How To Make An AIDS Virus

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AIDS is proving fantastically "beneficial" to cancer researchers. Was it created and unleashed on the world using cancer vaccine research programs to verify the immunodeficiency and viral theory of cancer causation?

This article describes the well-documented methods by which animal cancer and immunodeficiency viruses like AIDS were deliberately manipulated by cancer researchers to cross the species barrier and infect human cells shortly before the AIDS-induced cancer epidemic began in human populations.

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.*

How To Make An AIDS Virus

The Technology of Pseudovirus Construction

Jerry Leonard

> . . . on the immediate horizon are modern developments in molecular genetics which could result in manmade viruses for which there would be no natural immunities and against which no reasonable defense could be mounted. . . . It is not difficult to imagine the consequences if such agents should fall into the hands of a future Hitler.[1]
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> -*Congressional Record*, 1970 (10 years before AIDS)

In 1990 the prestigious journal *Science* published a technical paper (coauthored by the infamous Robert Gallo—co-discoverer of HIV) describing in detail successful attempts at *creating* a "super" AIDS virus with greatly enhanced infectious properties.[2] This new and highly dangerous AIDS virus was deliberately engineered—through a sophisticated mixing of human and animal viruses—to infect a greater number of cell types than the "natural" AIDS virus had been capable of infecting. Additionally, this newly engineered AIDS virus was thought to be transmissible through the air.[3]

In this study it will be proposed that a procedure similar to that used to create this enhanced form of AIDS virus was used to manufacture the original cancer-causing AIDS virus in the 1970s to complement an ongoing line of dangerous experiments which employed modified monkey cancer and tumor viruses to induce tumors in man. This procedure (known as "pseudotyping") was originally developed to coax animal cancer viruses to cross the species barrier as a means of understanding causes of human cancer to benefit the development effort for human cancer vaccines.

The methods developed by the cancer research community to create these cross-species animal cancer and immunodeficiency viruses will be reviewed below followed by a discussion of how such procedures may have been used to create HIV, an animal immunosuppressive virus that suddenly gained the ability to cross the species barrier and cause an epidemic of useful cancers in humans. (The reasons immunosuppressive viruses like AIDS were developed for human cancer vaccine research well before AIDS began are discussed in greater detail in another work by the author.[4])

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[1] "Report of the Subcommittee on National Security Policy and Scientific Developments of the House Foreign Relations Committee," placed in the *Congressional Record-Senate*, June 25, 1970, 21393.

[2] P. Lusso, F. Veronese, B. Ensoli, G. Franchini, C. Jemma, S. E. DeRocco, V. S. Kalyanaraman, R. C. Gallo, "Expanded HIV-1 Cellular Tropism by Phenotypic Mixing with Murine Endogenous Retroviruses," *Science*, vol. 247, 16 February 1990, 848-852.

[3] Decades previous to this, cancer virus researchers had developed animal cancer viruses capable of inducing cancer through the air: "During the past year it was shown that highly potent laboratory strains of mouse leukemia viruses can be carried in the air and that inhalation, by mice, of contaminated air results in infection and leukemia." F. Rauscher and R. Reisinger, *Special Virus-Leukemia Program, Progress Report #4*, Program Staff, National Cancer Institute, Etiology Area, May 1967, p.15.

[4] See: *AIDS: The "Perfect" Disease* at http://www.authorhouse.com/BookStore/

Manipulating viral cancer

By "isolating" cancer-causing viruses and studying the various conditions under which these viruses could cause cancer in animals, researchers hoped to gain insight into the cause of human cancers. For example, by using techniques developed in animal cancer research, cancer researchers sought to determine whether there were *human viruses* capable of causing cancer in man as well as whether known *animal cancer viruses* themselves could cross the species line and *cause human cancers*. The motivation for these experiments was the development of a means of human cancer prevention through viral vaccination, as had been successfully accomplished for leukemia viruses in mice (for example, the Rauscher leukemia virus[5]) and cats.[6]

But before researchers could create human cancer vaccines by modifying known cancer viruses, viruses capable of causing cancer had to be discovered. How might this be done? In the course of the early animal cancer studies, methods of inducing tumors were developed so that their growth and interaction with the immune system could be routinely studied in the laboratory. In mice, tumors and leukemias were intentionally induced in various mouse strains through exposure to radiation or chemical agents. Cells from these induced tumors (or naturally occurring varieties) were typically filtered and grown in tissue cultures. These processed cells were then transplanted and grown in other mice. When cancer resulted from these transplanted cancer cells, cells from the new tumors would then be processed and cultured and the transplantation process would be repeated. (This process of refining and injecting cancer viruses was sometimes referred to as the "bio-assay" method of determining the cancer-causing potential of viruses.[7])

By filtering out everything but virus-sized particles, increasingly infectious cancer "viruses" were eventually isolated from cell lines developed through this process of serially passaging cancer cells from one animal to another. These viruses were then kept in tissue cultures and used throughout the cancer research community to evaluate the different conditions under which they could induce tumors. As part of this research, scientists measured cancer susceptibility due to these viruses as a function of simultaneous exposure to immunosuppressive treatments such as radiation, chemicals, surgeries, and co-infection with other viral agents—including immunosuppressive viruses.[8] Numerous experiments were conducted in which animal immunosuppressive

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[5] M. A. Fink, F. J. Rauscher, "Immune Reactions to Murine Leukemia Virus. I. Induction of Immunity to Infection with Virus in the Natural Host," *Journal of the National Cancer Institute*, vol. 32, no. 5, May 1964, 1075-1082.

[6] Due to the similarities of animal cancer viruses isolated from different species (including mice, cats, and primates), some researchers hypothesized that most animal and even some human cancers may have had a common origin from a single cancer virus. This hypothesis provided motivation for animal studies in several dimensions—if human cancer was found to be due to animal viruses, then some forms of human cancer might be prevented by reducing human contact with these animals *or by creating human versions of animal cancer virus vaccines*.

[7] See chapter entitled "The Search for Oncogenic Viruses in Human Tumors and Lymphomas," Ludwik Gross, *Oncogenic Viruses* (Oxford: Pergamon Press, 1983), 1003.

[8] Some of the mouse cancer viruses isolated in this manner were found to be immunosuppressive and to aid the growth of other cancer viruses. One strain of mouse cancer virus called the Duplan virus (isolated and tested decades before AIDS) has been manipulated to cause a mouse version of AIDS (called MAIDS) in mice injected with this virus. D. C. Aziz, Z. Hanna, P. Jolicoeur, "Severe Immunodeficiency Disease Induced by a Defective Murine Leukaemia Virus," *Nature*, vol. 338, 6 April 1988, 505-508; M. Huang, P.

viruses were co-injected with animal cancer viruses to determine how immune system dysfunction aided cancer virus growth. Additionally, scientists created vaccines against these cancer viruses and measured their ability to prevent cancer in animals injected with known cancer-causing viruses.

By using this method, researchers seeking to develop human cancer vaccines developed a powerful laboratory model of viral cancer in mice. Modifying cancer viruses to serve as immunosuppressive viruses or vaccines allowed them *to increase or decrease viral cancer growth on demand*. If a similar viral cancer model were developed in humans, this would provide a powerful tool in the development of human cancer vaccines—just as it did in animal research. In other works by the author, *it is argued that this animal cancer model was indeed replicated in humans merely by modifying animal cancer and immunodeficiency viruses for growth in human subjects, and that this is why AIDS and the associated epidemic of viral cancer is proving so useful to cancer researchers.*[9]

The methods by which animal cancer viruses and immunodeficiency viruses were modified for human cell growth to serve this purpose will be discussed below.

Expanding the Host-Range of Cancer Viruses

The cancer virus studies in mice were expanded to include studies of whether the mouse cancer viruses might cause cancer *when injected in other species*. In these experiments to investigate the "host-range" of mouse cancer viruses, it turned out to be difficult to use mouse cancer viruses to cause cancer in species other than mice.

Mouse viruses were not the only animal viruses used in this type of cancer virus research. Similar research was conducted with chickens and cats (cancer viruses isolated from cats included feline sarcoma and feline leukemia viruses). The *feline* cancer viruses had unique properties that were particularly intriguing for researchers seeking the cause of human cancer among animal cancer viruses. One of these intriguing properties was that, unlike the mouse sarcoma viruses, the feline sarcoma virus had a naturally wide host-range. For example, *researchers were able to use feline sarcoma viruses isolated from cats to induce tumors in dogs, rabbits, marmosets, macaques, monkeys, and sheep.* Feline leukemia viruses were also able to replicate in and infect cell cultures of *human* origin.[10]

The ability of some animal cancer viruses to cause cancer in species other than that of their origin intrigued cancer researchers. Researchers surmised that if such animal cancer viruses could naturally cross the species barrier, they might be the cause of some human cancers. If this was the case, then revolutionary human cancer vaccines might be developed in exactly the same manner that animal cancer vaccines had been developed. Thus, as a means of investigating a potential cause (and cure) of human cancer, *researchers invested much time in attempts to determine the precise mechanisms that allowed some cancer viruses to jump the species barrier*. As research progressed into

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Jolicoeur, "Characterization of the gag/Fusion Protein Encoded by the Defective Duplan Retrovirus Inducing Murine Acquired Immunodeficiency Syndrome," *Journal of Virology*, Dec. 1990, 5764-5772.

[9] This would also explain why HIV is so similar to animal immunodeficiency viruses which were created prior to the useful human epidemic.

[10] G. H. Theilen, "Feline Leukemia-Sarcoma Complex: A Model for RNA Viral Tumorigenesis," in ed. F. F. Becker, *Cancer: A Comprehensive Treatise* (New York: Plenum Press, 1975), 169-205.

the mechanism by which this cross-species viral transfer could take place, scientists ultimately invented ways of *forcing* viruses such as the mouse cancer viruses to cross the species barrier artificially and cause cancer in numerous animal hosts—just as the feline cancer viruses were capable of doing naturally.

Human cancer experiments

Unfortunately, the procedures described above were not limited to experiments with animals. ***Processes similar to those just described for inducing cancer in mice and cats were also applied in human cancer experiments.*** For example, in one line of research, cancer cells from human cancer patients were removed and grown in tissue cultures. Researchers such as Chester Southam and Alice Moore[11] then induced these human cancer cell lines to grow in human subjects (as well as animals).

In experiments similar to those conducted in animals, researchers also studied the ability of these human cancer cell lines (which were allegedly later found to contain numerous viruses[12]) to induce tumors in humans in combination with immunosuppressive treatments such as radiation and chemical treatments. For example, in some of these experiments human patients undergoing chemotherapy or radiation therapy were given cancer cell transplants (in parallel with "conditioned" rats which were given the same cells) to see how the cancer cells grew as a function of immune system manipulation.[13]

In later *cross-species human experiments*, which were similar to those conducted in mice, researchers studied the cancer-causing potential of *animal* cancer viruses in *human* subjects! In numerous experiments, monkey tumor and cancer viruses were injected into human patients to determine whether they would cause cancer and tumors (they did). These studies also included determining how immunosuppressive treatments aided cancer growth *due to animal cancer viruses*. For example, one study described how the tumor-forming effects of animal cancer viruses such as SV40 (mixed with human cancer cells) varied in human subjects undergoing immunosuppressive chemotherapy treatments.[14]

It has been argued elsewhere that, in addition to *exploiting immune system damage* from chemical and radiation treatments to enhance the growth of deliberately induced cancer in humans, researchers also used *active* immunosuppression caused by human immunosuppressive viruses in such studies—just as they did in the cancer virus experiments with mice. Such studies would allow researchers to "dissect" the human immune system and determine how it responded to cancer viruses, thus providing clues in the race to induce immunity to cancer viruses in humans.

It has also been argued that the immunosuppressive viruses (such as HIV) used to induce immunosuppressive states in these proposed human cancer experiments were

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[11] C. M. Southam, A. E. Moore, C. P. Rhoads, "Homotransplantation of Human Cell Lines," *Science*, vol. 125, 1957, 158-160.

[12] H. Gelderblom, H. Bauer, H. Ogura, R. Wigand, A. B. Fischer, "Detection of Oncornavirus-like Particles in HeLa Cells," *Int. J. Cancer*, vol. 13, 1974, 246-253.

[13] A. Koike, G. E. Moore, C. B. Mendoza, A. L. Watne, "Heterologous, Homologous, and Autologous Transplantation of Human Tumors," *Cancer*, August, vol. 16, no. 8, 1963, 1065-1071.

[14] F. Jensen, H. Koprowski, J. S. Pagano, J. Ponten, 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.

derived from animal immunosuppressive viruses which were engineered to cross the species barrier to infect humans.

Was the technology used to engineer animal *immunosuppressive* viruses to cross the species barrier and infect humans derived from the research which resulted in the creation of mouse and monkey *cancer* viruses capable of crossing the species barrier and infecting human cells and human subjects (as described above)?[15]

To understand how HIV may have been adapted from animal viruses to infect humans, the mechanisms by which mouse and monkey cancer viruses were systematically engineered to cause cancer in animals other than mice and monkeys will be reviewed in detail below. *It will then be shown that this technology was indeed used to modify animal immunosuppressive viruses for human growth just before AIDS began in human populations and it will be argued that these same mechanisms were exploited to create HIV itself from animal immunosuppressive viruses. It will then be shown that these same procedures were later used by the elite of the cancer research community to modify the HIV virus to make it even more infectious and deadly than it already was*.

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Pseudovirus Construction

During the course of the animal cancer virus transplantation research with chickens and mice, scientists discovered that sarcoma viruses were often more infectious when a *leukemia* virus from the same species was "co-injected" with the tumor-causing *sarcoma* virus.[16] It was also discovered in this type of research that by growing *sarcoma* viruses and *leukemia* viruses in the same cell culture, a hybrid virus (or combination of the two viruses) with remarkable properties could be created. The combination virus formed in these types of experiments was called a "pseudotype" virus, and the *leukemia* virus (often called the "replication competent" component) was said to "rescue" the *sarcoma* virus—thus making it more infective.[17]

The remarkable infectious properties of these artificially created cancer viruses were the result of the manner in which the leukemia virus and the sarcoma virus came together to form the new virus. It was thought that the leukemia virus provided the outer part of the hybrid virus (the virus envelope) while the sarcoma virus provided the inner material (the genetic material in the viral core) that induced disease. Since it is the envelope of the virus that determines what cells the virus can infect and since the leukemia virus provided the envelope of the new combination virus, *the infectivity of the*

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[15] The immunosuppressive *mouse* viruses developed for cancer research would most likely be unsuitable for use in a covert human cancer experiment conducted using a vaccination program as a cover. Although such human-adapted, immunosuppressive mouse viruses themselves would have been unsuitable for covert experimentation, the research techniques that were developed to create such mouse viruses would have been useful to create more suitable animal immunosuppressive viruses for use in such human experiments. Specifically, the research which was conducted in mice involving the construction of infectious, transspecies cancer viruses provides some insight into how other animal immunosuppressive viruses (such as bovine visna) may have been created and adapted for growth in human hosts.

[16] Similar findings were made with *feline* leukemia and sarcoma viruses.

[17] Researchers determined that sarcoma viruses were "defective" viruses which required the help of leukemia viruses to replicate efficiently.

new pseudovirus could be artfully manipulated by choosing the appropriate type of leukemia virus (also called the helper virus) to combine with the sarcoma virus.

Originally, in this type of pseudovirus research, *mouse* sarcoma viruses were rescued with *mouse* leukemia viruses. The resulting pseudotype (consisting of a mouse sarcoma genome core surrounded by the mouse leukemia virus envelope), when injected into other mice, was capable of infecting and causing cancer in the mice more efficiently than the sarcoma virus alone.[18] This "rescuing" process was used to create infectious mouse sarcoma virus pseudotypes using a multitude of different mouse leukemia virus strains, including Moloney, Rauscher, Friend, and Gross leukemia viruses.[19]

This research involving pseudotype viruses was also used in attempts to determine under what range of conditions these cancer virus combinations could grow in different species (that is, determining the host-range of the sarcoma virus). By precisely determining the conditions under which a given cancer virus was capable of crossing the species barrier, researchers could more effectively evaluate the merits of the hypothesis that human cancer, much like the human flu virus, was the result of a naturally occurring, cross-species transfer of an animal virus to human populations. The more that was understood about the conditions under which animal cancer viruses could combine and jump species *in laboratory conditions*, the more scientists might understand about the origins of human cancer *in nature*.[20] As a result, this would make human cancer vaccines more likely to become reality.

The research to determine the host range of animal cancer viruses naturally evolved into research designed to *force* cancer viruses to jump the species barrier. In attempts to coerce a given sarcoma virus from one species to infect the cells of another species, the basic experimental procedure used in the "same-species" cancer virus experiments was modified slightly. For example, in "cross-species" experiments with mouse cancer viruses, instead of rescuing the mouse sarcoma virus with a mouse leukemia virus, *a leukemia virus from a different species was used*.

The specific experimental procedure used to increase the host-range of established mouse cancer virus cell lines in this manner typically consisted of the following:

- Mouse sarcoma viruses were injected in hamsters, resulting in tumor growth.
- Cells from the hamster tumors induced by the mouse sarcoma virus were then "rescued" with leukemia viruses from a given species by mixing or co-cultivating the hamster tumor cells with the leukemia virus in cell cultures.
- The resultant combination or pseudotype virus in the cell culture was then injected in various test animals, including the species from which the *leukemia* virus was derived, so that the infectivity and tumor-inducing capability of the virus could be evaluated.

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[18] R. J. Huebner, "The Murine Leukemia-Sarcoma Virus Complex," *Proc. N.A.S.*, vol. 58, 1967, 839.

[19] R. J. Huebner, J. W. Hartley, W. P. Rowe, W. T. Lane, W. I. Capps, "Rescue of the Defective Genome of Moloney Sarcoma Virus from a Noninfectious Hamster Tumor and the Production of Pseudotype Sarcoma Viruses with Various Murine Leukemia Viruses," *Proc. N.A.S.* vol. 56, 1966, 1164-1169.

[20] In addition to this type of work, researchers methodically attempted to compare cancer viruses (type C viruses) isolated from numerous species including mice, fowl, cats, cows, sheep, and primates to determine if they share DNA sequences which might indicate that they had the same or a similar origin.

This very process was successfully used to form pseudotypes of *mouse* sarcoma viruses capable of crossing the species barrier to infect *cats*. In one case,

- A mouse sarcoma virus was first used to induce tumors in hamsters in the manner described above.
- Viruses in these hamster tumor cells were then *rescued using a feline leukemia virus* (instead of the mouse leukemia virus), thus forming a murine/feline hybrid or pseudotype virus consisting of a mouse genome core surrounded by a feline leukemia virus protein envelope.
- Since it was the feline leukemia virus envelope that determined the host range of the combination sarcoma/leukemia hybrid, *these mouse sarcoma viruses were then able to induce sarcomas in kittens as well as grow in feline cell cultures*.[21]

Thus, with the use of this procedure, the "species barrier" was *artificially* broken using mouse cancer viruses.

It was repeatedly verified in these types of experiments that the host-range of a given sarcoma virus could indeed be successfully increased by changing the type of leukemia virus making up the outer envelope of the pseudotype virus.[22] By using this powerful process for manufacturing pseudotype viruses, a sarcoma virus from one species could be made to infect and cause tumors in a different species by rescuing the mouse sarcoma virus with a leukemia virus derived from the different species. (In other words, the process allowed the foreign viral genetic material derived from species A and placed within a leukemia virus envelope of species B to infect species B and cause tumors characteristic of those typically observed in species A.[23])

For example, the use of *leukemia* viruses from cats and primates to rescue mouse *sarcoma* viruses allowed the host range of the mouse sarcoma virus to be extended to both cats and monkeys. (The procedure described above was used to create mouse/primate pseudotype viruses by rescuing murine sarcoma viruses with a monkey virus. In a similar manner, a murine helper leukemia virus could be used to rescue a monkey sarcoma virus.[24])

These experiments, demonstrating that *numerous cancer viruses could be artificially made capable of jumping the species barrier (including that of primates)*, tended to support the theory that cancer viruses from animals might be able to cross the species barrier under certain *natural* conditions and cause cancer in humans. If this were true, then human cancer vaccines might be made possible by modifying any such animal

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[21] P. S. Sarma, T. Log, R. J. Huebner, "Trans-Species Rescue of Defective Genomes of Murine Sarcoma Virus from Hamster Tumor Cells with Helper Feline Leukemia Virus," *Proceedings of the National Academy of Sciences*, vol. 65, no. 1, January 1970, 81-87.

[22] Huebner, "The Murine Leukemia-Sarcoma Virus Complex."

[23] In addition to sarcoma viruses, this same process was used to create pseudotypes of other viruses such as vesicular stomatitis virus (VSV) consisting of the VSV genetic core surrounded by a mammalian type C helper virus envelope. M. M. Lieber, G. J. Todaro, "Mammalian Type C RNA Viruses," in F. F. Becker, ed. *Cancer: A Comprehensive Treatise* (New York: Plenum Press, 1975), 91-127.

[24] E. M. Scolnick, W. P. Parks, "Isolation and Characterization of a Primate Sarcoma Virus: Mechanism of Rescue," *Int. J. Cancer*, vol. 12, 1973, 138-147.

cancer viruses capable of causing cancer in humans.[25] (This would represent an interesting variation on the revolutionary process of creating vaccines against the deadly disease smallpox in man using cowpox viruses.)

Human Pseudovirus Construction

As early as 1964 some researchers advocated using procedures similar to those just described to make infectious pseudotype cancer viruses capable of infecting *human* cells. (Such experiments were designed to prove that a helper virus was involved in causing viral forms of human cancer.[26]) These viruses would be created by using a human leukemia virus instead of an animal leukemia virus as the rescuing agent.[27] However, since there were no known human leukemia viruses with which to rescue an animal sarcoma virus (and thereby produce a human cancer pseudovirus), the procedure described above was modified in various ways *to produce animal cancer viruses capable of replicating in human cells*.

In one successful attempt at getting *mouse* pseudotype viruses to infect human cultures, a pure mouse sarcoma/leukemia pseudotype virus (Kirsten murine sarcoma virus) was created in the manner described above and used to infect normal human cell culture lines. It was shown in this experiment that this pseudotype cancer virus became very proficient at growing in human cells.[28] In fact, the ease with which the human cells could be infected with the mouse pseudotype virus was so striking that researchers postulated that human genetic information from a latent virus in the human cell culture was picked up by the mouse pseudotype to form a new recombinant virus consisting of a combination of both human and murine genetic information.[29]

In another experiment of this type, *Moloney* sarcoma virus derived from mice was used to infect hamsters in the manner described above. However, instead of rescuing the virus with a pure mouse leukemia virus (as in the experiments described above) or a human leukemia virus (which didn't exist), the sarcoma virus was rescued with a *human cell culture independently infected with the* Rauscher *mouse leukemia virus*. Interestingly, this resulted in a pseudotype virus capable of infecting human cells but not those of

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[25] In an interesting twist on this phenomenon, it had been reported that vaccination of hamsters with the human wart virus was effective in preventing cancer due to the monkey cancer virus, SV40. C. W. Potter, J. M. Hoskins, J. S. Oxford, "Immunological Relationships of Some Oncogenice DNA Viruses," *Archiv für die gesamte Virusforschung*, vol. 27, 1969, 74.

[26] In its 1967 report, authors associated with the government program to isolate human cancer viruses and manipulate them for use in vaccines noted that the animal research with helper viruses might also apply to cancers infecting man: "A highly potent mouse sarcoma virus was discovered this year. The major significance of this new laboratory model is the finding that this virus requires the help of a leukemia virus for the induction of malignant sarcomas. These studies parallel those previously reported with several sarcoma viruses of chickens and suggest that the helper phenomenon may contribute to the occurrence of tumors in other animal species—including man." *Special Virus-Leukemia Program, Progress Report #4*, Program Staff, National Cancer Institute, Etiology Area, May 1967, p. 13.

[27] C. Jensen, A. J. Girardi, R. V. Gilden, H. Koprowski, "Infection of Human and Simian Tissue Cultures with Rous Sarcoma Virus," *Proc. Nat. Acad. Sci.* vol. 52, 1964, 53-59.

[28] S. A. Aaronson, "Common Genetic Alterations of RNA Tumour Viruses Grown in Human Cells," *Nature*, vol. 230, April 16, 1971.

[29] Ibid. The author of the paper remarked, "The most exciting possibility is that recombination has occurred with a latent C-type human virus which itself may exist in an integrated state. The new viral surface antigens of the murine viruses described here would then reflect genetic information of this latent virus."

animals.[30] This development was thought to be due to the fact that the pseudotype virus had obtained a virus envelope of human origin.[31]

Expanding host ranges

In addition to these two experiments with mouse pseudotypes grown in human cells, the process of creating pseudotype viruses with greatly expanded host ranges capable of infecting human cells was also adapted to monkey viruses. One example is particularly noteworthy. In this example, a rat *sarcoma* virus (Kirsten sarcoma virus) adapted for human cell growth was combined with a *noncancerous* baboon virus (a virus known as M7[32]). This pseudotype murine/baboon combination virus proved to have extraordinary characteristics. In addition to being capable of infecting human cell cultures, this pseudotype virus (as was discussed in an earlier section) was capable of inducing tumors in a wide range of animals including dogs and monkeys.[33]

This experiment had far-reaching implications. If *noninfectious cancer* viruses were capable of combining with *infectious noncancer* viruses resulting in *infectious cancer* viruses capable of infecting human cells, *a huge potential for the accidental creation of contagious human cancer viruses existed*. The researchers who made this breakthrough[34] realized that infectious, latent primate viruses (which they claimed were present in all experimental baboon tissue cultures[35]), might combine with other *noninfectious* cancer viruses in this same manner to form infectious, transspecies cancer viruses. If this were indeed possible, it meant that any human vaccines made using primate cell cultures had the potential to combine with latent human cancer viruses or animal cancer viruses in vaccine recipients and become sources of infectious *human* cancer-causing viruses![36] Thus, the resulting pseudotype virus combinations might possibly be capable of causing widespread cancer in man through vaccination procedures *even though the contaminating baboon viruses present in the vaccines weren't originally cancerous and the human or animal cancer viruses present in the vaccine recipients*

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[30] D. V. Ablashi, G. R. Armstrong, W. Turner, "Production and Characterization of Human Cell-Adapted Murine Rauscher Virus Pseudotype of Murine Sarcoma Virus," *Journal of the National Cancer Institute*, vol. 50, no. 2, February 1973, 381-385.

[31] The researchers who conducted this experiment also noted that such viruses might be useful as a diagnostic tool to rescue potential cancer-causing viruses from humans which otherwise might go undetected due to their latent nature. Such a tool might aid researchers in their quest to prove that human cancer viruses did indeed exist.

[32] This virus, also referred to as Baboon endogenous virus (or BaEV), was isolated from baboon placenta in 1974. R. Benveniste, M. Lieber, D Livingston, C. Sherr, G. Todaro, *Nature*, vol. 248, March 1, 1974.

[33] S. S. Kalter, 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, Dec. 1976. *Develop. biol. Standard.*, vol. 37, 219-228.

[34] It is interesting that this breakthrough was announced at a symposium of the World Health Organization on vaccine cell culture standardization. A similar technology may have been used to create a human immunosuppressive virus which was implemented in a WHO-supervised smallpox vaccination program. The evidence for this scenario is presented in other works by the author.

[35] "It is now apparent that any baboon tissue, under appropriate conditions, will yield an endogenous virus. Electron microscopy studies also suggest that the baboon is not unique in harboring these agents. It is highly probable that all animal species contain viral genes in their tissues which may be expressed to one degree or another depending on the physiologic conditions."

[36] For this reason, the researchers who made this discovery strongly cautioned the medical community about using such cell cultures for vaccine development.

weren't originally infectious (the contaminating monkey virus would merely make the latent cancer viruses more infective by "rescuing" them).

The discovery of "rescuing viruses" and their role in the creation of pseudotype viruses was a very important development in cancer research. The accidental or intentional creation of pseudotype viruses provided not only a mechanism by which defective or nonreplicating cancer viruses could become infectious within a given species, but a mechanism by which *defective* viruses from one species could be turned into *infectious* viruses in another species! *This technology would, for example, allow noninfectious sarcoma viruses from one species of animal to be used to infect and induce cancer in another species by gaining the host range of the rescuing viruses.* Such a capability would also open up the possibility for unscrupulous researchers to infect human populations with a wide range of experimental animal viruses to test the theory that human cancers were in fact caused by animal cancer viruses which had somehow *naturally* acquired the ability to cross species lines.[37] Following such a development, researchers could then set about trying to create vaccines against such human cancer viruses, just as they did in animal research.

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Searching for human cancer viruses

In addition to these types of experiments in which animal cancer viruses were manipulated to infect human cells, researchers continued to search for naturally occurring viruses in human cancer cells which, if found, might provide support for the theory that human cancer was caused by a viral source. Using techniques similar to those developed in the search for and characterization of animal cancer viruses, researchers probed human cell culture lines derived from various human cancers for viral particles which might be shown to be cancer-causing viruses.

Some successes were claimed in this type of research. For example, the Epstein-Barr virus was isolated from patients with a form of cancer known as Burkitt's lymphoma. Additionally an alleged virus was isolated from a patient with histiocytic lymphoma.[38] In the latter example involving histiocytic lymphoma, the putative *human* cancer virus was unsuccessfully used in attempts to create human/mouse pseudotype viruses by rescuing the defective *mouse* sarcoma viruses with the newly discovered *human* cancer virus. This was done in the same manner that murine leukemia viruses as well as feline leukemia, ape leukemia, and simian sarcoma viruses had been used to rescue defective murine sarcoma viruses.[39]

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[37] One can only imagine what might have happened if Chester Southam and his colleagues who experimented with human cancer cell injections (or those researchers who followed up on Southam's research by injecting animal cancer viruses—such as the SV40 monkey cancer virus—mixed with human cells in human subjects) had had such viruses during their cancer transplant/vaccine studies in human subjects. Perhaps they did exactly that, although unwittingly, when they mixed human cancer cell lines with animal cancer viruses and injected the resultant mixtures in human subjects.

[38] H. S. Kaplan, R. S. Goodenow, A. L. Epstein, S. Gartner, A. Declève, P. N. Rosenthal, "Isolation of a type C RNA virus from an established human histiocytic lymphoma cell line," *Proc. Natl. Acad. Sci. USA*, vol. 74, no. 6, June 1977, 2564-2568.

[39] Ibid.; N. M. Teich, R. A. Weiss, S. Z. Salahuddin, R. E. Gallagher, D. H. Gillespie, R. C. Gallo, "Infective Transmission and Characterisation of a C-type Virus Released by Cultured Human Myeloid Leukaemia Cells," *Nature*, vol. 256, 1975, 551-555.

Eventually researchers pursuing this line of research claimed to have isolated C-type cancer viruses from human patients with leukemia.[40] Extensive immunological and biochemical analyses were conducted on these alleged human leukemia viruses. For example, analyses such as DNA hybridization tests were conducted to determine the relationship of this virus to similar viruses from other species including apes. Through these tests, the alleged human "leukemia virus" was found to be related to a virus known as a simian sarcoma virus (SSV).[41] As had been recommended by earlier researchers, these scientists used this alleged human leukemia virus as a rescuing virus for a murine sarcoma virus to create a transspecies pseudotype cancer virus.[42] (This virus turned out to be a contaminating animal virus in the cell culture and was not of human origin. The other "human" leukemia viruses discovered in the 1970s were also suspect.)

Implications

A precedent was described previously in which combinations of the animal cancer virus SV40 and human cancer cells were produced and injected into immuno-impaired human cancer patients, resulting in tumor formation. Given that cancer viruses were later isolated from such human cancer cell cultures (these viruses were named HTLV—human T-cell leukemia viruses), it is possible that pseudotype viruses of the type described above were unintentionally created in the earlier experiments which mixed human cancer cells and animal cancer viruses such as SV40.

If the more sophisticated process described above, involving the ***deliberate*** fabrication of infectious pseudotype ***cancer*** viruses, was used to create an animal ***immunosuppressive*** virus capable of infecting humans, then a repeat of the aforementioned cancer injection experiment (which used injections of the modified monkey sarcoma virus SV40 in humans) on a wider scale using injections of such immunosuppressive viruses might very well be a plausible theory for how the AIDS epidemic and the associated epidemic of sarcoma began in human populations. This scenario might also explain the close relationship of the AIDS viruses to simian and bovine immunosuppressive viruses which were being grown in human cell cultures just prior to AIDS as well as the mechanism of cross-species transfer.

Such experiments with synthetically created immunosuppressive or cancer viruses that were derived from animals—yet which were capable of infecting humans—would allow researchers to reproduce in humans the powerful experimental procedure used in the mouse immunosuppressive cancer experiments described earlier.[43] Could this scenario explain why AIDS is proving to be so "beneficial" to cancer vaccine researchers?

Is the HIV a Manufactured Pseudotype Virus?

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[40] K. Nooter, A. M. Aarssen, P. Bentvelzen, F. G. De Groot, F. G. Van Pelt, "Isolation of Infectious C-type Oncornavirus from Human Leukaemic Bone Marrow Cells," *Nature*, vol. 256, 1975, 595-597; R. E. Gallagher, R. C. Gallo, "Type C RNA Tumor Virus Isolated From Cultured Human Acute Myelogenous Leukemia Cells," *Science*, January 31, 1975, 350-353.

[41] N. M. Teich, et al.

[42] Ibid.

[43] Recall that in these experiments mice were injected not only with cancer viruses but with immunosuppressive viruses designed to make the animals more susceptible to the cancer viruses.

Although *mouse* cancer viruses and mouse/primate pseudotype cancer viruses were created which were capable of infecting human cell cultures and acquiring human genetic information during infection of human cell cultures, it is doubtful that such viruses were used in widespread, *in vivo* human experiments.[44] The covert experimental use of mouse viruses in human populations would have drawbacks.[45]

It was noted above, however, that at least one strain of the human immunosuppressive virus was reported to be similar to an immunosuppressive virus of cattle called bovine-visna[46] (or BIV). Where did this virus come from? Could one strain of this virus have been created artificially and adapted for human cell growth for use in an immunosuppressive cancer experiment in human populations? Might a process similar to that described above, which was used to create pseudotype mouse *cancer* viruses capable of infecting human cells, have been used to make *immunosuppressive* animal virus combinations, such as bovine-visna, capable of infecting human cells?

The mouse pseudotype experiments described above provide illustrations as to how this might have been done. One possibility would have been to adapt the bovine leukemia and the visna viruses separately for growth in human cells followed by growth of the combination virus in human cells. A process similar to this was used to obtain mouse pseudotype cancer viruses capable of growing efficiently in human cell cultures.

Yet another way such a virus might have been created would have been first to create a hybrid ovine/bovine virus through growing visna sheep virus in cattle cell cultures[47] or alternatively growing the bovine leukemia virus in sheep cell cultures.[48] The host range of such a hybrid cattle/sheep virus might then be expanded to the human species by "rescue" of the virus with a human leukemia virus (which would provide the virus protein envelope) by growth of the hybrid virus in the appropriate human cell cultures. For example, such a virus might have been adapted for *human* cell growth by rescuing the bovine/visna virus with potential human leukemia viruses hidden in cell cultures from human cancer patients. (As described above, during the late 1970s researchers meticulously searched such human cell cultures derived from human leukemia patients for human leukemia viruses.[49] The infectious nature of such cell

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[44] An *in vivo* experiment is one that is conducted in human *subjects*. Experiments conducted in human *cell cultures* are referred to as *in vitro* experiments.

[45] For example, if they were discovered, there would be no rational explanation other than unethical experimentation for these viruses being in humans. Viruses from other animal species (e.g., cows and monkeys) would not suffer from this drawback since cell cultures from these animals are routinely used in medical research and production. Accidental contamination of cell cultures or even food could be used as an excuse or cover story for these viruses being found in humans.

[46] This virus is thought to be a combination of bovine leukemia and the visna virus from sheep.

[47] The visna sheep virus was successfully propagated in calf cell cultures as far back 1962. H. Thormar, B. Sigurdardóttir, "Growth of Visna Virus in Primary Tissue Cultures from Various Animal Species," *Acta. Pathol. Microbiol. Scand*, vol. 55, 1962, 180-186.

[48] Gross has remarked at how surprisingly easy it was to get the bovine leukemia virus to infect and cause cancer in newborn sheep. In fact, it was easier to infect newborn sheep with the cattle virus than to infect newborn calves. Ludwik Gross, p. 703.

[49] Robert Gallo published the first paper conclusively identifying human leukemia viruses in such human leukemia cell cultures in 1981. Robert Gallo, *Virus Hunting: AIDS, Cancer & the Human Retrovirus* (New York: Basic Books, 1991), 105.

cultures was demonstrated when researchers injected the leukemic material in monkeys—allegedly causing an infectious form of leukemia.[50])

Is this hypothetical process for the creation of a human version of the immunosuppressive bovine-visna virus feasible? It is highly interesting in light of this speculation that an experiment similar to that just proposed was conducted and published *(just before AIDS broke out in human populations)* in which bovine-visna virus, an immunosuppressive animal virus, was grown in cell cultures of human bone marrow obtained from patients with leukemia![51] Human leukemia viruses present in the leukemic marrow may have resulted in the creation of a pseudotype virus consisting of a combination bovine-ovine-human virus. Since the resultant virus was found to be capable of growing in human cells, it may have been capable of infecting human subjects and thereby causing an immunosuppressive infection similar to that which it causes in cattle.

Could this technique be responsible for the creation of an AIDS virus—the virus which causes an infection in humans which has such remarkable similarities to that caused by visna virus in sheep? (In addition to belonging to the same virus subfamily as HIV,[52] the visna virus infection itself has many similarities with the HIV infection. Numerous researchers have noted the eye-opening similarities in the effects on the immune system between the visna virus and HIV. For example, the viruses produce similar brain and central nervous system disorders as well as autoimmune effects. And both have a long latency period immediately following infection followed by long-term T-cell responses.[53])

Dr. Robert Strecker focused on the bovine-visna virus and a process similar to that just described as one source of HIV. Strecker has theorized that the bovine and visna viruses were combined to form bovine-visna virus (also known as bovine immunodeficiency virus, or BIV), that this combination was then grown in human leukemia cells derived from human bone marrow, and that the resulting virus combination was used to induce experimental immunosuppression in humans in the form of AIDS. Strecker concisely summarized his theory regarding the origins of HIV in a letter to the *Journal of the Royal Society of Medicine* as follows:

> Most likely the AIDS virus arose by hetrodimer recombination of bovine leukaemia virus and visna virus in a commonly infected host cell. Furthermore, it seems more probable that the virus expanded its host range and perhaps replicative rate (trivialities to those initiated in reaction rate

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[50] L. A. Yakovleva, "Studies on the Conjectural Virus Nature of Human Leukemia in Experiments on Monkeys," *Bibl. Haemat*, no. 36, 761-772.

[51] J. A. Georgiades, A. Billiau, B. Vanderschueren, "Infection of Human Cell Cultures with Bovine Visna Virus," *J. gen. Virol.* (1978), 38, 375-380.

[52] Both the HIV virus and the visna virus have been identified as belonging to the same subfamily of retroviruses known as lentivirus (a slow virus which is characterized as having a long latency period prior to the development of symptoms–similar to the AIDS virus).

[53] G. D. Harkiss, D. Veitch, L. Dickson, N. J. Watt, "Autoimmune Reactivity in Sheep Induced by the Visna Retrovirus," *Journal of Autoimmunity,* vol. 1, 1993, 63-75.

kinetics of retrovirus recombination) by culture growth in malignant bone marrow tissue.[54]

It is noteworthy that a procedure very similar to that which Strecker suggests was used to create the AIDS virus (mixing animal cancer viruses with human leukemia material) was mentioned decades ago by cancer researchers as one which might be used in an area of ongoing experimentation to demonstrate that helper viruses were involved in human cancer development.[55, 56] (This recommendation was made by authors including Hilary Koprowski, whose experimental polio vaccine using contaminated monkey cell cultures has been identified by some researchers as being responsible for the introduction of one strain of HIV into human populations.)

If AIDS viruses were indeed created in the manner described above (through the growth of animal immunosuppressive viruses in leukemic bone marrow),[57] the experimental injection of these viruses in human populations to deliberately increase susceptibility to cancer might be viewed as sophisticated versions of much earlier experiments in which human leukemic bone marrow was injected into human subjects in unsuccessful attempts to induce cancer in man.[58] Similar experiments were conducted by Chester Southam in his experiments to determine the susceptibility of humans to cancer. For example, Southam passaged human cancer cells that were thought to contain human cancer viruses through animal cells [heterologous hosts] and then systematically injected them in humans to measure the tumor nodules that formed. As was described in one paper: "Southam et. al. showed that human cancer cells carried serially in tissue cultures or conditioned heterologous hosts exhibited growth when inoculated into advanced

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[54] R. B. Strecker, "Aids Virus Infection," *Journal of the Royal Society of Medicine*, Volume 79, September 1986, 559.

[55] Researchers pondering the possible role of defective animal cancer viruses such as Rous Sarcoma Virus (RSV) and helper viruses in human leukemia noted as far back as 1964 that "should a defective-helper virus system be species-specific, *only exposure of RSV-infected human . . . cultures to human leukemic material may result in the appearance of infectious RSV. This, at the same time, would serve as a demonstration for the presence of a 'helper' virus in human leukemia. Experiments bearing on this problem are currently in progress*." [emphasis added] C. Jensen, et al.

[56] A National Cancer Institute report noted in 1971, "It is now believed that defective sarcoma virus-leukemia virus interactions may be more widespread in nature than originally thought and that similar systems may be found in man." How might such viral "systems" be found in man? Artificially created, cross-species pseudotype viruses were one possible tool. The NCI report noted that "a sarcoma virus of the mouse, artificially changed to one possessing infectivity for cat cells, can now be used in cultures for the detection of cat leukemia viruses." Similarly sophisticated viral engineering procedures were thought to be applicable in the search for human leukemia viruses: "The possibility exists that the cat-adapted mouse sarcoma virus can be hybridized with the human agent to produce an indicator system for the detection of human leukemia viruses." *Special Virus Cancer Program, Progress Report #8*, 1971, Etiology Area—National Cancer Institute, 21, 22.

[57] Curiously, researchers have used a process similar to that just described to modify the human HIV virus by replacing its normal viral coat with that of a cattle virus. This increased the ability of the virus to infect different types of human cells. Andrew Pollack, "Scientists Enlist H.I.V. To Fight Other Ills," *New York Times*, 1/19/99.

[58] J. B. Thiersch, "Attempted Transmission of Acute Leukemia from Man to Man by the Sternal Marrow Route," *Cancer Research*, vol. 6, 1946, 695-698.

cancer patients."[59] The addition of animal immunosuppressive viruses to such injections may well have been the key to reliably inducing cancer in man, just as the injection of animal immunosuppressive viruses overcame the long-term difficulty of reproducibly inducing cancer in animals.[60]

In addition to bovine-visna, it is possible that similar combinations of human viruses with *primate* immunosuppressive or cancer viruses were also manufactured and tested in human populations as cancer research tools to determine the infectivity of cancer viruses as a function of immune system health. Scientists had experience manipulating such monkey viruses to cause cancer in humans. An experiment with an "active" immunosuppressor would merely be a more sophisticated version of the experiment described previously in which the primate cancer virus SV40[61] was mixed with human cancer cells and injected into *pre-immunosuppressed* cancer patients so that researchers could measure its ability to cause human cancer.[62] Such a scenario may explain the dominant strains of HIV, which are reported to be very similar to simian immunosuppressive viruses, and why the cancer epidemic caused by AIDS is proving to be so lucrative to cancer researchers.

Were such transspecies immunosuppressive viruses created from *simian* immunosuppressive viruses in addition to or instead of *bovine* immunosuppressive viruses? Could these viruses have been modified for human cell growth and used in human experiments creating a highly useful AIDS/cancer epidemic?[63]

An experiment *conducted after AIDS was already raging in human populations* provides an interesting perspective on the types of viruses and techniques that may have been available to unscrupulous researchers trying to develop such human immunosuppressive viruses from monkey viruses *prior to AIDS*. In one case published in 1998 a transspecies simian immunodeficiency virus capable of infecting human cells was created by mixing a *monkey* SIV with a *mouse* Moloney leukemia virus. The mouse-modified immunosuppressive *monkey* virus (a pseudotype virus) was found to be capable of targeting *human* CD4+ cells, the very type of T-cells[64] that the human immunodeficiency virus attacks![65]

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[59] J. T. Grace, T. Kondo, "Investigations of Host Resistance in Cancer Patients," *Annals of Surgery*, vol. 148, no. 4, October 1958, 633-641.

[60] A similar progression in human cancer transplant research took place when injections of human cancer cells by themselves were supplemented with injections of mixtures of animal cancer viruses (such as SV40) and human cancer cells in the experiment described earlier.

[61] The SV40 monkey virus was reported to be the "first demonstration of a malignant oncogenic quality for a virus of primate (monkey) origin." H. C. Chopra, M. M. Mason, "A New Virus in a Spontaneous Mammary Tumor of a Rhesus Monkey," *Cancer Research*, vol. 30, no. 8, Aug. 1970, 2081-2086.

[62] F. Jensen, et al.

[63] The benefits of the AIDS/cancer epidemic to cancer vaccine research are described in AIDS: The "Perfect" Disease (available at http://www.authorhouse.com/BookStore/).

[64] T-cells dominate the immune response in fighting cancer and play a major role in hypersensitivity reactions and graft rejection.

[65] The authors of this study combined a mouse leukemia virus with a monkey immunosuppressive virus (Moloney murine leukemia virus and MPMV virus) and found that the targeted "lymphocytes belonged almost exclusively to the CD4+ subset." In fact, the virus was so effective at targeting the cells attacked by SIV (and HIV), that the researchers proposed using it in gene-based HIV research in animals. S. Indraccolo, S. Minuzzo, F. Feroli, F. Mammano, F. Calderazzo, L. Chieco-Bianchi, A. Amadori,

This experiment raises fascinating questions: Were such deadly immunosuppressive monkey viruses available to unscrupulous researchers before they suddenly broke out in human populations causing the AIDS crisis? Could similar modification of such monkey viruses have been the source of human AIDS?

Indeed, there were simian immunodeficiency viruses available to researchers before AIDS broke out in humans, and indeed researchers were busily modifying these immunosuppressive monkey viruses for human cell growth just prior to the AIDS epidemic! In fact, a monkey immunosuppressive virus was being modified for human cell growth by mixing it with human cancer cells and mouse cancer virus cell cultures—a procedure similar to that described above which rendered *the same monkey virus* capable of targeting human CD4+ cells.

For example, while the public is completely ignorant of this fact, *simian immunosuppressive viruses* such as the Mason Pfizer Monkey virus (one of three types of SIV[66]) were available to researchers as early as 1970—when such a virus was grown in cell cultures.[67] Well before AIDS, the Mason Pfizer Monkey Virus (MPMV) was shown to induce immunodeficiency states in monkeys in the early 1970s[68, 69] as well as the 1980s.[70]

"Pseudotyping of Moloney Leukemia Virus-based Retroviral Vectors with Simian Immunodeficiency Virus Envelope Leads to Targeted Infection of Human CD4+ Lymphoid Cells, *Gene Therapy*, vol. 5, 1998, 209217.

[66] MPMV is one of "three independent virus isolates" of "simian acquired immunodeficiency syndrome (SAIDS) in macaque monkeys." One group of researchers summarized, "When inoculated into young monkeys, MPMV produced a spectrum of nononcogenic disease associated with an immunodeficiency condition." R. M. Thayer, M. D. Power, M. L. Bryant, M. B. Gardner, P. J. Barr, P. A. Luciw, "Sequence relationships of type D retroviruses which cause simian acquired immunodeficiency syndrome," *Virology*.,1987 April 157 (2), 317-329.

[67] This immunosuppressive virus was isolated from a mammary tumor of a rhesus monkey in 1970. H. C. Chopra, et al.

[68] The virus not only had immunosuppressive properties; it was reported to act like a slow virus—just like AIDS. D. L. Fine, J. Landon, R. Pienta, M. Kubicek, M. Valerio, W. Loeb, 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.

[69] In 1986 one group of researchers described how immunodeficiency states were induced in monkeys in the early 1970s using MMPV. Since the virus did not immediately induce cancer and since AIDS hadn't started in human populations, the curious effects of the virus (immunosuppression and fatal infection by opportunistic diseases) did not attract widespread notice: "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." [emphasis added] M. L. Bryant, M. B. Gardner, P. A. Marx, D. H. Maul, N.W. Lerche, K. G. Osborn, L. J. Lowenstine, A. Bodgen, L. O. Arthur, E. Hunter, "Immunodeficiency in rhesus monkeys associated with the original Mason-Pfizer monkey virus," *J. Natl. Cancer Inst*, 77(4), Oct 1986, 957-965.

[70] Cryogenically preserved samples of this virus, isolated in the 1970s, were shown to induce a disease (SAIDS) very much like that due to simian immunodeficiency viruses (SIVs). The researchers who published the experiment in 1986 reported, "MPMV produces an acquired immunodeficiency similar to that caused by the recently described simian acquired immune deficiency syndrome type D retroviruses." [emphasis added] Ibid.

While this may be startling to some, even more provocative is the fact that cancer researchers were coaxing this immunosuppressive monkey virus to grow in human cells in the late 1970s—just before AIDS broke out in human populations. Just as the SV40 monkey sarcoma virus had been grown in human cancer cells, researchers associated with biological warfare facilities grew the immunosuppressive Mason Pfizer Monkey Virus in human cancer cells. They reported "that a variety of human cells from both primary and continuous cultures established from normal and neoplastic tissues were permissive for MPMV replication."[71]

In one of these experiments, MPMV was mixed with SV40—a monkey cancer virus (which had already been injected in humans to cause cancer) and the Rous sarcoma virus in human cell cultures.[72]

This research raises the questions:

- Were these pre-AIDS viral combinations also capable of targeting human T-cells, like the SIV/mouse leukemia virus combinations were demonstrated to be, after AIDS had broken out?
- Was the creation of these cancer/immunosuppressive combinations an attempt to replicate in humans the experiments in animals in which combinations of cancer and immunosuppressive viruses were used to induce cancer for vaccine experiments?

Not only did experimental immunosuppressive viruses exist at the time AIDS began (based on later experiments with these viruses, these viruses were capable of inducing AIDS-like results), but at just about the time AIDS began in human populations, researchers were investigating the immunosuppressive properties of various monkey and human viruses on human cells. For example, the effects of a virus known as PMFV, an alleged virus isolated from human breast tumors, were investigated as to its potential to inhibit (*in vitro*) the response of human lymphocytes—the cells that HIV attack. Likewise, a baboon virus was also investigated for this same purpose.[73]

Could human experiments with these viruses explain how the famous simian AIDS monkey virus (SAIDS), reported to be the cause of AIDS, suddenly attained the ability to infect human CD4+ cells efficiently and wreak such convenient havoc for cancer researchers? (These CD4+ cells were the very ones that cancer researchers were interested in targeting since they were the ones suspected to be capable of preventing cancer.) This seems a more plausible explanation for AIDS and its benefits than the more commonly accepted one (natural infection) and may be why the media has completely

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[71] These cancerous human cells included the HeLa cancer cell line derived from a human cervical carcinoma and Kirsten murine sarcoma virus-infected human osteosarcoma cells. D.L. Fine, G.C. Clarke, L.O. Arthur, "Characterization of infection and replication of Mason-Pfizer monkey virus in human cell cultures," *J Gen Virol*, Aug;44(2), 1979,457-69.

[72] H. Ogura, T. Tanaka, M. Ocho, T. Kuwata, T. Oda, "Detection of Mason-Pfizer monkey virus infection by syncytia formation of human cells doubly transformed by Rous sarcoma virus and simian virus 40," *Arch Virol*, 57(2), 1978, 195-198.

[73] J. Denner, V. Wunderlich, D. Bierwolf, "Suppression of human lymphocyte mitogen response by disrupted primate retroviruses of type C (baboon endogenous virus) and type D (PMFV)," *Acta Biol Med Ger*, 39 (11-12) 1980, K19-26.

blacked out discussion of the immunosuppressive techniques and transspecies viruses that had been created by cancer researchers at the time the AIDS epidemic began.

This laboratory process would explain why a highly useful epidemic due to a rare type of virus known as a lentivirus suddenly exploded in human populations just at the time scientists were playing with them in cell cultures—and never previously (or naturally). This artificial method for creating human versions of monkey retroviruses seems more likely than natural transmission. John Seale noted, "The theory popular amongst many molecular biologists that HIV-1 has been endemic and largely non-pathogenic, in an isolated group of people in Africa for millennia, is not scientifically credible." Seale explained the reasons he adopted this view:

> **When cross-species infection of a retrovirus is dependent only upon chance, and natural selection, as opposed to artificial selection in the laboratory, cross-species infection appears to be a rare historical event.**

Seale also noted that *man-made mechanisms* were capable of *enhancing* the cross-species transfer of such rare viruses:

> **Experimentally, however, cross-species transfers of these atypical viruses and the diseases they cause usually have been achieved by inoculation; adaptation of the new strains of virus to new host species was then perfected artificially by serial passage by further inoculations.**

This is exactly what was done with immunosuppressive cattle and monkey viruses, as described above.[74] Speculating specifically with respect to AIDS, Seale wrote, "On the other hand, HIV-1 may have evolved rapidly from known animal lentiviruses replicating in the highly artificial, selective conditions of serial passage in human cell cultures."

After reviewing numerous cases where experimental viruses, which had been modified through "artificial selection" in cell cultures, had been involved in catastrophic transfers into new hosts, Seale went on to add cryptically:

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[74] A monkey tumor virus known as the Yaba virus had also been serially passaged in man. The authors of one of several papers published in these studies summarized:

"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."

R. S. Metzgar, J. T. Grace Jr., and E. E. Sproul, "Immunological Studies of Subcutaneous Virus-Induced Histiocytomas In Primates," *Ann. N. Y. Acad. Sci.*, vol. 101, 192-202.

It would appear that the AIDS epidemic may be just one of the latest of several mammalian cross-species viral transfers triggered by the techniques of virology developed in the 20th century, which subsequently spread out of control in the new host species.[75]

A "Super" AIDS Virus Created by Cancer Researchers

The thesis developed throughout this series is that the AIDS virus was developed through the combined efforts of the cancer and biological warfare research establishments to develop improved cancer-causing viruses (and aerosol delivery methods) through immunosuppressive animal research and transspecies cancer experimentation. If the process proposed above to explain the creation of the original AIDS virus sounds far-fetched, it may be illuminating to discover that cancer researchers, including Robert Gallo, have recently used the very tools developed in this type of research (immunosuppressive animal research and transspecies cancer experimentation) to create an AIDS virus which is much more infectious than the natural form of the cancer-causing virus and which can most likely be transmitted through the air. (This has been a long-range goal of the biological warfare research infrastructure.)

The newly created and highly infectious form of AIDS was made so infectious by taking the "normal" AIDS virus and mixing it with human leukemia cells which had previously been infected with a mouse leukemia virus. (These human leukemia cells were infected with the murine viruses by injecting them into immunosuppressed mice.) The "common" AIDS virus, when combined with the mouse viruses present in the human leukemia cells, formed a new virus which became much more pathogenic and was able to reproduce more rapidly than the original AIDS virus.[76] Additionally, the newly manufactured super-AIDS virus was able to infect a whole new range of human cells, including those of the respiratory tract.[77] Jean Marx wrote in *Science*:

As a result, the AIDS virus, also known as HIV-1 (for human immunodeficiency virus 1), acquires some new biological characteristics, including the ability to reproduce much more rapidly than it normally does and to infect new kinds of cells.

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[75] J. Seale, "Crossing the Species Barrier–Viruses and the Origins of AIDS in Perspective," *Journal of the Royal Society of Medicine*, vol. 82, 1989, 519-523.

[76] Other researchers at about the same time devised similar methods for making the AIDS virus more infectious by mixing it with mouse cancer viruses. For example, one group mixed HIV with a mouse virus named Ampho-1B to make it more infectious. These researchers reported, "Our results demonstrate that infection of a cell with both HIV and Ampho-1B generates HIV progeny that can efficiently infect and replicate in a broad range of CD4 cells." D. H. Spector, E. Wade, D. A. Wright, V. Koval, C. Clark, D. Jaquish, S. A. Spector, "Human immunodeficiency virus pseudotypes with expanded cellular and species tropism," *J Virol,* 1990 May, 64(5), 2298-2308.

[77] J. Marx, "Concerns Raised About Mouse Models for AIDS," *Science*, vol. 247, 1990, 809.

Researchers surmised that this was because the AIDS virus formed a pseudotype virus by mixing with the murine leukemia virus.[78]

This cross-species viral experiment which was conducted by mixing animal and human immunosuppressive and cancer viruses to create a highly infectious form of AIDS virus is very suggestive of the procedure proposed above to account for the creation of the original AIDS virus.[79] Was a process similar to that just described used by Robert Gallo[80] or his colleagues to create the *original* AIDS virus by combining simian and/or bovine-visna virus in human leukemia cells?[81] Or, in other words, *were the published procedures which were used to increase the infectivity of the AIDS virus merely a more sophisticated version of the procedure which produced the AIDS virus originally?*

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[78] The remarkably expanded infectious properties of this manufactured AIDS virus may have been due to the formation of a pseudotype virus of the type described above (with the HIV genome surrounded by the mouse leukemia virus, MuLV). The researchers who created the new AIDS virus said, "The observed broadening of the cellular tropism of HIV-1 may be ascribed to the generation of pseudotypes containing the HIV-1 genome coated by the envelope of X-MuLV." P. Lusso, et al.

[79] It was proposed that in the creation of the original AIDS virus, an animal immunosuppressive virus was mixed with human leukemic and/or mouse cancer virus cells to make it capable of growing in human cells. In the documented creation of the "super" AIDS virus, a human immunosuppressive virus was mixed with human leukemia cells infected with an animal cancer virus. The procedures and viruses used to create infectious immunosuppressive viruses of both humans and monkeys have interesting similarities. In the case published in 1998 an SIV was mixed with an animal cancer virus, rendering it capable of infecting human CD4+ cells (turning an SIV into an HIV). In the case published in 1978 the SIV known as MMPV was mixed with mouse cancer cells, rendering it capable of infecting human cells. In the case just described by Gallo and company, the HIV was mixed with mouse cancer cells as well, rendering it capable of infecting a wider range of human cells.

[80] It is interesting that Robert Gallo, the controversial "co-discoverer" of the HIV virus in humans, was involved in finding the first human leukemia viruses as well as manufacturing cross-species pseudotype viruses. He would later create super-infectious AIDS virus offshoots using immunosuppression and transspecies infection. If Gallo was involved in the creation of the original AIDS virus as well, that might explain why he was able to "discover" the viral agent which causes AIDS. It might also explain why he is being given permission, as head of the University of Maryland's Institute of Human Virology, to conduct a massive experimental HIV vaccine campaign on human subjects in Uganda using a vaccine consisting of a novel combination of bacterialogical/virological materials. (The vaccine consists of a hybrid comprised of a "strain of salmonella bacteria responsible for typhoid [which] is genetically altered to be less infectious and to carry portions of the DNA of the HIV virus.") Alex Dominguez, "AIDS Vaccine to Be Tested in Uganda, *Associated Press*, Saturday, May 20, 2000.

[81] This research raised severe safety issues with respect to the use of animal models for AIDS research. By demonstrating that HIV could interact with latent animal viruses and thereby become more pathogenic, these researchers showed that the routine injection of animals with HIV in attempts to mimic human infection (as an aid to experimentation with AIDS treatments) may accidentally result in new and unpredictably dangerous infectious immunosuppressive viruses. Perhaps the exposed dangers inherent in such research will eventually be used to lend credence to the *accidental* AIDS creation theory, should knowledge of its artificial nature ever become widely known.

Summary

The technology for creating human/animal pseudotype cancer-causing viruses with greatly expanded cross-species infectious ranges was used to create a greatly enhanced AIDS virus, raising the possibility that such a process was used to create the original AIDS virus from an animal immunosuppressive virus which would be capable of causing immunodepression in human subjects for the purposes of cancer research.

Whether or not such a process was used to create the pathogens responsible for the AIDS plague will be determined only with further research. *Such viruses and techniques unquestionably existed at the time AIDS broke out in selective human populations*. Given the progress made in the field of pseudovirus creation, the frightening history of cancer researchers injecting humans with cancer-causing viruses (including combinations of human and monkey viruses), the benefits of the AIDS epidemic to cancer researchers and the harrowing warnings of scientists associated with pseudovirus technology regarding the danger of possibly creating infectious cancer viruses during the manufacturing of vaccines in contaminated animal cell cultures, this scenario warrants substantial investigation. The evidence linking the international AIDS pandemic to international vaccine initiatives conducted by international organizations with documented interests in exploiting immunosuppressive states on an international scale should also be investigated (this topic is discussed in other works by the author).

AIDS: The "Perfect" Disease

A more detailed study, titled *AIDS: THE "PERFECT" DISEASE*, which examines not only the cancer research benefits of AIDS mentioned above, but the benefits to the national security establishment, is available at 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 US 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 US government under the auspices of cancer research.