A Strange Case of Certainty

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Abstract

The unresolved debate about the origin of HIV/AIDS is used as a case study investigating the power of institutional interests to control or influence what J M Keynes called the encroachment of ideas and Michel Foucault referred to as the battle over regimes of truth. The case study examines the creation of unreasonable public certainty about an unresolved scientific dispute.

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I. Introduction

In 1885 Ulysses S Grant died from an oral cancer widely believed to have resulted from his long term heavy consumption of cigars. However, despite continually mounting scientific evidence for the link between tobacco and many diseases, the public notion that smoking was safe, or even healthy, persisted for another 100 years.

Benjamin Franklin discovered that hot lead in printer's type was dangerous to his health, yet 200 years later we were still putting lead into gasoline and house paint.

In 1881 President James Garfield was hit by an assassin's bullet. The wound was serious, but should not have been fatal. Garfield died 11 weeks later from infections caused by doctors repeatedly searching for the bullet with unwashed fingers inserted into the wound. Scientific studies by the Hungarian physician Ignaz Semmelweis had shown more than 30 years earlier that hand washing reduced the death rate from puerperal fever of mothers giving birth in clinics, but Semmelweis's evidence was not widely accepted. The United States had much evidence of wound treatment and survival rates from the Civil War, but the evidence was largely unexamined and unanalyzed. In France Louis Pasteur had developed the germ theory of disease, but the theory was still rudimentary, viruses had not been discovered, and the link between infectious substances and specific bacteria was not established. In the US doctors retained a practical certainty that the inconvenience of hand washing was unnecessary.

These are examples of discord between scientific evidence and practical opinion. In the first examples the evidence is one sided, but the popular view was conflicted. There were anti-smoking campaigns, as well as pro-smoking advertising and product placement by the tobacco industry that maintained public uncertainty about the health effects of smoking. The effects of tobacco and lead don't show up immediately, so the public was uncertain.

In the hand washing example, the evidence was as yet uncertain, lacking an accepted theoretical basis, but doctors were certain (the wrong way) and took risks that they could (and should) have avoided.

The first kind of discord is the more easily identified and studied. Climate science and evolution are examples of firm scientific evidence being disputed by an uncertain public. Evidence that conflicts with economic, political, or religious interests or ideological beliefs tends to be ignored by the interested parties.

The second kind of discord is a little harder to identify and analyze, but it is perhaps even more common and its effects can be even more costly. There is a natural tendency to seek certainty and resolution and to seize upon answers to questions even when those answers are tentative and the science, the evidence, and the analysis behind them are still open to scientific dispute.

This second discord occurs when scientific issues are uncertain or unresolved, but the public holds and acts upon beliefs that are not backed by science, especially when those false certainties are supported by institutional interests.

Not so long ago blood-letting was widely accepted as the preferred medical treatment for numerous conditions. George Washington died from it. Benjamin Franklin accepted it. We recently avoided eggs, then butter, then salt. Now, instead, sugar is the concern. We used to remove children's tonsils, now we leave them in except in extreme cases. We banned marijuana as dangerous, now we prescribe it. There is a wide gap between what is believed at a particular time and what is actually known. Faith is said to be pretending to know things you don't know. When people say they have faith in science, they are pretending to know things they don't know about science. Science is not something to be taken on faith.

Science is a method of inquiry, not a set of certainties. Science applies to things we are uncertain about. Science aids decision-making by providing evidence. Most decisions can't wait for certainty.

Science also enriches life apart from immediate practicality. We are consumers of information. There is a problem, though. Much of our information, many of the things we think we know, especially things we unthinkingly accept without question, are going to turn out to be false.

Desire for certainty makes us gullible. We want the truth, but what we have is information, evidence, and theories. We live in a reality that includes powerful institutions that have a strong interest in what we accept as true. Private corporations and other business entities make their profits by selling products, and by avoiding government constraints, and they spend large sums on advertising and lobbying and the creation or manipulation of public opinion. Political parties and government itself are powerful institutions that have interests in public information and the formation of public opinion. Nonprofit institutions can be just as strong as business institutions, and have similar interests. Religious institutions have obvious interests in the formation of beliefs. Public opinion relating to economics, politics or religion are hot topics where strongly held opinions often lead to conflict.

Our lives are constrained by institutions that saturate world society, and the institutions in turn are constrained and interact with public opinion. Public opinion based on facts and evidence, rather than institutional interests, is necessary for wise and beneficial relationships among the public and the institutions we rely on. Science should contribute facts and evidence. Science might be presumed to be an neutral arbitrator, until it is realized that science, too, is a matter of institutions. The method of science is available to all of us individually, but the practice of science is complex, interpersonal (or inter-institutional), and expensive.

Discussing all this in the abstract has limited appeal. A case study on how science, institutions, and public opinion interact can shed more light on the situation than a purely theoretical discussion.

The case study presented here takes on a topic that is less personal and possibly less inflammatory than one directly involving politics, economics or religion, but which has the same kind of powerful institutional influence in the background. The case study is about the origins of the HIV/AIDS epidemic,[1] a subject of interest in itself, and which could have important consequences for the fight against the epidemic in Africa and other places where inadequate funding has been a constraint.

II. The origin of AIDS case study

This essay is an inquiry into the manufacture of certainty in the public discourse. The subject of the case study is the debate over the origin of the worldwide HIV/AIDS epidemic. The debate has raged unresolved since 1983 when the AIDS virus (actually a family of viruses called HIV for Human Immunodeficiency Virus) was first isolated and identified. Since the early 1990s the subject of debate has been whether the self-regulated medical research community in America and Europe (as it existed in the 1950s with continuity to the present day) was responsible for starting the epidemic.

This case study is of interest because of the following elements:

1. The debated issue can be stated clearly: Did the HIV/AIDS epidemic result from medical trials of a vaccine prepared using chimpanzee tissue cultures in the 1950s?

2. There are influential institutional interests involved in the outcome of the debate.

3. There is widespread public agreement that the debate has been resolved with a result favorable to the institutional interests.

4. The debate has not been resolved.

"Institutional interests" is an unwieldy term. The medical research community that is the subject of the debate is not a formal organization. Instead, it is made up of inter-related institutions that comprise a loosely related community of interests that would be affected by the outcome of the debate. For convenience the more concise term "medical industry" as defined below will be used for this essay.

For this essay the medical industry is defined to include government health agencies, research labs and regulators, public and private universities and research facilities, private for-profit and non-profit pharmaceutical and other medical supply and service providers, companies providing testing equipment, protocols and computer software, and

[1] Epidemiologists may prefer the term pandemic for a widespread epidemic like HIV/AIDS. The terms are often used interchangeably, as will be the case here.

journals that publish medical research. Entities in this community have shared interests, but also compete for funding, recognition, and primacy. Sources of funding are an important part of this community. Funding comes from government, from foundations, and from private corporations. While the boundaries are not well defined, it is clear that there are people who are within the community, and outsiders who have difficulty accessing this community. The community does not speak with a single voice.

For this discussion direct providers of medical services are not included within the definition of the medical industry.

Not all of the medical industry is involved in the debate, but the debate could have far-reaching implications that could affect the industry as a whole.

The medical industry has been very consistent, although not necessarily forthright, in denying possible responsibility for the HIV/AIDS epidemic. As discussed in more detail below, the industry has made a long series of claims of proof in its defense. While these claims of proof have helped create the public perception that the debate is over, the claims of proof have not held up under scrutiny. Given the strong institutional interest in a favorable resolution to the debate we may be assured that if firm proof existed, it would be produced. Such firm proof has not been produced, and the debate has not been resolved.

III. Background

The possible responsibility of the medical industry for starting the AIDS epidemic was first suggested by an article published in *Science* on November 22, 1985.[2] The article noted that primate kidney tissue cultures were used as growth mediums for the preparation of oral polio vaccines (OPVs), and that donor primates (African green monkeys were specifically mentioned) harbored Simian Immunodeficiency Viruses (SIVs) that could be the precursors of the AIDS viruses (HIVs). This possibility was taken up by several others, including Eva Lee Snead, Jennifer Alexander, Billi Goldberg, Mike Lecatsas, Blaine Elswood, Raphael Stricker, and preeminently, Louis Pascal. The hypothesis (or theory) was that medical research trials in the late 1950s in the Congo and other regions of Africa provided the mechanism by which SIVs spread to humans, adapted into HIVs and caused the AIDS epidemic. This theory was brought to public attention by an article in *Rolling Stone* in March 1992 by the writer Tom Curtis.

The reaction of the medical industry, and especially people and institutions directly involved with the medical trials conducted in Africa (and America) in the late 1950s (and early 1960s), has included very aggressive denial (including a lawsuit against Curtis and *Rolling Stone*, and threats of other lawsuits), a continuing series of published "final" proofs against the theory, denial of access to publication for critics rebutting those proofs, and funding and support for research defending the medical community.

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[2] Kanki PJ, Alroy J, Essex M, "Isolation of T-lymphtropic retrovirus related to HTLV-III/LAV from wild caught African green monkeys, *Science*, 1985 Nov 22;230(4728):951-54, PMID: 2997923.

It has become clear that the HIVs originated from Simian Immunodeficiency Viruses (SIVs) infecting chimpanzees (the various HIV-1 viruses) and African sooty mangabey monkeys (the various HIV-2 viruses). The question is how did the SIVs jump species to humans and evolve into the HIVs?

A little background is needed to understand the situation. The HIV/AIDS epidemic first came to attention in the 1981, and its cause was unknown at that time. In the U.S. members of the gay community, hemophiliacs, and others became sick from the new disease which suppressed victims' immune systems, leaving them vulnerable to a variety of opportunistic diseases. The virus that was making people in the U.S. fall ill has since been identified as a retrovirus labeled as HIV-1-M-B to distinguish it from other related strains of HIV. HIV-1 is the family of viruses that almost certainly evolved from SIVs infecting African chimpanzees. HIV-1-M identifies the main branch of this family that has been the predominant cause of the worldwide AIDS epidemic (or pandemic). HIV-1-M has many more or less related subtypes, A, B, C, D, and so on, nine or ten in all, depending on definitions. All of the HIVs are retroviruses that tend to change rapidly by recombination, as well as more slowly by mutation. This is one of the factors that has made AIDS hard to fight, and make it difficult to develop vaccines against the HIVs.

One illustrative open question in AIDS history is where did the HIV-1-M-B branch of the virus originate? The origins model supported by the medical industry holds that in the early part of the 20th century a native of what is now Cameroon picked up a chimpanzee SIV as a result of chimpanzees being hunted and consumed for food. A single early transfer from chimpanzee to human is used in that model to explain the branching of the HIV-1-M family over time to produce the array of related viruses found today. According to that model or theory, a second single transfer occurred to bring the HIV-1-M-B virus from Africa to Haiti around the early or mid 1960s, and a third single transfer brought the virus to the U.S. a few years after that, where it simmered for over a decade before being discovered.

This theory is known variously as the African hunter, the cut hunter, or the bushmeat theory of the origin of AIDS. The theory covers all the various HIV-1 and HIV-2 varieties and subtypes. In particular, the theory proposes that all HIV-1 varieties (including HIV-1-M, the cause of the worldwide AIDS pandemic; HIV-1-O, which seems to have infected some tens of thousands of people; HIV-1-N which has apparently infected six people; and HIV-1-P, which appears to have infected just two persons) each resulted from single transfers from chimpanzee to African hunter or bushmeat consumer. That theory also proposes that HIV-2-A and HIV-2-B, which have both caused tens of thousands of AIDS cases, mainly in West Africa, resulted from roughly contemporaneous transfers from African sooty mangabeys to different African hunters or consumers.

The HIV-1-M-B bushmeat theory has some obvious weaknesses, and others not so obvious. To an outsider, the series of single transfers, first from chimp to human, then to Haiti, and then to the U.S. seems like an unlikely reach. This is compounded by all those years of undetected simmering.

There is also the curious absence of the HIV-1-M-B subtype in African blood and tissue samples until 1997, when the so-called Euro-American strain could have been imported (or reimported).[3] (The medical industry has claimed that all of the HIV-1-M subtypes were originally present in Kinshasa, but the basis of this claim for subtype B is a single sample taken in 1997 in Bwamanda 435 miles up the Congo River from Kinshasa.)[4]

In response to these problems the medical industry published a paper in 2007 that reported in conclusion that it was 99.8% certain that HIV-1-M-B had entered the U.S. from Haiti in the 1960s. This paper, Gilbert et al., "The emergence of HIV/AIDS in the Americas and beyond,"[5] was based on HIV positive blood samples taken in 1982-83 from 5 Haitian immigrants who had arrived in the U.S. after 1975, but who, it was assumed, had contracted HIV-1-M-B in Haiti. (This assumption has been criticized by Dr. Paul Farmer of Harvard Medical School, who is well known for his work in Haiti, along with five coauthors who claim that the epidemiology of AIDS in Haiti refutes the claim that these five immigrants contracted the virus while in Haiti.[6])

Gilbert et al. compared the five HIV-1-M-B genetic sequences from the Haitian-Americans to a limited selection of other U.S. and European HIV-1-M-B existing sequences using several complex statistical software packages to conclude with a reported statistical significance of 99.8%, that the virus came to America through Haiti. Accordingly, the theoretical hypothesis is that a Haitian visiter to Zaire, possibly a medi-

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[3] In searching for evidence of early HIV-1-M-B in Africa only one study was found that reported finding a single "unusual B/D virus" from mid-1980s Zaire; it was apparently unclassifiable, possibly degraded, and only some gene fragments were analyzed, but which the authors say clustered with what they call subtype B/D. Subtype B/D is not defined, but appears to be a theoretical phylogenetic reconstruction. "Recombinant Viruses and Early Global HIV-1 Epidemic," Marcia L. Kalish, Kenneth E. Robbins, Danuta Pieniazek, Amanda Schaefer, Nzila Nzilambi, Thomas C. Quinn, Michael E. St. Louis, Ae S. Youngpairoj, Jonathan Phillips, Harold W. Jaffe, and Thomas M. Folks; full access at: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3323344/>. (Note: internet links are provided for convenience, but the actual links may change over time, and a name search may be necessary to reach the referenced sites.)

[4] "Unprecedented Degree of Human Immunodeficiency Virus Type 1 (HIV-1) Group M Genetic Diversity in the Democratic Republic of Congo Suggests that the HIV-1 Pandemic Originated in Central Africa," Nicole Vidal, Martine Peeters, Claire Mulanga-Kabeya, Nzila Nzilambi, David Robertson, Wantabala Ilunga, Hurogo Sema, Kazadi Tshimanga, Beni Bongo, and Eric Delaporte, J Virol. 2000 Nov; 74(22): 10498–10507; full access at: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC110924/>.

[5] M. Thomas P. Gilbert, Andrew Rambaut, Gabriela Wlasiuk, Thomas J. Spira, Arthur E. Pitchenik, and Michael Worobey. The paper can be accessed at: <http://www.pnas.org/content/104/47/18566.full>. The study used what is known as phylogenetic analysis to investigate the evolution of the HIV-1-M-B virus. Phylogenetic analysis makes assumptions about the rate of molecular evolution to construct trees showing the evolution of a genetic family over time. It is based on complex statistical analysis made possible by high speed computers. The analysis is also referred to as molecular clock analysis in this essay.

[6] "The epidemiology of AIDS in Haiti refutes the claims of Gilbert et al.," Jean William Pape, Paul Farmer, Serena Koenig, Daniel Fitzgerald, Peter Wright, and Warren Johnson. The paper can be accessed at: http://www.pnas.org/content/105/10/E13.full>.

cal technician there on a humanitarian mission, must have contracted the HIV-1-M-B virus in Africa in the 1960s.

This 2008 Haiti paper is central to the medical industry defense. If HIV-1-M-B cannot be traced to Africa there is a serious hole in the theory.

As discussed below eminent Stanford Professor John P. A. Ioannidis takes the position that unreasonably high reported statistical significance is a measure of bias rather than confirmation of the test results themselves. The Haiti paper would seem a candidate for that opprobrium.[7]

The non-obvious problems with the foregoing HIV-1-M-B theory involve the whole origins debate and are rather technical. According to the medical industry defense there have been other transfers of SIVs to humans in addition to the one that led to the HIV-1-M family. All of these transfers are proposed to have been caused by direct contact between humans and SIV-carrying primates. Some or most transfers don't become HIVs because the SIV cannot or does not adapt to the human host. Other transfers persist for a while, but then fade away without causing an epidemic. Two transfers of sooty mangabey SIV did persist to become HIV-2 Group A and HIV-2 Group B (two different strains of HIV-2), but at least 6 groups of HIV-2 apparently died out because these sooty mangabey SIV strains failed to adapt to humans after the initial transfer.

Two basic problems with the industry theory are that the locations of early AIDS cases do not match very well with the locations of the suspected SIV hosts, and that the close timing of the various proposed transfers is too coincidental to go unquestioned.

The debate between the medical industry and various outsiders simmered until 1999 when The River was published. The River is a carefully documented, detailed investigation into the origins of the HIV/AIDS epidemic. Its author, Edward Hooper, was and is an independent science writer and investigator without medical industry funding or preconceptions about the source of the epidemic. Hooper's conclusions gained the tentative support of some prominent scientists and intensified the debate. Hooper's views at that time are summarized by him in a Royal Society (London) publication that is available on the internet.[8]

Hooper's research findings presented at the The Accademia Nazionale dei Lincei conference on "Origin of HIV and Emerging Persistent Viruses" in Italy later in 2001, and

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[7] The reported statistical significance is a measure of the possibility that the result of the computer simulations was not a matter of mere chance. The 99.8% claim is very misleading. The result was not by chance, but the reported probability implicitly assumes that the analysis was based on strictly accurate data, including being certain that the 5 Haitian-Americans had contracted HIV-1-M-B in Haiti, that the molecular clock model and assumptions were correct, and that the statistical model was appropriate.

[8] Edward Hooper, “Experimental oral polio vaccines and acquired immune deficiency syndrome,” Philos Trans R Soc Lond B Biol Sci 2001 Jun 29 356(1410: 803-814, full text available at: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1088470/>.

published with further findings in 2003 as Edward Hooper, “Dephlogistication, Imperial Display, Apes, Angles, and the Return of Monsieur Emile Zola,”[9] and also the text of his remarks at that conference, both available on his website,[10] are recommended as a more detailed and updated exposition of his research.

The River was Hooper's second book about the AIDS epidemic, and he was among the first to write about the epidemic in Africa and to understand the implications of the early AIDS history in central Africa. He was also involved with the process of retesting the oldest HIV sample ever found, and, as a result was included as a coauthor with Bette Korber, Paul Sharp and 3 other prominent researchers of a paper on phylogenetic analysis using the 1959 sequence known as ZR59.[11]

The River garnered good reviews and award nominations for science writing. Hooper's study of the early epidemiology of AIDS strongly supported what British virologist and medical industry advocate Robin Weiss soon called an ugly theory: that the African trials of an experimental oral polio vaccine called CHAT, developed at the Philadelphia based Wistar Institute by Hilary Koprowski,[12] and administered to nearly 1 million subjects in Africa from 1957-1960, including infants with undeveloped immune systems, older children and adults, was the most likely source of the HIV-1-M family of AIDS viruses.[13]

Could the conclusion of The River be true? People and institutions connected to the African CHAT trials have been active in their denials. A series of proposed proofs defending the medical industry have been prominently published, and then refuted, mainly by Hooper. Some preserved CHAT was tested and came up negative for HIV, but that CHAT turned out not to be samples of CHAT as used in Africa. AIDS cases said to predate the vaccine trials turned out to be based on lab contaminations or otherwise found not to be AIDS. A chimpanzee compound at Camp Lindi in the Congo used for CHAT

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[9] Edward Hooper, Atti dei Convegni Lincei; 2003; 187; 27-230 (ISBN 88-218-0885-8).

[10] Edward Hooper’s AIDS Origins website: <www.aidsorigins.com>.

[11] An African HIV-1 sequence from 1959 and implications for the origin of the epidemic, Zhu, Korber, Nahmias, Hooper, Sharp, and Ho, *Nature*, 1998 Feb 5;391(6667):594-7.

[12] Another organization, Lederle Laboratories, then part of American Cyanamid employed the Polish-American virologist Koprowski before 1957. Oral polio vaccines called Fox and CHAT were originally developed at Lederle by Koprowski. The connection between the polio vaccines that Koprowski developed at Lederle and those he used in the Wistar trials is obscure, as is much of Koprowski's vaccine development. Lederle became part of the American Home Products conglomerate in 1994, which soon took on the Wyeth name from a subsidiary. Wyeth was acquired by Pfizer in 2009.

[13] Another Wistar OPV, Fox Type III, was also fed to thousands of African subjects. And smaller trials of Wistar OPVs were also conducted at times and places in the U.S. Partial records show 88 newborn children of inmates at Clinton Farms woman's prison being fed various Wistar OPVs, including CHAT, often in multiple doses, between 1955 and 1958. Other evidence shows that the Clinton Farms polio vaccine trials continued from 1955 to 1966, that other U.S. trials of up to 850 subjects were held in Philadelphia and New Jersey, and that the trials included adults, children, premature babies, and infants. The focus has been on the CHAT trials, but the other Wistar polio vaccine trials are not excluded from consideration.

experiments was said to hold the wrong subspecies of chimpanzees, but then Hooper showed that the experimental Camp Lindi chimpanzees came from several different parts of Africa, and included the subspecies found in Cameroon where the chimpanzee SIV strain that most closely matches HIV-1-M (thus far) has been found.

In 2008 genetic sequences from fragments of a newly discovered sample of early HIV-1-M, identified as DRC60, were analyzed and compared with the only other such early sequence, ZR59.[14] These sequences, from 1960 and 1959, are about 16 years older than any other HIV samples known to exist. Both were from the Leopoldville (now Kinshasa) capital city of what is now the Democratic Republic of the Congo. Molecular analysis of fragments from the two samples showed that each is within the HIV-1-M family of the virus responsible for most of the AIDS cases worldwide, but were sufficiently different from each other to suggest that either they had evolved from a common ancestor decades earlier, or that they were the result of a more recent punctuated event, such as might have occurred in batches of vaccine produced using chimpanzee tissue cultures. The medical industry seized upon the former possibility and gave prominence to a series of articles claiming dates of origin in the early 20th century.[15]

It is important to understand the possible role of a punctuated event in the debate over the origins of AIDS. The term was used at a conference on the origin of AIDS held in London by the Royal Society in 2000 by geneticists to describe a disruptive event in the evolution of HIV from SIV.[16] They proposed that a disruptive (punctuated) event could have caused the starburst of HIV-1-M subtypes to appear in the genetic history around 1960. A prime candidate for the punctuated event would be the African CHAT vaccination trials of 1957-60. The OPV theory proposes that the trials introduced 10 or more

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[14] "Direct Evidence of Extensive Diversity of HIV-1 in Kinshasa by 1960," Michael Worobey, Marlea Gemmel, Dirk E. Teuwen, Tamara Haselkorn, Kevin Kunstman, Michael Bunce, Jean-Jacques Muyembe, Jean-Marie M. Kabongo, Raphaël M. Kalengayi, Eric Van Marck, M. Thomas P. Gilbert, and Steven M. Wolinsky; Nature. 2008 Oct 2; 455(7213): 661–664; full access at: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3682493/>. Using the ZR59 and DRC60 gene fragments with other more recent selected genetic sequences, and the assumption of a reliable molecular clock, the authors estimate the time of the most recent common ancestor of HIV-1-M to be near the beginning of the 20th century with ca. 1908 apparently being their most likely estimate.

[15] A recent contribution to the series is Faria et al.: "The early spread and epidemic ignition of HIV-1 in human populations," Nuno R. Faria, Andrew Rambaut, Marc A. Suchard, Guy Baele, Trevor Bedford, Melissa J. Ward, Andrew J. Tatem, João D. Sousa, Nimalan Arinaminpathy, Jacques Pépin, David Posada, Martine Peeters, Oliver G. Pybus, and Philippe Lemey, *Science* 3 October 2014: 56-61. Faria et al. use somewhat different sets of gene sequences, and a somewhat different methodology (incorporating geography into the analysis) than Worobey et al., *supra* at footnote 14, and conclude that all HIV-1-M cases originated from a single individual in Leopoldville (Kinshasa) in ca. 1920. This is a more restricted proposition than found in Worobey et al. which could be seen to trace the pandemic back to a single zoonosis in Cameroon (or to a single individual elsewhere). Once a most recent common ancestor is reached it is not possible to extend the phylogenetic analysis of a data set further back.

[16] For the original Royal Society conference paper discussing the punctuated event, see: "The origin of acquired immune deficiency syndrome: Darwinian or Lamarckian?", by Burr T, Hyman JM and Myers G; Phil. Trans. R. Soc. Lond. B; 2001; 356; 877-887.

slight variations of chimpanzee SIV into the human population, resulting in what is now characterized as the ten subtypes of HIV-1-M.

This is the essence of the origin of AIDS debate as it stands today. However, that is not the public understanding of the situation. Popular writers and mass media have declared the debate over, and have claimed that the medical industry has been vindicated and absolved of responsibility. The creation of this public certainty is the subject of this case study.

IV. Uncertainty, Certainty, and Causation

A scientist or a statistician might say that nothing is absolutely certain, but what is analyzed in this essay is a relative certainty found in popular media and public discussion. Most people who have at least a passing interest in the subject have heard that the origins of AIDS debate has been resolved and that the resolution absolves the medical industry of responsibility. Not everyone agrees, but the dissenters don't have media access and have little incentive for voicing their views. In fact there are disincentives to opposing the industry position.

It should be understood that only a relatively small number of scientists have been directly involved in the debate. To contend, as has been contended in the media on behalf of the industry, that scientists agree that the debate has been resolved is a pointless expression where very few independent scientists have had any reason to study the issues and form a considered opinion. Scientific debates are not resolved by opinion polls, but rather by a contentious back and forth among specialists.[17]

The strange certainty over the AIDS origins debate could also be characterized as a soft certainty around the periphery of the debate that increases with distance from the actual terms of the debate. The certainty that the debate has been resolved shows up in editorial comments in science magazines, in popular science literature, on internet websites, and in the mass media.

With this public certainty in mind, it is necessary to establish that the debate has not been resolved. It is irresponsible to accept without question the industry's present position that genetic analysis proves that HIV predated the Wistar Institute's CHAT vaccine trials, especially given the medical industry's past history of flawed proclamations of final proof on this topic. Much of the following discussion addresses the inconclusiveness of the phylogenetic analyses published by the medical industry. This is not for the purpose of taking sides in the debate (although the industry is sure to find fault in any discussion that undermines the public closure of the debate). It is to establish that the debate has not been resolved, and to trace the creation of public certainty and investigate its causes.

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[17] One can see this process in the recent claims and retractions about the possible detection of gravity waves from the big bang where the scientists involved have been forthright about the uncertainties.

V. Evidence Gets Twisted

The ZR59 and DRC60 sequences are touted in defense of the medical industry's African hunter theory not merely as evidence, but as firm proof that the hunting and eating of wild chimpanzees caused an SIV to cross over to a human, survive and eventually mutate into the viruses identified as ZR59 and DRC60, and into the starburst of subtypes dating to around 1960 that became the HIV-1-M AIDS pandemic. According to medical industry defenders, the SIV to HIV transfer must have happened a significant number of years before 1959 in order for the original virus to mutate into ZR59 and DRC60. The CHAT trials involving those million African subjects, took place between 1957 and 1960. According to the industry hypothesis, HIV-1-M was already around by the time of the CHAT trials.

The genetic difference between ZR59 in 1959 and DC60 1960 suggests either an early date of origin or a near contemporary punctuated event. When the defenders go a step further and assign theoretical dates and time spans to when the SIV to HIV jump occurred they consistently ignore the alternative possibility. Complex, theoretical statistical analyses compare HIV sequences dating from 1985 and later, with genetic fragments from the two early samples, and the publication of the resulting specific dates without appropriate qualifications has resulted in positive publicity for the industry theory. [18]

At about the same time, other research in defense of the African hunter theory compared various HIV genetic sequences to SIV samples from various tribes of chimpanzees and concluded that it was chimpanzees in Cameroon, not those near the CHAT research sites, that were the most likely source of HIV-1-M (although there were still significant differences between the Cameroon SIV and the HIV-1-M family of viruses).[19] The findings of the phylogenetic research, and the studies of chimpanzee SIVs were published in prominent journals and promptly reported in the public media.

Meanwhile, in the years after publication of *The River*, Edward Hooper continued his research, with new findings and analysis published on his AIDS origins website.[20]

Hooper remains convinced that the early 20th century African hunter hypothesis as now formulated requires too unlikely a series of events and coincidences to be a viable alternative to the CHAT origin hypothesis. The African hunter hypothesis lacks a coherent

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[18] The 2008 Worobey et al. paper discussed at footnote 14, above, is the first to use the DRC60 sequence, but the phylogenetic studies using genetic clock theories to put dates to the origin of HIV have a longer history. HIV molecular clocks can be calibrated without the old sequences, but the problem is getting the calibration right and dealing with the theoretical issues posed by retroviruses and the possible intervention of punctuated events. Korber et al. (discussed below at footnote 31) is exceptional in its willingness to consider and discuss the possibility of a punctuated event. The Faria et al. paper (footnote 15) like most of its predecessors ignores this possibility, as does Worobey et al.

[19] This paper is discussed below at footnote 33.

[20] Hooper's site is an authoritative and well documented alternative to the medical industry point of view and is a convenient source for documents and information about the debate; <www.aidsorigins.com>.

explanation of how chimpanzee SIV in Cameroon around 1908 adapted to humans to become HIV-1-M, but failed to show up as AIDS until the late 1970s and early 1980s in the regions along the Congo River where the CHAT trials took place, far from Cameroon.[21] It also fails to explain why the mooted 1908 transfer developed into a starburst of subtypes dating from around 1959-60.

The industry's African hunter theory also leaves unexplained, except by coincidence, why the second strain, HIV-2, appeared in areas of West Africa during the same time frame as HIV-1, following trials in that region of alternative experimental polio vaccines conducted by other researchers (not from the Wistar).[22]

The genetic separation between the ZR59 and DRC60 virus fragments does not disprove the CHAT origins theory. Genetic analysis of the various subtypes of HIV-1-M (the main group) relate the starburst of subtypes back to around the time of the CHAT experiments. According to the CHAT hypothesis, the starburst, including ZR59 and DRC60 (which fall into different HIV-1-M subtypes), was the result of SIV/HIV evolution, adaption, recombination, or a combination of these, caused by the use of locally made chimpanzee tissue cultures to prepare the CHAT vaccine used in various experimental vaccination trials.

It is known that hundreds of chimpanzees from different parts of Africa were housed together and sacrificed for the CHAT experiments at Camp Lindi near the Stanleyville (now Kisangani) laboratory in central Congo. There is specific evidence of locally produced chimpanzee kidney tissue culture (CKTC) being used for vaccine preparation.[23]

The evidence for local CKTC use at the Stanleyville laboratory has been challenged by medical industry proponents, even to the point of denying that the lab was capable of producing tissue culture. However, in addition to eyewitness accounts, there is the fact that a Polish veterinarian, Alexandre Jezierski, was also doing polio vaccine research using local tissue cultures at an even more remote laboratory in the mountains of eastern Congo in the early 1950s. And local preparation was the routine practice for polio vaccine programs at that time.

The chimpanzee compound at Stanleyville and the subsequent CHAT trials offer a viable explanation (a punctuated event) for the starburst of HIV-1-M subtypes that include the ZR59 and DRC60 sequences. The appearance of the various subtypes could be explained by the co-caging of chimpanzees infected with different SIVs, which then recombined in vivo. Or it could be explained by the recombination of those SIVs in various batches of tissue culture, or it could be that once transferred to human subjects in

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[21] There is much speculation and hypothesizing about this could have happened, but without firm evidence for any of the competing theories.

[22] *The River*, (US 2000 paperback edition), revised postscript, pp. 827-877.

[23] Extensive discussion of the evidence about the use of chimpanzee kidneys for tissue cultures can be reviewed on Hooper's website (including shipment of chimpanzee kidneys to America and Europe).

the CHAT trials, the SIVs adapted differentially. Or it could have been some combination of the above.

Danish geneticists Mikkel Schierup and Roald Forsberg have analyzed the possible effect of recombination of divergent SIVs in human hosts. They concluded that if two different SIV sequences had been transmitted from chimpanzees to humans and subsequently recombined, then it would not be valid to use a phylogenetic method to date the MRCA event. Moreover, by ignoring the recombination, the bushmeat geneticists would tend to place the date of the viral crossover earlier in time than was warranted.[24]

In remarks at the Lincei Conference in 2001 Schierup concluded that if just two SIVs differing by about 5% to 10% (a relatively small difference) transferred to humans and recombined, this could be the basis for the range of HIV-1-M variants seen today.[25]

The starburst of HIV-1-M subtypes that appear around the time of the ZR59 and DRC60 sequences in 1959-60 do present problems for the medical industry theory of the African hunter in the early 20th century.

In an apparent triumph of circular reasoning, advocates for the medical industry make the crucial assumption that there was not a punctuated event at around the time of the CHAT experiments to upset a steady, clock-like mutation of the HIV virus. With that assumption the defenders place the most recent common ancestor (MRCA) of HIV-1-M at various dates ranging between 1894 and 1946, well before the mass testing of the experimental CHAT vaccine. Industry defenders then assert that the CHAT theory is refuted. Since the molecular clock theory only works if there was not an intervening punctuated event, this analysis simply assumes its conclusion.

But this is not the only problem with the African hunter theory. The molecular clock pushes the MRCA too far back in time for a comfortable theory of African hunter origins. As medical writer Jacques Pepin concluded, it requires a lot of bad luck to get from 1921 to the outbreak of the AIDS epidemic.[26] (Pepin uses the 1921 MRCA assumption; 1908 is even harder to justify.) Advocates offer no persuasive theory of how SIV evolved to survive in humans as HIV to start with, and the explanations of how a single HIV-1-M virus from Cameroon, which had not spread locally, allegedly travelled down to Leopoldville, and there differentiated into the HIV-1-M subtypes that are claimed to have traveled up the Congo River to Rwanda, Burundi, and the Ruzizi Valley, where the epidemic appeared many years later, are ad hoc without supporting evidence.

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[24] Atti dei Convegni Lincei; 2003; 187; 231-245, 243; full access to the paper is available at: www.bmartin.cc/dissent/documents/AIDS/Schierup03.pdf. (MRCA or TMRCA is a commonly used abbreviation for time of the most recent common ancestor. It is the theoretical date of origin of a genetic tree.)

[25] See Hooper, "Dephlogistication . . ." at page 172 of the Lincei papers, supra; or see p. 91 of the “Dephlogistication . . . “ article on Hooper’s website, <aidsorigins.com>.

[26] Pepin, *The Origin of AIDS*, Chapter 4. Pepin accepts that the required pieces of bad luck did occur.

All of this is to suggest that the medical industry has not proved its case.

Nevertheless, it is a medical industry talking point that the OPV/AIDS has been "firmly" rejected. This talking point shows up in newspaper stories, and in various Wikipedia pages and edits, and in recent mass market books, including Jacques Pepin, *The Origin of AIDS* (Cambridge University Press 2011), David Quammen, *Spillover* (Bodley Head 2012), and Craig Timberg and Daniel Halperin, *Tinderbox* (Penguin 2012).

The authors of these three books are the greatest interest here (See section VIII, below), but first let us take a look at Wikipedia as an example of the promotion of certainty.

VI. The Case of Wikipedia

Wikipedia, valuable and useful as it is, is not always a reliable source for information on controversial subjects as the public editing can result in manipulation of content.[27]

An investigation of the rates of change on controversial Wikipedia pages as a measure of material interest in the topics of controversy might be interesting. For the present purposes a brief look at the current page as of July 10, 2015 on the OPV/AIDS hypothesis (as amended through April 30, 2015) is sufficient. As of July 10, 2015[28] the two lead paragraphs of that page are as follows:

> **The oral polio vaccine (OPV) AIDS hypothesis** suggests that the AIDS pandemic originated from live polio vaccines prepared in rhesus macaque tissue cultures and then administered to up to one million Africans between 1957 and 1960 in experimental mass vaccination campaigns.[1][2]
>
> Data analyses in molecular biology and phylogenetic studies contradict the OPV AIDS hypothesis; consequently, scientific consensus regards the hypothesis as disproven.[3][4][5][6] The journal Nature has described the hypothesis as "refuted".[7]
>
> Footnotes:
> 1. Courtois G, Flack A, Jervis GA, Koprowski H, Ninane G (July 1958). "Preliminary report on mass vaccination of man with live attenuated poliomyelitis virus in the Belgian Congo and Ruanda-Urundi". Br Med J 2 (5090): 187–190. doi:10.1136/bmj.2.5090.187. PMC 2026116. PMID 13560820.

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[27] See, e.g., a recent article in the NY Times, “A pr firm alters the wiki reality of its star clients” at: <http://www.nytimes.com/2015/06/23/business/media/a-pr-firm-alters-the-wiki-reality-of-its-star-clients.html?hp&action=click&pgtype=Homepage&module=second-column-region&region=top-news&WT.nav=top-news>.

[28] The Wikipedia excerpt quoted here was rechecked July 10, 2015. The Wikipedia page was originally excerpted August 19, 2014, as updated August 10, 2014. The excerpt and footnotes 1-7 were unchanged over that period. Note that there are several other Wikipedia pages that discuss the origin of AIDS. The others take a similar pro-industry view; the analysis here is specific rather than comprehensive.

2 ^ LeBrun A, Cerf J, Gelfand HM, Courtois G, Plotkin SA, Koprowski H (1960). "Vaccination with the CHAT strain of type 1 attenuated poliomyelities virus in Leopoldville, Belgian Congo 1. Description of the city, its history of poliomyelitis, and the plan of the vaccination campaign" (PDF). Bull World Health Organ. 22 (3–4): 203–13.

3. ^ Hillis DM (2000). "AIDS. Origins of HIV". Science 288 (5472): 1757–1759. doi:10.1126/science.288.5472.1757. PMID 10877695.

4. ^ Birmingham K (2000). "Results make a monkey of OPV-AIDS theory". Nat Med 6 (10): 1067–1067. doi:10.1038/80356. PMID 11017114.

5. a b Cohen J (2001). "AIDS origins. Disputed AIDS theory dies its final death". Science 292 (5517): 615a–615. doi:10.1126/science.292.5517.615a. PMID 11330303.

6. ^ Origin of Human Immunodeficiency Virus (HIV/AIDS) Centers for Disease Control and Prevention website , Accessed 30th January 2007

7. a b Worobey M, Santiago M, Keele B, Ndjango J, Joy J, Labama B, Dhed'A B, Rambaut A, Sharp P, Shaw G, Hahn B (2004). "Origin of AIDS: contaminated polio vaccine theory refuted". Nature 428 (6985): 820–820. doi:10.1038/428820a. PMID 15103367.

Many factual errors are squeezed into these few words. The OPV/AIDS hypothesis suggests that batches of CHAT were prepared in Africa using chimpanzee tissue cultures (not rhesus macaque tissue cultures). This is a crucial misdirection as the use of chimpanzee tissue cultures is itself an alarm that would be recognized by readers.

The first two footnotes do not support the statement they are attached to, and therefore are false and misleading. Footnote 1 is to an article published in 1958 by the people who conducted the CHAT trials, led by Hilary Koprowski, which describes several mass vaccination trial that involved 215,504 children and adults in Aketi, Banalia, Stanleyville, and the Ruzizi Valley in 1958. It includes no information about either the OPV/AIDS hypothesis or the tissue cultures used to prepare the CHAT vaccines. It says only that Rhesus monkeys and chimpanzees were used to test the vaccine prior to administration to the human subjects. Similarly, Footnote 2 cites a 1960 report by essentially the same team on a 1958-60 vaccination program in Leopoldville (now Kinshasa in the DRC), and it also contains no information about either the OPV/AIDS hypothesis or the tissue cultures used to prepare the CHAT vaccines.[29]

The first sentence of the second Wikipedia paragraph makes two claims. The first claim is that the OPV/AIDS hypothesis is contradicted by data analysis and phylogenetic studies. The second claim is that scientific consensus regards the hypothesis as disproven. Footnotes 3-5 refer to three essays that are not data analysis or phylogenetic studies,

[29] The two articles do provide information on the dates, locations, and numbers involved in some of the trials, over which there is no controversy.

but instead are opinion pieces about other people's work. All three essays were published in 2000 or 2001. Footnote 3 is the most interesting and will be discussed further. Footnotes 4 and 5 refer to opinion pieces based on work that has since been rebutted. Footnote 6 cites to a Centers for Disease Control Vaccine Safety site as of January 30, 2007. A search of that vast site did not find information in support of the proposition about the OPV/AIDS debate to which it is attached. The CDC has recently improved the accessibility of its website. The updated site contains this statement about the origin of AIDS: "Studies show that HIV may have jumped from apes to humans as far back as the late 1880s." This is not a strong statement from an institution that may have an interest in the outcome of the debate, and does not assert a firm conclusion.[30]

Footnote 3 refers to an opinion piece in *Science* that exemplifies just what this essay is studying. The opinion piece takes the findings of a scientific research paper and expands them as scientific fact. The actual research paper is not cited as a Wikipedia source for the statement at footnote 3, which is curious because the actual paper is high quality research, well written, and is perhaps the best of the series that supports the medical industry position. The actual paper (Korber, et al.[31]) assumes a uniform rate of evolution and a steady molecular clock to place the MRCA of HIV-1 at around 1931. The authors give reasons for why they doubt that a punctuated event occurred as a result of the CHAT trials to interrupt the molecular clock, and give reasons to support their hypothesis that it is unlikely that those trials were the source of HIV-1 in humans.

Why cite to secondary sources when the primary source is equally available? Perhaps it is because the secondary sources make claims of finality that are not found in the original paper.

The authors of the original paper state their assumptions. They tested their model against the ZR59 sequence, treating the ZR59 date of origin (1959) as the unknown, and the model predicted that it was from 1957 with a 95% confidence range of 1934-1962. They ran several other such tests of their model and they discuss the many variations of the model that they ran, and some of the difficulties involved.

The authors confront the fact that their analysis did not specifically address the question of whether the MRCA occurred before or after transmission to humans, i.e., the research, strictly speaking, did not set bounds on the date or dates of actual zoonosis.

For the CHAT hypothesis to be consistent with their research the authors conclude that at least nine genetically distinct viruses would need to have been transmitted by the vaccine. The authors note that so far as was known at the time SIV prevalence in

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[30] The CDC statement is posted at: <www.cdc.gov/hiv/basics/whatishiv.html>.

[31] "Timing the ancestor of the HIV-1 pandemic strains," Korber B, Muldoon M, Theiler J, Gao F, Gupta R, Lapedes A, Hahn BH, Wolinsky S, Bhattacharya T., Science, 2000 Jun 9;288(5472):1789-96; available at <http://www.ncbi.nlm.nih.gov/pubmed/10846155>. Wikipedia does cite to this paper later on its page among other papers dating "the introduction of HIV-1 to humans as occurring between 1915 and 1941."

chimpanzees was only about 1% (based on a limited sample of captive chimpanzees), that not many kidneys were needed for vaccine production, and that most of the chimpanzees used in the CHAT program were juveniles unlikely to have developed SIV infections.[32] Given these considerations they concluded that the vaccine trials would be an unlikely source of the HIV-1 transmission to humans.

The authors also discuss hurdles to successful SIV transfer to a new species that might occur during initial infection and onward transmission. If HIV-1 did enter the human population around 1931, then for many years the virus must have gone undetected while it diversified (and, as the authors neglect to mention, traveled across Africa to the sites of the CHAT trials where it was first detected). The authors implicitly recognize that this, too, could be considered unlikely. They conclude that given conditions in rural Africa during those years it is feasible that small numbers of HIV-1 infections could go undetected, and that colonial practices (in French Equatorial Africa) provided an opportunity for the subsequent expansion of the virus.

Citation to the actual paper (Korber, et al.) would not be useful if the purpose is to suppress the origins debate, because the paper is balanced and nuanced. The authors find that on the one hand, the CHAT theory is possible, but seems unlikely. On the other hand, the medical industry theory has hurdles and is just feasible. The statement could be reversed without changing its content: the industry theory is unlikely while the CHAT theory is just feasible. If there were chimpanzee SIV in some of the million doses fed in the CHAT trials, then multiple SIV transfers could feasibly have adapted and survived in humans to be passed forward as differentiated HIV-1-M subtypes.

The paper predates the sequencing of the DRC60 sample and comparison with ZR59 in 2008, as discussed above, which appears to push the molecular clock calculation another 25 years further from the onset of the AIDS epidemic (further reducing the feasibility of the virus remaining undetected for those many years), and the paper was written prior to research on wild chimpanzees that revealed much higher SIV incidence, typically around 14%, rather than 1%. Juvenile chimpanzees are now known to acquire SIV from their mothers, and fresh kidneys would have been required for the preparation of each new new batch of vaccine. This new information changes the weighting that should be given to the alternative theories; the feasibility of the industry theory is reduced, and the likelihood of the CHAT vaccine theory is enhanced.

Nevertheless, the paper remains a good summary of how the debate stands today: an unlikely series of events caused the AIDS epidemic. Whether it was through the CHAT trials in the late 1950s or through an unfortunate series of events beginning around 1908 has not been determined.

Wikipedia's claim of "scientific consensus" is empty, unsupported, and meaningless.

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[32] As discussed below, subsequent evidence, available from many sources on the internet using a search engine, has invalidated these assumptions about chimpanzee SIVs.

The third sentence of the Wikipedia excerpt (see above) claims that the journal *Nature* has described the OPV/AIDS hypothesis as refuted. One has only to follow the footnote, Footnote 7, to see that what *Nature* did was publish an article that makes that claim in its title. The abstract for that article, published in 2004, is the following:

> *Nature*. 2004 Apr 22;428(6985):820.
>
> **Origin of AIDS: contaminated polio vaccine theory refuted.** Worobey M1, Santiago ML, Keele BF, Ndjango JB, Joy JB, Labama BL, Dhed'A BD, Rambaut A, Sharp PM, Shaw GM, Hahn BH.
>
> Abstract
>
> Despite strong evidence to the contrary, speculation continues that the AIDS virus, human immunodeficiency virus type 1 (HIV-1), may have crossed into humans as a result of contamination of the oral polio vaccine (OPV). This 'OPV/AIDS theory' claims that chimpanzees from the vicinity of Stanleyville--now Kisangani in the Democratic Republic of Congo--were the source of a simian immunodeficiency virus (SIVcpz) that was transmitted to humans when chimpanzee tissues were allegedly used in the preparation of OPV. Here we show that SIVcpz is indeed endemic in wild chimpanzees of this region but that the circulating virus is phylogenetically distinct from all strains of HIV-1, providing direct evidence that these chimpanzees were not the source of the human AIDS pandemic.
>
> PMID: 15103367 [PubMed - indexed for MEDLINE][33]

The abstract starts with an inflammatory and false use of the modifier "the" in "the oral polio vaccine" rather than merely "an oral polio vaccine," or more accurately "an experimental oral polio vaccine." The medical industry has at times willfully suggested that for anyone to claim that a vaccine has been contaminated is to undermine vaccine campaigns generally, and has impugned the motives of outsiders who want to investigate questionable vaccine research practices and history.[34] One suspects that the use of the wrong modifier is not accidental.

There is a word for turning an issue around to gain rhetorical advantage. It is called spin. It is important to un-spin the debate. It is not about polio or about polio vaccine. The defenders of the medical industry would prefer to be seen as defenders of vaccination in general rather than as defenders of their prerogatives of independence and self-

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[33] The PMID number can be used for accessing the original abstract via the internet.

[34] My children get their recommended vaccinations. I hope the readers' children do, too (allowing for exceptions). Edward Hooper is also on record for his support of vaccination. The safety of present vaccines is not at issue in this discussion. This does not mean that the powerful vaccine industry should be immune from oversight.

regulation. There was a recklessness about many vaccine experiments in the 1950s. People were experimented on without their informed consent. Risks were taken affecting all of us without our consent. Risks were driven by the desire to be first, to win the prize, to make the fortune, rather than to serve humanity. The debate is about the possible consequences of something that was in some respects ugly.

A needed debate about oversight and practices of the medical industry is curtailed by failure to acknowledge the past and the possible consequences of past practices. Suppression of the origin of AIDS debate helps to suppress discussion about regulation and outside oversight of the medical industry. This is one of the institutional interests referred to in the introduction. The medical industry appears to fear the debate itself, beyond any regard for its possible outcome.

Getting back to Wikipedia, note that while a "final death" of the OPV theory is claimed by Wikipedia Footnote 5, with a publication date of 2001, a new refutation is claimed in 2004, and the earlier claims are only "strong evidence" by the time of the 2004 abstract cited at Wikipedia Footnote 7.

The *Nature* abstract quoted in full above continues with a false assertion about the basis of OPV/AIDS theory. The theory refers not to local wild chimpanzees near Stanleyville/Kisangani, but rather to a chimpanzee compound at a place called Camp Lindi 10 miles outside Stanleyville where more than 400 chimpanzees were housed and sacrificed for the CHAT vaccine experiments in the Congo. As Hooper has demonstrated, the chimpanzees were brought there from all over central Africa.[35]

The OPV/AIDS theory hypothesizes that HIV-1-M came out of the Camp Lindi chimpanzee compound through the use of chimpanzee kidney tissue cultures used to prepare batches of CHAT vaccine for the Congo vaccination trials.[36]

The finding that SIV was endemic among the wild chimpanzees found near Stanleyville (now Kisangani, DRC) is interesting. Some of those local chimpanzees would have been in the mix at Camp Lindi, possibly adding to the genetic variations of SIV available for recombination, but these were not the the only chimpanzees involved at Camp Lindi. This brief paper is, therefore, not a refutation of OPV/AIDS theory.

It is worth noting that 2 years after the article was published the last-named author, Beatrice Hahn (also a joint author of the 2000 Korber, et al., paper), was quoted by the

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[35] See "The Hollywooding of Science" at <aidsorigins.com>. (Reference [4], M.M. Vastesaeger et al., “L’atherosclerose experimentale du chimpanze. Recherches preliminaires”; Acta Cardiol.; 1965; Supp. 11; 283-297, evidences the presence of the non-local chimpanzee ptt subspecies at the Lindi camp.)

[36] It may also turn out to be relevant that scientists working on CHAT were also working on human cell line tissue cultures, and also that the robust and invasive HeLa cell line was available to the Stanleyville laboratory by 1954 and known to be present there by 1959. Human amniotic cells were also available for experimental tissue cultures. Investigation of the possibility of cross contamination of human and chimpanzee cell cultures might be warranted if the evolutionary path of SIV to HIV remains unsolved.

pbs.org science writer Mary Carmichael as admitting that to truly disprove OPV/AIDS theory researchers would have to find HIV in tissue samples that predate the African CHAT trials.[37] (No such samples have ever been found.)

The cited article is one of a continuing progression of claimed refutations of OPV/AIDS theory that have turned out on examination to be false. For example, as far back as 1992 the Wistar Institute (the Philadelphia institution behind the CHAT trials in the Congo) claimed that David Carr of Manchester, England had died of AIDS in 1959, and on the assumption that he must have acquired the virus some years earlier, prior to the CHAT trials, proclaimed, "Therefore, it can be stated with almost complete certainty that the large polio vaccine trial begun in 1957 in the Congo was not the origin of AIDS."[38]

It subsequently turned out that David Carr never had AIDS, and that the initial lab test mixed his sample with that of another person who had acquired the virus much later.[39] Nevertheless, Wikipedia continues to feature the 1992 report based on David Carr, including the above quotation.

Some years later it was claimed that scientists had tested a preserved sample of CHAT for HIV and for chimpanzee cells and that the tests were negative. This finding was proclaimed at a London Royal Society conference on the origin of AIDS in 2000 as final proof against the CHAT hypothesis, and published in the proceedings the next year. But this also turned out to be false. No laboratory has ever tested any OPV batches that have been prepared and used in Africa, or at least no one has ever reported the results of any such tests.

(Hilary Koprowski also claimed that records that might shed some light on his methods of OPV production had been lost. When Hooper mentioned this in an interview with a famous British scientist who had been involved with polio vaccine production, the scientist "roared with laughter".[40])

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[37] Mary Carmichael also reports that, “Proponents of each theory have acknowledged (albeit grudgingly) that the other is scientifically possible.” “How it Began: HIV before the Age of AIDS,” (2006) full access at: <http://www.pbs.org/wgbh/pages/frontline/aids/virus/origins.html>.

[38] Report from the AIDS/Poliovirus Advisory Committee, Claudio Basilico, et al., Wistar Institute independent panel, September 18, 1992. This report is available at: <http://www.bmartin.cc/dissent/documents/AIDS/Wistar92.html>.

[39] Zhu and Ho, “Was HIV present in 1959?” *Nature;* Apr 6, 1995; 374, 6522; ProQuest Health & Medical Complete pg. 503. See, also, *The River*, 1999, pp. 601-603. HIV experts David Ho (with colleague Tuofu Zhu) and Gerald Myers established by 1995 that David Carr did not have AIDS. A good summary of their discovery, by journalist David Connor, is available at: <http://www.independent.co.uk/news/uk/how-scientists-discovered-false-evidence-on-the-worlds-first-aids-victim-1612471.html>.

[40] *The River*, 1999, at p. 806, see also pp. 462-463.

The Wikipedia page goes on to make more factual misrepresentations, and even stoops to rehash the reprehensible claims that to even consider the OPV/AIDS hypothesis undermines ongoing polio vaccination campaigns, and that only "the hardened conspiracy theorist" would doubt the industry theory and defense after 2001.

One may argue that this is merely about a Wikipedia page. Deconstructing the whole obfuscation of the origin of AIDS debate on Wikipedia is not the focus of this analysis. But there is a lesson here. Someone has sufficient interest in suppressing the origins debate to actively monitor and edit Wikipedia (e.g., August 10, 2014 and as recently as April 30, 2015) without correcting pro-industry errors. Actually there are some very strong interests at play in the debate. Among them are those responsible for the conduct of the Congo CHAT trials, and institutions that might have or fear consequential financial liabilities, including powerful government agencies. There are also those who perceive any outside criticism of the medical industry as an existential threat.

VII. Sociologist Brian Martin on the Suppression of Dissent

Brian Martin, a sociology professor at the University of Wollongong, Australia, has been studying the suppression of dissent since the late 1970s. He has published extensively on the subject. One recent publication is, "How to attack a scientific theory and get away with it (usually): the attempt to destroy an origin-of-AIDS hypothesis."[41] Martin provides a useful summary history of the decades long debate over whether the medical industry vaccination trials in Africa were the origin of the AIDS epidemic. He uses a framework developed for analyzing techniques used by perpetrators of injustice to inhibit outrage at their actions. The techniques fall into five categories: coverup, devaluation, reinterpretation, official channels, and intimidation. His paper is a case study applying the framework to a scientific debate. In his conclusion Martin states:

> Opponents of the OPV theory have used a wide variety of techniques: they have blocked publication in peer-reviewed journals, delayed releasing polio vaccine samples for many years, disparaged the theory (mostly behind the scenes), put the onus of proof on OPV-theory proponents, claimed repeatedly that the OPV theory has been refuted, used an expert committee and scientific conferences to claim authoritative rejection of the theory, and used legal actions to discourage publications about the theory. These techniques fit within the five types of methods commonly used by perpetrators to reduce public outrage from a perceived injustice or norm violation. . . .
>
> In many public policy disputes, scientific theories are attacked as part of a wider struggle involving politics, economics and ethics. In such situations, it is naive to assume that scientific theories will be evaluated neutrally and fairly. Being pre-

[41] Brian Martin, *Science as Culture*, Vol. 19, No. 2, June 2010, pp. 215-239.

pared means that the attackers won't always get away with it. (Emphasis added.)[42]

VIII. Three Mass Market Books

Naivety enters the picture with the publication of the books by Jacques Pepin, by David Quammen, and by Craig Timberg and Daniel Halperin (T&H). (These are the three books listed at the end of Section V, above.) These three books reduce scientific debate to one-sided certainty. These three books both reflect and contribute to the public certainty that the origin of AIDS debate has been resolved in favor of the medical industry.

There are at least two other similar books (by Nicoli Nattross and by Gareth Williams) published within the same timeframe that promote the same false certainties. The three books by Pepin, Quammen, and T&H are singled out here because of their prominence and commercial success, and because the problem with these books is representative of the overall media coverage of the issue.

One thing that has made the three books successful is their sense of resolution and finality. Each author takes the position that the debate over the origin of AIDS has been firmly resolved and the medical industry absolved of responsibility. They naively assume that the analysis presented to them by the industry defenders has been fairly and neutrally evaluated. They did not look deeply into the defensive claims or the uncertainty involved, and they ignored opposition research. I have attempted to contact the authors with questions. I also asked Edward Hooper if any of them had contacted him. Hooper says that none of the four writers ever tried to contact him, although they refer to him and his research in their books.

Each of the authors would probably assert that they were justified in relying on papers published in scientific journals like *Nature* and *Science*, that they had no cause to look further. But, in fact, it is clear that they did go further; they had personal contacts and support from one side of the debate, the side representing the medical industry and the people with a stake in the outcome of the debate. They looked further, but only on one side of the question.

The authors have clearly benefitted from their one-sided proclamations, and they have served the interests of those interested in suppressing the debate. They were willing to go one step further than established scientists have gone. The authors proclaimed certainty, and they devised fictional representations of the, in reality, uncertain series of circumstances and events implied by the medical industry's origin of AIDS theory.

Were these authors justified in converting published research findings into certainties? Pepin and T&H did not respond to my inquiries. Here is what T&H say in their book:

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[42] Martin, *id.*, available at <www.bmartin.cc/pubs/10sac.html>.

> We now know where the epidemic began: a small patch of remote southeastern Cameroon. We know when: within a couple decades either side of 1900. We have a good idea of how: someone caught an infected chimpanzee for food, allowing the virus to pass from the chimp's blood into the hunter's body, probably through a cut during the butchering.....It was here, in a single moment of transmission from chimp to human that a strain of virus called HIV-1 group M first appeared.[43]

This statement is not just untrue (we don't know those things), but biologically impossible. HIV did not appear in a single moment of transmission. SIVs changed and adapted to humans to become HIVs.[44] The process of adaptation has not been sufficiently explored or analyzed, but, given the genetic difference between all known SIVs and every HIV-1-M the process could not have been instantaneous.

T&H worked closely with the medical industry defenders, and specifically thanked Beatrice Hahn and Michael Worobey (researchers closely associated with the African hunter–early 20th century theory) for "vital perspectives." Worobey and Hahn must know, as Hahn herself acknowledged in 2006, that the origins debate has not been truly resolved. One wonders, then, why T&H were allowed to naively persist with their level of certainty, especially glossing over major problems within the African hunter hypothesis.

Jacques Pepin, in his book, asserts that, "there is now overwhelming evidence that it (OPV/AIDS) did not happen," and that such theories "can be firmly rejected."[45]

Pepin is a medical doctor and educator, but not an expert in molecular genetics, microbiology, virology or phylogenetics. His opinion that the evidence is overwhelming is not an independent expert evaluation. He relies on the papers and assertions of his contacts in the medical industry, some of whom were connected with the CHAT trials.

David Quammen in a recent column in the New York Times stated a view that is also found in his book, *Spillover*:

> We now know from molecular evidence (published by Beatrice H. Hahn, Michael Worobey and their collaborators) that the pandemic strain of H.I.V. went from a single chimpanzee into a single person (presumably by blood-to-blood contact when the chimp was slaughtered for food) around 1908 or earlier, in southeastern Cameroon. The virus then must have passed slowly downriver, human to

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[43] Timberg and Halperin, *Tinderbox*, pp. 2-3.

[44] See, e.g., “Adaptation of HIV-1 to Its Human Host,” Wain LV, Bailes E, Bibollet-Ruche F, Decker JM, Keele BF, Van Heuverswyn F, Li Y, Takehisa J, Ngole EM, Shaw GM, Peeters M, Hahn BH, Sharp PM., Mol Biol Evol., Aug 24(8):1853-60. Epub 2007 Jun 1; <http://www.ncbi.nlm.nih.gov/pubmed/17545188>.

[45] Pepin, *The Origin of* AIDS*,* pp. 50-53.

> human, into the large population centers of the Congo basin before spreading worldwide.[46]

In Spillover Quammen goes further to create a long fiction of a hunter in Cameroon who is infected with chimpanzee SIV through a cut, and who then passes it on to a sex partner before the hunter is gored by an elephant. There follows sexual transmission to someone called The Voyager, the theft of two elephant tusks, a trip down the Sangha River, a murder, the disposal of the tusks, and finally a virus that crosses the Congo River from Brazzaville to Leopoldville. It is all rather embarrassing from an otherwise serious writer, and seemingly out of character for Quammen.

I sent Quammen a lengthy email saying that I was writing this essay on speculative science and popular certainty with a series of detailed questions about how he came to his conclusion that the CHAT theory of the origin of AIDS had been "directly refuted." He had the courtesy to reply as follows (quoted in its entirety):

> Thanks for your interesting message. I'm glad that you're interested in the relationship between refereed science, popular science writing, and public opinion. Very tangled, I agree. Witness the case of new discoveries in molecular biology about horizontal gene transfer, their implications for challenging the classic Darwinian idea of a unitary Tree of Life, and the confused abuse of that debate by Creationists who thrill at news that some evolutionary biologist has said that Darwin was wrong about SOMETHING.
>
> Yes, I've read a lot of that other AIDS/OPV literature to which you allude. All of it? No. Your email implies that, in order to have an opinion on Hooper's (and others') OPV theory, I'd be obliged to contact him after reading through his book. Not true. Having read his entire 1070-page opus, I'm entitled to the presumption that I know his argument. There are better ones. Have a good day.

David Quammen, email May 14, 2014.

I will discuss this response in detail since I believe it proves an important point. The first paragraph may seem tangental at first, but conflating and comparing "refereed science, popular science writing, and public opinion" with Creationist confusion is to suggest a parallel to the origins debate and the medical industry's defense. This is not likely to be unintentional from an experienced and careful writer. The message is that research published in science journals trumps outsider investigations and theories, just as evolution trumps creationism.

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[46] David Quammen, “Disease, the Next Big One,” October 15, 2013: <http://www.nytimes.com/2013/10/15/opinion/disease-the-next-big-one.html?pagewanted=all>. Note that Quammen has taken Worobey et al.’s mid-range estimate of 1908 and converted it into “around 1908 or earlier,” and poses this as a known fact. The entire statement recites hypotheses, not known facts.

Directly confronted, Quammen's analogy does not hold up. The theory that the medical industry was responsible for the origin of AIDS is not like creationism. The OPV/AIDS theory is based on an analysis of facts, not on an a priori system of belief. It can be confirmed or refuted by facts.

One of my unanswered questions to Quammen was whether any particular fact if proven would give him second thoughts about the debate being resolved.

In his second paragraph Quammen does not address my specific questions, but responds instead to inferences that he draws from them, or, perhaps to be more accurate, to inferences he draws just from the fact that I have asked him questions about his sources and contacts (on both sides of the issue). For the record, I do not agree that anyone in a scientific debate is entitled to presumptions.

What Quammen confirms in his email is that he has an opinion on the HIV/AIDS origins debate, and that his writing is based on his opinion that among the various origins arguments, the medical industry has the "better ones." He is entitled to his opinion, but his book and his New York Times piece convert his opinion into certainty, into fact.

This is no doubt the case for the other authors as well. They have formed an opinion favorable to the medical industry. From their opinions they jump to expressions of certainty. Their certainty is not the result of careful independent expert evaluation of the evidence, because they are not experts. Their certainty must come from something else. For Quammen it seems to come from faith in refereed science, and, perhaps, although he does not answer my questions about it, from off-the-record contacts and representations from within the interested medical industry, such as is acknowledged by T&H, and as is likely in the case of Pepin.

There is a vast gulf between opinion and certainty. Opinion is open to debate, but certainty closes off debate. Surely these authors know this difference. Papers published in scientific journals (including *Science* and *Nature* where much of the medical industry's contentions about the debate have been published, and where many publication decisions are made by editors without referees) make claims, but those claims are qualified by probabilities and assumptions. There is a myth that refereed papers have passed some kind of general truth test. The referee process includes some assurance of technical competence, but not certainty of results.

Science and *Nature* have come under severe criticism. University of California, Berkeley cell biologist Randy Schekman used the occasion of his Nobel Prize award in 2013 to announce that his lab would no longer submit papers to *Science*, *Nature*, or *Cell* for publication. He gave reasons: the editors are not working scientists, they have a preference for headlines over solid work, they artificially restrict the number of papers they accept, they look for impact factors and introduce distortion. He deemed the journals a toxic influence that damages science. "While they publish many outstanding pa-

pers, they do not publish only outstanding papers. . . . [T]hey accept papers that will make waves because they explore sexy subjects or make challenging claims."[47]

IX. Ioannidis: Most Published Research Findings Are False

Stanford University Professor of Medicine, Health Research and Policy, and, by courtesy, of Statistics, John P. A. Ioannidis has also been critical of *Nature* and *Science.*

Science does not normally proceed in an orderly fashion from truth to truth to truth. Better to say it advances from error to correction to correction. The recent proposed measurement of gravity waves supported one model of inflation and the big bang, but would prove others false (and has yet to be confirmed). The recent discovery of the Higgs particle added to knowledge of quantum physics by narrowing and winnowing the range of scientific theories that remain viable. The falsification of published research is especially relevant to empirical work that relies on statistical analysis, including molecular biology and phylogenetics.

Louis Pascal is credited with formulating the first comprehensive OPV (CHAT) theory of the origin of AIDS in 1987. His follow-up paper "What Happens When Science Goes Bad, The Corruption of Science and the Origin of AIDS: A Study in Spontaneous Generation" was published as University of Wollongong Science and Technology Analysis Research Program Working Paper No. 9, December 1991. The full text is available on line.[48] The first section of Pascal's paper is a discussion of the history of a cell culture called HeLa.

HeLa was the first successful long-term human cell culture. The cell line was created in 1951 from human cervical cancer cells (taken and used without informed consent) from a woman named Henrietta Lacks. HeLa was very robust and became widely used for research. More widely used, it turned out, than was recognized at the time. Pedigreed cell lines from around the world were contaminated and taken over by HeLa. The result was that uncounted scores of research findings based on what were represented to be other kinds of cells were in fact false.

> 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 con-

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[47] Randy Schekman, "How journals like Nature, Cell and Science are damaging science," *The Guardian*, December 9, 2013.

[48] The Louis Pascal paper on line: <http://www.bmartin.cc/dissent/documents/AIDS/Pascal91.html>.

> nected 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.[49]

Reports of research findings subsequently found to be false are not uncommon. What Pascal particularly deplored about the HeLa debacle was the response of the scientific community to the discovery of the widespread contamination that falsified a very large body of research: denial and coverup. Many important HeLa disclosures made in 1966 by Stanley Gartler were carried forward by the head of a cell bank at the University of California, Walter Nelson-Rees. (It was Nelson-Rees who made the discovery of the HeLa contaminations in the cultures from the Soviet Union.) Pascal reports that because of his efforts Nelson-Rees had his funding cut off and he was forced into early retirement in 1981 at the age of 52. One supplier of biological products continued to sell a HeLa-contaminated culture for 13 years after being told of the contamination. It appears that many discredited papers were not withdrawn.

Published research findings that turn out to be false are a common occurrence in science publishing, sometimes with serious consequences and resistance to correction. A few examples are discussed below.

A story in the New York Times provides one example. Renal denervation treatment for extreme hypertension had been widely approved in Europe and other countries (not including the US) based on published case studies and research. However, a new study published in the prestigious *New England Journal of Medicine* using double blind controls has debunked the previous studies. Renal denervation had no greater effect than a placebo (sham treatment).[50]

False research findings played a major role supporting economic policies of austerity that have cost the world economy trillions of dollars of lost output since the Great Recession of 2008.[51] Research published in 2010 by Carman M. Reinhart and Kenneth S. Rogoff, professors of economics at Harvard University, claimed to find that government debt exceeding 90% of national output exerted a severe drag on economic growth. These findings were seized upon by politicians seeking justification for pursuing debt reduction (for their own usually unstated political reasons) rather than economic stimulus. But then a graduate student, Thomas Herndon, discovered that it was only due to data and design errors that Reinhart and Rogoff found their severe relationship of debt

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[49] Pascal, *id.*, at Part I.

[50] The March 30, 2014 New York Times article can be viewed at: <http://www.nytimes.com/2014/03/30/health/setback-for-promising-high-blood-pressure-treatment.html?hp>.

[51] Political leaders in Europe, often supported by a majority of voters, continue to believe with strange certainty that austerity in depressed economies will stimulate growth and relieve debt.

and growth (which in fact was never shown to be causal, as many economists pointed out at the time of publication).[52]

An article in The Guardian exposed false research findings prominently published in the leading psychology journal, *American Psychologist*. The error in this case was in the use of complex mathematics not properly understood by the authors, editors, reviewers, or by the authors of over 350 articles citing it. Again, the error was discovered by a graduate student.[53]

It is worth looking a little deeper into the popularity of this psychology article ("Positive Affect and the Complex Dynamics of Human Flourishing" by Fredrickson and Losada, October 2005[54]) and its false research. It offered support to a lucrative area of psychology supported by major academic figures promoting "positive psychology". An earlier, rather mild criticism of the paper submitted to *American Psychologist* was declined for publication.

When *American Psychologist* finally published the debunking by grad student Nick Brown, joined by two psychology professors, it gave Fredrickson the last word where she accepted the errors, admitting that she never understood her co-author's mathematics, but put a positive spin on her research claiming that empirical evidence strongly supported her case. When Brown et al. tried to respond to Fredrickson's further statement they were turned down by *American Psychologist*. Brown et al. appealed, and their rebuttal was finally published in September 2014, nine years after the original paper was published.[55]

The *Guardian* coverage also recalls the case of Diederick Stapel, dean of faculty at Tilburg University who was caught by his graduate students having published research over a period of 15 years based on falsified data. "The environment was conducive to it," he later explained.[56]

Examples of research findings later overturned are a recurring feature of published medical research, especially research depending on complex statistical analysis such

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[52] "Does High Public Debt Consistently Stifle Economic Growth? A Critique of Reinhart and Rogoff," Thomas Herndon, Michael Ash, and Robert Pollin, full access at: <http://www.peri.umass.edu/236/hash/31e2ff374b6377b2ddec04deaa6388b1/publication/566/>; see also <http://www.nytimes.com/2013/04/30/opinion/debt-and-growth-a-response-to-reinhart-and-rogoff.html>.

[53] <http://www.theguardian.com/science/2014/jan/19/mathematics-of-happiness-debunked-nick-brown>.

[54] The original article can be viewed at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3126111/>.

[55] "The persistence of wishful thinking," Brown, Nicholas J.L.; Sokal, Alan D.; Freidman, Harris L., *American Psychologist*, Vol. 69(6) Sep 2014 629-632.

[56] There is a lengthy article about Stapel in the New York Times Magazine, available at: <http://www.nytimes.com/2013/04/28/magazine/diederik-stapels-audacious-academic-fraud.html?pagewanted=all>.

as in molecular biology. Although many publicized cases involve academic fraud, data falsification, or research misconduct, and result in the retraction of published articles, these cases are regrettable outliers of the scientific process and not the typical reason research findings do not hold up to further research.[57] (*Nature* has been particularly hard hit with retractions.[58])

Professor Ioannidis is looking beyond research misconduct when he concludes that most published research findings are false. Two of his influential papers that are frequently cited are (1) "Why Most Published Research Findings Are False"[59], and (2) "Why Current Publication Practices May Distort Science" (with Neil S.Young, corresponding author, and economist Omar Al-Ubaydli)[60], both published online by PLOS Medicine.

Ioannidis applies standard statistical theory to consider the effect of hidden, unknown, and unstated factors, including pre-study probabilities, statistical power, bias, multiple studies, and data mining to conclude that even where appropriate statistical methods and valid data sets yield statistically significant results, the post-study probability of the results being true can be low, even lower than 50-50 for a majority of published results.

Ioannidis studied results in biomedicine and genetic analysis. His analysis has been widely applied to other fields of statistical research. His papers have been cited thousands of times, and "Why Most Published Research Finding Are False" is the most viewed PLOS publication with over 1 million views and counting. Ioannidis is solidly within the scientific mainstream.

In a companion note to (1) the PLOS editors say that "Ioannidis argues convincingly that many published finding will turn out to be false," and concluded that: "The major issue about the truth of research findings would therefore seem to concern the conclusions, and Ioannidis's claim that most conclusions are false is probably correct."[61]

Here is Ioannidis's Summary of (1):

> There is increasing concern that most current published research findings are false. The probability that a research claim is true may depend on study power and bias, the number of other studies on the same question, and, importantly, the ratio of true to no relationships among the relationships probed in each scientific

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[57] At least we hope this is the case. The pharmaceutical industry has come under some intense criticism in recent years for their published research in support of drug approvals. Marcia Angell, Robert Whitaker, Ben Goldacre, and Peter Gotzsche are among the respected severe critics.

[58] See <retractionwatch.com> and <ori.hhs.gov>.

[59] Full article available at: journal.pmed.0020124, August 30, 2005.

[60] Available at: journal.pmed.0050201, extended version 0050201.sd001.pdf, October 2008.

[61] Minimizing Mistakes and Embracing Uncertainty, August 30, 2005, journal.pmed.0020272.

> field. In this framework, a research finding is less likely to be true when the studies conducted in a field are smaller; when effect sizes are smaller; when there is a greater number and lesser preselection of tested relationships; where there is greater flexibility in designs, definitions, outcomes, and analytical modes; when there is greater financial and other interest and prejudice; and when more teams are involved in a scientific field in chase of statistical significance. Simulations show that for most study designs and settings, it is more likely for a research claim to be false than true. Moreover, for many current scientific fields, claimed research findings may often be simply accurate measures of the prevailing bias. In this essay, I discuss the implications of these problems for the conduct and interpretation of research.
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> Published research findings are sometimes refuted by subsequent evidence, with ensuing confusion and disappointment. Refutation and controversy is seen across the range of research designs, from clinical trials and traditional epidemiological studies* to the most modern molecular research*. There is increasing concern that in modern research, false findings may be the majority or even the vast majority of published research claims*. However, this should not be surprising. It can be proven that most claimed research findings are false. Here I will examine the key factors that influence this problem and some corollaries thereof.[62]

Statistical significance attempts to measure the likelihood of a finding being not merely the product of chance. It is not the only factor determining whether a research finding will hold up as being true. Other factors affecting the truth of a research finding include, as Ioannidis points out, the pre-study probability of the finding being true, the statistical power of the study (sample size, effect size), the prejudice, financial interests, and bias involved, data mining and multiple studies conducted, and the non-publication of negative findings. (The choice of the appropriate theoretical model is also crucial in statistical research.)

Ioannidis's research area is biomedicine and statistical genetics, and he is particularly critical of publication practices within this area.

Ioannidis has much to say, including the following excerpts:

> As has been shown previously, the probability that a research finding is indeed true depends on the prior probability of it being true (before doing the study), the statistical power of the study, and the level of statistical significance.
>
> The greater the financial and other interests and prejudices in a scientific field, the less likely the research findings are to be true.

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[62] Ioannidis (1) at p.1 (*footnotes omitted).

> Or prejudice may prevail in a hot scientific field, further undermining the predictive value of its research findings. Highly prejudiced stakeholders may even create a barrier that aborts efforts at obtaining and disseminating opposing results.
>
> Despite a large statistical literature for multiple testing corrections, usually it is impossible to decipher how much data dredging by the reporting authors or other research teams has preceded a reported research finding.[63]

It is not necessary for our purpose here that most published findings be false or that the odds of published papers being true be less that 50-50. It is sufficient to emphasize that mere publication of statistically significant results does not justify or support certainty. This is especially so when negative results and rebuttals are suppressed.

Given the history of repeated publication of findings falsely or prematurely absolving the medical industry of responsibility for the AIDS epidemic, the failure to publish refutations of those findings, and the financial and other interests involved in the matter, especially careful consideration should be paid to Ioannidis's analysis of bias in published research.

The publication of research findings is not sufficient cause for certainty.

X. Certainty in the Face of Uncertainty is Naive

I credit Quammen, Pepin, and Timberg & Halperin with naivety rather than something more malign. I don't think their intent is to hinder the progress of science, or to shill for persons willfully suppressing a needed scientific debate. That they refuse to respond to questions that may challenge their views is more indicative of how much their views have been influenced by the medical industry defense than of fear of being exposed.

Yet their work has served to help suppress the debate. To make amends they should take the time to study the issues they have so glibly reported on, not for the purpose of changing where they might believe the truth lies, but to reconsider the certainty with which they purvey those views.

As H. Allen Orr, University of Rochester biologist, wrote recently:

"Science demands unrelenting skepticism about purported facts and theories, and science journalism demands an ability to make the complex clear."[64]

I suspect that the Medical industry has not published their best defensive arguments. Those arguments are likely to be based on very complex genetic analysis and not be conclusive. Such arguments would open doors to rebuttals and counter-arguments,

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[63] Ioannidis (1) at pp. 1, 4, 5, and 7, respectively.

[64] H. Allen Orr, “Stretch Genes,” New York Review of Books, June 5, 2014, first paragraph.

and perhaps lead to suggestions for further research into the origins question. Not only would this open up the origins debate, it might highlight a debate we should be engaged in regarding the degree of independence and self-regulation granted to the medical industry. In this view the medical industry has been suppressing both sides of the debate.

Edward Hooper in his Lincei Conference paper and Brian Martin in a paper published evaluating the 2000 Royal Society Conference on the origin of AIDS detail the pressure from the industry on scientists expressing any interest in the origins debate.[65] Both of these papers are recommended in their entirety. It is difficult to summarize all the detail provided in those papers.

The pressure has not let up in the ensuing years and has probably intensified. The industry position from the beginning, from even before the *Rolling Stone* lawsuit, has been independent of the state of the evidence. Evidence itself has been suppressed, as in the case of a movie called *The Origin of Aids* which contains interviews with some of the participants in the CHAT trials. The producers and possible distributers have been pressured and threatened with lawsuits, and have been reluctant to make the movie available for showing on television or at film festivals. (It has been and may still be available on Youtube and is well worth watching.)

Science and *Nature* have continued to give prominence to papers supporting the industry theories, and to refuse publication of any criticism of that theory or those papers.[66]

The recent paper by Faria et al. is an example.[67] The paper has 14 coauthors, most of them established industry theory supporters. The coauthors cite 10 separate medical industry funding sources. Three things about this paper stand out. 1) It continues the industry failure to recognize or acknowledge the unresolved origins debate. 2) It undermines the certainty expressed by the four popular authors who claimed that the questions about when, where and how the epidemic began already have proven answers. 3) It differs only in a few details from previous bushmeat research, but gives some unacknowledged support for the OPV (CHAT) punctuated event theory of the origin HIV-1-M.

Faria et al. discuss only one side of their results, the side favoring the African hunter hypothesis and an HIV-1-M origin predating the CHAT vaccine trials. The authors report

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[65] Edward Hooper, “Dephlogistication . . . ,” Atti dei Convegni Lincei; 2003; 187; 27-230 (ISBN 88-218-0885-8); and Brian Martin, “The Politics of a Scientific Meeting: The Origin-of-AIDS Debate and the Royal Society,” 2001, PLS 20 (2): 119-130, Association for Politics and the Life Sciences.

[66] *Science*’s intransigence is especially curious. They refuse to publish correspondence from scientists, such as the prominent evolutionary biologist William D. Hamilton, or evidence from Edward Hooper, if it contradicts the industry position. Yet, Jon Cohen, an award-winning staff writer for *Science* who has written extensively about HIV, said when interviewed about the origins question, "Everybody's always looking for certainty. It doesn't exist [in this field]. In a sense it's all theory." Quoted by Mary Carmichael at pbs.org, footnote 37, *supra*.

[67] See Faria et al. at footnote 15.

that they conducted a new phylogenetic analysis of selected HIV-1-M genetic sequences collected between 1985 and 2004, plus the ZR59 sequence fragments, and that their basic finding is that the pandemic originated from a single individual circa 1920 in Leopoldville in the Belgium Congo (Kinshasa in the DRC now). From there the virus reportedly spread through the Congo basin along rail and river routes before coming to worldwide attention in the 1980s.

If a computer is programed to coalesce a given set of sequences to a theoretical common ancestor, it will do so. Different input sequences will give different results. The Faria et al. results differ somewhat from previous phylogenetic studies. They tweaked the methodology to include the locations as well as the dates at which the sequences were sampled. Thus they claim to have identified the most recent common ancestor of the HIV-1-M family as having been from the one individual in Leopoldville in 1920.

This is certainly speculative research. But if the result is assumed to be true for the moment, where does that leave the when, where and how of the species jump from chimp to human?

Jacques Pepin is credited as one of the Faria co-authors; he is quoted by *Popular Mechanics* writer Joshua A. Krisch as saying, "Where did the very first patient, the one who got it from a chimp, live? I don't know whether anyone has any plan to investigate this now."[68]

As for their geographical analysis, apparently Faria et al. did not have good information on rail and river transportation connections in the Congo in the years before 1960, making their geographical conclusions generally questionable.[69]

One Faria et al. claim is that HIV-1-M probably spread from that single person in Leopoldville in 1920 up the Congo River to Stanleyville by about 1953 (range 1926-1970). They reached this result using 23 HIV-1-M sequences collected in Kisangani (formerly called Stanleyville) from 1985 and later. The 23 sequences are spread among several HIV-1-M subtypes. It is only by assuming that there was no punctuated event that the

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[68] Joshua A. Krisch, “How Technology Traced HIV to Its Very Beginnings,” <http://www.popularmechanics.com/science/health/a11361/how-tech-traced-hiv-to-its-beginnings-17270225/>.

[69] Edward Hooper has roundly criticized the factual basis of the Faria et al. geographical analysis. See Hooper’s aidsorigins.com website for details on this and other aspects of Faria et al.

model is forced into the single tree that goes back to 1953. If the subtypes are looked at separately what shows up is the starburst of subtypes around the 1960s.[70]

The authors acknowledge that something unusual happened at that time, that the rate of spread of HIV in their model must be adjusted after 1960, but they don't acknowledge that their results also suggest the possibility of a punctuated event around the time of the CHAT trials.[71]

Faria et al. reports a clear anomaly occurring around the time of the CHAT trials. They reported an exponential growth rate for HIV-1-M before 1960 of 0.1 per year, but that around 1960 "group M transitioned to a second, faster phase of exponential growth" with a growth rate of .27 per year.

Could this almost three-fold increase in the growth rate at around 1960 be an artifact of a punctuated event around 1960 interfering with their molecular clock assumptions? Faria et al. contains no mention of the CHAT trials or the punctuated event alternative. The possible anomalous result is not linked to the origins debate either in the paper itself or in published commentary accompanying the paper.[72] Faria et al. does not move the debate between the two competing theories of origin any closer to resolution, but it does raise new questions about the industry theory.

Professor Dan Graur, a microbiologist at the University of Houston and coauthor of *Fundamentals of Molecular Evolution,* who writes the judgestarling blog at tumbler.com*,* has questioned the prominent publication of Faria et al. in *Science.* He quotes Beatrice Hahn as saying in an interview that, "It's really nice that a new group of investigators has worked on this using the most cutting-edge molecular phylogeographic methods and come up with the same conclusions [as she and Paul Sharp published as long as 14 years ago]".[73] According to Graur, Hahn refused to say more, implying a measure of sarcasm in her comment. Graur calls the Faria et al. publication pretentious.

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[70] Faria et al. has a color coded graph showing the phylogenetic trees by location and HIV subtype. The graph shows most of the subtypes coalescing around 1960 in the Congo. The possible exception is HIV-1-M subtype B which appears to coalesce later in the 1960s in the Americas. It is these trees that, when forced to coalesce further, go back to the hypothetical single case in 1920 in Kinshasa/Leopoldville. For an earlier analysis of an individual HIV-1-M Group see "Timing and Reconstruction of the Most Recent Common Ancestor of the Subtype C Clade of Human Immunodeficiency Virus Type 1", Travers, Clewley, Glynn, Fine, Crampin, Sibande, Mulawa McInemey, and McCormack, J Virol. 2004 Oct: 10501-10506; full text at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC516391/>.

[71] Nor do the authors mention that the entire population of Kisangani were fed the CHAT vaccine in 1959.

[72] Edward Hooper has written extensively about Faria et al, but his analysis has only appeared on his website. He discusses the anomalous result in considerable detail.

[73] Professor Graur's referenced blog: <http://judgestarling.tumblr.com/post/99344494676/old-data-a-great-deal-of-pretentiousness-a]>. The quote from Hahn is behind the *Science* paywall. Graur is another scientist who is very critical of *Science* and *Nature.*

Nevertheless, the prominent placement of Faria et al. in *Science,* and the resulting media coverage, has served to reinforce the public perception that there is no remaining debate about the origin of HIV/AIDS. There is a very strong institutional interest in this result, an interest that seems to outweigh the desire for truth.

In 1995 Hooper interviewed a senior virologist at the US CDC. Part of the interview was off-the-record, and the scientist promised to deny what he had said if Hooper named him. What he said was that HIV could be acquired by mouth, that if chimp kidneys were used, the CHAT vaccine could have been infected, and that, "If the theory's correct, it would make the U.S. responsible for the AIDS epidemic, and [we would] have to go and take care of those people over there with AIDS." He concluded by telling Hooper that, "I'm not sure you're going to get anywhere with [the CHAT theory] . . . I don't know if truth in itself has merit. I think that truth which makes a contribution has merit."[74]

XI. Observations and Conclusions from the Case Study

For more than 20 years the medical industry has made strong claims about the origin of AIDS based on weak evidence that has repeatedly been refuted. Suppression of the debate has been more important than searching for the truth. That this was true up through the mid 2000s is pretty clear from the record of false claims of final proofs. More recently the claims have become more complex, as have the rebuttals, complexities which serve to quell the debate, at least for the general public. The medical industry appears content to sit back and let non-expert popular writers state their non-expert opinions as fact, thus achieving a goal without putting scientific integrity at greater risk.

It is worth noting that there has been no overall balanced evaluation of the origin of AIDS published by a technically qualified researcher from within the medical industry that reviews and considers the whole body of existing research.

The Royal Society conference on the origin of AIDS held in London in 2000 is instructive. The original idea for the conference came from the preeminent British evolutionary biologist W. D. (Bill) Hamilton who had written the foreword to Hooper's *The River*. Hamilton was one of three persons (along with virologists Robin Weiss and Simon Wain-Hobson) delegated by the Royal Society to organize the conference. But Hamilton died before the conference was held and the conference was taken over by medical industry insiders and dominated by defenders of the CHAT trials, and faulty evidence (as discussed throughout this essay) was accepted and presented to the media as conclusive.

A persistent tactic of suppression has been to demonize any questioning of the medical industry's favored conclusion that there is no remaining debate over the origin of AIDS. To question published research, it is claimed, is to become a "conspiracy theorist," and undermine beneficial vaccination campaigns. This is not an atmosphere conducive to scientists in junior positions or dependent on funding to come forward with ideas or re-

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[74] *The River*, 1999, p. 806-807.

search proposals that are in opposition the the established position, or even to speak out freely.

It is often suggested that to oppose the industry position on the origin of AIDS is to oppose vaccination, and thereby harm public health. This is argument by calumny, and should be dismissed out of hand. Public health depends on the search for scientific truth, not the protection of industry interests.

Kathleen Sebelius while U.S. Secretary of Health and Human Services has been quoted as saying, "We have reached out to media outlets to try to get them to not give the views of these people [vaccine safety critics] [sic] equal weight in their reporting to what science has shown and continues to show about the safety of vaccines."[75] If Health and Human Services has also determined that the origin of AIDS debate is not in its interest it could easily be contributing to suppression of that debate through its media outreach.

Such positions appear to have had effect with the public media as evidenced by *The Guardian* coverage of the Faria et al. paper.[76]

Two psychologists at the University of Kent offer this useful definition: "Conspiracy theories are attempts to explain events as the secret acts of powerful, malevolent forces."[77] They go on to discuss anti-vaccine conspiracy theories as attempts to explain away overwhelming scientific evidence that vaccines are safe and effective.

It should be clear that the origin of AIDS debate is not an attempt to explain away scientific evidence, nor are secret acts of powerful malevolent forces used to explain events. It is not conspiracy theory to compare and contrast the evidence.

It is the medical industry defenders that fail to acknowledge or publish adverse facts and evidence. They do no honor to science or the search for truth by this failure.

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[75] Holland, Mary S., Reconsidering Compulsory Childhood Vaccination (September 15, 2010). New York University School of Law Public Law Research Paper No. 10-64, p. 30. Available through SSRN at: <http://papers.ssrn.com/sol3/papers.cfm?abstract_id=1677565>. Holland then says, “Failure to report criticism of vaccine safety is unlikely to resolve the serious questions that surround it.”

[76] *The Guardian* in England covered the publication of Faria et al. and opened its coverage for public comments. *The Guardian* redacted comments critical of the study, including thoughtful responses from people with science credentials. Checking back some months after the original article, some of the redacted comments were restored (but not one from Edward Hooper). Comparing those restored comments to others that were not redacted but did not question the Faria et al. story shows that it was not an issue over community standards as set forth by *The Guardian* that caused the redactions. It was purely a matter of censorship. One suspects that *The Guardian* science editor was warned by the Faria et al. co-author he interviewed to beware of conspiracy theorists critical of the study. *The Guardian* coverage is at: <http://www.theguardian.com/science/2014/oct/02/hiv-aids-pandemic-kinshasa-africa>.

[77] Daniel Jolley and Karen M. Douglas, “The social consequences of conspiracism: Exposure to conspiracy theories decreases intentions to engage in politics and to reduce one's carbon footprint,” published online 4 Jan 2013, <http://onlinelibrary.wiley.com/doi/10.1111/bjop.12018/abstract>.

One hopes for truth, whatever that may be, or, if not for truth, at least for free and open debate and scientific investigation. Suppressed theories can reappear as accepted fact. The history of astronomy and cosmology provide classic examples of this.

The idea that doctors were spreading puerperal fever by not washing their hands, and the depressing idea that poor disposal of human waste caused cholera are examples of theories once rejected as too ugly to be true, but are now accepted as undisputed.

This case study has attempted to untangle a situation where scientific uncertainty has been changed into an artificial social truth. The reality is that issues are becoming increasingly complex as science advances, and there is a public need to consider how society is to interact with this complexity.

If institutions are able to suppress and control public debate of issues in which they have an interest, then the public will be ill served by placing its full trust in those institutions, even when those institutions cloak themselves in science and good intentions.

It seems clear that from the very beginning the medical industry has not been interested in a genuine debate over its possible involvement in the origins of the AIDS epidemic. This case study shows the ability of the medical industry to control and limit scientific debate through the creation of public certainty. This does not mean that all members of the public have accepted the preferred final resolution of the debate, but that possible dissenters have been marginalized. Edward Hooper's research in particular has been devalued and excluded because he has refused to submit to the industry view.

Beyond this is the threat of marginalization of dissenting members within the medical industry if they were to go public with disagreements with the industry position.

This case study is presented as an instance of a pervasive phenomena. Governance has always been the prerogative, or at least the expectation of the elites of society. Today the elites include complex international institutions, as well as individuals whose wealth is so great that they themselves operate in the manner of institutions. Institutions naturally resist erosion of their independence and self-governance. Even small changes in the regulatory balance require exceptional motivation, commitment, and effort in the face of institutional ability to control the terms of public discussion and debate.

XII. Final Words– Applying the Case Study

When John Maynard Keynes wrote, "Practical men who believe themselves to be quite exempt from any intellectual influence, are usually the slaves of some defunct economist"[78] he was taking note of the kind of phenomenon examined in this case study. Strange certainties occur when the strength of popular opinion exceeds the weight of the supporting evidence. Strange certainties abound in our public discourse, and often determine, or at least influence our public and private actions.

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[78] Keynes, *General Theory of Employment, Interest and Money*, last paragraph of Chapter 24.

Weak evidence and strong opinions supported the U.S., U.K, and coalition of the willing invasion of Iraq in 2003. From defunct economists arose the strange certainty that austerity after the 2008 recession would be expansionary. Strange certainties led to the worldwide war on drugs. Our multi-trillion dollar so-called war on terror that includes extensive domestic spying on citizens may involve a complex of strange certainties.

Ideas and public certainties can be manipulated. Institutions spend billions of dollars to spread ideas. Internet platforms like Google and Facebook give away their own products in order to collect information and sell access to advertisers.

I think Keynes missed something important when he wrote, "I am sure that the power of vested interest is vastly exaggerated compared with the gradual encroachment of ideas."[79] This case study on the unresolved origin of AIDS debate presents an example of the power of vested interests to control the encroachment of ideas.

Effective control or manipulation of ideas in the public arena is an ultimate political power. Such control would allow institutional interests to affect the outcome of elections, or allow a narrow one party system or dictatorship to maintain support. Democracy would not threaten elite control so long as ideas were controlled. It is this kind of control that French philosopher Michel Foucault was probably getting at in theorizing about what he called power/knowledge being discursive, diffused, dispersed, and pervasive rather than concentrated and coercive.

Foucault was an early victim of the AIDS epidemic. He did not survive to finish his work. His life-long interest was in examining what he called regimes of truth. This case study can be seen as an example of what Foucault described as a battle over regimes of truth, or as a battle about the status of truth and the economic and political role it plays.[80]

However one feels about Keynes and Foucault, the importance of institutional interests and the manipulation of ideas in contemporary society stands on its own. I hope this case study has been a contribution to understanding the creation of strange certainties.

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[79] Keynes, *id.*

[80] I am indebted to the powercube <www.powercube.net> at the University of Sussex for helping condense Foucault's ideas, although the powercube disclaims compatibility with Foucault's understandings of power. Foucault's analyses are complex, difficult, often misunderstood, controversial among philosophers, and were evolving while he was alive. Two sources for Foucault on power in English are *Discipline and Punish: the birth of the prison*, and *The EssentialWorks of Foucault, 1954-1984,* Paul Rabinow, Series Editor (vol. 3, *Power,* James D. Faubion, ed.). His published series of lectures at the College of France and elsewhere show the development of his thought and what he called power/knowledge and regimes of truth.