AIDS is proving fantastically beneficial to cancer researchers. In this e-book the author proposes that these benefits are not coincidental and reveals that cancer researchers not only created animal immunosuppressive viruses to do exactly what HIV does but even modified them for growth in human cell cultures, shortly before the epidemic began.

Moreover, published human experiments with deadly monkey cancer viruses are exposed and proposed as smaller-scale versions of the cancer experiment which culminated in the useful epidemic of immunosuppression and cancer associated with HIV. The surprising manner in which this catastrophic epidemic is systematically fulfilling the long-term goals of the cancer vaccine establishment—the very group that invented immunosuppressive viruses—is also revealed.
For more information on how cancer research is related to AIDS and the Gulf War Syndrome, please see the following site

**AIDS: The “Perfect” Disease**

at

http://www.authorhouse.com/BookStore/

In this study the author argues that AIDS and the associated epidemic of cancer are caused by a type of virus specifically developed and tested by cancer researchers (prior to AIDS) to increase susceptibility to cancer. Moreover, it is proposed that AIDS is the culmination of increasingly sophisticated experiments which were conducted intentionally in animals and humans to induce cancer as a means of developing and testing cancer vaccines. An eye-opening series of published experiments, including one in which immunosuppression was exploited to cause sarcoma tumors in human subjects injected with monkey cancer viruses, is revealed as a precedent for the experiment proposed to explain the explosion of immunosuppression and useful sarcomas in AIDS victims.

Evidence is also presented to support the thesis that the AIDS epidemic was deliberately started as an exercise in biowarfare conducted under the cover of an international human cancer experiment. This would explain why the HIV epidemic is selectively depopulating regions of the globe that the national security establishment had targeted for depopulation in the 1970s as a means of maintaining access to resources in the developing world. Additionally, the “cancer vaccine experiment” theory of the origin of AIDS could also explain not only why the AIDS virus selectively depopulates the exact components of the immune system that cancer researchers had been targeting for decades prior to AIDS but why homosexual populations are disproportionately infected by HIV and rare cancers. The relationship between AIDS research and the Gulf War Syndrome are also reviewed in this work.
Why AIDS Was Invented

Jerry Leonard

“As disastrous as the spread of HIV is, the insights that the AIDS epidemic provides into the causes of cancer may ultimately lead to new and successful approaches to cancer prevention.”

On the heels of one of medical science’s greatest triumphs—the vaccine against polio—researchers pursued the ultimate goal: a vaccine against human cancers. Unfortunately, unlike the case with the poliovirus, there were no known cancer viruses with which to make or test human cancer vaccines. Therefore, researchers engaged in a crash government program to isolate and characterize animal and human viruses capable of causing human cancer so they could manipulate them into serving as vaccines.

In this long-range human cancer vaccine research program, scientists consistently followed the path that had already led to cancer vaccines in animals. In the effort with animal cancer viruses, tumor cells were extracted from chemically or radiation-induced tumors and turned into so-called “cell-free filtrates.” These filtrates were then injected into other animals to see if the tumors could be “transplanted.” If transplanted tumors successfully grew, the researchers assumed that these injections had contained cancer viruses (since all other cells had been filtered out).

As part of this effort, researchers injected these “synthetic” cancer viruses into experimental animals with differing immune system health and genetic backgrounds. This allowed them to observe how viral cancer growth was controlled by the immune response under different host conditions. After watching how the immune system reacted to the injected cancer viruses, researchers were able to develop procedures for artificially

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2 This program was called the Special Virus Cancer Program. The goals of this massive government research effort were outlined in its 1967 Progress Report:

“The main objectives of this Program are: (1) to determine whether viruses comparable to those now known to be associated with avian and murine leukemia are etiological agents of human leukemia, and (2) to develop an effective vaccine or other means for the prevention and/or control of human leukemia and lymphoma when such etiological agents are found.”

boosting the immune system reactions they observed in the form of vaccines. By manipulating into vaccines the cancer viruses they had created, researchers now had a laboratory model for the creation and prevention of viral cancer in animals—they could give lab animals combinations of the experimental cancer viruses and vaccines (based on these or other viruses) they had created to determine the effectiveness of the cancer vaccines.

In order to replicate this successful research technique in humans, researchers needed a way to induce human cancer on demand. This would allow researchers to observe cancer growth from its earliest stages as well as develop and test potential human cancer vaccines. As a means of reaching this lofty goal, virologists engaged in a long-running research effort to isolate human cancer viruses from cancer patients using procedures developed in animal research. The cancer-causing potential of these viruses was tested by injecting them in lab animals.

In some cases researchers went even further and conducted barbaric human “cancer transplant” experiments in which an array of human and animal cancer cells and viruses were isolated, combined and injected in human subjects to induce and measure tumor growth directly. This gave researchers the opportunity not only to monitor carefully the critical transition from health to cancer growth in human subjects, but also to look for natural immune responses to the initial challenge with cancer which might be artificially amplified with vaccines.

A similar procedure had been used in vaccine research for hepatitis (an alleged cancer-causing virus in humans). In this case mentally retarded children were injected with hepatitis so that vaccine researchers could watch the disease progress from its earliest

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4 Virologists were anxious to extend the cancer vaccine procedures successfully developed for use in animals to man: “It has already been shown that a vaccine prepared against one particular virus will immunize mice not only against that virus but against others as well. This is important because it shows that (a) animal leukemia can, in fact, be prevented with vaccines and that the causative viruses from which the vaccines were prepared can be attenuated and/or killed by procedures already established for the influenza, polio, and measles diseases of man… This approach may make possible more effective control of the human disease than that attained by drug therapy alone.” Special Virus-Leukemia Program, Progress Report #4, p. 14.

5 Cancer researchers have long coveted the ability to watch cancer grow from its earliest stages in a large number of human subjects. To this end, human victims of naturally occurring immunosuppressive disorders have been the subjects of numerous monitoring programs in which the relationship between immune system health and cancer could be correlated. By watching people predisposed to cancer, researchers could eliminate the drawbacks of epidemiological studies, in which such correlations were typically made after cancer had already been manifested. R. A. Good, “Relations between Immunity and Malignancy,” Proc. Nat. Acad. Sci. USA, vol. 69, no. 4, April 1972, 1026-1032.

6 Scientists affiliated with the government’s cancer virus program correctly predicted that deadly human cancer viruses would be created using the same techniques by which animal viruses had been isolated and made more potent: “Experience with animal tumor viruses suggests that it may be possible to establish and enhance the disease-inducing potency of candidate particulates from human neoplasma by use of virus passage, concentration and purification procedures. As with murine and avian tumor viruses it must be anticipated that increasing potency of candidate viruses from human and other animal neoplasms will confer on these viruses…” Special Virus-Leukemia Program, Summary Progress Report for the Period September 1964 through June 1965, p. 9. [emphasis added]
Noting the high incidence of hepatitis among the residents of the school, nearly all of whom were profoundly mentally impaired children and adolescents, Krugman and his colleagues injected some of them with a mild form of hepatitis serum. The researchers justified their work on the grounds that the subjects probably would have become infected anyway, and they hoped to find a prophylaxis for the virus by studying it from the earliest stages of infection.\(^7\) [emphasis added]

Human experiments involving vaccines against hepatitis would continue for years to come and can be tied directly to the outbreak of AIDS in homosexuals chosen for experimental hepatitis vaccine trials shortly before they began dying of previously rare cancers. Vaccines against hepatitis would also later be used in an ambitious international human cancer vaccine experiment (recently declared a success), which will be discussed shortly.

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Unfortunately, clues to the hypothesized naturally occurring human immune response to cancer, which might be harnessed for vaccines, proved elusive. Therefore, an indirect way of determining which immune system components were critical in fighting off human cancer was investigated. This effort—which was similar to the approach used in human hepatitis research and animal cancer research—consisted of injecting cancer cells in patients with various immune system deficiencies. The hope was that if cancer cells grew more consistently in patients with immune system components that were damaged in specific ways, then these components were the keys to fighting off cancer in healthy patients. Follow-up efforts that focused on strengthening these identified components might be the key to training the human immune system to prevent cancer with vaccines.\(^8\) Thus, it was hoped that if researchers traveled the path through “immunosuppression,” it would lead to the ultimate goal of cancer “immunocompetence” in the form of vaccines.\(^9\)

In the 1950s researchers began to use this procedure systematically in humans in the same manner it had been used in lab animals. For example, researchers deliberately

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8 One author stated the goal: “Here, then, we have a wide possibility: if there is such a biological mechanism as a defense against cancer, then it may be possible to stimulate it either before cancer strikes or perhaps even later when the cancer has taken hold.” Earl Ubell, “Injecting Cancer Cells—the Case for the Defense,” *New York Herald Tribune*, January 26, 1964, 29.

9 It was in order to generate organisms with various immune system defects for these studies that numerous means of inducing immune system deficiencies (or immunodeficiencies), including the use of viruses designed for this purpose, were eventually developed.
induced tumors in human subjects with compromised immune systems by injecting them with cancer cells thought to contain human cancer viruses. Clues were sought as to the immune system deficiencies responsible for increased tumor transplant growth by correlating tumor rejection rates with other measures of immune system health.

Chester Southam was a leading researcher who published numerous papers describing the use of this technique in human subjects. Southam described his methodology as follows:

The procedure, as I explained, requires simply the hypodermic injection of a suspension of tissue-cultured cells at two sites on the anterior thigh or arm and observation of the sites at about weekly intervals for six weeks or until regression is complete. These cells are of two or more cancer cell lines. These cancer cell lines were chosen because they have the necessary growth capacity to produce a measurable reaction.\[emphasis added\]

Southam injected hundreds of healthy and diseased patients\[11\] with “cancer transplants” so he could measure the tumor growth and correlate it with the immune system health of the cancer transplant recipient.\[12\] One of the results of Southam’s dangerous tests was that the tumors he transplanted in healthy human subjects seemed to regress faster with repeated implants while the implants in cancer patients showed less favorable results:

In brief, normal recipients responded to implanted human cancer cells with a marked local inflammatory reaction and rapid complete regression of the implants within a maximum period of 3 to 4 weeks. . . In striking contrast, however, those recipients that had advanced cancer showed little or no acute inflammatory response, the implanted cancer cells grew progressively for a period of 3 weeks or more before regression started, and some individuals failed to reject implanted cancer cells over periods of observation between 6 weeks and 6 months.\[13\] [emphasis added]

Southam assumed that the different reactions to cancer injections were due to the differences in the test subjects’ immune systems.\[14\] After watching how these tumor transplants grew in healthy patients, patients with cancer, and patients with diseases other

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11 Southam summarized this procedure: “During the past 10 years, as we have seen, cancer cells have been implanted in almost 600 well persons and cancer patients. . . . The technique for measuring immune reactions is now standardized and the results are predictable.”
12 Southam explained, “Other than the two hypodermic injections and observation of the reaction, the only other procedure would be drawing serum for study of antibody reactions to the transplanted [cancer] cells at approximately two-week intervals during the observation period.”
than cancer (at the Jewish Chronic Disease Hospital), Southam claimed cancer patients had an immune system defect that contributed to cancer growth:

All the patients in the study undertaken at the Jewish Chronic Disease Hospital rejected the transplants as promptly as did the healthy persons. Thus it has been demonstrated that cancer patients lack an immune mechanism present in other individuals, including chronically diseased patients. [emphasis added]

More specifically, Southam postulated that the reason the cancer patients were more susceptible to tumor growth was due to defective cellular-based immune reactions. Southam also assumed that the reason healthy patients seemed to reject the repeated tumor cell implants with greater efficiency was that they had a natural mechanism for preventing cancer that he was stimulating through injections of controlled amounts of cancer—resulting in an induced immunity. Southam wrote:

The growth of a repeat implant of the same cell type has been studied in normal recipients. The repeated implants formed smaller nodules and regressed more rapidly as judged by gross and microscopic examination. This accelerated rejection of a second implant is presumably the result of an induced immunity. [emphasis added]

Southam hoped that the defense mechanisms against cancer he claimed to be monitoring in these studies might be further manipulated to control cancer growth:

The studies also demonstrated a correlation between the rate of rejection of homotransplanted cancer cells and the patient’s apparent ability to restrain his own disease, thus providing additional direct evidence that patients may have immunological (defense) mechanisms to restrain their own cancer. These results, of course, give hope that, through further clinical research, methods of stimulating such mechanisms to greater efficacy can be developed. [emphasis added]

The question was how to make such human cancer vaccines. One of the goals of these tumor transplant studies was to identify potential human cancer viruses in the tumor transplant cells so that these viruses could be tested as vaccines. Chester Southam summarized, “If and when a causative virus of human cancers is found, it may provide a source of antigen for cancer prophylaxis, but this is merely a hope for the future.”

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15 “The immunologic derangement responsible for the comparative slowness of rejection in patients with cancer is still unidentified, but the search is narrowing down and an impairment of cell-associated immune mechanisms now seems probable.”
16 Ibid.; C. M. Southam, et al.
17 Katz, 37.
While Southam and other researchers were able to induce tumor growth in human subjects using human tumor transplants,\textsuperscript{19} they were not able to identify human cancer viruses in these studies.\textsuperscript{20} Researchers had considered the possibility that animal cancer viruses that had acquired the ability to jump the species barrier—like the flu virus—might be responsible for human cancer. If it were true that animal cancer viruses could cause human cancer, then existing animal cancer vaccines might be directly applicable to humans.

As a means of examining this intriguing hypothesis, researchers began to study how cancer viruses from one species could cause cancer in other species. Cancer researchers also began to join cancer viruses from various species \textit{and grow them in human cell cultures}.\textsuperscript{21} They also began to study how animal cancer viruses might cause cancer directly in humans—\textit{by injecting human subjects with the animal cancer viruses} they grew in cell cultures.

In one of these tests, researchers used a procedure similar to the one that Southam and his colleagues had used in human guinea pigs. However, in addition to injecting subjects with human cancer cell growths (such as the cancer cell line known as HeLa), they also injected a monkey cancer virus known as SV40.

\textsuperscript{19} It was determined after much of this type of human cancer transplant experimentation had been completed, that Southam and his colleagues had conducted many of his experiments on cancer patients at Sloan-Kettering and the Jewish Chronic Disease Hospital in Brooklyn \textit{without their consent}. The prestigious journal \textit{Science} published a scathing review of the controversy which ensued (along with an inset of the Nuremberg Code) in which it was revealed that many of the patients involved in the cancer transplant experiments were told only that they would be involved in "skin tests" or that they were to receive "some cells." (Some of these patients were senile and some didn’t even speak English.) As the Attorney General of the State of New York noted during an investigation into Southam’s experiments in 1964/65: "An analysis of the patients selected amply illustrates that a substantial number of them had not sufficient mental or physical ability to comprehend what was being told to them or what was being done to them; and those who may have had the capacity to understand were not given the full and true nature of the experiment.” Louis J. Lefkowitz, Attorney General of the State of New York, Petitioner’s Post-Hearing Memorandum, published in: Jay Katz, \textit{Experimentation with Human Beings}, (N.Y., New York, Russel Sage Foundation, 1972), 45; E. Langer, “Human Experimentation: Cancer Studies at Sloan-Kettering Stir Public Debate on Medical Ethics,” \textit{Science}, vol. 143, 551-553.

\textsuperscript{20} The \textit{New York Times} summarized the overall situation in 1975 as follows: “[M]ore than 100 different viruses have been proved capable of causing some kind of cancers in some animal species under some circumstances. Many viruses have been suggested as possible causative factors of some cancers in man, but no such case has been proved to date.” Harold M. Schmeck Jr., “Scientists Find Virus Linked to Human Leukemia; Diagnostic Value Seen,” \textit{New York Times}, January 9, 1975.

\textsuperscript{21} One example is particularly noteworthy. In this example a rat sarcoma virus (Kirsten sarcoma virus) adapted for human cell growth was combined with a \textit{noncancerous} baboon virus (a virus known as M7). This pseudotype murine/baboon combination virus proved to have extraordinary characteristics. In addition to being capable of infecting human cell cultures, this pseudotype virus was capable of inducing tumors in a wide range of primates including dogs and monkeys. Since the virus proved so infectious, researchers thought it would readily cause cancer in man. S. S. Kalter and R. L. Heberling, “Primate Endogenous Viruses: Their Role in Oncogenesis and as Biohazards,” Joint WHO/IABS Symposium on the standardization of cell substrates for the production of virus vaccines, Geneva, December 1976. \textit{Develop. biol. Standard.}, vol. 37, 219-228.
In a twenty-page paper in the *Journal of the National Cancer Institute*, researchers published table after table of the results of their attempts to induce tumors with a monkey virus in human subjects (cancer patients) of various ages and in various disease states. They began by using separate injections of human cancer cells and animal cancer viruses in different regions of the same subject’s body. Because initially only the human cancer cell implants resulted in tumors while the SV40 implants did not, researchers began to investigate systematically the mechanism by which the monkey virus SV40 could be made to cause human tumors. Experimental parameters investigated included: the length of time the SV40 was cultured in human cells, the type of human cancer cells the SV40 cancer virus was cultured with, the length of time the recipient subject had been on immunosuppressive drugs, and the dose of cancer virus cells injected.\(^{22}\)

The experimentation with human subjects using the human cancer cell/monkey cancer virus cultures eventually paid off. Researchers found that by using the right mixture of human cancer cells and the SV40 virus (cultured for the proper length of time and given in sufficient quantities), the SV40 monkey cancer virus could not only reproducibly cause tumors (referred to as “nodules”) in human subjects but could also be retrieved in cells removed from induced cancer growths.\(^{23}\) The authors summarized:

> The formation of nodules after . . . implantation of cells from simian virus 40 (SV40) transformed human tissue culture could be correlated with the stages of the transformation process, number of cells implanted, and possibly the presence of infectious virus in the implants.

While initially the researchers had more difficulty inducing human tumors with the SV40 virus than they did with just human cancer cells (such as HeLa cancer cells), eventually they found the SV40 monkey virus, when mixed and cultured properly, could produce human tumors (characterized as sarcomas) with comparable ease:

> Judging by the results of homologous implantation, HeLa cells and late passage, virus-free SV40-transformed cells had a comparable neoplastic potential. Histologically, nodules produced by SV40-transformed human cells were sarcomas.

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\(^{22}\) The caption of Table 5 read: “Growth of nodules after homologous implantations of SV40-transformed cultures related to cell dosage and length of time spent in tissue culture after exposure to SV40.” The subheading read: “Ratios represent number of sites showing growth of nodules over number of sites inoculated.” F. Jensen, H. Koprowski, J. S. Pagano, J. Ponten, and R. G. Ravdin, “Autologous and Homologous Implantation of Human Cells Transformed In Vitro by Simian Virus 40,” *Journal of the National Cancer Institute*, vol. 32, no. 4, April 1964, 922-937.

\(^{23}\) In this manner researchers attempted to verify experimentally that the SV40 monkey virus fulfilled Koch’s postulates in human cancer growth. This meant that for an agent to be responsible for a disease, 1) it had to be present in all cases of the disease, 2) it had to cause the disease when intentionally placed in healthy subjects, and 3) it had to be present in the intentionally infected subject. J. Cohen, “Fulfilling Koch’s Postulates,” *Nature*, vol. 266, 1994, 1647.
Researchers now had a model for inducing cancer in humans using animal cancer viruses. This model allowed them to watch such cancers grow from the earliest stages under laboratory conditions.\(^{24}\) They also had a model for testing vaccines for this virus, which had already been tested in animals.\(^{25}\)

In yet another series of experiments of this type, but published by different authors, both human cancer patients and test monkeys were injected with a monkey tumor virus known as the Yaba virus. The Yaba virus had been tested in monkeys with the following result:

intonavenous inoculation of virus in the rhesus monkey produces widespread tumors in the subcutaneous tissues, skeletal muscle, lungs, and heart and occasionally results in the death of the monkey.\(^{26}\)

Nevertheless, the virus was injected in human test subjects so that the induced tumors and immune system reactions to the virus could be measured—hopefully identifying an immune response that might be manipulated for use in human cancer vaccines. Following these virus injections, the researchers observed that “the humans developed lesions quite similar to those of the monkeys” and that “inoculation of virus produces only local tumors in the monkey and human.”\(^{27}\)

The depraved nature of these experiments is compounded by the fact that the researchers used human beings as walking tissue cultures for the monkey tumor virus they were studying—alternately inducing human tumors through injection of the monkey virus, removing tissue from the induced tumors, re-injecting the virus removed from the human tumors induced, and finally inducing more tumors in other human subjects with the transplanted and processed virus. The authors summarized this process:

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\(^{24}\) One researcher summarized the importance of such a model in developing immunological means of fighting cancer: “We needed to know more about the earliest transforming events if we were ever to be able to make meaningful determinations of specific changes in cells which reflected their conversion to transformed and subsequently to malignant cells. This basic information about early tumor cells is essential also if we are to ever ‘unravel’ the mystery of the very complex immunologic responses which occur in the host experiencing the cancer event.” Joseph H. Coggin Jr., University of South Alabama, Annual Progress Report Contract EE-77-S-05-5601, Report Number ORO-5601-034, Period: August 1, 1978 to May 1, 1979, Project Title: Radiation and Chemical Effects on Viral Transformation and Tumor Antigen Expression.

\(^{25}\) Declassified documents from the government’s human radiation experiments reveal that researchers had developed methods for inducing “transplantation resistance” to SV40 in animals. One researcher reported, “We could reproducibly detect transplantation resistance and a significant inhibition of tumor development in animals sensitized to an intermediate level of the tumor extract and given a relatively large tumor cell challenge.” Human cells infected with SV40 were also tested as vaccines in hamsters. One report stated, “We are able to readily confirm that human cells transformed by SV40 in vitro were capable of interrupting oncogenesis in the hamsters.” Were such anticancer measures tested in humans following the development of transplantable SV40 tumors? Report titled: Induction of Transplantation Resistance with Soluble SV40 Induced Hamster TSTA, Joseph Coggin Jr., University of South Alabama College of Medicine. Declassified by the Department of Energy: Internet location: [http://tis.eh.doe.gov/ohre/](http://tis.eh.doe.gov/ohre/) (12/31/01)


\(^{27}\) Ibid., 1123-1128.
A cell-free filtrate of a monkey tumor was injected into three sites on the left forearm, and a tumor suspension into three sites on the right forearm of Patients 1, 2, and 3. Tumors developed at all sites. A single site was excised from the left forearm of Patient 2 at 17 days and from Patient 3 at 10 days. A suspension of each of these tumors was then injected into patient 5. . . . By this method, replication of the virus in the human was established.  

This experimentation in which animal viruses were passaged and tested in human subjects in vaccine research is reminiscent of the Nazi concentration camp studies with typhus. In these experiments “inmates of the camp were infected with typhus for the purpose of procuring a continuing supply of fresh blood taken from persons suffering from typhus.” Having a fresh supply of such contaminated blood allowed Nazi doctors to inject patients with disease-causing blood to test vaccines. Some of these vaccines, subsequently tested directly in humans deliberately infected with the pathogenic virus to be vaccinated against, were developed using mouse viruses. As described in the Nuremberg Trials: “Other inmates, some previously immunized and some not, were infected with typhus to demonstrate the efficacy of the vaccines.”

Active Immunosuppression

While researchers developed ways of causing cancer in animals—and extended these techniques to humans, the specific immune system dysfunctions which supposedly aided cancer growth still eluded them.

In addition to injecting cancer cells and viruses into subjects with naturally occurring immune system defects, there was another, more sophisticated way of determining which defects of the immune system resulted in increased cancer growth. This involved developing ways of deliberately inducing such defects so that the immune system damage could be correlated with cancer growth due to cancer transplants. Once again, if certain viruses damaged the immune system and resulted in increased cancer growth, researchers might assume that by boosting the damaged components through vaccines, cancer could be prevented.

The procedure for inducing controlled immunodeficiencies allowed researchers the ability to create immune system defects on demand in the laboratory and then measure the transplantability of the cancer viruses they had developed. In addition to allowing them the flexibility of not having to find animals with natural immune system defects, it also allowed them to watch cancer grow from its earliest stages under controlled conditions.

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30 Additionally, if there were naturally occurring immunosuppressive viruses that aided cancer growth in this manner, cancer might be immediately reduced by reducing exposure to such cofactors.
This procedure essentially represented a four-step process in cancer vaccine research. The first step would be to inject test subjects with both immunosuppressive agents and cancer agents. The second step would be to determine which induced immune system deficiencies resulted in greater cancer growth. The third step would be to develop means of stimulating these immune system mechanisms. The fourth step would be to test these immune system-stimulating procedures in the presence of cancer injections as a means of evaluating their effectiveness as cancer vaccines. Thus, by carefully inducing immunodeficiency, researchers might ultimately develop immunoproficiency against cancer.

By injecting animals with different immune system-damaging viruses along with different cancer viruses, researchers hoped to identify combinations of immunosuppressive and cancer viruses which might be occurring naturally to cause cancer in humans. Thus, animal immunosuppressive and cancer viruses provided a laboratory model for cancer creation that allowed researchers to mimic processes that they suspected were causing cancer naturally in man.

As part of this research, immunosuppressive viruses with properties amazingly similar to AIDS were created and tested extensively in mice throughout the 1960s.

In these experiments, researchers would inject mice with both sarcoma viruses and immunosuppressive leukemia viruses. Once the leukemia viruses damaged the immune systems of the experimental mice, researchers measured the resulting cancer susceptibility (due to the sarcoma viruses) in the leukemia virus-impaired test subjects.

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31 This branch of research is actively and successfully pursued in animal cancer research today.
32 Similarly, it had been suspected that malaria created an immunosuppressive state in humans, which aided the growth of cancer allegedly caused by the Epstein-Barr virus. To test this hypothesis, researchers injected primates with combinations of a virus capable of inducing Burkitt’s lymphoma and agents capable of inducing malaria. The 1967 report of the Special Virus-Leukemia Program noted that such studies were being conducted in monkeys: “A special project in progress involves induction of malaria in the recipient prior to inoculation with Burkitt virus.” Special Virus-Leukemia Program, Progress Report #4, p. 21. See also: D. P. Burkitt, “Etiology of Burkitt’s Lymphoma—an Alternative Hypothesis to a Vectored Virus,” J. Natn. Cancer Inst. 42, 19-28 1969; Whittle, Brown, Marsh, Greenwood, Seidelin, Tighe, Wedderburn, “T-cell Control of Epstein-Barr Virus-Infected B Cells is Lost During P. Falciparum Malaria,” Nature, vol. 312, 1984, 449-450.
33 One of these viruses, called the Duplan virus, was created by cancer researchers in 1962 by irradiating mouse leukemia viruses. According to an article published in 1989, this virus induces “a severe immunodeficiency disease with striking similarities to human AIDS.” D. C. Aziz, Z. Hanna, and P. Jolicoeur, “Severe Immunodeficiency Disease Induced by a Defective Murine Leukaemia Virus,” Nature, vol. 338, 6 April 1988, 505-508. Other researchers who tested the nearly forty-year-old virus reported, “This crude virus stock induced a severe immunodeficiency syndrome which has been designated murine acquired immunodeficiency syndrome.” M. Huang and P. Jolicoeur, “Characterization of the gag/Fusion Protein Encoded by the Defective Duplan Retrovirus Inducing Murine Acquired Immunodeficiency Syndrome,” Journal of Virology, December 1990, 5764-5772.
35 Using methods similar to those used in experiments which were conducted with mice using combinations of leukemia and other cancer viruses, experiments were also conducted with cats from the early 1970s in which attenuated or inactivated feline leukemia viruses were used to induce immunosuppression in
Since immunosuppressive viruses were able to increase cancer growth dramatically in mice and cats, researchers suspected that immunosuppressive viruses were also able to increase cancer growth in humans.\[^{36}\]

In order to gain a better understanding of the role of immunosuppression in human cancer, researchers began to conduct international studies of humans with immunodeficiency syndromes so they could watch cancer grow from its initial stages and correlate the type of cancer growth with viruses detected and the type of immune system dysfunction observed.\[^{37}\]

In parallel with cancer studies of naturally occurring immunodeficiency diseases in humans, in the 1970s cancer researchers began growing immunodeficiency viruses they had created for animal studies in human cell cultures. They also combined the monkey virus they had used to induce human sarcoma tumors with these immunosuppressive viruses.

In the 1980s an epidemic of sarcoma cancer due to an animal immunosuppressive virus began to explode in human populations and alert scientists to the impending AIDS epidemic.\[^{38}\] As it turns out, this cancer epidemic due to AIDS is proving to be highly combination with cancer virus injections. Following the induction of immunosuppression with the modified feline leukemia virus, experimental cats were found to be much more susceptible to cat cancer viruses. R. G. Olsen, E. A. Hoover, J. P. Schaller, L. E. Mathes, and L. H. Wolff, “Abrogation of Resistance to Feline Oncornavirus Disease by Immunization with Killed Feline Leukemia Virus,” *Cancer Research*, vol. 37, July 1977, 2082-2085.

\[^{36}\] Immunosuppressive drugs used in kidney transplants resulted in increased cancer rates in humans. This fact, combined with the fact that naturally occurring immunodeficiency diseases also resulted in higher rates of cancer in humans, caused researchers to postulate that the immunodeficiency model in animals would provide clues to human cancer growth and prevention.

\[^{37}\] By watching cancer grow in subjects predisposed to cancer, researchers could learn more about the initial stages of cancer growth (and the factors that caused it) than conducting epidemiological studies in subjects who had already contracted cancer. By watching only those patients known to be predisposed to cancer growth due to naturally occurring immunodeficiencies, the observation time required could be drastically shortened in crucial experiments designed to monitor tumor growth from its very beginning stages. These advantages (for naturally occurring forms of immunodeficiencies) were summarized by Joseph Fraumeni in a National Cancer Institute Monograph as follows:

> “Future attempts at identifying etiologic agents in human leukemia and lymphoma may involve the longitudinal study of individuals over a period of time that covers the critical phase of tumor induction. Such a project would be extremely difficult to carry out, perhaps impossible, if all persons had an identical risk of developing these otherwise uncommon cancers. *Epidemiologic and experimental advantages would result from the use of patients at exceptionally high risk of leukemia and lymphoma, such as those affected with certain constitutional diseases.*” [emphasis added]

Fraumeni also noted, “There are significant advantages to the use of constitutional disorders and other ‘high-risk’ conditions in laboratory and epidemiologic research on these neoplasms.” One of these advantages was that researchers could probe the immune responses of such patients and then correlate the exact form of their deficiencies with cancer growth. J. P. Fraumeni Jr., “Constitutional Disorders of Man Predisposing to Leukemia and Lymphoma,” *National Cancer Institute Monograph* 32, August 1969, 228.

\[^{38}\] Lawrence Altman wrote in the *New York Times*, “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.”
useful to researchers seeking to develop human cancer vaccines—the group that invented and tested immunosuppressive viruses for this very reason.

The Benefits of AIDS
The AIDS virus has allowed cancer researchers to make major breakthroughs—providing long-awaited proof that viruses cause sarcoma in humans and that such cancers can be controlled by the immune system (the so-called immunodeficiency theory of cancer susceptibility).

Headlines in the New York Times have announced that long-sought human cancer viruses have finally been isolated from AIDS patients suffering from the most prominent form of AIDS-related cancer called Kaposi’s sarcoma. Although it is not known for sure that the virus is totally responsible for the growth of Kaposi’s sarcoma in AIDS victims, this fortuitous development is extremely encouraging to those studying the viral causes of human cancers. Lawrence Altman, writing in the New York Times, recently predicted, “Ultimately, the new findings about the Kaposi’s sarcoma virus could help unravel many unknowns about how viruses cause cancers.”

This line of research, which at long last correlated a herpes virus with the growth of Kaposi’s sarcoma in human subjects, encouraged another group of researchers to look for other forms of cancer that might be caused by this same virus. These researchers have recently announced another major breakthrough in viral cancer research—finding a link between this same virus and a common form of blood cancer known as multiple myeloma, the second most prevalent form of blood cancer in the U.S.

How fortunate! As a direct result of the rare cancers that form in HIV victims, cancer researchers are gaining insight into the manner in which cancer viruses and viruses which cause immune system impairment interact to cause cancer. And they are able to do this

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39 Recall that researchers had been systematically searching for such human cancer viruses for over forty years. Up until the advent of the AIDS epidemic, this search had been in vain. Schmeck wrote in the New York Times in 1975: “... more than 100 different viruses have been proved capable of causing some kind of cancers in some animal species under some circumstances. Many viruses have been suggested as possible causative factors of some cancers in man, but no such case has been proved to date.” Schmeck.

40 The source of Kaposi’s sarcoma, the leading form of cancer that develops in immunosuppressed AIDS patients, has recently been traced to a type of herpes virus. Lawrence K. Altman, “Virus Linked To a Cancer Is Identified: Malignancy Is Found In Gay AIDS Patients,” New York Times, March 1, 1996.

41 Cancer researchers had long theorized that human cancers could be caused by herpes viruses. One government report summarized in 1967: “The virus or group of viruses most persistently detected and isolated from human leukemia and lymphoma materials is a Herpes-type agent.... This virus appears to be an important candidate for consideration as an etiologic agent of human cancer...” In the 1970s researchers wrote numerous papers speculating on the manner in which such viruses might cause human cancer. AIDS is providing the perfect research vehicle for proving these theories. F. Rauscher and R. Reisinger, Special Virus-Leukemia Program, Progress Report #4, p. 3.

thanks to the same type of viruses which government researchers had isolated, purified and made more virulent and infectious for this very purpose.\textsuperscript{43}

Lawrence Altman, who has been chronicling the AIDS epidemic through the \textit{New York Times}, recognized the benefits of AIDS to cancer researcher at the very beginning of the epidemic. Altman predicted that the rare cancer caused by AIDS would provide insight into the causes of other types of cancer:

\begin{quote}
The sudden appearance of the cancer, called Kaposi's sarcoma, has prompted a medical investigation that experts say could have as much scientific as public health importance because of what it may teach about determining the causes of more common types of cancer. [emphasis added]\textsuperscript{44}
\end{quote}

This prediction came true. Another researcher more recently summarized how AIDS-related cancers are providing valuable insight into the cause of cancer in general: “The etiology and mechanism of the specific cancers that are increased with HIV infection are proving highly informative for our general understanding of cancer etiology.”\textsuperscript{45}

As a result of this convenient new insight into the mechanisms of cancer growth, researchers are now predicting that the AIDS epidemic and the associated cancer epidemic will assist in the development of cancer prevention strategies. One author suggested, “The AIDS-malignancies have provided insights into the pathogenesis of neoplastic disease in general and into strategies for further therapeutic intervention.”\textsuperscript{46}

Other authors in the \textit{European Journal of Cancer} summarized the benefits of AIDS to cancer vaccine research in the following manner:

\begin{quote}
As disastrous as the spread of HIV is, the insights that the AIDS epidemic provides into the causes of cancer may ultimately lead to new and successful approaches to cancer prevention.\textsuperscript{47} [emphasis added]
\end{quote}

\textit{Indeed, the AIDS epidemic appears to be perfectly suited to providing a research vehicle to the viral cancer research effort, which invented immunosuppressive viruses to determine the links between cancer, viruses, and immunodeficiency so that cancer}

\textsuperscript{43} Herpes-type viruses were isolated from human cancers and mass-produced to determine their ability to cause cancer as far back as 1967: “The production of herpes-type virus from a culture of the African Burkitt lymphoma has been increased at Chas. Pfizer and Co., Inc. by a substantial increase in the virus content of the culture.” Suspected cancer-causing viruses were isolated from humans and injected into various species to assess their cancer-causing potential: “Materials from human patients have already been inoculated into 600 newborn monkeys and chimpanzees of various species, and into large numbers of dogs, cats, mice, hamsters, etc.” Special Virus-Leukemia Program, Progress Report #4, pp. 12, 20.
\textsuperscript{47} V. Beral, et al.
vaccines could be created. One author wrote of the utility of AIDS-related cancers or neoplasms to cancer prevention efforts:

The occurrence of these neoplasms offers an opportunity to study the role of viruses and immunodeficiency in development of these tumors. Also, the pathogenic mechanisms leading to specific malignancies can be elucidated. This information ought to guide development of strategies for prevention of virally determined cancers.48 [emphasis added]

Altman provided a fitting summary of the situation:

. . . scientists saw in the tragedy of the AIDS epidemic an extraordinary opportunity to study the interplay of viruses, an impaired immune system and the development of cancer. In a way, AIDS research was an extension of the war on cancer that the Government declared in 1971.49 [emphasis added]

It should be clear that the HIV epidemic is providing cancer researchers with extensive knowledge of human cancer viruses and the immune system reactions to them. This knowledge will certainly assist in the development of human cancer vaccines, but HIV itself may even be used in such vaccines if recent animal research is applicable to human cancer prevention. Recent cancer experiments with HIV in animals have shown that the virus can be used not only to cause cancer but to inhibit its growth in the form of cancer vaccines!

By using components of HIV in experimental vaccines against tumors (which were induced by modified forms of HIV), researchers have reportedly dissected the role of CD4 and CD8 T-cells in tumor prevention through immunosurveillance. One group of scientists reported, “This model has allowed us to begin to dissect some of the mechanisms mediating and regulating tumor immunosurveillance.” The researchers who conducted this experiment optimistically noted that this line of experimentation with HIV “may provide a successful concerted approach to cancer immunotherapy.” 50

Whether HIV will be used directly as a cancer vaccine in humans in a similar manner remains to be seen. However, HIV may have much more far-reaching medical uses than cancer vaccines—as stunning as that achievement might be. Specifically, the use of HIV in the brave new world of genetic therapy in humans looks promising.

Indeed, HIV has the potential to be a very useful experimental and therapeutic virus with broad biomedical applications. Amazingly enough, researchers have begun using the HIV virus itself as a viral “vector” for the controversial line of medicine known as gene therapy—at least in the laboratory. By deleting certain genes in the deadly AIDS virus, researchers have been able to exploit its unique infectious properties while eliminating its harmful capacity. Andrew Pollack wrote in the *New York Times*:

> In a bold but potentially frightening effort to turn one of the world’s most virulent killers into a cure, scientists and biotechnology companies are trying to tame the AIDS virus and harness it to treat disease. The scientists say they have stripped the human immunodeficiency virus of its ability to cause disease, while leaving intact its ability to infect human cells. Such a crippled virus, they say, *could be used to deliver genes in to human cells for gene therapy.* [emphasis added]

As a result of laboratory tests, the subfamily of viruses to which HIV belongs is thought to be one of those most suited for the emerging technology of genetic therapy. This type of virus, the lentivirus, seems to have infectious properties which render it superior to other viruses for the insertion of genes in human DNA. This includes the DNA of nondividing cells, which pose a barrier to other types of viral vectors. Pollack noted:

> H.I.V., on the other hand, is both cunning at evading the body’s immune defenses and can carry large genes. Most important, it is one of a small class of viruses, known as lentiviruses, that can incorporate genes into the chromosomes even of nondividing cells. [emphasis added]

This convenient infectious property, together with its carrying capacity, makes HIV one of the more promising ones for use in human clinical gene therapy trials.

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It should be clear that the cancer epidemic due to AIDS is proving fantastically beneficial to cancer researchers, but the question needs to be asked: Are these stunning developments merely due to coincidence? Altman had noted, “In a way, AIDS research was an extension of the war on cancer that the Government declared in 1971.” Is there more to Altman’s comment than meets the eye?

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51 Cancer researchers are not the only ones who are benefiting from the medical knowledge gained in the fight against AIDS. The entire medical research community stands to reap tremendously useful knowledge from the AIDS catastrophe. Several knowledgeable authors summarized in the medical literature: “Furthermore, an unforeseen dividend for HIV research is the unraveling of the rich, intricate pathways of gene regulation utilized by the virus, which may well illuminate novel, fundamental cellular processes. The understanding of basic biology that we gain from the studies of HIV may be one major legacy of this epidemic to medical science.” [emphasis added] Y. N. Vaishnav and F. Wong-Staal, “The Biochemistry of AIDS,” *Annu. Rev. Biochem*, 1991, vol. 60, 580.

Could researchers have caused the AIDS/cancer epidemic using the tools they had developed in the government’s war on cancer? Did they continue in their human experiments with cancer-causing monkey viruses and escalate to the use of “active” immunosuppression with the monkey immunosuppressive viruses they developed for animal cancer experiments?

In the human experiments with SV40 tumor transplants, researchers mimicked active immunosuppressive experiments that were conducted by Chester Southam in animals. In Southam’s animal experiments, he gave mice treatments that disabled their immune systems in an attempt to increase tumor growth due to cancer virus injections. In the SV40 experiments with humans, patients undergoing immunosuppressive treatments for cancer were injected with the cancer-causing monkey virus SV40 to determine both whether such viruses were capable of causing human cancer and whether immunosuppression aided such cancer growth.

The SV40 researchers even got to use one of the same immunosuppressive treatments (an immunosuppressive chemical known as Cytoxan) in their human transplant studies that Southam used in cancer transplant studies in mice.

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53 Southam reported, “It seemed reasonable to test this possibility by studying quantitatively the transplantability of syngeneic tumours under conditions which suppress or enhance immunological responses.” J. Reiner and C. M. Southam, “Effect of Immunosuppression on First-Generation Isotransplantation of Chemically Induced Tumours in Mice,” *Nature*, vol. 210, 23 April 1966, 429-430.

54 Although a *New York Times* reporter [Altman] recently noted, “[I]t would be unethical to inject humans with a virus to prove that it can cause cancer,” as documented in this work, scientists have conducted numerous experiments using just such a procedure. Lawrence K. Altman, “A Virus Associated With AIDS.”

55 Some of the human patients used in the monkey virus experiment were undergoing immunosuppressive treatments with Cytoxan for their cancer: “Seventy-five percent of the patients had been given cytotoxic drugs, including cyclophosphamide (Cytoxan), 5-fluorodeoxyuridine, and 5-fluorouracil in full therapeutic doses.” Researchers were able to study the injected cancer cell growth as a function of how long it had been since the immunosuppressive treatments had been halted: “[Cancer] Cells were inoculated into patients 1 to 31 days after drug administration was terminated, except for 2 patients who received their last dose of Cytoxan on the day of implantation.” F. Jensen, et al.

56 Southam explained, “Such an investigation has been undertaken in this laboratory and the results obtained with cyclophosphamide (‘Cytoxan’) are sufficiently interesting and reliable to merit this preliminary report. Cyclophosphamide (‘Cytoxan’) was chosen for this study because its immunosuppressive effect had previously been demonstrated in a variety of experimental systems and because its pharmacological characteristics seem well suited to the objective.” This chemical had the advantage that it “acts very rapidly after systemic administration to initiate cytotoxic changes, including destruction of those cells responsible for immunological reactions.” Conveniently, “if administration of the drug is stopped 24 h or more before a transplant is injected, it results in a host in which immunosuppression will continue for a considerable time, but in which there is no persistence of a cytotoxic drug which might inhibit the tumor transplant.” J. Reiner and C. M. Southam, “Effect of Immunosuppression on First-Generation Isotransplantation of Chemically Induced Tumours in Mice,” *Nature*, vol. 210, 23 April 1966, 429-430.

57 Experiments similar to these were also conducted in monkeys in which immunosuppression and infection with the SV40 virus were studied to determine the cancer-causing potential of SV40. G. A. Cole and K. V. Shah. “Experimental Simian Virus 40 Infection of Normal and Immunosuppressed Spider Monkeys,” *Acta virol*, vol. 18, 1974, 65-69.
Since cancer researchers routinely used experimental cancer-induction procedures in humans that they developed in animals, it is natural to ask whether, in addition to immunosuppressive chemicals, researchers have also used monkey immunosuppressive viruses in conjunction with SV40 or other cancer virus transplants in humans.

Curiously, monkey immunosuppressive viruses were not only available to researchers prior to AIDS but were mixed with the SV40 virus and propagated in human cell cultures!

While the public is completely ignorant of this fact, simian immunosuppressive viruses (SIVs) such as the Mason Pfizer Monkey Virus (MPMV)—one of three types of SIV—were available to researchers as early as 1970 when the virus was grown in cell cultures. Well before AIDS, MPMV was shown to induce immunodeficiency states in monkeys in the early 1970s as well as the 1980s.

In one of these early experiments, MPMV was mixed with SV40—the monkey cancer virus (which had already been injected in humans to cause cancer)—and the Rous

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60 The virus not only had immunosuppressive properties, it was reported to act like a slow virus—just like HIV. D. L. Fine, J. Landon, R. Pienta, M. Kubicek, M. Valerio, W. Loeb, and H. Chopra, “Responses of Infant Rhesus Monkeys to Inoculation With Mason-Pfizer Monkey Virus Materials,” *Journal of the National Cancer Institute*, vol. 54 no. 3, March 1975, 651-658.

61 In 1986 one group of researchers described how immunodeficiency states were induced in monkeys in the early 1970s using MPMV. Since the virus did not immediately induce cancer and since AIDS hadn’t started in human populations, the effects of the virus (immunosuppression and fatal infection by opportunistic diseases) at that time did not attract widespread notice. The researchers reported in one paper, “However the results were disappointing at that time because tumors were not induced by inoculation of MPMV into newborn rhesus monkeys and other nonhuman primates. Instead, many of the inoculated neonatal animals developed persistent lymphadenopathy, thymic atrophy, and weight loss and eventually died of undue susceptibility to facultative organisms. Because of the absence of transmissible tumor and the lack of occurrence at that time of human AIDS, this nononcogenic but immunosuppressive result attracted little scientific attention.” M. L. Bryant, M. B. Gardner, P. A. Marx, D. H. Maul, N. W. Lerche, K. G. Osborn, L. J. Lowenstein, A. Bodgen, L. O. Arthur, and E. Hunter, “Immunodeficiency in rhesus monkeys associated with the original Mason-Pfizer monkey virus,” *J. Natl. Cancer Inst.*, 77(4), October 1986, 957-965.

62 Cryogenically preserved samples of this virus, isolated in the 1970s, were shown to induce a disease (simian acquired immune deficiency syndrome, SAIDS) very much like that due to simian immunodeficiency viruses (SIVs). The researchers who published the experiment in 1986 stated, “MPMV produces an acquired immunodeficiency similar to that caused by the recently described simian acquired immune deficiency syndrome type D retroviruses.” Ibid.
sarcoma virus in human cell cultures. Researchers also mixed the immunosuppressive virus with simian sarcoma virus.

Is it not curious that cancer researchers were mixing immunosuppressive monkey viruses with sarcomas and coaxing them to grow in human cells in the late 1970s—just before HIV broke out in human populations and caused an epidemic of immunosuppression and sarcoma? Could this epidemic have been an extension of the human tumor transplant experiments with SV40, using the technology perfected over decades of research with animal immunodeficiency viruses?

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If researchers were going to use animal immunosuppressive viruses to conduct such experiments in man, how might they have been conducted? It would make sense for researchers to use vehicles that would allow them not only to inject subjects with viruses but to conduct follow-up studies to see how the immunosuppressive and cancer viruses resulted in immune system destruction and cancer growth. Vaccine programs meet these criteria and are the perfect vehicles for implementing such experiments.

Prior to AIDS, the world’s medical elite was well aware of the usefulness of immunosuppressive viruses to cancer research:

- World Health Organization (WHO) representatives had made statements expressing their desire that the organization get involved in research with immunosuppressive

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65 Such research would not be unprecedented. A long-running program exploiting unsuspecting black men naturally infected with syphilis was supervised by the government for decades. This program, known as the Tuskegee study, was conducted “to see how the syphilis germ destroys the body. In the late stages, the germ can cause paralysis, gradual blindness and dementia.” Sanjay Bhatt, “The anthrax vaccine: Government’s shot in the dark,” Palm Beach Post, December 30, 2001. In this case, natural cases of syphilis were deliberately left untreated so victims of the disease could be monitored for medical research purposes. A government report summarized the experiment: “By the mid-1940s it was becoming clear that the death rate for the infected men in the study was twice as high as for those in the control group. The study was reviewed by PHS officials and medical societies and reported by a number of journals from the early 1930s to 1970. In the 1960s a growing number of criticisms began to appear, although the study was not stopped until 1973. Thus, men with a confirmed disease were not told of their diagnosis and were deceived into participating in the study under the guise of its being therapeutic for unspecified maladies. In addition to exposing the subjects to the additional harms of participation in the study, the false belief that treatment was being administered prevented subjects from otherwise seeking medical care for their disease. Over this forty-year history, at least 28 participants died and approximately 100 more suffered blindness and insanity from untreated syphilis before the study was stopped.” Advisory Committee on Human Radiation Experiments - Final Report, Chapter 3, Section: “The Development of Human Subject Research Policy at DHEW,” http://tis.eh.doe.gov/ohre/roadmap/achre/chap3_2.html (12/31/01)
viruses capable of selectively targeting T-cells.\textsuperscript{66} (Researchers associated with WHO were optimistic that the immunosuppressive research conducted in animals could be usefully extended to human beings.\textsuperscript{67})

- Members of WHO had also recommended that experimental agents be placed in vaccines as a vehicle for measuring the human immune response on a global scale\textsuperscript{68} (as a means of separating genetic from environmental determinants of immune system function).

- A report by a WHO study group had also recommended an international study of naturally immunosuppressed humans with \textit{immunological deficiency syndromes} for cancer research purposes (this was prior to the emergence of the international \textit{Acquired Immune Deficiency Syndrome} epidemic)\textsuperscript{69}

Did WHO achieve all of these goals \textit{simultaneously} by placing immunosuppressive agents in some of its vaccines, thereby creating a highly useful international epidemic of \textit{acquired} immunodeficiency syndromes and cancer resulting from selective T-cell depletion?

The possibility that increased cancer rates due to infectious agents present in vaccines would be useful for cancer vaccine research was evident in the 1967 report of the Special Virus-Leukemia Program. Projects were initiated to \textit{correlate cancer deaths of children and veterans with vaccines they may have received} (as compared to “matched control groups”). One project recommended that a massive database be constructed and maintained by obtaining “death certificates for all U.S. children under 15 years of age who have died of cancer, 1960-1964” so that cancer deaths could correlated with names

\textsuperscript{66} The following chilling recommendation for future research was made in a World Health Organization publication from 1972: “An attempt should be made to ascertain whether viruses can in fact exert selective effects on immune function . . . or by affecting T cell function as opposed to B cell function. The possibility should also be looked into that the immune response to the virus may itself be impaired if the infecting virus damages more or less selectively the cells responding to the viral antigens.” [emphasis added] This is exactly what HIV does. A. Allison, W. Beveridge, W. Cockburn, J. East, H. Goodman, H. Koprowski, P. Lambert, J. Van Loghem, P. Miescher, C. Mims, A. Notkins, and G. Torrigiani, “Virus-Associated Immunopathology: Animal Models and Implications For Human Disease,” \textit{Bulletin of the World Health Organization}, vol. 47, no. 2, 1972, 259.

\textsuperscript{67} “Recent studies on virus-induced immunopathological reactions in domestic and experimental animals have led to the development of concepts and technical methods that may be useful in investigating certain viral diseases in man, including hepatitis.” [emphasis added] \textit{Ibid.}, 258.

\textsuperscript{68} “In relation to the immune response, \textit{a number of useful experimental approaches can be visualized. One would be a study of the relationship of HL-A type to the immune response, both humoral and cellular, to well-defined bacterial and viral antigens during preventive vaccination.”} [emphasis added] \textit{Federation Proceedings}, vol. 31, no. 3, May-June 1972, 1102.

\textsuperscript{69} The pre-AIDS WHO report recommended that an “international co-operative study of patients with \textit{immunological deficiency syndromes} should be carried out.” It also suggested that “[o]bservations on patients with immunological deficiency diseases should be as complete as possible, and it is desirable that they should enable valid comparisons to be made between patients studied in different institutions.” [emphasis added] A. C. Allison, B. A. Askonas, B. Benacerraf, R. Ceppellini, R. A. Good, E. S. Lennox, H. O. McDevitt, R. S. Nezlin, and M. Seligmann, “Genetics of the Immune Response,” \textit{World Health Organization Technical Report Series}, no. 402, 34, 52.
“of children believed to be at unusual risk of cancer; e.g., those given certain vaccines in the past.” Were similar databases created for vaccine recipients who were deliberately predisposed to cancer through vaccines?70

Unfortunately, there is evidence to support the radical hypothesis that humanitarian vaccine programs were used to conduct inhumane cancer experiments. Promiscuous homosexuals were selectively targeted with an experimental vaccine against hepatitis B beginning in the late 1970s.71 And the AIDS epidemic in promiscuous homosexuals began almost immediately after the first of these vaccine experiments.72 It has been documented that recipients of this experimental hepatitis vaccine have been disproportionately afflicted with AIDS73 and that immunosuppressed homosexuals are being targeted with numerous follow-up studies to monitor how cancer develops in immunosuppressed victims versus healthy populations.74

Interestingly enough, the suspect experimental hepatitis B vaccine given to homosexuals just prior to the AIDS epidemic appears to have been a subset of a proposed global cancer vaccine experiment administered by the World Health Organization.75 It was thought that hepatitis B was the cause of a form of cancer called hepatocellular carcinoma and that by immunizing people against hepatitis, immunity to this form of cancer might be obtained.76 Hepatitis B vaccination trials were conducted as a means of developing

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70 Special Virus-Leukemia Program, Progress Report #4, Program Staff, National Cancer Institute, Etiology Area, May 1967, pp. 29, 31.
75 The hepatitis vaccine developed in these trials has been referred to by some observers as the first human cancer vaccine.
76 Researchers affiliated with the WHO noted in one publication, “The Meeting considered that there were good grounds to suppose that vaccination of children with hepatitis B vaccine at birth, or shortly thereafter, would confer long-term protection against the development of hepatocellular carcinoma.” “Prevention of Liver Cancer,” World Health Organization Technical Report Series 691 (Geneva: World Health Organization, 1983), 27.
vaccines for use in these international hepatitis/cancer vaccine trials.\textsuperscript{77} (Hepatitis was also mentioned in WHO publications discussing the experimental merits of extending animal immunosuppressive techniques to human research.)

Given the admitted experimental nature of this cancer vaccine effort employing hepatitis vaccines, and, given the useful immunosuppression subsequently associated with one branch of it, it seems logical to ask: Was this direct attempt at cancer vaccination using the hepatitis B vaccine augmented through the addition of immunosuppressive viruses, as was done in experimental animal populations?\textsuperscript{78} In other words, was there a covert cancer vaccine experiment within the overt cancer vaccine experiment associated with the hepatitis B program? Could this explain why immunosuppression in homosexual populations is proving so beneficial to cancer research?

Such a procedure would represent the replication in human populations of a published experiment conducted in cats in which kittens were injected with a “vaccine” containing a mixture of immunosuppressive viruses and cancer agents.

The authors of this study noted:

\textbf{Four-week-old specific-pathogen-free cats were immunized with a combined vaccine composed of killed feline leukemia virus and killed feline oncornavirus-associated cell membrane antigen-containing tumor cells.}

The authors further explained that the immunosuppressive leukemia virus present in the vaccine increased susceptibility to the oncornavirus cancer agents: “\textit{Thus, it appears that the protective immunity to feline oncornavirus disease was hindered rather than enhanced by inclusion of inactivated, purified FeLV as a vaccine component.}”\textsuperscript{79}

\textit{The Source of the International AIDS Epidemic}

In addition to the AIDS outbreak in American homosexuals, there is evidence supporting the theory that the international HIV epidemic is also the result of a premeditated international “cancer vaccine” experiment implemented using existing vaccine programs associated with the World Health Organization.

While gay recipients of the hepatitis B/cancer vaccination program have been disproportionately stricken by the AIDS virus, according to a front page article in \textit{The Times of London}, it is a segment of the recipients of the international smallpox

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\textsuperscript{78} This process might allow long-term immunocompetence (through vaccine development) to be attained through short-term immunocompromise induced in a group of unfortunate human test subjects.

\textsuperscript{79} R. G. Olsen, et al.
\end{footnotesize}
vaccination administered by the WHO which have been disproportionately afflicted with AIDS in the developing world. Pearce Wright summarized in the Times:

The World Health Organization, which masterminded the 13 year campaign, is studying new scientific evidence suggesting that immunization with the smallpox vaccine Vaccinia awakened the unsuspected, dormant human immuno defence virus infection (HIV).\(^{80}\)

The Times elaborated, “The smallpox vaccine theory would account for the position of each of the seven Central African states which top the league table of most affected countries; why Brazil became the most afflicted Latin American country; and how Haiti became the route for the spread of Aids to the U.S.”

**Conclusion**

AIDS is providing a fantastic laboratory model on a large-scale—essentially duplicating research efforts conducted earlier on a smaller-scale to prove the immunodeficiency and virus model of cancer growth. For example, AIDS is providing answers that studies of immunosuppressed transplant patients and children with naturally occurring immune deficiency syndromes sought with respect to the viral nature of cancer and the breakdown of the immunological surveillance mechanism.\(^{81}\)

Indeed, the AIDS epidemic is providing a near ideal research vehicle for proving theories about cancer causation that were popular shortly before the epidemic broke out. In 1972 the *Proceedings of the National Academy of Sciences* published numerous articles speculating not only on the relationship between immunodeficiency and viruses in cancer formation, but specifically on the relationship between herpes viruses and cancers. \(^{82}\) AIDS has made international headlines apparently showing that immunodeficiencies can be a major contributor to cancer due to viruses and that one of the main forms of cancer due to such immunodeficiencies—Kaposi’s sarcoma—is due to a herpes virus.

Thus, in addition to providing evidence that the “breakdown of the immunological surveillance mechanism” can lead to cancer, AIDS is helping to identify the specific

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\(^{81}\) One author summarized the situation in 1969: “Although further analyses are obviously required, the results suggest that children with immune deficiency syndromes or adults treated with immunosuppressives have a greatly increased risk of developing tumours, especially of the lymphoreticular system. Presumably this represents breakdown of the immunological surveillance mechanism, allowing proliferation of tumour cells that would otherwise be restrained by an immune response. Whether one or more viruses are involved cannot be stated on existing evidence.” A. C. Allison, “Immune Responses to Virus-Induced Tumours,” *Proc. Roy. Soc. Med.*, vol. 62, September 1969, 956-958.

\(^{82}\) R. A. Good, “Relations Between Immunity and Malignancy.”


viruses involved in the resultant conversion to cancer. Consequently, the damage inflicted by HIV on the immunological surveillance mechanism will be useful in the development of cancer vaccines and, as is evident in animal experiments, the deadly AIDS virus itself may even be used in experimental human cancer vaccines.

In summary, it seems improbable that, by sheer chance, an unprecedented epidemic infection due to a type of virus invented by cancer researchers began benefiting cancer researchers almost immediately after they grew it in human cell cultures.

Given the immense benefits of the disease in combination with the history of human experimentation with cancer-causing viruses by the cancer research establishment, it seems more likely that AIDS is the result of human experimentation and not accident. This type of human experimentation would be directly in line with the type of cancer research perfected over decades of animal experiments with immunosuppressive viruses. It would also represent the culmination of a long series of experiments in humans in which immunosuppression was exploited as a mechanism to induce cancer by various means (including monkey sarcoma viruses) in the search for cancer vaccines.

While this conclusion may seem extreme, the open medical literature contains many surprises that run contrary to what a blind faith in the medical research establishment would foster. One can only wonder what surprises the classified records of the CIA (which had an entire wing of a hospital to experiment in as part of its MKULTRA program\(^{85}\)) and the National Cancer Institute hold. It is hoped that this brief study will provide insight into what areas should be further investigated.

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A more detailed study, titled *AIDS: THE “PERFECT” DISEASE*, which examines not only the cancer research benefits of AIDS described above, but also the benefits to the national security establishment, is available at [http://www.authorhouse.com/BookStore/](http://www.authorhouse.com/BookStore/).

In this study, disturbing links between the national cancer research establishment and the biowarfare establishment are revealed as is the manner in which AIDS is helping to fulfill long-standing national security goals related to international population control in addition to fulfilling the goals of the cancer vaccine research establishment. Additionally, the sordid history of previously secret national and international testing programs of the U.S. government is reviewed. The proposed cancer experiment to explain the AIDS epidemic is placed within the context of this backdrop of unethical human experimentation under humanitarian pretexts. These unconscionable testing programs serve as a warning to those who would doubt that an experiment of the size and scope of that proposed to explain the AIDS epidemic could and would be carried out by the U.S. government under the auspices of cancer research.

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\(^{85}\) “In another MKULTRA project, CIA secretly provided funding for the construction of a wing of Georgetown University Hospital in the 1950s so that it would have a locale to carry out clinical testing of its biological and chemical programs.” *Advisory Committee on Human Radiation Experiments*, Appendix E: [http://www.gwu.edu/~nsarchiv/radiation/dir/mstreet/interim/intret.txt](http://www.gwu.edu/~nsarchiv/radiation/dir/mstreet/interim/intret.txt) (12/31/01)