Heinrich Kremer: The Silent Revolution in Cancer and AIDS Therapy

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Chapter VII

Collective Tunnel Vision

Why "HIV characteristics" are the outcome of evolutionary biological programming, and are not specific causes of strong and/or continuous immune stress – what the "HIV test" really measures

The Nobel Prize winner for Chemistry in 1993, the American research scientist Karry Mullis, gave an account of his experiences while searching for scientific references for the disease theory "HIV is the probable cause of AIDS". His account is a staggering document of contemporary history and highlights the mass psychological staging of "the most devastating epidemic of the 20th Century" (Gallo 1991).

"In 1988 I was working as a consultant at Specialty Labs in Santa Monica, setting up analytic routines for the Human Immunodeficiency Virus (HIV). I knew a lot about setting up analytic routines for anything with nucleic acids in it because I had invented the Polymerase Chain Reaction. That's why they had hired me."

The Polymerase Chain Reaction (PCR) is a laboratory process in which the tiniest DNA fragments can be amplified. The prerequisite is that the DNA sequences, which flank both ends of a given region of interest in DNA, are known and that the corresponding short start sequence is available for the beginning of the amplification process. PCR is today one of the most important methods in all medical and molecular biological laboratories (Wirthmüller 1997). Karry Mullis received the Nobel Prize for Chemistry in 1993 and in his acceptance speech addressed the problem of evidence for the hypothetical retrovirus HIV as supposed cause of AIDS. His speech is the only one in over 100 years of the Nobel Prize that was not published.

The futile search of the Nobel Prize winner Mullis for the original publication showing "HIV is the probable cause of AIDS"

"Acquired Immune Deficiency Syndrome (AIDS), on the other hand, was something I did not know a lot about. Thus, when I found myself writing a report on our progress and goals for the project, sponsored by the National Institutes of Health, I recognized that I did not know the scientific reference to support a statement I had just written: "HIV is the probable cause of AIDS". So I turned to the virologist at the next desk, a reliable and competent fellow, and asked him for the reference. He said I didn't need one. I disagreed. While it's true that certain scientific discoveries or techniques are so well established that their sources are no longer referenced in the contemporary literature, that didn't seem to be the case with the HIV/AIDS connection. It was totally remarkable to me that the individual who had discovered the cause of a deadly and as-yet-uncured disease would not be continually referenced in the scientific

papers until that disease was cured and forgotten. But as I would soon learn, the name of that individual - who would surely be Nobel material - was on the tip of no one's tongue. Of course, this simple reference had to be out there somewhere. Otherwise tens of thousands of public servants and esteemed scientists of many callings, trying to solve the tragic deaths of a large number of homosexual and/or intravenous (IV) drug-using men between the ages of twenty-five and forty, would not have allowed their research to settle into one narrow channel of investigation. Everyone wouldn't fish in the same pond unless it was well established that all the other ponds were empty. There had to be a published paper, or perhaps several of them, which taken together indicated that HIV was the probable cause of AIDS. There just had to be. I did computer searches, but came up with nothing. Of course, you can miss something important in computer searches by not putting in just the right key words. To be certain about a scientific issue, it's best to ask other scientists directly. That's one thing that scientific conferences in faraway places with nice beaches are for. I was going to a lot of meetings and conferences as part of my job. I got in the habit of approaching anyone who gave a talk about AIDS and asking him or her what reference I should quote for that increasingly problematic statement, "HIV is the probable cause of AIDS". After ten or fifteen meetings over a couple years, I was getting pretty upset when no one could cite the reference. I didn't like the ugly conclusion that was forming in my mind: The entire campaign against a disease increasingly regarded as a twentieth century Black Plague was based on a hypothesis whose origins no one could recall. That defied both scientific and common sense.

Finally, I had an opportunity to question one of the giants in HIV and AIDS research, Dr Luc Montagnier of the Pasteur Institute, when he gave a talk in San Diego. It would be the last time I would be able to ask my little question without showing anger, and I figured Montagnier would know the answer. So I asked him. With a look of condescending puzzlement, Montagnier said, "Why don't you quote the report from the Centers for Disease Control?" I replied, "It doesn't really address the issue of whether or not HIV is the probable cause of AIDS, does it?" "No," he admitted, no doubt wondering when I would just go away. He looked for support to the little circle of people around him, but they were all awaiting a more definitive response, like I was. "Why don't you quote the work on SIV [Simian Immunodeficiency Virus]?" the good doctor offered.
"I read that too, Dr Montagnier," I responded. "What happened to those monkeys didn't remind me of AIDS. Besides, that paper was just published only a couple of months ago. I'm looking for the original paper where somebody showed that HIV caused AIDS". This time, Dr Montagnier's response was to walk quickly away to greet an acquaintance across the room" (Mullis 1996).

The astonishing confession from the "HIV" discoverer Montagnier, that the self-defined standards for genuine retrovirus isolation were ignored

Beside Dr Gallo, the former director of the Laboratory for Tumor Biology of the National Cancer Institutes in the USA, Dr Montagnier is a worldwide authority on HIV. Since 1983 he is considered to be the discoverer of the "retrovirus HIV". Together with Gallo he is the patent holder for the "HIV test" and earns a certain share on every "HIV test". Montagnier has led the research team for viral cancer research at the world-famous Pasteur Institute in Paris since 1972 and has been the leader of the AIDS and retroviral department at the same institute since 1991. Dr Montagnier has also been President of the World Foundation for Aids Research and Prevention since 1993.

In 1997 Dr Montagnier gave a detailed scientific interview in which in answer to the final question about his belief in the existence of "HIV" he stated: "Oh, it is clear. I have seen it

and I have encountered it". In the same interview Montagnier explains why the electron microscopic photographs of the supposed "new agent" produced and publicized by his research team were made only from cells in the cell culture and not, in accordance with the standard rules of virology, after the purification of the supposed virus particle from the remaining cell debris: "There was so little production of virus it was impossible to see what might be in a concentrate of virus from a gradient" (density gradient: the separation of the tested material in a sucrose solution after ultracentrifugation of the cell fluids of the stimulated cell culture. This process is one of the standard rules of isolation of a retrovirus).

"There was not enough virus to do that. Of course one looked for it, one looked for it in the tissues at the start, likewise in the biopsy. We saw some particles but they did not have the morphology typical of retroviruses. They were very different. Relatively different. So with the culture it took many hours to find the first pictures. It was a Roman effort! It's easy to criticise after the event. What we did not have, and I have always recognised it, was that it was truly the cause of AIDS" (Tahi 1997).

Montagnier was referring in his interview in 1997 to findings in cell cultures of T-helper immune cells. These experiments were carried out by Montagnier's viral cancer research group at the Pasteur Institute in 1982/1983 and publicized in the leading scientific journal *Science* in March 1983, the same month as the historic conference in New York postulated a "new agent" as the cause of AIDS. The findings of the Montagnier team were not presented at the historic conference, but only the discovery by the Gallo team of a supposed retrovirus HTLV-1 in T-lymph cells of two American AIDS patients. The T-lymph cells analyzed by the Montagnier team originated from the blood serum of patients who had shown clinical and immunological signs of a Th1-Th2 switch. The cells were treated in a culture with a growth factor (type-1 cytokine interleukin-2) and heavily oxidized substances like PHA as mitogens. After a few days physical and biochemical analyses were carried out both on the surface of some of these cells and in the cell fluids. These resulted in unspecific findings. In 1972 researchers at a symposium at the Pasteur Institute - Montagnier has led the research team for viral cancer there since 1972 - established the standard practice for the isolation of retroviruses from cell cultures, which were supposed to exclude the possibility that unspecific findings from cell cultures and cell fluids of human cells could be confused with specific characteristics of retroviruses (Sinoussi 1973, Toplin 1973, Bader 1975, Papadopulos-Eleopulos 1993a). In the publication of the Montagnier team in *Science* of March 1983, which later was deemed to be the first publication about the discovery of the "human immune deficiency virus HIV", the authors emphatically refer to the standard rules of isolation of retroviruses in human cell cultures from 1972, as proof for the isolation of a "new retrovirus" in human T-helper immune cells (Barré-Sinoussi 1983, Montagnier 1985).

The most important standard rule for isolation of a retrovirus in human cell cultures is the separation of the cell material gained in the cell culture from all cell debris other than the cell particles thought to be retroviral, which after stimulation of the cell culture have budded out of the cell membrane. The cell particles that are considered retroviral particles have to be separated from the cell culture fluids by ultracentrifugation and absorbed in a sucrose solution. From experimental analysis it is known that during this process retroviruses band at a certain level known as the density gradient. 1.16 gm/ml is the accepted level. Molecules, cell debris, virus particles and non-virus particles from the centrifuged cell fluids of different cell cultures all band at this density gradient as the components separate not according to molecular weight but according to the density of the components in the sucrose solution. In order to be sure that, as far as possible, only the supposed virus particles have banded at the 1.16 gm/ml density gradient, purification and concentration procedures have to be carried out,

as only the particles at the density gradient may be tested as to whether they whether correspond in diameter and volume to the supposed retroviral particles that were ascertained by studying electron-microscopic (EM) photographs during maturation from the cell membrane. As there are many non-viral particles in stimulated cell cultures whose shape, structure and appearance cannot with absolute certainty be distinguished from actual retroviruses, the contents of the particles have to be biochemically processed after isolation by purification. The proteins of the particle's membrane together with proteins from the inner particle membrane, including the characteristic enzyme proteins of retroviruses and nucleic acids, have to be exactly identified by a routine molecular biological process.

Only if structurally completely identical proteins and nucleic acids are present in the isolated and purified particles, and only if the nucleic acids in these particles form RNA instead of DNA molecules, can it be assumed as a probability that they are retrovirus particles. The findings can only be considered to be sure proof of the existence of a retrovirus in human cells when the RNA molecules in these particles construct genes which present the coded instruction for the biosynthesis of proteins within this particle and if these proteins can actually be identically synthesized. Once these findings have been verified it is still not possible to say whether these retroviral particles are exogenic, transmittable and infectious retroviruses as they could be endogenic retroviruses that have often been identified in the center of the genotype of many human cell types and are not infectious. In order to enable a differentiation between exogenic and endogenic retroviruses in human cells the properly isolated and biochemically characterized retroviruses would have to be transferred to human cell cultures, mature out of the cell again, it has to be separated again from any cell material by means of proper isolation and purification, successful isolation would have to be confirmed by EM photographs, the biochemical identity of the proteins and nucleic acids have to be substantiated and the RNA of the particle as coded genotype for the specific protein synthesis of the retrovirus particle would have to be verified.

In an interview in 1997 Montagnier admitted that he and his colleagues did not carry out purification of the cell particle. "I repeat, we did not purify" (Tahi 1997). Montagnier also conceded that only an "assemblage of properties" of the cell particle was detected by his team on the cell membrane and only components were found in the density gradient. No EM photographs were published to show which cell material had banded at the density gradients. Nevertheless, non-identified proteins from the density gradients were used as substrate for the "HIV test" patented by Montagnier in 1983 with the claim that they were retrovirus proteins of the "newly isolated HIV".

The financial battle between the Pasteur Institute in Paris and the National Cancer Institute of the USA, litigation over the patent rights to the "HIV test"

The Pasteur Institute receives half its finance from the state and the other half through the production of vaccines, test diagnostics etc. The Pasteur Institute, in competition with the Gallo team from the National Cancer Institute in the US, very definitely had commercial interests in capturing the worldwide market for test substrates against the "new AIDS agent". What in fact happened was that in 1983, three months after the first publication of the Montagnier team's "isolation" of a new retrovirus in T-helper lymph cells from the blood serum of risk-group AIDS patients, the license for the Pasteur Institute's vaccine against hepatitis B was cancelled, for example, in Germany and Switzerland. Instead the use of hepatitis B vaccines from the US was recommended. This hepatitis B vaccine was approved in the US in October 1981 and in Germany and Switzerland in October 1982. The justification for the international ban on the Pasteur Institute's substrate was, according to a confidential

official memo: “suspicion of AIDS contamination of the French vaccine”. Although the American and French vaccines were extracted from comparable human cell cultures the National Health Authority’s suspicion of “AIDS contamination” only fell on the Pasteur vaccine. In the same issue of *Science* in which the Montagnier team reported about the “isolation” of a “new human retrovirus”, the Gallo team published about the “isolated retrovirus HTLV” in T-helper lymph cells of homosexual patients, allegedly carried out in 1980 (Marx 1983, Barré-Sinoussi 1983). By mid-1983 the irrational notion of a “lethal AIDS/sex plague” was psychologically programmed on the basis of a few hundred cases of “infection” amongst anal receptive homosexuals with a long-standing abuse of nitrite inhalants and antibiotics. By mid-1983 an interaction between retroviral and cancer laboratory specialists, national health authorities and the mass media had long established that AIDS diseases were the consequence of a “new agent” and a “lethal new plague of sex and blood”. It was only a question of who would control the “invisible hand of the market” for the marketing of the worldwide test substances. The Gallo team evidently needed to win time to come up with the decisive laboratory trick of how to isolate enough “HIV” in order to produce enough “HIV proteins” for mass testing. “HIV production” in a test tube was not sufficient for this purpose. In September 1983, Gallo announced that the Montagnier team could not have discovered a “new retrovirus” as they could not provide evidence of continuous “HIV production”. In an interview in 1997 Montagnier stated: “For example Gallo said: “They have not isolated the virus...and we (Gallo et al.), we have made it emerge in abundance in an immortal cell line.” But before making it emerge in immortal cell lines, we made it emerge in cultures of normal lymphocytes from a blood donor. That is the principal criterion” (Tahi 1997).

Gallo and Montagnier’s statements are both objectively misleading and are based on deliberate acts of deception.

But in the economic war between the French and the Americans the Americans initially retained the upper hand. The denunciation of the Pasteur Institute’s hepatitis B vaccine as “AIDS contaminated” had its effect. Montagnier’s patent application for an “anti-HIV antibody test” was rejected in the USA, the patent application of the National Cancer Institute for Gallo’s “anti-HIV antibody test” was authorized in record time even before Gallo had published one line in scientific journals about the “isolation of HIV” and the development of an “anti-HIV antibody test” on the basis of proteins from his “isolated HIV”. Only after year-long litigation between the USA and France were the patent charges for the “anti-HIV antibody test” awarded equally to Gallo and Montagnier at a conference between President Reagan and the then Mayor of Paris, Chirac and then given in an apparently noble gesture to the World Foundation for AIDS whose president Montagnier was to become. In reality this absurd dispute over the patent rights served as a distraction from the real problem – namely the fact that neither Gallo nor Montagnier had “isolated” a human retrovirus and that the source of the proteins for “HIV tests” had by no means been established as having a retroviral origin. The worldwide public was led to believe that when two specialists from such renowned research institutes like the Pasteur Institute and the National Cancer Institute quarrel about the honor of discovery, then “Public Enemy No.1” (President Reagan 1984) must really exist and be the cause of the “most devastating epidemic of the 20th Century” (Gallo 1991), and also that the “AIDS test” would protect the world population from this “deadly mass plague”.

The ramifications of the psychological mass suggestion of a “new plague of sex and blood”

Nobel Prize winner Mullis' "little question" as to the original references that demonstrated that "HIV is the probable cause of AIDS", which neither Montagnier nor any other specialists would or could answer, answered itself by virtue of scientific historical facts. Fear of plagues is deeply anchored in the archaic subconscious of mankind as a legacy of evolutionary experience. It is very easy to trigger especially in association with sex and blood. When non-stop images are shown on television and in other mass media of condemned, relatively young people in connection with a "puzzling plague" and it is suggested that this "deadly incurable mass plague from the malicious depths of Nature" affects homosexuals today and tomorrow every man, woman and child, then people are scared. Then the question of whether the causes of the disease are correct will already have been answered, before a rational analysis could even be initiated. The all-or-nothing approach is adopted when confronted with archaic fears. You are either frightened of not. Reason confirms with hindsight what apparently the emotions already had known. With the continuous barrage of horrific images in the media and the statements of medical authorities, apparently in the know, terms like "HIV" and "AIDS" become provisory triggers whose objective truths can no longer be queried. The majority of people surrender helplessly to suggestive manipulation. There are plenty of object lessons in the history of the 20th Century in totalitarian systems and also in the world of modern media. Psychologists name this process "operant conditioning". When a non-specific, diffuse prospect of fear is associated contemporaneously with enough concrete stimuli or projections, the appearance of the stimulus without a concrete reason for fear is sufficient in the future, to search for and find a collective consensual protective mechanism for fear. There is no need to seek out original scientific references to prove whether the statements associated with these fears are justified. They are secure as they have been collectively internalized as a "healthy strategy". The people who are considered dangerous are those who reject the apparent dangers through rational analysis. The standard answer, since the "outbreak of AIDS epidemic" amongst uninformed doctors remains to this day "Do you want send millions of people to a certain death?" or "You should be happy that the young are frightened of something during sex and are more careful". When reasons are given as to why the false disease theory "HIV causes AIDS" is a danger to millions of people and that making the sex life of young people neurotic by means of HIV/AIDS propaganda detracts from the real dangers, you get at most a look of "condescending puzzlement" just as Nobel Prize winner Mullis did from Dr Montagnier in reply to his "little question" (Mullis 1998). After verifying that there were no scientific references to show that "HIV is the probable cause of AIDS", Mullis publicly described this fact as one of the greatest scientific scandals of the 20th Century (Mullis 1998).

The self-delusions and other mistakes of the "HIV discoverers" and the "disastrous results"

In the first publication about the "isolation of a human retrovirus" in the T-helper immune cells of AID and AIDS patients in 1983 the Montagnier team claimed to have followed all the procedural steps according to the standard rules of isolation of a retrovirus with the exception of electron-microscopic photographs of the density gradients. Such photographs are, however, of crucial importance to verify which proteins are present in the band of the density gradient. An arbitrary choice of these proteins may not be used as antigen substrate for the "HIV test". If decay proteins from the stimulated human cells in the cell culture have banded in the density gradient, then "HIV positive" will register as increased quantities of antibodies are found in the blood serum of test subjects which formed naturally against decay proteins inside the organism, against alloantigens (e.g. foreign semen after anal intercourse) or against microbial antigens. These antibodies in "HIV positive tests" then also react against decay proteins from the foreign human cells of the cell culture. This means, however, that millions of patients have received the medical death sentence of a "lethal retroviral infection" as a

consequence of a natural antibody reaction and in countless cases the patients have been treated with highly toxic drug cocktails which have been proven to trigger AIDS and cancer. The electron-microscopic control of the protein components at the density gradient in the "isolation of HIV" and the construction of the "HIV test" is of immense importance to a large number of people and their dependents.

Yet, between 1983 and 1997, the EM photographs of the protein components of the density gradient were not published either by Montagnier, Gallo, or any other retro-virologist. The very first EM photographs of the density gradient of "HIV isolation" were published in March 1997 by two research groups, fourteen years after the first apparent "HIV isolation" by Montagnier and Gallo. These EM images showed "disastrous results", as judged by Professor De Harven, one of the pioneers of the use of electron-microscopy for the control of the isolation of retroviruses in mammalian cells (De Harven 1998 a). The first EM photographs, as truth test for the cell material of the density gradient after "HIV isolation" from human cells showed "practically only cell material" from human cells of the cell culture (Papadopulos-Eleopulos 1998 a). So, fourteen years after the alleged "first isolation of HIV" and thirteen years after the introduction of "HIV tests" it transpired that the retrovirus cancer researchers Montagnier and Gallo feigned the "isolation of HIV" and that the cell proteins for the protein antigens of the "HIV test" were nothing more than decay proteins from human cell cultures. The test result "HIV positive" meant the reaction of natural, if increased, antibody levels in the blood serum of test subjects.

Whatever else the retrovirus cancer researchers fabricated by laboratory trickery, the omission of EM photographs of the test proteins for the "HIV test" objectively amounted to grievous bodily harm, in uncounted cases leading to death, as patients as a rule were treated with highly toxic pharmaceutics, which could trigger AIDS and cancer. The explanation of Montagnier in an interview in 1997 is significant in understanding the grave charge that is based on firm scientific documents. Montagnier suddenly denies that he and his research team adopted the standard rules of isolation of retroviruses during the "first isolation of HIV". This statement is a direct contradiction to the claims in several publications by Montagnier and his colleagues in which it is explicitly reported, with reference to the rules determined by the Pasteur Institute in 1972, that these procedures were carried out for the "isolation of HIV", excluding the crucial EM cell material images at the density gradient after centrifuging the cell fluids of the suspected retrovirus cell culture of lymph cells from the AID and AIDS patient's blood serum (Barré-Sinoussi 1983, Brun-Vezinet 1984, Vilmar1984, Rey 1984, Klatzmann 1984, Montagnier 1985). As to the question of whether the standard rules for the isolation of retroviruses (Sinoussi 1973), defined at a symposium at the Pasteur Institute in 1972 and which he and his colleagues referred to in the initial publication of the "Isolation of HIV" were respected – namely:

- "culture [the suspected T-lymph cells], purification of the material by ultracentrifugation, Electron Microscopic (EM) photographs of the material which bands at the retrovirus density, characterisation of these particles, proof of the infectivity of the particles"

Montagnier answered unequivocally in an interview in 1997: "No, that is not isolation" (Tahi 1997).

There are only two possible explanations for this response from one of the leading HIV specialists in the world:

- Either Montagnier and his team, contrary to the claims in their publications, did not follow the standard rules of retrovirus isolation during the “isolation of HIV”, in which case these publications are scientific falsifications
- Or they did follow standard procedures and realized that the required results – the existence of human retroviruses in the T-immune cells of AID and AIDS patients – were not to be proven, in which case we are dealing again with a serious case of scientific falsification.

The fact that Gallo and his team published the same procedure as the Montagnier team for “HIV isolation” and that the Pasteur team sent the cell fluids from its cell cultures to the Gallo laboratories in 1983, speaks against the first possibility (Popovic 1984). In an interview in 1997 Montagnier admits that in 1983 he had seen only unspecific factors for the existence of retroviruses, but that through an assemblage of unspecific factors his mind’s eye perceived the retrovirus “HIV” and this speaks for the latter possibility: “It is not one property but the assemblage of the properties which made us say it was a retrovirus of the family of lentiviruses. Taken in isolation, each of the properties isn't truly specific. It is the assemblage of them. So we had: the density, RT, pictures of budding and the analogy with the visna virus. Those are the four characteristics” (Tahi 1997).

All four characteristics are absolutely unspecific, they are found in many human cells, normal cells and cancer cells in cell cultures (overview with Papadopulos-Eleopulos 1998 a). For fifteen years (1982-1997), both Montagnier and Gallo and all other retrovirologists dispensed with the only specific characteristic, a highly specific characteristic, namely the EM control of the density gradient after purification by ultracentrifugation, in order to be able to determine whether human cell proteins or retrovirus proteins were to be found in the density gradient for the “HIV test” construction. This is a routine matter in laboratories. “Dangerously enough, EM was progressively dismissed in retrovirus research after 1970. Molecular biologists started to rely exclusively on various “markers”, and what was sedimenting in sucrose gradient at density 1.16 gm/ml was regarded as “pure virus”. It is only in 1997, after fifteen years of intensive HIV research, that elementary EM controls were performed, with the disastrous results recently reviewed in *Continuum*. How many wasted efforts, how many billions of research dollars gone in smoke... Horrible. Errare humanum est sed diabolicum perseverare... [To err is human. To repeat error is of the Devil]” (De Harven 1998 a).

So Montagnier and Gallo must have blindly fished out of their “cell soup” protein antigens for the test substrate of the “HIV test”, a test which in positive cases meant horrific suffering and death for millions of people, for the healthy and ill “HIV positives”, for those with symptoms and those without. “How sad it is to think that a simple EM control of such “bands” (which takes about two days, and costs a few hundred dollars, but has never been done before 1997) could have prevented these highly misleading interpretations of “markers” (De Harven 1998 a).

It is, however, very difficult to imagine that of the thousands of highly specialized laboratory scientists in countless retrovirus research laboratories all of which have an electron-microscope on hand, with a budget of over 200 billion dollars in research funds, nobody in 15 years thought of running an EM control of the sucrose gradient in the “cell culture soup”. The acceptance of such a syndrome, in light of the alleged threat to mankind of a “deadly mass HIV plague”, required a devout deference to authority. Gallo, Montagnier and a few of their colleagues were proclaimed as “HIV discoverers”, consultants in chief and lords of the greatest capital investment in modern medical history. The majority of retrovirologists profited knowingly and silently from the funding. “It was like having a money-printing

machine in the basement, when you based a funding application on HIV, irrespective of what you wanted to research, the funds flowed. Without HIV every funding application was a lottery" (anonymous laboratory researcher about research practices in the AIDS era).

The most absurd thing about the statements from Montagnier and his co-workers is the fact that every interpretation of the observed phenomena in the cell cultures, the cell fluids, the density gradients and the interaction of cell cultures had to be objectively false. In the 1980s there was still a lack of knowledge amongst laboratory researchers and clinicians about the fundamental processes in human cell systems. The less they definitely knew, the more dictatorial the assertions that the research findings had been properly rendered (Epstein 1996, Lang 1996).

"The emergence of acquired immunodeficiency syndromes (AIDS) in 1981 gave the retrovirus establishment an opportunity to transform what could have been only an academic flop into a public health tragedy"

The first trailblazing insights of NO research, cytokine research, mitochondrial research and many other research fields have heralded a radical change in the understanding of many apparently puzzling phenomena. For the first time it is possible to recognize all phenomena in the context of "HIV isolation", "HIV test construction" and "HIV test findings" as being within the bounds of evolutionary biological laws. The insights suggest a more than strong suspicion that Montagnier, Gallo and their colleagues had not only examined the four unspecific standard characteristics for the isolation of retroviruses in human cells, named by Montagnier in an interview in 1997, but had also made up the standard characteristic – the EM control of the material in the sucrose solution in the density gradient. The logical reason for this assumption is the fact that the Montagnier and Gallo teams had made publications of EM photos of budding particles from the cell membrane of human cell cultures that they had stimulated with strong oxidizing substances. Why, then, would they not be interested in checking these EM findings with a control of the purified materials in the density gradient. Both teams have, to this day, sold these unspecific EM photos of budding from the cell membrane as evidence for the existence of "HIV". The budding of particles from stimulated cells is not specific evidence for retrovirus particles, not even if this particle arouses unspecific initial suspicions that it could be a retrovirus particle. Presenting the unsuspecting public with budding EM photos as evidence of the "existence and isolation of HIV" is a premeditated act of deception and a deliberate scientific falsification. Noted virologists have clearly proven that: "budding virus-like particles from the cell membrane can be ascertained in numerous non-infected normal cell lines and transformed cell lines, in T-cell lines, in transformed B-cell lines; and in cultures of primary human lymphoid cells from cord blood, which were either PHA stimulated or not and grown with or without serum and in cord lymphocytes directly after Ficoll separation". (Dourmashkin 1991, overview with Papadopulos-Eleopulos 1993 a, 1993 b, 1996, 1998 a, 1998 b). Such budding particles have been demonstrated in the cell membranes of cells in enlarged lymph nodes in 90% of cases of "HIV associated" AID and AIDS patients as well as in the cell membranes of cells in enlarged lymph nodes in 87% of cases of non-"HIV associated" AID and AIDS patients (O'Hara 1988). In the same way as the embryonic umbilical cells and the transformed cells, all cases were concerned with cells after a Th1-Th2 switch and a type-2 cytokine dominance. Montagnier and Gallo knew nothing at the beginning of the 1980s about the existence of the two T-helper lymph cell populations, Th1 and Th2. They also did not know that these cells synthesize different cytokine profiles and that this was the reason why they showed differing patterns in the regulation of cellular symbiosis. They also did not know that T-helper cells produce or do not produce NO depending on the redox status. They also did not know that,

depending on the redox status and the stimulation by oxidizing substances, T-helper cells activate counterregulations that either accelerate or inhibit apoptosis/necrosis and activate glycolitic energy production from outside the mitochondria.
The latter, however, meant an increase in lactate formation and the export of cell debris, accrued by proteolytic enzymes activated by the lactate. The budding of virus-like particles from the cell membrane is the garbage collection of the type-2 cytokine cells. The proof is the findings in typical Th2 cells: transformed T- and B-cell lines, embryonic lymphoid cells in umbilical tissue, productive lymph nodes independent from antibody levels obtained in the "HIV test" (Dourmashkin 1991, O'Hara 1988).

But Montagnier and Gallo definitely had a dire need to confirm, by means of EM photos of the materials in the sucrose solution, their initial suspicion, on the basis of the budding EM photos, of being on the trail of a "new retrovirus". If, after ultracentrifugation, there was a banding at the density gradient of predominantly retrovirus-like particles, then it could be possible by biochemical identification of the proteins and amino acids of this particle to prove that it was a new retrovirus. The disappointment must have been immense when, exactly like the first EM photos from 1997, practically only cell debris from the human cells of the stimulated cell culture was represented (Bess 1997, Gluschankof 1997). Such EM photos were the counter-evidence of the "isolation of a new agent". Proteins from human cell debris are not suitable to be sold as protein antigens for a worldwide antibody test. So Montagnier and Gallo declared the four unspecific characteristics for "HIV" for specific by a generous interpretation of the set theory– four times minus equals plus (Montagnier in interview 1997, Tahi 1997). They expounded the unspecific budding EM photos as specific evidence of a "new agent HIV". The specific EM photos of the sucrose gradient were not published; the unspecific budding EM photos were sufficient to quench the thirst of the mass media craving for images of the plague. Photofit images of "HIV" were later supplied together with computer designed monster-like excrescences, so-called knobs and spikes, to stimulate the collective plague fantasies, how the "new agent of the deadly mass plague" enters the immune cells and how it goes about its deadly deeds (see illustration: HIV photofit [table IX]). In reality the best-known electron microscopist for "HIV" EM photos had published that the "HIV particle" on average only exhibited 0.5 knobs per particle. These protein complexes on the cell membrane of the "HIV particle" are supposed to be decisive for the infectiousness of "HIV". This protein with a molecular weight of gp120 is one of the proteins - the alleged "HIV protein" in the "HIV test" – that reacted to the antibodies in the human serum and can produce positive results in the test. The EM researchers established that knob-like structures could be observed, even when gp 120 proteins were not present, the EM results could then be falsely positive (Layne 1992). But how is "HIV" without the gp 120 configuration supposed to dock onto the immune cells, if, according to the unanimous view of the "HIV" researchers it could not possibly be infectious without it? The act of deception with the budding EM photos and the gp 120 protein complex for the "HIV test" is of crucial importance to the virus hunters for the optical staging of the trick series. As the human sensory organs receive some 11 million bits per second, of which roughly 10 million are optical bits, pictorial presentation is the best means to generate fear or fancy. From a mass psychological viewpoint it is not to be assumed that Montagnier and Gallo had forgone the opportunity to produce considerably more specific EM photos of the sucrose gradient as there were very convincing EM photos of retrovirus particles in the sucrose gradient after purification by ultracentrifugation even in the 1960s. Such EM photos were published by, amongst others, the Pasteur Institute under Montagnier and by the National Institute of Cancer in the USA under Gallo. These EM photos were taken of cell cultures of mice suffering from leukaemia. EM photos of particles from human cancer cells and other cell types after purification could, however, never be demonstrated. The scientific contemporary witness, De Harven, who as one of the pioneers of

retrovirus research had already shown in the 1950s convincing EM photos of purified retrovirus particles from animal cancer cells at the most famous American cancer research center – the Sloan-Kettering Institute in New York- stated: “In the 1950s and 1960s many EM cancer research centers [in the USA and Europe], were spending a considerable amount of time in attempts to demonstrate virus particles associated with human cancer cells. “Virus-like particles” were occasionally reported but convinced nobody. Typical viruses were never conclusively demonstrated. This was in sharp contrast with the highly reproducible demonstration, by EM, of viruses in a variety of murine and avian leukemias and tumors. Very few papers were published to report on these negative findings in human cancers and leukemias. However, Haguenau, in 1959, reported on the difficulty of identifying any typical virus particles in a large series of human mammary carcinomas. Bernhard and Leplus, in 1964 in an EM survey of cases of Hodgkin's disease, lymphosarcomes, lymphoid leukemias and metastatic diseases failed to recognize virus particles associated with these malignant conditions. At Sloan Kettering in New York, I decided, in 1965, to stop surveying cases of leukemias and lymphomas by EM for the presence of viruses in view of our entirely negative results. This was reported at a conference on Methodological Approaches to the Study of Leukemias held at the Wistar Institute, in 1965 (Haguenau 1959, Bernhard 1964, De Harven 1965). Publication of these negative findings failed to discourage fanatical virus hunters! An explanation for these negative results had to be found somewhere! Perhaps the technique of EM by the thin section method was not the best approach? (although it worked perfectly for mice!). Preparing thin sections was time-consuming and skill-demanding! Who had time for that, when research funding was getting difficult, and when major pharmaceutical corporations were starting to finance “crash programs” for speedy answers? ... It became acceptable to postulate that when viruses cannot be seen by EM in cancer cells, biochemical or immunological methods supposedly identifying viral “markers” were enough to demonstrate viral infection of the cells under scrutiny. Such markers can be an enzyme (RT), an antigen, various proteins, or some RNA sequences. Never seeing the viral particles was conveniently explained by the integration of the viral genome into the chromosomes of the alleged infected cells. To accommodate such interpretations implied complete oblivion of all we knew from previous research on cancer of experimental animals …” (De Harven 1998 b).

The fact that the leading lights of “HIV isolation” and the patentees of the “HIV antibody test”, Montagnier and Gallo, were not in the position to produce a specific EM photo of purified cell debris and instead of this were able to feign for 17 years the unspecific budding EM photos as the official “wanted” poster for “HIV” just goes to show that counter controls no longer work in medical research: “As far as scientific policy is concerned, research on potentially oncogenic viruses was dominated by the retrovirus hypothesis. Federal funding took the same direction, amplified by the incredibly naive idea that success was primarily a matter of money! Unusually large levels of federal support resulted in the creation of a retrovirus research establishment. Large numbers of research jobs were created in this venture. The intellectual freedom to think along other avenues of cancer research was rapidly dwindling, especially when major pharmaceutical companies started to offer tantalizing contracts to support polarized retrovirus research... The top priority was to demonstrate, at any cost, that retroviruses had something to do with human cancer, a hypothesis, however, which didn't receive the slightest support throughout the 1970s. Such a misdirected research effort would have been relatively inconsequential as long as public health was not involved. Unfortunately, the emergence of acquired immunodeficiency syndromes (AIDS) in 1981 gave the retrovirus establishment an opportunity to transform what could have been only an academic flop into a public health tragedy” (De Harven 1998 b).

The remaining unspecific characteristics can also be explained by the concept of cell dyssymbiosis. The Montagnier team and the Gallo team specified as second characteristic, besides the budding of cell particles from the stimulated human T-helper lymph cells, evidence of the repair enzyme reverse transcriptase (Barré- Sinoussi 1963, Popovic 1984). Such enzymes transcribe the RNA sequences to DNA sequences. From an evolutionary-biological viewpoint, information storage began in living cells, with less stable RNA codings, as “RNA world” (overview with De Duve 1991). DNA coding is, however, more stable and in all living cells the carrier of information for protein synthesis in the cytoplasm. Mobile messenger RNA, after redox dependent activation, is transcribed to the DNA form. Up until 1970 the biological dogma was that flows of genetic information always ran from DNA to RNA. As Temin and Baltimore discovered in 1970 that there is also a counter-flow of information from the RNA form to DNA form, they termed the enzyme involved reverse transcriptase (RT). At first it was thought that this enzyme was present exclusively in animal cell identified RNA tumor viruses (retroviruses). This assumption was soon recognized as a mistake. RT is active in all eukaryotic cells and in bacterial cells (Temin 1972, 1974, 1985, Baltimore 1985, Varmus 1987, 1988).

The claim by Montagnier and Gallo that RT was fished out of the density gradient 1.16 gm/ml, which was shown to contain Montagnier’s impure cell debris from the cell fluid (Tahi 1997), and that this find is exclusive proof of the presence of “HIV” and so the proteins from the same density gradient are exclusively “HIV proteins” is a grave scientific deceit. As Montagnier and Gallo did not purify the material from the banding by ultracentrifugation and also did not wish to verify the presence of retrovirus particles with EM photos, in order to disguise the absence of retrovirus particles (!) the whole process becomes a dreadful game with concealed cards. Montagnier said in an interview in 1997, after the 1997 EM photos (Bess 1997, Gluschankof 1997) had exposed the non-existence of the retrovirus particle in the sucrose gradient which had been intimated since 1983: “I repeat, we did not purify … Gallo? … I don't know if he really purified. I don't believe so” (Tahi 1997).

The safest and clearest procedures for the isolation of retroviruses from stimulated human cells was not employed and substituted by the objectively false statement that evidence of RT is exclusive proof of the “isolation of HIV”. As the discoverer of RT, the Nobel Prize winners Temin and Baltimore and Nobel Prize winner Varmus (Temin and Baltimore 1972, Varmus 1987,1988) had unequivocally shown that RT features in all cells, the presence of RT in the cell debris of cell fluid from human cell cultures, which were stimulated with strong oxidizing substances, was to be expected.

Montagnier and Gallo as well as all the other HIV researchers were only able to demonstrate “HIV characteristics” in stimulated cells (Klatzmann 1986, Papadopulos-Eleopulos 1993, 1998 a). The stimulation of the cell cultures took place as a result of the addition of strong oxidizing mitogens (PHA, Con A etc.) and type-1 cytokine interleukin-2 (IL-2). IL-2 activates, however, Type-1 cytokine interferon-γ (IFN-γ). IFN-γ stimulates cytotoxic NO production. As the Montagnier team and the Gallo team stimulated T-helper lymph cells (TH cells) from homosexual AID and AIDS patients and it has been demonstrated that such patients show a TH-2 dominance (overview with Lucey 1996), these cells have mostly been counterregulated, this means on activation of NO synthesis by interferon-γ these cells either die by apoptosis or necrosis, as IL-2 also stimulates tumor necrosis factors and as a consequence increasingly ROS. Or the cells increase the counterregulation to protect against apoptosis/necrosis which means that by activating the type-2 cytokine profile (IL-4, IL-5, IL-10 etc.) the formation of the enzyme COX-2, prostaglandin (PGE) and the repair enzyme, transforming growth factor (TGF) is increased. PGE and TGF suppress the NO synthesis and

activate from arginine the metabolic path for the synthesis of polyamines that stimulate the proliferation and repair mechanisms. The synthesis of reverse transcriptase, the enzyme for the neosynthesis of DNA from RNA, also belongs to the repair sequence.

“HIV positive” tested people have a conspicuously low thiol pool in T-helper cells. Both the cysteine levels in the blood plasma are lower as well as the intracellular glutathione levels in the T-helper lymph cells that are on average 30% lower (Dröge 1988, Eck 1989, Buhl 1989, Gmünder 1991, Roederer 1991 a, 1991 b, Harakeh 1991, Kinscherf 1994, Dröge 1997 a, 1997 b, Herzenberg 1997). Thiol depletion is to be expected in long-term stress in which a number of factors are involved and is a specific expression of a profound cell dyssymbiosis in AID and AIDS patients. The antioxidative and antinitrosative emergency status of the T-helper immune cells can only be remedied by massive counterregulation or apoptosis/necrosis. The stimulation by IL-2 and heavily oxidized mitogens and antigens of these thiol depleted immune cells in the cell culture triggers oxidative and nitrosative stress. Considerable cell damage can be expected through this provocation of the synthesis of cytotoxic NO gases, which in the cell culture also diffuse to the neighboring cells. The net effect is that the RT levels also increase in counterregulated cells.

In plain language the procedures of the virus hunters meant that they placed the immune cells that were already dyssymbiotic under artificial prooxidative/nitrosative stress, inevitably provoking apoptosis/necrosis and /or increased counterregulation. Finally the released cell products, including RT and other cell specific proteins, RNA and DNA molecules as well as the export particles of the stressed cells (budding) are interpreted by chicanery as unspecific, but then again apparently *in toto* specific, characteristics of a “new retrovirus”. The “HIV test” is assembled using disintegrating proteins with the full awareness that people with an increased antibody count (Th2 status with increased antibody production) and/or cross-reacting antibodies and autoantibodies according to the natural laws could show an antigen antibody reaction on contact with human foreign protein. The selection of the “HIV positive” stigmatized is governed by an arbitrary specification of the measuring threshold of the test, this means at which point of sensitivity the antigen antibody reaction counts as “positive”. With this technique of measuring a targeted haul can be made within the population as a whole as it is already known which particular subgroups will be stigmatized. Subsequently the necessity was suggested to those who had been scared to death at being stigmatized, for a long-term therapy with highly toxic pharmaceutical combinations to combat the phantom retrovirus that a few thousand laboratory specialists with a simple EM photo costing “a few hundred dollars” were apparently unable to conceive for 17 years. Then again the extremely expensive pharmaceutical cocktails, the unfailing implementation of the “planned experiments on human beings” promoted at the historic conference of the *manqué* retrovirus cancer researchers in March 1983, have been shown to cause prooxidative/nitrosative stress and compound cell dyssymbiosis. The circle closes, the pharmatoxic consequences, to the point of fatal organ failure, are interpreted as “specific characteristics” of the “lethal HIV infection”.

The “HIV” test measures an antibody reaction against what is put into the test substrate: indeterminate proteins excreted by repeatedly stressed human immune cells

Countless experimental studies with all kinds of cell types have proven that the “four HIV-specific characteristics” (Montagnier) observed in cell cultures can equally arise in “HIV negative” cell cultures under stimulation stress. When HIV researchers say that they have infected previously “non-infected cell cultures” with “HIV” it always means that they have stimulated oxidative/nitrosative stress (without stimulation stress there are no “HIV

characteristics"!) and that these cells, dependent on cell type or the state of cell symbiosis, have reacted with counterregulations. The subsequent diagnosis of "HIV positive" in the cell culture is reached in that budding was observed, a portion of RNA (or DNA!) was re-modelled to DNA in an artificial initial sample, and that this is regarded as evidence for the presence of the enzyme RT, that the one or the other protein is proven by molecular weight, as occurs in every cell, but that this one is defined as an "HIV protein" in advance and that possibly these proteins will react to antibodies in animal or human blood serum. In the last case it is claimed, from a scientific viewpoint incorrectly, that these antibody reactions are specific and exclusively a reaction to "HIV proteins".

In reality there are no "specific" antibodies. Rather there are special protein molecules that are formed by the million each day by B-lymph cells in every human being. They are positively charged whereas T-lymph cells are negatively charged. Antibodies have varying charging centers on the molecular surface. When they come into contact with an antigen, mostly proteins but also other molecules, then one or more of the positively charged centers on the antibody react to one or more negatively charged centers on the antigen. The antibodies are, so to say, the molecules with the positive plug that fit into the antigens' negative socket. If you have a negatively charged antigen many different antibodies with roughly the same positive "plug" can fit the negative "socket" of the same antigen. Conversely many different antigens with the same negatively charged "sockets" can couple with positive "plugs" of the same type of antibody. This process is called cross-reaction. The logic is that the more different antibodies a person has formed in the blood serum in increasing amounts, the higher the statistical probability is that they will react on contact with antigens. It is not known, however, whether these antibodies were originally produced "specifically" against these particular antigens or against other antigens. As antibodies remain a long time in blood serum, sometimes lifelong, it cannot be stated for certain whether the antibody activating incidents are backdated or persist from the time of the test (Guilbert 1985,1986, Pontes de Carvalho 1986, Chassagne 1986, Termynck 1986, Matsiota 1987, Parravicini 1988, Gonzalez-Quintial 1990, Berzofsky 1993, Fauci 1994, Owen 1996, Papadopulos-Eleopulos 1998 a).

The claim of the retrovirologists that a positive "HIV test" signifies a "specific" antibody reaction against "HIV proteins" is thus consciously misleading. As Montagnier admitted in an interview in 1997, originally he and Gallo brought the "unpurified" protein mixture from their "real cell culture soup", as Montagnier described it in the same interview (Tahi 1997), into contact with the serum from AID and AIDS patients. If there was shown to be an antigen antibody reaction in the serum, which was by no means the case in all the serums, then the reaction as such would be published as evidence that "HIV" must exist and that the patients must be suffering from AID and AIDS because they had become infected with "HIV". This meant that T-helper immune cells extracted from the serum of immune-stressed AID and AIDS patients were exposed to a further immune stress through oxidizing substances in a cell culture. The highly oxidized proteins, released by the overstrained immune cells by cell disintegration or cell particle export were ultracentrifuged and then banded in a sugar solution, but not purified and identified. Finally a sample was taken from the material that had banded at a particular density (a level at which according to procedures in earlier experiments with animal cancer cells, retrovirus particles, amongst other things, had concentrated) – blindly and without identification of the material. Taking the protein as antigen from the sample it was allowed to react with antibodies from the blood serum of likewise immune-stressed AID and AIDS patients. Nobody, not even the most imaginative retrovirologist, could even with the remotest semblance of certainty say which proteins from the cell culture had reacted to which antibodies from the serum of AID and AIDS patients. It could only be said that if the proteins from the immune cells of immune-stressed patients were treated

prooxidatively/nitrosatively in a laboratory and the released proteins were made to react with antibodies from the serum of equally immune-stressed AID and AIDS patients, then an antigen antibody reaction can be seen. This arcane process and the alleged evidence that unspecific repair enzyme RT is released in immune-stressed immune cells after immune stress in a cell culture is up until now the only evidence for the "existence and isolation of HIV" as well as for the "infection with the immune cell killing HIV".

The first "HIV test" process was called the ELISA test. It was said later that the ELISA test was wrongly positive in 90% of the positive results. In reality all ELISA tests are wrongly positive as without proper isolation of a retrovirus no test can be constructed that can display the antibody formation against a retrovirus. Later the proteins from the blind sample from the cell culture soup were run through an electric field and defined as "HIV proteins" a handful of proteins that stood out distinctively according to particular molecular weights. In laboratory jargon this process is called Western Blot. The ELISA test becomes the screening test and Western Blot the confirmatory test. If the ELISA test is positive twice then a subsequent Western Blot test is carried out. If this is also positive then the patient is considered to be "HIV positive". In reality the Western Blot test also does not measure antibody formation against a "retrovirus HIV". It measures an antibody reaction against what was put into the test substrate: undefined proteins, released by repeatedly stressed human immune cells.

The screening test also cannot find what it is looking for, namely the human antibodies against a "retrovirus HIV" that Dr Gallo and Dr Montagnier also failed to find. The confirmatory test also cannot confirm the antibody formation against "retrovirus HIV", as Dr Gallo and Dr Montagnier only saw "unspecific characteristics" and for the only specific characteristic that could confirm the existence of a "retrovirus HIV", namely EM control after purification of the cell fluids, they evidently did not want to expend "two days time and a few hundred dollars" (De Harven 1998 b).

Nevertheless, can the "HIV test" give information about the condition of a human's immune system? As the test substrate contains human proteins it can only indicate the degree of sensitivity that a test subject reacts to human proteins. From this alone it cannot be deduced why a proband reacted to a particular protein with antibodies of a particular intensity. There might be, for instance, a cross-reaction between antibodies against tubercular bacteria, leprosy bacteria, pneumocystis carinii fungi, candida fungi and the proteins of the "HIV test". There are many other cross-reactions that have still been little researched (Mathews 1998, Calabrese 1989, Müller 1990, 1991, Ezekowitz 1991, Tumijama 1991, Kion 1991, Kashala 1994, O'Riordan 1995, Fraziano 1996, Papadopulos-Eleopulos 1997 c). The test cannot provide information about the concrete significance of an antibody reaction against the proteins of the "HIV test" for the past, present or future of test subjects. At the very most a test can indicate that the proband could have Th2 dominance with an increased antibody production. The "HIV test" has no more informational value than the simple DTH test that measures the reaction of the skin lymphocytes to antigen stimulation. As was demonstrated with surgical patients (Christou 1986) the DTH skin reaction could give significant indications about which patients could develop sepsis after severe traumata, burns and operative intrusions. In this respect the DTH skin reaction is superior to the "HIV test" as it indicates the readiness of stimulation of the Th1 immune cells. As the "HIV test" merely shows an increased antibody sensitivity, which can have a lot of different or evident causes in excessive immune stress, and as antibodies are not decisive for the development of opportunistic infections and certain cancer forms but for the elimination capability of Th1 immune cells for an intracellular agent and the antioxidative capacity for the efficiency of cell symbiosis, the "HIV test" is unsuited as an aid to diagnosis and prognosis. But as the construction of the "HIV test" is based on sham

particles this test should be banned internationally as quickly as possible and the groundbreaking work of NO research, cytokine research, mitochondria research and other seminal research fields should be implemented into medical practice.

The experimental findings of the Montagnier team as counter-evidence against the disease theory "HIV is the cause of AID and AIDS"

From the numerous experiments with different cell cultures, two experiments from Gallo and Montagnier should demonstrate how they themselves have shaken their "HIV causes AIDS" theory (Zagury 1986, Laurent-Crawford 1991). At the time of the cell experiments the fact of the existence in humans of different cytokine profiles (type-1 and type-2) of the T-helper lymph cells, Th1 cells and Th2 cells, were not yet published. Likewise the fact that during excessive or long-term immune stress of the redox milieu a re-programming of the cytokine profile of the T-lymph cells can take place. These facts were finally verified in 1991 (Romagnani 1991). The additional fact that Th1 cells and Th2 cells can be differentiated as Th1 cells produce cytotoxic NO gas after stimulation with interleukin-2, while Th2 cells suppress the formation of cytotoxic NO gas was only demonstrated in 1995 (Barnes 1995). In this respect the consequence of a Th1-Th2 switch to Th2 dominance was also not known then, namely the fact that after severe immune stress Th2 dominance impairs or totally prevents the elimination of intracellular agents. The further consequence that with a Th2 dominance massive counterregulations are triggered resulting in a cell dyssymbiosis in mitochondria and that these cells export highly oxidized proteins by transporting particles from the cells as well as producing an increase in repair enzymes instead of NO gas production was also at that time not understood.

The Montagnier team (Laurent-Crawford 1991) provoked by mitogen and cytokine stimulation (PHA, interleukin-2) in three human cell lines the appearance of "unspecific characteristics".

The experiments of the Montagnier team (Laurent-Crawford 1991) show that the four "HIV characteristics" cited by Montagnier as evidence supply the counter-evidence; that we are dealing with cell products under prooxidative and nitrosative stress (see illustration: Experimental findings of the Montagnier team as counter-evidence against the disease theory "HIV is the cause of AID and AIDS" (see table X)).

The results demonstrate the following findings:

1. In T-helper immune cells and other human blood cells that have been acutely stimulated in cell cultures with highly oxidized mitogens (PHA) and the Type1 cytokine interleukin-2 (IL-2) cell disintegration always occurs (cell culture A, C, D).
2. Those cells in the cell culture showing no signs of cell disintegration are those that have reacted with counterregulations to stress provocation by PHA and IL-2 or those that were already counterregulated (cell cultures A, B, C). The counterregulations lead to a shutting down of cytotoxic NO production and to the choking of ROS production in the OXPHOS system of the mitochondria (cell dyssymbiosis).
3. The maximum cell disintegration appears a few days before the maximum appearance of "HIV characteristics" (cell culture A).
4. Cell disintegration occurs after stimulation by PHA and IL-2 even when there are no cells in the cell culture that had previously been provoked for the formation of "HIV characteristics" by stimulation with PHA and IL-2 (cell culture D).

Explanation: In case A the cell culture was stimulated with mitogens and IL-2 for as long as it took some cells from the cell culture to show signs of “HIV characteristics” (in a cell culture it is always only individual cells that are “HIV positive”). This cell culture is then declared as “HIV infected”. After further stimulation the cells that are not counterregulated (or have not been counterregulated through the additional stimulation) die (apoptosis/necrosis). These are mainly the Th1 cells. If these Th1 cells are thiol-depleted, as has been proven in numerous studies on AID and AIDS patients (Herzenberg 1997 et al.), they die even more quickly as the provoked NO gas and the ROS in the Th1 cells can no longer be neutralized quickly enough. Thiol-depleted Th2 cells have a better chance of survival as with a type-2 cytokine profile they can shut down the enzyme for cytotoxic NO synthesis and slow down calcium-dependent NO synthesis in the cytoplasm. In doing this the level of the powering gases for the mitochondrial chanels in the mitochondrial membrane sinks. The import and export facilities of the mitochondria are reduced, the mitochondrial membrane relatively or totally closed, the Ca^{2+} level in the cytoplasm is lowered, apoptosis or necrosis (depending on the diminished ATP level) is prevented. In the cell culture there remain, in essence, the cells that already before stimulation (“HIV characteristics”) or during stimulation (“HIV characteristics”) were no longer capable of apoptosis or necrosis (cases A and B). The cell culture is “chronically HIV infected” and shows no apoptosis /necrosis. Logically the maximum apoptosis/necrosis has to precede the maximum “HIV production” as is the case in cell culture A. The length of time for maximum apoptosis/necrosis after prooxidative/nitrosative stimulation (case A) is well known: an attack of flu in immune healthy people lasts a week, with or without treatment. The Th1 cells react to the intracellular agents. For people with a Th2 dominance, for instance the elderly, a flu infection can be lethal because hitherto compensated cell dyssymbiosis can decompensate which could lead to organ failures ending in death. The same critical period of time was also apparent in the surgical sepsis patients. If the anergic DTH skin reaction as expression of Th2 dominance failed to improve after seven days a significant sepsis developed (Christou 1986). In cases C and D apoptosis/necrosis occurred in Montagnier’s experiment, independent of whether the T-helper lymph cells for the provocation of “HIV characteristics” had been placed under immune stress. In case D without previous “HIV stimulation” apoptosis/necrosis occurred somewhat later than in case C. That is easily explained by the thiol usage of the preceding prooxidative/nitrosative stimulated cells of cell culture C some of which, although not the majority, had developed “HIV characteristics”. Subsequently through further stimulation with PHA/IL-2 even more cells died even more quickly. As can be seen in all four cases it is the stimulation by PHA/IL-2 that is decisive in cell disintegration and not the formation of “HIV characteristics”. These are in fact the results of counterregulation and not the cause of cell disintegration (see especially cases A and B).

As the “HIV researchers” measured the cell effects after stress stimulation only as a net effect of the total amount of cells in the cell culture, the differentiated reaction and regulation (or as the case may be the counterregulation of individual cells) remained concealed. Up until now there have been no publications from the “HIV researchers” about NO-induced effects on an individual cell level. Mitochondria researchers, however, have carried out such studies with the fluorescence-activate cell sorter. In human myelomonocytes from bone marrow after a direct dose of a NO-donating substance at 2mM some 30% of the cells showed necrosis and 30% apoptosis depending on time and dose. The loss of the electric membrane potential of the cell symbionts (mitochondria) preceeded in each case cell disintegration (Richter 1996).

The experimental findings of the Gallo team as counter-evidence against the disease theory “HIV is the cause of AID and AIDS”

The findings of the experimental studies of Gallo and his assistants confirmed, in principle, the results of the Montagnier team (see illustration: Expermental findings of the Gallo team as counterevidence of the disease theory “HIV causes AID and AIDS”, table IX).

The Gallo team stimulated three T-lymph cell cultures with oxidizing mitogens (PHA) and type-1 cytokine IL-2. Previously cell culture A showed signs of “HIV characteristics” in some cells after stimulation with PHA and IL-2 but the majority of the cells in cell culture A showed no “HIV characteristics”. Cell cultures B and C were not previously stimulated with PHA and IL-2 and had no signs of “HIV characteristics. The Gallo team then exposed the two cell cultures, the “HIV infected” cell culture and one of the two “HIV negative” cell cultures, to the same amounts of immune-stress-triggering PHA and IL-2. The third “HIV negative” lymph cell culture was not stimulated as a control. All three T-lymph cell cultures had a 34% proportion of T-helper cells (Zagury 1986). The AEDS concept (acquired energetic cell dyssymbiosis syndrome = acquired cell dyssymbiosis) now predicts that:

1. Cell cultures A and B will feature a cell loss of T-helper cells after stimulation with PHA and I-2,
2. Cell culture C will show no loss of T-helper cells,
3. Cell culture A will show a greater loss of T-helper cells than cell culture B as the cells from cell culture A were previously treated with oxidizing substances (PHA, IL-2) and some cells had shown signs of “HIV characteristics”.

The latter is a sign of a relative thiol deficiency. These cells will respond to stress stimulation with a stronger apoptosis/necrosis as the previously damaged cell symbioses will react more sensitively to the induction of cytotoxic NO gas and oxygen radicals with cell disintegration (as they had not previously responded with counterregulation). Their cell receptors alter in such a way that they respond to PHA and IL-2 in a reduced way. This would mean that a proportion of the Th1 cells do not die but can no longer be measured as Th1 cells due to monoclonal antibodies. The results must have been as follows:

- T-helper cell loss in cell culture A > cell culture B > cell culture C
- T-helper cell loss in cell culture C = 0

The data of the Gallo team were unequivocal (Zagury 1986). The findings confirmed the later results of the Montagnier team that apoptosis/necrosis in T-helper immune cells is dependent on stress stimulation. The strength and duration of the reaction of the T-helper immune cells, either as a type-1 reaction (apoptosis, necrosis) or a type-2 reaction (counterregulation with loss of Th1 characteristics) is dependent, amongst other things, on the thiol pool. In the Gallo team’s experiment there were probably amongst the 3% of T-helper cells, those that could be traced a few days after cultivation through stress stimulation, cells that featured signs of “HIV characteristics” (the publications, however, do not mention this). These cells on further cultivation will “continuously produce HIV” (Laurent-Crawford 1991) as in case B of the Montagnier team’s T-lymph cell cultures, and show no signs of apoptosis or necrosis. This process of counterregulation has been demonstrated by NO researchers. When liver cells were pretreated with low, tolerable amounts of NO gas (NO-donating substances) then a resistance to apoptosis/necrosis was subsequently induced by the addition of higher amounts of NO gas. This resistance was linked to an increased counterregulation through the synthesis of heat shock proteins, the enzyme haemoxygenase and ferritin proteins that can store iron in a non-redox active form (Kim 1995).

The system of genetic and supragenetic counterregulation against nitrosative oxidative stress is proven in all animals

As already documented the system of genetic and supragenetic counterregulations is much more comprehensive and complex. A crucial point is that a part of the counterregulation for the stabilization of the redox milieu is the export of highly oxidized proteins by “retrovirus-like” particles and an increased synthesis of the repair enzyme RT. Montagnier identified both of these factors as being “unspecific HIV characteristics” (Tahi 1997). They actually occur in AID and AIDS patients not as causes, but on the contrary as consequences of a severe or long-term immune stress status and have nothing to do with an “HIV infection”. This evolutionary-biological switching from a Type I reaction to a Type II reaction in states of highly acute or long-term immune stress has been demonstrated in all animals from microbes to human beings. As the cell symbionts in human cells were former bacteria they are also confronted, under severe and/or long-term stress, with the strategic dichotomy, literally, of “gas pedal” or “brakes”. The net result of the counterregulation of the gas-driven micro-Gaia milieu are subject to intricately networked, non-linear adherence to the rules that have only recently, at least in principal, been recognized. The knowledge that all plant cells also react to stress stimuli by producing NO as a protective gas is helpful here. What is remarkable is the fact that plant cells using the unique messenger substance NO and its capacity to diffuse, can relay alarm signals to distant plant cells within its cell system. These primarily unaffected plant cells become subsequently and in increasing numbers resistant to the same initial stress stimulus, as the warning signal triggered an adequate counterregulation in good time. Plants have also developed a dual strategy although they do not have a specific immune cell network. They react by closing down NO production and the formation of ROS (superoxide anion, hydrogen peroxide) when confronted with microbial toxins that trigger a physical/chemical reaction, with a too strong UV irradiation or with heat stress. The affected plant cells respond to acute stress attacks with a hypersensitive Type I reaction and programmed cell death or necrosis. Simultaneously a series of transcription factors are stimulated via a highly networked alternating cooperation between NO and ROS that activate, on a genetic level, a multitude of expressions for the biosynthesis of enzymes. These effect a Type II protective reaction that inhibits NO/ROS/ONOO production and lowers the increased calcium level (Delledonne 1998, Dangl 1998, Hachtel 1998). The ratio of cyclic guanosine monophosphate (cGMP) to cyclic adenosine monophosphate (cAMP) plays an important role in this process. The cGMP, activated by NO, participates in many productive metabolic processes, while the cAMP, as opponent, brakes overreactions. The ratio of cGMP to cAMP is regulated in human cells by corticosteroid hormones (glucocortico steroids) from adrenal glands in favor of cAMP. Practically all stress influences provoke in T-lymph cells as net effect a strong increase of cAMP. The result is a loss of function and the dwindling of T-helper lymph cells (AID) (Fauci 1974, 1975, Hadden 1977, Haynes 1978, Cupps 1982, Coffey 1985, Calvano 1986). Glucocorticoids infiltrate the T-helper lymph cells, bond with transcription factors and inhibit via a chain reaction the synthesis of all cytokines, type-1 cytokines and type-2 cytokines. The only exception is the synthesis of certain repair phase cytokines, like transforming growth factor (TGF) and platelet derived growth factor (PDGF) (Brattsand 1996). Every doctor knows this fact, including the laboratory specialists Gallo and Montagnier.

Polyamines are synthesized by TGF and prostaglandin PGE2 from arginine in competition with the NO synthesis via the enzyme arginase. These inhibit the production of cytotoxic NO and activate repair mechanisms. Included in this process is an increased production of the repair enzyme reverse transcriptase (RT) (overview with Lincoln 1997).

Gallo left a lead as evidence for his ploy. In 1984 the Gallo team had used T-helper lymph cells from homosexual AID and AIDS patients “as evidence of the isolation and continuous

production of cytopathic retroviruses" as well as for the production of the "anti-HIV antibody test" (Popovic 1984, Gallo 1984, Schüpbach 1984, Sarngadharan 1984). External colleagues participated in the laboratory work. Two of these assistants worked for Litton Bionetics, Kensington, MD, USA. In 1987 they reported on the way the Gallo team had treated the T-helper lymph cells of the AID and AIDS patients. Amongst other things they disclosed: "Stimulation in vitro [in cell cultures] could be provided by mitogen or added cells (allogenic antigens). … Certain manipulations of culture conditions were found to improve the outcome, e.g., cocultivation of patient cells with mitogen stimulated peripheral blood leukocytes from uninfected donors. Isolation of virus from cultured cells also was substantially facilitated by inclusion of hydrocortisone in the culture media" (Sarngadharan 1987). The statement by scientists participating in the "HIV isolation" in Gallo's laboratory confirmed the simulated "HIV isolation" and the use of released proteins from human cells from the cell culture as protein antigens for the "anti-HIV antibody test" (Kremer 1998 a, 1998 c):

1. Hydrocortisone is a glucocorticoid.
2. Glucocorticoids inhibit the increase and proliferation of human T-helper cells. They effect in all physiological, psychological and pathophysiological states of stress an effective immune suppression (Gabrielsen 1967, Machinodan 1970).
3. Real existing retroviruses in human T-helper lymph cells can only proliferate when the enzymes for the duplication and division of the DNA strand of the T-helper cells – the DNA polymerases – are present and active (Levine 1991).
4. Glucocorticoids inhibit the synthesis and activities of the DNA polymerases of the T-helper lymph cells (Gillis 1979 a, 1979 b).
5. Glucocorticoids inhibit the genetic expression of the enzyme NO synthase for the production of cytotoxic NO on the genetic transcription level and at the translation stage of RNA transcripts in the biosynthesis of proteins (Kunz 1996).
6. Glucocorticoids promote the synthesis of repair enzymes and the repair processes in T-helper lymph cells (Brattsand 1996, Lincoln 1997).
7. The indispensable prerequisites for the production of "HIV" in T-helper lymph cells are stimulation by the type-1 cytokine IL-2 (Gallo 1984, Montagnier 1985) and mitogens. Glucocorticoids block the effects of IL-2 and mitogens (Gillis 1979 a, 1979 b).
8. The production of type-1 cytokines in the human organism is subject to a day/night rhythm. When the glucocorticoid (cortisol) level in the blood serum is at its lowest during the night and in the early morning, the production of inflammatory type-1 cytokines is at its peak (Petrovsky 1998).
9. Glucocorticoids are employed clinically for the treatment of type-1 cytokine overreactions in countless inflammatory and autoimmune diseases, leukaemia, and tumors as well as for organ transplant patients to prevent rejection of the transplant (Cupps 1982).

The statement: "Isolation of virus from cultured cells also was substantially facilitated by inclusion of hydrocortisone in the culture media" (Sarngadharan 1987) is objectively misleading. All specialists are agreed that the indispensable prerequisite for the cultivation of retroviruses from human T-helper lymph cells is blocked by the glucocortico steroid hydrocortisone. In order to be scientifically correct the quoted passage should read: " The production of the repair enzyme reverse transcriptase (RT) in human cells, which were cultivated by stimulation with mitogens and type-1 cytokine IL-2, was substantially facilitated by the inclusion of hydrocortisone in the culture media".

In his original publication in 1984 Gallo kept quiet about the manipulation of the cell culture of T-helper lymph cells of AID and AIDS patients with hydrocortisone for “isolation of HIV” (Gallo 1984). After publication of these facts (Kremer 1998 a, 1998 c) Gallo was asked at an international press conference at the World AIDS Congress in Geneva in 1998 whether it was the case that: 1. He and his assistants had added hydrocortisone to the culture media “for proof of isolation and the continuous production of HIV” in T-helper lymph cell cultures of homosexual AID and AIDS patients. 2. Whether the “HIV isolation” from cells that had been previously activated by stress stimulation with mitogens and interleukin-2 had been substantially facilitated by the addition of hydrocortisone.

Gallo countered with the question: “What are you implying?” After persistent questioning by journalists Gallo admitted that it was correct and that during the first experiments for “isolation of HIV”, hydrocortisone was added to the “HIV infected” culture media. All other comments as to how the “retrovirus isolation” had been substantially facilitated when he and is team had blocked the proliferation of “retroviruses” with hydrocortisone, were declined by Gallo. The questions to Gallo, Montagnier and their colleagues, posed by representatives of the German Research Group for Investigative Medicine and Journalism (regimed) have remained unanswered to this day. A few weeks after the World AIDS Congress it was prematurely announced in the press that Gallo was to be awarded the most prestigious medical research prize in Germany. The HIV/AIDS establihment was alarmed, the award ceremony was planned for a number of months later in 1999. A statement from the president of the Paul-Ehrlich-Stiftung, which awards this research prize, declared: “This prize is not being awarded for the discovery of HIV” (Paul-Ehrlich-Stiftung 1998). An absurd statement as bar the “isolation of HTLV-I, HTLV-II and HTLV-III (HIV)” by Gallo there is no “discovery of human exogenic retroviruses” (the reason for the prize). HTLV-I and HTLV-II were produced by Gallo with the same laboratory techniques as the “retrovirus characteristics” in leukaemia cell cultures and in contrast to “HIV” play no role clinically. “HIV”, however, was supposed to be threatening all of mankind, why then should the most prestigious German research prize be awarded to Gallo “for not discovering HIV”? In his acceptance speech for this special honour Gallo does not mention a single word explaining whether “HIV isolation” had been substantially facilitated by hydrocortisone. He also did not waste a single word about concrete isolation techniques for “retroviruses”, the “discovery” of which, under his auspices, he described in detail. In the listing of “retroviruses” that he had discovered, Gallo remained silent about the “very first human retrovirus”, HL23V, propagated by Gallo in 1976. This oversight is interesting because for the “isolation “ of that particular “human retrovirus” Gallo was still using the standard rules that he ignored for the “isolation of HIV”.

The absurdity of this “first human retrovirus” is, however, that this particle of “HL23V” did not constitute a “retrovirus” and also that Gallo no longer tried to maintain this assertion (Papadopulos-Eleopulos 1996, 1998 a).

Also, it has also to be said that it is an irresolvable logical contradiction to claim that retroviruses find it considerably easier to propagate in human T-helper lymph cells with the addition of hydrocortisone.

The assumption that evidence of the repair enzyme RT under the influence of hydrocortisone, is the result of the activation of TGF and prostaglandins as a Type II counterregulation of cell dyssymbiosis against oxidative and nitrosative stress stimulation, has been supported by an overwhelming amount of clinical and experimental research data. The fact that Gallo remained quiet about the inclusion of hydrocortisone and to this day is not prepared to give an

explanation proves that he has systematically suppressed all evidence that contradicts this claim of “HIV isolation” and all evidence that the protein antigens used for the “HIV test” could be exposed as human cell proteins. The only possible explanation is that Gallo had sold the products of evolutionary-biological counterregulation of human T-helper lymph cells under oxidative and nitrosative stimulation stress as “retrovirus HIV” and as “anti-HIV antibody test” to the scientific community and the population of the world as a whole.

But how could Gallo “cultivate the virus en masse” (Montagnier in an interview 1997, Tahi 1997) in order to gain enough “HIV proteins” as protein antigens for the “HIV test”?

The laboratory trickery behind the mass production of “HIV Proteins” needed for the assembly of the “HIV test”

T-helper lymph cells only have a limited lifespan. As Gallo had made his “retrovirus HTLV-I and HTLV-II” appear in leukaemic lymph cell lines, it seemed appropriate to establish the mass production of “HIV proteins” by having the “HIV particles” mature out of unlimitedly cultivatable, transformed leukaemia cancer cells via prooxidative and nitrosative stress stimulation. For this purpose Gallo chose cell line H9 (HUT78) that came from patients with T-helper cell leukaemia. Gallo cultivated this H9 cell line together with T-helper lymph cells from AID and AIDS patients and claimed that the observed “unspecific characteristics” were proof that “HIV” had relocated from the T-helper lymph cells to the T-helper lymph cells leukaemia cells (Gallo 1984). This process is called co-cultivation. As the cancer cells are highly counterregulated cells in a state of decompensated cell dyssymbiosis we can say according to the AEDS concept that these leukaemia cells in a co-cultivated cell culture will respond to the NO gas production in the T-helper immune cells by increasing counterregulation. The NO gas produced after oxidative and nitrosative stress stimulation also diffuses between the two cell types. The “immortal” T-leukaemia cells that as a consequence of the decompensated cell dyssymbiosis (heavily reduced vitality and number of mitochondria) no longer show signs of programmed cell death, behave in the mixed stimulated cell culture similarly, but even more distinctly, to cell culture B in the Montagnier team’s experiment in 1991. The leukaemia cells increase the already running counterregulations and display chronic “HIV characteristics” (increased RT production, increased budding of export particles for oxidized cell proteins). And exactly that was the case. Cancer cells respond to NO exposure depending on the dosis with cell inhibition (cytostasis), subsequently with a heightened counterregulation (Brüne 1996, Lincoln 1997). Up until now nobody has been able to demonstrate actual “HIV production” in the H9 leukaemia cells, as was also the case of the T-helper lymph cells of AIDS patients. Also the H9 cells were by no means destroyed by “HIV” as they should have been according to the “HIV/AIDS theory”. Again in these limitlessly cultivatable leukaemia cells unspecific “HIV characteristics” remained, nobody has demonstrated the actual presence of “HIV retroviruses” in these cells. Though in this way any old cell debris from the cell fluid from this “immortal” cell culture can be centrifuged out and some of these scrap proteins can be used as antigens to assemble the “HIV test” (Sarngadharan 1984). In “HIV tests” these proteins from the special refuse of co-cultivation (foreign proteins from human cells) could react with increased amounts of antibodies in the blood serum of probands. An absurd vicious circle with tragic consequences.

The “invisible hand of the market”

Unspecific pseudo evidence was published for the “isolation of HIV” and the “existence of HIV” - unspecific “HIV characteristics” and unspecific “molecular markers” – but never

challenged in scientific discourse. The scientific contemporary witness, Professor De Harven, after decades of laboratory experience and as an internationally renowned expert in the field of detection and isolation of retroviruses categorically stated:
"When around 1980, R. Gallo and his followers attempted to demonstrate that certain retroviruses can be suspected of representing; human pathogens, to the best of my bibliographical recollection, electron microscopy was never used to demonstrate directly viremia in the studied patients. Why? Most probably, EM results were negative and swiftly ignored! But over-enthusiastic retrovirologists continued to rely on the identification of "viral markers", attempting to salvage their hypothesis. When retrovirus particles are legion, the study of molecular markers can be useful, and provide an approach to quantification probably better than direct particle counting under the EM (which I always found very difficult). But when, using EM, retrovirus particles are absent relying exclusively on 'markers' is a methodological nonsense. 'Markers' of what? … In conclusion, and after extensive reviewing of the current AIDS research literature, the following statement appears inescapable: neither electron microscopy nor molecular markers have so far permitted a scientifically sound demonstration of retrovirus isolation directly from AIDS patients" (De Harven 1998 c).

The "invisible hand of the market" that had produced the conditions for heavy and long-term immune stress (AID) (nitrite gases, the raw materials of recreational drugs, pharmaceutical substances for antibiotic, chemotherapeutic and analgesic abuse, highly contaminated coagulating protein preparations and blood products etc. etc., conditions for poverty), then arranged the patented marketing of "unspecific HIV characteristics" – the subsequent symptoms not the causes of acquired immune stress syndrome: "anti-HIV antibody tests", "anti-retroviral" substances for "anti-HIV prevention" and "HIV synthesis inhibition".

The agencies of the "invisible hand of the market" (Centers for Disease Control, CDC, National institutes for Health, NIH, Food and Drug Administration, FDA) had already issued warnings about the "human immunodeficiency virus" (CDC and NIH 1982, CDC, FDA, and NIH 1983) before the desired product design was developed and marketable (Popovic 1984, Gallo 1984, Sarngadharan 1984, Schüpbach 1984).

"Unfortunately, the emergence of acquired immunodeficiency syndromes (AIDS) in 1981 gave the retrovirus establishment an opportunity to transform what could have been only an academic flop into a public health tragedy … Soon after the first cases of "Gay related immune deficiency" were described by Gottlieb it was obvious for all observers that Gallo and his associates were going to jump on the new syndrome as a Godsent opportunity to attempt to justify the lavish federal budgets they had consumed on retroviruses over the past 10 years. In 1980, the scientific community was getting more and more concerned about the absence of results in "The War against Cancer" based on retrovirus hunting. The minor episode of HTLV 1 was not enough, by far, to calm the fears of grossly misdirected federal research funds. The fact that the syndrome, soon tactically renamed "AIDS", had nothing to do with cancer was apparently of little embarrassment for Gallo. Frequent association with Kaposi sarcoma helped to blur the difference in the eyes of the public. Dominated by the media, by special pressure groups and by the interests of several pharmaceutical companies, the AIDS establishment efforts to control the disease lost contact with open-minded, peer-reviewed medical science since the unproven HIV/AIDS hypothesis received 100% of the research funds while all other hypotheses were ignored. The general public and the medical community were made to believe that the presence of circulating antibodies is diagnostic of this disease … And to ensure that the AIDS establishment could profitably continue to flourish, research on any dissenting (i.e. non-HIV) hypothesis was carefully prevented by tight control of research funding and by the extreme difficulty of publishing anywhere any

dissenting views... In the late 1980s, I was considering adding to my research program in Toronto more EM observations on samples from AIDS patients. Unfortunately, by that time the media and the CDC had so perfectly orchestrated the panic of a plague-like epidemic that I was quickly made to understand that my assistants would all transfer out of the lab if I had insisted to activate such a program... The HIV seropositivity test was still at that time regarded as providing reliable diagnostic data. Since then, Papadopulos and the Australian team have demonstrated that this is very far from the truth" (De Harven 1998 b, Papadopulos-Eleopulos 1993 a).

In comprehensive scientific analyses the internationally renowned research group of Eleni Papadopulos-Eleopulos and her colleagues at the Royal Perth Hospital and the University of Western Australia have published numerous critical publications about the "isolation of HIV", the construction of the "anti-HIV antibody test", AIDS indicator diseases, their causes and proliferation as well as about HIV/AIDS therapy (Papadopulos-Eleopulos 1988, 1992 a, 1992 b, 1993 a, 1993b, 1995 a, 1995 b, 1995 c, 1996, 1997 a, 1997 b, 1997 c, 1998 a, 1998 b, 1999, 2000 a, 2000 b, Turner 1998).

In German-speaking countries the most important critical studies on the same questions about "HIV isolation", "HIV test", AIDS diseases, AIDS therapy and AIDS politics have been published by the study group Ernährung und Immunität (Nutrition and Immunity) in Bern and the research group regimed (Research Group for Investigative Medicine and Journalism) in Stuttgart as well as the Zentrum zur Dokumentation für Naturheilverfahren (ZDN) (Center for the Documentation of Natural Healing Processes) in Essen (Hässig 1993,1994 a, 1994 b, 1996 a, 1996 b, 1997 a, 1997 b, 1998 a 1998 b, Kremer 1990, 1994, 1996 a, 1996 b, 1998 a, 1998 b, 1998 c, 2000 b, 2000 c, Lanka 1994, 1995, 1997, ZDN 1995, 1998).