VIRUSMYTH HOMEPAGE

15 YEARS OF AIDS

The continuous failure in the prevention and treatment of AIDS is rooted in the misinterpretation of an inflammatory auto immune process as a lethal, viral venereal disease

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May 1998

Summary - The question of the specificity of the anti-HIV antibody test has to be re-evaluated as it was shown that the viral enrichment obtained from co-cultivations of patients lymphocytes with fetal cord blood by BARRÉ-SINOUSSI et al. and leukaemia cells by GALLO et al., exclusively consisted of proteins of the cell types used in the cell culture. This precludes a clear separation of presumed retroviral and cellular proteins or extracellular matrix proteins. In this context it was shown that the anti-HIV antibody test detects autoimmune antibodies directed against cyto-skeletal proteins e.g. the liver cells. Strongly augmented anti-actin autoantibodies is considered close to pathognomonic for chronically active hepatitis. The original assumption that "reverse transcription" from RNA to DNA is evidence for the existence of retroviruses, was wrong. In fact, "reverse transcription" is a vital mechanism for the maintenance the genome. The decrease in numbers of circulating CD4 lymphocytes can be explained by a stress-induced hyper-cortisolism. Up to date, direct HIV-mediated destruction of CD4 lymphocytes could not be proved. The same is true for measuring of the "viral load". Shortcomings of the applied method to quantify the "viral load" do not permit definitive conclusions. Possibly, it may be taken as an expression of a stress-induced weakening of the cellular immune reactions, in the course of which the nucleoside fragments resulting from the current cell turnover are inadequately eliminated. Furthermore, the treatment of patients with nucleoside analogues has a toxic effect on both the genome of the cell-nucleus and the mitochondria. The latter, therefore, may produce insufficient amounts of ATP, causing organ failure and, eventually, death. The synthetic protease inhibitors used these days are associated with serious side-effects. Therefore, it seems worthwhile, in these patients, to bring back the catabolic situation due to whole body inflammation to homeostasis by administering anabolic phyto-polyphenolic compounds.

AIDS is the abbrevation for acquired immunodeficiency syndrome. AIDS, as a term for an illness, originated in the search by the American Centers of Disease Control for sick homosexual men, also suffering from Kaposi's Sarcoma (KS) and/or Pneumocystis carinii pneumonia (PCP). In 1983 BARRÉ-SINOUSSI et al. reported on a T-lymphotropic retrovirus which they allegedly isolated from an enlarged lymphnode of a homosexual patient (1). In 1984 GALLO et al. reported the alleged isolation of an identical retrovirus from CD4-lymph cells from homosexual patients, clinically diagnosed as suffering from AIDS (2). BARRÉ-SINOUSSI et al. co-cultivated these patients lymph cells in question with fetal cord blood, and GALLO et al. co-cultivated theirs with leukaemia cells. Initially, these laboratory methods must raise doubts as to whether the isolation of a new human retrovirus is evident just by these data. GALLO et al. stated that their allegedly isolated retrovirus caused the destruction of CD4 lymphocytes in those patients, whose heterogenic illness was taken as an after-effect of the CD4-cell destruction and so subsumed as AIDS. Besides, GALLO et al. announced that in due time a vaccine would be available for the formation of antibodies against the discovered virus (2). Today, fifteen years later, the question still remains open, whether HI retroviruses actually do exist or whether the postulated retroviral HIV-antigens as

well as the postulated HIV reverse transcription are a matter of human protein molecules derived from cells in co-cultured cell cultures used by both BARRÉ-SINOUSSI et al. and GALLO et al.. The most extensive investigations in this regard are owed to ELENI PAPADOPULOS-ELEOPULOS and her group in Perth, Australia. In 1993 they published a review concluding that there is no evidence for the existence of HI viruses (3). In 1994 LANKA demonstrated that all retroviruses, including HIV, are biologically inexistent and their phenomenology is based on laboratory artefacts (4-6). ELENI PAPADOPULOS confirmed LANKA's interpretation in a recent comprehensive review (7).

These fundamental counter-statements to the current HIV-AIDS-theory have been strongly supported in the last few years. Upon investigations in order to develop a vaccine against HIV it became apparent, that the enrichment of presumed HIV-1 preparations, considered as pure, consist of proteins of the cell types used in the cell cultures, which resist a clean separation into presumed retroviral and cellular proteins, i.e. extracellular matrix proteins. Above all, these cell proteins, also occur in the inside of extracellular particles, which have been misread as it seems as so-called HI virions by the retrovirologists (8-10). These findings were to be expected as GALLO et al., when developing the AIDS test, did not investigate the presence of cells own proteins in the protein mixture, released during the co-cultivation of patients lymphocytes and leukaemia cells. Upon developing the ELISA- and Western Blot tests it should have been imperative to consider proteins released from stimulated leukaemia cells, not being mixed with patients lymphocytes, and to differentiate these from the ones, released only after addition of patients lymphocytes.

In view of this it seems to be mandatory to re-evaluate the question of the specificity of the anti-HIV-antibody test.

What is the laboratory finding "anti-HIV-positive" based on?

In a series of preceding reports we have discussed this question in detail (11-14). We came to the conclusion: the laboratory finding "anti-HIV-positive" is primarily the expression of an autoimmune activation of the immune system linked to a persistant catabolic state of metabolism. In view of the fact that the diseases grouped under the term AIDS are limited to risk groups such as homosexuals, drug addicts and recipients of blood products contaminated with parenterally transmitted hepatitis inductors, the question is raised, whether the anti-HIV test determines autoantibodies directed against cell envelope structures with a specificity to the body's own proteins of the host cells. It has been known for over twenty years that chronically active hepatitis (currently hepatitis B, hepatitis C and autoimmune hepatitis without evident antiviral antibodies) react by the formation of autoimmune antibodies directed against cyto-skeletal proteins of the liver cells. Thus, the raised anti-actin-autoantibodies are pathognomonic for chronically active hepatitides (15). JOHNSON et al., in 1965, were the first to report on anti-actin-autoantibodies (16). They described autoantibodies directed against smooth muscle cells and showed that this had to be considered as characteristic indication of "lupoid hepatitis". In 1973 GABBIANI et al. demonstrated that autoantibodies directed against smooth muscle cells react with actin-containing microfilaments (17). Further investigations indicate that autoantibodies with anti-actin specificity are to be classified within the big group of autoantibodies against filamentous proteins of smooth muscle fibres. 3 - 18% of healthy individuals present low titer autoantibodies against cyto-skeletal proteins (18). High titer anti-actin autoantibodies, on the other hand, are only found in patients suffering from chronically active hepatitis and/or biliary cirrhosis (19). In 1994 BERMAS et al. showed that both sera from patients with lupus erythematosus and from mice suffering from the same illness react with glycoprotein 120 and peptides of the postulated HIV-1 envelope (20). They further proved that control sera of healthy individuals and patients with other autoimmune diseases contain small amounts of the same autoantibodies. Last but not least, they showed that autoantibodies reacting with glycoprotein 120 do not possess antinuclear specificity. They refrained from investigating a specificity against cytoskeletal proteins of these autoantibodies.

Evidence is given that the anti HIV test does not indicate antibody formation against the postulated retroviruses as, during the last decade in Germany, not a single seroconversion has been observed in inprisoned drug addicts. All sero-positive drug addicts acquired their anti HIV "positivity" before their imprisonement. In opposition to this, seroconversion by hepatitis B inductors was recorded in intravenous drug addicts (21-23). The same was also observed in haemophiliacs, i.e. despite continuous substitution with hepatitis-contaminated blood products approx. a third of these individuals never become anti-HIV positive. This is characteristic for the individual response to autoimmune reactions against cyto-skeletal proteins in the host cells, in which GIRARD and SEN? CAL observed a polyreactivity (24). The individual autoimmune reactivity either appears at first contact or fails to appear even at multiple contacts.

We conclude that a positive anti-HIV test does not indicate an antibody formation against "retroviral HIV antigens". Low titer "anti HIV" antibodies are common even in healthy individuals. High titer "anti HIV" antibodies are pathognomonic in chronically active hepatitis. The anti HIV test does not answer the question whether anti HIV antibodies occur or not; the test differentiates between "plenty = positive" and "few = negative".

Rethinking as to "Reverse transcription"

The error, taking proteins resulting from "HIV" isolation for retroviral proteins, dates back to 1970. The paradigm, that DNA codes information and programs relating to all physiological and phenomenological aspects of all organisms, resulted in the postulation of the irreversibility of the genetic flow of information for the synthesis of proteins - from DNA via messenger substance (RNA) to proteins. This was the crucial genetic dogma (25). Despite the proven reversibility in 1970, that from RNA DNA can remerge, this fact was postulated as an exception that proves the rule by stating the existence of retroviruses, qualified for this reversibility, which, at this time, were only considered as tumour viruses (26, 27).

With the discovery of this enzymatic activity in all living cells it soon became clear that the evidence of the function of "reverse transcription" from DNA into RNA was not a proof for the existence of retroviruses, because the genome of all eukaryotic cells is clearly marked by this activity (28, 29). Retrospectively, it seems rather astonishing that in 1983 MONTAGNIER and in 1984 GALLO still postulated a new retrovirus despite the fact that a new viral entity had never been isolated or described, according to the standard regulations in virology. As a matter of fact, the enzyme Reverse Transcriptase from ÒHIVÒ has never been isolated or described, but only inferred from functions to its existence, when new formation from DNA into RNA was proven by laboratory techniques.

Since 1985 it has been known that the "Reverse Transcription" plays a decisive role in the maintenance of the structure of the genome, by repairing chromosome fractures and, especially, by limiting the loss of chromosomal end components, the telomeres occurring at cell replication (30-33). The respective enzymes for this kind of reverse transcription, the telomerases dispose of a type-specific RNA matrix for the formation of the repeating telomer units. Somatic human cells, that do not belong to the reproductive path, cannot adapt to the shrinking of their telomeres when replicating and so cease the replication at a certain degree of depletion.

Up to date, the influence of nucleoside analgues on the action of telomeres at replication has obviously not been investigated. We could trace only two publications, in 1996, which describe an in vitro investigation of nucleoside analogues inhibiting the telomerase activity. In our view, the knowledge, already gained in 1980, on the vital physiological function of "reverse transcription", should have lead to a rethinking as to the establishing of nucleoside analogues as pharmacological inhibitors of the "Reverse Transcription" of the postulated HI viruses and, according to knowledge of that time on the physiological function of the "Reverse Transcription", it should have been rejected (34,35).

What is the decrease of CD4-lymphocytes in AIDS based on?

The decrease of the circulating CD4-lymphocytes in the blood stream during progressive immune deficiency in AIDS has generally been explained by the progressive destruction caused by HI viruses (36). Four years ago CARBONARI et al. showed in an in vitro investigation that the apoptosis of the lymphocytes in AIDS patients is mainly related to CD8 - T-cells and CD19 B-cells (37). FINKEL et al. then pointed out that apoptosis concerns mainly "bystander" cells and spares supposedly infected cells from so-called HIV- and SIV-lymphnodes (38). These reports remind us of FAUCI's classical publications of the 70's in which he and his working group clearly demonstrated that, in persisting hypercortisolism, an increasing number of CD4-cells leave the blood stream and can thus activate B-cells in the marrow (39-44). The "migrated" CD4-cells return to the blood stream upon dropping to normal values of the cortisol level.

At the beginning of 1995 WEI and HO et al. published a report in which they declared, that the extremely fast multiplication of HI-1 viruses produces a raised turnover of CD4-lymphocytes (45, 46). Towards the end of 1996 WOLTHERS et al. showed that the telomere length of CD4-lymphocytes in anti- HIV positive individuals remains normal, whereas the one of CD8-cells decreases (47).

During the latest international congress of leading HIV scientists the long-term criticism of the HIV/AIDS theory has been confirmed: despite intense and precise investigations there was no proof of a pathophysiological mechanism explaining the different reaction of CD4- and CD8-lymphocytes to the postulated retrovirus HIV (48). It was literally stated: "The riddle of CD4 cell loss remains unresolved." Paul Johnson of the Harvard Medical School in Boston voiced in a disillusioned way the helplessness of the conventional AIDS scientists: "We are still very confused about the mechanism that leads to CD4 depletion; but at least now we are confused at a higher level of understanding." In other words, FAUCI's pioneering work, based in the seventies, on experimental traumatology fell into oblivion. CALVANO clearly documented in a review published in 1986 that the selective depletion of CD4-lymphocytes is induced by neuroendocrine mechanisms in traumatic conditions such as injuries and burns, whereas its proportions depend on the degree of hypercortisolism (49). It remains enigmatic why FAUCI, after having joined the AIDS research, never again mentioned his own reports.

What does the "viral load" measure?

Immediately, after the publication of WEI's and HO's reports in January 1995 (45,46) in which they put forward the hypothesis that HI viruses multiply at raving speed destroying a similar number of CD4 helper cells, quantitative tests based on the genetic multiplication method PCR were introduced and so a large number of HIV in the blood stream was postulated. It has been well known among HIV scientists that the so-called viral load, i.e. the measurement of the "viral load" is no evidence of the entire virus genome or intact viruses (50). The "viral load" measures short components of the messenger substance RNA, attributed to the HI viruses. Because the HI virus genome per se never could be described it is impossible to designate these RNA fragments as viral. Going through the records of the presumed characterisation of HIV it can be inferred that all components - proteins and genetic substance - attributed to “HIV”, are of pure cellular origin (3-7). Therefore, the results of the "viral load" can only have an indirect significance, such as the measurement of an increase or decrease of cellular RNA, as it can be observed as increasing in catabolic conditions of cell disintegration and decreasing in anabolic conditions. However, these results cannot be considered as clinically relevant as, besides the technical inadequacy, control investigations with both non-positive defined healthy and ill individuals have never been published.

The polymerase chain reaction (PCR) is a technique for a manifold multiplication of short DNA fragments, developed by Nobel prize winner for chemistry, Kary Mullis. Upon measuring the "viral load" the RNA fragments in the blood stream first have to be converted into DNA and then

multiplied as such. The single, developing technical steps of this method are prone to failure. The slightest impurity, drugs, such as heparin and other substances interfere with a reproducible functioning of the PCR method, especially with quantification (53). Kary Mullis, the inventor of this method does not miss any occasion to critizise the application of his technology in the context of AIDS (52). Further more, it is concealed that it does not make sense, either practically nor theoretically, to initially multiply manifold fragments of genetic structure and then to postulate their manifold presence. In case they were actually present in the blood samples it would cause no problem to prove their existence by simple, quick and cheap standard methods (51) and, if de facto, viruses did exist in the blood stream, scientists certainly would have been successful in making them visible. Hence, up to date, no scientist claims this achievement, a fact which has been confirmed under coercive evidence by the German Health Ministry in 1996. After the report of a produced "positivity" in the "viral load" during a vaccination test with proteins (54) of a previously "negative" defined test person it is now frankly admitted that repeated wrong-positive results in the viral load are quite a wellknown phenomenon (55).

Impairment of energy formation in mitochondria by nucleoside analogues such as AZT (Azidothymidine, Zidovudine)

AIDS patients quite often demonstrate a weakening of their skeletal muscles. Up to 1990 this was considered a HI-virus-caused impairment of muscles. In 1990 DALAKAS et al. demonstrated that this kind of muscle disease is due to an administration of AZT, weakening the mitochondria within muscle cells. With the excessive release of free radicals the mitochondria are affected in their function of forming ATP as key substance in metabolic energy (56). HAYAKAWA et al., in 1991, demonstrated important changes in the mitochondrial DNA (mtDNA) in the liver of mice after the administration of AZT. The final sentence of this paper reads: "However, for AIDS patients it is urgently necessary to develop a remedy substituting this toxic substance AZT" (57). These results were confirmed by histochemical methods in the same year by CHARIOT and GHERARDI (58).

The toxicity of nucleoside analogues in the treatment of viral diseases was thoroughly dealt with in the following years and, it was proven that the toxic effect causes multiorganic impairments in heart muscle, brain and kidney, as well as in liver and pancreas (59). Further, it was shown that successor drugs of AZT such as ddI and ddC cause the same mitochondrial impairment (60).

Since 1991 it would have been mandatory that not only the pharmaceutical industry but also the registration authorities seriously consider these impairments caused by long-term administration of nucleoside analogues and provide proof of the incoherence of AIDS patients death with this drug treatment; in general this obligation has been avoided and now they face upcoming connected liability questions.

HIV-proteases inhibitors: A new therapeutic principle in the prevention and treatment of AIDS

According to the HIV-model long precursor molecules of proteins along the multiplication process, have to be cut at certain interfaces in order to create functional HIV proteins upon which, ultimately, new HI viruses form. Synthetically produced short protein molecules, reproduced after the interface to be cut from the precursor protein but which cannot be cut should, according to the model again, inhibit the natural activity of the HIV protease and thus prevent the formation of new HI viruses. As a matter of fact the HIV protease has not been isolated, but has been reconstructed by genetic engineering upon which it was observed that this enzyme is very similar to the human digestive enzyme, pepsin of the class of the asparate proteases.

The problem of the model is that the one and the very same HIV protease would have to be cut at completely different interfaces in order to form functional proteins and, ultimately, HIV. Practically, this is not conceivable and has been explained as: "enzymes do not have a high sequence specificity" although it has been postulated that: "a therapeutically applicable inhibitor

has to be specific, and should not inhibit human enzymes of this class of substance." (61) Considering these explanations of the head of the chemical department of the scientific laboratories at BAYER's, it becomes obvious that, theoretically, it is not possible to exactly target the postulated HIV protease. Further, it is impossible not to interfere in the cellular processes of integration and disintegration of a variety of proteins. The inhibition of the active protease in AIDS per se makes sense. However the pharmacological administration of high doses of distinct aromatic substances is a non-physiological measure, connected with serious side-effects which excludes their pharmacological use.

Indeed, up to date, the pharmacological HIV protease inhibitors prove to be connected with side-effects, which demand the absolute necessity of their replacements by phyto-therapeutic mixtures. Apart from side-effects such as kidney stones, damage of the liver, an increase of diabetic impairment of metabolism, CMV retinitis and haemolytic anaemias these protease inhibitors, after a short-term administration, also demonstrate a loss of effect on the inflammatory process which was misinterpreted as a result of an acquired resistance by HIV as well as an incompatibility with many drugs, especially with the ones of the group of Cytochrom-P450-inhibitors and inductors (62).

Nutritional possibilities in the prevention and treatment of AIDS

Looking at the formula of structure of the synthetic protease inhibitors, it becomes obvious that these are artificially produced aromatic compounds. As we have suggested lately, polyphenols as well as tannins and flavonoids are phyto-protective substances against harmful external influences. As aromatic substances, they cannot be synthesized by the animal organism. The nutritional supply of a variety of phyto-polyphenols to the animal organism has the function of operating as a redox buffer and rebalancing oxidative stress conditions with their catabolic alteration of metabolism to the anabolic-catabolic state of equilibrium (63).

Flavonoids and tannins are effective with respect to:

- 1 Inhibition of lipid peroxidation
- 2 Scavenging of oxygen radicals
- 3 Binding and inactivation of pro-oxidative transition metals such as Fe and Cu
- 4 Binding of proteins including attenuation of their enzymatic activity (protease inhibitors)

Upon these reductive activities flavonoids and tannins are oxidized themselves; a well known example is the reduction of vitamin E by vitamin C or coenzyme Q. These mechanisms are at the beginning of a cascade of recycling. This example demonstrates, that the multitude of almost 5000 different flavonoids and tannins is used to overcome the oxidative state of the ex-antioxidative molecules at the end of the cascade of recycling, by transferring it to a variety of native molecules.

The stress-induced state of catabolic metabolism in AIDS is in the center of the pathogenesis. The correction of the connected whole body inflammations, caused by oxygen radicals and protease activation, is a compelling preventive and therapeutic action which urgently demands the use of phyto-therapeutic polyphenol compounds.

Possibilities and limits of treatment of hepatitis in anti-HIV positive individuals

A symptomlessness and the stress-induced activation of liver inflammation in healthy individuals are characteristic of parenterally transmitted inoculation hepatitis (hepatitis B and C). The classic example for this occurrence is posttransfusional hepatitis caused by blood and blood products of clinically healthy blood donors. At the occasion of a study, made in the early fifties at the blood transfusion service of the Swiss Red Cross on recipients of lyophilized pools of mixed plasma of 50 - 70 healthy blood donors, it was observed that this caused serious, sometime even lethal hepatitis in many ill recipients (64-66).

It is emphasized that in a contaminated organism with parenterally transmitted hepatitis inductors (now called hepatitis B and C), the aim of treatment has to be reduced to just reach a normal state of health. The administration of virucide, cytotoxic drugs is not able to eliminate these inductors from the organism. Proceeding from this knowledge, in Poland, for two decades, BRZOSKO et al. have been collecting data with the Tibetan prescription of phyto-therapeutic formula, PADMA 28 (67). They showed that this phenol-rich plant compound is able not only to reduce the serum level of hepatitis B antigens in hepatitis B patients but also to augment the serum level of hepatitis B antibodies. At the same time an amelioration was observed in these patients regarding their clinical condition and the biochemical and histological results from their hepatitis. Based on these pioneer results, today, in patients suffering from chronically active hepatitis, the substitution with phyto-polyphenolic mixtures has priority over other treatments.

How does the nucleoside analogues treatment of AIDS patients influence their course of disease?

After having examined 8 reports on HIV positive long-term non-progressors, who stayed clinically symptomless for over 10 years, we realized, that, without exception, they had not been treated with nucleoside analogues (68-75). We consider this as a confirmation of our above-mentioned caution as to the prophylactic and therapeutic administration of these cell toxics, originally developed for treating cancer, in the autoimmune course of disease in AIDS.

Nutritional supply of polyphenolic mixtures as basic treatment of anti-HIV positive individuals and AIDS-patients

As initially showed, a positive anti-HIV test is an indication of an augmented formation of autoantibodies against cytoskeletal proteins, i.e. actin. This condition is pathognomonic for chronically active hepatitis. AIDS, as serious immuno-deficiency-syndrome is the expression of a persistant hypercatabolic state of metabolism along with a stress-induced whole body inflammation. A successful treatment of such conditions consists of the nutritional supply of a sufficient quantitiy of antioxidative and antiproteolytic phyto-phenolic mixtures, consisting of flavonoids and tannins. As neither the animal nor the human body are able to synthesize aromatic compounds they are fully dependent on a sufficient supply of anabolic effective phyto-polyphenolic mixtures, in order to adjust catabolic states of metabolism. These mixtures are present in drugs made of teas and spices. Padma 28 proved to be the most effective one. Additionally, it is recommended to balance other possible states of deficiency of vital nutritional components such as polyanions and essential fatty acids.

Completing this review we came across the publication by PADIAN et al. which remarkably emphasizes the insignificance of heterosexual intercourse in transmitting "HIV". In this study, extended to 10 years, the authors say: "male-to-female transmission was approximately eight times more efficient, than female-to-male transmission and male-to-female per contact infectivity was estimated to be 0.0009".

Obviously, AIDS is not a viral venereal disease, but an inflammatory autoimmune process (76).

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