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Answers to Questions raised by South Africa's President Thabo Mbeki and Health Minister Manto Tshambalala-Msimang, M.D., on the active Mechanism of AZT of 23 February 2000 and HIV/AIDS of 6-7 May 2000.

Q - Is AZT incorporated in DNA?

A - Azidothymidine (AZT) is a nucleoside into which adverse to the natural nucleoside thymidine an acido group (=N3) was incorporated. Like all natural and synthetic nucleosides, AZT can only be incorporated as a nucleotide in combination with three inorganic phosphorus atomes into DNA or provirus DNA. Numerous experimental studies have demonstrated that up to 99% of the nucleoside AZT is not metabolized in the nucleotide azidothymidine triphosphate (AZT-TP). Therefore, in theory, 1% of the AZT accepted by the human cells could be incorporated in the DNA nucleus or in some DNA provirus. However, in living human cells, nobody to date has proved the actual incorporation of AZT-TP in the DNA nucleus or in any DNA provirus. Other assertions are completely without foundation.

Q – Can AZT stop replication of the so-called HIV viruses?

A - The theoretical possibility of 1% of the AZT absorbed being incorporated in the DNA as AZT-TP means that 5 mg of the prescribed minimum dose of 500 mg or 15 mg of the prescribed maximum dose of 1,500 mg AZT could be incorporated in the DNA nucleus or in some DNA provirus. According to data from the AZT manufacturer Glaxo-Wellcome, the nucleoside analoge AZT is accepted after absorption in the alimentary canal in numerous immune cells and non-immune cells. Only a fraction of this low amount of substance from the 5-15 mg of AZT would be available for incorporation in the so-called HIV infected TH1 lymphocytes (identical to the T4 or CD-4 cells of type 1). According to the HIV/AIDS theory valid since 1995, the HIV virus should increase a billion-fold daily. *So* the portion of the AZT triphosphate taken up by the HIV-infected TH1 lymph cells in comparison to the amounts of AZT-TP taken up by all the non-infected TH1 lymph cells as well as other immune cells and non-immune cells would be theoretically and actually too low that replication of the so-called HIV virus could be stopped. However, the fixing of the active dose of AZT to inhibit the so-called HIV virus presumes the objectively refuted assertion that ATZ is incorporated as ATZ-TP with a high affinity exclusively into the so-called HIV virus's DNA provirus.

Yet the active mechanism of AZT is another matter. The 99 times higher amount of AZT that cannot be incorporated in DNA, which does not combine with three inorganic phosphoric atomes, actually reacts in a much shorter time with non-DNA molecules in the alleged HIV-infected TH1 lymph cells and in TH1 lymph cells not infected by HIV as well as in other immune cells and non-immune cells. The reactive azido molecule group is used in the experimental mitochondrion research in order to block the cytochrome oxidase enzyme in the mitochondria respiration chain. The intact mitochondria (former bacterial cell symbionts that appear in all human cells

except red blood corpuscles) work with molecular oxygen (0_2) to produce 90% of the adenosine triphosphate (ATP) energy carrier molecules necessary for human life.

The blockade of the respiratory enzyme cytochrome oxidase by AZT prevents the transmission of electrons on O_2. The immediate result is reduced ATP production and an increased synthesis of toxic oxygen radicals. The cells suffer from loss of energy. Within a few minutes and at latest within three hours, there is a reaction to the AZT that cannot incorporate in the DNA. Meanwhile, replication of the DNA nucleus or any kind of DNA provirus (that always depends on DNA nucleus replication of active cells) would require 40-72 hours after the suggested incorporation of AZT-TP. The blockade of oxygen respiration and energy production in T-helper lymph cells (T4 cells or CD-4 cells) resulting from AZT medication *leads* specifically to premature death of immune cells or, under certain conditions in accordance with the law of nature to switching of maturing T-helper immune cells to type 2 T-helper immune cells (TH1-TH2 switch) as part of a type II counter regulation.

Both reaction forms result in immune system weaknesses. The premature dying particularly affects TH1 cells. Their depletion is the principal immunological characteristic of so-called HIV-positive and AIDS patients. It is the function of TH1 cells to eliminate intracellular pathogens such as parasites, fungi, mycobacteria, and viruses. Since the discovery of nitric oxide (NO) production in human cells (Furchgott and Ignarro, 1987, Nobel Prize, 1998), it has been beyond any reasonable doubt that NO gas production in TH1 cells is indispensable for elimination of intracellular pathogens. If the TH1 cells producing NO gas are lacking, opportunistic pathogens may develop (AIDS). The function of the TH2 cells is to prompt antibody formation. TH2 cells produce no NO gas to eliminate intracellular pathogens.

Numerous studies have proved that so-called HIV-positives show a loss of TH1 cells and a dominance of TH2 cells at the earliest possible stage in so-called HIV seroconversion. It is biologically unimaginable that all T-cells should infested at the stage of the so-called HIV infection by HIV, since the prevailing TH2 cells are intact, and antibody production is even increased. The TH1-Th2 switch, which leads to cellular immunity weakness, must therefore have other causes according to the laws of logic.

The active mechanism of NO and AZT (=N3) is identical: inhibition of cytochrome oxidase in complex IV of the mitochondria respiratory chain is the essential physically and patho-physiologically active factor in human cells through NO and also AZT. Depending on duration and dose of increased NO production as well as medication of AZT, special cell types and disposition of patients, a concurrent rise occurs in cell death (apoptosis, necrosis) and/or TH2 cell dominance (opportunistic infections = AIDS), tumor formation (e.g., Kaposi's sarcoma, lymphoma, carcinoma), or degeneration of the skeleton and heart muscle cells as well as nerve cells).

The causes of AIDS in Western countries have been clarified epidemiologically and patho-physiologically without any reasonable doubt in thousands of experimental and clinical studies. Without any doubt, unusual accumulating load factors for exogenously and/or endogenously induced NO over-stimulation have been proved in all so-called risk groups. There is no rational, comprehensible biological reason to

assume that the combination of these immune stress factors in Western civilization should have remained completely ineffective and without recognizable disease results. Strong or lasting NO over-stimulation leads as a counter-reaction to increased cell death and/or in the case of T-helper cells to TH1-TH2 switch with inhibition of the cellular production of NO and disturbance of the mitochondria's oxygen respiration.

The clinical results (including AIDS) are in no way puzzling but rather programmed in accordance with law of the biology of evolution. The so-called HIV — the virus that nobody has actually isolated so far according to the standard rules of retrovirology and that was deduced to exist only due to unspecified molecular markers to postulate it hypothetically as cause of disease— is neither sufficient nor necessary. This postulate veils the real cause of AIDS. At the point in time when the "HIV causes AIDS" disease theory was developed, the researchers did not know of NO production in human cells or the existence of two forms of T-helper immune cells with and without NO gas production. Nor were we aware of *the* dependence of the function of eliminating intracellular pathogens to TH1 cells and their NO gas production or *of* the regulation of oxygen respiration in the mitochondria by NO and its derivatives. Failure to consider these research data by HIV/AIDS-researchers can only be based on ignorance or unwillingness to learn.

This explanation of AIDS' causes and the active mechanism of AZT is supported by the fact that after introducing clinical medication in malignant forms of lymph cell cancer with substances analogous to nucleosides (that show the same active mechanism as AZT), a massive loss of TH1 cells appeared in the same form among all treated, and so did the inverse ratio of T4/T8 lymph cells and opportunistic infections. Exactly these immunology data and clinical symptoms define the AIDS syndrome. Since presentation of the conclusive data from NO research, cytokine research, mitochondria research, and other experimental and clinical research sectors from the mid-1990s, there are no longer grounds for rational doubt about the actual causes of AIDS in Western countries.

African clinical standards for diagnosing AIDS and test-procedure standards for antibodies against the so-called HIV are in no way congruous with those of Western countries. However, regardless of race and country-specific diagnostic practices, all human beings are identical in the response that evolution has programmed into human immune and non-immune cells when affected by nitrosative and prooxidative stress conditions.

In Africa it is particularly chronic inflammatory and infectious processes, protein shortages, and malnutrition (nutritional AIDS), contamination of drinking water with nitrifying bacteria, and nitrosamine burdens in nutrients that can lead to clinical symptoms of opportunistic infections (AIDS) as result of induced TH1-TH2 switches.

Chronic infections stem from (1) mycobacteria, such as chronic tuberculosis or the leprous form of leprosy, (2) spirochete bacteria such as the tertiary form of syphilis, (3) malaria pathogens, trypanosomal, toxoplasmic, and other parasites, (4) fungi pathogens such as pneumocystes, candida forms, histoplasms, crytococcus, and

many others. These always result from too weak a TH1 immune response and a switch in the TH1-TH2 immune cell balance to TH2 immune state with increased antibody production. Infections such as worm parasites trigger a priori a TH2 immune response that can become chronic.

If the clinical symptoms of unspecified sort and duration become chronic, these have been diagnosed in Africa since 1985 as AIDS, based on the Bangui definition, without test evidence of so-called anti-HIV antibodies. This pragmatic procedure led to the apparent proof of a suddenly rapid increase in Africa of so-called HIV-caused AIDS indicator diseases.

Arbitrary projections from small random samples of so-called HIV-positive serum tests and sweeping clinical AIDS diagnoses in Africa still serve the World Health Organization, UNAIDS, the Western countries, and the international news media as basic proof of the HIV pandemic in Africa and the threat deduced from that pseudo medicine it poses to all humanity.

Since children, women, and men can suffer from chronic inflammatory and infectious processes due to general living conditions in developing countries, these AIDS cases can be manipulated at will in terms of sweeping medical statistics as proof of heterosexual transfer and mother-child transfer leading to the so-called HIV in Africa. Since these undoubted facts are logical in view of the high scientific standards of Western medicine and can be understood with little intellectual effort, there is no rational reason to assume that it involves a tragic scientific error in case of an intended mass poisoning with the proven mitochondria inactivator azidothymidine (AZT). No HIV-AIDS researcher and no medical specialist to date has been able to answer the inescapable medical-ethics question of why medicinal application of AZT and other substances that trigger the loss of TH1 immune cells, reverse the ratio of T4/T8 lymph cells, and opportunistic infections should be indicated to treat patients in danger to develop Pre-AIDS and AIDS.

That AZT is effective as inactivator of mitochondria can be derived from the bio-logical fact that azidothymidine was isolated from herring spermatozoa in 1961. The spermatozoa of vertebrates are not allowed to transmit these cellular symbionts to the ovum and have to inactivate their mitochondria before penetration into the ovum. In the case of vertebrates, only the mother's cellular symbionts will be transmitted to daughter cells. AZT was produced synthetically in 1964 but banned from human trials after tests on mice and rats diseased with leukemia resulted in developing lymph-cell cancer. AZT was used clinically for AIDS patients from 1986 on, but without evidence of its actual incorporation in any provirus DNA and without testing for possible mitochondria damage.

The question if AZT can stop replication of the so-called virus is inseparably linked with the question concerning evidence of the so-called virus.

The so-called anti-HIV antibody test has been stocked with nitrosatively and oxidatively stimulated human stress proteins as antigens from lymph cell cultures of patients manifesting AIDS and from co-cultivated lymphatic leukemia cells. The test substrates have been calibrated so that a positive test result will particularly appear

from a certain amount of unspecified antibodies which in the blood serum of test subjects characteristically indicate an outlasting TH2 immune-cell response and an increasing antibody reaction. The test reaction level and the number of test antigens in the so-called anti-HIV antibody tests have been determined arbitrarily. For this test reaction level and the required test-antigens there are no internationally obligatory standards. In Africa, for example, a reaction in so-called HIV tests is usually rated a positive test result with fewer and different test antigens than in Western countries. Since no antibodies form in the human immune system that react only with those antigens against which they were originally formed, the statement that so-called anti-HIV antibody tests react exclusively with antibodies formed in human organisms against antigens of the so-called HIV is already objectively in error for this biological reason. So-called HIV test antigens, for example, react demonstrably with antibodies against tuberculosis, malaria, and pneumocystis pathogens as well as many other antibodies against microbial and non-microbial antigens.

In Western countries too, applied determination of so-called viral load with the help of the PCR laboratory technique is completely unsuitable to detect the so-called HIV's RNA according to the inventor of this DNA-multiplying method, the Nobel Price laureate Kary B. Mullis. Nobody to date has actually isolated a natural RNA sequence or a provirus DNA sequence of so-called HIV. All publications on the so-called isolation of so-called HIV show nothing else than findings of unspecified molecular markers that are arbitrarily interpreted as "finger prints" of the so-called HIV.

Other data and scientific findings can not be expected in view of the pressing epidemiological, immunological, cell-biological, and clinical proof that type II counter-regulation of human immune and non-immune cells as well as development of AIDS-indicating diseases are evolutionary programmed under certain conditions. For physiological and patho-physiological understanding of these immunological and clinical phenomena, the assumption of an infection with so-called HIV is neither sufficient nor necessary but objectively redundant. It was assumed at the conference of leading HIV-AIDS researchers in 1997 that no disease mechanism of the so-called HIV could be proved (M. Balter, 1997; *Science* 278: 11399-11400).

The question is often raised whether AIDS could be transmitted in another way, sexually, into the bloodstream, via the respiration system or other infection routes if one assumes that the so-called virus is not the cause of AIDS.

Many people have mental difficulty in separating certain facts of the immune system, since it is suggested to them that the immune cells of so-called HIV-positives and AIDS patients react primarily to infectious pathogens that are usually transmitted sexually or from a so-called HIV-positive mother to her children. However, the biological truth is that human immune cells are influenced by a number of non-microbial immune stressors as well as microbial immune stressors (antigens and toxins). Thus AIDS indicator diseases must not always be triggered primarily by infections of any kind, as demonstrated by the examples of nutritional AIDS, transplantation AIDS as a result of immunosuppressive therapy, or by AIDS after AZT medication or after medication with other nucleoside analog drugs.

A homosexual African, for example, can also become ill of nutritional AIDS, even if he never takes the risks of an anal-receptive homosexual from the West. However, he could be registered in Africa as a heterosexual HIV-AIDS patient. Nor would the apparent mother-child transmission of AIDS have to be caused primarily by infection in any way. Since the immune cells and non-immune cells of the fetus show a dominance of TH2 or type 2 cytokines respectively during pregnancy, the disposition for opportunistic infections after birth (AIDS) depends mainly on whether the mother has transmitted enough intact maternal antibodies and a stable TH1-TH2 immune cell balance can be adjusted in the child during the first months of life.

In case of the mother lacking nutrition, being poorly nourished, or suffering toxic damage before and during pregnancy, the maturing of the infant's T-helper immune cells will be substantially affected. Opportunistic infections (PCP) were already diagnosed during the 1940s among prematurely born children and orphans in Europe. Opportunistic infections also appear among children with congenital thymic aplasia. That children of nutritionally, infectiously, and toxically damaged mothers in Africa should suddenly be infected by so-called HIV if they develop opportunistic infections cannot be comprehended rationally, not even if the so-called anti-HIV antibody test shows a positive result for the reasons mentioned above. Treating such children preventively or therapeutically with AZT or other nucleoside analogues would then be inhumane treatment in the sense of the UN Declaration of Human Rights — even when one works from the assumption that the postulated so-called HIV would exist and would be transmitted from mother to child. The restricted or unrestricted treatment of the not yet matured immune cells of a newborn child by means of substances that demonstrably largely damage the maturity of immune cells fulfills the facts of the case for deliberately inflicting bodily harm with fatal results and must be condemned as especially inhumane treatment internationally.

President Mbeki's questions to the conference of experts on 6-7 May 2000 in Pretoria show some of the basic misunderstanding that can arise from viewing AIDS as the exclusive result of a sexual infection and taking no account a priori of all other immune stressors (whether or not sexually associated, infectious, or noninfectious). For example, more than 90% of those older than age six in Western countries indicate/show antibodies that also react to pneumocystes. But only a few human beings fall ill of pneumocystis carinii pneumonia (PCP), the most frequent AIDS-indicating disease in Western countries. The pathogen is a fungus that is transmitted airborne from person to person. If a human being becomes diseased from such an opportunistic PCP, it depends entirely on whether enough TH1 immune cells are available to produce the cytotoxic NO gas in order to eliminate PCP pathogens after specific signals from antigen presenting cells and toxin stimulation through pathogens. In case of the disease, the PCP pathogens benefit from cellular immunity weaknesses, regardless if the precedent TH2 dominance was resulting from stressors, that were infectious or noninfectious, sexually transmitted or not.

Gender and manner of sexual transmission may play a role, but they may just as easily not be a factor. Other pathogens that trigger opportunistic infections may gain advantage from previous chronic infections, even though they produce no opportunistic infections themselves. One knows such interactions in Western countries, for example, among surgical patients after operations and trauma as well

as among patients in intensive-care wards. Such interactions of chronic and opportunistic pathogens are frequent in developing countries. They may have nothing to do with so-called HIV, not even if the so-called HIV-test is positive and the T4 cell count is low. On the contrary, such laboratory findings can be given in case of any marked TH2 immune-cell dominance and existing chronic infections without the presence of any so-called HIV. However, an AZT medication would even be counter-indicated if the existence of the so-called HIV were proved, since such so-called HIV would die only when immune cells die and because it would kill more immune cells that were not infected with the so-called HIV. But these biological conditions do not mean that AIDS is “transmitted”, since AIDS is the clinically resulting syndrome and not the cause of the acquired TH1 immune cell weakness and the inhibition of toxic resistance-gas production. Transmitted are pathogens that can primarily be involved in developing a TH1 immune weakness or can profit secondarily in case of an existing TH1 weakness.

Yet even among homosexuals, these transmissions in no way result only via the sexual channel but though all possible access channels.

The superficial differentiation between heterosexually and homosexually associated transmission of so-called HIV serves Western HIV/AIDS propaganda as a manipulative suggestion of a fatal so-called HIV infection transmittable to anybody through sexual contact. It ignores infectious immune stressors and involvement of non-infectious immune stressors, trivialised as cofactors (which have been very effective disease triggers for millions of years without so-called HIV). However, the predominantly homosexual transmission of so-called HIV infections in Western countries and the heterosexual transmission of so-called HIV infections in African countries are not explained by HIV/AIDS researchers as result of special infectious and non-infectious risks of a minority of homosexual patients and the general living conditions in Africa countries but as a result of the demonstrably special sexual compulsiveness of homosexuals and African men and women.

During the past 15 years the international mass media have not left out any cliché on the fantasized abnormal sex life of African men and women. In order to avert the alleged pandemic for all humanity, it is demanded quasi in sheer solicitousness that pregnant women and newborns in Africa be treated with AZT. As start-up help and for seemingly humanitarian reasons, pharmaceutical concerns in cooperation with the WHO and Western countries are offering AZT and other nucleoside analogues at dumping prices. The crucial question in this economic who-done-it is no longer if AZT can stop so-called HIV but if South Africa can serve as the gateway for AZT in developing countries.

A problem that first came to public light through the change in knowledge of medical research during the past decades is intensifying for Western countries as well as developing countries: the increasing abuse of chemo- antibiotics and mass *vaccination* since the end of World War II. Both factors can favour a predisposition for the long-term prevalence of TH2 illnesses such as allergies, ectopic skin diseases, chronic arthritis, certain autoimmune diseases, AIDS, cancer, etc. The reason for this is the lack of training on TH1 immune cells and the shift in the balance of TH1-TH2 immune response. An indicator for this two-edged change in the

infectious load profile in Western countries is the fact that practically only patients from age groups born after World War II were diagnosed as AIDS patients. The same shift in the ballance of TH1-TH2 immune response applies to patients with organ transplants (without genetic disposition) who have developed transplantation AIDS. This acquired disposition has an even graver impact under the general living conditions in developing countries than in Western countries where the change in disease has already been clearly recognized in the growing number of chronic diseases compared acute illnesses.

During the ongoing decade, evolutionary biology laws of co-evolution must be discussed anew in light of medical research's fundamental findings in the 1990s. A future-oriented and rationally based health-care and social policy must be oriented within this context — not in the context of irrational theories that have caused the waste of enormous scholarly and economic resources.

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