

SACRIFICE OF THE VIRGINS, AND THE OTHER FIRST ANTI-CANCER VACCINE

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By Andrew Maniotis, PhD.

The Hall of the Virgins, Parthenon, Athens, Greece, 2005. South side of the Erechtheum, the famous porch of the Korai (maidens). On a pedestal, are six statues of young girls, well built and strong, proudly standing, their chests thrown out, attired in long Ionic tunics.

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SACRIFICE OF THE VIRGINS, AND ANOTHER FIRST ANTI-CANCER VACCINE

SUMMARY

Cervical cancer became an AIDS-defining illness in 1993, and HPV (the **H**uman **P**apilloma **V**irus) is now said to be responsible for approximately 70% of human cervical cancers. We have been assured that our daughters will become immune to cervical cancer later

in life if we subject them to gardasil or cervarix vaccination when they are virgins. We have been informed also, that if they test positive for more than 100 “HPV” molecular markers other than what are called the 16 or 18 DNA sequences of HPV DNA, then there is nothing to fear regarding the development of cancer.

These kinds of claims and mandates are advanced with such campaign slogans as “just one less,” and the vaccine maker, Merck, claims 100% protective efficacy for its gardasil vaccine, unless “the patient” isn’t a virgin. If this is the case, we are told, than all bets are off, according to Merck.

This analysis explores how these kinds of prophetic claims are advanced by scientists, doctors, vaccine industry, and Public Health Officials with such absolute certainty, in the context of human cancer-associated viruses.

In this context, the histories, hypotheses, and evidence that HPV DNA sequences 16 and 18 are associated with cervical cancer in humans, and how their detection can predict the development of that cancer, need analysis that is provided here. The evidence that gardasil, cervarix, other anti-cancer vaccines, and molecular testing can protect against the development of cervical cancers and others also requires a little reflection.

It will become clear that there are numerous common themes in the histories, assumptions, and politics surrounding “HPV,” “HBV,” “HBC”, and “HIV” research, medical practice, and standards of care, and I hope to show how these common themes actually are part of a single story that has directly led to the modern vaccines such as gardasil, cervarix, the hepatitis B vaccines, the 64 failed and in some cases abruptly halted “HIV” vaccine trials, and, the 210 \$500 million dollar/year “HIV” vaccine trials that are currently at some spot in that “HIV” vaccine pipeline

(Please see <http://aidsinfo.nih.gov/clinical-trials/search/q/1/category/299/vaccine-research/300/hiv-preventive-vaccines>; <http://aidsinfo.nih.gov/clinical-trials/search/q/1/category/299/vaccine-research/301/hiv-therapeutic-vaccines>).

As children, some of us were taught that the Sabin sugar cube vaccine was invented so children didn’t have to get shots that hurt, but we now know that in fact Sabin and Salk were bitter rivals, and each knew that both vaccines might be the cause of cancers due to a contaminant called Simian Virus 40. The Salk vaccine was immediately halted as soon as it was implemented to the masses because it increased the rate of polio, as Salk testified, along with the CDC, before Congress on several occasions.

In this context, you might ask, if one vaccine worked, why would they need to be developing no less than 210 different vaccine trials on humans in the context of “HIV” (145 preventative ones and 65 “therapeutic” ones)?

It is in this context where we must bravely consider the rationales and evidence underlying what has been described as “Challenger-sized disasters” such as the STEP and recently halted Thailand-US-military trial, and, also, the \$500 million dollar a year efforts to push forward the 220 current “HIV” vaccines that are in “the pipeline,” and others. Some critical analysis also is required to understand the results obtained so far in the context of vaccinating our infant children against cancers that are thought to arise from illnesses like hepatitis B with the hepatitis B vaccines, Merck’s claimed first anti-cancer vaccines, that it developed during the hepatitis B era of Baruch Blumberg.

I hope that as a researcher of cancer and as a cell biologist, that the following story will cast light on some fascinating and repeating assumptions that have become institutionalized during these several medical eras, and also, that this information will be useful in helping the layman, the medical professional, and Public Health Service officials alike to better understand these syndromes’ link to cancers. Finally, by presenting the story that follows, I hope to alleviate certain shame-generating stigmas surrounding these medical interventions and practices directly aimed in many cases at Africans, Americans of African descent, women, gay persons, and other groups.

Finally, before we explore in-depth the issues regarding Merck's and GSK's Gardasil and Cervarix vaccines for "HPV," it would be instructive to review the history leading up to these so-called second anti-cancer vaccines (the first being of course hepatitis B, which Merck said was its other first anti-cancer vaccine against liver cancer).

PART I: SOME HISTORY ABOUT CANCER VIRUSES

UNCERTAIN BEGINNINGS

In 1910, Payton Rous, Nobelist and celebrated discoverer of "RSV" or "Rous Sarcoma Virus" published that he could induce cancers in some chickens after transferring filtered juices called "extracts" made from tumors he obtained from cancer-afflicted barnyard chickens. Using the best filtering technology of his day, all that was possible to know was that something smaller than a bacterium in the filtered extracts could go through the filters to induce cancers, and in the minds of Rous and others, the most likely culprit was some unknown protein(s), probably made by the tumors that could pass through the filters. At the time of his discovery, and indeed many decades later, he himself didn't accept or acknowledge that his work had proven that any virus was responsible for chicken cancers, but those who celebrated and awarded Paton Rous's discovery with a Nobel prize did. Rous's December 13, 1966 Nobel lecture is perhaps the most articulate account of a possible role that viruses have had in "the initiation" or "promotion" of human cancers that can be found anywhere, according to all the evidence he was aware of in 1966, with statements like:

*"What can be the nature of the generality of neoplastic changes, the reason for their persistence, their irreversibility, and for the discontinuous, step-like alterations that they frequently undergo? A favorite explanation has been that oncogenes cause **alterations in the genes of the cells** of the body, somatic mutations as these are termed. But numerous facts, when taken together, decisively exclude this supposition"¹.*

Rous is referencing himself here, his 1957 article entitled "Surmise and fact on the nature of cancer," (Nature, 183, 1959, page 1357)," and he is using the term oncogenes to mean cancer promoting agents or chemicals, not oncogenes, or so-called cancer-associated genes. Yet he believed, before the term oncogenes was termed, as he emphasizes, that the genes of the cells were not the targets of such carcinogens.

"No virus has as yet been found that indubitably actuates neoplasms in man. Yet this is not to say that viruses play no part in initiating them occasionally. Now and again a human cancer arises where a virus has persistently wreaked inflammatory injury. The virus causing "fever blisters" often next to the mouth of persons who are notably susceptible to its action provides an instance in point, cancer sometimes arising after a while from the epidermal tissue long kept in a disturbed state. One sees the same course of events occasionally on skin where the severe virus causing herpes zoster ("shingles") has left tissue permanently damaged. Yet these instances tell no more, as concerns causation, than do the cancers that now and then arise on the skin of old people where it was burned in youth. Sunlight provides yet other examples, cancer arising from skin that it has been kept inflamed instead of tanned. In all these instances the tumors have been merely initiated. No virus actuating any of them has been discovered."

When discussing "mouse mammary tumor viruses" and how they were believed to have been discovered, Rous emphasized the following:

*"This virus lay latent within them until they matured, and then it caused orderly, benign tumors in the breasts of the females; and from these growths carcinomas derived by step-like changes like those already described. **To this day no virus directly responsible for the cancers has been obtained from these, but only the "milk virus" producing the benign growths.** The problem of the actuating cause of the cancers remains unsolved like that of the V x 2 carcinoma of rabbits⁴. In both*

instances the virus causing the benign tumor was no more than an initiator of the malignant growth deriving from them.

In reference 4 given here that Rous says is an unresolved problem, a footnote or qualification is provided. In this qualification (reference 4 of his Nobel lecture), Rous goes out of his way to state that:

“4. Despite protracted search, aided by the electron microscope, no tumor virus has ever been found in human milk, and family histories definitely rule it out.”

and it should be noted that it is Rous’s own emphasized surmise here that *“In both instances the virus causing the benign tumor was no more than an initiator of the malignant growth,”* is just that: surmise, not fact. A virus is a suspected but not a demonstrated initiator *“not directly responsible”* for the cancers,” if you read the statement carefully.

In an analysis of Dr.s Eleni Papadopulos-Eleopulos, Valendar F. Turner, John M Papadimitriou, and David Causer of The Royal Perth Hospital, Australia below, we are reminded of the important fact that in 1911 Rous did not consider his experimental evidence as proof that the cause of the chicken sarcoma was a virus:

“The first tendency will be to regard the self-perpetuating agent active in this sarcoma of the fowl as a minute parasitic organism...But an agency of another sort is not out of the question. It is conceivable that a chemical stimulant, elaborated by the neoplastic cells, might cause the tumour in another host and bring about in consequence a further production of the same stimulant.”

“In 1999, two medical historians writing about Duran Reynals, “most likely the first Catalan to become a research scientist of world renown”, and his close collaboration with Peyton Rous, wrote “...even though the Nobel Committee recognised the “agent” as a virus when it awarded Rous the Nobel Prize for Medicine, he still refused to recognise it as such.”

In any account about Rous’s experimental genius regarding his keen descriptions of animal cancer induction and transplantation experiments, he pointed out another important problem during his Nobel lecture with respect to tying viruses to cancer initiation or promotion. Although he attributes the first discovery of tumor viruses to a discovery in 1908, he cautiously qualifies the discoverers’ evidence that it was a cancer virus they found. Instead, Rous points to a persistent problem that all researchers of this cancer-virus hypothesis would thereafter and presently encounter, namely, that the disease attributed to the discovered virus, or virus-like particles, was in fact a confirmed or recognized cancer:

*“Two Danes, Ellermann and Bang, reported the first tumor virus in 1908². It caused leukemia in chickens and they made six successive passages of it from fowl to fowl, producing the same disease each time. They studied it until 1923, meanwhile reporting upon a second virus causing a chicken leukemia of a different sort. Yet though their work was convincing **its findings were written off because the leukemias were not then realized to be neoplastic diseases nor indeed until after 1930.**”*

In other words, if these sources and statements are to be seriously considered, Rous himself left the door open to the possibilities that no virus had been linked to a definitive cancer even in the original report, and, work employing the so-called filtered tumor virus extracts of Ellermann and Bang may have shown instead the ability of foreign proteins to initiate lesions instead (long before the invention of electron microscopy to show there were any viruses in the first place). Most significantly in this context, the diseases transferred from animal to animal might not constitute cancers. The morbid syndromes that resulted through filter extracts were not at that time recognized as cancers to begin with, until 1930. If you want to demonstrate that a virus causes cancer, you need to at least have a cancer that is recognized as a cancer, but, such are the beginnings of much of biomedical research. Retrospectively, at least, we cannot even conclude as Rous suggests, that Ellermann and Bang discovered the first tumor virus because nobody considered their work to show the induction of a cancer for another 22 years, and that the condition harbored by the chickens and

induced in other chickens with filtered extracts was leukemia. Because of these uncertainties, during a time when no equipment or electron microscope was yet invented that could even show viruses, it is of perhaps more significance to realize that the so-called first cancer causing extracts exerted their filterable tumor-causing activity as toxins produced by a tumor as Rous suggested, or simply as foreign material transferred into a non-self context, rather than as the so-called integrating viruses or retroviruses that are believed to exist today. Of equal interest perhaps, was Rous's strong feeling and warning that oncogens (toxins) do not disturb or disrupt cellular genes as quoted above because, "*numerous facts, when taken together, decisively exclude this supposition.*"

By proposing a process or mechanism of cancer induction that did not involve the disruption of genes or somatic mutation being important in cancer induction rather than a consequence of it, Rous's admonishments and elegant work on tumor extract cancer induction in chickens also raise issue with modern genome analyses that point to the imagined genome structure of both "RSV" and "HIV," with their currently defined gag, pol, and env sequences that such "viruses" are believed to harbor, according to molecular analyses and genome database searches. This is odd because if similar to "HIV"'s genome structure, it becomes enigmatic in this context that "HIV" was first described to be "a variant of a known human cancer virus" thought to peacefully infect cells and make them cancerous, rather than an immune suppressive virus that killed cells. It is odd also because Rous's sarcoma virus is thought to harbor the same basic retroviral genome structure as "HIV." These modern molecular assumptions couldn't be more at odds since now "HIV" is believed to cause lymphoid cells first to proliferate and through direct and indirect mechanisms, kill those same cells instead of making them cancerous, leading to extreme immune suppression, and not cancer, but more about this later.

WARTS ENTER THE PICTURE

No one knows how long the stories about the existence of horned cottontail rabbits that looked to people like small goats in the Southwest were passed down as folk legend. The rabbit "horns," it was claimed, would form only on "naked areas" of the rabbits. The horny wart-like growths were observed on the eye region, anus, ears, or mouths. By 1933, Richard Edwin Shope, had been an active promoter of the viral cause, rather than the postulated bacterial or vaccination causes of the Great Spanish Flu epidemic of 1918-1919, and an experimenter with flu vaccines in animals. As a pathologist turned virologist, Shope tested whether extracts of these horn-like protuberances or growths could induce lesions in non-lesion-bearing domestic rabbits. He used filtered extracts, as Rous had done with chicken tumors, from fibromas (benign growths) obtained from the horned rabbits.

In certain strains of cottontail rabbits from the Southwest and from California (also Iowa and Illinois), Shope demonstrated he could reproducibly induce papillomas (warts) on the rabbits' ears, especially in these strains of rabbits, but not in others. He concluded that susceptibility to tumor formation and cancer development was rabbit strain-dependent. While some of Shope's rabbits developed lethal cancers and not only benign growths, most wild strains of rabbits other than cottontails, except a strain from California, were not susceptible to the development of any lesions when the tumor extracts were applied to their skin. Shope's rabbit experiments with warts, papillomas, and cancer set the stage for our modern science of cervical cancer, complete with the ongoing conundrum regarding the power of "the seed" (the cancer) versus "the soil:" the organism with cancer. His observations also raised issue with the cancer "virulence problem." Like Pasteur's rabies where some strains seemed to cause great harm while others didn't, Shope identified the question regarding how deadly or harmless various extracts seemed to be, versus their variability in being able to transmit any lesions, or not, to another organism. And of course, the possibility advanced by Rous, that there was a toxin and not necessarily any transmissible virus that induced cancers and growths, had not yet been addressed experimentally, other than to say the "agent" whatever it might be, was able to pass through a filter smaller than a bacterium.

A noted biochemist and his group in 1939 improved upon Rous's and Shope's tumor extraction process, and Beard and his colleagues added sophisticated biochemical and analytical chemical methods in an attempt to better define the active cancer-inducing principal contained in the horny-wart extracts. Beard found they he could induce warts and growths using only the protein extracted from what they believed was the active substance in the filterable agent. In so doing, Beard and colleagues showed that the protein alone

contained in the extracts, which sedimented at a specific density, was the likely carcinogenic substance.

This realization though was much like Percival Pott's surmise had been as far back as 1779. Pott surmised that coal tar or chimney dust induced scrotal cancer in young boys that had been forced to clean the soot out of chimneys naked, so their clothes wouldn't get blackened. Such substances, especially hydrocarbons, acted like irritants when smeared on the skin of certain kinds of rabbits. Beard also "played" with the activity of the extracted and sedimented protein material, and perhaps was the first to begin characterizing it using the best chemical methods of his day (which were considerable). He concluded in one paper that the material lost its wart-causing or lesion causing activity after the protein was denatured with excess low or high pH, and heat. Very high titres also were typically needed to evoke the lesions, but low titres also could sometimes induce growths.

Again, Rous's Nobel lecture is helpful to weigh the impact of this work:

After working out the complex relationship existing between the RSV, the cells it affects and their hosts, I tried for several years to get causative viruses from the transplantable tumors of rodents but with the same failures as previous investigators and hence quit the neoplastic problem for others in pathology which proved rewarding. Not until 1933 did a virus opportunity come my way again. Richard Shope of the Rockefeller Institute (a man already renowned for his discovery of animal diseases with human implications) reported then on a virus causing the giant warts often present on the skin of wild "cottontail" rabbits in the southwestern U.S.A. On inoculation this virus proved effective only in rabbits, and it produced far more vigorous warts on animals of domestic breeds than on its native host, the cottontail. When describing the growths of both species Shope remarked that they **might be** true tumors. He knew of my fruitless search for a mammalian tumor virus, and he and I had long been friends. Hence he asked me to determine the character of the warts, saying that he knew nothing about tumors and already had more than he could do, what with possessing four new viruses responsible for animal diseases of other sorts. Thus it came about that I experimented as his deputy throughout many later years. Now and again he reported on the peculiarities of the papilloma virus as such, but never concerned himself with its relationship to tumors until after the work in my laboratory had ended. He died – and of cancer – less than a year ago. One of his last papers, written while ill, ranks as a classic³. It is concerned with "the many sly and devious ways that viruses may behave in causing tumors," **and he stressed, as example, an extraordinary finding reported by him early, namely that the papilloma virus can rarely be recovered from the prodigiously active growths it yields in domestic rabbits although immunological tests show it to be present and it can regularly be got in quantity from the cottontail growths.**

*Experimentation carried out in my laboratory together with Beard, Kidd, Friedewald and MacKenzie showed the "warts" produced by the virus to be genuine tumors, **benign epidermal papillomas** in which the virus persists **although eliciting an antibody capable of neutralizing it on direct exposure.** The same anomalous state of affairs had previously been found to exist in chickens carrying the first tumor that yielded a virus and experiments had disclosed the fact that the phagocytes of normal blood can protect ingested **bacteria** from antibodies present in the surrounding medium, as long as they themselves remain alive and will even shield from **a markedly hemolytic serum** the phagocytosed red cells from **a foreign** species.*

*These findings enable one to understand why the Shope papilloma **virus** flourishes in the proliferating cells of cottontail growths **despite** the strong antibody that it engenders, **and why it never causes the normal cells around it to become neoplastic** but instead produces tumors which grow by intrinsic cell multiplication, "caus sich heraus", to use the German phrase, like the neoplasms of unknown cause.*

*Some of the carcinomas arising from the papillomas of domestic rabbits were serially transplanted, **and one** that soon became anaplastic and exceedingly malignant has now been maintained for 28 years by transfer from rabbit to rabbit. **Never has it yielded a virus of any sort, and tests of the blood of its early successive hosts showed that the antigen inducing***

an antibody against the papilloma virus gradually disappearing. Now it has been gone for many years. This cancer, known as the V x 2, is studied in many laboratories today because, like the generality of those due to other causes, it yields no sign of what actuates it. Obviously the Shope virus had been merely an initiator when producing the V x 2, though some antigenic remnant of it had persisted for a while. In this relation a remarkable recent discovery by the Kleins of the Royal Caroline Institute of Sweden deserves mention, namely that certain polycyclic hydrocarbons initiating mouse tumors put a specific, antigenic mark on these, which persists despite repeated transplantation of the growths.

All of these statements (especially the emboldened ones) are critical pieces of cancer virology that in some form or another still heavily influence present day thinking. For instance, and almost wraith-like, Rous believed these suspected filterable cancer-inducing virus extracts didn't behave like other viruses, such as baculoviruses in silk worms that had been known to have decimated the silk industry as far back as at least the 1500's. Massive cellular destruction and large quantities of microscopic particles, thought to be cellular breakdown products, had always been seen to be associated with high concentrations of sub-microscopic particles at all times, rather than like "actuating" ghosts that can't be seen to do their damage before they disappear. As we shall later see, the same can be said for "HIV," whose proponents first had us accept that they only could find one virus in one out of every thousand or ten thousand cells, or indeed why PCR needs to be used to detect "HIV" in the blood of someone said to harbor nearly a half-million "viral load" in their blood.

Rous's explanation of antibody immunity in the context of papillomas in his animal experiments is also critically important to understand because it accounts not only for why virus-like particles don't spread from cell to cell or cause "*normal cells around it [an infected cell] to become neoplastic.*" His explanation about an antigen evoking a potent neutralizing yet waning antibody begs the question in both the contexts of hepatitis B and "HIV/AIDS" as to why no cellular damage or virus particles are noted in the liver in the first case after experimental infection of chimps or mice with HBV, and why in the second case, a positive "HIV" test for human is something to fear, since one would surmise that since the ELIZA (Enzyme Linked Immunoabsorbant Assays) and WESTERN blot tests for supposed host-made antibodies against "HIV," that these antibodies would keep these entities, like Shope's and Rous's imagined containment mechanism of HPV, also from infecting other cells, or from harming its host in any way.

To end this section about the principal research and investigators that are said to have established that viruses cause cancer in animals, it is fitting to end with a contrary statement from Payton Rous's Nobel lecture once more:

"Ignorance could scarcely be more stark."

HOW CERVICAL CANCER CELLS CHANGED THE WORLD AND HELPED WIN THE BIOLOGICAL ARMS RACE

During the 1950's, the third major push for a polio vaccine that might prevent rare childhood paralysis became coupled to the unsubstantiated belief that a virus also must be a cause of human cancers with renewed vigor after World War II. Oddly, some of the cells first used to try to grow the intestine-derived polio "agent" were cancerous cervical tissue cells, cells that came to be known as HeLa cells. The also advanced the idea at this time that suspected human cancer viruses also could be immunized against to rid cancer from the face of the world via vaccinations made against them.

This belief in the existence of human cancer viruses during the 1950's polio era helped shape a campaign to launch a "War On Cancer" during the 1960's and 1970's. This initiative was begun by Mary Lasker, **Mathilde Krim**, and others. They had close personal connections to the most powerful political circles inside Washington, and also to Hollywood and to the Hollywood stars. Mary Lasker was the wife of a prominent New York businessman who had died of cancer, Albert D. Lasker. Mathilde Krim had married the director of Orion Pictures who also had developed countless connections in both Hollywood and Washington D.C.

In 1956, the Albert and Mary Lasker Foundation, Inc., along with George Gund, the George Gund Foundation, and David M. Levy,

bank-rolled the James Stevens Simmons Memorial Fund at the Harvard School of Public Health. (Gifts to Harvard, January 1 to March 31, 1956. Nathan March Pusey. ctr/50001545-1580.) Simmons was the Dean of the Harvard School of Public Health from 1946 to his death in 1954. From 1940 to 1946, he was a founder of **The Armed Forces Epidemiological Board,** with then-**Colonel Stanhope Bayne-Jones** as his executive assistant. (The Genesis of the Board for the Investigation and Control of Influenza and Other Epidemic Diseases **in the Military.** US Army-History of the National Cancer Institute's Viruses and Cancer Programs / National Institutes of Health (pdf, 379pp).

Members of the NACC in 1958 were **Lane Adams of the American Cancer Society**; Dr. Murray M. Copeland; Dr. Charles A. Evans; Dr. Henry S. Kaplan; **Mrs. Mary W. (Albert D.) Lasker**; Dr. Robert A. Moore; Dr. Isidor S. Ravdin; Dr. Leo G. Rigler; Dr. Joseph F. Ross; Dr. Harold P. Rusch; Dr. Richard S. Schreiber; Dr. Howard E. Skipper; Dr. Warren Weaver; Mr. James E. Webb; and Dr. Sidney Weinhouse. **Bayne-Jones was a member of the National Advisory Cancer Council from 1959 to 1961, and had maintained ties with his Yale “skull and cross-bones” connections thereafter for many years,** such as some of the members of the President's Cancer Panel, 1976, and National Advisory Cancer Council, 1957-71 (J Natl Cancer Inst 1977 Aug;59 (2suppl):763), and The President's Cancer Panel (J. Natl. Cancer Inst 1977 / tobacco document). Colonel Bayne-Jones was appointed to the Surgeon General's Advisory Committee on **Smoking and Health** (US Department of Health, Education, and Welfare, Press Release, Nov. 11, 1962). Colonel Bayne-Jones acted as their quasi-chairman and was senior-most member, and he'd been administrator as Yale medical school dean: he was 'the father figure' for the group" (Richard Kluger's notes for "Ashes to Ashes," interview of SGAC member Leonard M. Schuman, public health epidemiologist from University of Minnesota; July 15, 1988). Mary W. Lasker's friend Florence Mahoney had close ties to **President Kennedy.**

These people, and their connections and beliefs served to codify much of what has become the modern “science” and views of cancer viruses that still are prevalent today. Study of their initiatives and world views reveals that their sociological, militaristic, philanthropic, and cultural views were of influence equal to, and arguably far exceeding, any scientific hypotheses or theories about cancer biology, as will become clear by consideration of the following information.

“Scientifically,” there was a strong influence from Dr. Mathilde Krim, who was not only instrumental in establishing a “war on cancer” but also among the first and most vocal to found AmFAR during the AIDS era (the American Foundation for AIDS research that was ideologically conceived of and begun under similar pretenses and fanfare as the launching of the War On Cancer, including the recruitment of Hollywood stars and producers). Along with Mary Lasker, Krim first convinced Lyndon Johnson of the importance of defeating the virus she believed might cause human cancers based on no evidence that one existed in the first place, as Rous recounted in his 1966 Nobel lecture. Early in Nixon's regime, Lasker and Krim finally triumphed and convinced the president, and other heads of State, to direct hundreds of millions of dollars of taxpayer money to rid the world of cancer. (It is interesting to note that AmFAR is similarly now directed by the shoe-salesman and fashion magnate, Richard Cole). With her “Human Cancer Virus Program” now funded to identify and contain the first imagined “human cancer virus” believed to transform normal cells into cancerous ones, and then to develop a vaccine against it, Krim and her cancer-virus-theorizing promised to make the world cancer free. Nixon's administration then launched the intensely funded Special Cancer Virus Program, along with its special buildings created to contain that cancer virus when, and if, it ever was found. It was a venture christened by Nixon as an initiative similar to splitting the atom or putting men on the moon. These political maneuvers also helped launch a new biological arms race during The Cold War between the Soviet Union and the U.S. About the same time, the Soviets were given a small collection of what the Americans believed were **animal** tumor viruses during a visit by a Soviet-American scientific delegation, and thus the Soviets were thus made aware that the race was on to find “the first Human tumor virus” with all it could mean in terms of a biological arms race, or Sputnik-like competition.

How did Mathilde Krim, a prime instigator of “the war on cancer” and eventually the founding chairman of AmfAR, The American Association For AIDS Research, precisely influence the beginning of “the War On Cancer?” According to her biography:

Krim joined the research staff of Cornell University Medical School following her 1958 marriage to Arthur B. Krim—a New York attorney, head of United Artists, and founder of Orion Pictures—and advisor to Lyndon Johnson. It was at the Krim NYC home on May 19, 1962 that the famous 45th birthday party for President John F. Kennedy was held, with many famous persons in attendance (Robert Kennedy, Marilyn Monroe, Jack Benny, Harry Belafonte). In 1962 Krim became a research scientist at the Sloan-Kettering Institute for Cancer Research and, from 1981 to 1985, she was the director of its **interferon** laboratory.

In an Israeli newspaper story by Amos Elon published in Haaretz on Tuesday, June 29, 2010 we learn just how connected to the Washington power structure Krim was during the Johnson administration:

Incredible intimacy

Tom Segev documents this historic tragedy – brilliantly and authoritatively – as no one has before. Combining keen political awareness and understanding of human foibles, and weaving together political, social and cultural history, he paints a vibrant picture of Israel in 1967. The book reads like a chapter from Barbara Tuchman’s “March of Folly: From Troy to Vietnam.” Tuchman defines folly as “the pursuit of policy contrary to self-interest.” This is a long book, but it is hard to put down. It is not a raging broadside, but the work of a serious historian who says what he has to say without raising his voice, basing himself on sources in Israel and the United States never published before. His chief sources are records recently opened to the public in the Israel State Archives, including official protocols of government meetings before, during and after the war; the personal papers of Ben-Gurion, Levi Eshkol and Lyndon Johnson; and the files of Yaakov Herzog, then director general of the Prime Minister’s Office. Herzog’s files are particularly important because he was a politically independent civil servant and a shrewd observer, who filed away every scrap of paper that landed on the government’s desk. Segev’s probe has turned up material that attests to an incredible intimacy between Israel and the White House during and after the war. Such a relationship was obviously a great help to Israel, although President Lyndon Johnson strenuously objected to it. “It was almost idyllic,” writes Segev, thanks, in large measure, to such powerful and influential Jews as U.S. ambassador to the United Nations Arthur Goldberg; two key figures in the Democratic party – industrialist Abe Feinberg and Washington attorney David Ginsberg; and Supreme Court justice Abe Fortas, who enjoyed free access to Johnson and often used it to promote Israel’s interests. The president himself was anxious to gain the support of American Jewry for the war he was waging in Vietnam.

Segev adds a little glamour and romance to the relationship with his disclosure that President Johnson had a beautiful Israeli girlfriend – a Weizmann Institute scientist who married the Hollywood producer Arthur Krim. Mathilde Krim was a frequent guest at the residential wing on the third floor of the White House, with or without her husband. There is no question that she goaded Johnson into supporting Israel, and she seems to have done so, writes Segev, in coordination with the Israeli embassy. According to historian William Quant, only two people could say they spent more time on the phone with President Johnson than Mathilde: secretary of state Dean Rusk and secretary of defense Robert McNamara. On the day the Six-Day War broke out, there Krim was again, sleeping over at the White House, in Room 303. At 6 A.M. in the morning, there was a loud knock on her door. She opened it in her nightgown and there was Johnson, visibly agitated, with the news that the war had started. What they said to each other next is anyone’s guess. Segev, interviewing Krim for the book, asked her if she and Johnson had been romantically involved. She denied it, but added that it wasn’t the first time she had heard that question.

It is with admiration and resolve that Krim recounts in a BBC documentary how Mary Lasker, as a prominent socialite, captured the attention of Lyndon Johnson and persuaded him to launch a “space-like” program on cancer. With statements like, “more money is spent on chewing gum than on cancer research,” Lasker and Krim were successful with the connections they had with Washington and Krim’s Hollywood husband. With stars like Being Crosby, William Tallman who had lung cancer (of Perry Mason fame), and The American Cancer Society, the stars promised they would create the fund to end cancer (The Undead Henrietta Lacks And Her Immortal Dynasty documentary by Adam Curtis <http://www.bbc.co.uk/blogs/adamcurtis/2010/06>

[/the undead henrietta lacks and.html](#)).

Also, as it turned out, Richard Nixon's favorite "Aunt Elisabeth" had died of cancer, and in 1971, Mary Lasker (and Mathilde Krim) triumphed as Nixon announced his 100-million dollar/year war on cancer, to be waged with the same kind of concentrated effort that was used to split the atom and take men to the moon.

HOW RACISM KILLED THE FIRST HUMAN CANCER VIRUS AND THEN GAVE BIRTH TO OTHERS

The Americans were now fighting their new "war" to try to find and "root-out" a suspected hiding mythical human cancer virus terrorist with Mathilde Krim and her interferon laboratory and others leading the charge. The Soviets shortly thereafter announced to the world that after they had played around with small collection of animal "tumor viruses" given to them by the American delegation, that they had inadvertently found by luck the first human cancer virus. However, a little chink in the human cancer virus theory then emerged full force from an immortal cervical cancer cell line obtained from an African American woman that was named HeLa.

These cells came from Henrietta Lacks, an African American woman whose family had been "plantation workers" for 3 generations. Her father, the plantation's owner, was white while her mother was a black woman who worked in his fields. She grew up in rural Virginia, and she moved to Baltimore, Maryland, as an upwardly mobile young African American woman during the Second World War.

During her thirties, Henrietta showed up at Baltimore's principal city hospital with a malignant cervical cancer. Her cervical cancer cells would be the "first" human cells it would be claimed that could be grown perpetually outside the human body, so that dedicated and self-less scientists and doctors could isolate "the cause" of cancer and defeat it. Dr. Gey was able to culture her cervical cancer cells in Petri dishes using chicken serum as Henrietta lay dying of disseminated cervical cancer, and after "proven mainstream remedies" promoted by the American Cancer Society and National Cancer Institute, such as radium seeds and radiation treatments, had failed to cure the young woman, who by the way, died of urea poisoning. Henrietta Lacks died in 1951, and her "HeLa" cells were sent to cancer researchers all over the world by Dr. Gey for research purposes. During a televised broadcast before the American public where Gey introduced his findings that he could grow cancer cells outside the human body, and when he spoke about how he would solve the cancer problem, it became clear that for reasons other than patient confidentiality, he felt he needed to conceal the true origin of the cervical tissue from Henrietta Lacks' family. Dr. Gey changed the name of the patient he had isolated the cervical cancer cells from "Henrietta Lacks" to "Helen Lane," and called the cells "HeLa" for short. Although her cells had been obtained without her permission or her family's, it has been insinuated in personal interviews in a BBC documentary that Dr. Gey was afraid of being sued by Henrietta's family for not gaining their consent to use Henrietta's cells, which is why he changed her name to Helen Lane, and the cells he had isolated, to HeLa.

The cervical cancer cells of Henrietta Lacks that Dr. Gey sent out to the world ended up at NASA and they were sent into outer space into zero gravity where they were said to grow faster and faster; they were blown up by atomic bombs during the above ground testing of nuclear weapons in order to irrationally determine if the atomic bomb radiation would induce chromosomal changes in cancer cells (rationally, one would want to test normal cells because all tumor cells have distorted aneuploid chromosome sets); they irrationally constituted the cellular testing material of the cosmetic industry (again, one would want to know how normal cells could be affected when cosmetics were applied); and during the early 1950's they were used without success to generate yet another failed "polio vaccine."

Perhaps most significantly, HeLa cells were injected into some 80 or more Ohio prisoners' forearms by a Dr. Chester Southam. Notably, this inoculation of live cancer cells into humans followed the first hepatitis B vaccine trials on prisoners in that same Ohio prison that were conducted by Nobelist Dr. Baruch Blumberg and Saul Krugman and others (who would launch the hepatitis B era

that will be discussed later). Yet before Chester Southam would eventually concede that his cancer cell injections failed to induce metastatic cancer in any of those 80 prisoners, 3 Jewish student doctors under Southam's charge at the Jewish Chronic Disease Hospital in Brooklyn had refused to follow Southam's orders to do the same experiment on the sick and old who were hospitalized there, and they alerted the hospital's top administrator. Southam had and wanted to continue to perform the same experiment on 40 old sick Jewish folks at this Jewish Chronic Disease Hospital in Brooklyn, who only spoke Yiddish, and couldn't understand the purpose of Southam's experiment if they wanted to. It also would become revealed that Southam also would subject more than 600 gynecological surgery patients to his inoculations of live HeLa cancer cells and other cells at Sloan-Kettering Memorial Hospital.

Why were Henrietta Lack's cervical cancer cells injected into the arms of 40 non-informed and aged and quite ill Yiddish-speaking Jews at the Jewish Chronic Disease Hospital in Brooklyn, and also, without their knowledge, into more than 600 women in New York who had undergone gynecological surgeries of various kinds? Because Dr. Southam wanted to know if cancer cells were needle-stuck into doctors or researchers, if they would acquire cancer, since doctors and scientists were special soldiers on the front lines to rid the world of that disease.

The work was published. It made little impression on those doctors or scientists that an African American woman's living malignant cervical cancer cells didn't cause cancer even when directly injected into a human being's skin by a tuberculin syringe and repeatedly into some 80 healthy white prisoners, into 40 morbidly sick and aged old persons some of whom already had cancer, or into 600+ unsuspecting females. All that happened was a small lesion formed at the inoculation site, and the lesions became rejected and the injection site healed in every human case that he followed.

After the student doctors refused to aide in Southam's experiment at the Jewish Chronic Disease Hospital, the ethical conundrum Dr. Chester Southam's "human experiments" created became the focus of the world's attention, that led to many our modern experimentation rules and informed consent laws that are currently in place when human beings are ever used in medical research.

Was it possible, perhaps, that malignant cancer cells didn't cause cancer by themselves without the proper "soil?" Most importantly, however, an initiative was begun "to grow" the suspected polio agent or virus in HeLa cells for reasons that aren't completely clear, but which failed as an attempt to amplify the suspected viral polio agent, as the findings of John Enders and his use of normal embryonic cells would show. Enders derived the cells that would propagate the polio agent from human embryos, for which his virological accomplishment would bring him the Nobel. Although, after he was caught, Dr. Southam was reprimanded and his work was stopped for about a year, he was elected as Vice President of the American Cancer Society.

Southam believed that he had found a test for human immune suppression, as the old sick Jewish patients took the longest to reject their tumors, and Southam imagined standardizing the injection of live cancer cells into everybody to "know their status" (their cancer potential status) and to identify potential cancer patients harboring latent, yet non-morbid-symptom-inducing tumors, by measuring the length of additional time needed for them to reject his injection of HeLa:

Isoantibodies to Human Cancer Cells in Cancer Patients Following Cancer Homotransplants

Tetsuo Itoh and Chester M. Southam. The Journal of Immunology, 1964, 926-936. From the Division of Clinical Chemotherapy, Sloan-Kettering Institute for Cancer Research, New York 21, New York.

Abstract

*Homotransplants of human cancer cells were made in patients with cancer of various types and in various stages of progression. Tissue-cultured cancer cell lines (HEp 2, HEp 3 or RPMI 41) were used for 28 recipients, and cell suspensions prepared **from surgical specimens** were used for another 12. Homologous leukocytes or other non-neoplastic cells were admixed with some of*

the inocula. Sera collected at frequent but irregular intervals were tested for reaction with antigens prepared from HEp 2 cells, using the tanned erythrocyte agglutination technique.

All pretransplantation sera gave negative tests.

Twenty-three of the 28 recipients of tissue-cultured cells developed antibody at serum dilution titers of 1:10 to 1:40, usually by 14 days after transplantation. Five of the 12 direct homotransplant recipients developed antibody, but titers were only 1:5 or 1:10.

*As previously reported, rejection of these cancer cell homografts in cancer patients **was significantly delayed** in about half of the recipients—as compared with rate of rejection in healthy recipients. There was no consistent relationship between homotransplant behavior and antibody response.*

The role of serum antibody in homograft rejection, and the ability of cancer patients to produce serum antibody, are discussed briefly.

Footnotes

These studies were supported in part by research grants from the National Cancer Institute of the United States Public Health Service and the American Cancer Society.

It was in this context of Southam's confusing and unethical medical practice on both ill patients and healthy people alike, that the University of Washington geneticist, Stanley Gartler, believed something was wrong with Krim's Cancer Virus Program's theory that "the cancer enemy" was a virus that caused the cancers, and was in error, regardless of the fact the notion had been advanced by heavy weights such as Mathilde Krim, Mary Lasker, the Hollywood Stars, and the Washington power-brokers, along with Presidents Johnson's and Nixon's efforts.

The problem, according to geneticist, Stanley Gartler, was black people. As a geneticist at the prestigious University of Washington in Seattle, he believed that only blacks carried the so-called G6DP-A variant -a marker of interest in the context of pathological anemias that had been tied in the past to blacks, and malaria.

This mistaken belief stood in stark contradiction to both the cherished ideas of a mythical human tumor virus of Krim, and ideas regarding spontaneous transformation of cells as being the causes of human cancers, that was an idea also being advanced by noted cell biologists of that day.

Walter Nelson Rees, a cell collector and cell bank archivist in Oakland California did a survey of the cells in his collection using Gartler's "black" G6DP-A enzyme marker, and to his surprise, many "normal" cells such as lung or liver cells in his collection tested positive for the molecular marker as HeLa cells had, as shown by their expression of the G6DP marker.

When other investigators used the G6DP-A test reagent in their labs, even when they were protected labs built during the early days of the "War On Cancer" era to contain the potentially contagious and deadly "first human cancer virus" if and whenever it was found, it also was concluded by Gartler and Rees, to the surprise of all of the cancer research community, that this black woman's immortal cervical cancer cells must have somehow contaminated the cell culture stocks in these buildings as well. But how could this contamination of the "Fort-Knox-like" buildings have occurred even in theory, not to mention, in reality? These were labs which never had had cervical tumor cells or HeLa cells in them at any time, and which as emphasized, were highly biologically secure installations.

Nelson-Rees announced at the meeting that he had found HeLa cells everywhere: even in the liquid nitrogen cell storage containers of “building 41,” which was the highest security laminar flow lab in the world into which HeLa cells had never been brought. His alarm about Henrietta’s supernatural cells was taken seriously by his superiors such as Head of the American Type Tissue Culture Collection, Robert Stevenson, who believed Stanley Gartler’s and Nelson Rees’s interpretation that the G6DP molecular marker was specific for black persons.

At an emergency meeting that was convened to discuss these supernatural black woman’s cells that were now believed to have integrated themselves throughout America’s cell repositories, Gartler and Rees proposed, and incorrectly of course, that Henrietta’s cells had wafted around the country riding on the backs of dust particles (the absence of an aqueous medium would kill any and all mammalian cells including cancer cells). According to Gartler and Rees, and being the strong black-woman’s cancer cells they were, and through Darwinian selection of course, HeLa cells had wafted around the country, they infiltrated laminar flow protected labs of the Cancer Virus Program, and they grew like weeds contaminating and out-dividing (being so prolific) all other [white] cell culture collections in the nation. The “black” cervical cancer cells “infiltrated” “everywhere.”

But Henrietta’s cervical cancer cells were not unique just because they grew immortally in Petri dishes. A gifted cell biologist named Leonard Hayflick, and many other cell biologists, also had grown human cells outside of the body in Petri dishes, including some “spontaneous transformants” (normal cells that turn into cancer cells) that were occasionally found in his Petri dishes of normal cell cultures, and no viruses were required. Hayflick, for instance, had obtained cells from his new-borne daughter’s amniotic fluid, taken them immediately back to his lab, and established the so-called “WISH” cells in culture. It was with similar techniques that could not possibly have introduced contaminant HeLa cells with which he went on to discover in various normal cell isolates what is known in modern cell biology as “The Hayflick limit,” which is a period of time and number of cell divisions relative to the lifespan of organism(s) from which normal cells are obtained.

The Hayflick limit is still used to explain why primary culture cells (cells derived directly from the normal cells and tissues living organisms) have the potential to divide in Petri dishes only a set number of times that is relative to the lifespan of the donor organism. According to the Hayflick limit, the doubling capacity of normal cells derived from living organisms are thought to somehow be mysteriously linked to the length of their lifespan (to this day we don’t know why). For example, in humans this ability of most normal cells constitutes approximately 52 cell divisions or generations (depending on the cell type), and about no more than 20 generations or geometrical cell doublings in mice. But the Hayflick limit, as it was called, applied only to normal cells. Modern molecular biologists claim that the Hayflick limit has something to do with the telomere lengths of chromosomes, and the amount of vanishingly rare and hard to detect “reverse-transcriptase-like” enzyme they called “telomerase.” Telomerase supposedly is present in all cells and the enzyme somehow miraculously is thought to regulate all chromosome telomere lengths. Its abundance or lack of abundance is also believed to immortalize cells, especially in the context of cancer. The telomerase hypothesis, however, failed to explain why mouse cells have longer telomeres than we do or how double-knock-out telomerase deficient mice live at all, yet can only live like all mice, no more than two years, while we typically live 70+ years. (Also, I have found, for instance, most malignant melanoma cells on earth can cells only be propagated for a few years, if at all, without supplying new frozen stocks-see Ref’s 1-6 of this document). WISH cells were not cancer cells and they obeyed the “Hayflick cell division limit” of only about 50 doublings.

Since Dr. Gey’s first pronouncements that he’d successfully cultured cancer cells for the first time, Gartler surmised that since he had failed to find new descriptions of new cell lines of normal origin published or discussed in the literature over a several year period since HeLa had been isolated and announced to the world, that this interruption in the published scientific record describing new cell lines signaled the fact that no new cell lines had been isolated since HeLa, because Henrietta Lacks cervical cancer cells must have infiltrated everywhere.

Henrietta’s immortal cervical cancer cells continued to generate confusion in scientific circles. Hayflick, and his functional

characterizations of such things as the doubling capacity of cells, were becoming increasingly marginalized as being “less objective” measures of reality, while the “more objective” yet invisible worlds of genetics, especially biochemical genetics, began to flourish and hold dominion over the other biological sciences. As an example of this genetic and biochemical bias in the minds of the emerging biologists like Gartler and Rees, and despite at that emergency called meeting surrounding the possibility of a “black” cervical cancer cell contaminant that had integrated into all American cell collections, Hayflick at that meeting pointed out: how he was able to isolate and grow normal cells outside the body; how not only cancer cells, but normal cells and spontaneous transformation of normal cells had been observed not only in his own work but the work of others; how no viruses were required; and how there was no need for cells to be initially derived from a tumor to become immortal. But the scientists, doctors, and Federally-backed administrators at the meeting liked the Gartler-Rees GCDP “black enzyme” hypothesis more to account for the believed HeLa contamination of the world, because they religiously believed Garler’s enzyme was a “more objective” marker of cells than anything that Hayflick or the cell biologists had presented or had to offer in defense of their years of work on isolating and growing normal cells, or Hayflick’s occasional observations or descriptions of spontaneous transformation In Vitro of normal cells. G6DP was a “genetic” and “biochemical” measurement, and therefore it was more inherently objective, and even despite the fact that it was a race-based marker hypothesis instead of a logical or rational one, they ignored Hayflick, and promoted the idea from then on that HeLa cells had supernatural abilities, and had wafted around on dust particles and integrated themselves into every cell culture collection in the nation.

How could this have happened among such intelligent scientists and doctors? Adam’s “Immortal Life Of Henrietta Lacks” cited before provides the answer. A table containing a list of Hayflick’s WISH cells, along with 17 other different human cell lines (i.e. human liver cells obtained from a Harvard lab, and a lab in Texas, where a cell line had been obtained that established the human chromosome number) were flashed on the screen at that special emergency meeting by Stanley Gartler. Gartler, then armed with his certainty about genetics and biochemistry and backed by the biases and fears of Federal Administrators, proposed that Hayflick’s daughter’s WISH cells, as well as the other 17 lines he listed, must all have been derived from a black person or from black people, because the specificity of his GCDP marker, and its association with black persons could not be in error as a biochemical certainty.

So persuasive was the new belief in Gartler’s “objective” biochemical genetic marker among the scientists, that after he presented the shocking news that Hayflick’s daughter’s cells and the 17 others he showed must have come from a black person, and with his assurances that his “genetic and biochemical” G6DP-A molecular marker couldn’t be mistaken about this fact, the bewildered Hayflick was heard to comment that, although he didn’t believe that his daughter’s cells could have come from a black person, Gartler’s presentation at that meeting had rattled him and prompted him to call his wife between meeting sessions from the meeting center, to ask her if she had been sleeping with a black man! And although Hayflick’s cell culturing expertise was second to none during his day, and as many would agree today, are still regarded as pioneering and valuable contributions, Hayflick’s admission about his phone call to those meeting participants evoked only uncontrollable laughter from the geneticists and others at that meeting (See **Appendix 1**).

In the hands of the Soviets, the “animal cancer viruses” given to them by The Americans only produced cancers in certain laboratory animals, as reported by the Americans previously (I suppose the Soviets didn’t have the nerve to inject live cancer cells into the forearms of their own Gulag prisoners as we had done with HeLa cells in Ohio, or 600 or more women undergoing gynecological surgery, or into 40 chronically ill old Jewish patients, to produce only benign lesions that were all rejected)?

When the Soviets announced their miraculous finding of the first human tumor virus they had believed they isolated from 5 different persons’ cancers throughout the Soviet Union, where HeLa cells had never been sent behind the Iron Curtain by Dr. Gey, the Soviet’s cells were immediately checked to see if they tested positive for G6DP-A, and, sure enough, they were found to be positive for “the black enzyme” also. The Soviet cancer delegation was angry, but they also conceded to Gartler’s genetic racial theories as had the Americans. Gartler’s and Rees’s finding had embarrassed the entire Soviet cancer effort, yet their pinning the Soviet finding of the

first Human cancer virus on a black person's supernatural cells due to a mistaken molecular marker in effect later saved the Sputnik-like race in favor of the Americans to find the first Human cancer virus. The finding of G6PD in the Soviet's claimed human cancer virus containing cells, meant to the scientists that they were HeLa or from a black person, and not transformed by "the first" human cancer virus. Still, nobody could explain precisely how Henrietta's cells could have wafted across the Pacific and into Soviet laminar flow hoods, or how it could be that there were so many clumsy soviet cell culturists to begin with.

As Wade Parks, a tumor virologist in the Special Virus Program, and a member of the American-Soviet delegation recounted in that Lacks documentary cited before, secrecy from then on surrounding the finding of HeLa cells everywhere and especially in the Soviet Union became a matter of National security.

Thus, like "HIV" would later become regarded as a matter of national security under the Clinton regime, HeLa cells, 3 decades earlier, became an issue of National security, as this secrecy helped to further foster the now HeLa-troubled Special Cancer Virus Program of Krim and others.

But even though the Special Cancer Virus Program tried to suppress to the best of their abilities the information about the supernatural HeLa cells wafting around the world in favor of the human cancer virus theory for reasons of National security, it later was revealed by Dr. Wade Parks in that same documentary interview, that another very important "virus-specific" enzyme would eventually be found in the Soviet's reported human tumor cells. This enzyme would come to be regarded as a functional relative of the imagined telomerases, and it would be called **reverse transcriptase (RT)**, and attributed to **a monkey virus contaminant**.

The belief that this so-called monkey virus contaminant-that wasn't really a virus but another molecular marker like G6DP in the Soviet's cells that expressed reverse transcriptase is critically important to appreciate. RT is the same enzyme that would become identified as a hallmark for ALL retroviruses in 1970 by Howard Temin and David Baltimore, and which would again lead to more Nobel prizes for cancer virus research.

How is it thought that this monkey virus came to infect G6DP-A-positive cells that were in the hands of the Soviets, who had proposed they found the first human cancer virus? The "monkey virus contamination" of G6DP-A-positive "black cells" was due, it was rumored, either to an unsubstantiated trip Henrietta was rumored to have made to visit African black friends of hers living in Moscow, or alternatively, to these black persons' association with monkeys in Africa before they emigrated from Africa. Park's disclosure about these suspected reasons that reverse transcriptase was found in HeLa cells, and the Soviet's cells foreshadowed the sad belief, that RT would become the principal molecular hallmark, and most important molecular characteristic thought to be present as a component of ALL retroviruses, especially several other imagined human rumor viruses yet to be "discovered" or described, that would become known as "HTLV-I" and "HTLV-II," and eventually of course, "HTLV-III," (which became "HIV," "a known variant of yet another "known human cancer virus").

Another reason why this reference by Parks about Henrietta's cells having reverse transcriptase is so crucially important to this story is because it is still constantly advanced today that the original transmission of "HIV" ("HTLV-III") must have occurred from zoonotic (from animals to man) exchanges between apes and monkeys to black Africans preceding the AIDS era. Therefore Park's disclosure as a Special Human Tumor Virus insider, that Henrietta's cells and the Soviet's cell stocks also harbored this monkey-contaminated retrovirus reverse transcriptase enzyme, presented the same kind of racist notion as to whether Henrietta Lacks, as an upwardly mobile black woman living in Post World War II Baltimore, or whether the African immigrants living in Moscow, also had any such "close associations" with non-human primates. It cannot be emphasized enough how important RT's role would play in both technical and sociological contexts, as it would become the hallmark of "the AIDS virus," and others, in the AIDS era that would unfold during the late 1970's and early 1980's.

Against all reason or logic, and with the subsequent irrational and almost immediate acceptance of the Garler's and Rees's genetic

and biochemical notions and G6PD-A marker as proof that HeLa cells had contaminated the world along with the dismissal of Leonard Hayflick's reservations, the cancer virus hypothesis began to lose steam. Yet, with the now mistaken belief that this black woman's cells had miraculously invaded even our cold-war enemy's labs, during the next decade, a second, third, and then a fourth human cancer virus was "discovered."

But unknown to those scientists, including Dr. Hayflick, the molecular marker G6PD-A was not specific to black persons. The imagined G6PD-black person specificity was merely an extension of a contemporary genetic hypothesis regarding (heterozygously manifested) anemia bestowing a survival advantage in both sickle cell anemia and thalassemia in malaria-infested regions of the world. And it was their faith in this so-called specific "black" marker that eventually led the scientists (not Hayflick) to the "inescapable" conclusion that Henrietta's cells had "integrated" themselves into every cell repository on earth, including Hayflick's, the Soviet's, and even into the most guarded Fort-Knox-like cell biological labs in Washington D.C., like Building 41, by riding on the backs of dust particles, or by riding on scientist's lab coats, or gloves.

We now know, from the pens of other geneticists, why the G6PD-A HeLa contaminant described by Gartler and Rees, and which didn't have these supernatural qualities, couldn't infect labs although frozen, couldn't survive atomic bomb blasts, and couldn't have penetrated the Fort Knox equivalent of the biomedical world (building 41), couldn't have wafted across the Pacific to infect the entire Soviet Union's cell collections, or emerged from black person's close associations with monkeys and apes in Africa, etc. It is because these cells were typified using a widespread non-specific molecular marker. Even if you believe in, or assume for a moment for argument purposes, that the "racial" specificity of this or any molecular marker was specific for any race or group of people, then you'd need to admit today that any cells that test positive for G6PD are more likely derived from cells obtained from Greek people living on the Isle of Rhodes, or perhaps from a Kurdish Jew. The so-called contaminants were less likely derived from any "negro's cells," that ruined the entire world and the war on cancer. From "*Emery and Rimoin's Principles and Practice of Medical Genetics*," Volume 1, David L. Rimoin, J. Michael Connor, Alan E.H. Emery, Reed E. Pyeirtz, we now read:

Glucose-6-Phosphate Dehydrogenase (G6PD) Deficiency Prevalence and Geographic Distribution

*This is the most common red blood cell enzyme abnormality associated with hemolysis (16, 72, 108, 117). This RBC enzyme disorder affects millions of people throughout the world, the highest frequency occurring in Mediterranean countries, Africa and Asia. **Approximately 10% to 15% of African Americans are deficient in erythrocyte G6PD activity and this is similar to 2% incidence in Sicily to over 20% on the Greek island of Rhodes. As many as 60% to 70% of Kurdish Jews may be affected, and in the male Asian population, the incidence of G6PD estimated to be 14% in Cambodia, 6% in South China, 3% in India, and less than 0.1% in Japan.***

1. *G6PDA+ is found in 20% of African Americans. It has normal catalytic properties and does not cause hemolysis (Class IV). It differs from G6PDB in that it has a much faster electrophoretic mobility. The structure of the G6PDA+ differs from that of G6PDB by the substitution of one amino acid, an asparagine for aspartate.*

2. *G6PDA- is the most common variant associated with mild to moderate hemolysis (class III), and is found in 10% to 15% of African Americans. Its electrophoretic mobility is identical to that of G6PDA-. This is an unstable enzyme and its catalytic activity is decreased in older RBC. Hence this variant is designated G6PDA- compared with G6PDA+.*

3. *G6PDMediterranean is the most common abnormal variant found in whites, particularly those whose origins are in the Mediterranean area and the Middle East. The electrophoretic mobility of G6PDMediterranean is identical to that of G6PDB, but its catalytic activity is markedly reduced and hemolysis can be severe (class II).*

4. *G6PDCanton is a variant seen in Asians. Its biochemical properties are very similar to those of G6PD-Mediterranean*

So do these G6DP distributions signal that perhaps Henrietta Lacks was part Kurdish Jew or perhaps part Greek? Modern population genetics teaches us that there is no such thing as any molecule that can specifically designate any “races” among populations within the human species, as no blood or genetic marker is known to specifically characterize any living human group in exclusion of any or all others. Gartler’s mistake, and its willing acceptance by the white biomedical community of his day, and the continued ignorance held today regarding many kinds of genetic and biochemical markers would suggest that indeed, if you believed in such markers, that Henrietta was most probably from a Kurdish background, or perhaps she derived from people of the Greek Isle of Rhodes. And although “genetic” studies recently were done on the blood taken from members of Henrietta’s surviving family members to see if her cancer was “genetic,” her brother claims they were not told Henrietta had cancer.

Genentech’s CEO, Dr. Raab, testified in that documentary that the money generated in biotech from her cells is incalculable, for which her family recently and politely received a plaque of recognition at Morehouse University (and not Johns Hopkins), and a fund was started so they could buy health insurance, while the world turned away for a moment, once again, from exogenous infectious human tumor viruses.

As the viral theory of cancer causation and cell transformation theory of cancer causation was being debated by the American scientific community and dismissed in favor of these supernatural black woman’s cells contaminating the world, most “legitimate” scientists became thereafter focused on oncogenes, where they remain largely unproductively engaged to this day, according to Harvard’s Richard Lewontin

In the oncogene (not oncogens of Rous) hypothesis of cancer, genetic damage is thought to arise in “the inside” of our cells rather than from outside. In fact, the concept of oncogenes is more consistent with Hayflick’s hypothesis proposing the spontaneous transformation of genetic instructions, and contrary to the “overwhelming evidence against this idea” mentioned by Rous in his Nobel speech, when he disparages “somatic genetic changes” as being a cogent reason for cancer. But this sad state of affairs did not become fully appreciated in the new molecular biologists minds before beliefs in supernatural cancer viruses and supernatural African American cells swept through the entire world. The idea became “fixed” amidst the latently problematic racist notion of an African American woman’s supernatural cancer cells’ abilities to float from lab to lab “on dust particles,” and at the same time, concerns over who would win the new arms race to find or perhaps even create a human cancer virus for the purposes of warfare consumed the genocidal and eugenic scientists, medicine men, and arms merchants on both sides of the Pacific.

POLYOMA VIRUSES CAUSE NON-SPECIFIC DISEASES AND ALMOST CAUSED THE SOVIETS TO LOSE IN THE 1964 OLYMPICS BECAUSE THEY ALL MIGHT HAVE CAUGHT CANCER FROM SV-40 IN THE SALK AND SABIN VACCINES

Before reviewing the evidence of cancer causation from oncogenes, there is yet another important discovery that needs to be discussed first, that also began during the 1950’s Special Cancer Virus, polio, HeLa, and spontaneous transformation era. This discovery involves yet another proposed group of so-called animal and human tumor viruses, known as “polyoma” viruses that are said to cause multiple forms of cancer. There were proposed to exist by Gross in 1953. This group of so-called animal tumor-promoting agents are extremely significant because of an inadvertent experiment performed on millions of humans, in which these polyoma viruses are said to have contaminated the first polio vaccines. But how could such a catastrophe happen?

During the Salk polio campaign in 1955, cells from the African Green monkey kidney were used to “grow” polio viruses in culture. But unbeknownst to the vaccine makers of that decade, a “latent” “SV-40 (simian cell vacuole-producing “virus” 40) molecular marker that these African green monkey kidney cells naturally harbored, was first suspected, and then were convincingly shown as biochemical markers like G6DP, to have contaminated the broths generated during mass production of the polio agent in pharmaceutical houses. Rous, in his Nobel speech, describes how this group of polyoma viruses was first proposed:

In 1953 Gross (Ludwik Gross, *Oncogenic Viruses*, Pergamon, Oxford, 1961) discovered a neoplastic virus [in mice] that has greatly widened knowledge. He obtained it from a carcinoma arising spontaneously in the parotid gland of a mouse and on inoculation into other mice it proved capable of producing tumors **of more than 20 kinds**, and some as well in rats, rabbits, guinea pigs, hamsters and ferrets. Because of these widely various neoplastic effects it has been aptly termed the polyoma virus. The growths it induces can be maintained indefinitely by transplantation, **and nearly always the virus disappears from them as time goes on**, yet the activity of the tumor does not lessen. Obviously in such instances its role is no more than that of an initiator, comparable in this respect to the chemical and physical oncogens [carcinogens]. Very occasionally though, it is an actuating agent as well, persisting and multiplying in a parotid cancer like the one originally providing it, and from **this** favorable growth it can be recovered anew and started again on its polyomatous career. **Under natural conditions the virus maintains itself as an infectious agent widely prevalent in mice but causing only a trivial, scarcely perceptible illness of non-neoplastic sort save in those rare instances in which it produces a tumor.**

Gross himself, however, initially published that his virus was not a virus, but, **a filterable “agent:”** [like **a protein** perhaps rather than a virus]?

GROSS L (June 1953). “A filterable agent, recovered from Ak leukemic extracts, causing salivary gland carcinomas in C3H mice”. Proceedings of the Society for Experimental Biology and Medicine. Society for Experimental Biology and Medicine 83 (2): 414–21.

This is a significant admission because of what we already have discussed in the contexts of Rous, Shope, Beard, Krim’s promised and imagined human cancer viruses, and of course our discussion regarding how HeLa cells ruined the Special Cancer Virus Program because of racism, which in time, erected new hunts for human cancer viruses as we will discuss later. What is most significant to appreciate about the so-called “polyoma cancer viruses” is how they became suspected of contaminating the polio vaccines, and despite the horror this discovery caused, the contaminant was injected directly into millions of human arms or buttocks during the polio crusades, probably without causing cancers in human populations that numbered in the millions, in a way very similar perhaps, to the manner in which Chester Southam injected live cancer cells into scores of sick Jewish people, prisoners, and about 600 women without causing cancer.

Immediately following the polio vaccination program of 1955-60, Bernice Eddy, Maurice Hilleman of Merck, and others at the Fox Chase Cancer Institute in Philadelphia discovered what they thought was **a putative primate** “polyoma cancer virus” for the first time in (a primate’s cells) monkey kidney cell cultures used to generate the vaccine. This “contaminant” that they termed SV-40 was similar, they believed, to the “agent” Gross had described, because Bernice Eddy also noted that the monkey cell-derived filterable agent could induce multiple types of lesions in small lab animals (again no specific single cancer or disease was produced). SV-40 did **NOT** induce cancer(s) in the African green monkey kidney cells in which it was initially discovered, and only caused lesions in small lab animals. However, it both amused and horrified these researchers to realize that the first polio vaccines were actually made in these same cultures with these same African green monkey kidney cells that harbored this same “simian SV-40” agent, used to make vast quantities of the first Salk vaccines injected into millions.

Thus, in John Enders day, an almost pathological and psychotic fear of the biomedical research establishment was realized by all of the scientists, and this alarm of theirs was even published in the New York Times. A potential human tumor virus, derived from a primate’s kidney cells known as SV-40 had inadvertently been given to millions of human beings on several continents by accident during the Salk and Sabin polio vaccine crusades. After Eddy’s discovery of SV-40 in primate kidney cell cultures, it was feared that entire nations would come down with cancers, and particularly mesotheliomas and brain cancers, that affect the lungs and brain.

After she alerted her superiors that this new cell vacuolizing agent could induce mesotheliomas and other cancers in hamsters, Dr. Eddy was severely censored and marginalized for experimentally demonstrating this phenomenon. Several years later, after the Salk vaccines had been deemed a failure, and the huge Sabin vaccine campaign using live viruses which also was made initially in

cultures contaminated with SV-40 was developed to replace it, jokes were advanced by certain politicians that the Soviets would lose the 1964 Olympic games because they all would come down with cancers. There had been massive Sabin polio campaigns executed there with demonstrated SV-40 contaminants obtained from nasal swabs following inoculation with “live poliovirus” via the oral route, and from seroepidemiological studies. As with most, if not almost all vaccine trials, the polio vaccine trials were immediately halted in many nations. Despite the fact that other vaccine makers weren’t so willing to stop producing the “polio vaccine,” Merck decided to stop its polio vaccine program and eventually most other vaccine makers followed Merck’s lead. Unfortunately, many doctors never received the official edict to stop their live “enteriform” (feces)-derived-polio and SV-40 contaminant vaccination experiments on their patients, and they continued to administer them to millions.

But many doctors were confused, and still are. In 1955-6, and before SV-40 had been discovered as a contaminant in the polio vaccines, the nation was still terrified by the increases in paralysis following those first Salk polio vaccination crusades in California, Idaho, Massachusetts, and the South. It was widely known that the Salk polio vaccine made by Cutter and by several other vaccine makers, had increased paralysis rates as much as 3-600 times above normal background incidence in some of these states. The first hard evidence that paralysis was being caused by the Salk polio vaccine occurred when children became paralyzed in the limb where they were injected, at such institutions as Michael Reese Hospital in Chicago, and then in California.

The African green monkey kidney-cell-derived “vacuolating agent” had induced many kinds of different tumors and tumor-like growths in experimental animals, which is why it was believed to be “a polyoma virus” like Gross’s that he found in mice. And like Gross’s filterable agent, SV-40 also was a “filterable agent.”

Consistent with the oncogene hypothesis to be discussed next, it is now believed that the cellular and oncogenic large and small T-antigens are thought to interact with SV-40, and that p53 and retinoblastoma expression in SV-40-“infected” cells becomes disturbed on exposure to the “simian vacuolizing agent, to cause transformation (cancer).

Eventually, Enders avoided the problem of growing the feces-derived “polio virus” extracts in the reportedly SV-40 “cancer virus”-infected African green monkey kidney cells or HeLa cells as had been tried by others, by using human embryonic cell cultures instead, for which he received the Nobel. Yet today, during the 2000-2008 Bush Administration, and because Ender’s cells are human embryo-derived, these cells have been outlawed for biomedical research on moral grounds. Yet this use of human embryonic cells by John Enders rather than primate kidney cells (or HeLa), circumvented the imagined possibility that future putative contaminating “cancer viruses” or other viruses thought to come from non-human primates, or especially from “African green monkey kidney cells,” wouldn’t again be injected into millions of people in future vaccine crusades, while it was of little concern to anyone, that the polio agent itself was derived from extracts of human excrement. I worked in his building for about 6 years, at Harvard, but it was a subject nobody wanted to discuss.

Eventually, however, even the selfless front line doctors, and researchers, and infectious disease warriors, realized that the SV-40 vacuolizing agent apparently did not induce cancers in the vast human polio experiment when SV-40 was supposedly co-injected along with formalin fixed or attenuated live poliovirus directly into the bloodstreams or mouths of millions of unsuspecting children and adults. Yet to this day, it still frightens many scientists (and public health officials) that SV-40 genomic sequences are found in many tumors, but it puzzles them as to why the SV-40 molecular profile also is found in a plethora of non-cancer contexts, and in people without cancer and who never will develop cancers. For many, association still means or implies causation like the possibility that skid marks cause car accidents, or the fact that building fires and fire trucks are frequently associated, and therefore fire trucks must cause fires?

Vaccination does not induce an immunological state equivalent to natural immunity. Many investigators have suspected and written volumes about the possibility that in many communicable illnesses in which antibodies that are considered protective and are generated as a response to a microbe, that in most cases, naturally acquired infections are not accompanied by significant morbidity,

or indeed, any clinical symptoms at all, in the vast majority of those who will test positive as evidence of exposure.

Similarly, the molecular profiles or markers of “HTLV-1” “HTLV-II,” “HIV,” hepatitis B and C, Papilloma (HPV), herpes, SV-40 are exhibited by those who “test positive,” and yet most live in a healthy “carrier” state all their lives. Clearly, in so many different contexts, molecular signatures (antibodies as evidence of disease exposure or vaccination) do not equal protection afforded by natural infections, nor do they predict the future occurrence of actual disease.

From “The Virus and The Vaccine-The True Story Of A Cancer -Causing Monkey Virus-Contaminated Polio Vaccine, And the Millions Of Americans Exposed, by Debbie Bookchin and Jim Schumacher, St. Martin’s Press, 2004, we read:

*“The first notice to the general public about SV40 came in July 25, 1961. Associated Press filed a story announcing the surprise cessation of Salk vaccine production by both Parke Davis and Merck. The story ran in the New York Times on page 26. Its placement in the newspaper and the fact that the Times did not assign any of the several science writers on its staff familiar with polio to cover the story suggests that **DBS’s (Division of Biologic Standards) effort to downplay SV40 had paid off.** The article quoted directly from the DBS press release in several places; the Times subhead to the story said SV40 was ‘believed harmless,’ and the body of the story repeated the NIH reassurance that ‘there was no evidence that small amounts [of SV40] when introduced through the vaccine produced illness in man.’ **The words ‘cancer’ and ‘tumor’ never appeared in the AP write-up.***

*The story behind the story was much more interesting. Merck had stopped shipping Purivax (its ‘purified’ version of the Salk vaccine) as soon as its own tests in May 1961 confirmed that the vaccine was contaminated with SV-40. **Its unilateral withdrawal of vaccine from the market had not been well received by the DBS.** If Merck recalled vaccine, then everyone else would have to. That would have resulted in public panic and would have run counter to the Technical Committee’s May 18 directive that polio vaccination ‘continue to be pursued with vigor with the materials presently available.’ In June, after the Girardi cancer results had come in, Hilleman (Merck’s science director) had tried one more time to get all vaccine production halted. That suggestion, as we have seen, was rebuffed. Merck had already suspended production and was trying to figure out how to screen SV40 out of the vaccine when DBS tests on vaccine samples indicated that Parke-Davis supplies were also badly contaminated. Parke-Davis now also stopped vaccine manufacture. The truth was that by the time the Associated Press reported the ‘news’ in late July, both companies had not produced vaccine for several weeks. Parke Davis eventually resumed production, but Merck would soon decide that producing a polio vaccine that at times might be contaminated was not worth the risk. In vaccine circles, Purivax was now derisively being called ‘Imprurivax,’ and Ben Sweet was labeled the ‘million dollar man’ because that was the cost of the vaccine program that had just been killed by his discovery of SV40.*

*...But other than the reports in the Associated Press and the National Enquirer, there was no more news for the remainder of 1961 about SV40. Hull was in the midst of conducting his own experiments at Eli Lilly on SV40. He had found, just as Eddy and Girardi had, that the simian virus caused cancer in suckling hamsters, but his results were never published as a scientific paper. At Merck, Girardi and Sweet began a different set of SV40 experiments, **but these were halted before completion. The pair had discovered that when SV40 was injected into tissue cultures of normal human cells it ‘transformed’ them into cancer-precursor cells.** Hilleman decided, however, that this alarming development was not going to emanate from Merck. There was only so much self-inflicted damage (‘hanging out dirty laundry’ were Hilleman’s words according to one of his subordinates) that the company could take about its SV40-contaminated polio vaccine. Instead, to Sweet’s displeasure, Hilleman contacted John Enders at the Harvard Medical School and sent him some SV40 and encouraged Enders to undertake the same experiment.*

Girardi had also started another experiment that was never to be completed. From throngs of monkeys that came through Merck, he had found nine non-rhesus pregnant females. After they had given birth, he injected six of their newborns with SV40, leaving

three as controls. The significance of this experimental design was that monkeys are far closer to humans than hamsters. Whatever might happen to them after SV40 exposure would provide a strong signal of what the virus might do after it had been injected into people. **Before Girardi could continue much farther with the live monkey experiment, word came down from higher up at Merck to quit the project.**

The next big news about SV40 came in mid-April 1962. The American Association for Cancer Research, the organization that still publishes the influential scientific journal *Cancer Research*, was holding its annual meeting in Atlantic City, New Jersey. The association's annual weekend meetings were often the occasion for the announcement of important breaking news on the cancer front, and lay press interest in the conference was considerable. On Sunday, the last day of the scientific gathering, Girardi presented a summary of his Merck experiments that had showed SV40 produced tumors in newborn hamsters. At the very end of his report, he announced that he and Sweet had also found SV40 transformed human cells *in vitro* (in tissue culture as opposed to *in vivo*, in a living organism—my emphasis). Earl Ubell, the president of the National Association of Science Writers at the time wrote up the Girardi presentation for the *Chicago Sun Times*:

'Polio Vaccine Virus Puzzles Scientists'

Atlantic city, N.J.—Those strange viruses found floating alive in both live and killed polio vaccines display increasingly disturbing peculiarities...

A year ago, it was reported for the first time that something in the monkey cell culture broth could cause cancer in hamsters. A few months ago, scientists at Merck & Co., identified that 'something' as SV40. Now, these same Merck researchers have found that SV40 will grow in human tissue kept alive in a test tube. They will make the cells in those tissues multiply at a greater rate.

Sunday, another report said SV40 can get into human tissue cells growing in test tubes and change the microscopic chromosomes, destroying one of the 46 (chromosomes)...

*Ubell's article finished with a description of three theories under debate at Atlantic City after Girardi's report. One was that SV40 was a human carcinogen, a prospect Ubell described as 'the most frightening idea' since 'millions of persons' had received Salk injections and SV40 had been administered alive to them. A second conjecture was that SV40 was totally harmless. The third was the somewhat wishful notion that the massive exposure to SV40 would somehow act as **an anticancer vaccination** for Americans. (Ubell labeled this 'a far out idea'). Time magazine also reported on the conference, noting that SV40 was **'the first primate virus shown to cause cancer in any animal.'** In the article, Bernice Eddy, for one of the few times in her career, was publicly credited for her contributions to the understanding of the relationship between viruses and cancer.*

*...Girardi joined the staff of the Wistar Institute a year later. One of the many discoveries he made about SV40 during the next several years was that it sometimes took **more than a month** before **the virus** grew out and could be detected in tissue culture. The DBS, based on Gerber's experiments, believed that **fourteen days** was a sufficient observation period to detect SV40 and had drafted its new regulations for oral polio vaccine **on that assumption**. Manufacturers were required to look for evidence of SV40-induced cell damage for only **two weeks** in various tissue culture safety tests. Any slower growing SV40, such as the kind Girardi had discovered, would not be uncovered by following these new regulations. Girardi says that his findings about how long it sometimes took SV40 to appear in tissue culture were communicated to the DBS as soon as he discovered them in the early 1960's. **The agency, however, never changed this section of the vaccine regulations to lengthen the observation time.***

Having secured another promising scientist for Wistar and some crucial knowledge, Koprowski rushed back to Philadelphia and personally oversaw the completion of research already underway at Wistar on SV40 and human cells. Cultures of skin and cheek-

lining cells had been infected with SV40 by a team of five Wistar researchers. **The SV40-infected cultures multiplied at out-of-control rates and piled up on top of each other-bizarre behavior when compared to healthy human tissue.** The SV40 cultures also had readily discernable chromosomal abnormalities, confirming the unpublished Girardi research. (The unpublished Wistar paper included photomicrographs of rearranged and fragmented chromosomes). All were unmistakable signs that the cells were no longer normal and were well on the way to malignancy. Worse, the Wistar team concluded that the 'transformed cells' seemed to have a selective advantage over 'normal cells.' **Apparently, not only could SV40 turn normal human cells cancerous, it also turned them into bullies-completely overgrowing uninfected cells until they were suffocated to death.**

The Wistar human tissue study appeared in midsummer 1962, shortly before the human tissue study that Enders had completed at Hilleman's urging. Enders and his collaborator, another Harvard researcher, Harvey Shein, reached essentially the same conclusions as the Wistar group, with a different kind of tissue, human kidney cells. Koprowski had rushed the Wistar study into press **hoping to scoop Enders and gain some publicity for Wistar.** But in the end, despite being second, the Enders study attracted a good deal more attention because it was published in the prestigious Proceedings of the National Academy of Sciences. A lengthy New York Times story on August 10, 1962, reported the Enders study:

'A cancer-causing virus has for the first time produced cancer like changes in human cells...

Changes that the virus produced in cultures of human kidney cells included greatly accelerated growth patterns and chromosomal aberrations..

The virus, the Times said, was SV40. The Times story described Eddy's SV40 discovery, noting that 'fortunately' her original findings had come before the use of any commercially licensed oral vaccines. But, the story continued:

There is no doubt, however, that a large part of the Salk vaccine and the live-virus Sabin vaccines that were used in clinical trials throughout the world were contaminated with SV40 virus.

*The Koprowski and Enders studies fit the last pieces into the SV40 puzzle. At first there had been a question as to whether SV40 was even a concern, since it was believed that it had not actually made it into the final Salk vaccines. **It was now known that it had contaminated most of the polio vaccine ever produced.** Next there was debate about whether SV40 was infectious in humans. The Anthony Morris study on prisoners showed that the monkey virus, **when ingested or inhaled,** multiplied inside humans. A 1962 study by Baylor University's Joseph Melnick, which found that **children who had taken contaminated Sabin vaccine excreted the virus in their stools for up to five weeks,** reinforced the point. Obviously, when injected-a far more potent transmission route than oral or nasal exposure-SV40 would infect and multiply in humans. Then, there had been doubt whether the virus would produce anything beyond the mild subclinical illness that Morris had observed among **his prisoner volunteers.** The Eddy and Girardi experiments both demonstrated that the virus and lethal consequences for laboratory animals. Presumably, the virus could do something similar to man. Still, **skeptics pointed out, tumors in hamsters were not necessarily relevant to human beings.** Now that point, too, had been addressed. What SV40 did to hamsters, it could do to human cells **in a test tube.** Chromosomes had been damaged, and normal cells had been rendered malignant. SV40 was as dangerous as many of attendees at the American Association for Cancer Research had suspected four months earlier.*

*When Enders and Koprowski's studies on human cell transformation by SV40 were published in the spring and summer of 1962, it seemed as if everyone's darkest apprehensions about the polio vaccine contaminant had suddenly come to life. By the fall of 1962, as news of the most recent SV40 research spread, the anxiety that had been growing in scientific circles about the simian virus reached its zenith. **'It was the worst thing in the world,' Hayflick recalls of the news.** 'Please tell me: What else could we*

find worse in monkey kidney cells? In Britain, Wellcome Laboratories decided to stop inactivated vaccine production and switch entirely to live polio vaccine production.

As in the United States, however, both the British and Canadian governments decided not to recall old stocks of Salk vaccine. Britain had a surplus of 6 million injections in 1961. In Sweden, the concern was about Sabin-type vaccine. There were plans to give monkey gamma globulin to **four thousand children** who had received oral vaccine **in the belief** that it would contain antibodies against any simian viruses, including SV40, which might have contaminated the oral doses. In the Soviet Union, site of the most extensive use of Sabin's vaccine, tests were conducted to determine the spread of SV40. Many of the technicians and scientists involved in Chumakov's massive vaccination trial proved to have been infected by **the virus**, and the Soviets were now fearful of SV40's possible long-term effects. **Among American research and health officials, a joke with gallows-type humor began to make the rounds: The Soviets would lose the 1964 Olympics because their athletes would all have tumors thanks to SV40.**

But in Bethesda, even this jibe at the cold war enemy was of little comfort. The DBS's own research was suggesting that SV40 could no longer be downplayed as a health threat to the American public. The division, to its credit, had become quite busy research SV40 during the past year. **Gerber's study confirming that the virus was not killed in Salk's vaccine** had been published in the spring of 1962, and there were a dozen or so other SV40 research projects now under way. None seemed to offer reassurances that the virus was as inconsequential as Murray and Shannon had believed (or hoped) in 1961.

A young DBS researcher named Alan Rabson-future deputy director of the National Cancer Institute-found that SV40 caused ependymomas, a rare brain cancer, in a species of rats. This was the first proof that the virus could cause cancer in a mammalian species other than hamsters. Another DBS experiment led by Rabson determined that when human thyroid tissue was infected with SV40, it became cancerous. When the infected human thyroid cells were, in turn, transplanted into the brains of hamsters, the hamsters developed ependymomas. Ependymomas were also induced in hamsters by Gerber, who inoculated the animals directly with SV40. In a third Rabson experiment, SV40 was found to produce kidney cancer in hamsters. Interestingly, a coauthor on all of these newer DBS tumor studies was Rabson's wife, Ruth Kirschstein, the pathologist who two years earlier had refused to participate in Bernice Eddy's original hamster tumor study.

Gerber, meanwhile, confirmed that SV40 was a DNA virus -and that it had a preference for invading the nuclei of infected cells. He also found that SV40 seemed to go into hiding once inside the cell, yet could reemerge much later and still cause cancer in the hamsters he was using for his experiments. This seemed to suggest that the virus could perhaps 'go underground' in humans, as the New York Times termed the phenomenon, and theoretically do damage long after initial exposure.

Now that the NIH's own research had established the potential carcinogenicity of SV40 virus, the health agency was confronted with a very frightening public health question. **Almost half of the American population had received Salk vaccine by 1963. Were the nearly 100 million Americans who had been potentially injected with live SV40 in contaminated Salk vaccines going to contract cancer?** Attempting to answer that question became a complex research project that fell to a young physician named Joseph Fraumeni.

The group decided to focus just on children who had been enrolled in the National Foundation's 1955 spring and summer immunization program. Gerber would test samples of 1955 vaccine and determine which lots were contaminated with SV40. Lanmuir would figure out where the contaminated lots had gone. Fraumeni would collect **the mortality data** from an HIIH data repository. He would look at overall cancer mortality in general and for cancer deaths specifically attributable to brain, kidney, and connective tissue cancers-the kinds of cancer that Eddy, Rabson, and other DBS researchers had most frequently induced with SV40.

...When it was published in JAMA at the end of August, 1963, the conclusion of Fraumeni and his boss, Robert Miller, the study's only authors, was that, despite the 'questions about SV40's oncogenic potential in man,' their investigation had found there were 'no significant alterations **in mortality rates for cancer** for **the three cancer types surveyed** and that overall cancer mortality rates appeared unaffected. There had been **a blip** upward in leukemia rates, but as far as the authors could determine, SV40-exposed and unexposed children alike had higher rates, so contaminated vaccine was not at issue.

The take-home message from the Fraumeni study was obvious: Despite all the fears and worries of the past three years, **SV40 had no measurable consequence for human health**. Even though Fraumeni was careful to state in the JAMA paper that 'it would be premature to conclude from this study that SV40 is innocuous to man,' that was exactly how his study was interpreted.

Because it seemed to definitively dispel the SV40 anxieties, the NIH was eager to share the Fraumeni study with the public, releasing it to the press even before its JAMA publication—a move designed to heighten interest. Just as in July, 1961, when news about SV40 first became public, mainstream media coverage ran true to form—an uncritical presentation of the NIH's interpretation of the results. '**Public reassured on Polio Shots; US Finds No Links to Cancers**' ran a New York Times headline to a story about the Fraumeni study.

...Especially with the passage of time, most physicians stopped worrying that they might have harmed their patients with tainted Salk vaccine. Future generations of medical practitioners, if they learned about SV40 at all during their medical training, would find it related to them as a novel bit of medical history—an odd virus that had once contaminated the polio vaccine, but had proved to be inconsequential. Indoctrination within the medical establishment about the putative harmlessness of the virus had begun.

Despite the import attached to it, the 1963 Fraumeni study design was clearly lacking when measured against rigorous epidemiological standards. Fraumeni himself says that '**the study had lots of limitations and caveats**'.

...The first limitation was acknowledged by the authors. **They had only followed the children for four years, so any cancer that took more than four years to develop, even if SV40 were the culprit, would have gone undetected**. Many cancers have more than a four-year latency period—a fact known at the time. In fact, it would be fairly unusual for any carcinogen—unless applied in very high doses, such as radiation after a nuclear explosion—to produce cancers quickly on a large scale. This limitation alone suggests that a much longer follow-up of the children was warranted. This never occurred [yet at this time—my emphasis—it would later be—see Pankhurst's study described below].

Another limitation was also acknowledged by the authors: The study would have failed to detect 'small differences' in cancer rates caused by SV40; it was sensitive enough to notice only 'gross variations' in cancer occurrences...

A third limitation concerned the types of cancers surveyed—just three types—brain, kidney and connective tissue. **A number of cancers with which SV40 was later associated were not included in the study. Mesothelioma which has a two-to-four decade latency period, was not included, nor were lymphomas**.

A fourth limitation centered on how 'a cancer' was defined. Only cancer deaths—as opposed to cancer diagnoses—were included. This excluded any cancer contracted by children who were ill, but still alive, in 1959, four years after vaccination. Another problem lay in determining who was defined as having contracted cancer. Cancer diagnoses and statistics are considered inherently unreliable the further back one goes in time. The federal government did not even begin to maintain its own database of cancer cases until **1972**....

The final limitation was the study's definition of who was exposed to SV40 and who was not. The study assumed, on the basis of Gerber's tests of polio vaccine samples, that the continental United States could neatly be divided into three SV40-exposure

'cohorts,' or population study groups: high SV40-exposure states, low SV40-exposure states, and no SV40-exposure states...All of the study's conclusions on SV40's influence on cancer incidence were predicated on the accuracy of these cohort assignments. The validity of Fraumeni's cohort definitions, in turn, was dependent upon the assumption that the Gerber tests of vaccine lot samples always detected SV40 if it were present-an assumption that Tony Giraldi ...proved false. At the time, the DBS detection methodology was to observed tissue cultures **for only fourteen days**. However, as Girardi discovered, some strains of SV40 take longer to manifest themselves in culture. The DBS detection protocols would have missed any 1955 vaccine lots that contained such slower growing SV40. These lots would have been erroneously defined as SV40-free in the Fraumeni study-thus casting into doubt Fraumeni's entire basis for comparing SV40-exposed states to SV40-unexposed states. (There was also the possibility that in some states defined as 'high exposure' states, some of the vaccine used was actually free of the virus at times).

Even assuming that the paper's state-by-state assignment of SV40 exposure levels were flawless, there were still other problems with how SV40-exposed cohorts were defined for the study. Fraumeni, for example, did not really know the number of children aged six to eight during May and June 1955 for any given state. He instead took census data from 1950 and 1960, state by state, and, using that data, **estimated** the population of children aged six to eight for each state-extrapolating the 1955 ages six to eight population as the midpoint between 1950 and 1960. Secondly, Fraumeni did not really know who received polio vaccine in this population. He assumed that everyone age six to eight was vaccinated in 1955-or at least that the rate of vaccination for this age group did not vary from state to state. **But with the Cutter incident dominating the news at the time, many parents withdrew their children from the National Foundation's free immunization program.** Rates of withdrawal (and therefore vaccination) did vary from state to state, thereby making it impossible to assume that the percentage of children vaccinated in state A was the same as state B. The final flaw with the cohort design was that Fraumeni assumed that none of the children moved from one state to another from 1955 to 1959-or if they did, they conveniently always moved from a high SV40-exposure state to another high SV40-exposure state, and never to a low SV40-exposure or no-exposure state.

Taken together, the flaws in the Fraumeni cohort selection add up to one important shortcoming. Defining who was exposed to live SV40 in contaminated vaccines is impossible. **This same flaw has been present in every subsequent attempt to use epidemiology retrospectively to determine whether or not the virus is causing cancer in human beings.** In 1963, Fraumeni had no way of being certain which children actually received live SV40 in their polio shots; no epidemiologist since has been able to clear this technical hurdle. Looking backward in time, it is simply impossible to know for sure which individuals were exposed to SV40 and which were not.

Taken as a whole, the flaws in the 1963 study suggest that its conclusions were open to challenge, if not highly suspect. Yet no effort was made to do a more precise or more thorough subsequent study on Salk vaccinees, despite the fact that one-half of the American population had received potentially SV40-contaminated Salk vaccine...

Interestingly, after Fraumeni's 1963 study, there were epidemiological studies that showed cause for concern in connection with Salk vaccines. A 1968 Australian study of several hundred hospitalized children with malignancies showed they were more likely to have received polio vaccine, while two American studies in the 1970's found an increased brain cancer incidence among groups of children born from mothers who had received Salk vaccine during pregnancy. Even though these epidemiological investigations contradicted Fraumeni's findings, the studies by the NIH researcher held sway...

...One more startling paper about SV40 was still to come from Wistar. In April 1964, Koprowski reported at the American Association for Cancer Research that a Wistar team had injected SV40-transformed human cells under the skin of terminal cancer patients, and lumps had formed that, while not specifically cancerous, appeared precancerous in nature. But even when reporting this development, the New York Times stressed that polio vaccines were now free of SV40 and 'that there has been no evidence that its former presence has done any harm.' This would

prove to be the last concerted effort for nearly three decades to determine whether the simian virus could cause human disease. Koprowski was more interested in vaccines than in cancer, and no other private researchers picked up where he and his Wistar team had left off. In the minds of most public health officials, doctors, and science writers, the virus reverted to its June 1960 status: an annoyance to vaccine makers, a virological curiosity because of its cancer-inducing properties in animals, and of no consequence to humans.”

A carefully controlled study, conducted for 35 or more years, that followed SV-40-contaminated polio-vaccine recipients has been reported. Its authors claimed that despite the fact that SV-40 was injected directly into the arms or buttocks of millions of infants and children, or given orally, an increase in cancer from it apparently has not resulted. In their 2001 publication, Pankhurst et al. warned that their 35-year post-polio vaccine mortality study of infants injected with the vaccines contaminated with SV-40 has not been long enough to unequivocally determine if SV-40 is contributing to escalating cancer rates. Merck, oddly enough, which now aggressively advertises that after only about 5 years, their vaccine Gardasil is 100% effective, employs different standards of science to claim their cervical cancer vaccine can prevent cancer in as little as 5 years. But as a biologist, one couldn't ask for a more convincing experiment relevant to human beings, that to date, a virus or agent that can be shown to cause cancer or many ill-defined diseases thought to resemble cancers in inbred lab animals, such as hamsters, or which is peacefully situated in monkey kidney cells without causing cancer, may not be able to cause cancer in Humans either, even as potentially devastating as this mass human experiment could have turned out, if it did, due to vaccination crusades.

The thirty-five year mortality study on people now in middle age following receipt of SV40-simian-(cancer) virus-contaminated and feces-derived polio vaccine show that out of 1073 newborns that were vaccinated and carefully followed for 35 years, (which the authors cautiously claim is not really long enough to determine if the agent causes cancer in humans), among these infants and among the millions of individuals who were given this “cancer virus-contaminated vaccine ” between 1959 and 1963, there has been no apparent increase in cancer above the expected background incidences in this carefully followed subgroup, or in the world, according to Carroll-Pankhurst et al., in her 2001 study published in the British Journal of Cancer. There are some scientists, however, such as Michele Carbone, and a group in Australia, that believe SV-40 has caused increases in mesothelioma, brain cancers, leukemias, and other cancers in Humans, but their groups have not been studied as long as Pankurst's.

Cancers that are diagnosed based upon sequence data, instead of viral isolation, and demonstrated transformation ability, cannot be considered cancers due to exogenous viruses or retrovirus.

SV-40, the contaminant of the polio vaccine that was feared might cause epidemic cancers was placed into the family of viruses known as polyoma viruses because, as stated in dictionary definitions:

*[Polyoma viruses] are DNA-based (double-stranded DNA, ~5000 base pairs, circular genome), small (40-50 nanometers in diameter), and icosahedral in shape, and **do not have a lipoprotein envelope**. They are **potentially** oncogenic (tumor-causing); they often persist as **latent infections** in a host without causing disease, but **may** produce tumors in a host of a different species, or **a host with an ineffective immune system**. The name polyoma refers to the viruses' ability to produce multiple (poly-) tumors (-oma).*

But, most important to this discussion regarding the imagined effect of this “cancer causing agent” in humans, it is now thought that there are at least several polyomaviruses found in humans. In addition to SV-40, It is now believed that:

*JC virus, can infect the respiratory system, kidneys, or brain (sometimes causing the fatal progressive multifocal leukoencephalopathy in the latter case), and BK virus, which produces a mild respiratory infection and can affect the kidneys of **immunosuppressed transplant patients**. Both viruses are very widespread: approximately **80 percent of the adult population in the United States have antibodies to BK and JC...** but yet 80% of us do not have cancer.*

The polio vaccine era taught three lessons. 1) That a potential human cancer-causing contaminant, SV-40, has not increased cancer rates among millions of human polio vaccine recipients after direct injection into their bloodstreams or through oral application, although 80% of adults are said to “test positive” for at least one type of cancer-causing polyoma “virus” or another; and that 2) polio vaccines increased the rates of polio. The evidence for this second lesson comes from the CDC, because, as recently as in 1992, America’s Centers for Disease Control (CDC) in Atlanta admitted that the polio live-virus vaccine had become the main cause of polio in the United States. Specifically, the CDC asserted that, from 1973 to 1983, 87% of all (non-imported) cases of polio resulted directly from vaccine administration. Even more amazingly, it was asserted that every non-imported case of polio in the United States from 1980 to 1989 was vaccine-induced (Strebel, P. M., et al., *Epidemiology of Poliomyelitis in the U.S. One Decade after the Last Reported Case of Indigenous Wild Virus Associated Disease*, *Clinical Infectious Diseases*, CDC, February, pp. 568-579, 1992). 3) A third potential lesson that the SV-40 contaminated polio vaccine era teaches us is that known escalating cancer rates that are occurring in all human populations as shown by statistics kept during the past 40 years may be more directly attributable to environmental toxins and carcinogens, than any rumor viruses that are believed, but yet have not yet been shown, in any context, to cause cancers in humans, which will be elaborated on later in the discussion regarding how to stop cancer before it starts.

REVERSE TRANSCRIPTASE AND ONCOGENES- NOT ROUS’S ONCOGENES

For the so-called discovery in 1970 of the supposedly retrovirus-specific enzyme, reverse transcriptase (**RT**), later found to be another non-specific molecular marker present throughout the normal biological world, and now known to be a normal protein associated with normal chromosome structure, Howard Temin and David Baltimore received a Nobel Prize only 5 years later in 1975. This is the enzyme we discussed earlier, that was thought to have infected HeLa cells, because Henrietta Lacks was rumored to have gone dancing at a Moscow dance club, where somehow her cells became infected with a monkey retrovirus, which ended up destroying Mathilda Krim’s Special Cancer Virus Program because of Stanley Gartler’s racist beliefs about the GCDP-A marker, which the scientific community accepted. It was during this decade that the detection of RT in HeLa cells had been advanced as a principal reason why The Soviets had failed to find the first human cancer virus, as HeLa cells derived from the Soviet collection were found to be riddled with RT activity. With this announced discovery, biochemical acceptance of RT, and Nobel bestowed for what would be thought to be a specific retroviral marker, and indeed the hallmark of retroviruses, a new cellular oncogene hypothesis began to flourish in the reductionistic minds of the new generation of molecular scientists and cancer virologists.

During the oncogene and reverse transcriptase era of the 1970’s, it had become increasingly clear to many, that Watson and Crick didn’t discover “the secret of life” in the model they had built of DNA, because it had come to the attention of many biologists that any DNA model made of such geometrically arranged chemical building blocks couldn’t explain how living things actually worked. Legions of examples in biology show how knowing genetics or the the genetic code can explain little, if anything, regarding life, other than transcription and molecular inheritance. However, there was no hypothesis sufficiently chemical and reductionistic in nature that could challenge Watson and Crick’s “central dogma,” and thus a need was created to augment or even challenge the explanatory power of The Central Dogma of Watson and Crick, but for the wrong (chemical) rather than obvious biological reasons.

Thus the announcement and acceptance of the discovery of reverse transcriptase both augmented and challenged the Watson and Crick central dogma model, and was welcomed. There was now indeed more to life than an explanation employing the concept of genetic inheritance and a one way flow of genetic information, and it became possible, with RT, that other information could or might flow from “the environment,” back to “the genes.” The RT hypothesis suggested now that the Watson and Crick model merely explained how information is geometrically stored in the DNA molecule, and how this information is chemically inherited, and at the same time, flows outward from DNA to form RNA.

The discovery of RT also helped gain wide acceptance in the biological community of the oncogene or genetic mutation concept of cancer that had been dismissed by Payton Rous in 1966 in his Nobel lecture, with his statement:

*A favorite explanation has been that oncogenes cause alterations in the genes of the cells of the body, somatic mutations as these are termed. **But numerous facts, when taken together, decisively exclude this supposition**"¹.*

This new gold rush was directly the result of, and would be permitted by the Nobel Prize-worthy imaginations of yet two other virologists, Harold Varmus (who would become NIH director and name HIV "HIV" during the AIDS era), and Stephen Bishop. Using the same reductionistic arguments that underscored the need to chemically explain the reverse flow of information that RT was thought to provide to make DNA from RNA, Varmus's and Bishop's oncogene concept attempted to reduce cancer itself down to specific, ultimately inherited, molecular interactions instead of the kinds of cancer induction phenomena, as Rous and Shope and others had pioneered. Along with the discovery of reverse transcriptase, the oncogene hypothesis formed a new genetic theory of cancer based on the belief that "oncogenes" had become integrated into cells by retroviruses when they infected them, during a process fancied to have occurred **perhaps a billion years ago**, and which made these hijacked "cancer genes" by retroviruses part of "self" through inheritance. As recounted in a historical review of the contributions of Harold Varmus:

*The fact that...[Rous's] the chicken sarcoma oncogene is found in a wide range of species and has been preserved for more than a billion years indicated to Varmus and Bishop that it originated **in normal cells, not in the retrovirus that carries it**. They concluded that the [cancer causing] SRC...oncogene... was **captured** from the genome of a host cell **by an invading retrovirus far in the evolutionary past in a chance event known as viral transduction. As retroviruses insert themselves into the DNA of host cells and from there direct the synthesis of new virus particles, they can on rare occasions make copies not just of their own viral genes, but capture adjacent cellular genes—including cellular proto-oncogenes—which they then carry, or transduce, into other organisms they subsequently infect. The process by which retroviruses capture proto-oncogenes damages these genes in ways that can turn them into full-fledged oncogenes, which induce malignant growth when the virus infects another cell.***

*Through an accident of nature, retroviruses had **singled out** proto-oncogenes, extracted them from cells, and brought them to the attention of scientists, well before scientists could have found these genes among the convolutions of the immensely long chain of human DNA. (This accident, which was of such benefit to science, brought no evolutionary benefit to the viruses themselves—they can reproduce and live even when they lose their oncogene **through mutation**.) Retroviruses remain **vital tools** for the isolation of oncogenes **and** for elucidating fundamental processes within human cells.*

Although these so-called oncogenic proto-oncogenic DNA sequences and retroviruses were found in both cancerous and non-cancerous cells alike, and in contexts such as worms that rarely if ever develop cancers, and even in bacteria that do not produce cancers as they are single celled organisms, Harold Varmus and Stephen Bishop received their Nobel Prize in 1989, simply on the basis of their rumor virus suggestions, and colorful imaginations, that through some hypothesized accident involving invading retroviruses, perhaps a billion years ago by chance, during which time a few of these terrorist retrovirus 'hijacked' a cellular gene, messed it up (mutated it), reinserted it into another cell, and now that cell could become cancerous, except when that same oncogene and retrovirus infected non-cancerous cells that didn't become cancerous, in which the same genes and virus sit quietly to this day without causing cancer. This hypothesis was relentlessly pursued for two decades as cancer research looked more and more for these oncogene "mutations." All that was typically found were differences in the levels of expression of normal cellular proteins (see **Refs 1-6**).

In the oncogene, retrovirus-mutation-cancer world view, it was also believed that numerous and different "invading" oncogenes other than src, the so-called chicken retroviral sequence attributed to Rous, without his agreement on the matter that he had discovered a virus, would be discovered by the in earnest and highly funded emerging oncogene program, and that these imagined oncogenes would also be able, with the help of other retroviruses and other reverse transcriptases like telomerase of course, to find a plethora of other important cellular regulatory genes out of a background of thousands of others they could somehow selectively avoid, and specifically alter these regulatory genes in consistent ways to produce a tumor, and in the case of the imagined Hayflick-

controlling telomerase, reverse transcribe RNA into DNA to “protect” the mythical “reverse-transcribed ends” of chromosomes.

The proposal of this attractive random mechanism of cellular disruption by “invading” infectious terrorist cancer retroviruses, despite the fact that cancer gene swapping never has been observed in living organisms, and regardless that topologically the “ends” of chromosomes aren’t really ends, the mutation-oncogene-retrovirus –reverse transcriptases hypotheses thus provided the newly emerging and powerful virus and gene reductionists with a beautifully complex story that reduced the complexity of cancer into chance invading terrorist viruses from a billion years ago, along with their RT-empowered ability to make DNA from RNA.

Thus, exogenous (coming from the outside of an organism) and currently existing imagined human “cancer viruses” didn’t need to be caught in recent times from infected black folk to cause cancers, as had been postulated by Wade Parks regarding the “monkey virus” that was said to infect Henrietta’s cells, but virally transduced cellular oncogenes could exist in our own cells for evolutionary periods of time, without causing cancer, but then suddenly, somehow, induce them. Thus, the oncogene hypothesis served as the basis of what was to follow in the era of AIDS, because it associated cancers both in animals and humans to “infectious” “retroviruses,” and their hypothetical ability to hijack cellular or viral oncogenes, and thereafter, hopelessly ever since, has blurred the distinction between “self” and “non-self” in all biological contexts that ascribed to these Nobel-worthy ideas to explain cancer, concepts such as apoptosis, where even “death genes” have been proposed (as opposed to “life genes” not working correctly resulting in cellular failure or death).

Last, but not least, reverse transcriptase, telomerase, and its association with constellations of erroneous hypotheses of diseases, then emerged during the oncogene era full force, to explain cancer, cancer cell glycolysis, the Hayflick limit, and aging itself, including a variety of premature aging syndromes that are associated with short telomeres. Some of these and others include: Werner syndrome, Ataxia telangiectasia, Ataxia-telangiectasia like disorder, Bloom syndrome, Fanconi anemia and Nijmegen breakage syndrome, Ashkenazi Jews that live a long time, progeria, a way to reverse liver damage from alcoholism, resistance against “HIV,” why mice with elevated telomerase don’t live longer, why mice without active telomerase don’t live longer, stem cell behaviors, aplastic anemias, Cri du chat Syndrome, dyskeratosis congenital, skin pigmentation, leucoplakia, why heart attacks occur so suddenly, and even how psychological stress contributes to chromosome damage. And predictably, more Nobel prizes, such as The Nobel Prize in Physiology or Medicine 2009 have been awarded jointly to Elizabeth H. Blackburn, Carol W. Greider and Jack W. Szostak “for the discovery of how chromosomes are protected by telomeres and the enzyme telomerase” (for alternative models of how chromosomes are continuous and do not have telomeres, please see References **7,8,9**).

The mutation-oncogene-retrovirus-reverse-transcriptase-telomerase hypotheses (MORRTT- these hypotheses might be called collectively-denoting the Latin word for death or dead) also were used to explain the Rous sarcoma virus hypothesis that began the entire virus-cancer era. As noted earlier, first the Rous sarcoma filterable extract had a shaky beginning. It took more than 15 years to convince the scientific community of his day that his filtered chicken tumor extracts could induce cancers in several chickens but not mammals. Shope’s papilloma work had more success perhaps, in convincing the scientific community that the papilloma extracts he used could induce warts and a few malignant tumors in rabbits, and perhaps a few other mammals, although this was thought to be a DNA virus. SV-40, a contaminant of the polio vaccine, that disturbed even human chromosomes in Petri dish experiments, but not hundreds of millions of vaccinated humans, and with its DNA-oncogene-like Large T and small -T antigen mechanism that was said to invade the nuclei of cells and were envisioned to compete for cellular pools of important oncogenic molecules (the oncoproteins p53 and retinoblastoma) to cause cell deregulation, didn’t induce cancer after the polio vaccine crusades despite the injection or sugar cube delivery of the vaccines into hundreds of millions of humans.

By the same token, in a gene-centric universe, where genes and transducing viruses are given billion year-old supernatural and chance abilities that direct all living process and which drive everything living such as cells, and non-living entities such as viruses, while the physiological mechanisms outside the genes in the cell cytoplasm or extracellular matrix are viewed with contempt for how

they hold both structural and biochemical information outside the genes and control them, the door was opened to billion year old, nearly supernatural invading retroviruses and oncogenes, that could through mutation evade drugs, defeat vaccine trials, and kill cancer and AIDS victims as effectively and as assuredly as a Los Vegas card sharp could reshuffle a deck of cards. As Harvard's Richard Lewontin concluded though, the oncogene era failed to explain cancer. Before we review HPV and Merck's claimed "First" human anti-cancer vaccine, "HPV," it would be instructive to review briefly the race to develop Merck's previous other "First" claimed human anti-cancer vaccine and virus during the hepatitis B era.

–BITTNER'S MOUSE MAMMARY TUMOR VIRUSES CAUSE BENIGN TUMORS IN MICE, AND SOMETIMES LEUKEMIAS, AND ARE SAID TO CAUSE HUMAN BREAST CANCERS AND LIVER CIRRHOSIS.

Before delving into the hepatitis B era and the so-called first recombinant (molecular) vaccines, you need to know the history, beginning in 1936, of the so-called mouse mammary tumor virus. For brevity's sake, a time-line is presented, and notice how antibodies and DNA sequencing technology replace virus isolation, Koch's postulates, and fly in the face of Rous's warning that:

“Despite protracted search, aided by the electron microscope, no tumor virus has ever been found in human milk, and family histories definitely rule it out.”

Bittner is credited as discovering the “milk factor” or so-called mouse mammary tumor “viruses” in 1936, but I have chosen to relate the story (briefly) now, following rather than preceding the postulation of oncogenes, since mammary tumor viruses are thought to only be able to induce cancer(s) (or cirrhosis!) by altering or insertion at or near known oncogenes. Such imaginings first needed Varmus, and his ideas, to bring forth the concept of transactivation of genes of a billion years ago, but more about this topic later. Moreover, like “HIV,” as a believed “retrovirus,” MMTV requires strange accessory proteins, like “HIV” is said to require Rev, to do the job, although, as mentioned, it cannot or does not induce malignant tumors or metastases in mice. It even is imagined that HMTV, or Human Mammary Tumor Viruses exist, and women living in close proximity to certain mice and in certain areas of the world can catch these “cancer causing viruses” and spread them about or that they accelerate their disease. However, as you will hopefully discern latter, these stories are just that: stories or imaginings of persons who appear more often than not, to live in fear of invisible terrors.

Finally, and importantly, MMTV was confounded in the development of its science by the problem of HERVs, or what are known as Human Endogenous Retroviral sequences, which are problematic artifacts of the modern molecular era. Only a timeline of MMTV and HMTV reports will be presented here, because, if for no other reason, the science becomes tedious, and shows again, that no evidence has been convincingly demonstrated, that viruses can cause or has been linked as an efficient causal inducer of human cancers. By contrast, historical developments in virology reveal that a principal contribution of rumor virology has principally been to mislead the PHS and NCI/NHI granting committees, and to dominate and marginalize other disciplines that attempt to experimentally elucidate the complexities of the natural world and human health. In fact it could be cogently argued that billions have been squandered through provision of resources and research programs, as well as public health policies, that have provided nothing in terms of any rigorous science or human benefit. Not to abandon thoroughness, however, an impartial list of the principal developments and claims about MMTV and HMTV is presented below, beginning with a list of confounding Human Endogenous Retrovirus Sequences (HERVs) and the so-called diseases or contexts to which they have been linked:

ENDOGENOUS HERV (RETROID) SCIENCE FOR THE TECHNICALLY MINDED:

HERV'S (retroids) ARE FOUND:

1. IN XENO-TRANSPLANTATION OF PIG ORGANS

2. IN AUTOIMMUNE DISEASES LIKE ARTHRITIS
3. IN THE SERUM SAMPLES OF 70% OF “HIV-POSITIVE” HUMAN PATIENT’S SERA
4. IN 30% OF PREGNANT WOMEN
5. IN 45% OF TESTICULAR CANCER
6. IN 26% OF LYMPHOMAS
7. IN LUNG CANCERS
8. IN BREAST CANCERS
9. IN TERATOCARCINOMAS
10. IN MELANOMA PATIENTS
11. DURING PHASE I TUMOR REJECTION
12. IN ZEBRAFISH THYMUS GLANDS
13. IN CELL FUSION EXPERIMENTS
14. TO STIMULATE CYTOTOXIC T-LYMPHOCYTES
15. IN HUMAN URINE
16. TO BE REQUIRED FOR MOUSE MELANOMA GROWTH
17. TO BE SENSITIVE TO AZT AND DDI AND ARE FOUND AT LOW LEVELS IN NORMAL BLOOD DONORS AND THEIR TISSUES
18. IN 1 OR 10% OF THE HUMAN GENOME
19. TO HAVE OR NOT HAVE GAG OR ENV PROTEINS (“HIV-specific but not “HIV”).
20. IN OLD WORLD MONKEYS AND CHIMPS
21. TO BE RELEASED AFTER GLUCO-CORTICOID STIMULATION
22. TO PROTECT THEIR HOSTS FROM INFECTION WITH “EXOGENOUS” RETROVIRUSES
23. TO BE THE SOURCE OF MOST RT ACTIVITY IN HUMAN CELLS
24. TO PROMOTE THY1 RESPONSES

25. TO BE RESPONSIBLE FOR ALS, SCHIZOPHRENIA, SJOGREN'S SYNDROME, OPITZ SYNDROME
26. TO BE IMMUNE SUPPRESSIVE
27. ON PART OF THE Y CHROMOSOME
28. IN A FRACTION OF THE CAUCASION POPULATION
29. TO CAUSE MICHAEL JACKSON'S AUTOIMMUNE VITELIGO DISEASE, BUT IN CHICKENS
30. IN THE DEVELOPMENT OF "PLACENTALNESS" IN MAMMALS

These are some of the key papers said to define MMTV scientific history:

From: <http://www.thepinksquadron.org/research-library/39-mousehuman-mammary-tumor-virus-time-line-of-discovery>

[Mouse/Human Mammary Tumor Virus: Time Line of Discovery](#)

[Bittner J, Some Possible Effects of Nursing on the Mammary Glad Tumor Incidence in Mice, Science, August 14, 1936, 84: 2171, p 162](#)

1936 John Bittner discovers that "***an infectious agent***" carried in breast milk from one strain of mice can cause (non-metastasizing-my emphasis) breast cancer in another strain of mice.

1942 [Bittner J, The Milk-Influence of Breast Tumors in Mice, Science, 1 May 1942, 95:462-463](#)

John Bittner isolates his "milk agent," and feeds it to mice that have **a low incidence** of breast cancer. This causes **an increased** number of breast cancers in the mice. **He thinks** the milk agent ***might be*** a virus.

1966: Rous's Nobel Lecture: When discussing "mouse mammary tumor viruses" and how they were believed to have been discovered, Rous emphasized the following:

"To this day no virus directly responsible for the cancers has been obtained from these, but only the "milk virus" producing the benign growths. The problem of the actuating cause of the cancers remains unsolved like that of the V x 2 carcinoma of rabbits⁴. In both instances the virus causing the benign tumor was no more than an initiator of the malignant growth deriving from them."

"Despite protracted search, aided by the electron microscope, no tumor virus has ever been found in human milk, and family histories definitely rule it out."

1969 [Links J, The Growth Accelerating Effect of Bittner Virus in Monolayers of Baby Mouse Kidney Cells, J. Gen Virol, 1969, 5:547-550](#)

Researchers took the Bittner "**virus**," known **now** as *mouse mammary tumor virus*, and exposed baby-mouse **kidney cells** to it: the virus caused the **kidney cells** to grow faster? And one might ponder, "grow faster" than what? Than if you didn't contaminate them with proteins derived from somewhere other than the kidney?"

1978 [Mesa-Tejada R, Detection in human breast carcinomas of an antigen immunologically related to a group-specific antigen of mouse mammary tumor virus, Proc Natl Acad Sci USA, 1978,75:1529-1533](#)

Researchers find evidence of the **mouse** mammary tumor virus in **human breast cancer** specimens. But then in the title of the work, why is it spelled out, “immunologically related,” if indeed they found any specific virus? How do they surmise that what they have found is nothing but a non-specific promiscuous antibody interaction?

1980 [Zotter S, Mouse Mammary Tumour Virus-related Antigens in Core-like Density Fractions from Large Samples of Women's Milk, Euro J Cancer, 1980, 16:455-467](#)

Researchers find evidence of mouse mammary tumor **virus-related antigens** in human breast milk. Again, why the antigens instead of a virus?

1981 [Witkin S, Antigens and Antibodies Cross-Reactive to the Murine Mammary Tumor Virus in Human Breast Cyst Fluids, J Clin Invest, 1981, 67:216-222](#)

Researchers find evidence of the mouse mammary tumor virus in breast cyst fluid. Who cares about cross-reactive antigens and antibodies which by definition are promiscuous and often non-specific chemical interactions without proof that there is a virus present? Where is the virus?

1981 [Day N, Antibodies reactive with murine mammary tumor virus in sera of patients with breast cancer: Geographic and family studies, Proc Natl Acad Sci USA, 1981, 78:2483-2487](#)

Researchers find evidence of mouse mammary tumor virus **antibodies** in the blood of women with breast cancer. Where is a virus?

1984 [Westley B, The human genome contains multiple sequences of varying homology to mouse mammary tumour virus DNA, Gene, 1984, 28:221-227](#)

Multiple copies of **gene sequences** of the mouse mammary tumor virus are found **in the human genome**. Better than single copies of non-identified gene sequences I suppose. Where is the virus?

1991 [Nusse R, Insertional Mutagenesis in Mouse Mammary Tumorigenesis, Current Topics in Microbiology and Immunology, 1991, 171:43-65](#)

Nusse **hypothesizes** that mouse mammary tumor virus produces breast cancer by activating otherwise **silent oncogenes**, thus triggering malignant transformation of normal breast cells. It sure would have been good to show us a virus rather than an hypothesis, and high titres of identified MMTV particles at the sight of supposed transformation?

1993 [Shackleford G, Mouse mammary tumor virus infection accelerates mammary carcinogenesis in Wnt-1 transgenic mice by insertional activation of int-1/Fgf-3 and hst/Fgf-4, Proc Natl Acad Sci USA, 1993, 90:740-744](#)

Researchers identify **which genes** are activated by insertion of the mouse mammary tumor virus. So the infection doesn't cause the cancer....it accelerated them after they already had begun by something else?

1995 [Wang Y., Identification and expression of MMTV-like sequences in human breast cancer, Proc. Am. Assoc. Cancer Res. 37, 1996, 565](#)

Another portion of the mouse mammary tumor virus is found **in the genome** of human breast cancer cells. It would be good to have the whole entire virus someday? And the fact that the authors point to “a portion” of “the virus” in the genome suggests that it is a partial DNA sequence they are detecting rather than an intact, infectious, cancer-causing entity. In effect, they assume an unusual genome sequence they are detecting in human breast cancer specimens is responsible for that cancer. Much like fire trucks are present at fires and it is then assumed that fire trucks cause fires, or skid marks from tires cause auto accidents, as fire trucks are frequently seen at fires and skid marks are frequently seen at car accidents?

2000 [Stewart T, Breast Cancer Incidence Highest in the Range of One Species of House Mouse, *Mus Domesticus*, *British Journal of Cancer*, 2000, 82:446-451](#)

Researchers study the geographic range of the mouse that **carries** the mouse mammary tumor virus and find that the incidence of **human breast cancer** is highest in areas where the mouse is most prominent. It is truly amazing how these viruses can jump species to cause cancer in humans. Maybe Rous should have been looking at the mouse milk rather than electron micrographs of human milk where he claimed nobody to date (1966) ever could find evidence of a virus? Where aren't mice “prominent”?

2003 [Ford, CE, Mouse mammary tumor virus-like gene sequences in breast tumors of Australian and Vietnamese Women, *Clinical Cancer Research*, 2003, Vol 9: 1118-1129.](#)

Increasing evidence of the mouse mammary tumor virus in human tissue samples is found to be associated with progression of abnormal and malignant breast cells in clinical survey samples of human tumors. Yet once again, tumor virus-like gene sequences instead of a whole intact virus do not prove viral causation, or even that a virus is present at any time.

2004 [Theodorou V, Fgf10 is an oncogene activated by MMTV insertional mutagenesis in mouse mammary tumors and overexpressed in a subset of human breast carcinomas, *Oncogene*, 23; 2004, 6047-6055](#)

Researchers discover that the mouse mammary tumor virus activates **a specific oncogene**. This oncogene is found to be **overly active in a certain portion** of human breast cancers. It is a good thing Harvard researcher, Lewontin dismissed oncogenes, as did the most of the War On Cancer Warriors following a decade or two of unproductive research speculating about their existence from transduction a billion years ago and their role in cancer. In other words, MMTV can't cause cancer without oncogene interactions (how does the “virus” know how to find an oncogene sequence amidst 25 thousand genes) , and why don't they cause metastatic cancers in mice (or humans)?

2006 [Lawson J, Presence of mouse mammary tumour-like virus gene sequences may be associated with morphology of specific human breast cancer, *J Clin Pathol*, 59; 2006, 1287-1292](#)

Researchers study different kinds of breast cancer and find that certain forms of human breast cancer have evidence of mouse mammary tumor virus **genes**. Where is the virus?

2007 [Indik, S, Rapid spread of mouse mammary tumor virus in cultured human breast cells, *Retrovirology*, 2007, 4, 73](#)

Researchers note the rapid spread of mouse mammary tumor virus from one cell to the next in normal human breast cell cultures. Where is the virus? And were the cultures transformed?

2007 [Melana S, Characterization of viral particles isolated from primary cultures of human breast cancer cells, *Cancer Res*, 2007, 67:8960-8965](#)

Cultured breast cancer cells produce viral particles **similar** to mouse mammary tumor virus. They are now referred to as human mammary tumor viral particles. The scientists hypothesize that human mammary tumor virus **may play a role** in human breast cancer.

2009 [Melana S, Detection of human mammary tumor virus proteins in human breast cancer cells, Journal of Virological Methods 2009](#)

For the first time, human breast cancer cells are found to produce human mammary tumor virus proteins. Does this imply that the cells are making the “viral like sequences” of HMTV-believed gene sequences that produce these proteins they think they have found, without any virus ever being present?

Oncogene thinking and gene sequencing along with non-specific antibodies as evidence of the presence of a virus does not prove any virus has ever been causally linked to any mouse or human breast cancer. The literature is polluted, even in the titles as the above list shows, with assumptions whereby cancer cell gene sequences, non-specific antibody interactions, wrong tissue tropisms (such as kidney), and wild assumptions about women living near mice or not and species jumping comes to take the place of any shred of actual proof that any virus has initiated any cancer, whether it be benign and not truly a lethal cancer in mice, or in surveys of human breast cancer tissue.

LIVER CANCER IS CAUSED BY PEANUTS AND BOOZE, AND CAN BE PREVENTED WITH THE HEPATITIS B VACCINE-THE FIRST ANTI-CANCER VACCINE

During the 1950's and 60's, the chemical process of DNA replication through conservative (with both strands of a chromosome) versus **semiconservative** replication (with only one strand of a chromosome), was finally resolved by J. Herbert Taylor in favor of semiconservative DNA replication. Taylor had used the high-resolution method of tritium (^3H) autoradiography instead of C^{14} autoradiography that Danial Mazia did. His radioactive images of radio-labeled chromosome revealed that only one strand in a replicating chromosome became labeled (with tritium). Taylor's higher resolution tritium versus C^{14} labeling clearly showed that only one DNA strand of a chromosome became labeled with radioactive tritium, because its decay was much shorter compared with C^{14} -labeled autoradiography of chromosomes, which fogged the film, making it appear to Mazia as though both chromatids labeled. Thus it became clear that only one DNA (or RNA) strand was needed to form another.

An idea was proposed by a doctor, who perhaps was not schooled in molecular genetics, regarding the possibility of “replicating” protein molecules, instead of Taylor's now demonstrated molecular templating ability of single-stranded DNA. D. Carlton Gajdusek's imagined instead, the idea that infectious protein particles were the most likely cause of horrid neurological diseases in both animals and man, and his idea became not only accepted as plausible by the medical establishment. He, along with another physician, Dr. Baruch Blumberg, were awarded with yet another virological Nobel Prize. As a new infectious disease model, the idea that proteins could be infectious and propagate themselves without nucleic acids, served as a basis to justify the slaughtering of hundreds of thousands of suspected scrapie-infected cattle in England. It also served to prevent English persons born after 1980 from donating blood in the U.S., as Creutzfeldt-Jakob disease or CJD was thought to be a degenerative neurological disorder that is incurable, and invariably fatal, and also, oddly enough, a syndrome that was said to “run” in English families and inherited.

Now attributed to Gadjusek's infectious proteins, CJD was imagined to be a human version of transmissible bovine spongiform encephalopathy in England, and, together with the English cattle industry noticing what appeared to be new outbreaks of scrapie in cattle, both of these phenomena were then attributed to Gajdusek's “slow viruses” he thought he had found in Papua New Guinea cannibals. It was further believed that these proteins could hide out in a latent form in cells or tissues for many years, and then one day, leap forth (like oncogenes) to cause the grotesque neurological demise that is seen in CJD and kuru in man, and scrapie in sheep

and cattle, or that was inherited and ran in English families.

The discovery and promotion of these so-called “slow viruses,” such as the kuru or scrapie agent, and “retroviruses” during the middle to late 1970’s, only further challenged the Watson and Crick dogma regarding the one way flow of information from the genes outward. With the ability of inherited information to now flow in both directions, toward DNA, or toward proteins, or now for proteins themselves to possess the remarkable ability to form templates, the Watson/Crick paradigm, based upon semiconservative replication, was viewed as definitely incomplete, and it needed to be expanded. In fact, there was much excitement and interest regarding the way proteins might be able to seed or “nucleate” cellular reactions in other biological contexts as well, thanks to Gajdusek’s speculations, or even in organelle biogenesis (such as the centrioles which are part of the normal cellular division mechanism), in the absence of a nucleic acid template (i.e. not requiring genes).

While the biochemists and Nobelists continued to receive unlimited intellectual and financial support for their notions regarding how viruses, genes, and now proteins could replicate and cause human illnesses, cogent observations by cattle experts such as The Late Mark Purdy and others, explained how changes in food processing for cattle had introduced potent neurotoxins during that decade. The possibility of toxins introduced during food processing, as well as the association with organophosphate fertilizer poisoning, was ignored in favor of an infectious cause. Purdy had noticed how English farmers changed the way they processed bone meal using highly toxic solvents, which could cause these same spongiform encephalopathies. For several decades now, the developments in “prion” science and hepatitis B science have stood as formally accepted exceptions to a ubiquitous and exclusive focus on genetic determinism (that genes produce and ultimately control everything). Not only that, but what was to be named the prion agent couldn’t be killed by heat, radiation, strong acids, or practically anything. It was branded as a true “Andromeda Strain” by the biological community, and it was feared in laboratories throughout the world.

The Nobel Committee’s designation of “new mechanisms of infectious disease transmission” (the reason of the Noble Prizes given to Gajdusek and Blumberg) also signaled that the postulated mechanisms of hepatitis B infectiousness, by co-Nobelist Baruch Blumberg, did not need to conform to Koch’s postulates or any microbiological or rational standards that prove that a virus or virus-like agent causes a disease. Nor did these notions necessarily even need to conform to the idea that genetic information in the form of nucleic acids are required at all for infectious transmission. It goes without saying how much Dr. Gajdusek’s contribution greatly influenced what transpired during the oncogene, retrovirus, reverse transcriptase era, because of this notion that a “slow virus” or virus-like agent might exist.

After the acceptance of his discovery, and after he and Dr. Blumberg were awarded the Nobel in 1976 (a year after Baltimore’s and Temin’s Nobel for RT and retroviruses), Dr. Gajdusek would serve as the chief NIH Neuroscience Program Director for many years. And although his ideas would continue to be, and still are, worshiped and heavily funded, unfortunately, Dr. Gajdusek’s reputation became less than God-like, after he pled guilty to charges against him following an FBI-directed investigation. Dr. Gajdusek admitted (and defended the fact) that he had sexually abused his adolescent male cannibal research subjects that he had brought back from New Guinea in his home’s living room in Maryland, before he fled to Europe to evade serving his sentence.

Although “infectious proteins” and prions are not relevant to cancer, their so-called “slow-virus like” incubation for years before they manifested disease was, and their “slow-nature” served to codify the thinking that microscopic agents thought to cause diseases can “hide out” and somehow **not biochemically interact** with cells and fluids of “infected” organisms, until one day, of course, when they decided ‘to bite.’

Although Gajdusek’s, and later Stanley Pruisner’s theorizing about their “slow viruses” now dubbed prions began to capture the minds and funding of the Federal agencies as infectious agents that threatened the food supply and also caused horrid human degenerative illness, the fact that such agents might be inherited and run in families flew in the face of any such infectiousness. Truth be told, their molecular sequences were, like oncogens, subsequently found in both healthy and diseased hamsters (for which

Pruisner received another Nobel prize a few years later). Gajdusek's co-Nobel recipient, Baruch Blumberg, and his hepatitis B contributions, would become a key further justification for the wraith-like nature of, and imagined "slow incubation," of tumor viruses.

But Dr. Baruch Blumberg's major focus at first was not directed toward studying liver hepatitis, hepatitis B, infectious agents, or even cancer. Blumberg was fascinated by inherited genetic (blood) polymorphisms in the great population genetic and biochemical marker traditions of G6DP and sickle-cell and thalassemia anemias, and their possible relationship to malaras, as well as Boyd's blood group designations, and other population studies using protein or genetic markers. It was in this context that, like Stanley Gartler and G6DP, where Blumberg first found that the so-called Au (Australian antigen-HBsAg surface antigen) polymorphism. He declared that it was rare in the West, but was present in "**a black man**" who was a healthy Australian aborigine whose blood he had stored in his freezer.

The HBsAg molecular marker was also present on rare occasions in blood samples obtained from healthy Micronesians, healthy Vietnamese, healthy Taiwanese, and, in non-healthy patients with leukemia (not hepatocellular carcinoma), or Down syndrome (Baruch Blumberg, PNAS, Vol. 94 pp 7121 -7125, page 7122-23, 1997, top of page).

But how was this possible? Nowadays, "Hepatitis B virus" markers (HBsAg and HBeAg) are thought to be transmitted primarily through sex, dirty needles, and perinatal transmission, or through transfusions. These "3rd World and disparate groups or diseases either all must share these risks or risk behaviors as mentioned-such as IV drug use and sexual promiscuity, or as I would argue instead, were and are detected in these groups simply because like G6DP, it was a poorly or incompletely researched genetic polymorphism at that time, or, in the context of disease processes, it represented a stress-induced immunological reaction. In these contexts, the hepatitis B antigen(s) can be induced in hepatitis patients and found at high frequency in Down Syndrome persons because of tissue destruction, and perhaps, for many other poorly understood immunological reasons.

The HPSAG (hepatitis) antigen may merely represent a simple and relatively rare (in the West-not so rare in Asia or Australia or Africa) blood polymorphism, which is supported by the fact that most people (>95%) who harbor the HBsAG molecular signature never become sick, develop hepatitis, or cancer of any kind. In the context of hepatitis B and cancer, and this gets very bizarre indeed, Blumberg's hypotheses regarding HBsAG antigen also suggested to him that:

"i) Individuals with Au have an increased susceptibility to leukemia (NOT HEPATOCELLULAR CARCINOMA as is continuously touted by The WHO and the PHS), and this susceptibility is inherited;"

"ii) Leukemia causes Au (HbsAg)" (again not hepatocellular carcinoma);

"iii) Au is related to "the virus" that has been postulated to be the cause of leukemia.

Where is the link between Au and "hepatocellular carcinoma?" Do Hepatitis B viruses cause leukemia? Again we are faced with Rous's dilemma: that these cancer viruses aren't linked to consistent diseases.

And why did Blumberg initially think "family studies" of carriers (of HbsAg) "were consistent with a genetic hypothesis of inherited polymorphism," yet allowed the idea to be advanced that hepatitis B was transmitted as an infectious viral agent? How could an inherited trait be infectious? This question, stimulated a lot of controversy and dissent during the 1980's.

Why did Blumberg believe that HbsAg was an infectious agent linked to leukemia when in his writings he was fully aware that "certain individuals were susceptible both to the development of Au-positivity and leukemia" (not hepatocellular carcinoma) and that "children with Down syndrome have a rate of HBsAg of approximately 30% compared to other patients housed in mental

institutions whose rate of HbsAg was approximately 5%?” Does trisomy (Down Syndrome) have an infectious basis also? Are Down people more promiscuous? Do they share needles? Are they exposed more frequently to blood products? In mental institutions, do inmates eat feces and get infected, as was once suggested by a gifted virologist?

Blumberg in the PNAS article also tells us about his collaborative work with Bayer and Werner in their Philadelphia laboratory, who identified particles with the “appearance” of a virus in the serum of individuals with Au (HbsAg antigen) again on page 7123, (paragraph 3-4 at the bottom of the first column). Why does Blumberg then emphasize that these particles were subsequently shown to “not contain any nucleic acid” or even “core proteins,” and tell us that they were “neither infectious or pathogenic?”

Why does Blumberg tell us about how I. Millman and V. Coyne in his lab were able to “localize Au” in the liver cells of patients with hepatitis, and yet elaborate as to why it was so “difficult to confirm” they were able to sustain the growth of “the virus” in liver cells obtained from patients with liver disease associated with Au? A related question is, why don’t chimps show any liver pathogenicity, cellular damage, or develop anything resembling hepatitis in modern studies when they are infected with hepatitis B?

Why does Blumberg in this Proceedings of The National Academy of Sciences paper make a big point emphasizing that:

“We subsequently realized that the virus had been transmitted to the leukemia patients (not hepatocellular carcinoma patients) by transfusion of blood contaminated with HBV, and that the high prevalence in the Down syndrome population was a consequence of crowding in large institutions.”

Why does he then tell us that: *“But the answer was not that simple. Down syndrome patients were more likely than patients with different diagnosis within the same institution to become **carriers**.”*

When Millman joined Blumberg’s laboratory from Merck in 1967, why did he calculate that “the amount of Au (HbsAg) in the serum of carriers to be 1% of the serum proteins,” and why did he feel that this amount of virus would be incompatible with the life of the carrier? [1% is a huge amount].

One percent of anything in the bloodstream would be a gargantuan quantity. Millman thought that the hep B antigens constituted 1% of the serum. Is this consistent with current hepatitis viremia estimates? Hardly. On page 7124, paragraph 6 of the PNAS article, why does Blumberg again state that:

*“Collaborating with M. Bayer and L. Loeb, we found that the small particles visualized in the electron microscope did not contain nucleic acid,” ...“We inferred that these were non-replicating, incomplete forms of the virus made up entirely of surface antigen, and that there **must be** additional particles which contained nucleic acid, that were replicating, infectious, and pathogenic.”*

It is not that proteins injected into a recipient cannot induce a myriad of autoimmune syndromes, or severe toxicity. Transfusion medicine is rife with such examples. For instance, almost 10% -22% of transfusion patients experience some form of toxicity or lung infections and exhibit a syndrome doctors call TRLI (Transfusion Related Lung Injury), that can be fatal. Foreign protein-containing blood transfusions a few years back were proposed to be used prior to heart transplantation precisely because they are known to suppress the immune system so well that cyclosporins or other profound immune suppressive drugs don’t need to be used right away to avoid rejection and stress out the patient immediately following heart transplant surgery. Transfusions profoundly suppress this natural process of organ rejection by crippling the immune response (a worthy subject of another book)?

How can particles or proteins that lack nucleic acids replicate? They can’t. They are believed to catalyze or polymerize pre-existing normal proteins and somehow change them into infectious and disease causing proteins via these catalyses. And although it is chiefly through Blumberg’s and Gajdusek’s infectious protein hypotheses and slow virus theorizing that these beliefs became entrenched,

and/or because of their celebrated “new mechanisms of infectious disease transmission,” were so Nobel worthy, that a few years later, and as mentioned earlier, another Nobel prize would be awarded the Prize once more for his so-called “prion” hypothesis, which was an elaboration of Gajdusek’s infectious protein model, known as the PRP^C/PRP^S (prion) hypothesis.

Yet, is there any proof that the hepatitis B causes hepatocellular carcinoma? It might be an advantage to Public Health to have at least some evidence that it does, before the PHS and WHO goes and vaccinates an entire planet with an isolate derived from an ill-defined soup originally found in a healthy black Australian aboriginal gentleman that lacked viral nucleic acids, or before the USDA or similar agencies kills every cow that grazes in the US, Canada, or England for fear they carry prions in the context of prion disease avoidance or management? As will be discussed later, this is very important because the hepatitis B era constituted the basis or foundation upon which “molecular recombinant vaccines,” as well as many of the mistaken theories and dangerous pogroms of the AIDS era would be based upon.

If hepatitis B is an infectious virus that causes liver cancer, then why did Blumberg in his personal PNAS-published account of the Hepatitis B era make a point of stating that:

*“In animal experiments, Millman and London found that partially purified Au particles, which we had not **yet** visualized, could be transmitted by inoculation into experimental animals....The fully purified particles from which {THE WHOLE VIRUS} had been removed were not infectious.”*

Were these “visualized” particles again isolated, and shown to cause cytopathic effects in yet newly injected animals to fulfill Koch’s postulates? Were they even found in every (or any) inoculated animal who had been subjected to the “hepatitis B-infected sera?”

All subsequent studies using hepatitis B since Millman and London’s studies also have borne out this same result in mice of all kinds, chimps, and other experimental animals: (Guidotti et al., Viral clearance without destruction of infected cells during acute HBV infection. *Science* Apr 30; 284(5415):825-9, 1999):

*Viral clearance during hepatitis B virus (HBV) infection has been thought to reflect the destruction of infected hepatocytes by CD8(+) T lymphocytes. **However, in this study, HBV DNA was shown to largely disappear from the liver and the blood of acutely infected chimpanzees long before T cell infiltration and most of the liver disease. These results demonstrate that noncytopathic antiviral mechanisms contribute to viral clearance during acute viral hepatitis by purging HBV replicative intermediates from the cytoplasm and covalently closed circular viral DNA from the nucleus of infected cells.***

From Yamamura et al., HBV production in transgenic mice we read (5. *Gastroenterol Jpn* 1990 Sep;25 Suppl 2:49-53):

*We produced transgenic mice by microinjecting a partially duplicated copies of hepatitis B virus (HBV) gene into fertilized eggs of C57BL/6 mice. One mouse was a high producer of HBV surface antigen (HBsAg) and HBV e antigen (HBeAg) in the serum. All offspring carrying HBV DNA were positive for both antigens in the serum. The HBV DNA was expressed in liver- and kidney-specific manner. The normal process of HBV replication, including the packaging of the pregenome 3.5-kb RNA into a nucleocapsid, the reverse-transcription of the complete minus strand DNA, and the release of Dane particles into the serum before the completion of synthesis of plus strand, occurred in the liver of these transgenic mice. **These results suggest that the species specificity of HBV infection is not due to the inability to replicate in a nonnatural host but to the lack of receptors or factors needed for virus adsorption and internalization. The founder mouse is now 19 months of age but shows no clinical or pathological change, suggesting that HBV itself is not cytopathic.***

Much like Rous’s “actuator” ideas regarding the wraith-like nature of cancer viruses that never can be found associated even with

established and serially-transferred animal cancers, Blumberg here is repeating these same notions, now in the context of hepatitis B “infection.”

Then disappointingly, we are left with the sad “co-factorial” argument by Blumberg, that it isn’t so much the virus that causes liver cancer (hepatocellular carcinoma-HCC-not leukemia), but things such as peanut butter sandwiches that can be contaminated with cancer-causing aflatoxins (like those typically produced on moldy bread). From the same PNAS Blumberg recollections we read:

“The probability of HCC increases if the chronic carriers also are exposed to other agents, such as the carcinogen aflatoxin, which is produced by Aspergillus fungus that contaminates poorly stored foodstuffs.”

When Blumberg mentions carcinogens that contain aflatoxins, is he indeed talking about things like peanut butter sandwiches that could contain aflatoxin if it were laced with Aspergillus fungus? If so, how many peanut butter sandwiches harboring Aspergillus nidulans mold in the peanut butter is required as a “co-factor” to help the hepatitis B virus along with promiscuous sex or IV drug abuse or transfusions, to cause HC cancer in old age? Is he really saying here that you need to eat a lot of peanut butter sandwiches in order to acquire hepatitis B? Do they eat a lot of peanut butter sandwiches in China or down under in the Australian outback where hepatitis B was first found in a black person, and where it is said to be “endemic?” There is some evidence that aflatoxins at high concentrations will induce non-metastasizing cancers in some genetically-weakened lab animals under some conditions. What does this have to do with a supposed viral infection that causes liver cancer (HCC) 30-50 years after exposure in humans eating peanut butter sandwiches? The same co-factorial arguments have also been repeatedly advanced for HCV (hepatitis C virus) and its imagined link with hepatocellular carcinomas, and Baruch Blumberg warns us that:

Studying HCV has been difficult, in part, because of the lack of a reliable tissue culture for testing neutralizing antibodies or for passage and expanding of the virus. Historically, the invention of such a tissue culture system allowed the development of other vaccines such as the polio vaccine.

Using chimpanzees has presented numerous problems in that they appear to respond to HCV differently than humans. Mother-infant transmission has been reported in humans but not chimpanzees. Chronic infection occurs approximately 75% of the time in humans but only 30-50% of the time in chimpanzees.

Humans progress to liver fibrosis and cirrhosis while chimpanzees do not. This may in part be due to environmental differences between the species; e.g., humans consume large quantities of alcohol while chimpanzees do not. Other environmental differences also exist. Humans suffer from hepatocellular carcinoma as a result of HCV. Hepatocellular carcinoma after HCV infection is very rare in chimpanzees. The course of HCV is highly variable in the chimpanzee, as it is in humans, but is the variability secondary to the same causes in both species?

So it can be safely concluded that diseases such as hepatitis B and C are caused by too much peanuts and booze, as the title of this section claims, and cannot be caused only by hepatitis B viruses or hepatitis C viruses, as neither of these “first human cancer viruses” cause cancer in an animal model or even cause cellular pathogenicity. This is why peanut butter and booze need to be outlawed.

Other considerations should be emphasized as well before we review the AIDS era next and then Merck’s other “first” anti-cancer vaccine, Gardasil they will advance 20 years after they forgot about their first cancer vaccine claim, about their hepatitis B vaccine being the “first” anti-cancer vaccine.

However, before we proceed, the following questions need to be asked and answered:

Why does the WHO and CDC continue to insist that “the Hep B vaccine” is the first vaccine that can prevent liver cancer (not leukemia) in 350 million people worldwide” “who will eventually get liver cancer because they are carriers of the hep B antigen?” Remember, the hepatitis B antigen was found in the blood of a healthy black Australian aboriginal as well as in healthy people all over Australia and Asia, when Blumberg was studying genetic blood polymorphisms.

Why do modern EM micrographs of Hepatitis B virus show 3 different forms—a comet-like shape, a round filled shape, and a hollow round shape, whereas other viruses appear to have one and only one morphology—which allows virologists to classify them, and explain X-ray crystallographically, how the genes that encode them, make them?

Why have not the efforts of Mark R Geier M.D. Ph.D. and former President of The Genetic Centers of America, not been successful in obtaining the raw clinical trial data at The Institute of Medicine’s (IOM) hearing in May, 2004? Dr. Mark R. Geier and his son David Geier were part of a research team requested by Congress to review the CDC’s Vaccine Safety Datalink (a database kept by the CDC to monitor vaccine health issues). The Geiers were successful at gaining access to the CDC database only twice. When they presented the adverse findings from their limited access to the IOM Committee, they were immediately barred from the database under the guise of “potential confidentiality issues.” “The patient information is coded,” said Geier, “there is no possibility of a confidentiality breach.” In this context why wasn’t Dr. Geier’s testimony before the Institute of Medicine not taken seriously? Dr. Geier, a board certified geneticist who has published over 30 studies on this issue recounted how he asked only for the primary data from the hep B clinical trial (that was used to get FDA approval for the vaccine). After his request, he was first told that “the media that the data is kept on is too old to be accessed by modern computers.” He responded by saying that he would obtain a translation program to update that data, and that he wanted it anyway. Dr. Geier was then told that “the disk containing the data was damaged.” He said that that was OK because he would obtain a Norton Utilities Disk and fix the damaged disk. Dr. Geier was then told that “the disk couldn’t be found.” Where are the safety data? We will keep asking until they provide it.

The Vaccine Adverse Events Reporting System (VAERS) shows that the hepatitis B vaccine damages far more individuals than there are persons who exhibit the hepatitis B syndrome. Evidence obtained from the American Association of Physicians and Surgeons (AAPS) and other physicians, vaccine-monitoring agencies such as the National Vaccine Information Center (The 1999 statement of the National Vaccine Information Center-NVIC-regarding hepatitis B vaccination and adverse events), the CDC and World Health Organization, the Illinois Vaccine Awareness Coalition, the vaccine manufacturers Merck and GallaxoSmithKline, and evidence from the peer reviewed scientific literature, all show that the risk of groups such as infants and children acquiring liver hepatitis associated with hepatitis B is nearly 0%. In all comprehensive statistical surveys available, the actual incidence of the hepatitis B syndrome in the US has remained constant at about 2-4 cases/ 100,000 individuals despite widespread mandated and aggressive vaccination programs in all but 4 states.

A statement of the Association of American Physicians and Surgeons (AAPS) regarding the hepatitis B vaccine to the Subcommittee on Criminal Justice, Drug Policy, and Human Resources of the Committee on Government Reform U.S. House of Representatives also called for an immediate halt to mandatory vaccination, and AAPS also urged an immediate investigation in 1999 into reports regarding the vaccine’s danger to child health (Yazbak’s testimony before the Massachusetts House of representatives Committee on Education, Arts, and Humanities. Dr. Philip Incao’s testimony before Ohio House of Representatives).

Why does all the available data show instead, that the hepatitis B syndrome, when it does occur in non-vaccinated individuals, spontaneously resolves in almost 100% of those who become seropositive for the “hepatitis B antigens.” By contrast, the data also show that there are 100 victims of the hepatitis B vaccine who experience serious, life-threatening, and life-long adverse effects for every 1 person the vaccine **is claimed** to protect (according to the Merck package inserts, the figure is 10.4% experience adverse reactions). Some of the severe adverse effects include autism, Stevens-Johnson Syndrome, arthritis (both transient and permanent), Guillain-Barre Syndrome, myelitis including transverse myelitis, seizure, febrile seizure, peripheral neuropathy including Bell’s palsy,

diabetes mellitus, pancreatitis, encephalitis, multiple sclerosis, thrombocytopenia, systemic lupus erythematosus, lupus-like syndrome, vasculitis, optic neuritis, radiculopathy. Lesser vaccine effects include vomiting, abdominal pains, vertigo, dizziness, pruritis, angioedema, urticaria, lymphadenopathy, insomnia, dysuria, hypotension, herpes zoster, migraine, severe muscle pain and weakness, hypesthesia, alopecia, petechiae, increased sedimentation rate, tinnitus, conjunctivitis, visual disturbances, syncope, tachycardia, keratitis, irritability (see the Merck and GallaxoSmithKline package inserts).

The “cryptic argument,” that every person on the planet must be vaccinated because the hepatitis B “virus” can hide in cells in “chronic carriers” for decades without causing clinically detectable disease ignores the fact that in seropositive individuals without liver disease, the presence of the hepatitis antigens may represent non-specific markers of immunological stress, or merely represent a normal genetic polymorphism, as was originally thought by Blumberg. Therefore, because > 95% of cases spontaneously resolve, the presence of the Au antigen did (does) not predict who would (will) develop clinically detectable hepatitis or hepatocellular carcinoma, and thus the Au antigen cannot be a specific marker for the development of liver cancer any more than skid marks cause auto accidents. Yet the genetic polymorphic “cause,” or physiological stress “cause” of hepatitis as a form of autoimmune dysfunction has been ignored. Instead, a viral cause for hepatitis was advanced because it is yet again, “a filterable agent.” Blumberg and his colleagues reasoned that hepatitis might be caused by a virus because something smaller than bacteria that was associated with inducing transfusion hepatitis that could pass through filter pores too small for bacteria to pass (such as immunologically toxic serum proteins).

Despite widespread mandated hepatitis B vaccines for more than a decade, and claims that it can prevent hepatocellular carcinoma, no evidence whatsoever exists linking hepatitis B causally with hepatocellular carcinoma, as no animal models have ever exhibited this carcinoma after experimental infections, and no liver cell culture of normal human or animal liver cells has ever been induced to change into cancerous cells after adding the “hepatitis B agent” to them. Moreover, despite widespread mandated hepatitis B vaccination, liver cancer rates have increased in the US from 4 cases/ 100,000, in 1992, to 5.5 cases / 100,000, since at the end of 1999, according to the NCI and CDC’s official records.

This kind of fear mongering and propaganda not only ignores evidence showing that these possibilities are without foundation, but functions to stifle legitimate questions about the biology of the hepatitis B syndrome, or legitimate questions concerning the benefits and consequences of vaccination that should have been addressed before this (or any) vaccine was universally mandated.

1. Why is such alarm regarding hepatitis B sweeping across the planet now as a sexually transmitted syndrome, when jaundice (and assumed hepatitis) has been recorded in the medical literature since the time of the Ancient Greeks?
2. Because it is claimed that hepatitis B can only be spread through venereal contact or through exposure to infected fluids, is hepatitis B a new human disease or, if not, are humans more promiscuous now than they were during the Eleusinian Orgies and Roman Bacchanals chronicled by the ancient poets?
3. What evidence is there to substantiate that 350, 000,000 people in the world are “carriers,” and that 1,000,000 people in the US are carriers?
4. In this regard, why are the projected figures for hepatitis B given, when data recording the actual incidence of hepatitis B have been available for 20 or more years?
5. If the hepatitis B antigens are specific for the hepatitis B syndrome, and if these antigens don’t simply represent markers for certain physiological stress responses such as cancer or long term alcohol, drug use, or transfusion(s) with foreign proteins, or if the presence of the hepatitis B antigens don’t merely represent the different incidence and expression of certain Human genetic polymorphisms (differences in the kinds of molecules found in the blood of different peoples, as was originally thought by

Blumberg), then why did Blumberg and his collaborators find the hepatitis B antigens present in a vast majority of healthy people who never develop hepatitis (such as healthy Australian aboriginals or Micronesians or Vietnamese), or in patients experiencing other non-liver related illnesses or genetic disorders?

6. Do leukemia, hepatocellular carcinoma, and Down syndrome share something in common, or is the antigen merely expressed in these disease states in persons whose immunology is altered by cancer, substance abuse and dependency, genetic disturbance, or altered for some other cause of physiological stress?

7. If a hepatitis B (or C) virus could cause liver cancer decades after infection, then why does the microscopic percentage of those who exhibit the hepatitis B antigens and who develop chronic clinically detectable liver disease, require carcinogenic co-factors “such as fungal aflatoxins” (or “a lifetime abuse of alcohol or drug consumption”) to develop cancer, as indicated by Blumberg?

8. Why can't the hepatitis B virus (and C virus) be isolated according to standard isolation techniques even though as claimed by Blumberg, it is resistant to proteinases, even after a Roman effort and after decades of trying?

9. What substance(s), then, were actually isolated from sick persons, and used by Merck as antigenic material to make hepatitis B the first “molecularly derived recombinant” vaccine?”

10. Why hasn't any “HBV” isolate induced liver disease in chimpanzees, mice, or other organisms upon experimental infection? Why doesn't it induce either liver cancer or leukemia in animals? Why have there been no instances reported where “the hepatitis B virus” generated a pathological effect in animal models or in liver cells infected in vitro that even remotely resembles the hepatitis B syndrome's hallmarks in those tiny fraction of seropositive individuals who exhibit morbidity consistent with the so-called hepatitis B syndrome?

11. If the recombinant vaccine is molecularly specific against a hepatitis B virus and if the vaccine confers long-term immunity, then why does the vaccine ‘wear off’ after only several years? By contrast, when the real hepatitis B syndrome resolves in the vast majority of persons in nearly 100% of all cases who develop jaundice and demonstrable liver pathology, then why does this mild and transient syndrome provide lifetime immunity, and produce detectable antibody titres of the hepatitis B antibodies for at least 50 years? If these data are correct and acknowledged even by the vaccine manufacturers, then what is the logic behind vaccinating newborns when their immune and digestive systems are developing and fragile, and when their often hypothesized membership into in IV injecting, multiple sex partner, or blood product exposure risk group might occur a decade or more after the antibodies generated by the vaccine can no longer be detected?

12. Why have some studies shown an increase in the hepatitis B syndrome in vaccinated populations, such as The Gambian Study?

13. Why is the hepatitis B vaccine still mandated after a congressional hearing that put its safety in question, and why aren't are parents given any information at all about the possible adverse effects of the hepatitis B vaccine that are listed on the manufacturer's package inserts?

Let us conclude the hepatitis B era with a few choice selections from Baruch Blumberg's own PNAS and Nobel-worthy considerations and quotes, about hepatitis C and “HIV,” and leave an era that Blumberg began because he found a new molecular marker (the AU antigen) in a healthy Australian aboriginal black man's blood:

The Control of Post-Transfusion Hepatitis B

The clinical implications of the findings were quickly realized. In the United States in the 1960s a large percentage of blood donor

units were obtained from paid donors, although this was not the case in many countries in Europe, in Canada, and elsewhere. The incidence of post-transfusion hepatitis, both clinical and occult, was **enormous**. In some studies, **post-transfusion** hepatitis occurred in up to **50% of patients** who were receiving **large numbers** of transfusions for extensive surgical treatments such as open-heart surgery.

There were **casual observations** that recipients of donor blood containing Au developed hepatitis, but there were no systematic observations on the extra risk imposed. Okochi undertook such a study in Tokyo and reported a significantly increased risk for hepatitis in recipients of the positive donor units. We initiated a study at Philadelphia General Hospital, where the incidence of **post-transfusion** hepatitis was high, to determine the efficacy of a blood screening program. The results were very convincing. Before the screening program the incidence had been **17.9%**, and after screening it had been reduced by two-thirds to **5.9%**. In retrospect the **residual cases** were a consequence of the **low sensitivity** of the immunodiffusion method and cases due to **non-B hepatitis**.

Hepatitis C infection (non-B hepatitis) is **estimated** to have infected **3% of the population of the planet** or **170-200** million people worldwide. It is the most common cause of **chronic liver disease in some** countries. In the United States 40% of chronic liver disease is related to HCV. HCV infection can lead to cirrhosis, liver failure, and **hepatocellular carcinoma**. Between 1990 and 1992 routine antibody testing by enzyme-linked immunoassay (EIA) became available. Recombinant immunoblot assay (RIBA) is also available as are viral RNA detection tests for HCV. Treatment **with interferon in combination with ribavirin, is ineffective in the majority of cases** hence **the need** for a **vaccine**. [Mathilda Krim should have been thanked here of course for the interferon gift-my suggestion].

HCV is **a single-stranded RNA virus** and was **cloned** in 1989 at which time it was found to be the cause of **80% to 90%** of cases of non-A, non-B hepatitis. **Like HIV, numerous genotypes exist** and the virus can **mutate** rapidly, **this means, again like HIV, that developing a vaccine will be difficult**.

We knew that the Au particles, which we had isolated, and which Bayer and Werner had visualized in the electron microscope, did not contain nucleic acid (see below). We also observed that the HBsAg, which **signified** the presence of the virus, and the antibody against the surface antigen (anti-HBs), were never detected in the same individual. This was consistent with the explanation that anti-HBs was protective. Further, Okochi in Tokyo had showed that transfused patients who had anti-HBs, or acquired it after transfusion, were **less likely** to develop post-transfusion hepatitis than transfused patients who had not developed the antibody. [Less likely means many of them developed post-transfusion hepatitis despite antibodies]? The significance of this finding can be appreciated by considering the present status of vaccine research for **HIV, the AIDS-causing virus**.

Although the molecular biology of HIV is well understood, and recombinant virus proteins can be readily produced, it is not clear which of these antigens can generate a protective antibody or protective cellular immunity. (The development of an HIV vaccine is further complicated by the successful assault by the virus on the immune cells and the extreme mutability of the virus). Collaborating with M. Bayer and L. Loeb, we found that the small particles visualized in the electron microscope **did not contain nucleic acid**.

We **inferred** that these were **nonreplicating, incomplete forms** of the virus made up entirely of surface antigen [instead of meaningless cellular debris-my addition], and that **there must be** additional particles, **which contained nucleic acid, that were replicating, infectious, and pathogenic.** The **surface antigen particles** were highly resistant to proteinases, which allowed their purification from the serum proteins in the blood. In animal experiments Millman and London found that partially purified Au particles **that presumably** also contained the whole virus particles, **which we had not yet visualized**, could be transmitted by inoculation into experimental animals. The fully purified particles from which the whole virus had been removed

were **not** infectious. **The implication** was that we could separate the noninfectious particles containing only the surface antigen from the pathogenic whole virus particles. **Based on these findings we submitted a patent application in 1969** for the extraction of the surface antigen particles from the serum of human **carriers** of the virus...In 1980, the results of the first extensive field trial were published by W. Szmunes and his colleagues in New York City...HBV vaccine is the first, and so far, **only vaccine** to be produced commercially **by recombinant methods**. Vaccine **still** is produced **from the blood of carriers in China, Korea, and elsewhere**, and at present, remains a major source of supply.

This hepatitis B history shows how doctors try to come up with cures to reverse the damage that they had done through transfusions...but that is the subject of another book and horror story regarding the carnage of the hepatitis B vaccine (that will be visited later). Now, let us cast our nets broad and wide, my lads and ladies, because as a famous writer once said, “Freedom Lads is neither wine, nor maids, nor sweet sons in cradles: it is a lonesome mornful cry the wind has taken.” Let’s move on to hear the birth-pains of the AIDS era, known as HTLV-I and II fiascos, the First Human Retroviruses!

ART WORK RATHER THAN SCIENTIFIC EVIDENCE PROVES THAT BLACK SLAVES CAUGHT A CANCER VIRUS FROM MONKEYS IN THE 16TH CENTURY DURING THE SLAVE TRADE WHICH CAUSED JAPANESE LIVING NEAR NAGASAKI TO CATCH CANCER CENTURIES LATER, AFTER THE ATOMIC BOMB WAS DROPPED ON THEM

In 1975, Robert Gallo published a paper saying he had isolated a new human virus – human leukaemia virus 23 (<http://www.virusmyth.net/aids/data/javirus.htm>):

Gallo was jubilant, it was the justification for years of dedication. ‘We got permanently growing cell lines eventually, and it was a great eureka. We succeeded ten times in ten different cell lines, and we thought we had made the discovery, the genuine article, that retroviruses exist in humans. A year or more of analysis went by. We thought it was a triumph.’

This period of research turned from being Gallo’s greatest triumph to date into his greatest disaster. When other scientists looked at this virus they discovered it was a mixture of three animal viruses: from a gibbon, a baboon and a woolly monkey.

*As Gallo said: ‘I was depressed, dumbfounded, angry. It was the low point of my whole career. It was almost the last nail in the coffin of the field of retrovirology. The programme died, and all the good that came out of it, like interleukin-2, **which would be so important in fighting cancers**, didn’t seem to matter, to me or to the world. I became more cynical, tougher, less happy. I mean, what could it be but sabotage? One contamination can occur, but three? In fifteen years I had had one contamination from a mouse. But three?’*

After this so-called mistake, in 1978-9 Gallo’s group believed again they isolated a new human retrovirus they thought might cause leukemia, and called it “HTLV-I.” The results were published in 1980 and early 1981. The molecular marker of “HTLV-1” and the way it was obtained, was nearly identical for what was to be used to pursue of “the AIDS virus” a few years later. Descriptions of these initial “HTLV” and “HIV” characterization methods can be found in a new book, “Dissecting a Discovery” (<http://dissectingadiscovery.com/>) by Nikolas Kontaratos. Kontaratos is a security guard and filter salesman Gallo hired to write his recollections of the Dingell and HHS investigations regarding Gallo’s alleged theft of the virus from the Pasteur and Luc Montagnier’s group. Kontaratos wrote a compelling account of this collaboration in favor of Dr. Gallo’s positive role in the NIH-Pasteur collaboration, rather than about the widely held belief that Gallo stole the virus isolate from Montagnier’s group (see also an analysis by a Semmelweis Society-hired investigator) (<http://exlibhollywood.blogspot.com/2009/05/doctors-without-boundaries.html>).

Gallo is credited with isolating and describing the first human retrovirus. Japanese and American researchers confirmed by

analyzing the RNA of both isolates that the Japanese and American viruses were related strains of the same virus. They could never be exactly the same **because of the mutations** which occur as the virus replicates, but the RNA sequence was **close enough** in the two isolates.

Once the virus had been described, other laboratories looked for it. It was found **in black patients** born in the US, Caribbean countries or South America; Caribbean-born **black people** in England, **Africans and Japanese** [living 175 miles from Nagasaki]. What could tie these disparate regions together mused Gallo.

The answer he came up with was **the slave trade**. Miyoshi in Japan found Japanese macaques had **antibodies** to HTLV-I and he suggested the monkeys had the disease first and infected people. Researchers at Gottingen, Germany, and in Gallo's lab found that many species of **African** monkeys had antibodies which reacted with HTLV-I. **African green monkey** [same ones that contaminated the polio vaccine with SV-40-my emphasis here] and chimpanzee **viruses** [antibodies-my correction here] were most closely related to the virus Gallo had found in leukaemic cells.

Gallo suggested: HTLV-I originated in Africa where it infected many species of Old World primates including human beings. It reached the Americas along with the slave trade.

Curiously, it may well have arrived in Japan the same way. In the sixteenth century Portuguese traders traveled to Japan and stayed specifically in the islands where HTLV-I is now endemic. **Along with them they brought both African slaves and monkeys, as contemporary Japanese works of art show, and either one or the other may have carried the virus.**

The discovery of HTLV-I infection on Hokkaido, one of the northern islands of Japan, immediately challenged this view of events but Gallo and his colleagues have remained attached to the monkey-virus theory. [The monkey-African slave-virus theory-my translation here].

So, why is it thought that this virus causes the leukaemia? 'First because of the coincidence between virus and leukaemia – find one and you will find the other,' Gallo says. [Like skid marks causing auto accidents or fire trucks causing fires-my emphasis here].

The incidence of adult T-cell leukaemia in Japan is estimated to be **only 0.06** per cent based on **339 cases of T-cell leukaemia among 600,000 subjects** who are antibody-positive for HTLV-I. Why is this? Because of **the latency period**, responds Gallo. It **will cause** leukaemia, but it may take as long as **forty years**.

Thus, despite the art work evidence that proved to Dr. Gallo that "**The first human leukemia retrovirus**" originated during "the slave trade," these hypotheses and notions of Gallo regarding the origin of "HTLV-1, HTLV-II, hepatitis B, hepatitis C, and later, "HIV," should be considered in light of what Africans and African scholars have written (see **Appendix 2: Rosalind Harrison-Chirimuuta and Richard Chirimuuta: Racism and "AIDS from Africa."**)

"THE AIDS VIRUS ORIGINATED FROM BLACKS!"

The amplification and characterization of "HIV" as another first Human cancer retrovirus, an "Acquired Immune Deficiency-causing" rather than a cancer virus in this case, was based on Dr. Gallo's claims and techniques used by molecular biologists of that day to detect **reverse transcriptase**, and later of course, from art work evidence he saw proving that black slaves and monkeys brought "HTLV-1" and "HTLV-II" to the Japanese near Nagasaki to cause leukemia there in 0.06%, of 600,000 "carriers" as just described. Thus, a second mistaken putative human cancer retrovirus, and then a third and fourth mistake, "HTLV-I and II," or rather, their assumed reverse transcription markers, were brought to the world's attention by Dr. Gallo, his collaborators, and the U.S. government during the late 1970's and early 1980's.

Also, as will be briefly discussed later, the first toxic treatments devised to quell Acquired Immunity Deficiency Syndrome would become erected on the same theorizing about mutation, racism, and war-inspired science used during “the War on Cancer,” and cocktails would be designed to defeat “mutation” in the same manner and in some cases, with the very same failed drugs, like the currently regarded mutagen, AZT, developed for toxic cancer therapy, but which had been shelved for its toxicity a decade before it was used at high dosages to kill a generation of mostly gay men and others, like Arthur Ashe, or Kimberly Bergalis. Today “life saving” AZT is now listed on California’s Office of Environmental Health Hazard Assessment registry as a cancer-causing mutagenic chemical and carcinogen since 2009 (http://www.oehha.ca.gov/prop65/prop65_list/files/P65single100810.pdf).

Yet all the evidence there is regarding profound immune suppression since the beginning of the AIDS era suggests the detection of “HIV,” when it can be ruled out as a non-specific molecular reaction, did not arise as an infectious entity from African children playing with monkeys as toys, from African “bush-meat monkey food” that infected people’s mouths or intestines, or from “bizarre African sexual practices,” as has been stated in high-priced journals such as *Nature* or *The Lancet*. Nor did it emerge from a government “special virus program aimed at depopulation,” as Boyd Graves, Leonard Horowitz, and other conspiracy theorists have advanced. Other hypotheses and work with other pathogens were advanced as causes and co-factors for AIDS before and during the AIDS era, such as mycoplasma infections, “hepatitis B” vaccinations of gay men guinea pigs in New York, smallpox and flu vaccinations, and autoimmune syndromes, but these and many other potentially immune-suppressive phenomena such as drug dependency or popper use (amyl nitrites) were not welcomed as cogent reasons for AIDS by our Churches of Modern Medicine.

A close look at the history of events during the AIDS era, with it’s 30 or more hypotheses as to what causes a collection of 58+ syndromes subsumed under the AIDS umbrella, were all known before the era of AIDS, and together with the wars on cancer and epidemics that preceded it, reveals that it is clear that cultural biases and faith based thinking, rather than a scientific method, has guided much of this viral “science” and medical interventions in the context of these “new diseases.” These hypotheses have paved the way for some imaginative, yet latently racist, homophobic, or faith-based theories of “HIV’s” origins and pathogenesis. Especially important, cogent immunological constructs like immune chain reactions, or imbalances of the T-cell ratios, or the effect of multiple foreign antigens on the human immune system that are measurable, also have been ignored, unfunded, or were not pursued in favor of a one cancer-like virus fits all theory.

Other “top AIDS scientists,” “experts,” and high priced gloss-covered science and medical journals even have advanced the idea that Africans smeared monkey blood on their loins to enhance their sexual experience, or have engaged in “dry sex,” and we still are reminded occasionally by the government media that these African men continue to promote to this day, the raping of their own child virgin daughters to prevent “HIV”. As stated in *The Lancet*:

“Sir: The isolation from monkeys of retroviruses closely related to HIV strongly suggests a simian origin for this virus... Several unlikely hypotheses have been put forward... In his book on the sexual life of people of the Great Lakes area of Africa Kashamura wrote: to stimulate a man or a woman and induce them to intense sexual activity, monkey blood [for a man] or she-monkey blood [for a woman] was directly inoculated in the pubic area and also the thighs and back. These magic practices would therefore constitute an efficient experimental transmission model and could be responsible for the emergence of AIDS in man.” (9. Noireau F. HIV transmission from monkey to man-*Lancet* (i):1498-1499, 1987).

Although nobody has ever seen a cancer or AIDS virus “jumping” from a non-human primate to an African or an African American woman’s HeLa cells, as recently as 2008, and according to their new *genome* analyses, and their comparisons between what they think are “HIV” and “SIV” “genome” sequences, we even frequently still find that the high priced magazine *Nature* publishing a claim that “HIV” and AIDS was the fault of dark-skinned African peoples and their “close associations” with non-human primates.

These imagined zoonotic exchanges are said to have first occurred in places such as Cameroon, when the Africans there built cities near chimps, and had “high risk” behaviors near them, **or with them**, 125 years ago. It is my contention that these now

institutionalized racist theories of the origin of “HIV” arrived at via sequence analyses still may not demonstrate beyond all doubt that “HIV” “jumped” from monkeys and apes to blacks, and then somehow of course to gays in California and New York who visited Haiti, and then to many white American victims like the AZT-killed Kimberly Bergalis, Ryan White, or to Tommy Morrison the prize fighter whose career was ruined after an “HIV” diagnosis before he tested negative multiple times to restore his boxing career, or the mother and daughter of the Glaser family, or AZT-killed athletes such as tennis star Arthur Ashe, and the ballet master and National treasure, Rudolf Nuryev, and indeed approximately 33 million others. Instead, my contention is that these ideas of initial species jumping, rather than phylogenetic conservatism or non-conservatism of genetic sequence homology form the flawed foundations of both modern cancer and “AIDS science.”

In the context of imagined molecular phylogenetic conservatism, “SIV” has always been a better animal model of “HIV” than “HIV,” precisely because no animals have ever been shown to get sick by direct injection of “HIV” or the sera from AIDS patients, similar to hepatitis B.

When the Promoters of AIDS continue to ignore phylogenetic conservatism among molecules evolved among primates, and continue to claim that “HIV” undoubtedly emerged from Black Africans “close associations” with chimps and gorillas, perhaps when they built cities with them 125 years ago” as described in prestigious peer-reviewed journals such as *Nature*, *Nature Medicine*, *The Lancet*, or *the New England Journal of Medicine*, which claim they ate them as “bushmeat,” or that AIDS is a problem of too many smallpox vaccines or not enough of them, or due to limited “social mobility,” or bizarre sexual practices, then one might out of common sense ask if there is good cause to perhaps consider other hypotheses regarding the emergence and phenomenon of AIDS?

A good example of this racist thinking was advanced as recently as 2008. An African woman tested “HIV” positive and was said to have had a high probably of “exposure(s)” with a gorilla or a chimp. And in doing so, she somehow caught the virus and it rapidly mutated in her body (although she didn’t have any symptoms-she is a healthy “carrier” of a **New AIDS virus**). Now, because doctors would say the black woman is probably lying because she claims she had no such association with a gorilla and is not ill, she is creating the threat of yet a new global pandemic AIDS virus, and a new virus that causes AIDS.

But why would any scientific journal publish any such claim that, although she is not sick, this woman in Cameroon definitely caught “HIV” from a gorilla or from a gorilla through chimp exposure, and like the Andromeda strain, it has mutated immediately in her body to form a new, rapidly growing, probably widespread, deadly pandemic virus, as reported in *Nature Medicine* and news-flashed throughout the world on government-controlled media like ABC news?

NEW HIV STRAIN DISCOVERED IN WOMAN FROM CAMEROON, Randolph Schmidt, AP Science Writer (<http://www.dailymail.co.uk/news/worldnews/article-1204035/New-strain-HIV-gorillas-woman-Cameroon.html>).

“ A new strain of **the virus that causes AIDS** has been discovered in a [black-my emphasis] woman from the African nation of Cameroon.

*It differs from the three known strains of human immunodeficiency virus and **appears** to be closely related to a form of simian virus recently discovered in **wild gorillas**, researchers report in Monday’s edition of the journal **Nature Medicine**.*

*The finding “highlights **the continuing need to watch closely for** the emergence for new HIV variants,” said the researchers, led by Jean-Christophe Plantier of the University of Rouen, France.*

*The three previously known HIV strains are related to **the simian virus that occurs in chimpanzees**.*

*The **most likely explanation** for the new find is **gorilla-to-human transmission**, Plantier’s team said. But they added they*

cannot rule out the possibility that **the new strain started in chimpanzees and moved into gorillas and then humans, or moved directly from chimpanzees to both gorillas and humans.**

The **62-year-old patient** tested positive for HIV in 2004, shortly after moving to Paris from Cameroon, according to the researchers.

She had lived near Yaounde, the capital of Cameroon, but **said she had no contact with apes or bush meat**, a name often given to meat from wild animals in tropical countries. **The woman currently shows no signs of AIDS** and remains untreated, though she still **carries** the virus, the researchers said. How widespread this strain is **remains** to be determined. Researchers said **it could be** circulating **unnoticed** in Cameroon **or elsewhere**. The virus' **rapid replication** indicates that it is **adapted to human cells**, the researchers reported. Their research was supported by the French Health Watch Institute, the French National Agency for Research on AIDS and Viral Hepatitis and Rouen University Hospital."

As February 11, 2010, *The New England Journal of Medicine* published:

AIDS IN AMERICA: FORGOTTEN BUT NOT GONE, BY WAFAA M. EL-SADR, M.D., M.P.H., KENNETH H. MAYER, M.D., AND SALLY L. HODDER, M.D.

"Over the past decade, **limited attention** has been paid to the human immunodeficiency virus (HIV) **epidemic** in the United States. The global epidemic—particularly the epidemic in sub-Saharan Africa, where approximately two thirds of the world's population living with AIDS resides—has **rightfully** received most of the focus. Meanwhile, however, the prevalence of HIV infection within some U.S. populations now rivals that in some sub-Saharan African countries (see graph). For example, **more than 1 in 30** [black-my emphasis] adults in Washington, D.C. are HIV-infected—a prevalence higher than that reported in Ethiopia, Nigeria, or Rwanda. Certain U.S. subpopulations are particularly hard hit. In New York City, **1 in 40** blacks, **1 in 10** men who have sex with men, and **1 in 8** injection [drug abusers] **are** HIV-infected, as are 1 in 16 black men in Washington D.C."

Why such a disparity between Washington D.C., New York City, African AIDS, and the rest of the developed world, according to the views of these esteemed authors and world-renowned NEJM journal editors who monthly publish these kinds of articles? El-Sadr, M.D. et al continue:

"For the past decade, however, **progress** has been **stalled**. It had been anticipated that effective antiretroviral therapy, with its suppressive effect on **viral replication**, would reduce the overall rate of new infections, **but this expectation has not been realized**."

Why not?

"Many of the populations most affected (in the U.S.) tend to have limited **social mobility**..."

THAT A HUMAN "RETROVIRUS" "HTLV-1" CAUSES RARE CASES OF LEUKEMIA 175 MILES FROM NAGASAKI IS LIKE SAYING, "I HAVE 20,000 BIRDS..."

Due to this multifactorial singularity type of logic that advanced the much cherished cancer biology and retrovirology of the 1970'-1980's, and indeed today, we can now say that to advance the idea that the so-called cancer-associated Human Retrovirus, "HTLV-1" causes leukemia in Humans at a rate of 0.06% out of 600,000 "carriers," is like saying:

I have 20,000 birds. 10,000 of these birds molt once a year. The other 10,000 molt 3 times a year. Now, none of the 10,000 birds

that molt once a year died by hitting their head into utility poles.” However, 6 of the 10,000 birds that molt 3 times a year died by hitting their head into utility poles. Therefore, among these birds, their molting 3 times a year CAUSED them to hit utility poles and die. Molting 3 times annually → hitting utility poles → death.

Of course, this is a purely hypothetical and statistical argument, but it demonstrates how worthless such arguments are that claim that cancers occur at a rate of 0.06% of those hundreds of thousands who carry the identical molecular marker called a cancer virus without acquiring cancers. There simply aren't any controlled studies on HTLV-I and ATL that demonstrate what was needed to be demonstrated to advance the hypothesis that any of these “tumor viruses” could change a normal cell into metastatic cancer cell, or produce a tumor in an experimental animal using purified HTLV-I or HTLV-II. With this type of multifactorial singularity-style of logic, similar arguments apply to “HIV’s molecular signature(s), and for the exact same reasons. It is an inevitable consequence of statistics that happen at low frequency that are thought to cause something to happen.

Let me give a real example, rather than with birds. In 2004, the American Red Cross reported that even after repeated “HIV” testing using different test kit types, that “low-risk” populations, such as blood donors (or military recruits or nuns) will typically yield 12 (PCR) positive or 2 (ELISA) positive results out of 37,000,000 million units of blood, which means that 10 out of 12 were false positives. In a follow-up analysis of this Red Cross study, it was then claimed that 6 of the 12 PCR-positive subjects tests seroconverted within several months, thereby obtaining a “HIV” molecular signature in 8/12 cases, out of 37 million negatives. Again, these numbers could represent statistical artifact, or, the several who seroconverted may represent the detection of some kind of auto-immune condition in those who test positive, like psoriasis, arthritis, physiological stress, pregnancy, or a genetic polymorphism.

Multifactorial singularities like these arise and become institutionalized as causal forces or processes despite the contradiction of simple logic: if a virus X causes cancer in 1 in a million who also have that virus or its markers, then 999,999 times out of a million it doesn't cause cancer. Therefore virus X causes cancer. In simple language, a multifactorial singularity is “a cause” confounded by many factors that influence statistics. Although they frequently employ math and numerical data, multifactorial singularities have no predictive power. Sciences such as Newton's science “works,” and we can fly airplanes or space ships, or predict where a bullet or an intercontinental ballistic hydrogen bomb will land in advance, after accounting for all or most of the variables like weather, masses of objects, and the rest. In this context, because the forces are known and measurable, it does not matter that we are still seeking “the cause” of gravity, which becomes irrelevant. When multifactorial singularities like “HPV and cancer, or “HBV and cancer” or “HIV/AIDS” arise, and their “forces” or pathogenesis is unknown, all that becomes relevant is the imagined cause, and everyone is unequivocally assured that it is deterministic or causal that “HIV” causes AIDS, except of course, not in the minds of the hypotheses' initial framers, like Rous. Luc Montagnier, for instance, said in a recent documentary that: “you can catch it (“HIV”) many times and if your immune system is strong you can get rid of it” no problem! (This specific interview with the Nobelist, Luc Montagnier, discoverer of “HIV” can be watched at and the entire film seen, “House of numbers” in its entirety at:

Luc Montagnier:

*“...I believe HIV, we can be exposed to HIV many times without being chronically infected. **Our immune system will get rid of the virus in a few weeks, if you have a good immune system...***

“If you have a good immune system, then your body can naturally get rid of HIV?”

Luc Montagnier:

“Yes.”

See Montagnier's interviews:

<http://www.youtube.com/watch?v=WQoNW7lOnT4l>,

http://www.facebook.com/l.php?u=http%3A%2F%2Fhiv-dissidents.tabaru%2Fvideo%2F%2F26508_Dom_chisel.html&h=f6d5eYwjJV-GDsJMqI3gQp8u_Vg

And yet these statements are only a glimpse of similar thoughts and statements of those to whom we bestow science's highest Nobel awards. Yet we are repeatedly taught by the government and military-supported media campaigns such misinformation and fear. "Everyone is at risk," you can catch "it" through just one contact, you don't need co-factors, Africa's population is being decimated by a virus although it's population has no AIDS pandemic, that an antibody positive test is something to fear, that sexually-transmitted viruses jumped from non-human primates to black persons because biologists ignore phylogenetic sequence homologies in favor of racist thinking backed by phony science, etc. But when you check the primary sources of various Nobel discoverers as they recount their glory, their hard won hypotheses, and listen to them candidly discuss their caveats, however, it becomes clear that forces other than science and reason are at work in most if not all of our higher educational systems, medical schools, and indeed, even in elementary school science and health classes. In this context, apparently the views of Payton Rous, Luc Montagnier, and many others are worthy of some investigation and analysis and intense questioning, and for many of the reasons they themselves provided.

With these Nobel prize-worthy suggestions, Dr. Montagnier also has in effect dusted off the Century-old argument of his own country's biomedical forerunners, Louis Pasteur and Antoine Bechamp, regarding the supremacy of soil or terrain versus the seed, microbe, or toxin in the development of clinically visible disease. Thus among a few biomedical researchers Like Dr. Montagnier at least, it is becoming ever more widely acknowledged even by the Nobelist who is credited with first discovering "The AIDS Virus" that the soil or terrain of the organism, and not the "seeds" or germs (or "HIV"), ultimately determines if and how much illness will result from any seed, imagined or real.

And because Montagnier is now claiming that "we can be exposed to HIV many times without being chronically infected...*because our immune system will get rid of the virus in a few weeks, if you have a good immune system,*" then clearly, Montagnier has sided with Antoine Bechamp's argument, and not Pasteur's in the context of "HIV" and AIDS.

Concordant with Dr. Montagnier's interview, in a recent issue of *AIDS Clinical Care*, (*AIDS Clinical Care*, June 1, 2009) front-line AIDS clinicians published statements that should concern the Obama Administration, and indeed every person who wants to live in a world without AIDS. Dr. Abigail Zugar stated it in most clearly:

"The Puzzle of CD4-Cell Depletion Despite Good Viral Suppression. In some patients, CD4-cell counts fail to rise as expected. Could extensive lymph node fibrosis be responsible?"

In other words, "AIDS" is unexpectedly progressing despite treatments with "life saving anti-retrovirals" and in many cases without "HIV" being detected. Even more concerning statements followed:

"In a recent study, NIH researchers sought evidence to support **any** of several **hypothetical explanations** for the aberrant CD4-cell responses seen in four patients on combination ART whose CD4 counts had fallen from a median of 719 cells/mm³ to a median of 227 cells/mm³ **despite persistently undetectable plasma viral loads.**"

These patients were taking a double or triple AIDS-drug cocktail regimen, no "viral load" could be detected, yet their T-cells were plunging from relatively normal levels to the worrisome and CDC-defined level reached when doctors suggest that drug therapy

should begin, at around 200-300 CD4+ cells/mm. ³

Drug “resistance” was “checked” and found not to be an issue. However, what is most troubling is that Dr. Zugar then writes that:

*“Residual **replicating** HIV did not seem to be **the problem**: Results of **ultrasensitive** PCR and assays for peripheral blood mononuclear **cell-associated** HIV RNA and proviral HIV DNA — and of assays for cell-associated HIV RNA and proviral DNA in mononuclear cells from inguinal lymph nodes — were similar to those obtained in other, **successfully treated** patients.”*

It is not the first time that fibrosis in the lymph nodes has been discussed as being a chief issue in “AIDS patients,” as Dr. Zugar correctly stated:

“The single unusual finding was a striking abnormality in inguinal lymph node architecture...”

Or: *“...the unusual lymph node architecture documented in all four patients in this study may be related to (or even responsible for) the inadequate CD4-cell response — i.e., that CD4-cell depletion is independent of specific components of an antiretroviral regimen and is instead caused by lymph node fibrosis. They note that such architectural damage may well be “clinically **irreversible** with **currently available** interventions.”*

This also is why the language used to describe reality in science is so important and precise. Yet one must ever be on guard as to how fruitless scientific progress is typically buried in a language and odd results that attempt to conceal such “multifactorial singularities,” racism, and religion. One example might be useful here to understand this philosophical and scientific disconnection, which is why I have emboldened certain words here that attempt to modify and mollify a completely wrong but ardently defended so-called science in which the assumptions are buried or distorted in the invented language itself.

During the entire history of AIDS, although there have been five “successes” claimed by the biomedical establishment, there are recurring themes and principles to be learned from these claimed successes. The successes include:

- 1) Dr. Nancy Padian’s claims that she counseled no less than 175 “HIV-positive” persons who had unprotected sex over a 10-year period and who did not transmit even one positive test result to their non-“HIV-positive” serodiscordant partners, principally because of Padian’s ability to counsel them so well;
- 2) Dr. Robert Bailey’s ability to give good advice to STD-prone African “black” men during his African male circumcision studies, about which he claims the statistical incidence of documented “HIV transmission” was half of that measured before they were circumcised and intensively counseled (personal communication);
- 3) Dr. Gero Hütter’s claim that he saved one man from his leukemia and AIDS after he whole-body irradiated him, and after he gave him a bone graft to regenerate his immune system, and the man’s “HIV-positive” result disappeared for 2 years (how did irradiation get rid of this man’s “HIV?”), and 4), the many thousands of anti-retroviral studies, especially those which have claimed to block or prevent mother-to-child-transmission (pMTCT), and which have been said to greatly extend the length and quality of life with “life saving antiretroviral drugs.”
- 4) Antiretroviral therapies.
5. The newly disclosed Thailand-Military trial that didn’t achieve statistical significance yet Fauci claimed reduced AIDS by 50% although aluminum is probably cheaper to inject into populations.

With a little critical review, however, it is far easier to identify the enormous universal and colossal failures including the recently halted “HIV” STEP vaccine trial, which was likened to a “Challenger-sized disaster.” The even larger Thailand-US military vaccine trial that followed STEP on some 16,000 human beings also was halted.

What is the current state of medical care regarding “HV/ADS?” According to hopeful reports news flashed around the globe, we perpetually are bombarded with claims such as the following:

AIDS PATIENTS CAN LIVE ON AVERAGE 24 YEARS IF THEY PAY \$385,000 DOLLARS.

“In 2006, Dr. Bruce R. Schackman, chief of health policy at Weill Cornell Medical College in New York and lead author of a paper appearing in Medical Care in 2006, a journal published by the American Public Health Association claimed that “...patients can live average 24 years, if they pay \$385,000.”

SURPRISINGLY, NEUROCOGNITIVE FUNCTION (ABILITY TO THINK) IMPROVED IN HIV-INFECTED PATIENTS WHO INTERRUPTED ANTIRETROVIRAL THERAPY²⁵

*“Conclusion: Subjects with preserved immune function found that neurocognition improved significantly following antiretroviral treatment (ART) **discontinuation**. The balance between the neurocognitive cost of untreated HIV viremia and the **possible** toxicities of ART **require consideration**. Classification of evidence: This study provides Class III evidence that discontinuing ART is associated with **an improvement** in 2 neuropsychological tests (Trail-Making Test A & B and the Wechsler Adult Intelligence Scale–Revised Digit Symbol subtest) for up to 96 weeks. Resuming ART was not associated with a decline in these scores for up to **45 weeks**.”*

TRANSLATION: Stopping the anti-retroviral drugs causes people to be able to think again.

“HIV” CAUSES 15 “CANCERS” OR PROBABLY ALL OF THEM (which are a spectrum of diseases characterized by too many cells rather than too few cells)?

As mentioned in the beginning of this story about the sacrificing of virgins, in 1993, **cervical cancer** was included in the revision of the surveillance case definition for AIDS, without of course any evidence so far that any virus could cause human cancers. But after the suggestions that cervical cancer and Kaposi’s sarcoma are caused by “HIV,” an increasing number of cancers were attributed to have either been caused directly or indirectly by “HIV,” as long as antibodies, proteins, glycolipids, or genomic fragments indirectly associated with “HIV” are detected in a cancer patient.

At the first international meeting devoted to AIDS and cancer sponsored by the National Cancer Institute 4 years later, the reporter and Epidemiological Intelligence Service agent, Lawrence Altman of the New York Times wrote the following:

AIDS Patients Face Growing Threat From Cancer. New York Times May 6, 1997:

*As more people with AIDS survive longer, many are confronting yet another serious health problem: **cancer**.*

*Malignant tumors, particularly Kaposi’s sarcoma and certain cancers of the lymph system, have been common among AIDS patients since the disease was first recognized in 1981. But now the array of cancers is broadening **in mysterious ways**. Once rare cancers are appearing more often. Some kinds of cancer that were expected to increase in frequency **have not**. And **strange** geographical patterns of certain cancers are emerging.*

It is a baffling situation, yet scientists see hope in it. Researchers are looking at the puzzling trends with the aim of identifying and using biological clues to unlock the secrets of the myriad cancers that afflict people who **are not** infected with HIV, **the virus that causes AIDS**, and to devise new ways to treat them.

Like everyone else, people with AIDS can develop any kind of cancer. And to a doctor, a slide of a specific tumor looks the same under a microscope, regardless of whether it came from a person infected with HIV.

Now, though, the new patterns of disease have raised the possibility that further studies of **subtle** differences might eventually help researchers improve cancer therapy.

An emerging theory is that most AIDS-related cancers are somehow linked to viral infections. But doctors do not know if HIV enhances the ability of other viruses to produce certain cancers, scientists said here last week, at the first international meeting devoted to AIDS and cancer. The meeting was sponsored by the National Cancer Institute.

Dr. Richard Klausner, the director of the federal institute, said that at least **30 percent** of people with AIDS **also** developed cancer. The frequency is **estimated** from a variety of studies and cancer registries using information collected from hospitals and death certificates.

Dr. Valerie Beral of the University of Oxford in England said that studies had **definitely** linked five cancers with HIV infection: **[1]** Kaposi's sarcoma (skin and internal organs); **[2]** non-Hodgkins lymphoma (lymph system); **[3]** squamous cell cancer of the conjunctiva (eye); and in situ (early stage) carcinomas of the **[4]** cervix and **[5]** anus.

Three other cancers are probably related to HIV. **[6,7]** Two affect the lymph and blood system (Hodgkins disease and plasmacytoma) and **[8]** one affects muscle (leiomyosarcoma). Once rare, leiomyosarcoma has become the second most common cancer in HIV infected children but, for unknown reasons, not in adults.

Studies have also suggested an increase in **[9]** lung, **[10]** lip, **[11]** testicular and other cancers. Although the overall incidence of breast cancer is not increased in AIDS patients, several cases of unusual types of aggressive **[12]** breast cancers have been reported. But their link to AIDS remains uncertain.

...With more widespread use of **anti-retroviral drug treatment**, AIDS-related cancers are happening less often. **But as people with HIV are living longer, they are developing cancers that are not generally linked to HIV**, such as lung, **[13]** throat, **[14]** liver, intestinal, and anal cancers as well as Hodgkin disease and **[15]** multiple myeloma. Most of the time, treatment includes ART along with cancer treatments that have worked for people without HIV. At the same time, any other needed treatments for HIV (such as antibiotics to prevent infections) are used.

Most perplexing is why HIV-infected people are not developing liver and certain other cancers. For example, many HIV-infected people are also infected with either the hepatitis B or C viruses, which increase the risk of liver cancer, yet they are not unusually prone to liver cancer.

The meeting took place **at a time of optimism about AIDS**. Combinations of newer anti-HIV drugs like protease inhibitors, and older ones like AZT, **can suppress the virus** to levels below the limits of detection by the latest laboratory techniques.

But whether the new therapies will reduce **or increase** cancer rates among people with HIV **is not known**.

But there was little formal discussion about the adequacy of the current techniques to track cancer among people infected with HIV and to detect what proportion, **if any**, might be caused by the new combination anti-HIV drugs themselves. In hindsight, members of the program committee said in interviews, they **should have** added this problem to their agenda. The cancer institute's system to track cancers among AIDS cases relies heavily on studies and registries that link the two diseases. Two officials at the institute, Dr. Ellen Feigal and Dr. James J. Goedert said the existing system **should be** able to detect an increase in cancer rates among long-term survivors of HIV infection, though the information may be delayed.

The Centers for Disease Control and Prevention also tracks AIDS-related cancers through its own system. It includes information about certain cancers if they are detected at the time AIDS is diagnosed, said Dr. Harold W. Jaffe, an AIDS expert at the federal agency in Atlanta.

But if an AIDS patient develops a cancer after the initial report, he said, "we probably do not know that, and we need to do a **better job** of getting that information in a timely way."

If the frequency of AIDS-related cancers does increase or turns out to be linked to anti-HIV drugs, it would be important to get the word quickly to patients and their doctors, who are now being urged to begin combination therapy early in the course of HIV infection.

Dr. Alexandra M. Levine, an AIDS and cancer expert at the University of Southern California, said she was "extremely worried that we will see more and more cancer" among HIV-infected patients.

Treatment of HIV infection in its early stages **might prevent major damage** to the immune system, though it could still leave an individual with **a slightly** impaired immune system. "If therapies prolong lives, the immune system **may be** more effective at combating HIV than in controlling cancer cells" because different components of the immune system may be involved in fighting the two diseases, Levine said.

Earlier this year, a panel appointed by the National Institutes of Health urged careful long-term follow-up of all children whose mothers took AZT during pregnancy. **The use of AZT in pregnancy has been one of the most striking successes in the AIDS story because it greatly reduces maternal transmission of HIV to a newborn. But a study has shown development of tumors among the offspring of mice that received very high daily doses of AZT during pregnancy.**

It was the sudden appearance of the Kaposi's sarcoma cancer in large numbers of gay men in New York City that led doctors to recognize what is now called AIDS. Until then, Kaposi's sarcoma had been rare, and few experts suspected that it was related to a virus.

Then in late 1994, Dr. Patrick S. Moore and his wife, Dr. Yuan Chang, working at Columbia University without a federal grant, used **a new technique** to discover human herpes virus 8 (HHV-8). Further studies suggested that infection with HHV-8 **was needed** before Kaposi's sarcoma developed. Scientists have also linked HHV-8 to a rare type **of lymphoma** in people infected with HIV. **But scientists do not know how HHV-8 virus causes normal cells to become cancerous or precisely how it is transmitted, and they continue to debate the possible ways.**

Might other viruses help produce other cancers[in humans-Rous's emphasis, and mine]? Scientists are **now looking hard**. For example, virtually all lymphomas arising in transplant recipients are related to the Epstein-Barr virus. But only about 50 to 75 percent of the lymphomas arising in AIDS patients are linked to Epstein-Barr.

Dr. Robin Weiss of the Institute of Cancer Research in London said he was among those who had sought but failed to detect a new virus in the lymphomas linked to Epstein-Barr among people with AIDS. [Sounds like something Payton Rous might say in his Nobel speech]?

One enigma is why HIV-infected women have about four times the risk of developing in situ carcinoma of the cervix yet do not go on to have an increased incidence of the more deadly invasive cervical cancer.

“It defies logic,” Levine said.

*Experts said they doubted the explanation that it was because Pap smears and other tests were preventing the progression of the cervical cancers. Invasive cervical cancers **are not** being detected in Africa and other areas where such health care is not available.*

*Another enigma concerns the Burkitt’s type of lymphoma, which can produce grotesque tumors of the face and neck. Before the AIDS epidemic, Burkitt’s was common in central Africa, but rare in developed countries. Now an **estimated 2** percent of AIDS patients in developed countries get Burkitt’s lymphoma, said Dr. Alan Rickinson of the University of Birmingham in England.*

*Rickinson said he **had assumed** that doctors in Africa, **where there is an epidemic of HIV infection**, “should see a flood of Burkitt’s coming in.” But Rickinson said he was **astonished** to learn last week from doctors at a cancer registry in Kampala, Uganda, that the incidence of Burkitt’s **not increased** among Africans **infected** with HIV.*

“This is completely counterintuitive,” Rickinson said. Yet, he added, “They tell me that the spectrum of AIDS-associated malignancies in Africa is different from that which we see in the West.”

*Burkitt’s has historically occurred in regions of Africa where malaria commonly infects children. Rickinson speculated that HIV infection, **by mimicking the chronic effect of malaria through an unknown mechanism on the immune system**, might somehow protect against Burkitt’s lymphoma.*

***If** scientists can identify such protective mechanisms and other viruses that help produce HIV-related tumors, then they can target them in developing new strategies to prevent Burkitt’s and other cancers. For example, several scientists reported using such an approach in treating Epstein-Barr-related lymphomas.*

According to a recent revised 05/12/2008 update from the American Cancer Society, the current establishment thinking about “HIV” and cancer is as follows:

Certain types of cancer occur so often in people with AIDS that they are considered AIDS-defining conditions – that is, their presence in a person infected with HIV is a clear sign that full-blown AIDS has developed. They are also called AIDS-related cancers and include the following:

* Kaposi sarcoma

* lymphoma (especially non-Hodgkin lymphoma and primary central nervous system lymphoma)

* invasive cervical cancer

Other types of cancer that may be more likely to develop in people with HIV infection are invasive anal cancer, Hodgkin disease,

lung cancer, cancer of the mouth, cancer of the testicles, and skin cancers, including basal cell, squamous cell, and even malignant melanomas. **Of course**, people without HIV or AIDS can also have all of these types of cancer, even the ones that are better known as AIDS-related. They are only called AIDS-related cancers if they develop in people with HIV infection (“HIV” antibodies-not “infection”-my correction).

*In developed countries, about **4 people in 10 with AIDS** develop cancer at some time during their illness. However, the cancer picture in HIV is changing. Kaposi sarcoma and non-Hodgkin lymphoma have decreased **as anti-retroviral treatment has become more common**. Most other types of cancer do not seem to be slowed by HIV treatment, and have the same risk factors as those in people without HIV. For instance, **people who smoke** and have HIV are more likely to have cancers of the lip, mouth, throat, and lung than people who have HIV and don't smoke.*

The relationship between HIV and these other cancers is still not completely understood. However, it is believed cancers can grow quickly because people with HIV have weakened immune systems [just like Chester Southam's 40 chronically sick Jewish patients-my contribution].

*Unfortunately, cancer in people with HIV can be harder to treat. This is partly because of the decreased immune function caused by HIV and the lower white blood cell count that can result from HIV infection. Having AIDS can make it **hard** for a person to take chemotherapy because the bone marrow, which is needed to make new blood cells, is **sometimes** already damaged by **the HIV infection**. People with this problem often can't take **the full dose** of chemotherapy without serious **harm** [without death-my emphasis and observations]. The introduction of highly active anti-retroviral therapy or HAART in the late 1990s led to a decrease in some types of cancer among those with HIV and better survival with anti-cancer treatment. It has also allowed full chemotherapy doses to be used for treatment. Other types of cancer treatment, such as monoclonal antibodies and stem cell transplant, are being studied in people with HIV.*

STOPPING THE AIDS EPIDEMIC IN AFRICA USING THE POWER OF PUBLISHED REPORTS

Perhaps the best way to stop the AIDS epidemic in Africa, whose population has grown during the AIDS era more than the population size of the entire United States during the past 30 years, would be to immediately call a halt to the repeated implementations of interventions of the AIDS establishment, and provide Africans with the following information regarding “HIV”/AIDS?

Currently, most of the \$9.8 billion a year of U.S. tax dollars of the promised 50 billions presently going to Africa via George Bush's PEPFAR program (President's Emergency Plan For AIDS Relief), supports AIDS “activists” like TAC (the Treatment Action Campaign) of South Africa, “HIV-testing” campaigns, circumcision trials, microbicide trials that smear toxic drugs on African's genitals to increase the rate of “HIV-transmission,” breast feeding dissuasion counseling coupled with the dumping of Nestlé formulas on the third world, and of course, a lion's share of that money is earmarked for anti-viral roll-outs of deadly black-box label pharmaceutical drugs and other interventions. These are the same drugs that have been causing the mortality of Americans and Europeans during the last 15 years, year after year, with no solution in sight staying the present course. But as the global “heterosexual AIDS epidemic” came to an end, according to Kevin de Cock, except of course in Africa where some of the heaviest drugging and propaganda is occurring still, it becomes clear that the most promising strategy therefore, to end the AIDS epidemic even in Africa perhaps, might be to simply stop sending the African population all these interventions, and begin sending them a few boxes of publications to read?

But these facts have not deterred PEPFAR-in fact they have provided it's justification. Instead of critical examination of evidence, policies based on biblical rather than scientific evidence have been repeatedly advanced. Celebrated researchers such as Dr. Robert Bailey of the University of Illinois, Chicago, continue to be given lavish praise and funding for now promoting such ideas that all

African males should be circumcised to prevent the spread of the dreaded “virus,” “HIV.” It is beyond belief and human reason that these pogroms based on these bizarre interpretations of reality continue to claim that Egyptian and Hebrew biblical practices such as circumcision have won out over pharmaceutical technologies, vaccines, microbicide campaigns, and breast feeding dissuasion campaigns designed to diminish Mother-To-Child Transmission of “HIV” (each of which including circumcision have been halted by various oversight and human protection committees due to their tendency to **increase** rather than **decrease** “HIV” incidence in these various African lab rat test populations), or because of their “futility.” But a new AIDS ambassador under President Obama’s Administration, Mr. Crowley, might well ask if it is true that biblical approaches such as circumcision really reduces “HIV” incidence of African men in STD clinics typically presenting with multiple STD’s and genital ulcers simply because AZT, HAART, microbicides, vaccines, breast feeding dissuasion campaigns, nevirapine, condom crusades, and microbicides simply haven’t worked, and bizarrely, have increased the level of both positive testing results and morbidity in African “lab rats” before they were halted? Here are a few final examples of those interventions that, if stopped or defunded, would probably end the African AIDS epidemic immediately, as it is clear that Africans might surely perish if these and other interventions continue. And if these interventions aren’t halted, “the AIDS viruses” that Africans had initially acquired from their “close associations” with monkeys, chimps, or gorillas, could again spring forth again to infect the entire planet. To conclude this terrible chapter, let us review the successes of a few of these interventions.

CIRCUMCISION REDUCES “HIV” TRANSMISSION BY 50%, AND WAS HALTED FOR ITS SUCCESS AND “FUTILITY.”

On December 14, 2006, a New York Times editorial article appeared, entitled, “Rare Good News About AIDS”: *The announcement yesterday about the results in two African studies of male circumcision **may be** the most important development in AIDS research **since the debut** of antiretroviral drugs more than a decade ago. The National Institutes of Health **halted** studies in Uganda and Kenya when it became **overwhelmingly clear** that circumcision significantly reduces men’s chances of **catching** H.I.V.*

*A Kenyan AIDS trial was interrupted because **a 53 percent reduction** in acquisition of “HIV” among circumcised men was observed. Out of 2,784 men studied in the trial, 69 men were “HIV” positive: 22 of these were circumcised, and 47 uncircumcised. **Many, if not all 65 of them** had received prior (or concurrent) treatment **for penile infections**, and 28 of the 69 **had detectable serologic syphilis** at the outset. A year before, it was claimed that a trial of **4,996 HIV-negative men** in Rakai, Uganda, showed that HIV acquisition was reduced by **48 percent** in circumcised men. **Yet**, other prior studies regarding the ability of circumcision to protect against “HIV” acquisition in both males and females yielded conflicting data regarding the ability of circumcision to block acquisition of “HIV,” and the role that other STD’s or medical conditions play as cofactors in acquiring “HIV.”*

Although not spelled out here in this hopeful report, uncertainties existed regarding this hopeful report because: data had been acquired at STD clinics or from trial participants with genital ulcer disease (GUD) or other infections, and the relative roles (if any) of biological versus cultural practices that influence “HIV” acquisition were challenged by the WHO. Uncertainties regarding **the damage done by microbicides** also existed at that time, **which** apparently **increased the frequency** of reported genital lesions and the **fearedspread** of “HIV.” The ability or inability to neutralize “HIV” by washing with mild or concentrated detergents was **in question**, and the transmission of “HIV” from human to human by providing evidence of seroconversion **was yet to be provided in a form that constitutes as careful a study as the 10 year study that followed 175 serodiscordant couples for 10 years that found no (zero) seroconversions in “HIV”-serodiscordant couples** (see below).

Yet on July 16, 2009 (HealthDay News) – it was reported that:

“Circumcision doesn’t reduce transmission of HIV from infected men to women, says a study that included 922 HIV-infected men in Uganda.”

The men, who were circumcised before the start of the study, were randomly selected to undergo immediate circumcision or circumcision after two years. The study also included HIV-uninfected female partners of the men. The women were checked for HIV infection at six, 12 and 24 months.

The study was stopped early due to “futility.” The final analysis of 92 couples in the intervention group and 67 couples in the control group showed that **18 percent** of women in the intervention group became infected with HIV, **compared with 12 percent of those in the control group.** Cumulative probability of HIV infection at 24 months was 22 percent among women in the intervention group and 13 percent among those in the control group.

While these results **weren't statistically significant**, they were sufficient to stop the study, said Dr. Maria J. Wawer of Johns Hopkins Bloomberg School of Public Health in Baltimore and colleagues. “Circumcision of HIV-infected men did not reduce HIV transmission to female partners over 24 months; longer-term effects could not be assessed. Condom use **after** male circumcision is essential for HIV prevention,” they concluded.

The findings appear in the July 18 issue of **The Lancet**. The study results shouldn't deter programs working to increase circumcision services for men at risk for HIV, wrote Dr. Jared M. Baeten of the University of Washington in Seattle and colleagues in an accompanying commentary.

Involving women in decision-making about circumcision provides an opportunity to educate men and women about the risks and benefits of circumcision and to target risk-reduction counseling efforts to serodiscordant couples, where one partner is HIV-positive and the other HIV-negative, they noted

Thus, the ancient Hebrew and Egyptian practice of circumcision probably should no longer be pushed on Africans to prevent the transmission of “HIV.”

AFTER FIRST DECADE OF HAART IT IS REPORTED THAT IMPROVEMENT IN “VIRAL LOAD” MEASUREMENTS ARE OBTAINED BUT NO IMPROVEMENT IN MORTALITY IS ACHIEVED: THERAPY WAS A SUCCESS BUT THE PATIENTS DIED.

If “HIV/AIDS” is chemotherapeutically hit hard and early as a consequence of an impassioned crusade to provide in what amounts to toxic “anti-viral” cancer chemotherapy for life instead of cycles (see any AntiRetroviral-ARV- package insert) to millions who test positive, then how do the promoters of AIDS propose to solve the problem that these drugs pose, if and when they are “rolled-out” to those who live in Kenya and elsewhere, who only have only a single cup of diluted gruel paste/day as food-liquid with which to wash down their pills? -see Christine Amanpour's July 19, 2006 documentary on CNN (www.cnn.com/2006/WORLD/africa/07/17/amanpour.africa.btsc/index.html).

Most horrifying, is the fact that universal testing for “HIV” “infection” would increase morbidity and death amongst those designated as “HIV/AIDS” patients, rather than decrease morbidity and death. For example, de Martino *et al.* concluded that children born to ZDV-treated mothers (ZVD is AZT, or the AIDS drug, Azidothymidine): “**are more likely to have a rapid course of HIV-1 infection compared with children born to untreated mothers, as disease progression and immunological deterioration are significantly more rapid and the risk of death is actually increased during the first 3 years of life**”[de Martino et al., Rapid disease progression in HIV-1 perinatally infected children born to mothers receiving zidovudine monotherapy during pregnancy. AIDS. 13(8):927-933, May 28, 1999. The Italian Register for HIV Infection in Children AIDS, 13:927-933, 1999].

In the journal Pediatrics, Antoni Noguera et al reported that: “Almost **half** of the children (63 of 127) who were exposed to nucleoside analogues developed benign and self-limited hyperlactatemia when symptomatic, nucleoside analogue-induced

*toxicity affected **neurologic development***” [Antoni Noguera et al. Pediatrics, Vol. 114 No. 5 November, pp 598-603, 2004].

In 1992, The Veterans Affairs Co-operative Study Group reported that AZT disproportionately harmed Blacks and Hispanics, and provided no benefit to the quelling of advancing immune suppression in Caucasians, and harmed healthier subjects (early treated) more than persons considered to exhibit clinical symptoms of AIDS [JD Hamilton et. al. and the Veterans Affairs Cooperative Study Group. A controlled trial of early versus late treatment with zidovudine in symptomatic human immunodeficiency virus infection.” New England Journal of Medicine, 326: 437-434, 1992].

The Concorde trial, which was published without endorsement by Burroughs Wellcome’s Coordinating Committee who declined to endorse the final report, and which was the largest, longest, and best controlled adult AZT trial concluded:

*“The results of Concorde **do not** encourage the early use of zidovudine in symptom-free HIV-infected adults. They also call into question the uncritical **use of CD4 cell counts as a surrogate endpoint** for assessment of benefit from long-term antiretroviral therapy”* [Seligmann et al., Concorde: MRC/ANRS randomised double-blind controlled trial of immediate and deferred zidovudine in symptom-free HIV infection. Concorde Coordinating Committee. Lancet, Apr 9;343(8902):871-81, 1994].

Perhaps this sad legacy of drugging experiments on human subjects is nowhere made so obvious as in the report issued after the first decade of HAART, where it was claimed that improvements in “viral load” measurements were obtained but there were NO improvements in mortality:

Methods: We analyzed data from **22,217 treatment-naïve HIV-1-infected** adults who had started HAART and were followed in one of **12** cohort studies. The probability of reaching 500 or less HIV-1 RNA copies per mL by 6 months, and the change in CD4 cell counts, were analyzed for patients starting HAART in 1995-96, 1997, 1998, 1999, 2000, 2001, and 2002-03. The primary endpoints were the hazard ratios for AIDS and for death from all causes in the first year of HAART, which were estimated using Cox regression.

Interpretation: Virological response after starting HAART **improved** over calendar years, **but such improvement has not translated into a decrease in mortality.** [The Antiretroviral Therapy (ART) cohort Collaboration-www.thelancet.com Vol 368, 451-58, August 5, 2006].

PEDIATRICIANS SUGGEST FORCED COMPLIANCE USING GASTROSTOMY TUBES IN INFANTS TO FORCE THEM TO TAKE AS MANY AS 7 BLACK-BOX LABEL DRUGS:

2007, February. A report from American Academy for Pediatrics publishes:

“Gastrostomy Tube Insertion for Improvement of Adherence to Highly Active Antiretroviral Therapy in Pediatric Patients With Human Immune Deficiency Virus.”

If it is a child of a parent who “is infected” (despite a 60% sero-**reversion** rate after 9-18 months after birth), the parents can be held for criminal negligence unless they submit to having the children drugged to the extent that neurological demise develops, or drug-induced diseases occur, as in the case of the more than 300 orphans drugged through **g-tubes** with 7 black box label drugs at New York’s Incarnation Children’s Center (ICC), and elsewhere (Chicago’s Northwestern Hospital), or, when many of them are taken away from the natural parents or legal guardians to be made compliant with the drug regimens, after which only about 80 deaths were recently admitted in one trial (at ICC). To force this compliance if the children don’t like their drugs, then pediatricians have discovered an effective means of forcing compliance through inserting g-tubes in these recalcitrant children. Mothers and infants who are “HIV-positive” are dissuaded from bonding during the time of birth, and parents are brow beaten and threatened by The State in many cases to force their infant to imbibe black-box-label drugs through forced administration or through surgically-

implanted g-tubes with drugs that have been shown to be able to induce brain abnormalities, liver failure, stunted growth, and death. Breast feeding is forbidden! To do otherwise is, irresponsible. Inserting these g-tubes in Africans is too expensive though, so it probably doesn't represent too much of a threatening intervention for them at this time.

THE SUCCESSES OF BREAST FEEDING DISSUASION COUNSELING TO PREVENT “HIV” TRANSMISSION, POGROMS OF FORMULA DUMPING ON THIRD WORLD NATIONS, AND 532 DEAD CHILDREN

If stopping the HAART or nevirapine drugging by Max Essex and his group at Harvard, or if stopping AIDS research chiefs such as Ed Tremont and others from fudging of safety reports doesn't work to end the African “AIDS epidemic,” and if g-tubes inserted into infant and children's stomachs don't seem practical, then, as mentioned, there is another promising intervention touted as one of the most successful interventions to date, in both developing and developed nations, that if stopped, would decisively help end the AIDS era even in Africa. This intervention involves dissuading African women from breast-feeding their infants to avoid passing along the deadly AIDS virus through their breast milk, through the promotion of dumping mass quantities of Nestlé infant milk formulas on the third world:

African women who were dissuaded from breast feeding resulted in a 20 fold increase in infant mortality compared to breast-fed controls as it was reported late last year?

On Monday, July 23, 2007, in Nkange, Botswana, it was reported that in Botswana, step to cut AIDS proves a formula for disaster (Craig Timberg Washington Post Foreign Service):

“Doctors noticed two troubling things about the limp, sunken-eyed children who flooded pediatric wards across Botswana during the rainy season in early 2006: They were dying from diarrhea, a malady that is rarely fatal here. And few of their mothers were breast-feeding, a practice once all but universal.”

“After the outbreak was over and at least 532 children had died – 20 times the usual toll for diarrhea – a team of U.S. investigators solved the terrible riddle.”

“A decade-long, global push to provide infant formula to mothers with the AIDS virus had backfired in Botswana, leaving children more vulnerable to other, more immediately lethal diseases, the U.S. team found after investigating the outbreak at the request of Botswana's government.”

HETEROSEXUAL TRANSMISSION OF “HIV” IN NORTHERN CALIFORNIA: RESULTS FROM A TEN YEAR STUDY SUGGEST “HIV” ISN'T SEXUALLY TRANSMITTED

Sex and “HIV,” has always been the major problem during the era of AIDS. Shouldn't Africans therefore be informed about the scientific evidence regarding the sexual transmission of “HIV” by providing them with the following kinds of reports?

*We studied 50 sexually active couples with discordant antibody results [one positive, one negative]...seronegative partners continued to have **negative results in all tests** for a mean follow-up period of 17 months despite ongoing sexual relations with their seropositive partners....approximately one-half of each group reported some instances of unprotected intercourse...intercourse with outside partners was uncommon in both groups, as was current illicit drug use. (Clin Infectious Disease. July, 1995; 211).*

In April 1996 another study in *Nature Medicine* focused on 24 hetero- and homosexual men who've remained HIV negative despite “histories of multiple high-risk sexual exposures to HIV-1,” including “sex with multiple HIV-1-infected partners,” or “long-term

relationships involving unprotected sexual intercourse over many years [with] predominantly a single HIV-infected partner.” **“All subjects were HIV-1 negative,”** even though “several [of their] partners succumbed to AIDS.” (Nature Medicine. 1996 2(4)).

If these smaller non-“HIV-transmission studies don’t convince the Africans that it is OK to have sex and continue to grow their combined population numbers in 25 years by more than the population of the U.S., then perhaps Nancy Padian (mentioned before) might be summoned to go to Africa to assume her role of the world’s best AIDS-prevention counselor in the African context because of her extraordinary counseling skills? She perhaps could reduce the population just by talking to them. This suggestion is a cogent one, because, in 1997, the success of her published study of 175 sero-discordant pairs she followed over a 10-year period in Northern California became legendary amongst the promoters of AIDS. In this study, Dr. Padian reported that even though condom’s weren’t used by 25% of her 175 sero-discordant couples, 0% transmission occurred. Dr. Padian also reported that **“no seroconversions”** were observed among 175 HIV-discordant couples (where one partner tests “HIV” positive, one “HIV” negative), for a total of approximately 282 couple-years of follow up in that 10- year study [Padian, et al. Heterosexual Transmission of HIV in Northern California: Results from a Ten-Year Study.” American Journal of Epidemiology. August, 1997]. As mentioned, and which should be strongly emphasized here, the reason for no conversions described by Padian et al. to explain why “HIV” was not transmitted through sex by anyone during her 10 year study, was because the people in her study were instructed as to how to have sex and how not to have sex with each other, and despite non-use of condoms by 25% of her subjects, she repeatedly reinforced the “HIV-prevention wisdom, that if people would only listen, and behave properly, there wouldn’t be an AIDS pandemic in Africa or anywhere else.

PART II: HPV TESTING, GARDASIL, CERVARIX, AND THE SACRIFICE OF THE VIRGINS

In a front page Chicago Tribune story printed September 18, 2005, by T. M. Phung, and in a front page May 18, 2006, by J. Graham Chicago Tribune Story, we see how The Media has become big pharma’s biggest ad spinners, as Merck is given a free and unchallenged platform to tout the wonders of “GARDASIL,” (cleverly named I think), where they claim that this new vaccine against human papillomavirus, or “HPV,” was 100 percent effective in preventing precancerous cervical disease, but only when given to women and girls who had never engaged in sex at the time of the shots.”

How good is the science behind such claims that vaccines against viruses can prevent pre-cancerous cervical disease in the case of “HPV”? Unfortunately, instead of science or evidence, moralistic debates, fear, and authoritative opinions, simplifications, and distortions are routinely presented as fact through media campaigns regarding the safety and efficacy of vaccines.

In these Tribune articles, there are numerous distortions, inaccuracies, unqualified and unreferenced statements from Public Health Officials, a clinical pediatrician/ psychologist, moralistic and conservative Christian religious groups who oppose sex such as the Abstinence Clearinghouse, pharmaceutical drug-makers such as Merck and GlaxoSmithKline, a mom and her 3 children, the CDC, The American Cancer Society, The Illinois Department of Public Health, and NIAIDS. It is even advanced in the article that young children should at some point be asked about what they believe (regarding “HPV” and cervical cancer) in the context of their future sexual behavior:

“Julietta Bolivar of Little Village, a mother of three adolescents—ages 10, 12, and 15—said she does not know how she feels about the vaccines. I would have to think about it,” Bolivar said. “I want to learn about it before I make a decision. I guess I would have to talk to my doctor about it first, then talk to my kids too and hear what they think.

Aside from the possible future opinions of Mrs. Bolivar’s children about Merck’s new vaccine (which would probably be negative ones-what 10-year old do you know wants to go to the doctor to get a shot?), or the imagined association between “HPV” and cervical cancer, not a single reference is provided to validate the wild claim advanced by Merck that “a wave of experimental vaccines against sexually-transmitted diseases could revolutionize the prevention of such infections in the next few years.” Instead, what is advanced

is a threat: “but there is a catch: the shots will likely work best when administered to (our) children” (*my emphasis*).

In the May 18th, 2006 J. Graham article, there are also numerous claims advanced that are not supported by any evidence (I am not being inclusive here regarding all the flaws, lies, and distortions that appear in this story: I am only presenting the most egregious) that claim that:

1) HPV vaccine prevents cervical cancer—no evidence.

2) HPV causes cervical cancer—no evidence.

3) HPV is the first cancer vaccine—what about hepatitis B and hepatocellular carcinoma? Merck told us 20 years ago that that the hepatitis B vaccine was the first cancer-preventative vaccine, and the Taiwan, and Korean studies are often provided to support that the vaccine reduced cancer in these countries. Can’t these folks remember the history of their own vaccinology for 20 years prior to the present?

4) *“The unanimous recommendation inspired cheers from the medical community, which said the vaccine could help save the lives of tens of thousands of women worldwide.”*

But you don’t hear the parents of vaccine damaged children cheering (as many as 10% are severely damaged by the hepatitis B vaccine according to Merck’s own package insert-which in many cases is being co-administered along with GARDASIL-see VAERS reports toward the end of this article). Many of our colleagues who have worked directly with “HPV” diagnosis and treatment also aren’t cheering, because they diagnose and treat cervical cancers and they know that the molecular HPV tests have not been validated, nor have the cell based smears been elucidated (why would the ASCUS test mean Atypical Squamous Cell Carcinoma of UNDETERMINED SIGNIFICANCE if this were not the case)?

Certainly, the US’s chief and most respected workers in HPV research aren’t cheering because they stated in a CAP Today article (College of American Pathologists Today) there are grave medical concerns even regarding the validity of HPV test kits.

Moreover, we worked with HPV in my laboratory, and we were horrified at such a dangerous suggestion-the implementation of this vaccine will place the lives of millions of our daughters at risk. Where are the long-term safety data? Will they be conveniently lost like the hepatitis B data that we asked the CDC to show us several years ago?

5) *“...this is a watershed event ... that we hope will help usher in a new era of cancer prevention,”*

It is a “watershed event” designed to reap untold profits at almost \$900 dollars for the series at the expense of our daughter’s (and possibly son’s health).

6) *“Haupt doesn’t expect women to be tested routinely for HPV before being inoculated, in part because commercial tests can’t pinpoint which HPV strain a woman has.”*

How can any scientific study on vaccine efficacy be conducted without an accurate test to “pinpoint” the presence of a suspected pathogen?

7) Documents prepared by the FDA suggest some women with persistent HPV infections could be at higher risk of cervical cancer after taking the vaccine.

How could this be science based upon the immunology that we learned in school, or anything resembling science? Why should some women be at greater risk if they receive the vaccine? Is the HPV vaccine, GARDASIL, like the SV40-contaminated polio vaccines that increased the polio rate 3 fold in California, and 15 fold in Idaho, and hundreds of folds in certain outbreaks over normal summer background incidences of polio in the US weeks after the first vaccine wave was given? Does it increase disease like MMR and pertussis vaccines have, or is it due to the possibility that the HPV vaccine contains poisons, toxins, other viruses, or non-disclosed immune-disease-stimulating adjuvants (such as squalene) that cause autoimmune disease such as lupus, demyelinating syndromes, arthritis, or even cancer in animals?

What leading pathologists are saying about the molecular HPV kits and their validation as cancer diagnostics

Both of these Chicago Tribune articles differ considerably from a front-page statement and 5 page article published in the September 2005 issue of Pathology/Laboratory Medicine/ and Laboratory Management article released monthly by The Collage of American Pathologists (CAP). Instead of asking Merck, The Public Health Service, or Moms and their children what they think about the merits of the new Gardasil “HPV” vaccine, a highly pointed and critical 5-page article was advanced regarding the uncertainties of “HPV” testing. A few examples from the article make the point:

“Dr. Schiffman heads the HPV Troup in the Division of Cancer, Epidemiology, and Genetics at NCI and is a tenured senior investigator. In mid March, Dr. Mark Schiffman, MD, MPH, called CAP TODAY’s editor to voice a troubling concern: that laboratories are failing to clinically validate their HPV tests.”

In two subsequent interviews with CAP TODAY, Dr. Schiffman says labs are stumbling badly. His case is straightforward. Laboratories that use HPV tests need to make sure those tests are clinically validated. *“It’s amazing to me that someone would sell a product that’s influencing a patient’s life in terms of treatment for cervical cancer without being sure, based on data, that they can do it again and again and again with reliability.”*

Mark Stoler, M.D., professor of pathology and clinical gynecology and associate director of surgical pathology and cytopathology, University of Virginia Health System, Charlottesville, says the problem is a major concern, not ‘some’ concern. It’s beyond anecdotal.”

“I certainly see-in the chat areas of the different organizations, at conferences, on the Internet-advertisements and statements that are troubling, because they’re indicating an excessive faith in poorly validated assays.”

In the CAP TODAY article, Dr. Stoler also pointedly asks, “The essential question for ASCUS triage (atypical squamous cells of undetermined significance), is what is the sensitivity of the HPV test, and therefore its negative predictive value, in patients who have equivocal cytology for high-grade lesions? Many physicians, however, focus instead on the positive predictive value of the test, that is, the likelihood of finding high-grade lesions with colposcopy.”

“The problem,” he says, “is colposcopy is a terrible gold standard, missing anywhere from one-third to one-half of high-grade disease.” “Lots and lots of labs say, ‘We think the PCR test is more sensitive because we can pick up fewer DNA copies.’ That has nothing to do with what we’re talking about,” says Dr. Stoler. “If you’re going to bring forward a test, you’ve got to do a clinical validation trial that establishes its performance relative to these other benchmarks,” he continues. “And the standard is not analytical molecules of DNA. It’s not the analytic validation that matters, it’s the clinical validation-how does the test perform in the real world? How sensitive are you with finding high-grade disease in a population of minimally abnormal cytology patients?”

These are all very good questions and warnings. However, when the reference labs throughout the world, and the doctors who run them are asking what the tests mean *“in the real world,”* it should cause some pause, and certainly caution with respect to the

certainty with which HPV and cervical cancer and a potential vaccine against HPV and cervical cancer may have been worked out by Merck, and broadcast in the Chicago Tribune and L.A. Times with statements like: “...*this new vaccine against human papillomavirus, or HPV, was 100 percent effective in preventing precancerous cervical disease, but only when given to women and girls who had never engaged in sex at the time of the shots.*”

Even Attila Lorincz, PhD, chief scientific officer and senior VP of research development at Digene (one of the principal HPV test-kit makers) says that:

“much of the confusion simply boils down to analytical and clinical accuracy is not well enough understood or described by people who write or talk about it,” and that “the problem surfaces in the HPV literature with distressing regularity.”

Toward the end of the CAP TODAY article, Dr. Shiffman again is quoted as saying that:

*“What surprises me is that this {the certainty with which these tests for HPV and cervical cancer} could in any way be controversial, he says. “The issue is not so much controversial, of course, as it is **loaded-with money and competitive claims, scientific complexity, and grave medical concerns.**”*

If what Dr. Shiffman, a world expert on “HPV,” is describing the state of the art in “HPV” testing, then how could anyone suggest that: “...*this new vaccine against human papillomavirus, or HPV, was 100 percent effective in preventing precancerous cervical disease, but only when given to women and girls who had never engaged in sex at the time of the shots,*” unless madness about making money, rather than sound public health policy, were behind it?

THE ILLINOIS DEPARTMENT OF PUBLIC HEALTH VERUS THE PARENT TEACHERS ASSOCIATION OF ILLINOIS

A few years ago, small group of physicians and scientists gained the support of the Illinois PTA in a unanimous decision to support a halt to the current mandated hepatitis B vaccine. Another way of saying this is that every school representative present at the convention, when shown the data we had obtained, had agreed with our concerns, and immediately held a brief session to advance a motion to direct PTA funding to disseminate literature so that parents would be informed.

This group of perhaps a thousand parents (mostly women), appeared to have only one concern: the total welfare, protection, and education of the school children of Illinois. It should be stated emphatically, that the current hepatitis B mandate threatens not only our children’s health, but also serves to threaten our children’s education and admission to all kinds of institutions (day care and school admission), with the bluff that you don’t get your kid vaccinated against this STD, that is detected only in subpopulations of injection drug users and perhaps highly promiscuous persons, as well as in healthy black Australian aboriginal men, Micronesians, Vietnamese, Taiwanese, Native Americans, patients with Down syndrome, leukemia and transfusion recipients, he or she cannot enter school to learn how to read and write.

This is not overstating it. Children cannot gain admission into day-care, Kindergarten, elementary schools, junior highs, high schools, and now even colleges, without showing evidence of a mandated (federally-recommended), and dangerous vaccine (hepatitis B).

Pursuing these issues, we presented the current head of the IDPH (Illinois Department of Public Health) Director Whitaker and his staff, with the same publicly available data from Medline, the vaccine manufacturer’s package insert warnings, data from the Vaccine Adverse Events reporting System, the CDC, Vaccine-link, and other databases, that we had presented to the Illinois PTA convention. After visits with numerous Senators, and public officials during the past several years, over a year later, in June of 2005, we finally

were granted a brief meeting with the IDPH, after they could put us off no longer.

As a response to our pleas to institute informed consent regarding the dangers of the hepatitis B vaccine's side effects and safety record as it appears on the Federal governments VAERS database, and after many weeks of deliberation, Dr. Whitaker and his staff emailed us a one paragraph letter stating:

“Parents are currently given enough informed consent.”

Well, one may ask Dr. Whitaker, “how do threats that our children won't be admitted to school unless they are jabbed with the hepatitis B vaccine (a rare syndrome) and whose safety data we have yet to see, constitute, informed consent?”

Shouldn't parents at least be given a list of the adverse syndromes induced by the vaccines that are presented on the manufacturer's package inserts, as shown above on Merck's insert? Should parents be shown the VAERS data? Should a list of the hundred or so articles on Medline regarding adverse syndromes induced immediately after vaccination, by mostly private physicians? Shouldn't parents be informed that the data supposedly supporting the safety of the hepatitis B vaccine in neonates doesn't exist (Lewis E, Shinefield HR, Woodruff BA, Black SB, Destefano F, Chen RT, Ensor R; Vaccine Safety Datalink Workgroup. Safety of neonatal hepatitis B vaccine administration. *Pediatr Infect Dis J.* Nov;20(11):1049-54, 2001; Also, Testimony of Dr. Marc Geier at IOM hearing, Aug. 2004).

Somebody should tell the public, as we have tried to warn for the past several years, that parents have the right to refuse all vaccines or medical treatments on their children's behalf, with the aid of a publicly-available form on which either religious or philosophical objection to these experimental medical interventions can be declared. The school nurse and Public Health Department, or school admittance policies should not be used to threaten you that you cannot enroll your kid, based on the madness surrounding the possibility that your 5-year-old will transmit a sexual, or needle-borne, or blood-product-transmitted “syndrome” that has a 95% or greater spontaneous resolution rate, to someone else's 5 year old, (when they have sex or shoot heroin in the gym locker-room, or if they share razor blades-are the reasons typically given to support mandatory hepatitis B vaccination) as the pharmaceutical company and Public Health Service logic goes.

These same kinds of threats, mandates, and fear-mongering are already beginning to occur within the arena of GARDASIL and the undemonstrated link between the HPV molecular sequences and cervical cancer.

We beg the Public Health Service in the case of both hepatitis B and “HPV” to regard your own children as potentially at risk for becoming sexually promiscuous so they won't contract a rare disease that poses almost 0 risk, that will resolve without treatment in most cases, that simply presents as an ill-defined molecular sequence, or presents as harmless molecular signatures, or that may represent immunological stress or a simple genetic polymorphism. Please leave our infants and children alone.

Why in the face of all this damning evidence against the vaccine, does the hepatitis B vaccine mandate still stand with no end in sight? It is because new legislation has insured that there is no incentive, compensation laws, or mechanisms in place anymore to guard against dangerous universally mandated experiments.

THE FUTURE IS HERE: MEDICAL TERRORISM INTO LAW

From the “Biodefense and Pandemic and Vaccine and Drug Development Act of 2005”—a bill to amend the Public Health Service Act to enhance biodefense and pandemic preparedness activities, and for other purposes, SEC. 319F-3:

“(a) Authority- As provided in subsection (b), and subject to subsection (b)(1)(C), a manufacturer, distributor [sic; distributor], or

administrator of a security countermeasure, or a qualified pandemic and epidemic product, described in subsection (b)(1)(A) or a health care provider **shall be immune from suit or liability** caused by or arising out of the design, development, clinical testing and investigation, manufacture, labeling, distribution, sale, purchase, donation, dispensing, prescribing, administration, or use of a security countermeasure, or a qualified pandemic and epidemic product, described in subsection (b)(1)(A).”

Further, subsection (b)(1)(A)(i) reads:

“(i) IN GENERAL- No cause of action shall exist against a person described in subsection (a) for claims for loss of property, personal injury, or death arising out of, reasonably relating to, or resulting from the design, development, clinical testing and investigation, manufacture, labeling, distribution, sale, purchase, donation, dispensing, prescribing, administration, or use of a security countermeasure or qualified pandemic or epidemic product distributed, sold, purchased, donated, dispensed, prescribed, administered, or used in anticipation of and preparation for, in defense against, or in response to, or recovery from an actual or potential public health emergency that is a designated security countermeasure or a qualified pandemic or epidemic product by the Secretary in a declaration described in paragraph (2).”

What is being described here is almost carte-blanche freedom to use untested vaccines, drugs, medical products, or “security countermeasures”. And there is nothing you, or we, can do about it because it is in the interest of “National Security.” The costs of these giant Nuremberg-violating programs to insurers and to our society are catastrophic. We have met “slightly damaged” vaccine-damaged kids whose parents have spent more than 1.2 million dollars for several years of care.

In the modern scientific culture of molecular cancer research, the standards and experiments used to demonstrate such phenomena have radically changed from the classic methods of Rous, Shope, Beard, Gross, Eddy, and many others, as faith in molecular biology and molecular markers has grown. In addition, more and more scientists are beginning to accept the growing body of published evidence that the non-living matrix material outside of living cells principally controls cancer growth or its metastasis, and that in general, genes, viruses, or transformed cancer cells themselves are insufficient to cause or even promote cancers (1-6). Also, because the concept of “cofactors” in cancer induction has assumed such importance historically and in recent years, the link between any viruses and the induction of human cancers remains speculative at best. A few of these co-factors in the context of virus-associated cancers include, but are not limited to insufficiently cooked red meat but oddly enough not “smoked white-chicken.” Other cofactors that appear to always be said to play a role are alcohol consumption, smoking, social mobility or lack of it, and various vaccines, such as smallpox vaccine-induced tumors at the site of vaccination on human arms that generate basal carcinomas, or the 160,000 cats/year that acquire malignant cancers at their vaccination sites, according to the American Veterinary Medical Association and Feline Sarcoma society. These and other cofactors in fact were cited as recently as 2008 in a Nobel Prize acceptance speech by Harold Zur Hausen, arguably the world’s principal exponent of HPV’s role in cancer. During his Nobel speech, Zur Hausen’s admonishments and warnings about cofactors are cogent because Zur Hausen is credited for the claimed cancer-promoting ability of certain HPV DNA sequences, as he characterized what he believed to be the molecular mechanism to explain the destructive power of such DNA sequences (<http://nobelprize.org/mediaplayer/index.php?id=1052>). During the last minute of his 45 minute speech, Zur Hausen details as a speculative idea how his “virus” might cause cancer, and again, it is like reading Rous’s speech, or watching Luc Montagnier’s interview cited earlier. As I mention above, and the link between vaccination and cancer in both livestock and humans is covered at 25 minutes into his talk). More cogent reasons than persistent viral infectious causing cancers in humans are presented in **Appendix 3**).

HUMAN PAPILLOMA VIRUS VACCINE SAFETY INFORMATION IN 2007: ANALYSIS OF VACCINE ADVERSE EVENTS REPORTING SYSTEM REPORTS: ADVERSE REACTIONS, CONCERNS, AND IMPLICATIONS

(March) <http://www.909shot.com/Diseases/HPV/HPVrpt.htm>

This information is divided into three sections. The first section describes reaction reports for a number of reported adverse events: neurological symptoms including syncopal episodes and seizures, arthralgia and joint pain, Guillian-Barre Syndrome, and other immunological reactions. The second section addresses concerns related to vaccinating individuals already infected with HPV. The last section discusses issues that need to be addressed by government regulators and the manufacturer and considerations for clinicians and consumers.

On June 8th 2006, the Food and Drug Administration (FDA) announced the approval of GARDASIL, and on June 29th the Advisory Committee on Immunizations Practices (ACIP) voted to recommend adding GARDASIL human papilloma virus vaccine to the Centers for Disease Control's national childhood recommended immunization schedule.

On July 14th the first report of a serious reaction to the vaccine was filed with the federal Vaccine Adverse Event Reporting System (VAERS). A 16-year-old Illinois girl was vaccinated July 7th and 13 days later developed symptoms eventually diagnosed as Guillian-Barre Syndrome. A 14-year-old girl in the District of Columbia was vaccinated on July 11th and complained of severe pain immediately following the injection, fell off the examining table and experienced a 10 to 15 second fainting spell ending up in the emergency room with a headache and speech problems. The report of this reaction, the first in the nation, was filed on July 14th, 15 days after the ACIP vote. Six months later, 82 reports of GARDASIL reactions have been submitted to VAERS on behalf of at least 84 young girls and 2 boys. Reaction reports have come in from 21 states and the District of Columbia.[2] Reactions were reported for children and young adults ranging in age from 11 to 27. Of the reports indicating what day the vaccine was given and the reaction occurred, 63 percent stated that the reaction occurred the same day the vaccine was given. All but three of the reports were for reactions that occurred within one week of vaccination.

Reported Adverse Events:

Presumably, the reactions described below occurred after the first dose of GARDASIL. GARDASIL is given in a three-dose series. None of the reports stated that the children and adults experiencing problems had previously been vaccinated with GARDASIL.

Syncopal Episodes and Seizures. One-quarter of all reports filed after GARDASIL vaccination were for neurologic adverse events including loss of consciousness, syncope, syncopal events and seizures. An additional five reports included symptoms of dizziness and feeling faint.

Syncope is defined as a temporary suspension of consciousness due to generalized cerebral ischemia (inadequate blood flow and lack of oxygen). The reports of syncopal episodes and their descriptions are remarkable. A physician from Washington State reported that in one morning, three patients experienced syncopal episodes. On August 8th another physician's office reported that two patients experienced syncopal episodes on the same day.

Although these reports did not detail what happened to the individuals experiencing these syncopal episodes, other reports did. The 14-year-old DC girl mentioned earlier experienced a syncopal episode combined with amblyopia (poor vision in one eye), abnormal speech, vomiting, and headache. Also experiencing vision problems, a 17-year-old New York girl reported feeling dizzy and her vision went "black for a few seconds" and she turned pale and lips turned purple and she also had fever and chills. Similar to the DC girl, on July 18th immediately after being vaccinated, a 22-year-old Kentucky woman experienced slurred speech accompanied by pallor and shock. On August 29th, two hours after being vaccinated, a 15-year-old New York girl who had a history of asthma and was on four asthma medications experienced difficulty swallowing prompting a visit to the emergency room. On August 17th, 15 minutes after being vaccinated, a 14-year-old Pennsylvania girl passed out in the car on the way home.

Most of the reports do not describe what happened as a result of the syncopal episode but a few do. One 11-year-old Florida girl fell from the examining table and two Washington girls fell – a 16-year-old girl fell and hit her head on a carpeted concrete surface

and a 14-year-old girl fell down and broke her nose.

Whether the 22 girls who experienced syncopal episodes actually experienced atonic seizures cannot be determined from these reports. Four girls, however, displayed observable seizure activity. The 11-year-old Florida girl who fell from the table also displayed “tonic posturing.” Tonic posturing is a type of seizure where sustained contraction of muscles in the legs and arms occurs and consciousness is impaired. The 16-year-old Washington girl who fell and hit her head on the floor lost consciousness for one minute and displayed tonic posturing of her right hand. Additionally, a 15-year-old girl from Virginia was described as having “a mild seizure.” In California, a 13-year-old girl was walking down the hall after her vaccination, fell and had a 15-second tonic/clonic seizure. Tonic/clonic seizures are also known as “grand mal” seizures.

Additionally, there were reports of dyskinesia (difficulty or distortion in performing voluntary movements) and hypokinesia (slow or diminished movement of the body musculature) both of which have neurological implications.

Arthralgia, Joint Pain and Fever. Arthralgia is defined as pain in the joints. Concerns about arthritis were raised during the GARDASIL clinical trials. Reports of arthralgia in one or more joints accompanied by fever were noted in five instances from four young girls and women in Wisconsin, Texas and New York, and one 18-year-old New York male.

Guillain-Barre Syndrome. Reports state that two recently vaccinated 16-year-old girls – one from Illinois and the other from Mississippi – were diagnosed with Guillain-Barre Syndrome (GBS) following vaccination with GARDASIL. In both cases, the onset of symptoms occurred 13 days after vaccination. According to the National Institute for Neurological Disorders and Stroke:

GBS is a serious disorder in which the body’s immune system attacks part of the peripheral nervous system. The first symptoms of this disorder include varying degrees of weakness or tingling sensations in the legs. In many instances, the weakness and abnormal sensations spread to the arms and upper body. These symptoms can increase in intensity until certain muscles cannot be used at all and, when severe, the patient is almost totally paralyzed. ... Vaccinations can trigger onset of GBS.

The Illinois girl described earlier was vaccinated on July 7th and symptoms were evident by July 20th.

The girl also experienced gait abnormalities (trouble walking properly), asthenia (weakness without loss of strength), paresthesia (burning, prickling, tingling or numbness sensation usually felt in the hands, arms, feet and legs), and hyperkinesia (abnormal increase in muscle movement). The Mississippi girl was vaccinated on July 31st and by August 13th she had increasing numbness and tingling in her feet and hands and was subsequently evaluated by a neurologist and diagnosed with GBS. The current health status of these girls is not known. In both of these cases, the girls were also vaccinated with Aventis Pasteur’s Menactra, a vaccine for meningococcal infections. Menactra has previously been associated with Guillain-Barre Syndrome, and the FDA and others have issued alerts.

Other Adverse Reactions:

Additionally, a number of other reactions to GARDASIL are noted in VAERS reports and they include: urticaria (hives); pruritus (itching); macular and papular rashes; blisters and vesicles near the injection site; swollen arms; lymphadenopathy (swollen lymph nodes); red, hot swollen knots at injection site; burning, stabbing, severe and radiating pain at the injection site and in the affected limb during and after injection; nausea and vomiting; infections and skin ulcers, and other allergic reactions.

Other Considerations:

GARDASIL is marketed as a “cervical cancer vaccine” and intended to prevent infection with specific HPVs – common viruses

among sexually active women. It isn't clear what benefits or potential harms could arise from vaccinating sexually active women who have already contracted HPV. Of the 86 reaction reports filed with VAERS so far, 12 reports were generated by young women 18 and older who were taking hormonal contraceptives and presumably sexually active.

With respect to concerns related to vaccinating women with known HPV infections, adverse reaction reports were filed on behalf of a 17-year-old Texas girl who was already diagnosed with HPV and genital warts. Similarly, the 22 year-old Kentucky woman who experienced slurred speech following vaccination already had an abnormal pap smear with evidence of cervical dysplasia.

Implications:

The early reports of potential safety problems with GARDASIL raise concerns and questions that need to be addressed by government regulators, manufacturers and prescribing physicians. Specifically, the following concerns need to be addressed:

1. Syncope, seizures and Guillain-Barre Syndrome have now been reported in hours to a week after GARDASIL vaccination. GARDASIL manufacturer, Merck, should add these serious adverse events to the product manufacturer insert,
2. Considering that over 20 girls have experienced syncopal episodes sometimes combined with seizures and serious injuries, physicians should consider only giving GARDASIL when the patient is safely laying down on the examining table. Because there seems to be syncopal reactions up until 15 minutes after vaccination, patients should be asked to lie down for 15 minutes after receipt of GARDASIL.
3. The information provided by Merck indicates that it is safe to administer GARDASIL with Hepatitis B vaccine. The prescribing information states, "Results for clinical studies indicate that GARDASIL may be administered concomitantly (at a separate injection site) with hepatitis B vaccine (recombinant). Co-administration of GARDASIL with other vaccines has not been studied." Due to the small number of girls aged 9 to 15 who appear to have been evaluated for GARDASIL safety in Merck clinical trials (fewer than 2,000) and lack of publicly available information about how many of these girls were given GARDASIL and hepatitis B vaccine simultaneously, the safety of administering GARDASIL and hepatitis B vaccine to all pre-adolescent girls is uncertain.
4. Aside from Hepatitis B, Merck does not state that it is safe to simultaneously administer GARDASIL with any other vaccine. Considering that there are ongoing evaluations of a reported association between Menactra (meningococcal vaccine) and Guillain-Barre Syndrome, and Merck does not explicitly indicate that it is safe to administer GARDASIL and Menactra simultaneously, consumers and clinicians should question whether administering both GARDASIL and Menactra at the same time is safe.
5. Similarly, adverse reactions were reported when GARDASIL was administered with eight other vaccines: Hepatitis A, MNQ (?), MEN (Menactra), TD (Tetanus and Diphtheria Toxoids), DPP (Diphtheria/Pertussis/Polio), PNC Prevnar (Heptavalent pneumococcal conjugate), DTaP (Diphtheria And Tetanus Toxoids and Acellular Pertussis Vaccine), and TDAP (Tetanus, Diphtheria and Pertussis). Because Merck does not state that it is safe to administer simultaneously GARDASIL with any vaccine other than Hepatitis B, consumers and clinicians should question whether co-administration of GARDASIL and other vaccines is safe.
6. Most, if not all, of the reactions reported to VAERS were in response to the first of the three doses of GARDASIL. The Centers for Disease Control (CDC) Vaccine Information Sheet (VIS) developed for HPV vaccine states that severe reactions include "any unusual condition, such as a high fever or behavior changes. Signs of a serious allergic reaction can include difficulty breathing, hoarseness or wheezing, hives, paleness, weakness, a fast heart beat or dizziness." The CDC also states that "anyone who has ever had a life-threatening allergic reaction to yeast, to any other component of HPV vaccine, or to a previous dose of HPV vaccine should not get the vaccine." Which of the reactions reported to VAERS constitute a "life-threatening allergic reaction" and which, if

any, of the children and young adults who experienced reactions should receive additional doses of vaccine? At the October 2006 ACIP meeting, CDC staff stated that only “three serious reports were reported to VAERS after HPV vaccination in females 14 and 16 years of age. One of these patients had vasovagal syncope and was hospitalized overnight for observation.” [7]CDC’s summary of the first 76 VAERS reports suggests that CDC doesn’t regard the remaining reports as “serious.” CDC needs to clarify which of the reactions reported to VAERS constitute contraindications to further vaccination with GARDASIL and make this information available to the public and to prescribing physicians

7. What were the short and longer-term outcomes for the individuals who experienced the reactions reported to VAERS? Is there information available that would help to predict the characteristics that predispose one to be at greatest risk of experiencing a serious reaction?

8. The CDC’s Vaccine Information Sheet indicates that allergy to yeast is a reason to avoid taking GARDASIL. Merck notes that contraindications to the vaccine include “hypersensitivity to the active substances or to any of the recipients of the vaccine. Individuals who develop symptoms indicative of hypersensitivity after receiving a dose of GARDASIL should not receive further doses of GARDASIL.” The prescribing information provided by Merck does not specifically note that yeast allergy is a contraindication to taking GARDASIL. Government regulators and the manufacturer need to address the discrepancy between these documents and clarify the issues related to yeast allergy and make this information readily available to the public and prescribing physicians.

9. Additionally, Merck notes that vaccine ingredients include 225 mcg of aluminum (as amorphous aluminum hydroxyphosphate sulfate adjuvant), 0.78 mg of L-histidine, 50 mcg of polysorbate 80, and 35 mcg of sodium borate. These ingredients are not listed on the CDC’s VIS sheet. The public needs this information so that they can identify whether they have “hypersensitivities” to any of the ingredients and whether they are at risk of experiencing a serious allergic reaction. Hypersensitivities and known allergic reactions are critical pieces of information that need to be communicated to prescribing physicians in order to make the safest possible vaccination decisions.

Government regulators including the CDC and FDA, in combination with Merck, should address the above safety concerns as soon as possible. Medical groups advocating use of GARDASIL should effectively communicate to physicians and patients the potential risks of using GARDASIL along with precautions to improve the safety of patient care.

ADVERSE EVENTS REPORTED BY October 6, 2007:

Another eight deaths in just the past few months are being connected to Gardasil, Merck & Co.’s vaccine that targets the sexually transmitted human papillomavirus and is being considered by many states as mandatory for all schoolgirls, according to documents released by Judicial Watch. ARTICLE_ID=58004 (c) 2007 WorldNetDaily.com

There also have been another 1,824 adverse reactions to the drug, bringing the “known total” of such problems to 3,461, according to the public interest group that investigates and prosecutes government corruption.”In light of this information, it is disturbing that state and local governments might mandate in any way this vaccine for young girls,” said Tom Fitton, the group’s president. “These adverse reactions reports suggest the vaccine not only causes serious side effects, but might even be fatal.”

WND previously has reported how Merck was lobbying state lawmakers to require the vaccination, but gave that up after its activities were unveiled.

WND also reported when a key researcher into human papillomavirus, which is targeted by Gardasil, reported it needed more testing, and how even the Centers for Disease Control suggested the vaccine should not be mandatory.

The dispute primarily has been over proposed state and other governmental requirements that schoolgirls be vaccinated against an infection transmitted only by sexual contact.

The target of the vaccine is cervical cancer, since studies show that those who have HPV have a higher chance of later developing cervical cancer. However, opponents note that such cancers develop most often in older women, while the plan is to require girls as young as 11 or 12 years old to be inoculated. They cite the lack of evidence that the vaccine would have an impact later in life.

Judicial Watch said it obtained documents from the U.S. Food and Drug Administration under the Freedom of Information Act detailing the new 1,824 cases. Those cases include as many as eight deaths related to the vaccine, on top of the three deaths reported earlier among 1,637 earlier reports of adverse effects.

Among the new information Judicial Watch found:

“Information has been received concerning a 17 year old female who in June 2007 was vaccinated with a first dose of Gardasil. During the evening of the same day, the patient was found unconscious (lifeless) by the mother. Resuscitation was performed by the emergency physician but was unsuccessful. The patient subsequently died.”

“Information has been received concerning a 12 year old female with a history of aortic and mitral valve insufficiency who on 01-MAR-2007 was vaccinated IM into the left arm with a first dose of Gardasil. On 01-MAR-2007 the patient presented to the ED with ventricular tachycardia and died.”

“Initial and follow-up information has been received from a physician concerning an ‘otherwise healthy’ 13 year old female who was vaccinated with her first and second doses of Gardasil. Subsequently, the patient experienced paralysis from the chest down, lesions of the optic nerve. At the time of the report, the patient had not recovered.”

The flood of adverse reactions during 2007 reported to the FDA through the Vaccine Adverse Event Reporting System, included 347 serious reactions. “Of the 77 women who received the vaccine while pregnant, 33 experienced side effects ranging from spontaneous abortion to fetal abnormalities. Other serious side effects continue to be reported including, paralysis, Bells Palsy, Guillain-Barre Syndrome, and seizures,” Judicial Watch said. And these numbers may not even include all the cases, Judicial Watch said.

It filed a lawsuit this week against the FDA for failing to fully respond to its requests for information involving the vaccine.

Specifically Judicial Watch wanted access to correspondence between Merck and the FDA regarding the vaccine, communications between the FDA and GlaxoSmithKline, which is working on a similar vaccine, called Cervarix, and reports by consumers, health professionals and others regarding problems with the HPV vaccine.

*When the organization’s investigation into the HPV vaccine issue arose, and the first reports started coming in, Fitton described it as **“a catalog of horrors.”***

One earlier report, No. 275438-1, describes the reaction as coronary artery thrombosis, sudden cardiac death. “Given Gardasil vaccine dose #1 3/12/07. Collapsed and died on 3/26/07

Echocardiogram revealed very enlarged right ventricle, small left ventricle as well as large blood clots within both the right atrium & right ventricle.” Another report noted that the woman was vaccinated and “died of a blood clot 8 hours after getting the Gardasil vaccine.”

Officials with the Abstinence Clearinghouse noted in a position paper that groups including the Texas Medical Association, the American Academy of Pediatrics, the Association of American Physicians and Surgeons, and the American Academy of Environmental Medicine have come out publicly against mandatory vaccination.

“The reasoning of these medical associations is clear. They are not opposed to medical progress, and certainly support all efforts to combat life-threatening diseases. The problem, as these organizations see it, lies in the fact that the drug only went through three and a half years of testing, leaving the medical community somewhat in the dark as to what serious adverse effects might result in the long term,” the group said.

“Along with the potential of serious adverse effects is the question of efficacy. There is evidence that after approximately four years, the vaccine’s potency significantly declines. The long-term value of the vaccine has yet to be determined; if it wears off within six years, will girls and women need to repeat the battery of injections they originally received?” the organization wondered.

Michigan was the first state to introduce a plan to require the vaccine to be given to young girls, but the proposal failed. Ohio also considered a failed plan in 2006. Then in 2007, after Merck’s aggressive lobbying campaign and contributions to Women in Government, lawmakers in at least 39 states and the District of Columbia worked on sponsoring such plans.

September 26, 2007

Merck Set to Donate Cancer Vaccine Doses: By LINDA A. JOHNSON

<http://www.lasvegassun.com/sunbin/stories/thrive/2007/sep/26/092603125.html>

AP Business Writer

TRENTON, N.J. (AP) – Drug maker Merck & Co. plans to donate enough doses of its cervical cancer vaccine, Gardasil, to inoculate 1 million women in some of the world’s poorest countries (46. .

Merck announced the gift Wednesday at the third annual Clinton Global Initiative conference in New York.

The Whitehouse Station-based company said at least 3 million doses of Gardasil are to be distributed over the next five years. The vaccine is given in three shots spread over six months.

Cervical cancer, caused by a sexually transmitted virus, is the No. 2 cause of cancer deaths worldwide, with nearly 500,000 new cases and 250,000 deaths each year. Most deaths occur in poor nations, where women rarely get tests to detect cervical cancer early, when it is most curable.

Merck plans to partner with a nongovernment organization to set up programs to distribute the vaccine in countries yet to be chosen. “Our company is fully committed to making Gardasil available to those who need it,” Margaret McGlynn, head of Merck Vaccines and Infections Diseases, said in a statement.

*Gardasil is **99 percent effective** in preventing infection by two strains of the human papilloma virus that together cause about 70 of cervical cancer cases. It also protects against 10 other strains that cause cancer, plus another two that cause genital warts but not cancer. In the United States, Gardasil costs about \$360 for three doses, plus any fee for a doctor visit.*

Merck has the only cervical cancer vaccine approved in the United States, but rival GlaxoSmithKline PLC's vaccine, Cervarix, was approved Monday for sale in the 27 European Union member states and is awaiting U.S. approval.

Somebody should at least let these folks know that the hepatitis B vaccine was touted as being the first anti cancer vaccine, which in fact was made by Merck, the company now claiming the HPV is the first anti-cancer vaccine. If they believe HPV causes hepatocellular carcinoma (or is associated with leukemia), and if "HIV" causes 6 cancers (except of course for Kaposi's sarcoma, the first AIDS-defining illness that isn't caused by "HIV" but by "herpes-virus"), then even in a court of law, it could be argued that GARDASIL indeed is not the first anti-cancer vaccine given (or mandated) in the U.S. It represents, perhaps, another first anti-cancer vaccine whose effectiveness will be evaluated at least 55 years from now, given the certainty and uncertainties of the Pankhurst analysis of the 35 year post polio era data where SV-40 was monitored for its ability to cause cancer in the carefully followed group that included 1067 infants injected with the SV-40-contaminated poliovaccines.

IN JUNE 2008, THE REPORTED ADVERSE EVENTS TALLY ATTRIBUTED TO GARDASIL INCREASED TO 8,864 (only less than 10% of adverse reactions are typically reported for any vaccine) (except perhaps Hepatitis B and MMR):

Death toll linked to Gardasil vaccine rises. Complications include shock, 'foaming at mouth,' convulsions, coma (Posted: June 30, 2008 10:18 pm Eastern, 2008 WorldNetDaily).

"Anaphylactic shock," "foaming at mouth," "grand mal convulsion," "coma" and "now paralyzed" are a few of the startling descriptions included in a new federal report describing the complications from Merck & Co.'s Gardasil medication for sexually transmitted human papillomavirus – which has been proposed as mandatory for all schoolgirls.

The document was obtained from the U.S. Food and Drug Administration by Judicial Watch, a Washington group that investigates and prosecutes government corruption, and it has details of 10 deaths just since September.

"Given all the questions about Gardasil, the best public health policy would be to re-evaluate its safety and to prohibit its distribution to minors. In the least, governments should rethink any efforts to mandate or promote this vaccine for children," said Judicial Watch President Tom Fitton.

The organization's work uncovered reports of about one death each month since last fall, bringing the total death toll from the drug to at least 18 and as many as 20. There also were 140 "serious" reports of complications including about three dozen classified as life- threatening, 10 spontaneous abortions and half a dozen cases of Guillain-Barre Syndrome.

The document reveals the case of an 18-year-old woman who got the Gardasil vaccine, was found unconscious that evening, and died. Another woman, age 19, got the drug and the next morning was found dead in her bed. The new documents also reveal a total of 8,864 Vaccine Adverse Event Reporting System records, up from a total of 3,461 that had been reported in a document just last fall.

WND previously has reported how Merck was lobbying state lawmakers to require the vaccination, but said it would quit the campaign after its activities were unveiled. WND also reported when a key researcher into human papillomavirus, which is targeted by Gardasil, reported it needed more testing, and how even the Centers for Disease Control suggested the vaccine should not be mandatory. That, however, has not diverted the building campaign to have legislatures adopt mandatory vaccination plans.

Judicial Watch said one of the reports, VAERS ID: 310262-1 (D), had this to say:

“Information has been received...concerning a 20-year-old female with no medical history reported, who on 01-APR-2008 was vaccinated with a dose of Gardasil....The patient died four days after...patient sought unspecified medical attention. An autopsy was performed which ruled out suicide and anything suspicious.”

Another report said, “Information has been received from a physician concerning a female patient who on an unknown date was vaccinated with a dose of Gardasil. Subsequently, the patient experienced a coma and is now paralyzed. At the time of this report, the patient’s outcome was unknown. VAERS ID: 303188-1”

The target of the vaccine is cervical cancer, since studies show that those who have HPV have a higher chance of later developing cervical cancer. However, opponents note that such cancers develop most often in older women, while the plan is to require girls as young as 11 or 12 years old to be inoculated. They cite the lack of evidence that the vaccine would have an impact later in life.

A Judicial Watch report said, “Even though Gardasil will not be fully tested for safety until 2009, physicians are already pushing it as a routine, harmless vaccine. Merck’s aggressive advertisement campaign tells young girls that their lives could be ‘one less’ affected by cervical cancer and that, ‘It’s your turn to help guard against cervical cancer.’”

The report also estimated it will cost as much as \$2 billion to buy vaccinations for the nation’s poorest girls.

“This vaccine will be more expensive than all other childhood vaccines put together,” concluded John Schiller, a National Cancer Institute investigator.

Judicial Watch earlier uncovered reports such as this: “Initial and follow-up information has been received from a physician concerning an ‘otherwise healthy’ 13 year old female who was vaccinated with her first and second doses of Gardasil. Subsequently, the patient experienced ... paralysis from the chest down, lesions of the optic nerve...At the time of the report, the patient had not recovered.”

Officials with the Abstinence Clearinghouse noted in a position paper that groups including the Texas Medical Association, the American Academy of Pediatrics, the Association of American Physicians and Surgeons, and the American Academy of Environmental Medicine have come out publicly against mandatory vaccination.

“The reasoning of these medical associations is clear. They are not opposed to medical progress, and certainly support all efforts to combat life-threatening diseases. The problem, as these organizations see it, lies in the fact that the drug only went through three and a half years of testing, leaving the medical community somewhat in the dark as to what serious adverse effects might result in the long term,” the group said at the time.

“Along with the potential of serious adverse effects is the question of efficacy. There is evidence that after approximately four years, the vaccine’s potency significantly declines. The long-term value of the vaccine has yet to be determined; if it wears off within six years, will girls and women need to repeat the battery of injections they originally received?” the organization wondered.

Michigan was the first state to introduce a plan to require the vaccine to be given to young girls, but the proposal failed. Ohio also considered a failed plan in 2006.

In 2007 Merck’s aggressive lobbying campaign and contributions to Women in Government resulted in proposals in at least 39 states to institutionalize such vaccinations.

GARDASIL LINKED TO SEVENTY-EIGHT OUTBREAKS OF GENITAL WARTS

Tuesday, November 11, 2008 by: Joanne Waldron

***The Gardasil vaccine has been linked to 78 outbreaks of genital warts**, according to an article in The Fiji Times entitled “Are our girls guinea pigs?” by Matelita Ragogo. In addition to all of the other adverse reactions to this controversial vaccine, children who receive it are subject to outbreaks of genital warts. Unfortunately, not too many doctors take the time to educate parents about some of these possible reactions prior to giving little girls this expensive jab.*

Deaths, Miscarriages and Other Adverse Events

While genital warts are certainly disgusting, parents who think that genital warts are the worst possible adverse reaction to the vaccine should think again. According to Ragogo, as of August 14th, including the 78 outbreaks of genital warts, there have been 9,748 adverse events reported as per Judicial Watch, a non-profit watchdog group. Judicial Watch also reports that there have been 21 deaths, not including the deaths (by miscarriage) of 10 unborn babies.

Vaccine No Guarantee Against Cell Abnormalities

“Hundreds of thousands of women who are vaccinated with Gardasil and get yearly pap testing will still get high-grade dysplasia (cell abnormalities),” Ragogo reports.

It’s not a cancer vaccine, as media hype may lead some people to believe. Ragogo also points out, “Gardasil has been shown to prevent precancerous lesions, but it has been impossible to ascertain whether it will actually prevent cancer because the testing period has been so short.”

80% of Cervical Cancer Deaths Happen in Developing Countries

According to an article by the King County Health Department in Washington, the “average age of women newly diagnosed with cervical cancer is between 50 and 55 years,” and “risk of developing this cancer is very low among girls less than fifteen.” How long will any possible immunity from a vaccine given to a tiny girl last? No one really knows. So, young girls are being vaccinated for potential problems that they may experience 40 years down the road, if at all, at which time any possible immunity conferred from the vaccine may be long gone. Of course, according to an article by the World Health Organization (WHO), 80% of all cervical cancer deaths happen in developing countries, anyway.

Diets Low in Fruits And Vegetables up Cervical Cancer Risk

The King County Health Department also points out that of the various risk factors for cervical cancer, many can be controlled. One risk factor for cervical cancer is HPV. Ordinarily, getting HPV is not exactly a huge risk for little girls that aren’t having sex. Other risk factors include smoking, HIV, race (African American, Latino, Vietnamese and Native American women are more likely to die from cervical cancer), reduced access to health care, and (possibly) the use of oral contraceptives. The article also states, “Diets low in fruits and vegetables are associated with an increased risk of cervical cancer and several other cancers.”

Could The Vaccine Cause Cancer, Genotoxicity or Infertility?

Another concern about the HPV vaccine is that it hasn’t even been evaluated for its potential to actually cause cancer. Nor has the vaccine been evaluated for the ability to cause genotoxicity (DNA damage). Moreover, it is also unknown as to whether or not this

vaccine could possibly cause future infertility. For all of these reasons, and many others that have been previously reported, it would appear prudent for all parents to think twice before subjecting their children to this “mystery” jab.

GARDASIL STILL WORKING MIRACLES IN 2010: REPORTS OF HEALTH CARE CONCERNS FOLLOWING HPV VACCINATION WITH GARDASIL AND CERVARIX

*There are two HPV vaccines, Gardasil and Cervarix, available to protect against the types of HPV that cause most cervical cancers. The safety of the HPV vaccine was studied in 5 clinical trials before it was licensed. There were over 21,000 girls and women ages 9 through 26 in these clinical trials. Cervarix was studied in over 30,000 females participating in several clinical trials performed all over the world. Cervarix has also been in use in other countries such as England and Europe prior to licensing from the Food and Drug Administration (FDA). CDC and FDA have been closely monitoring the safety of HPV vaccines. There are 3 systems used to monitor the safety of vaccines after they are licensed and being used in the U.S. These systems can monitor adverse events already known to be caused by vaccines, as well as detect rare adverse events that were not identified during a vaccine’s clinical trials. The 3 systems are: The Vaccine Adverse Event Reporting System (VAERS)—a useful early warning public health system that helps CDC and FDA detect possible side effects or adverse events following vaccination. The Vaccine Safety Datalink (VSD) Project—a project between CDC and 8 health organizations evaluate and monitor adverse events following vaccination. The Clinical Immunization Safety Assessment (CISA) Network—a project between 6 academic centers in the U.S. which conduct research on adverse events that might be caused by vaccines. VAERS Limitation A major limitation of VAERS data is that there is no proven causal association between the vaccine and the adverse event. The only association is in time, meaning that the adverse event occurred sometime after vaccination. Therefore, we cannot conclude that the events reported to VAERS were caused by the vaccine. Reports to VAERS Following HPV Vaccination To date, VAERS has not received any adverse event reports occurring in the U.S. following Cervarix. **As of January 1, 2010, approximately 28 million doses of Gardasil were distributed in the United States.** As of January 31, 2010, there were **15,829** VAERS reports of adverse events following Gardasil vaccination in the United States. Of these reports, 92% were reports of events considered to be non-serious, and 8% were reports of events considered serious. Based on all of the information we have today, CDC recommends HPV vaccination for the prevention of most types of cervical cancer. As with all approved vaccines, CDC and FDA will continue to closely monitor the safety of both HPV vaccines. Any problems detected with this vaccine will be reported to health officials, healthcare providers, and the public, and needed action will be taken to ensure the public’s health and safety.*

Non-serious adverse event reports

VAERS defines non-serious adverse events as those other than hospitalization, death, permanent disability, and life threatening illness. The vast majority (92%) of the adverse events reports following Gardasil vaccination have included fainting, pain, and swelling at the injection site (the arm), headache, nausea, and fever. Fainting is common after injections and vaccinations, especially in adolescents. Falls after fainting may sometimes cause serious injuries, such as head injuries, which can be prevented by closely observing the vaccinated person for 15 minutes after vaccination.

Serious adverse event reports

VAERS defines serious adverse events as adverse events that involve hospitalization, permanent disability, life-threatening illness, and death. As with all VAERS reports, serious events may or may not have been caused by the vaccine. All serious reports (8%) for Gardasil have been carefully analyzed by medical experts. Experts have not found a common medical pattern to the reports of serious adverse events reported for Gardasil that would suggest that they were caused by the vaccine. The following is a summary of selected serious adverse event reports that were submitted to VAERS between June 8, 2006 and January 31, 2010. Guillain-Barré Syndrome (GBS) Guillain-Barré Syndrome (GBS) has been reported after vaccination with Gardasil. GBS is a rare disorder that causes muscle weakness. It occurs in 1-2 out of every 100,000 people in their teens. A number of infections can cause GBS.

There has been no indication that Gardasil increases the rate of GBS in girls and women above the rate expected in the general population, whether or not they were vaccinated. Blood Clots There have been some reports of blood clots after receiving Gardasil. These clots have occurred in the heart, lungs, and legs. Most of these people had a risk of getting blood clots, such as taking oral contraceptives (the birth control pill), smoking, obesity, and other risk factors. Deaths As of January 31, 2010, there have been 49 U.S. reports of death among females who have received Gardasil. Twenty eight of these reports have been confirmed and 21 remain unconfirmed due to no identifiable patient information in the report such as a name and contact information to confirm the report. Confirmed reports are those that scientists have followed up on and have verified the claim. In the 28 reports confirmed, there was no unusual pattern or clustering to the deaths that would suggest that they were caused by the vaccine. More information is available at: <http://www.cdc.gov/vaccines/recs/ACIP/downloads/min-archive/min-oct08.pdf> [PDF – 2.37 MB] Summary of HPV Adverse Event Reports Published in JAMA Reports of adverse events after getting a vaccine can be submitted to VAERS by fax at 1-877-721-0366, online at:

<https://secure.vaers.org/VAERSDataEntryintro.htm>, or by mail to Vaccine Adverse Event Reporting System, P.O. Box 1100, Rockville, MD 20849-1100.

NEW STUDY IN 2010 SUGGESTS THE SPREAD OF SEXUALLY TRANSMITTED DISEASES MAY NOT APPLY TO HUMAN PAPILLOMAVIRUS, ALSO KNOWN AS HPV: HPV MIGHT NOT ACT LIKE OTHER STD'S

Released: 3/25/2010 2:15 PM EDT Source: Health Behavior News Service Newswise.— A small new study suggests that some common beliefs about the spread of sexually transmitted diseases **may not apply to human papillomavirus**, also known as HPV. “We shouldn’t assume that we understand transmission” of the virus, said study lead author Dr. Lea Widdice, an assistant professor of pediatrics at Cincinnati Children’s Hospital Medical Center. Widdice and colleagues found evidence that heterosexual couples who share **razors and towels** and engage in some seemingly risky sexual practices **aren’t** at higher risk of giving the disease to each other. “This might be counterintuitive since HPV is thought to infect cells near abrasions and cuts,” Widdice said. Still, Widdice said, the study findings are preliminary and based on an analysis of only 25 couples. **HPV is very common**, and research suggests more than **80 percent** of people will be exposed to it during their lives, Widdice said. It’s transmitted through skin and sexual contact, causing conditions like genital warts and abnormal Pap smears. “**Most people** will clear the infection,” she said. “But the reasons why some women clear the infection and others can’t aren’t well understood.” Women who fail to vanquish the virus can go on to develop **cervical cancer**. Widdice and colleagues examined **DNA tests** of the HPV strains found in 25 couples. The average age of the men was 25 and 23 for the women. The goal was to determine how many of the couples shared the same strain of HPV, which would suggest that one partner gave it to the other. The findings appear online in the Journal of Adolescent Health. Couples who shared towels or razors and had sex involving anal touching (six of the 50 people fell in that category) **were less likely** to share the same strain. “Sharing towels and razors and certain types of sex may transmit HPV, but the virus **doesn’t hang around** and cause an infection,” Widdice said. “So the person can be exposed to the virus but doesn’t get infected. When the couple is tested, they don’t share the same types.” It’s also possible that the immune systems of men and women respond differently to the virus, she said, with men perhaps being able to quickly eliminate it from their bodies. Dr. Anna Giuliano, who studies HPV, said that while the new study is useful, researchers still need to **figure out exactly how and when HPV is transmitted** between couples. “How many sex acts does it take to get a transmission event to occur? And over what period of time within a relationship: two weeks, one month? What is the probability that an infection will be transmitted from one partner to the next? That’s what we really need,” said Giuliano, chair of the Department of Cancer Epidemiology and Genetics at the H. Lee Moffitt Cancer Center in Tampa, Fla. That kind of information will shed light on things like the cost effectiveness of vaccinating **both men and women** against HPV, she said (Journal of Adolescent Health: Contact Tor Berg at (415) 502-1373 or tor.berg@ucsf.edu or visit <http://www.jahonline.org/Widdice> LE, et al. Human papillomavirus concordance in heterosexual couples. J Adolesc Health online, 2010. <http://www.newswise.com/articles/view/562681?print-article> Keywords: Hpv Infection, Sexually Transmitted Infections).

LETTER FROM LAYLA

Dear Dr. Andrew Maniotis,

Good afternoon, sir. Thank you for your reply. Yet I am still wondering, what does having HPV DNA really mean, then? I would assume it means in the past I was exposed to something of which I have now built natural immunity, thus the “normal” pap smears for several years. So WHY an HPV DNA test? I feel I was treated like a criminal. A total encroachment to my so called “civil” rights.

Obviously having HPV DNA (from 2006 testing) does not mean I am carrying active HPV, am I correct? I assume it means nothing. What would I ever tell a potential partner, or would I need to say anything at all? This type thing RUINS relationships!

ALL based on fraudulent DNA testing.

The last guy I was with (for almost three years) knew about the HPV, and did not care. But that relationship ended due to his not being able to remain monogamous, etc. A typical latin womanizer. I do feel that I was used as a human lab rat by various doctors, and it infuriates me. I am in great health and take excellent care of myself. I do not smoke. I run everyday. I am actually in far better physical health than ANYONE I have ever met. I also have lots of energy. I am drug free and do not drink much alcohol. Most people I know are addicts of one thing or another, and most I know are not in good physical health. And most people I know are totally ignorant when it comes to just about anything besides what they see on their TV sets.

But there is this stigma with the HPV DNA thing, from the 2006 “testing” I was given without my consent, twice in six months by the same, uh, you know what female doctor. I cannot stand her. She makes me so mad. You cannot imagine the humiliation. If I was used as a medical lab rat, does this not give me legal grounds to sue the doctor who did this to me, or perhaps Digene Diagnostics?

It really does not seem very fair. It has caused me extreme anxiety, depression, stress, and humiliation. This is why I will never trust another doctor again. Most are such arrogant and insensitive people. I am in Texas, and you can imagine how bad it is here. I hate Houston. We also have that creep Perry in office, who tried to mandate Gardasil vaccinations. Anyway, thank you so much for your reply. I really really appreciate it very much.

Regards, Layla

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Appendix 1-More information about “The Immortal Henrietta Lacks.”

Different versions of this story have been told by various reporters or agencies, and it is clear by all accounts that the “unwitting” isolation of Henrietta’s cells was not done with her family’s knowledge, despite her cervical cancer condition. For instance, in the BBC documentary on the life and times of the Lacks family which describes how HeLa cells corrupted the entire world directed by Adam Curtis, we find her brother being interviewed and saying, “nobody said she had cancer” during the time she was being “treated” at John’s Hopkins.

The story of Henrietta Lacks also is recounted in a new book by Rebecca Skloot. Because of the importance of HeLa to the generation of all current theories of cancer, some historical documentation is important to include here, and consider below, which I provide verbatim. Certain facts about this history, in addition, reveal much about the medical and social biases of the 1950’s and 1960’s surrounding cancer as well as how Lyndon Johnson’s and then Richard Nixon’s war on cancer became established.

On February 1, 1951, just days after a march for a cure for polio in New York City, Lacks visited Johns Hopkins because of a painful “knot” in her cervix and a bloody vaginal discharge. That day, she was diagnosed with cervical cancer, and the appearance of the tumor was unlike anything that had ever been seen by the examining gynecologist Dr. Howard Jones who, with

his wife Georgeanna, would go on to found the Jones Institute for Reproductive Medicine at Norfolk, Virginia's Eastern Virginia Medical School (Rebecca Skloot, *The Immortal Life of Henrietta Lacks*, 2010).

Prior to receiving treatment for the tumor, [the] cells from the carcinoma were removed for research purposes without her knowledge or permission, which was standard procedure at that time. At her second visit eight days later, Dr. George Otto Gey took another sample from her tumor and saved some. It is from this sample that the HeLa cells came (http://en.wikipedia.org/wiki/Henrietta_Lacks).

Lacks was treated with radium tube inserts, sewn in place, a common treatment for these types of cancers in 1951. After several days in place, the tubes were removed and she was released from Johns Hopkins with instructions to return for X-ray treatments as a follow up. Lacks returned for the X-ray treatments. However, her condition worsened and the Hopkins doctors treated her with antibiotics, thinking that her problem might be complicated by **an underlying venereal disease (it has been rumored that she had neurosyphilis and presented with acute gonorrhea at one point as well)**.

In significant pain, and without improvement, Lacks returned to Hopkins demanding to be admitted on August 8 and remained until her death. Though she received treatment and blood transfusions, she died of uremic poisoning on October 4, 1951 at the age of thirty-one (Smith, Van. April 17, 2002. "The Life, Death, and Life After Death of Henrietta Lacks, Unwitting Heroine of Modern Medical Science." *Baltimore City Paper*, 2007-08-21). A subsequent partial autopsy showed that the cancer had metastasized throughout her body.

George Gey "discovered that [Henrietta's] cells did something they'd never seen before: They could be kept alive and grow[n indefinitely]. Gey named the sample "HeLa", after the initial letters of Henrietta Lacks' name. As the first human cells that could be grown in a lab and were "immortal" (did not die after a few cell divisions), they could then be used for conducting many experiments.

This represented an enormous boon to medical and biological research.. According to reporter Michael Rogers, the growth of HeLa by a researcher at the hospital helped answer the demands of the 10,000 who marched for a cure to polio just shortly before Lacks' death. By 1954, HeLa was being used by Jonas Salk to develop a vaccine for polio. To test Salk's new vaccine, the cells were quickly put into mass production in the first-ever cell production factory. Demand for the HeLa cells quickly grew.

Since they were put into mass production, Henrietta's cells have been mailed to scientists around the globe for "research into cancer, AIDS, the effects of radiation and toxic substances, gene mapping, and countless other scientific pursuits". HeLa cells have been used to test human sensitivity to tape, glue, cosmetics, and many other products. Scientists have grown some 20 tons of her cells. Doctors still have not discovered why HeLa cells are so unique. There are almost 11,000 patents involving HeLa cells.

From, The "HeLa bomb"incident" chapter in "The Immortal Life of Henrietta Lacks," by Rebecca Skloot:

"I have not been able to ascertain the supposed racial origin of all eighteen lines," Gartler told the audience. "It is known, however, that at least some of these are from Caucasians, and that at least one, HeLa, is from **a Negro**." He knew this, because a few months earlier, he'd written George Gey:

'I am interested in the racial origin of the person from whom your HeLa cell line was initiated. I have checked a number of the early papers describing the development of the HeLa cell line but have not been able to find any information pertaining to the race of the donor.'

When Gey responded that HeLa cells had come from "**a colored woman**," Gartler knew he'd found the source of **the problem**.

“It seems to me the simplest explanation,’ he told the audience, “is that they are all HeLa cell contaminants.”

Scientists knew they had to keep their cultures free from bacterial and viral contamination, and they knew it was possible for cells to contaminate one another if they got mixed up in culture. But when it came to HeLa, they had no idea what they were up against. It turned out Herietta’s cells could float through the air on dust particles. They could travel from one culture to the next on unwashed hands or used pipets; they could ride from lab to lab on researcher’s coats and shoes, or through ventilation systems. And they were strong: if just one HeLa cell landed in a culture dish, it took over, consuming all the media and filling all the space.

Gartler’s findings did not go over well. In the fifteen years since George Gey had first grown HeLa, the number of published articles involving cell culture had more than tripled each year. Scientists had spent millions of dollars conducting research on those cells to study the behavior of each tissue type, comparing one to another, testing the unique responses of different cell types to specific drugs, chemicals, or environments. If all those cells were in fact HeLa, it would mean that millions of dollars had been wasted, and researchers who’d found that various cells behaved differently in culture could have some explaining to do.

Years later, Robert Stevenson, who became president of the American Type Culture Collection, described Gartler’s talk to me in this way:

‘He showed up at that meeting with no background or anything else in cell culture and proceeded to drop a turd in the punch bowl.’

Stevenson and other members of the Cell Culture Collection Committee sat stunned in the audience as Gartler pointed to a chart on the wall listing the eighteen cell lines that had been contaminated by HeLa, along with the names of the people or places he’d gotten them from. At least six of the contaminated lines came from ATCC. HeLa had penetrated Fort Knox.

At that point, the ATCC’s collection had grown to dozens of different types of cells, all guaranteed to be free from viral and bacterial contamination, and tested to ensure that they hadn’t been contaminated with cells from another species. But there was no test to see if one human cell had contaminated another. And, to the naked eye, most cells growing in culture look the same.

Now Gartler was essentially telling the audience that all those years researchers thought they were creating a library of human tissues, they’d probably just been growing and regrowing HeLa. He pointed out that a few years earlier, when scientists started taking protective measures against cross-species contamination—such as working under sterile hoods—it had suddenly become harder to grow new cell lines. And in fact, “very few [new human cell lines] have been reported since.” Not only that, he said, but there had been no new examples of “so-called spontaneous transformed human cell cultures” since. Everyone in the audience knew what that meant. On top of saying they’d possibly wasted more than a decade and millions of research dollars, Gartler was also suggesting that spontaneous transformation—one of the most celebrated prospects for finding a cure for cancer—might not exist. Normal cells don’t spontaneously become cancerous, he said; they were simply taken over by HeLa.

Gartler concluding his talk by saying, “Where the investigator has assumed a specific tissue of origin of the cell line, i.e. liver...or bone marrow, the work is open to serious question, and in my opinion would be best discarded.”

The room sat silent, dumbfounded, until T.C. Hsu, the chair of Gartler’s conference session, spoke. Hsu was the University of Texas geneticist whose earlier work with HeLa and other cells had made it possible to discover the correct number of human chromosomes.

“A few years ago I voiced some suspicion about cell-line contamination,” Hsu said. “So I am happy about the paper by Dr. Gartler and am also sure he has made many people unhappy.”

He was right, and those people quickly began asking questions.

“How long did you keep them in your laboratory?” one scientist asked, suggesting that Gartler had contaminated the cells himself after they arrived in his lab.

“They were analyzed before being grown in my laboratory,” Gartler responded.

“They didn’t send them to you frozen?” the scientist asked, knowing that contamination could have occurred while they thawed.

Gartler said that didn’t matter-**the cells didn’t have to be thawed to be tested.**

Another scientist wanted to know if the similarity Gartler was seeing between cell lines was just the effect of spontaneous transformation making all cells act the same.

Eventually Robert Stevenson of the Cell Culture Collection Committee spoke up, saying, “It looks like more detective work is needed to see...whether we are going to have to start all over again to isolate some new human cell lines.”

Hsu stepped in and said, “I would like to give particular priority to those who initiated the cell lines, whom Dr. Gartler has attacked. If there is any defense, we would like to hear it.”

Harvard’s Robert Chang-whose widely used Chang Liver Cell line was listed as a HeLa contaminant on Gartler’s chart-glared from his seat. Chang had used those cells to discover enzymes and genes specific to liver cells. If Gartler was right and the cells were actually from Henrietta’s cervix, Chang’s liver research using them was worthless.

Leonard Hayflick had an especially personal connection with his cell line, WISH, which Gartler had listed as contaminated: he’d grown it using cells from the amniotic sac in which his unborn daughter had once floated. He asked Gartler whether it was possible to find G6PD-A in samples from white people.

“Caucasian subjects with G6PD-A have not been reported” Gartler told him.

Later that day-in a talk chaired by George Gey-Hayflick delivered a paper, on the “facts and theories” of spontaneous transformation of cells in culture. Before beginning his talk, Hayflick stood at the podium and announced that, since WISH cells supposedly tested positive for a genetic marker found only in black people, he’d called his wife during the break to ask if he was, in fact, his daughter’s father. “She assured me that my worst fears were unfounded,” Hayflick said. The room erupted in laughter, and no one said anything else publicly about Gartler’s findings.

But a few people took Gartler seriously: before leaving the conference Stevenson met several of the top cell culturists for lunch. He told them to go back to their labs after the conference and start testing cells for the G6PD-A genetic marker, to see how widespread this problem might be. Many of their cell lines tested positive, including the skin cells George Hyatt had transplanted onto a soldier’s arm years earlier. Since Hyatt had no HeLa cells in his lab at the time, the cells in his experiment must have been contaminated before they arrived. And though few realized it, the same thing was happening in laboratories around the world.

Still, many scientists refused to believe HeLa contamination was real. After the conference where Gartler dropped what became known as “the HeLa bomb,” most researchers kept right on working with the cells he’d said were contaminated. But Stevenson and a few other scientists realized the potential scope of the HeLa contamination problem, so they began working to develop genetic tests that could specifically identify HeLa cells in culture instead of just testing for the presence of G6PD-A. And those genetic tests

would eventually lead them to Henrietta's family.

Appendix 2. Rosalind Harrison-Chirimuuta and Richard Chirimuuta **Racism and "AIDS from Africa"** (<http://www.virusmyth.net/aids/data/javirus.htm>).

The first black people diagnosed as suffering from AIDS in any number were Haitians living in the United States. The possibility that they may have caught the infection from Americans in the United States or in Haiti was not given serious consideration and Haiti was immediately accused of being the source of the epidemic. Soon Haitians with AIDS were being reported from all over the Western world and the Centers for Disease Control (CDC) in Atlanta, Georgia, included Haitians as a group at risk for AIDS along with homosexuals, intravenous drug users and haemophiliacs. It was only in 1985 that CDC, faced with overwhelming evidence that Haitians per se were no more at risk for AIDS than anyone else, removed them from the high risk classification, but not before Haitians en masse were dismissed from their jobs, evicted from their homes, and even housed in separate prisons. Abandoning Haiti, the researchers then turned their attention to Africa.

One of the reasons given by scientists for this turn to Africa was the high incidence of Kaposi's sarcoma (KS) in Africa, although it was clear from the beginning that the benign clinical course of African KS was very different from the aggressive, disseminated form of KS in AIDS patients. A number of AIDS-like cases were reported retrospectively, the most cited being a Danish surgeon who worked in Zaire and died in 1977. This patient was given prominence in the best-selling book by Randy Shilt's "And The Band Played On" where, under "Dramatis Personae" she is listed as "Danish surgeon in Zaire, first westerner to have died of AIDS," and is described in the following manner (<http://www.virusmyth.net/aids/data/reafrika.htm>):

"The battle between humans and disease was nowhere more bitterly fought than here in the fetid equatorial climate, where heat and humidity fuel the generation of new life forms... Here, on the frontiers of the world's harshest realities, Grethe Rask tended the sick."

It was claimed that she acquired the infection from her patients, at least one of whom had KS, but there was no firm evidence that she died of AIDS, and other diagnostic possibilities were not considered. In 1988, five years after the case was published, we learned that her serum had been tested and found human immunodeficiency virus (HIV) enzyme linked immunosorbent assay (ELISA) negative, but the author of the original paper has not published this information in the scientific literature.

Although such AIDS-like cases are presented as evidence that the human immunodeficiency virus existed in Africa prior to the American epidemic, only African cases are considered and the many instances of AIDS-like cases documented in Europe and America are conveniently ignored. Indeed, on the opposite page to the report of the Danish surgeon in the same issue of The Lancet was an account of an AIDS-like illness in a young German homosexual, but whilst non-AIDS in a Danish surgeon heads the citation index proving an African origin, the German case has been completely ignored.

The next source of support for the African hypothesis came from the seroepidemiological studies undertaken in Africa or on African serum stored in the West. This research, more than any other, has been at the foundation of all the fantastic stories of millions dying in Africa. Using an enzyme linked immunosorbent assay seropositive figures of 25% of patients attending a clinic in rural Zaire in 1984, 50% of the Turkhanas in Kenya from 1980-1984, and 66% of children in Uganda in 1972 were reported. As AIDS was rare or unknown in the areas where the serum was collected, one would expect the authors to have had serious doubts about the reliability of the tests, but sadly scientific scepticism has never been a feature of AIDS research in Africa.

One of the most cited studies was undertaken on serum collected in Zaire in 1959. Using a number of tests in addition to ELISA, only one of 1,213 plasmas was positive, but the identity of the donor, described as "rural Bantu", was no longer known. As with the sporadic AIDS-like cases, only seroepidemiology in Africa is considered relevant to the question of the origin of HIV. A study using

the same tests was undertaken on stored serum taken from “aboriginal” Amazonian Indians in Venezuela in 1968/69, and 9 of 224 samples were positive on all the tests. The results were challenged by other researchers as probable false positivity,(30) but the single positive sample from Zaire continues to be cited as evidence that the world AIDS epidemic began in Zaire 35 years ago.

In an interview shown on British television Professor Hunsmann, head of virology and immunology section and professor of medicine at the (West) German Centre of Primate Research at Gottingen, discussed the problems of seroepidemiology:

“We had conducted quite extensive experiments in respect to the epidemiology... first human retrovirus... HTLV [Human T-Lymphotropic Virus]... For this reason we had several thousand serum samples frozen and saved in our refrigerated stock. When the news came that there was another, and new human retrovirus discovered, the AIDS virus... we could immediately search among our stock and probe for an earlier presence of this virus in Africa... These tests quickly and clearly gave results, namely, that the first “positive” probes which we could find among our more than 7,000 serum samples are dated only after the beginning of the ‘eighties, from the years 1982-83; and that among samples from before that date — and we had quite a lot of that earlier time in our stock- not a single one proved positive. We have concluded from all this that most other researchers had probably fallen victim to the technical difficulties connected with the conservation and analysis of older serum samples. And the American authors who originally had produced those high percentage data had to correct them — but certainly, once some wrong information like that has been put into circulation it continues to go on. This has led to quite a lot of friction between some African states and the United States.”

Later in the same interview when asked why AIDS is not considered to have originated in the United States, Professor Hunsmann made the following comment:

“Testing of the kind being done in Africa and to that volume has never been done by anyone in America. Nobody has looked at the stocked blood serum in the USA and there certainly is much more there than in Africa. Nor has anyone asked what happened to the general population. Only one single group, the homosexual community in San Francisco, has been analysed and the results showed a high percentage of HIV positivity already by the mid 1970’s. But no other samples have been tested to the extent done in Africa. I think this should be clearly said.”

Why, then, if this research is valid (and there have not been any serious criticisms) have other AIDS researchers persisted in arguing that the African AIDS epidemic preceded the epidemic elsewhere in the world? And if the tests are unreliable, why are the predictions that millions of Africans will soon die from AIDS still presented without comment? How, indeed, is it possible that a virus could spread so much more rapidly by heterosexual contact in Africa than anywhere else in the world? It is here as in so many other aspects of AIDS research, that racist beliefs about the sexual propensities and promiscuity of Africans conflict with scientific evidence, and in such a confrontation belief is almost invariably the victor. Researchers had originally proposed that AIDS was an “old disease of Africa” that had reached the West via recent intercontinental travel , a rather curious notion given the enforced intercontinental travel of up to 100 million Africans in previous centuries. [Slavery-my emphasis]. As this hypothesis became increasingly untenable attention was diverted to the possibility of a monkey origin of the virus. Such ideas cohabit easily with racist notions that Africans are evolutionary closer to sub-human primates. Dr. Robert Gallo and his co-workers were among the pioneers of this line of research, both for HTLV-I and HTLV-III (later renamed HIV). Two of Gallo’s colleagues, Kanki and Essex, reported the isolation of a virus similar to HTLV-III in macaque monkeys who were suffering from an AIDS-like illness, and labeled it simian T-lymphotropic virus type III (STLV-III) of macaques. For those who were arguing an African origin of the AIDS virus, an Asian monkey like the macaque was not a suitable source, but less than six months later the same researchers reported finding the virus in “wild-caught” African green monkeys from Kenya and Ethiopia. This research, like most other research on AIDS in Africa, was motivated only by a desire to prove an African origin of the disease, and was greeted with enthusiasm by the Western scientific community. Discussion quickly moved on to the question of how the virus crossed the species barrier, and two

AIDS “experts” from St Mary’s Hospital in London even offered this explanation:

“Monkeys are often hunted for food in Africa. It may be that a hunting accident of some sort, or an accident in preparation for cooking, brought people in contact with infected blood. Once caught, monkeys are often kept in huts for some time before they are eaten. Dead monkeys are sometimes used as toys by African children.”

Are we seriously to believe that African parents are so desperate for toys for their children that they give them putrefying carcasses of dead animals? More fantastic suggestions were published in The Lancet:

“Sir: The isolation from monkeys of retroviruses closely related to HIV strongly suggests a simian origin for this virus... Several unlikely hypotheses have been put forward... In his book on the sexual life of people of the Great Lakes area of Africa Kashamura writes: “pour stimuler intense, on leur inocule dans les cuisses, la region du pubis et le dos du sang preleve sur un singe, pour un homme, sur une guenon, pour ne femme” (to stimulate a man or a woman and induce them to intense sexual activity, monkey blood [for a man] or she-monkey blood [for a woman] was directly inoculated in the pubic area and also the thighs and back). These magic practices would therefore constitute an efficient experimental transmission model and could be responsible for the emergence of AIDS in man.”

This came in for particular derision at the conference on AIDS in Africa held in Naples in October 1987:

When queried regarding the plausibility of a premise put forth in a letter to The Lancet suggesting that a bizarre tribal ritual of injecting monkey blood into the pubic region of young African men and women to stimulate intense sexual activity could be responsible for the emergence of AIDS in man, researchers from Zaire, Congo and Belgium were unanimous in declaring it to be preposterous....

It is hardly surprising that western AIDS researchers have become persona non grata in many African countries.

According to documented conversations with Dr. Gallo, his work with Dr. Montagnier was open and collaborative, and Gallo believes that his work established that AIDS and Montagnier’s group’s pre-AIDS from “Patient One” exhibited consistent molecular markers or profiles. These markers were first called lymphadenopathy-associated virus (LAV-BRU and LAV LAI), Human T-cell-lymphotropic virus type III (HTLV-IIIB), AIDS-related virus (ARV) and, finally, Human Immunodeficiency Virus (“HIV”).

The importance of these initial observations in shaping the AIDS era cannot be overestimated. From a later NEJM retrospective article written jointly by Gallo and Montagnier we learn that:

The story began in an unfavorable environment: during the late 1970’s many people thought that epidemic diseases caused by microbes, including viruses, no longer posed a threat in industrialized countries. Other prevailing beliefs were that viruses did not cause any human cancer and that there was no such thing as a retrovirus that infected humans. Some of these beliefs were justified, since attempts to find tumor viruses and, in particular, retroviruses in cancers and other diseases in humans had a troubled history, and many of the groups that had the greatest expertise in the study of retroviruses had turned their efforts toward research on oncogenes. Luckily, and rather amazingly, however, the conceptual and technical tools arrived in our hands just before the first patients with AIDS were identified in 1981. In addition, there remained a few heretical or “old-fashioned” groups—among which were our two laboratories—that persisted in searching for retroviruses in human cancers, particularly breast cancers and leukemias. This search finally paid off with the discovery of human T-cell leukemia virus types 1 and 2 (HTLV-1 and HTLV-2), the first of which was shown to cause an unusual T-cell leukemia. This discovery was made possible by 15 years of basic research on leukemogenic retroviruses in animals, including the design and development of highly sensitive biochemical assays that were based on reverse transcriptase—the enzyme that is present in all retroviruses, which was discovered in 1970 by Temin

and Baltimore.

An additional important contributor was the development of methods for growing T lymphocytes in culture for a period sufficient to allow the expression of putative latent retroviruses. This effort was helped greatly by the isolation of specific factors-in particular, the T-cell growth factor (now called interleukin 2) in Bethesda, Maryland. The role of interferon in repressing the production of retroviruses in mouse cells was demonstrated in Paris, and this discovery led to the use of anti-interferon serum in the search for human retroviruses. Thus, at the beginning of the 1980's, we had the essential tools required to search for a retrovirus in this new and menacing disease called AIDS. But why search for a virus, and specifically a retrovirus, in AIDS? The answer was far from obvious in 1982.

At that time, AIDS had already appeared as a long-lasting disease, with an extremely long lag time between exposure to the agent (through blood or sexual activity) and the profound state of immune suppression characterized by the occurrence of opportunistic infections or cancers. Many factors-fungi, chemicals, and even an autoimmunity to leukocytes-were invoked at that time as possible causes. However for us, there were clues. First, the various manifestations of AIDS were unified by a biologic marker: a decrease in the levels of a specific subgroup of T cells that harbored the CD4 surface antigen. CD4 and other CDs had been identified only a few years earlier with the use of specific monoclonal antibodies, thanks to the work of Milstein and Kohler. The findings regarding the T cell subgroup suggested an agent that specifically targeted CD4+ T cells, and HTLV (the putative cancer causing virus) was one such agent. Moreover there were animal models in which lymphotropic retroviruses caused not only leukemias or lymphomas, but also an AIDS-like wasting syndrome. Furthermore, HTLV was transmitted through blood and sexual activity, as well as from mother to infant, which was consistent with some of what we learned early on about the epidemiology of AIDS. Finally, the Centers for Disease Control and Prevention (CDC) reported cases of AIDS in patients with hemophilia who had received only filtered clotting factors, which seemed to eliminate the possibility that the agent was a microorganism larger than a virus.

This set of arguments convinced us, as well as Max Essex in Boston, each independently to start a search for an HTLV-like virus in patients with AIDS. We began conducting this research at the National Institutes of Health in Bethesda and at the Pasteur Institute in Paris. The theory that a retrovirus caused AIDS was correct, but the hypothesis that it was a close relative of HTLV proved to be wrong. In Bethesda, an earlier survey involving the use of molecular and immunologic probes seemed to favor a variant similar to HTLV-1 (the putative cancer virus). In fact, some patients with AIDS were doubly infected with HTLV-1 and the new agent, which complicated the interpretation of the nature of the virus causing AIDS.

Appendix 3. More cogent reasons for cancer induction than persistent or latent infections with rumor viruses: The above ground test-ban treaty based on Commoner's and Bauer's baby-tooth survey in St. Louis.

As a child growing up in St. Louis during the late 1960's and 70's, I learned of one of the truly powerful achievements of biology. The biochemist and ecologist, Berry Commoner, and the Pathologist Harold Bauer, collected some 300,000 children's teeth from St. Louis dentist offices and tested them for radioactive isotopes. The teeth were contaminated with radioactive strontium 90 because strontium and calcium chemically substitute, and, the Cold War atomic and hydrogen bomb testing had contaminated the grass of the Midwest's grain belt. The cows that ate this radioactive grass produced radioactive milk containing strontium 90. Children who drank that strontium 90-laced milk had radioactive teeth (and perhaps other tissues in which calcium is critical, such as nerve and brain tissue, and bone. Most living cells and processes in the body employ calcium). When published, the Commoner and Bauer study became the justification for the first above ground nuclear test-ban treaty. The treaty demonstrated the awesome power that a simple, comprehensive scientific study could have. It made a huge impact toward changing the human condition and the direction of society, and confirmed Mankind's long-term tendency from prehistorical times to strive toward "The Good," rather than toward self-destruction, self-extinction, and death.

Because of the acceptance of baby tooth study by the scientific community and eventually, the world of international politics, it was surmised that science, when it is well done and effectively presented, could vanquish even the powerful forces of politics and religion. It could overpower the arms merchants, the military industrial complex, and even The Church of Modern Medicine—a term advanced by Dr. Robert Mendelsohn, in his book “*Confessions of a medical heretic.*”

There was a group of 16 and 17 year-olds who scored at least 10-15 points lower on National standardized U.S. college tests than other kids had who were born earlier or later than 1956-7. Years later, these 16 or 17 year-olds were told that their poor performance on standardized tests was likely due to the fact the most nuclear test blasts had been detonated during the mid-1950 nuclear bomb-testing years during the beginning of the Salk vaccine era, and when they were In Utero or newborns, circa 1956-7. I was born in 1957 which explains a lot, perhaps, about my brain.

Early during my career, as part of the search to find new anti-seizure medications, Dr. Olney taught me electron microscopy, and how to induce seizures. I photographed the effects of excitotoxins on rat brains and documented, using the electron microscope, how they affected the dendrites, synapses, and pathways of connections in the brain and spinal cord. Some of this work on the effects of various compounds and drugs was published [Clifford, DB., Olney, J.W., Maniotis, A., Collins, RC., Zorumski, CF. The functional anatomy and pathology of Lithium-Pilocarpine and high dose pilocarpine seizures. *Neuroscience* Vol 23 (3): pp.953-968, 1987].

In the year-and-a-half that I was in that neuro-excitotoxin lab, a drama unfolded as Dr. Olney struggled to alert the medical and scientific community, and the nation, about the potential harmful effects of the artificial sweetener, aspartame, on the brain.

Donald Rumsfeld’s company Searle, made aspartame, and Searle had been accused of fudging safety data. Dr. Olney cautioned that, especially in children, these compounds were a significant potential threat, because they were routinely used at very low concentrations (5 ng/kg) to experimentally induce seizures in our animals (rats). When the substance was put into liquids like soft-drinks, it was reported that Nutrasweet, as it was commercially known, became unstable and broke-down into a [tumor](#) agent called diketopiperazine (DKP). It was claimed that this agent caused brain tumors.

The problem with Mr. Rumsfeld’s acidic amino acid-containing excitotoxin, Dr. Olney argued on two episodes of *60 Minutes*, was that the safety data released by Rumsfeld’s company and the FDA were not correct. They clashed with the neurotoxicity and cancer-causing data that Olney’s work, and the work that others in excitotoxin research, especially the Japanese, had previously reported. As presented by By James Turner, ESQ., Director of the National Institute of Science, Law, and Public Policy, the controversy unfolded as follows (NISLAPP):

In December 1965, while working on an ulcer drug, James Schlatter, a chemist at G.D. Searle, accidentally discovered aspartame, a substance that is 180 times sweeter than sugar yet has no calories.

In the Spring of 1971, Dr. John Olney (whose pioneering work with monosodium glutamate, was responsible for having it removed from baby foods) informed Searle that his studies showed that [the acidic amino acid] aspartic acid (one of the ingredients of aspartame) caused holes in the brains of infant mice. One of Searle’s own researchers confirmed Dr. Olney’s findings in a similar study.

On July 26, 1974, the FDA granted aspartame its first approval for restricted use in dry foods.

In August of 1974, Jim Turner and Dr. John Olney filed the first objections against aspartame’s approval.

On March 24, 1976, Turner’s and Olney’s petition triggered an FDA investigation of the laboratory practices of aspartame’s manufacturer, G.D. Searle. The investigation found Searle’s testing procedures shoddy, full of inaccuracies and “manipulated” test

data. The investigators report they “had never seen anything as bad as Searle’s testing.

On January 10, 1977, the FDA formally requested the U.S. Attorney’s office to begin grand jury proceedings to investigate whether indictments should be filed against Searle for knowingly misrepresenting findings and ‘concealing material facts and making false statements’ in aspartame safety tests. **This is the first time in the FDA’s history that they request a criminal investigation of a manufacturer.**

On March 8, 1977, G. D. Searle hired prominent Washington insider Donald Rumsfeld as the new CEO to try to turn the beleaguered company around. A former Member of Congress and Secretary of Defense in the Ford Administration, Rumsfeld brought in several of his Washington cronies as top management.

On August 1, 1977, The Bressler Report, compiled by FDA investigators and headed by Jerome Bressler, was released. The report found that 98 of the 196 animals died during one of Searle’s studies and weren’t autopsied until later dates, in some cases over one year after death. Many other errors and inconsistencies are noted. For example, a rat was reported alive, then dead, then alive, then dead again; a mass, a uterine polyp, and ovarian neoplasms were found in animals but not reported or diagnosed in Searle’s reports.

“On January 21, 1981, Ronald Reagan was sworn in as President of the United States. Reagan’s transition team included Donald Rumsfeld, CEO of G. D. Searle, who then hand picked Dr. Arthur Hull Hayes Jr. to be the new FDA Commissioner.

Rumsfeld, with his new FDA Commissioner thus ushered Nutrasweet through safety testing and Federal Regulation for use in the general food supply by 1983. I thus became aware for the first time how politics, economics, and corporate interests could be more influential at shaping health policy than science. (Two decades later, I would learn more about of the genius of Donald Rumsfeld’s scientific contributions, as the Chairman of the Board of Gilead Bioscience, the maker of tamiflu to treat “bird” and “swine flus,” and atipla, the biggest selling AIDS-drug during the past several years). All this was unlike what had happened during the comprehensive above ground nuclear test ban treaty and Commoner’s and Bauer’s baby tooth survey, and I began to lose my innocence.

Appendix 4: DIETHYLSTILBOESTROL (DES):

At The University of California at Berkeley I met Dr. Howard Bern, who hired my wife as the graduate program’s administrative assistant while she was pregnant with our first child. Dr. Howard Bern, was a distinguished endocrinologist, cancer biologist, and a member of the National Academy. Dr. Bern had exposed the dangers of DES (diethylstilboestrol)-a widely used synthetic oestrogen (Modified from DES action of Australia <http://www.desaction.org.au/aboutdes.htm>):

Synthesised in 1938, laboratory studies showed animals administered DES developed mammary cancer, with high rates of fetal death, sterility and cancer in the offspring. Despite this, DES was approved for use in humans in 1940. Initially used to treat late pregnancy complications, by the mid 1940s the use was widened to include the prevention of miscarriage, i.e. for prophylactic use by women who had a history of miscarriage. However by 1949 DES was seen as making “a normal gestation more normal”. By the early 1950s DES was being prescribed and marketed as a general pregnancy “tonic”, mixed with vitamins and recommended for all pregnant women to ensure healthier pregnancies with “bigger and stronger” babies. A 1953 study showed, to the surprise of the researchers, that the DES-treated group experienced higher rates of miscarriage, premature labour and neonatal death than the control group, and the 1953 Dieckmann study was ignored. DES was already entrenched as standard obstetric clinical practice. Also by this stage DES was being aggressively promoted by the drug companies for use in all pregnancies. In 1971 it was discovered that DES caused clear cell cancer of the vagina/cervix in DES daughters. DES was thus proven to be carcinogenic in humans. Regardless of these findings, it was continued to be used as a treatment for acne, to dry up breast milk, as a contraceptive

like a morning after pill, as hormone replacement therapy during menopause, as a treatment for “tall girls” to stunt their **adult height**, and to fatten up livestock to increase profits.

In 1981 a landmark publication, ‘Developmental Effects of DES in Pregnancy’ was edited by Arthur L Herbst and Howard A. Bern, which brought together leading experimental researchers and expert clinicians on DES. In an experiment **on mice**, Herbst and Bern showed that in later life, **the immune system of DES exposed mice was suddenly compromised**. Preliminary studies of **DES daughters** in the early 1980s indicated that DES exposure is linked with **immune system problems**, including a higher incidence of **autoimmune disease**, such as asthma, arthritis, diabetes, systemic lupus and thyroid dysfunction.

Appendix 5. Preventable causes of cancer.

For the past decade, one of my more pleasurable collaborations has been with Sam Epstein, a vocal opponent of the cancer industry, and author of the book, “*Stop Cancer Before It Starts*.” I’ve been following Dr. Epstein’s work with his organization, The Cancer Prevention Coalition, ¹ and at one point he asked me as a cancer biologist to sign his *Stop Cancer Before It Starts* book a few years ago. In it, there is a breathtaking compilation of the conflicts of interests and corporate stakeholders. ¹The book is free and available on the internet as an HTML file or as a PDF, and I have referenced it for you below. ² In it you will find huge amounts of information regarding the insanity wrought by the most toxic polluters of our world guarding the purses of federal cancer research money. Below I provide two of the tables you will find in the *Stop Cancer Before It Starts* book. In it you will also find some of our favorite protagonists, like Sam Broder, the pusher of AZT, and his ilk, calling the shots.

These professional mindsets are compounded by poorly recognized institutionalized conflicts of interest, particularly for the ACS. For decades, powerful groups of interlocking corporate interests, with the highly profitable cancer drug industry at their hub, have dominated the losing war against cancer. In a surprisingly frank statement, Dr. Samuel Broder, NCI Director from 1989 to 1995, stated the obvious: “The NCI has become what amounts to a government pharmaceutical company”. Broder resigned from NCI to become Chief Scientific Officer of Ivax, subsequently moving to become Chief Medical Officer of Celera Genomics; both companies are major manufacturers of cancer drugs. By linking their interests with those of major cancer drug companies, both NCI and ACS have directed their priorities away from research on prevention to virtually exclusionary emphasis on damage control. The professional mindset of the NCI is strikingly confirmed by the lack of expertise in prevention of its successive 3-member executive President’s Cancer Panels since their inception in 1971; *additionally, some members had deep personal ties with drug and petrochemical industries. Concerns on professional mindsets also apply to the successive 18-member National Cancer Advisory Boards, which “almost totally lack expertise in environmental and occupational carcinogenesis.” This is in clear violation of Section 407(a)(1)(B) of the National Cancer Act, requiring that no less than five Board members “shall be individuals knowledgeable in environmental carcinogenesis.”*

NCI and ACS Reliance on Industry-Biased Claims on Cancer Causation

The cancer establishment has long insisted that faulty lifestyle, particularly smoking, inactivity, and fatty diet—excluding recognition of its contamination with carcinogenic pesticides—is the predominant cause of cancer. This exclusionary or predominant lifestyle emphasis, also known as “blame the victim,” remains strongly reinforced by U.S. and international reliance on the biased and inept 1981 report on U.S. cancer mortality by U.K. epidemiologists, Drs. Richard Doll and Richard Peto; over the last three decades, Doll’s track record on prevention reveals strong pro-industry bias and conflicts of interest (Appendix VII). In the absence of any scientific data, Doll and Peto guesstimated that lifestyle factors are responsible for up to 90% of cancer mortality. This left a small balance, which they arbitrarily assigned to occupation, pollution, and “industrial products.” Strangely excluded from their 1981 guesstimates was any consideration of mortality for people over the age of 65 and for Black Americans, just those groups among whom cancer disproportionately impacts; also excluded was any consideration of cancer incidence.

Further excluded was recognition of the substantial evidence that a wide range of occupational carcinogens are major causes of many cancers, particularly lung; there is also clear evidence of additive or synergistic interactions between carcinogenic occupational exposures and smoking. Moreover, “non-smoking attributable” exposures, occupational and air pollution, are responsible for about 20% of lung cancers. Nevertheless, NCI and ACS continue to direct minimal research and emphasis on occupational and environmental causes of cancer, in spite of substantial data relating them to the escalating incidence of overall and site-specific cancers.

APPENDIX IV: PREVENTABLE CAUSES OF CANCER

FROM THE BOOK “STOP CANCER BEFORE IT STARTS, BY SAM EPSTEIN, UIC SCHOOL OF PUBLIC HEALTH:

The American Cancer Society track record on primary prevention. In the book, “Stop Cancer Before It Starts, by Sam Epstein:

- *In 1971, when studies unequivocally proved that diethylstilbestrol (DES) caused vaginal cancers in teenaged daughters of women administered the drug during pregnancy, the ACS refused an invitation to testify at Congressional hearings requiring the FDA to ban its use as a growth promoting hormone for cattle in feedlots.*
- *In 1977 and 1978, the ACS opposed regulations proposed for black or dark brown hair coloring products, containing coal tar dyes known to cause breast and liver cancer in rodents, in spite of evidence of human risk.*
- *In 1977, the ACS called for a Congressional moratorium on the FDA’s proposed ban on saccharin, and even advocated its use by nursing mothers and babies in “moderation,” despite clear-cut evidence of its carcinogenicity in rodents.*
- *In 1978, Tony Mazzocchi, then senior representative of the Oil, Chemical and Atomic Workers International Union, stated at a Washington, D.C. roundtable between public interest groups and high-ranking ACS officials: “Occupational safety standards have received no support from the ACS.”*
- *In 1978, Congressman Paul Rogers censured the ACS for doing “too little, too late” in failing to support the Clean Air Act.*
- *In 1982, the ACS adopted a highly restrictive cancer policy that insisted on unequivocal epidemiological evidence of carcinogenicity before taking any position on public health hazards. Accordingly, the ACS still trivializes or rejects evidence of carcinogenicity in experimental animals, and has actively campaigned against laws (the 1958 Delaney Law, for instance) that ban deliberate addition to food of any amount of any additive shown to cause cancer in either animals or humans.*
- *In 1984, the ACS created the October National Breast Cancer Awareness Month, funded and promoted by Zeneca, an offshoot of the U.K. Imperial Chemical Industry, a major manufacturer of petrochemical products. The ACS leads women to believe that mammography is their best hope against breast cancer. A recent ACS advertisement promised that “early detection results in a cure nearly 100% of the time.” Responding to questions from a journalist, an ACS communications director admitted: “The ad is based on a study. When you make an advertisement, you just say what you can to get women in the door. You exaggerate a point. Mammography today is a lucrative [and] highly competitive business.” Even more seriously, the Awareness Month publications and advertisements studiously avoid any reference to the wealth of information on avoidable causes and prevention of breast cancer.*
- *In 1992, the ACS supported a statement by the Chlorine Institute defending the continued global use of organochlorine pesticides—despite clear evidence of their persistence and carcinogenicity. Society Vice President Clark Heath, M.D., dismissed evidence of*

this risk as “preliminary and mostly based on weak and indirect associations.”

- *In 1992, the ACS launched the breast cancer “chemoprevention” program, in conjunction with the NCI, aimed at recruiting 16,000 healthy women at supposedly “high risk,” into a 5-year clinical trial with the highly profitable drug Tamoxifen, manufactured by Zeneca. Evidence of the claimed effectiveness of Tamoxifen is, at best, arguable. Furthermore, evidence of the drug’s life-threatening adverse effects in healthy women is trivialized. More seriously, information that Tamoxifen poses grave risks of liver cancer, as it is a highly potent liver carcinogen in rats in whom it also induces irreversible DNA adducts, remains undisclosed to women recruited into clinical trials.*
- *In 1993, The Chronicle of Philanthropy, the nation’s leading charity watchdog, warned that the ACS is “more interested in accumulating wealth than in saving lives.”*
- *In 1993, just before PBS aired the Frontline special entitled, “In Our Children’s Food,” the ACS came out in support of the pesticide industry. In a damage-control memorandum, sent to some 48 regional divisions and their 3,000 local offices, the ACS trivialized pesticides as a cause of childhood cancer. ACS also reassured the public that food contaminated with residues of carcinogenic pesticides is safe, even for babies. When the media and concerned citizens called ACS, they then received reassurances crafted by Porter-Novelli, a powerful PR firm for the agribusiness industry, and then rehashed and sent to another client, the ACS: “The primary health hazards of pesticides are from direct contact with the chemicals at potentially high doses, for example, farm workers who apply the chemicals and work in the fields after the pesticides have been applied, and people living near aerially sprayed fields. The American Cancer Society believes that the benefits of a balanced diet rich in fruits and vegetables far outweigh the largely theoretical risks posed by occasional, very low pesticide residue levels in foods.” In support of this ACS-agribusiness initiative, these reassurances were then rehashed for a third time by the right-wing group, Accuracy in Media (AIM), which published quotes from the ACS memorandum in an article with the banner headline: “Junk Science on PBS,” with an opening, “Can we afford the Public Broadcasting Services?”*
- *In February 1994, the ACS published a study designed to reassure women on the safety of dark permanent hair dyes and trivialize risks of fatal and non-fatal cancers, as documented in over six prior reports. However, the ACS study was based on a group of some 1,100 women with an initial age of 56 who were followed for seven years only. The ACS concluded that “women using permanent hair dyes are not generally at increased risk of fatal cancer.” However, risks of cancer in women over 63 are up to 20 times higher for non-Hodgkin’s lymphoma and multiple myeloma, 34 times for bladder cancer, and 8 times for breast cancer. As designed, the ACS study would have missed the great majority of these cancers, and excluded dark hair dyes as important risks of avoidable cancers.*
- *In September 1996, the ACS together with patient and physician organizations, filed a “citizen’s petition” to pressure FDA to ease restrictions on access to silicone gel breast implants. What the ACS did not disclose was that the gel in these implants had clearly been shown to induce cancer in several industry rodent studies, and that these implants were also contaminated with other potent carcinogens, notably ethylene oxide and crystalline silica.*
- *In 1998, ACS allocated \$330,000, under 0.1% of its \$678 million revenues, to research on Environmental Carcinogenesis, while claiming allocations of \$2.6 million, 0.4% of its revenues. Furthermore, in its annual publication, Cancer Facts & Figures, designed to provide the public and medical profession with “basic facts” on cancer, other than information on incidence, mortality and treatment, there was little or no mention of primary prevention. For breast cancer, ACS stated: “Since women may not be able to alter their personal risks factors, the best opportunity for reducing mortality is through early detection.”*
- *In May 1999, the ACS issued a statement trivializing cancer risks from consumption of genetically engineered, rBGH/BST, milk containing high levels of the growth factor IGF-1. This reassurance was in striking contrast to substantial published scientific*

evidence that elevation in blood levels of IGF-1 are strongly associated with excess risks of breast, colon and prostate cancers.

- *In the January 21, 2000, Cancer Letter, commenting on the ACS behind the scenes creation of a Legislative Committee to gain major control of national cancer policy, Dr. John Durant, former executive President of the American Society of Clinical Oncologists, charged: "It has always seemed to me that was an issue of control by the ACS over the cancer agenda. They are protecting their own fundraising capacity . . ." from competition by survivor groups.*
- *In the January 28, 2000, Cancer Letter, it was revealed that the ACS had close ties to the tobacco industry. Shandwick International, representing R.J. Reynolds Holdings, and Edelman, representing Brown & Williamson Tobacco Company, have been major PR firms for the ACS in its attempts to rewrite the 1971 National Cancer Act, and in conducting voter education programs in the past presidential campaign.*
- *In the ACS Cancer Facts and Figures 2002, the Community Cancer Control Section includes a "Look Good ... Feel Better" program to teach women cancer patients beauty techniques to help restore their appearance and self-image during chemotherapy and radiation treatment." This program is partnered by the National Cosmetology Association and The Cosmetic, Toiletry and Fragrance Association Foundation, which have failed to disclose the wide range of carcinogenic ingredients in toiletries and cosmetics. These trade organizations have also failed to disclose evidence of excess risks of breast and other cancers following long-term use of black or dark brown permanent and semi-permanent hair dyes. The ACS has failed to inform women of these avoidable risks.*
- *In the ACS Cancer Facts and Figures 2002, the Environmental Cancer Risk Section dismissively reassures that carcinogenic exposures from dietary pesticides, "toxic wastes in dump sites," ionizing radiation from "closely controlled" nuclear power plants, and non-ionizing radiation, are all "at such low levels that risks are negligible." There is a high probability that the ACS track record on primary prevention will be perpetuated in future policies of the NCI following the February 2002 appointment of Dr. Andrew Von Eschenbach as NCI Director; prior to this, Eschenbach was President-Elect of the ACS. Furthermore, as a condition of appointment as NCI Director, Eschenbach continued his leadership of the National Dialogue on Cancer.*

APPENDIX VII IN THE BOOK "STOP CANCER BEFORE IT STARTS, BY SAM EPSTEIN, UIC SCHOOL OF PUBLIC HEALTH:

SIR RICHARD DOLL'S PRO-INDUSTRY TRACK RECORD ON PREVENTION

In 1954, together with Dr. Bradford Hill, Doll warned that, besides smoking, exposure to nickel, asbestos, gas production tars, and radioactivity were major causes of cancer (12). In 1955, Doll published a landmark report warning of high cancer rates in asbestos workers (51). In 1967, in the prestigious Rock Carling Fellowship lecture, Doll further warned that an "immense" number of substances were known to cause cancer, and that prevention of cancer was a better strategy than cure (52). In the late sixties, Doll could have even been considered a radical. However, over subsequent decades, Doll drastically changed his views, and gradually emerged as a major defender of corporate industry interests. This role, still virtually unrecognized, has been reinforced by his key influence in U.S. and other cancer establishments worldwide. In these overlapping roles, Doll has trivialized or dismissed industrial causes of cancer, which he predominantly attributed to faulty lifestyle, particularly smoking. Furthermore, as the leading spokesman for U.K. charities, Doll has insisted that they should focus exclusively on scientific research, and not become involved in prevention research and education (12). Doll's track record speaks for itself:

- *In 1976, in spite of well-documented concerns on the risks of fluoridation of drinking water with industrial wastes (12), Doll declared that it was "unethical" not to do so (53).*

- In his 1981 report on causes of cancer mortality in the U.S. (13), in the absence of any scientific evidence, Doll trivialized the role of environmental and occupational causes of cancer. He claimed that occupation was responsible for 4% of mortality rather than at least 20%, as previously admitted by consultants to the American Industrial Health Council of the Chemical Manufacturer's Association (14).
- In 1982, as a longstanding consultant to Turner & Newall (T&N), the leading U.K. asbestos corporation, Doll gave a speech to workers at one of their largest plants (54). This speech was in response to a TV exposé that forced the Government to reduce occupational exposure limits to an allegedly low level (1f/cc). Doll reassured the workers that the new exposure limit would reduce their lifetime risk of dying from occupational cancer to "a pretty outside chance" of 1 in 40 (2.5%). This, however, is an extremely high risk. Doll also declined to testify on behalf of dying plaintiffs or their bereaved families in civil litigation against asbestos industries. Furthermore, Doll filed a sworn statement in U.S. courts in support of T & N (54).
- In 1983, in support of U.S. and U.K. petrochemical companies, Doll claimed that lead in petroleum vehicle exhaust was not correlated with increased blood lead levels and learning disabilities in children (55). Doll's research had been generously funded by General Motors.
- In 1985, The U.K. Society for the Prevention of Asbestos and Industrial Disease (SPAID) criticized Doll for manipulating scientific information in order to assure that only 1/100,000 people working in an office containing undamaged asbestos risked disease and death (56).
- In 1985, Doll wrote to the judge of an Australian Royal Commission, investigating claims of veterans who had developed cancer following exposure to the herbicide Agent Orange in Vietnam, in strong support of the defense claims of its major manufacturer, Monsanto. He stated that, "TCDD (dioxin), which has been postulated to be a dangerous contaminant of the herbicide, is at the most, only weakly and inconsistently carcinogenic in animal experiments" (57). In fact, dioxin is the most potent known tested carcinogen, apart from confirmatory epidemiological evidence. Doll's defense, resulting in denial of the veterans' claims, was publicized by Monsanto in full-page advertisements in worldwide major newspapers.
- In 1987, Doll dismissed evidence of childhood leukemia clusters near 15 U.K. nuclear power plants (58). Faced with evidence of a 21% excess of lymphoid leukemia in children and young adults living within ten miles of these plants, Doll advanced the novel hypothesis that "over clean" homes of nuclear workers rendered their children **susceptible to unidentified leukemia viruses** (59)!
- In 1988, Doll claimed that the excess mortality from leukemia and multiple myeloma among servicemen exposed to radiation from atom bomb tests was a "statistical quirk" (60). Doll revisited this study in 1993 and eliminated the majority of cases which developed within two years of exposure, claiming that such short latency disproved any possible causal relation (61).
- In a 1988 review, on behalf of the U.S. Chemical Manufacturer's Association, Doll claimed that there was no significant evidence relating occupational exposure to vinyl chloride and brain cancer (62). However, this claim was based on an aggregation of several studies, in some of which the evidence for such association was statistically significant.
- In a 1992 letter to a major U.K. newspaper, Doll pleaded the public to trust industry and scientists and to ignore warnings by the "large and powerful anti-science mafia" of risks from dietary residues of carcinogenic pesticides (63).
- In January 2000 depositions, Doll admitted to donations from Dow Chemical to Green College, Oxford, where he had been the presidential "Warden" (64). He also admitted that the largest "charitable" donation (£50,000) came from Turner & Newall, U.K.'s leading asbestos multinational corporation, "in recognition of all the work I had done for them." In spite of this explicit record of

pro-industry bias, Doll has recently attempted to challenge charges which have “impugned my scientific independence” (65). Doll’s long-standing domination of U.K. cancer charities (66) and government policy is exemplified by a 1999 letter (to the author) from the Ministry of Health stating that, based on Doll’s 1981 report (11), “relatively little of the cancer burden (5-10%) is attributed to occupational, environmental or consumer exposure to specific chemicals” (67). Faced with growing evidence of the scientific untenability of his virtual dismissal of causes of cancer other than smoking and lifestyle, coupled with highly damaging revelations of conflicts of interest, Doll has suddenly retracted his long-standing dismissal of environmental causes of cancer. As a member of a recent IARC scientific working group, convened to review evidence relating tobacco smoking and cancer, Doll finally admitted: “It does look as if it’s the cancers that are principally caused by hormones that are not affected by smoking. Most of the other cancers throughout the body are induced by exposure to chemicals, often environmental ones” (68). This retraction, countless cases of avoidable cancers and deaths, have been ignored by cancer establishments worldwide.

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