

Human Endogenous Retroviruses and AIDS Research: Confusion, Consensus, or Science?

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ABSTRACT

Human Endogenous Retroviruses (HERVs) are confounding factors in HIV/AIDS research that cannot be ignored. Evidence suggests that “viral load” may actually be measuring retroviral nucleoside sequences associated with HERVs. HERVs also provide a valid explanation for the presence of retroviruses recognizable by electron microscopy (EM) in the original 1983 publication from the Institut Pasteur, and may account for claims of innumerable “mutations” of the putative HIV pathogen. The interference of HERVs in AIDS research brings into question the subject of study in so-called “AIDS Research,” and the very existence of an exogenous HIV pathogen itself.

The HIV Consensus

The hypothesis that the acquired immunodeficiency syndrome (AIDS) is caused by an exogenous retrovirus, the human immunodeficiency virus (HIV), initially proposed in the early 1980s,¹⁻³ has exclusively dominated AIDS research for the past 25 years, although many investigators have repeatedly stressed the lack of scientifically acceptable verification of this hypothesis. Alerted to the numerous shortcomings of the official retroviral hypothesis by eminent retrovirologist Peter Duesberg,^{4,5} a group of AIDS “Rethinkers,” founded by molecular biologist Charles Thomas in 1991, called for the “Scientific Reappraisal of the HIV/AIDS Hypothesis” in 1996.

This group (www.rethinkingaids.com) released a mission statement co-signed by thousands of scientists and concerned citizens, including Nobel laureates Walter Gilbert and Kary Mullis. Other well-respected scientists, notably Sonnabend, Stewart, Lang, Papadopulos, Rasnick, and Gesheker⁶⁻¹² and distinguished scientific writers such as Celia Farber, John Lauritsen, Neville Hodgkinson, Joan Shenton, Christine Maggiore, Renaud Russeil, Djamel Tah, Jean-Claude Roussez, and Janine Roberts¹³⁻²¹ have also described the multiple failings of the HIV hypothesis. Between 1992 and 2000, another group based in London, UK, made highly significant contributions to scientific/public education by publishing *Continuum* magazine, under the leadership of Huw Christie.²² A medical team directed by Eleni Papadopulos in Perth, Australia, has also presented information questioning the validity of the HIV hypothesis.²³⁻²⁵ In May 2000, the controversy concerning HIV and the antiretroviral (ARV) drugs used to treat it became the topic of international inquiry when President Thabo Mbeki of South Africa, convened a debate between 35 academic scientists, “Orthodoxers” as well as “Rethinkers” together.²⁶ A similar debate took place in 2003 at the

European Parliament in Brussels, Belgium, when Paul Lannoye, a Belgian member of parliament, organized a public debate on “AIDS in Africa.”²⁷

Reports by AIDS Rethinkers are readily accessible on numerous websites, the early and most significant ones being www.virusmyth.com, www.rethinkingaids.com, www.theperthgroup.com, www.sidasante.com, and www.altheal.org.

In spite of innumerable scientific and public conferences and publications by AIDS Rethinkers, many in the medical community either ignore, or bluntly reject the existence of any HIV controversy,²⁸⁻³⁰ or claim that AIDS “denialism” undermines AIDS prevention.³¹ As a result, the monumental budgets allocated throughout the world to combat AIDS have been, and still are totally and exclusively restricted to HIV research. This can neither be explained nor justified by the lack of alternative hypotheses of AIDS causation, since nonviral factors (chemical, pharmacological, nutritional, and behavioral) associated with the clinical symptoms attributed to AIDS have been well documented and reviewed by others.³²

The retroviral hypothesis linking HIV to AIDS received a precipitous acceptance, not on the basis of scientifically verifiable data, but based on a so-called “consensus”—a consensus enthusiastically supported by the pharmaceutical industry. This review will focus primarily on the scientific facts (or artifacts) that impact the credibility of AIDS research.

Factors That Gave Apparent Credibility to the HIV Hypothesis

In the extensive HIV/AIDS literature, one finds that the claimed “evidence” that AIDS is caused by HIV-1 or HIV-2 is presumably “clear-cut, exhaustive and unambiguous,”³³ and comprises four groups of data: (1) identification of retroviral molecular markers, (2) observation of retroviral particles by transmission EM, (3) claimed efficacy of antiretroviral (ARV) drugs, and (4) epidemiological data.

(1) Identification of Retroviral Molecular Markers

In a long list of presumed HIV molecular markers, the most emblematic one is the enzyme reverse transcriptase (RT).^{34,35} Importantly, however, the activity of this enzyme has been readily demonstrated in practically all living cells of the biological universe,^{36,37} making it imperative to verify the purification of viral samples before making any claim for a specific link between RT and retroviruses. Sample contamination by cell debris can, by itself, explain the presence of RT activity. This is of considerable importance because attempts to isolate and purify HIV by sucrose gradient ultracentrifugation of supernatant from supposedly HIV-infected cell cultures have provided samples heavily contaminated with microvesicular cell debris, readily demonstrated by EM.^{38,39}

Anti-HIV antibodies are regarded as another class of molecular markers, used in so-called “HIV tests,” such as the enzyme-linked immunosorbent assay (ELISA).⁴⁰⁻⁴¹ The lack of specificity of this test, however, was clearly documented by C. Johnson⁴² who reported, as early as 1996, that almost 70 medical conditions having nothing to do with AIDS or HIV may result in a positive antibody test. These conditions include tuberculosis, malaria, leprosy, hepatitis, blood transfusions, influenza vaccination, multiple pregnancies, and others. Such a lack of specificity came as no surprise to those who were aware that the method used to prepare “HIV” antibodies was based on a circular argument, as discussed early on by Neville Hodgkinson.⁴³ Moreover, the method initially used in ELISA tests included a 400-fold plasma dilution. Without such high dilution everybody turned out to be “HIV positive,” as originally demonstrated by Roberto Giraldo in 1998.⁴⁴

Protein antigens of claimed retroviral origin represent a group of HIV markers used in another “HIV test,” the western blot test (WB). The WB test is used to confirm the ELISA test, and is based on the identification by electrophoresis on polyacrylamide gels of 10 presumably HIV proteins, such as p120, p41, p32, p24/25, and others. However, prior successful isolation and purification of HIV would be required to verify that all of these proteins actually originate from HIV particles, a purification that has never been achieved, as recognized by Luc Montagnier himself.⁴⁵

The considerable difficulty in isolating and purifying HIV was recognized, as early as 1993, by Eleni Papadopulos et al., who correctly concluded that without successful HIV purification, the retroviral nature of the “HIV marker proteins” was most uncertain. Papadopulos emphasized that these proteins are most likely cellular, originating from the abundance of cell debris in poorly “purified” HIV samples. The uncertainty and shortcomings of WB testing were already reported in 1991.⁴⁶ Soon afterwards, Papadopulos et al. raised the question: “Is a positive western blot proof of HIV infection?”⁴⁷ That WB tests are not reliable is evidenced by the variability of the protein criteria required for a “positive” test in different countries. The test is not even approved for diagnostic purposes in Great Britain.

The considerable difficulties experienced in attempts to purify “HIV” have never been resolved.⁴⁷ Recently, Henry Bauer has reviewed evidence that supports the conclusion that “HIV tests are not HIV tests.”⁴⁸ “HIV tests” only indicate the presence of antibodies supposedly directed against HIV. They do not indicate the presence of the virus itself.

The question then arises of whether the so-called “viral load” tests are more reliable, as they are based on polymerase chain reaction (PCR) technologies for recognizing and quantifying HIV. This appears highly questionable; Nobel laureate Kary Mullis himself, the discoverer of PCR, has indicated that his method is not expected to provide a reliable result in HIV diagnosis.

A second reason to question “viral load” data is that “viral load” implies the existence of viremia, i.e. the presence of virus particles in the peripheral blood, although no one has ever observed, by EM, one single retroviral particle in the blood of HIV/AIDS patients, even in those patients tagged as presenting with a “high viral load.”⁴⁹ Moreover, the PCR methods used for “viral load” determination bypass the problems of isolation of retroviral particles. The question

therefore arises: what is actually measured in “viral load” determinations? To date, no satisfactory answer has been provided.

Still, various amounts of claimed retroviral nucleotide sequences are routinely identified and quantified in a patient’s plasma. They are interpreted as originating from HIV, and used in the clinical assessment and therapy of AIDS patients. When Luc Montagnier was asked, “What is actually measured in viral load assessments?” during the discussion of a major HIV/AIDS debate in the European Parliament in 2003, his answer was less than clear and convincing.²⁷ The contradiction remains that genomic retroviral sequences are routinely recognized by PCR, and interpreted as originating from HIV particles, while nobody has actually visualized them by EM. More critical attention should be given to the true nature of these retroviral sequences, the origin of which is at present unclear.

(2) Observation of Retroviral Particles by Transmission Electron Microscopy

All the images of particles supposedly representing HIV and published in scientific as well as in lay publications derive from EM studies of cell cultures. They never show HIV particles coming directly from an AIDS patient.⁵⁰ The pictures are always embellished by computerized image reconstruction, with attractive colors and refined three-dimensional effects. The endless, worldwide publication in the media of these elegant artifacts has done much to persuade scientists and lay people alike to accept the existence of HIV as a key part of the orthodox consensus.

Cell cultures have been the major tool that permitted the development of modern virology. Unfortunately, these cultures are frequently contaminated by microorganisms such as viruses and/or mycoplasma, readily identifiable by EM. These contaminants, well known and documented for a long time,⁵¹ frequently made the interpretation of experimental data rather laborious, because to demonstrate the cytopathic effects of a given virus on cultured cells, it would have been much preferable to experiment with “clean” (i.e. virus-free) cells. Unfortunately, such cells are hard to obtain! Actually, it was difficult to study the Friend leukemia virus (FLV) in cell cultures, using murine cells, because EM readily demonstrated that most available murine cell lines were chronically carrying retroviruses!

The 1983 study from Institut Pasteur in Paris⁵² is illustrated by an EM (their Figure 2) showing unquestionable budding retroviruses on the surface of human cord blood lymphocytes. The interpretation of this figure by Luc Montagnier and his team, that these retroviruses originated from a pre-AIDS patient, was based on the fact that the cord blood lymphocytes were exposed to the cell-free supernatant of “infected” co-cultures. But the authors did not provide any evidence for “infection” in their co-cultures, nor for the presence of retrovirus particles in the supernatant of these cultures. Therefore, another explanation for the origin of the observed retroviruses on the surface of these cultured cord blood lymphocytes must be sought.

(3) The Claimed Efficacy of Antiretroviral (ARV) Drugs

Drugs such as azidothymidine (AZT), a DNA chain “terminator,” as well as non-nucleoside analog RT inhibitors (such as nevirapine) and protease inhibitors (such as ritonavir), are currently used in various combinations such as “highly active retroviral therapy”

(HAART), and repeatedly claimed to be “life saving.” Manufacturers of these drugs, however, strongly emphasize their toxicity. Lethal effects of AZT became dramatically evident when mortality of seropositive hemophiliacs suddenly increased sharply in 1987, precisely at the time high dosages of AZT started to be prescribed.^{53,54} Hopes that AZT might have preventive value were shattered by the Concorde study, when mortality of AZT recipients was found 25% higher than that of the untreated control group of symptom-free HIV-positive individuals.⁵⁵ These important studies have been reviewed by Duesberg,⁵ by Hodgkinson,⁵⁶ and others. Equally perplexing is that deaths of ARV-treated patients very frequently result from acute liver failure,⁵⁷ conflicting with the fact that HIV is not known for liver toxicity, while ARV drugs are.

If the effects of ARV drugs could still be regarded as proving that HIV is the cause of AIDS, one would at least expect some patients to be cured by these drugs. However, not a single case of “cure” has ever been reported. Instead, the clinical evidence points to the high toxicity of ARV drugs and their immunodepressive effects which actually mimic AIDS itself.⁵

Patients with severe AIDS have frequently been reported to be transiently, but remarkably, improved by ARV drugs. Such “Lazarus” type observations have been interpreted as evidence for an antiretroviral effect on HIV, supporting the existence/role of HIV. However, as most of these patients frequently suffer from pneumonia with *Pneumocystis carinii*, mycosis with *Candida albicans*, or both, and because protease inhibitors, introduced in antiretroviral therapy in 1996, have marked anticandidal⁵⁸ and antipneumocystis⁵⁹ effects, this interpretation is questionable at best. When anti-proteases help block such opportunistic infections, this has no direct relevance to HIV, and certainly does not “automatically support the “HIV model.”⁶⁰

(4) Epidemiologic Data

Maneuvering for major federal budget allocations, AIDS public health policies have been relying on media amplification of fear.^{61,62} Catastrophic prediction of heterosexual transmission of the disease, prophecies of a worldwide pandemic, and reliance on CDC and WHO statistical reports were all linked to the assumption that AIDS was a contagious disease, possibly transmitted in the general population by sexual intercourse.

Renowned epidemiologist Gordon T. Stewart did much, however, to dispel these erroneous predictions. In a letter to *The Lancet*,⁶³ he stated “the UK Government is beginning to retreat from its pessimistic certainty about pandemics of heterosexual transmitted AIDS” and exposed to scrutiny “the claim that AIDS has already spread by heterosexual transmission to the general populations.”⁶⁴ Stewart’s conclusions correlate well with the complete absence of HIV among female sex workers not using IV drugs. This “prostitute paradox” (i.e. no increased risk for AIDS among female sex workers) was reviewed from worldwide studies by Root-Bernstein in 1993,⁶⁵ and re-emphasized more recently by Etienne de Harven and Jean-Claude Roussez.⁶⁶ The lack of evidence for heterosexual transmission of AIDS was clearly presented by Padian et al., who could not observe one single case of seroconversion in a follow-up study of 175 HIV-serodiscordant couples over a period of six years.⁶⁷ That heterosexuals are not at risk for AIDS was stressed by Christian

Fiala in his 1997 book *Lieben wir gefährlich? (Do We Love Dangerously?)*.⁶⁸ Safe-sex practices (e.g. condoms) remain essential, however, for the prevention of diseases proven to be sexually transmitted, such as syphilis and gonorrhea.

Certain African countries, such as Uganda and Tanzania, had been regarded as epicenters of an AIDS “pandemic.” The lack of evidence supporting this, initially recognized by Philippe Krynen,⁶⁹ was clearly documented by Charles Gesheker⁷⁰ and by science writers Celia Farber⁷¹ and Neville Hodgkinson.⁵⁶ Twenty years later, national census figures have shown spectacular demographic increases in several sub-Saharan countries, clearly demonstrating that their populations had not been devastated, as officially predicted, by a deadly AIDS pandemic of historic proportions.⁷²

The most authoritative conclusions presented in 2008 by experienced epidemiologist James Chin, former Chief of the Unit of the Global Programme on AIDS of the World Health Organization (WHO) in Geneva, in his book *The Collision of Epidemiology with Political Correctness*⁷³ brought to a close any possible debate on heterosexual AIDS transmission. Chin stated that AIDS was, and still is restricted to a small population of homosexuals and intravenous drug users, and that the heterosexual population is not at risk. Chin’s conclusions have raised serious questions on the reliability of WHO statistics.

AIDS epidemiological data have been further confused by several consecutive changes in the official definition of the syndrome, and have failed to support the current HIV=AIDS dogma.

The hypothesis of an exogenous retrovirus “HIV” causing AIDS appears unsupported by the scientific evidence concerning molecular markers, EM findings, ARV drugs, and epidemiology. However, two intriguing findings deserve further attention: the identification of genomic retroviral sequences in AIDS patients’ blood (“viral load”) and the EM demonstration of retroviruses in cord blood lymphocytes.⁵² Simply concluding that “HIV does not exist” is not sufficient unless alternative, satisfactory explanations for these two observations are found.

“Viral Loads” and Retroviral Sequences

“Human endogenous retroviruses (HERVs) represent footprints of previous retroviral infection and have been termed ‘fossil viruses.’ They are transmitted vertically through the germline and are thus inherited by successive generations in a Mendelian manner,” stated Nelson et al. in a review entitled “Demystified... Human Endogenous Retroviruses.”⁷⁴ The molecular basis of HERVs was recognized 20 years ago.^{75,76} They appear defective, and rarely produce virus particles. As molecular footprints, they are “in all of us,” as recognized by Lower et al. in 1996,⁷⁷ and represent approximately 8% of the human genome, actually consisting of nucleotide sequences analogous to the retroviral genome.⁷⁸ Expression of HERVs, i.e. particle formation, seems to be a rare event, although it has been observed in placenta⁷⁹ and in tumor cell lines.⁸⁰ HERV retroviral sequences have also been detected by PCR in the peripheral blood mononuclear cells (PBMC) of healthy individuals.⁷⁵ The possible role of HERVs in human pathology (autoimmune diseases and cancers) has received considerable attention,⁸¹ as has the classification of their numerous families.⁸²

Since 1996, real-time PCR has been used to claim quantification of a postulated HIV viremia, termed “viral load,” in AIDS cases. These methods have been based on the study of patients’ plasma samples: initially, samples originated from nuclei of peripheral blood mononuclear cells, and later from low-speed centrifugation pellets of plasma.⁸³ The various methods applied to the PCR measurement of the so-called “viral load” have one point in common: they all bypass direct isolation of retroviral particles demonstrable by EM. These methods are not expected to isolate, nor concentrate any retrovirus. Moreover, as clearly stated during the South African 2000 conference,²⁶ not one single particle of retrovirus has ever been seen, by EM, in the blood plasma of any AIDS patient, even in those patients identified as presenting with a high so-called “viral load.” That statement, widely publicized, has never been refuted nor challenged.⁸⁴

Human plasma carries various amounts of circulating DNA. Suspected for a long time, this was first demonstrated by modern technologies in 1999, by P. Anker et al.,⁸⁵ in the blood of cancer patients. The significance of circulating nucleic acids, as possible molecular markers in the study of cancer, was extensively reviewed in a New York Academy of Sciences conference in 2006.⁸⁶ The origin of free circulating DNA is complex, and seems to depend primarily on cell apoptosis. “If the engulfment of apoptotic bodies is impaired or cell death is increased enough to produce substantial amounts of circulating DNA, inflammation would definitely be a problem and autoimmunity would occur frequently in cancer and other conditions involving increased circulating DNA.”⁸⁷

Apoptosis and a large spectrum of infectious diseases are constant components of all clinical AIDS cases. Circulating DNA is expected, therefore, in the plasma of all symptomatic AIDS patients. Amounts can vary, as a function of more or less rapid removal of DNA by clearance mechanisms. Apoptotic bodies and/or fragments of PBMC nuclei are certainly expected in low-speed centrifugation plasma pellets, such as those used in PCR “viral load” measurements, and most likely increase the amount of recognizable DNA. Human DNA always contains approximately 8% of retroviral nucleotide sequences. It’s no surprise, therefore, that RT-PCR study of plasma pellets shows, and amplifies, retroviral nucleotide sequences. Unfortunately, such findings are frequently misinterpreted as originating from hypothetical exogenous “HIV,” although, as stated above, not one single retroviral particle has ever been found by EM in plasma samples. Quantifying a presumed “viral load” has, therefore, probably nothing to do with an exogenous “HIV.” It simply reflects variable amounts of circulating DNA.

Retroviral sequences in plasma pellets being easily explained by the presence of variable amounts of circulating DNA, one should not, however, expect that these nucleotide sequences would be identical in all cases. Quite to the contrary, since “nucleotide sequences that diverged from co-linearity with the typical retroviral genome (LTR-gag-pol-env-LTR) considerably increase the number of HERV families,”⁸² the large number of HERV families resulting apparently from frequent recombinational deletions.⁸⁸ Expected variations in the observed nucleotide sequences have, unfortunately, often been misinterpreted as an indication for a high rate of HIV mutations! It seems much more likely, however, that the numerous variations in the observed retroviral nucleotide sequences in circulating DNA reflect the large number of HERV families they originate from, and have nothing to do with presumed “mutations” of a hypothetical HIV.

Reference to HERVs and/or to circulating DNA can hardly be found in the extensive literature on “viral load” measurements, interference of HERVs, and of circulating DNA being consistently ignored by the HIV/AIDS orthodoxy.

Conclusively, RT-PCR identification, and presumed quantification of so-called “HIV viral load,” can easily be explained by the variable amounts of HERV-derived retroviral nucleotide sequences present in the circulating DNA of AIDS patients.

Retroviruses on the Surface of Cord Blood Lymphocytes

In their 1983 *Science* paper,⁵² Barré-Sinoussi et al. failed to demonstrate, by EM, any retrovirus in their co-cultures. Still, the supernatant of these co-cultures has been used to “infect” human cord blood lymphocytes. This theory requires one to subscribe to infection via a virus that is not visible by EM. If the authors had included EM evidence for retroviruses in their co-cultures and their supernatant, their interpretation would have been more convincing. Unfortunately, such data were not provided.

Nevertheless, their Figure 2 unquestionably demonstrates “budding”⁸⁹ retroviruses on the surface of cultured human cord blood lymphocytes. Its origin needs to be better clarified.

Cord blood lymphocytes are placenta-derived cells. The human placenta is well known for its high content of HERVs,⁷⁸ with EM-recognizable retrovirus particles.⁷⁹ Cord blood lymphocytes are, therefore, likely to carry similar HERVs. The 1983 paper⁵² demonstrated that HERV particle expression had been successfully activated in cultured cord blood lymphocytes, under culture conditions that included 2g/ml of Polybrene. It does not demonstrate, however, that the EM-observed retroviruses originated from the studied pre-AIDS patient. A long-overdue control experiment would be to study, by EM, cultured cord blood lymphocytes under conditions that would reproduce exactly those used at the Pasteur Institute in 1983. Dourmashkin presented some data addressing this issue in 1992,⁹⁰ although his presentation did not satisfactorily resolve the problem, since his cord blood lymphocytes were not cultured under conditions identical to those used at Pasteur in 1983.

The EM observation of typical retroviral particles in the 1983 Pasteur paper can alternatively be explained by the presence of placenta-derived, Polybrene-activated HERVs. However, this EM observation does not support the existence of an AIDS-related, exogenous retrovirus.

Obviously, confounding by HERVs cannot be ignored in the objective analysis of clinical as well as basic HIV/AIDS research.

Discussion

All AIDS Rethinkers are united in the fundamental opinion that HIV is not the cause of AIDS.⁹¹ However, they diverge on the important question of the very existence of the Human Immunodeficiency Virus (HIV).

Some of them⁵¹¹ maintain that HIV is a “harmless passenger virus,” while others^{23, 25} claim that HIV “does not exist” at all. Since neither of these two positions explains the pertinent observations, an alternative interpretation, compatible with all the available scientific evidence, is needed.

Claiming that HIV is a harmless passenger virus raises at least two critical problems. First, if HIV is “harmless” it cannot be linked to immune deficiency (a very severe pathological condition), as implied in its name. Therefore, the name of the virus should at least be changed in order to fit with a claimed “harmless” character. Secondly, in the general classification of animal virology, very large numbers of viruses are nonpathogenic, as was well illustrated in the 1960s in a special conference, at the New York Academy of Sciences, under the title “Viruses in Search of Diseases.” Obviously, all nonpathogenic (i.e. “harmless”) viruses are clearly visible under the EM. Pathogenic and nonpathogenic viruses look identical under the EM. In AIDS research, retroviral particles were observed by EM only in complex cell culture systems,⁵⁰ never directly in the plasma, nor in the tissues of any AIDS patient.

Claiming simply “HIV does not exist” is not satisfactory either, because it fails to explain the two sets of data discussed in this review, namely the presence of retroviral genomic sequences in the plasma of AIDS patients, and the EM evidence for retrovirus particles in the “historical” 1983 Pasteur paper.⁵²

Others have previously emphasized that HERVs cannot be ignored and that they actually represent “confounding factors for human retrovirus discovery.”⁹² Their role having been confirmed and amplified, this review shows that HERVs, in addition, offer a rational, alternative interpretation for the two above-mentioned problems.

The existence of endogenous human retroviruses has been known for some time, but their interference in HIV/AIDS research has yet to be widely appreciated. Of course, HIV should not be considered an HERV, since the hypothetical HIV is supposed to be an exogenous, infectious microorganism, while HERVs are fundamentally endogenous, non-infectious, vertically transmitted, defective viruses. Still, HERVs have been a “confounding” factor in HIV/AIDS research,⁹² and have caused confusion in interpreting the concept of “viral load.” Moreover, HERVs put HIV researchers on the wrong track, creating the illusion of continuous HIV mutations—mutations that improperly served to explain the extreme difficulty in preparing anti-HIV vaccines. However, difficulties in developing anti-HIV vaccines might not be explained by a constantly mutating HIV, but rather by a lack of exogenous HIV.

As emphasized years ago by Papadopoulos,²³ Lanka,⁹³ and others,⁹⁴ there is no scientifically verifiable evidence to confirm the existence of a hypothetical exogenous HIV. However, stating simply that “HIV does not exist” is an incomplete statement that fails to explain the complexity of HIV/AIDS research. To that statement, one should always add that HERVs have heavily interfered with HIV/AIDS research in a way that cannot be ignored. Adequate understanding of HERVs as confounding factors opens the way to a better, more objective analysis of AIDS research.

Finally, the question as to whether HIV exists, or of whether researchers have been studying a harmless passenger virus, is a question that should be subject to open debate and careful consideration of scientific evidence or lack thereof. Alternative explanations for findings should be decided by the scientific evidence, not by consensus. The advancement of our understanding of AIDS demands nothing less.

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