# Can Electron Microscopy Resolve The HIV Battle? An Exclusive Interview with EM Pioneer Dr. Étienne de Harven

I have known Belgian scientist (pathologist and electron microscopist) Dr. Étienne de Harven since 1993, when he introduced himself to me at a conference, with characteristic graciousness and enthusiasm, clasped my hands, and said he had translated my 1992 SPIN article *Fatal Distraction* into French.

I was of course delighted. I was writing about this scientific battle for a rock and roll magazine, and he was a very distinguished scientist– one of the pioneers of Electron Microscopy. His utter lack of snobbery, his warmth and openness, set the tone for a friendship spanning all the years since– rooted in exchanges that are in no way sterile, not mere transfer of "information."

I wanted to understand de Harven's "poetic space–" that something I knew he was seeing when he peered into this abominable mess called HIV science.

Over the years, we've sat in bistros in New York, or on the patio of his marvelous house in Saint Cezaire, in the south of France, or in my cramped kitchen in New York City–and we've worked on it. "It" being the Mystery.

HIV.

Or rather, "HIV."

When I didn't exactly "understand" the science at hand, I understood the expressions on his face, the depth of emotion in his eyes when he spoke of "what Gallo did." For scientists like Dr. de Harven, "what Gallo did," and all that followed, represents a kind of holocaust on all they'd known and taken for granted: Empirical, classical science.

Now retired, Dr. de Harven's life's work has revolved around Electron Microscopy, a pioneering technology now at the center of the explosive trial in Buffalo New York, where Nushawn Williams, after serving 12 years in prison, and still being in state custody due to "mental abnormalities," he is said to possess, has been found to be HIV free on electron microscopy (EM) tests.

Amidst political machinations surrounding the attempted media blackout from the trial– coercion and even possible witness tampering; the internet is ablaze with new disputes as to whether being HIV negative on EM is a "valid" way to be considered HIV free, or negative. As we all know, the church is built entirely upon the now discredited HIV antibody tests and PCR viral load tests, both of which state as disclaimers in package inserts that they are not designed to test for HIV.

*The Truth Barrier* contacted Dr. de Harven as well as The Perth Group, with questions about the Nushawn Williams case and how it relates to the HIV existential debate, and these materials will be published as the story unfolds. There is disagreement about how EM tests must be performed in order to properly discern whether HIV is present or absent in the blood.

But what does this thing, not word, bit of code–"HIV"–mean?

The "debate" about HIV's causation has been, I'm devastated to realize, obscured by the sheer fact that we used as concepts, symbols, words that lacked meaning. We began by saying "Does HIV cause AIDS," when we should have said, "*What do we mean when we say "HIV*?"

If you have the mental stamina to unravel it, please read the writings collected at The Perth Group's website. I have certainly failed on this front–trying to unravel *this*. It was the hardest part of the gigantic knot, but it was the most important, by far. The knot ties all perceptions, illusions, shadows, linguistics, hallucinations, and stunning truths together at its center: HIV's existence (as exogenous "retrovirus," in human blood, not as technological artifact,) the "validity" of the "HIV test," the question of "cause" and pathogenicity, and the question of infectivity.

To accusations that I have not dealt with this adequately, I am guilty as charged.

In other words: I have mostly (not entirely) wasted the past 27 years of my life as an AIDS unraveler, because I did not start at the epicenter: The existential question–Not *does* it, but *is* it?

"Does HIV Cause AIDS?" already contains accumulated detritus, constructed words, and what William Burroughs called, the "set up."

*The Truth Barrier* is delighted to bring you this Q&A with a true expert on EM. Dr. de Harven is the former President of the Electron Microscopy Society of America.

[Note to readers: 1. The questions were emailed and responded to in writing, not asked in sequence, so there will be passages where the question asked suggests that the interviewer has not heard or comprehended the answers given.]

**INTERVIEW**

Q: Tell us about electron microscopy, EM: What is it? What need did it answer when

it was pioneered, and what role did you play in it?

A. There are several types of electron microscopes (EMs). One that brought, by far, the most important contributions to bio-medical research is the “transmission” electron microscope (TEM).

I shall, therefore limit my remarks to the birth of TEM. The inventor of TEM is Ernst Ruska, who constructed and successfully operated the first TEM, in collaboration with Knoll, in Berlin at the research laboratories of the Siemens CY, in 1931. The instrument was aimed at developing a microscope offering a “resolution” better than that of the optical microscope. By “resolution”, we mean the shortest distance separating two punctual objects that could still clearly be recognized as two, and not one. The resolution of the optical microscope is limited at the level of 0.2 micrometer (“microns”; one micron = one thousandth of a millimeter). This limitation was recognized as making the optical microscope totally unable to visualize viruses. By contrast, the resolution of the TEM is around one Angström unit. One micrometer equals 10.000 Angström. It follows that the resolution of the TEM is approximately one thousand times better than that of the optical microscope, making the direct visualization of viruses, and even of single atoms possible. Most viruses are definitely smaller than 0.2 micrometer. The size (diameter) of viruses was approximated before they were actually visualized, from the analysis of the average pore size of ultra filters through which these viral particles could go through, testing the infectivity of “ultra-filtrates.”
Ruska definitely demonstrated, in 1931-32, that his electron microscope had, indeed, a resolution better than that of the optical microscope. His microscope (the first TEM) was using a beam of electrons instead of a beam of light, and using electro-magnetic fields, generated by several magnetic “lenses”, to deflect there pathway instead of optical lenses. The technology involved in this instrument has several points of similitude with that of the cathodic TV tube. For this discovery, Ruska received the Nobel Prize in 1986, two years before he died. The hope of Ernst Ruska was that his new microscope could, possibly, visualize viruses, and he actually was thinking about the poliovirus! Still, in the mid 1930s, many biologists thought that this new microscope could never be useful in biological research, fearing that biological specimens would inevitably be destroyed by the electron beam–like burned out by a lightning.
This was demonstrated to be false at the University of Brussels, Belgium, in 1936, by Louis Marton who published the first EM images of cells, taken with an EM of his own making. The first EM made in America was constructed at the University of Toronto, Toronto, Ontario, at the Banting Institute, in 1938. Important improvements, aimed at correcting some image defects, were developed within the laboratories of the RCA CY, in Camden, PA, during WWII.
Just after WWII, Albert Claude (Nobel, 1976), using an RCA microscope and working at the Rockefeller Institute in New York, succeeded in imaging the Rous sarcoma virus, observed within infected, cultured cells. This was definitely the first application of EM to the direct visualization of what we now call retroviruses. In 1955, I was fortunate to operate the very first “Elmiskop I” from the Siemens CY in the USA, installed at the Sloan Kettering Institute, in New York City, and with which I made early contributions (1956-1960) to the ultrastructure of murine leukemia viruses, to their “budding” phenomenon, and to their purification from the blood plasma of leukemic mice. That microscope had a resolution far superior that of the US made RCA instruments.

By the early 1960s, all the known viruses had been well characterized under EM, and,

unquestionably, TEM has been a major factor in the emergence of modern virology, as well as of modern cell biology.
Nobelist André Lwoff recommended, at a Cold Spring Harbor conference in 1962, that the general classification of all viruses be primarily based on their morphology, as seen by EM.
[Emphasis mine, ed.]

By 1970 however, and in spite of a most extensive, worldwide research effort, not a single virus was ever demonstrated by TEM to be significantly associated with any form of human cancer or leukemia.

Q: What happened exactly vis a vis "HIV" and EM, in 1983/84? Was patient blood serum (HIV Elisa/WB positive) validated against EM? Ever? It seems to me they say that yes, it was, yet we have Montagnier saying, "We did not purify." Let me elaborate the question a little:

What should have happened, vs. what did happen, with the "new virus" and EM, in this period of time?

A. What happened in 1983 is a direct consequence of what developed since 1970, i.e. the highly predominant reliance on "molecular markers".

Unacceptably frustrated by the total lack of success in all attempts to demonstrate virus particles in human cancer by EM, the "impresarios" of the cancer/virus "dream" (Gallo, Fauci, and others) totally engaged in the molecular approach.

Consequently, they invented molecular markers to compensate for the missing viral particles... This would have been acceptable if the specificity of these new molecular markers would have been clearly established. Unfortunately, this was not the case.
The most misleading molecular marker was probably the first one, i.e. the enzyme reverse transcriptase (RT). Following Temin and Baltimore 1970 papers in "Science", the RT enzymatic activity has been, most abusively, used as a specific retroviral marker. Both Temin and Baltimore demonstrated RT activity in samples of supposedly "purified" retrovirus.
Embarrassingly, they both omitted to verify the "purity" of their samples by EM. Some of their samples were simply purchased from a commercial company... True, the label on the vials read "pure retrovirus"... However, it was known that these commercial "pure retrovirus" were heavily contaminated by cellular debris!

And since it is also known that all cells contain RT (see Varmus), cellular debris are most likely carrying similar RT enzymes.
Temin and Baltimore did not, therefore, prove that RT is a specific molecular marker for retroviruses. It would have been so simple to check, by EM, the degree of "purity" of the samples they used. This would have, most probably, shown important cell debris contamination, and would have obliged Temin and Baltimore to be much more cautious in the interpretation of their results. In 1975, the members of the Nobel Committee, most regrettably, failed to scrutinize this "purity" problem...

In 1983, at Pasteur Institute in Paris, reliance on the RT marker was a key element in the claimed "isolation" of a new retrovirus. Still, Montagnier himself recognized "We did not purify"... He dangerously omitted to consider the misleading interference of cell debris, just as Temin and Baltimore did in 1970.
But a paper on the discovery of a new retrovirus looks much better if it contains at least... one EM picture! So, members of Montagnier's team spent hours at the TEM,

looking at their mixed cell cultures, and they found the virus!
See Fig. 2 in their "historic" 1983 "Science" paper! It is, by the way, a good quality EM picture. It shows unquestionable retroviral particles, budding at the surface of a cell. But the legend of this Fig. 2 states that this cell is a cord blood lymphocyte. Indeed, cord blood lymphocytes were admixed to these complex cell cultures (why?) Montagnier and his co-workers should have known that human embryonic tissues, and the placenta in particular, are very rich in endogenous retroviruses (HERVs), and that cord blood lymphocytes should therefore be expected to carry the same endogenous retroviruses (under the TEM, endogenous and exogenous viruses, looking identical, cannot be distinguished.)
The budding of these particles has perhaps been stimulated by some of the growth factors also present in these cell cultures. An essential control would have been to repeat the experiment using lymphocytes from the peripheral blood instead of from cord blood. This control is unfortunately missing.

In short, I would frankly state that the Pasteur 1983 paper (whose 30th anniversary has just been celebrated in a "grand messe" of official HIV retro-virology!) contributed very little in AIDS research because its conclusion (i.e. "the isolation of a new retrovirus") is based on 1)the use of a non specific RT molecular marker, and 2) is falsely supported by EM pictures of, most probably, endogenous human retroviruses.

More details and appropriate references on this analysis can be found in my 2010 paper published in the Journal of American Physicians and Surgeons (www.jpands.org/vol15no3/deharven.pdf).

Q: When antibody and VL tests became widespread as diagnostic tools for "HIV infection" over the ensuing decades, what happened with EM inside of HIV science and literature? It is my understanding that nobody has ever found HIV in human blood, on EM. Is this an accurate way to say it?

A: In my views, Western Blot tests lost all credibility after the publication of Eleni Papadopulos's et al. (1993) paper, and antibody tests ("Elisa") after Christine Johnson's report( 1996). The notion of a "Viral load"(VL), however, brought a new parameter in AIDS diagnosis (Ho,1996). It called attention to the actual number of HIV particles supposedly present in the blood plasma of AIDS patients, PCR technologies being presumed to offer a way to quantify that number.

If such a viremia (i.e. presence of virus particles in the blood) is indeed present in AIDS patients, it reminisces the retroviral viremia well known in leukemic mice. In such case, retroviral particles should be readily demonstrable, by TEM, of appropriately prepared patient plasma samples. Unfortunately, it as never been possible to demonstrate by TEM one single retroviral particle in the blood plasma of any AIDS patient, even if one selects patients presenting with a so-called "high viral load."
I was apparently the first researcher to make that statement, during the opening session of President T. Mbeki's major AIDS conference, in Pretoria, SA, in May 2000. My statement to that effect has never been refuted.

How come?

That question must be answered because "something" is measured by PCR technologies in the blood of many AIDS patients. Actually, what is being measured is definitely not the number of retroviral particles (phantom-like, i.e. EM invisible!). In

fact, what is being PCR identified, amplified, and supposedly quantified is the number of genomic nucleotide sequences that are extremely similar to sequences known to be part of the retroviral genome. Most regrettably, these sequences were misinterpreted as an indication as a certain number of … HIV particles! This did a lot to consolidate the quasi-religious dogma of HIV as the cause of AIDS, a dogma that as been sharply criticized, a few years ago, by David Rasnick who wrote, authoritatively, about "The AIDS Blunder"…

This interpretation would have been acceptable only if retroviral particles would have been readily demonstrated, by EM, in the blood plasma of these patients; but, since this is not the case, another explanation for the presence of these nucleotide sequences has to be founded.

I presented at the RA conference in Oakland, CA, in 2009, and further developed in my 2010 JAPS paper such a much needed explanation for the presence of these retroviral-like nucleotide sequences. My explanation is based on the well known, variable amounts of circulating DNA in the blood of severely ill patients, and on the fact that we all carry retroviral-like sequences in our DNA, as endogenous, defective retroviruses, i.e. HERVs (HERVs, for "Human endogenous retroviruses")(See "Virus in all of us", R. Lower at al., 1996 PNAS paper).

No surprise, therefore, that these nucleotide sequences are recognized by PCR in the blood of many AIDS patients, who are indeed severely ill. As already demonstrated in 2008 in Robin Weiss laboratory, HERVs can interfere as confounding factors in the search for novel retrovirus in chronic human diseases.

In addition, "viral load" clinical data will remain very hard to interpret, as long as the essential control is missing. The essential control would be to search for "viral load" in serologically negative (HIV-), severely ill patients, ill from advanced cancer or from infectious diseases, NOT FROM AIDS. Unfortunately, this essential control has never been done, since the so-called "Viral load" has, so far, been exclusively searched for in HIV+ patients…

Conclusively, measuring "viral load" does not prove the existence of a hypothetical HIV.

Q: If so, if that is true, then what does it mean? Paint a picture for us. The story of the virus, the "new deadly virus," what happens first: What steps did they Montagnier, on one hand, Gallo on the other take to "find" the new entity? Then once they 'found' it, what shape was it in? It was not an entity, a thing, with a body, right? It was not coherent. Can we say that? So it lived where? It was seen only through the technologies developed to find it, Elisa, WB? Later PCR/VL? But what happened back THEN when they tried to see it on EM? Why didn't everybody look for it on EM? Too expensive?

A: No, EM is not cheap but not that expensive! And its cost has certainly nothing to do with the fact that it has barely been used for the past 30 years in AIDS research! It has not been used because "They" knew it was not going to show anything of retroviral significance in samples coming directly from AIDS patients. And since AIDS had become big business, the stocks of involved giant pharmaceutical companies could not be jeopardized! It had to be saved at all cost, even at the cost of trusting non specific molecular markers… Fear is good business, and viruses generate fear most efficiently… So, the HIV flag has to be maximally agitated. In worldwide medias, with thousands of computer-generated, colorful caricatures of an idealistic

retrovirus... By contrast, the medias have been dominated by the most rigorous censorship when it comes to inform the public about views of rethinking dissidents. This total censorship put a safety lock on any information that could jeopardize the colossal, entirely HIV derived profits of the major pharmaceutical companies.

But I am glad we have Internet!

Daring to say that HIV does not exist amounts to some sort of a capitalistic crime...

Yes, the HIV dogma is probably the darkest page in the history of modern medicine.

Q: What was your reaction when you saw the recent story that Nushawn Williams does not "have HIV," despite being repeatedly positive on antibody and VL tests over the decades.

A: I am not, unfortunately, familiar enough with phylogenetic analysis methodology to provide a critical view on this case. I was impressed, however, by the recent (June 1st and 2nd, 2013) e-mailings between Georg von Wintzingerode and David Crowe on this topic, and I would suggest to read that correspondence for elaborate questions on the analysis of Nushawn Williams story. Still, one point I wish to make is that, as far as I know (?), DNA/RNA phylogenetic analysis is currently performed exclusively on HIV antibody positive people(?). Where are the essential phylogenetic controls on antibody negative people? If indeed that control is also missing, then the significance of such studies is wide open for questions, just as much as the significance of the alleged HIV "viral load" (see above) that has also never been searched for among HIV antibody negative patients.

Q: Etienne, if you could sum up: Does HIV exist? If so, where and how and as what? If you could examine 1,000 HIV positive people's blood under EM, what would you expect to find? If you don't find HIV on EM in human blood, can any argument be made that the virus is "hiding" and so forth, or that the drugs suppressed the virus to undetectable levels? This is what the defenders of the orthodoxy seem to be saying about the results seen in the Nushawn Williams case.
A: This is the main question! Questioning the very existence of HIV is not something that should be debated only between specialized retro-virologists. It is an essential question that concerns all of us.

Why?

Simply because 100% of AIDS research funding is based on the dogmatically postulated existence of HIV. If HIV does not exist, it would follow that AIDS research is the most appalling case of total misappropriation of public research funds! And it would also follow that the monumental amounts of money, so far exclusively devoted to HIV research, would be much better used in other directions. Could you imagine what world we would live in, today, if the total amount of money wasted over the past 30 years on HIV research had been, instead, used for feeding starving Africans, for clean water supply equipment, for public hygiene infrastructures, and for public health education? This would happen only if HIV research is totally stopped! And for this, the scientific and public health organizations have to face the fact that, indeed, HIV does not exist!

If, to the contrary, one keeps talking about HIV as... possibly hiding, or possibly harmless, or possibly endogenous, then the waste of research funding on HIV research shall endlessly be tolerated.

Instead, we all have to, courageously, face the fact that the very existence of an exogenous HIV has never been scientifically verified.

True: to prove that something does not exist is never an easy task. But it is the responsibility of the scientific, orthodox establishment to bring us the proof of the opposite, i.e. the proof of the existence of HIV as an exogenous retrovirus responsible for the causation of AIDS. And such a proof has to be based on classic virology methods, not on the use of questionable molecular markers. So far, the hypothetical HIV has never been properly isolated, nor properly concentrated, nor even ever purified. As long as this remains the case, the specificity of HIV molecular markers shall not be accepted.

The Perth Group (PG, Eleni Papadopulos and Valendar Turner et al.) had repeatedly, in the early 1990, stressed the notion that in view of the considerable difficulty encountered in all attempts to isolate/purify HIV, the specificity of HIV molecular markers was most uncertain.

In 1994, German virologist Stefan Lanka raised major questions about the very existence of all exogenous retroviruses.

Soon afterwards, the “PG” strongly emphasized that HIV had never been properly isolated. During the international, large debate held at the European Parliament, in 2003, I further stressed the problems encountered in isolating HIV. But stressing the difficulty to isolate a virus remains short of stating that this virus does not exist, and is, consequently, not enough to stop all research on this virus.

As already emphasized, the appalling waste of HIV research funds must stop, and these enormous research funds should, most urgently, be re-affected towards completely different, non-retroviral aims. This will happen only if the worldwide scientific establishment courageously faces the fact that HIV does not exist. The fact that HIV does not exist is actually not be so surprising for all those who realize that, after almost 30 years of research, based most exclusively on a non-scientifically verified HIV hypothesis, 1) not one single AIDS patient has ever been definitely cured by ARVs, 2) apocalyptic epidemiological predictions never materialized, and 3) not one single efficient vaccine has ever been developed...

Obviously, we were, for 30 years on the wrong track!

ERRARE HUMANUM EST, SED DIABOLICUM PERSEVERARE, i.e. Error is human, but it is devilish to persevere!

Conclusively:

-we all carry, in our chromosomes, defective endogenous retroviruses (HERVs) that have interfered, most presumably, as misleading, confounding factors in AIDS research;

-“HIV” is not an endogenous retrovirus;

-“HIV”, as an exogenous, AIDS causing retrovirus simply does not exist, and this fact should be recognized as soon as possible for a complete, radical re-distribution of AIDS research funds, worldwide.

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[References: The relevant materials cited in this interview can be found here, here and here.]