HIV & AIDS - Did Dr. Gallo and his Colleagues manipulate the "AIDS-Test" to order?

VIRUSMYTH HOMEPAGE

DID DR. GALLO AND HIS COLLEAGUES MANIPULATE THE "AIDS-TEST" TO ORDER?

"The hunt for the virus" [1] has degenerated into "clean torture with fatal result" [2]

By Heinrich Kremer

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Who, for given reasons given below casts doubt on the theory that "HIV causes AIDS", is often confronted with the question, if it did not, how is it that a patient who has been diagnosed as "HIV positive" by the test sooner or later goes on to develop AIDS? To which the AIDS sceptic usually replies that a "HIV-positive" laboratory result, an arbitrary defined characteristic is part of the clinical diagnosis "AIDS".

This exchange does not advance the argument very much as to whether "AIDS" and "HIV" are scientifically-speaking biological entities and if between them a biological cause-effect relationship is possible. In other words, if either the term "AIDS" or the term "HIV", or neither, represents conceptually independent entities but rather purely semantic constructs, then biologically there can be no cause-and-effect relationship between these two terms, i.e. between the postulated pathogen "HIV" and the supposed definable disease entity "AIDS".

The causative factor, the "retrovirus HTLV-III" (later termed "HIV") was introduced by Robert Gallo in 1984 (then a retrovirologist in the Tumour Biology Laboratory in the National Cancer Institute at Bethesda). On May 4, 1984 together with collaborators from his own laboratory and other research centres and hospitals as well as workers at the pharmaceutical company Litton Bionetics, he published four basic papers in *Science* (3-6). These supposedly described the identification, isolation and continuous production of a newly discovered type of retrovirus (since 1987 called "HIV") as well as the serological analysis of this "HIV" and of tests "capable of detecting antibodies to HIV" in the sera of "patients with AIDS or pre-AIDS". The simultaneous publication of these four papers by Gallo et al was shortly preceded by a patent application for "HIV antibody tests" and by Reagan's US Health Secretary's announcement at a press conference attended by Robert Gallo himself before the world's media that Robert Gallo and his team had "discovered the probable cause of AIDS".

The first *Science* paper of May 4, 1984 begins with the fundamental assumption: "epidemiological data suggest that the acquired immunodeficiency syndrome (AIDS) is caused by an infectious agent that is horizontally transmitted by intimate contact or blood products" (3). The word 'probably' employed by the US minister only a few days before was no longer mentioned by Gallo et al.

The fourth and last Science paper of that date ends with the conclusion: "The data presented here and in the accompanying reports suggest that HTLV-III is the primary cause of AIDS" (6). (HTLV-III = HIV). Gallo et al's conclusion proves that they did not postulate a direct cause-and-effect relationship between "HIV" and "AIDS", declaring "HIV" to be only the *primary* cause of "AIDS": "Although the disease is manifested by opportunistic infections, predominantly Pneumocystis Carinii Pneumonia, and by Kaposi's Sarcoma, the underlying disorder affects the patient's cell-mediated immunity, resulting in absolute lymphopenia and reduced subpopulation of helper T lymphocytes (OKT4+)" (3). Gallo et al by no means, therefore, postulated that "HIV" was the *direct* cause of "AIDS", rather, they only claimed "HIV" is the cause of "AID" (AID = Acquired Immuno Deficiency = reduced sub-population of T-helper lymphocytes). The syndrome "S" ("manifested by opportunistic infections (OI), mainly Pneumocystis Carinii Pneumonia (= PCP), and Kaposi's Sarcoma (= KS)") was presented by Gallo et al like commonplace as the necessary consequence of "AID".

The scheme of Gallo et al is as follows:

1. "HIV" causes "AID" as a consequence of the infection and sooner or later the destruction of T-helper lymphocytes.

2. As a consequence of the decrease of cellular immunity, the control of opportunistic pathogens and cancer cells by T-helper lymphocytes breaks down as a result of which, syndrome "S" develops.

The short version of Gallo et al's plague formula is "HIV = AID = S".

The two part causal chain "HIV causes AIDS" actually turns out to consist of three parts, and Gallo et al's claim that "HTLV-III" (= "HIV") is the primary cause of "AIDS" (6) is a fusion of two hypothetical causal assertions, and a fictitious end-effect assertion. This is because Gallo et al's published data say nothing about whether "AID" really does cause "S"; they can at most suggest a cause-and-effect relationship between "HIV" and "AID". Whether "S" can be the result of "AID" is for several reasons highly doubtful. "S" is somewhat chameleon-like due to numerous re-definitions undergone, so that the existence of "S" as a "separate disease entity" (4), in the sense of a biological disease entity, can no longer be rationally made out. Individual, defined diseases, which initially made up part of the syndrome were years later expressly removed again. In the end a wild collection of 29 old infections and non-infectious diseases has been collected together to constitute the syndrome "S", of which several are part of "S" even if the "HIV" status is negative or indeterminate (7).

The latter means that "AID" cannot be the cause of "S" because "AID" is supposed to be the result of "HIV", in order that Gallo et al's plague formula "HIV = AID = S" as a causal chain is upheld, yet "AID" due to different reasons can exist independently of "HIV". Nothing is given whereby "AID" must be the cause of "S". "AID" and "S" could, instead, have a common cause which need have no causal relationship with a hypothetical "retrovirus HIV".

The pretence of a pseudo-biological cause-and-effect relationship expressed by the plague formula "HIV = AID = S" has made a leading AIDS critic, who has presented the most comprehensive clinical analysis of the AIDS phenomenon, say "AIDS, in short, has become a schizophrenic disease" (8).

How then, can a semantic construct of a collection of mostly contradictory diseases be the result of a supposed biological causal chain, which itself in turn is made up of hypothetical constructs as cause-and-effect factors? Because the premises and conclusions (3,6) which underlie Gallo et al's plague formula can be falsified convincingly.

Gallo et al have claimed that "epidemiological data prove that an infectious agent (3) is the cause of "AID", and "AID" is the cause of "S"." Essentially, Gallo et al arrived at this conclusion from the findings of the CDC that "S" ("OI, mainly PCP,

and KS") is significantly connected with very frequent promiscuity and predominantly receptive anal intercourse in homosexual men in the metropolitan areas in the US (3). However, this conclusion only demonstrates the arbitrary and selective interpretation of the clinical data by the CDC and Gallo et al.

Highly promiscuous and predominantly receptive (unprotected) anal intercourse are specifically indicators simultaneously for infectious and non-infectious causal factors for "S" ("OI, mainly PCP, and KS") as well as "AID" (decline in T-helper lymphocytes in blood serum). The conclusion of a new infectious pathogen and simultaneous exclusion of all non-infectious causal factors is by no means compelling, although it determines to this day the theory that "HIV causes AIDS".

Highly promiscuous behaviour and predominant receptive anal intercourse closely correlate with consumption of sexual stimulants, above all amyl and isobutyl nitrites. 95% of homosexual men in the US report regular use of nitrite (9,10). Nitrite inhalation relaxes the smooth anal muscles, raises blood flow to the penis, raises pain threshold, heightens orgasm and unleashes a mild state of intoxication in the brain. Nitrite use predominantly but not exclusively became known in homosexual sex partners, and has been approaching ubiquitous in surveyed homosexual men in Western countries since the mid-70s (11,13).

High frequency promiscuity and predominantly receptive anal intercourse very often entails concomitant increased multi-infectivity and provocation of administrating antimicrobials, chemotherapy, antibiotics, antiparasitica, antimycotica, virusstatica and corticosteroids (14). The first report by the CDC in June 1981 of five diseased homosexual men being treated for PCP contains some clinical information of their medical history and medication, because at the time, the all-encompassing description AIDS, masking the real symptoms, had not yet become entrenched: The five homosexual patients had not had sexual relations between themselves. All of the five patients used nitrites, and all five had been treated with TMP/SMX (TMP = trimethoprim, SMX = sulfamethoxazole) (15).

The substance TMP/SMX, also known as bactrim and septrin were introduced in the early 70s as a double chemotherapeutical folic acid inhibitor. Nitrite and SMX (a sulphonamide derivative) are strongly electrophilic oxidising agents. Both oxidise ferrous iron in haemoglobin to ferric, and thereby reduce oxygen-binding capacity of red blood cells. This causes methaemoglobulinaemia (16,20), a progressively life-threatening deficiency in oxygen supply into the respiration chain of the mitochondria. The latter are former bacteria, which, as multifunctional organelles, supply energy to the whole cell in form of adenosine triphosphate (ATP) produced in oxidative phosphorylation (21). Oxygen-dependent ATP synthesis and its resulting oxygen metabolites control the cell division cycle. If too little oxygen is transported to the respiratory chain, the ratio of oxidative ATP production in the respiration chain (normally about 90%) may become inverted in favour of the non-oxidative ATP production (normally about 10%). Latest experimental findings suggest that the redox balance controls the genetic expression of proteins for the enzymes of the non-oxidative ATP production (glycolysis) (22).

Under normal physiological conditions, there is a rhythm of phase-linked change between oxidative energy production in the mitochondria and the change to non-oxidative glycolysis during the late stage of cell division (the S-phase of mitosis). If, through lack of oxygen under conditions of methaemoglobulinaemia, the genetic expression of glycolytic enzymes is not sufficiently inhibited (23), the cell may, despite intact mitochondria, and the presence of residual molecular oxygen, switch to permanent non-oxidative glycolysis and cationic load reversal. This results in unrestrained cell division, which may ultimately lead to transformation to a tumour cell.

Along the oxygen transport route in the bloodstream, conditions in the most minute capillaries with a diameter below 100 nanometres, because of altered partial pressure of oxygen, are particularly favourable for the oxidation of the red

haemoglobin, which can only bind oxygen when being in reduced form. Through diffusion and association to essential fatty acids through transit routes of the basic-tissues it can deliver oxygen to individual cells. The mechanism of unrestrained activation of cell division (hyperplasia) in methaemoglobulinaemia, may, therefore, following hypoxaemic stress, above all in the smallest capillaries, affect the cells of the walls, – the endothelial cells. These endothelial cells are in direct contact with the hypoxaemic red blood cells. If hyperplastic conversion of endothelial cells occurs, that is called Kaposi's Sarcoma. On the other hand, especially in rapidly dividing cells such as in thymus-matured precursor cells of T-helper lymphocytes, ATP production can decline to a critical value, if oxygen turnover is reduced permanently even by a small amount. This is a control mechanism, which in turn may affect the rate of mitosis. This interaction of haemoglobin oxidation by nitrites and antimicrobial drugs with oxidative phosphorylation may, in a situation of increased simultaneous consumption of T-helper lymphocytes as a result of slowing maturation of T-helper lymphocytes, be in part a cause of "AID".

This chain of causal events is also supported by the "frightening possibility" (24) that nitrites may turn most classes of antibiotics into carcinogens (25). Excessive antibiotic consumption (whether prescribed or not; in a study 40% of male homosexuals admitted preventive use (26)) in conjunction with nitrites is a frequently encountered pattern of behaviour among male homosexuals especially in the large urban areas in Western countries (27).

Hypoxaemic stress can, therefore, explain the contradiction of simultaneous appearance of malignant hyperplasias (KS, lymphomas) and opportunistic infections, mainly PCP, in homosexual men (approx. 2/3 of "AIDS cases" in Western countries, excluding covered homosexual "AIDS patients" estimated by orthodox "AIDS"-doctors to amount to 50% of so-called heterosexual risk groups (28)), without ever introducing a hypothetical "retroviral" cause to explain the pathophysiology.

In contrast to this clear finding, Gallo et al tried to resolve the clinical contradiction between OI and KS by constructing a new "retrovirus HIV". Gallo et al's so-called retroviruses "HTLV-I" and "HTLV-II" are said to cause rare forms of leukaemia, i.e. cancers of the white blood cells, whereas "HTLV-III" (="HIV") is said to kill T-helper lymphocytes.

This concept has completely failed. The cytopathic effects of "HIV" demonstrated by Gallo et al have turned out to be laboratory artefacts (29). Gallo et al's claim that "HIV" kills T-helper lymphocytes could, despite changing the theories, not be confirmed (30-33).

The disease theory "HIV causes AIDS" is itself based on several serious clinical misconceptions:

1. The agent causing PCP is not as Gallo claimed a protozoon. The aetiology according to which after the destruction of T-helper lymphocytes by "HIV-infection", Carinii pneumocytes, the cause of PCP, could escape control by T-helper lymphocytes and multiply unrestrictedly, is objectively wrong. Such protozoa simply do not exist (34,35). What is involved are micro-fungi that are inhaled in the air, and which, for example, in the case of increased cell decay following hypoxaemic metabolic changes (including "AIDS" without "HIV"), find fertile terrain in the alveoli of the lungs. In this way, a harmless fungus (saprophyte) becomes the dangerous cause of PCP.

2. Contrary to what Gallo et al claimed, T-helper lymphocytes do not suppress the growth of cancer cells, because cancer cells do not have antigens through which T-helper lymphocytes could identify them (36). This means that the hypothetical destruction of T-helper lymphocytes by "HIV" and the ensuing disappearance of the suppression of KS cells cannot be the cause of KS. The predicted increase of all other types of carcinoma in "AIDS patients" resulting from the disappearance of the surveillance of cancer cells after the postulated destruction of T-helper lymphocytes by "HIV-infection" did not occur (37).

3. Contrary to the assumption of the CDC and Gallo, the hypothetical "HIV infection" of T-helper lymphocytes despite the postulated essentially alarm function of T-helper lymphocytes also for antibody production by B-plasma cells did not result in destroying defence capacity against *all* microbes. Unlike patients with impaired immune functions, E.G. intensive care patients in whom mortality following typical bacterial infections is up to 80%, strikingly in the "immune deficiency syndrome AIDS", bacterial infections are rarely seen. The CDC under the category "AIDS indicator diseases" states explicitly for "bacterial infections, frequent or repeated": "not applicable as indicator of AIDS in adults/adolescents" (37).

4. A fundamental pillar of the disease theory of Gallo et al according to which "HIV causes AIDS", is severely dented by the actual epidemiological situation over the 15 years 1982-1997. For example, in 1997 the German "AIDS Centre" registered 2736 KS cases in total with 2505 KS cases in the category "homosexuals". The remaining KS cases were in "heterosexual risk groups" or "no information on risk group". On average, therefore, there were 15 KS cases a year, which were not primarily classified as "homosexual". Because homosexual intravenous drug users are classified as intravenous drug users and at least 50% of the patients classified as "heterosexual men" and "not known" were subsequently reclassified as homosexuals (28,38), this is of the order of magnitude to be expected for KS cases classified as "non-homosexual men". Corresponding epidemiological data for the prevalence of KS are available for other Western countries (39).

Gallo et al's formulation "HIV = AID = S" is not, therefore, found to be true. "AID" (measurable decline in lymphocyte population in the blood, especially T-helper lymphocytes) though it can occur, in all members of "high-risk groups", is evidently not the cause of "S" ("OI, mainly PCP, and KS") because "S" can, first, occur without "AID" (29), and secondly, the combination of "S" (with KS) should, if the theory were correct, not exclusively be limited to homosexual patients. If, therefore, "S" is not necessarily the result of "AID", what then is the common pathogenic indicator of "AID" patients as defined by Gallo et al to be "high-risk groups" (4)?

The common factor of "AID" patients (without necessarily resulting in "S") is obviously the unusually high uptake of strongly oxidising substances (mitogens), and the huge variety of exogenous extraneous cells such as red blood cells, activated lymphocytes or sperm cells from individuals (allogenic stimulation (29,40)). It is beyond doubt that this oxidative stress (i.e. pro-oxidative vs. anti-oxidative metabolism) of "high-risk groups", can overload the detoxification capacity and waste disposal capacity of the body which is furthermore supported by the finding that asymptomatic "HIV positives" belonging to "high-risk groups" show a strong shift from reduced to oxidised glutathione (41).

The glutathione system is essential for the removal of oxygen free-radicals, especially in the mitochondria (42,43). The oxidation of the central molecule of glutathione, cysteine, to cystine, in a chain reaction reduces the build up of glutathione and accelerates the destruction. It follows that the systemic decline of glutathione concentration in HIV positives can be due to both reasons, because of decreased synthesis and increased disposal.

"The oxidative stress to which AIDS patients are subjected would lead to cellular anomalies in many cells, including lymphocytes, resulting in opportunistic infection, immunological abnormalities and neoplasia" (44).

Does this finding of the overload of redox potentials in members of "high-risk groups" mean that "HIV", too, or rather the "anti-HIV antibodies" are the result of oxidative bombardment on the cell-mediated immunity of the "high-risk groups"?

A specific load value of the diminution of the reduction force in the body of members of "high-risk groups" is hepatitis type B, in particular, in the chronically active form (45).

Gallo et al postulated in the first paragraph of the first publications in Science of May 4 1984 (except for the first rebutted premise: "Epidemiological data suggest that the acquired immunodeficiency syndrome (AIDS) is caused by an infectious agent" and the second (rebutted) premise: "AID" necessarily leads to "S"), a third premise: "Although patients with AIDS or pre-AIDS are often chronically infected with cytomegalo virus or hepatitis B virus, for various reasons these appear to be opportunistic or coincidental infections" (3).

This claim stands the clinical history completely on its head. "High-risk groups", in Gallo's definition "homosexual men with multiple sex partners, intravenous drug missusers, haemophiliacs, blood transfusion recipients and close heterosexual contacts of members of these high-risk groups" (6) were long before the so-called 'sudden' arrival of "HIV" (1978), recognised to be the most severely hepatitis-B affected groups of patients (46-50).

Hepatitis inducers (nowadays thought to be hepatitis-B, hepatitis-C) "appear to be thousands of times as infectious in clinical settings as HIV and represents a much more prevalent medical problem" (51). Hepatitis-B due to various patho-physiological reasons, especially in the chronically active form contributes significantly to oxidative stress, by restricting waste disposal and detoxification, and overloading of redox potentials. The body tries to compensate for this by increasing cortisol production. When this ultimately fails, hypercorticolism persists in a damaging way. A hypercatabolic metabolism results from this (i.e. excess cell decay vs. build up) (52). Cortisol as "synergiser" for a number of hormones and mediators effects activation of cyclic adenosine monophosphate (cAMP) and a displacement of the cAMP/cGMP ratio as principal indicator for increased cell turnover (53). The net effect is a dampening of cellular immunity and activation of humoral immunity. Resulting from the increased cell turnover, the decreased disposal of cell debris (because of the dampened cellular immunity, "AID") and the strengthened autoimmune activity, a significantly increased formation of autoantibodies occurs which above all specifically bind to cytoskeletal proteins and extra-cellular proteins of the cell matrix as antigens (54, 33).

Concluding, it is fair to assume that Gallo et al took these attributes (25) of "high-risk groups" into consideration, namely,

1. the excessive oxidative (mitogenic) stress

2. allogenic stimulation by foreign cell components

3. the sharply increased antigen auto-antibody load together with suppression of T-cell dependent immunity brought about by synergistic effects of persistent corticolism with resulting change in cAMP/cGMP ratio.

In their original paper ("Detection, isolation and continuos production .." (3)), Gallo et al were only able to cite indirect phenomena, such as reverse transcription, ultra-thin layer electron micrographs, banding of protein mixtures at given densities, which according to the established rules of virology are not acceptable as evidence for the existence of a virus or less a "retrovirus", because these indirect phenomena can also be obtained in the absence of any viral entity under certain cell culture conditions (55-60,33).

Then the question becomes increasingly pressing: how did Gallo et al manage to produce a protein mixture in cell cultures and in the test tube, which as the substrate in the "AIDS-test" when in contact with serum of people in "high-risk groups", resulted in a given rate of antigen antibody-reaction for single proteins (6)?

Gallo's papers, though written in highly technical language do not reveal this secret of test-constructing. Only in 1987 when the disease theory "HIV causes AIDS" led to the introduction of a highly toxic DNA chain terminator (azidothymidine = AZT = Retrovir), was some light shed on this matter when two of Gallo's former collaborators and co-

authors of the original publications in *Science* of May 4 1984 (3-6) revealed the essential details. Mangalasseril Sarngadharan and Phillip Markham (collaborators of Litton Bionetics, Kensington MD, USA) published the biochemical methods used by Gallo et al whereby they manipulated the protein mixture, which due to self-defined conventions are said to be "HIV antigens" (59).

To start with, Gallo et al biochemically prepared cell components obtained from members of "high-risk groups" according to the self-defined rules of "retrovirus production". This procedure only "from time to time" and only transiently (61) led to the production of unspecific phenomena as surrogates for the existence of a new "retrovirus". Then they mixed lymphocytes from patients in "high-risk groups" with exceptionally rapidly dividing leukaemia cells (3,4). This cell mixture was then subjected to the effects of certain biochemical substances. They go on to say that "in vitro stimulation was achieved by mitogens or added cells (allogenic antigens) ... Certain manipulation of culture conditions improved the result, for example, co-cultivation of patients' cells with peripheral white blood cells, which were stimulated by mitogens, from non-infected donors.

The "virus isolation" of cultured cells was also significantly facilitated by adding hydrocortisone to the culture medium" (61).

Knowing the specific antigen auto-antibody status of "high-risk groups" patients, it is possible, therefore, to trigger, on demand, an antigen mixture appropriate to the auto-antibody repertoire in serum from high-risk patients, in cell cultures of human lymphocytes, co-cultured with leukaemic cells when subjected to specific biochemical manipulation.

The apparent proof that in the antigen mixture one is dealing with "retroviral" proteins, brought about by the demonstration of a naturally occurring repair mechanism - reverse transcriptases, produced particularly copiously in cancer cell cultures to repair DNA and renew chromosome ends, hence co-cultivation with leukaemic cells in Gallo et al cell culture (3,4), as well as proof of exocytotic virus-like particles (frequently occurring transport particles to expel intra-cellular components from mitogenically stimulated cells) as proof of "isolation and continuous production" of supposed retroviruses is misinterpretation (33).

That Gallo et al's sensational discovery of a "new retrovirus" was in fact a laboratory artefact is made explicit by Gallo et al's expressly stating that "HTLV-I" (isolated from T-cells in 10% of "AIDS patients") and "HTLV-II" from the "family of retroviruses" in "AIDS patients", were also discovered and demonstrated (3,4). Later on, there was no further mention of "HTLV-I" and "HTLV-II" being "isolated from T-cells of AIDS patients". Nor were there noticeable occurrences of leukaemia in "AIDS patients". The "isolation" of "HTLV-I" and "HTLV-II" was a laboratory artefact due to the rules of "retrovirus-production" of Gallo et al. By analogy this finding accounts for "HTLV-III" (= "HIV") as well.

In effect, therefore, Gallo et al were adapting conditions which they knew to be conducive to antigen formation in the body of "high-risk patients", to laboratory conditions. The difference is that in cell culture as opposed to the body of "high-risk patients", no antibodies are present because the B-plasma cells are absent. Then it is possible, at a certain arbitrarily fixed auto-antibody level, to demonstrate an antigen-antibody reaction when the antigen mixture of the cell culture is brought in contact with sera of "high-risk patients". This is exactly the principle employed in "anti-HIV-antibody tests". In mirror image fashion, the artificially produced antigens bind to the auto-antibodies, whose presence was to be expected because of the well-known pathophysiological overload of "high-risk patients".

In describing the recipes of Gallo et al's, who covered their laboratory-tricks behind the dust screen of patents, the irrational reduction of "AID" to the effect of a seemingly new infectious cause (3) and the ignoring of the clinical effect of chronic hepatitis (3) becomes apparent: as a claim used to create pressure to introduce the patented "antibody test

system" of a "new retrovirus" found in the National Institute of Cancer.

The laboratory finding of "HIV positive" which may be diagnosed in those belonging to "high-risk groups" depending on the quantity and personal reaction pattern of antibodies, may also be made in rare cases in those not belonging to "high-risk groups" for a number of extremely diverse reasons.

Gallo et al's expectations regarding the dynamics of the spread of "HIV" have, contrary to the horrendous predictions, not been fulfilled in the real biological world. In Germany, for example, according to official figures for the 15 years 1982-97, out of a population of 82 million, 60.000 have been notified as HIV positive, i.e. more than 99.9% of the population are personally not affected by "HIV" and "AIDS". The official government forecasts, until now uncontradicted, spoke of there being more "AIDS cases" by 1996 than there were inhabitants. At least every other person was supposed to have died by 1996, unless a vaccine or drug against the "absolutely" fatal plague had become available (60). In the former East Germany, there have been a grand total of 252 cases in a population of 16 million, and that despite massive migrations (since the fall of the wall) up to the end of 1996. Over the past decade in the whole of Germany there has been a very constant 2-3.000 number of people diagnosed annually as HIV positive. 95 % of these have been classified as belonging to the "high-risk groups" of "homosexual men" and "IV-drug user" (homosexual IV-drug users are counted as ordinary IV-drug users). 5% of "HIV positives" are considered to be false positives, but cannot be identified as such by the test.

At most 2000 "HIV positives" develop AIDS annually, and 1300 patients die annually of "AIDS" (actual cause of death is not revealed). Of the supposed 60.000 HIV positives (figures are very unreliable because of unknown multiple reporting), 50.000 are still officially alive today. 54% of all "AIDS patients" gave their addresses to be one of the six largest cities, in which 10% of the general population also live. Opposed to that in 90% of the remaining inhabitants only 44% of the notified "AIDS cases" occur.

For example the disease rate and death rate of "HIV-positive" haemopholiacs registered in these six cities is twice as high as in "HIV-positive" haemophiliacs living outside of those cities. In these cities (Berlin, Hamburg, Köln, Düsseldorf, Frankfurt and München) the university clinical "AIDS-treatment centres" are located which report the highest "AIDS"-disease- and death-rates to the national AIDS-centre. As the positions of collaborators the "AIDS-ambulances" and "AIDS-stations" of these university clinics mostly are paid by the pharmaceutical companies, the connection between Medicine and market ("AIDS-test", "AIDS"-medications") becomes all too obvious. Very intriguing is the comparison between the "capitalist" West-Berlin and the former "socialist" East-Berlin.

In the period of 15 years from 1.1.1982 to the 1.1.1997 in West-Berlin (2,2 million inhabitants, which make less then 3% of Germany's population) 3083 "AIDS-cases" have been registered which are 20% of all German "AIDS-cases". In the same period (including 7 years of unification with West-Berlin after the fall of the Berlin wall 1989) in East-Berlin (1,3 million inhabitants = 1,6% of the German population) only 152 "AIDS-cases" are registered, which make 1% of all German "AIDS-cases". This very intriguing, by chance historical and model-like data (38) proofs wrong the premise of Gallo et al. that "epidemiologic data suggest that the acquired immunedeficiency syndrome (AIDS) is caused by an infectious agent". The disease rate when brought in connection with the whole population is obviously a very rare medical event, not dependent on a ubiquitous transmittable mass-virus, but determined by life-style in a largely commercialised subculture and/or by uncritical medical intervention in Western society of super-abundance.

Or pathophysiologically spoken: "AIDS-patients" come down due to a lack of power of reduction (caused by superoxidation and /or hypoxaemia) in the midst of a redundant medical over-supply.

To argue against Gallo et al. refers to Africa, which is uncritically presented by mass-media as the "dying AIDS-

continent". Too, in this context the world of facts seemingly is overwhelmed by a virtual, only imagined world of information.

In Africa south of the Sahara, the annual increase in population was about 100 million inhabitants over the last decade, even though the latest report on the world population states, that according to a lot of population experts "in the third world the plague supported the birth-planing more than any earlier programs" (63). Due to the lacking medical infrastructure and low budgets in the health care system (in most states south of Sahara the average annual spending per head of the population for providing health care is 6 US$, a single complete "AIDS-test" - 2x ELISA-test, 1x Westernblot - costs much more than 6 US$) the "AIDS-test" is not widely used. Instead of this the World Health Organisation (WHO) transfers certain amounts of money to the health authorities of the various countries for "AIDS-education" in order to get estimated incidental rates of "HIV-infection" and "AIDS-cases" which are not verified by the WHO.

WHO-experts use these estimates in calculations based on the supposed "dynamic of distribution" of the "HIV-plaque" and present the resulting numbers to the world media as "HIV-infection" and "AIDS-disease" in Africa. Usually, in the subsequent media reports the speculative "HIV-infections" and "AIDS-diseases" are lump-summed and wrongly reported as "AIDS-cases" in Africa. This is the way the manipulated numbers of more than 20 million "AIDS-cases" in Africa (app. 90% of the world-wide reported "AIDS-cases") came into existence without any substantial base of knowledge (64).

The fictious loom scene of a "people murdering AIDS-plaque" in the "global media village" acted again enhancing on the selling of "AIDS-tests" and "Anti-HIV-medications" (euphemicly termed "cocktail-therapy") in western countries, in a way that "poor Africa" unwillingly was misused to increase sales in the "rich West".

The data on the clinical, immunological, virological and epidemiological progress since 1984 show beyond any doubt that the disease-theory "HIV causes AIDS" has no concurrence with the biological reality. As a marketing strategy Gallo's manipulated "AIDS-test" has been extremely successful. But this at the cost of the health and life of uncounted children, women and men who, from a medical ethic point of view became victim to "clean torture with case of death" induced by the arbitrary medical death-sentence of a "HIV-positive" result. Medical ethical behaviour "according to best wisdom and conscience" must signify to make, out of your own, the effort to inform yourself on the basis of existing data about possible manipulations in diagnostic and therapy and to use the given alternative therapies instead of inducing fear blind with rage (33). *

Address of the author:
Dr. med Heinrich Kremer
Metzendorfer Weg 36
D 21224 Rosengarten (b. Hamburg)

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VIRUSMYTH HOMEPAGE