and favorably cites him.
- 49 a. Schmeck HM Jr. HeLa's legacy. *New York Times Book Review* 1986 Jun 15: 18. b. Gold M. Cancer cover-up. *Science digest* 1986 May; 94(5): 64-67, 80-81. c. Gold M. The immortal cells of Henrietta Lacks. *Reader's digest* 1986 Sep; 129(773): 49-53. Note that Gold's two articles greatly downplay the degree of cover-up. The closest thing to a negative review was Hicks J. The man who protested too much. *Science* 85 1985 Oct; 6(8): 90-92. This does not question Gold's scientific claims but blames the chaos on a few rotten apples. The obvious question of how a few rotten apples could have so thoroughly spoiled a whole field, despite the best efforts of the more responsible majority, is never addressed.
- 50 First see Sigurdsson B. Maedi, a slow progressive pneumonia of sheep. *British veterinary journal* 1954 Jul; 110: 255-270 and/or Sigurdsson B, Palsson PA, Tryggvadottir A. Transmission experiments with maedi. *Journal of infectious diseases* 1953 Sep; 93: 166-175. Follow with Lairmore MD, Rosadio RH, DeMartini JC. Ovine lentivirus lymphoid interstitial pneumonia. *American journal of pathology* 1986 Oct; 125: 173-181. John Seale deserves most of the credit for this alarming argument. I added the fact of increasing transmissibility. The argument was spelled out very carefully in my July 1987 paper rejected by *Nature*.

- 51 The alleged benefits of science are vastly exaggerated by the same improper accounting as has so distorted our perception of the AIDS mess, namely a failure to give due weight, or any weight, to negative consequences. Thus it may be claimed that antibiotics and other medical breakthroughs have already saved hundreds of millions of lives in this century. True, but they have also caused Earth's population to more than triple in this century, to a level well beyond what Earth's ecological systems can support. When those systems give out and population dies back to the level the by-then-pathetically-impooverished systems *can* support, those deaths must be subtracted from the benefits science is claiming. The resulting benefit will be a great deal less. Under reasonable assumptions about the magnitude of the ecological collapse, it will be a great deal less than zero. Thus this argument — the *only* one I can see that could be given as a half-way plausible rebuttal to my own accounting — is itself missing a fundamental piece. Even without AIDS, and whatever other diseases science may already have given us, it is far from clear that the overall impact of twentieth-century science would not be negative. It is more than a little ironic that science, which blindly caused the horrendous problem of overpopulation, may now have blindly solved it through its new diseases. But I am not sure we will like this possible solution a whole lot better than nature's own solutions of starvation, war, or natural pestilence. Reality is an impartial and unerring accountant, and it will set the books straight in the end.
- 52 There are many others listed in my previous unpublished writings on AIDS and in correspondence; more still I have not had a chance to investigate fully, some of which are surely true. And how many others have I missed entirely?
- 53 Mulder C. A case of mistaken non-identity. *Nature* 1988 Feb 18; 331: 562-563.
- 54 a. Gormus BJ, Murphey-Corb M, Martin LN, et al. Interactions between simian immunodeficiency virus and *Mycobacterium leprae* in experimentally inoculated rhesus monkeys. *Journal of infectious diseases* 1989 Sep; 160: 405-413. b. Desrosiers RC, Daniel MD, Li Y. HIV-related lentiviruses of nonhuman primates. *AIDS research and human retroviruses* 1989 May; 5: 465-473. I took these references from a most important unpublished paper of J Seale, submitted to *Nature* 12 Jan 1990. He says (p. 10): "Another 50 or so 'natural' cases in the mid 1970s occurred in a large group of stump-tailed macaques after many of them had been inoculated with sooty mangabey tissue during experiments related to kuru (Preston Marx personal communication)." Stump-tailed macaques would be still another species into which scientists inadvertently transferred SIV.
- 55 See Wain-Hobson S, Myers G. Too close for comfort. *Nature* 1990 Sep 6; 347: 18, for a probable four-level contamination involving HIV-1: Montagnier's LAV-1 contaminated Gallo's cultures giving HTLV-IIIb, which contaminated other cultures giving N1T, which contaminated other cultures giving MF. The 4 isolates are all virtually identical and the opportunity for contamination was present. It is difficult to imagine how rampant contamination would have to be for it already to have reached 4 levels, unless this strain is particularly prone to it. Other contaminations are mentioned. Wain-Hobson "has encountered more than a dozen cases of PCR [polymerase chain reaction] contamination." Since writing this, it has emerged that the strain Montagnier sent to Gallo was itself a contaminant from another of Montagnier's isolates! See Culliton BJ. Contaminated origin of AIDS viruses. *Nature* 1991 May 23; 351: 267.
- 56 a. Anonymous. A new chimp virus? *New scientist* 1990 Oct 27; 128(1740): 16. b. Huet T, Cheynier R, Meyerhans A, Roelants G, Wain-Hobson S. Genetic organization of a chimpanzee lentivirus related to HIV-1. *Nature* 1990 May 24; 345: 356-359.
- 57 The suspect batch was pool 13 of CHAT type 1 polio vaccine. Presumably there were 12 CHAT pools prepared previously. I have never seen what "CHAT" stands for, but "CHimpanzee ATtenuated" (i.e., producing no ill effects when tested in chimpanzees) seems a good guess. Sabin describes one of his experimental batches as "chimpanzee avirulent."
- 58 Reference 11b says the batch used in Leopoldville was tested in 5 chimpanzees. Reference 10 confirms this (noting that the same batch was used in the first campaign) and adds that vaccine against type 3 polio was also tested in 5 chimpanzees. Their

- central nervous systems were examined histologically, which would have necessitated killing them, and one chimpanzee was found to have mild lesions due to polio type 3. I am essentially certain this could only have been told if the 5 animals given type 3 vaccine were a different set of 5, giving 10 in all.
- 59 Corbitt G, Bailey AS, Williams G. HIV infection in Manchester, 1959. *Lancet* 1990 Jul 7; 336: 51.
 - 60 Cherfas J. Genes unlimited. *New scientist* 1990 Apr 14; 126(1712): 29-33. Also see reference 55 for PCR contaminations.
 - 61 Reference 1: 125-128 mentions Jonas Salk attempted to inject cynomolgus monkey heart cells into cancer patients, but later concluded his cells had been taken over by HeLa. I presume others tried similar experiments, but I have not looked for examples.
 - 62 Kolmer JA. Vaccination against acute anterior poliomyelitis. *American journal of public health* 1936 Feb; 26: 126-135.
 - 63 Kolmer JA. Active immunization against acute anterior poliomyelitis with ricinoleated vaccine. *Journal of immunology* 1937 May; 32: 341-356. See p. 344.
 - 64 Kramer SD. Active immunization against poliomyelitis. A comparative study. I. Attempts at immunization of monkeys and children with formalized virus. *Journal of immunology* 1936 Sep; 31: 167-182. "Dr. Kolmer now believes that the virus he uses is not modified appreciably, if at all, by this treatment — personal communication" (168).
 - 65 Essex M, Kanki PJ. The origins of the AIDS virus. *Scientific American* 1988 Oct; 259(4): 64-71. See p. 64.
 - 66 World Health Organization. T-lymphotropic retroviruses of non-human primates. *Weekly epidemiological record* 1985 Aug 30; 60: 269-270.
 - 67 See Sanford KK. Discussion. In: Merchant DJ, ed. *National Cancer Institute monograph 29 (Cell cultures for virus vaccine production)*. Bethesda, Maryland: National Cancer Institute, 1968: 41, for evidence that "lymphocytes can contribute quite substantial numbers of cells to a primary culture." It is possible this was true in 1957 and 1968 but is no longer true.
 - 68 a. Gear JH. Discussion. In: Reference 7: 86. b. Baker EF. Latent simian foamy virus. *South African medical journal* 1989 Oct 21; 76: 451-452.
 - 69 McDougal JS, Martin LS, Cort SP, Mozen M, Heldebrant CM, Evatt BL. Thermal inactivation of the acquired immunodeficiency syndrome virus, human T lymphotropic virus-III/lymphadenopathy-associated virus, with special reference to antihemophilic factor. *Journal of clinical investigation* 1985 Aug; 76: 875-877.
 - 70 Ohta Y, Tsujimoto H, Ishikawa K, et al. No evidence for the contamination of live oral poliomyelitis vaccines with simian immunodeficiency virus. *AIDS* 1989 Mar; 3: 183-184.
 - 71 For their test they tried to grow a monkey virus in human cells; and while this may often work, when it fails to work one cannot conclude it is because the monkey virus was not there. See Cheng-Mayer C, Seto D, Tateno M, Levy JA. Biologic features of HIV-1 that correlate with virulence in the host. *Science* 1988 Apr 1; 240: 80-82, for an indication of how tricky it can be to grow even the human-adapted virus HIV-1, with some strains infecting and others failing to infect various human tissue cultures, and indeed with later strains from the same patient infecting a greater range of cultures than earlier strains. They should have used many monkeys, many tissue culture types, and, above all, much longer periods of growth in culture (see following text). They did not use the PCR test. They did not even try so simple a test as injecting noninfected monkeys with the kidney cultures to see if they would develop the infection (potentially a very good test, since factor VIII was readily shown to be contaminated when it was injected into people). At the very least they should have conducted a careful search for the virus. There is no way they can claim to have done that.

- 72 See reference 68b.
- 73 a. Zuckerman AJ. AIDS in primates. *British medical journal* 1986 Jan 18; 292: 158. This letter does no more than summarize the WHO piece (reference 66). b. Hendry RM, Wells MA, Phelan MA, Schneider AL, Epstein JS, Quinnan GV. Antibodies to simian immunodeficiency virus in African green monkeys in Africa in 1957-62. *Lancet* 1986 Aug 23; 2: 455. These six FDA researchers cite both WHO and Zuckerman (above) as though they were two independent sources. c. Schoub BD, Dommann CJ, Lyons SF. Safety of live oral poliovirus vaccine and the origin of HIV infection in man. *South African medical journal* 1990 Jan 6; 77: 51-52. They cite Ohta (reference 70).
- 74 See reference 43.
- 75 See reference 73c and Lecatsas G, Alexander JJ. Professors Lecatsas and Alexander reply. *South African medical journal* 1990 Jan 6; 77: 52. The pieces in references 43, 66, 70, 73, and 75, along with 26, which proclaims the problem not to exist and cites no one, comprise a complete record of all published papers mentioning or discussing this question, in so far as I have been able to locate them.