Study Group AIDS therapy

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To those affected
Their doctors and carers
To institutions
To Media

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AIDBS: Antibiotic induced deficient bacteria syndrome: The HIV-activating bacteria

Dear Sir or Madam

The translocation of bacteria and its parts from the intestinal lumen into the tissue of the intestinal mucosa, its lymphatic tissues (GALT) and later via the blood circuit into the entire organism is considered nowadays as a central mechanism in the development of diseases defining the AIDS-syndrome.
Man has established a close symbiosis with the multitude of bacteria, fungi and other strains residing in his gut, that show a genetic variability which exceeds by far the one of his own genome. Segmented, filamentous bacteria (SFB) residing on the gut mucosa build the short chain fatty acids (SCFA) which are either used for the building of the gut tissues, as also for the building of natural defence substances (defensins), of various digestion materials and the building of substances such as lactic acid, which play an important role in the competition between bacterial strains on the resources. By this activities residing bacteria in the gut regulate via their products and its signalling the building of specialised cells in the gut mucosa, which from their side produce defence substances, hormones, growth factors and structure recognising receptors (termed as toll-like receptors), that induce the formation of specialised proteins (termed as tight junctions) and specialised immune cells in intestinal tissues, all of which are needed to maintain the inpermeability of the gut mucosa and the immunological border in the gut. Beside these activities and the reduction of sulphur compounds, residing gut bacteria produce from nutrients materials such as biotin, folic acid and vitamin K and the energy carrier molecule ATP, which are circulating throughout the hole gut tract, where the levels of reduced oxygen, transported into cells by means of glutathione molecules, nitric oxide, oxygen radicals and PH determine the composition of the gut flora and of the allocation single bacterial strains in certain sections of the gut. (**E1**) Frequently used large-scale antibiotics such as TMPSMX block the formation of the enzyme dihyropholate reductase, which is needed for the building of tetrahydropholate, used for the building of glutathione molecules in the liver, used for the stepwise reduction and transportation of oxygen into the cells and for building of tetrahydrobipterin (TH IV), needed for the building of nitric oxide gas (NO) used by killer cells for the attack on cells containing fungi, viruses and mycobacteria.

If bacteria can penetrate into the tissue of the gut mucosa under particular conditions they are caught up by specialised macrophages, which can recognise them by means of toll-like receptors, which helps to prevent the their entrance into local and distant lymphatic tissues and into the bloodstream and the activation of immune cells. If at this point bacteria cannot be

eliminated completely they can enter in lymph nodes in the mesentery of the small intestines and finally into the liver, which is specialised on the elimination of foreign, potentially pathogenic structures. If parts of bacteria despite these defence mechanisms can enter the blood stream, they are recognised as antigens by specialised immune cells. If the translocation of bacterial structures into the intestinal mucosa persists, an ongoing activation of antigen-presenting dendrites takes place, so that after prolonged activation of CD-4-T-cells there is a decline in CD-4-T-cells, in T-regulatory cells, Th17 T-cells CD-8 cells and macrophage, all of which are controlling immune reactions in the entire body. (**E2**)

After a continuous translocation of bacteria from the intestinal lumen into the tissues of the gut mucosa, a diminished transportation of reduced oxygen into the cells due to the blocking of the building of glutathione molecules in the liver and of the building of NO by antibiotics and the blocking of immune cells after their over-activation, changes in the bacterial flora in the intestinal mucosa and the mucosa of the oral cavities and genital tract are occur, resulting in an overgrowth of certain strains which leads to changes in the interaction between its metabolic products (for example butyrate) and immune cells present in these tissues. In this situation anaerobic, butyrate producing bacteria can induce the building of the so called HIV-transcription activator (HIV-1-Tat), which is seen as a product of the "human immune deficiency retrovirus (HIV) considered to be a independent, transmittable virus, causing the fast course of more than 30 infectious diseases, that can defining the AIDS-syndrome with the presence of a positive HIV-test result. (From another viewpoint the "HIV-1-transcription attractor" can be seen as cellular signalling occurring after the contact of cells with parts of oxidised, genetically mutated bacterial or fungal strains, which is activated under certain conditions. When this activation of the HIV-1-Tat has happened, and a positive result in HIV-testing occurs, the supporters of the HIV-AIDS- hypothesis speak of a co-infections of HIV with the various strains that cause the AIDS-defining infections. (**E3**)

Various bacteria of the gut (for example Clostridium difficile, Fusobacterium nucleatum, Clostridum cochlearum, Eubacterium multiforme), of the oral cavities (for example P.Gingivalis and of the vagina (for example Anaerococcus tetradius, A. vaginalis, Peptoniphilus assachrolyticus and A. lactolyticus can trigger in this way the activation of the HIV-transcription activator, at which two kinds of enzymes are considered to play a important role.

As it is known since many years, agents of various sexually transmittable infections such as N.gonorrhoeae, T. vaginalis, C.trachomatis, and Herpes Simplex which are resistant to various antibiotics, can induce via the activation of the toll-like receptor 2 the building of the HIV-transcription attractor (HIV-1-Tat). The same is done by various mycobacteria such as M.avium and M.tuberculosis or by the causing agent of Hepatitis C.

Mycoplasma, the smallest causative agents in the organism, which nowadays show resistance to various antibiotics, play a mayor role in genital infections, that cause in women infertility and complications in pregnancy, activate via lipid bound membrane proteins (LAMP) toll-like receptors releasing inflammatory messengers, which from their side activate the so-called HIV-terminal repeats (HIVLTR) and by this way the replication of the products of the alleged HIV-retrovirus.

Bacterial translocation and the following over activation of immune cells only takes place when bacteria in the outer and the smaller intestines, which produce the materials for the building of intestinal tissues and the substances for its protection are damaged by the repeated administration of antibiotics. In various trials carried out since the late 1980ies it has been

demonstrated that bacterial translocation could be induced neither by weakening the macrophages nor by the inhibition of T-cells, but easily by the administration of antibiotics. (**E4**)

When under ART-treatment bacteria in the gut are decimated day by day due to its bacteriostatic effects, in many patients a decrease in the activation of immune cells and of circulating bacterial parts is occurring, resulting in an increase of CD-4-T-cell counts. These values do not increase in so called ART-non-responders and never increase to the values measured in sane persons in all receivers of ART. Even after the complete elimination of the so-called HIV-viruses by ART ("viral load 0" the amount of bacterial liposacharides and bacterial DNA remain elevated with the consequence that after an increase in the defence capacities and the immune border in the gut for some time, a progression of AIDS-defining diseases takes place. The massive daily elimination of potentially pathogenic bacteria by ART is apparently faced by an ongoing damage of the commensally acting bacteria producing materials for the building of the gut mucosa and the protecting film on it. The suppression of bacterial growth by protease inhibitors in ART is faced by the suppression of the growth of proteases in cells of the gut mucosa, which need a high degree of cell division to maintain the inpermeability of the gut. Even at a total elimination of the so-called HIV-retroviruses by ART (viral load 0), which are the product of the activation of immune cells by bacterial lipposacharides after bacterial translocation, bacterial lipposacharides remain elevated.

As it has been shown in various trials, the damage to segmented, filamentous bacteria in the inner and outer intestines cause a sever decline in short chain fatty acids (SCFA) followed by a decline in acetate, butyrate and proprionate. When antibiotics such as Penicillin induce changes in the morphology of bacteria and their adherence on the intestinal mucosa in the small intestines, there is a decline in the defence capacity against pathogenic bacteria such as Salmonella enterica, Escheria Coli and Clostridia difficile, so that certain pathogenic strains can propagate themselves more easily. Beside this effect, penicillin augments the inflammation enhancing effect of streptococcus pneumoniae on the toll-like recptor 2, which plays an important role for the formation of all immune cells.

Many of the nowadays used antibiotics achieve their bacteriostatic effect by enhanced production of oxygen radicals (ROS, which facilate its attack on the bacterial cell wall, what due to following changes in the bacterial metabolism and DNA strand breaks causes its death. Other antibiotics block directly the formation of the cell skeleton and of DNA in bacteria, so that after each administration of antibiotics a higher level of oxygen radicals occurs in the organism. Oxygen radicals which are normally built in cellular energy production by means of reduced oxygen (ATPasis) in which also superoxide (O2) hydrogen peroxide (H2O") and the highly destructive hydrogen radicals are produced. While superoxide and hydrogen peroxide can be reduced enzymatically, this is not possible in hydrogen radicals, which directly attack proteins causing thereby genetic mutations. If in phases of oxidative stress O2 is released on the cell membrane as a by-product of cellular energy production, it can be either transformed by superoxide dismutase into H2O2 or reduce Fe3, which then as E2 can be transformed by H2O2 into aggressive hydrogen radicals and Fe3. In this manner under continuous administration of antibiotics against which bacteria defend themselves by the building of H2S a vicious cycle between the damage by oxygen radicals and the formation of new oxygen radicals can occur, which can cause severe dysfunctions such as the activation of herpes viruses inducing Kaposis'Sarcoma which is an AIDS-defining disease.

If the building of oxygen radicals (ROS), superoxide radicals (H2O2) and hydrogen radicals (OH) augments in the entire organism, bacteria activate their protection mechanisms against

oxidative materials (oxidative stress response), release specific antibacterial molecules (SOS DNA- stress response) and heat shock proteins, which leads to the development of resistance of certain bacteria and mycoplasma to certain antibiotics, which are passed over to their descendants or transmitted it by plasmids or introns to members of different bacterial strains. Under such conditions bacteria change their morphology, their metabolism and their products such as the proteins and liposacharides which are used for the building of the tissues in mucosa and the protecting film on it, that play a crucial role in the exchange of cellular signalling. In the course of this process strains can gain advantages in the competition with other strains, so that the composition of the gut flora (for example the ratio between aerobic and anaerobic bacteria) changes in certain fractions of the gut**. (E5)**

If after administration of antibiotics bacteria become cell wall deficient bacteria (CWDB) with filamentous forms they can not be recognised and eliminated anymore by immune cells sufficiently, in the course of inflammatory reactions, and then can settle in immune cells and other cells, where they cause over time a latent inflammation, first occurring in the mucosa of the oral cavities and the genital tract, and later triggering chronic infections after the contact with foreign non-residing bacteria, finally causing organ failure.
While some cell wall deficient bacteria (for example the mycobacterium tuberculosis) return to their original morphology at the decline of antibiotic pressure, others remain after their transformation in filamentous forms and make a fast emergence after activation of the immune cells they settle in. (For this reason in the autopsy of the first persons declared to be AIDS-cases, various cell wall deficient bacteria could be found in affected organs.) (E6)

After the administration of antibiotics to TB-patients, changes in the membrane of the bacterium induce a enhanced activation of CD-4-T-cells and of regulatory T-cells due to the activation of toll-like receptors. Besides this, mutations in the enzyme Dihyropholate-reductase and Thymidilate synthase by antibiotics induce resistance in M. tuberculosis to various antibiotics. The repeated administration of antibiotics also induces resistance in fungi and bacteria that play a mayor role in blood stream infections in which Salmonella enterica, Streptococcus pneumoniae, Staphylococcus aureaus und Streptococcus pneumoniae, Escheria coli and M. tuberculosis show resistance to various antibiotics, which are administrated frequently without a labour analysis detecting the causing agent. Genetic mutations in agents such Pneumocystis Jirovecii, Pneumocystis Carini und Streptocccus penumoniae induced by the administration of co-trimoxazoles, which block the building and the release of folic acids, cause emerging resistance, which makes the treatment of bacterial infections more and more difficult.

The treatment of the so-called co-infections to the HIV-infection, in particular tuberculosis, Malaria, hermits, Hepatitis C, Cytomegalo virus, Leishmania, Gonorrhoeae, Mycoplasma hominis and mycoplasma genitalium, Ureaplsma urealyticum, Chlamydia, Syphillis, Herpes causing viruses and parasites, such as Trichonomas vaginalis by means of specific antibiotics lead to a marked decline in the so called Hi-viral load and to a decrease in “viral replication” and “HIV-transmission. Persons with Chlamydia infections, doing a positive result in HIV-Testing show markedly lower CD-4 T-cell counts than those without this infection. As bacterial translocation persists in the receivers of ART persisting high levels of bacterial liposacharides in the plasma induce high levels of pro-inflammatory messengers and activated immune cells. After bacterial translocation the content of bacterial liposacherides (LPS) in the plasma, which may come from parts of cell-wall deficient bacteria (CWDB), built after the administration of antibiotics, is decisive about the activation of immune cells and the induction of the so-called HIV-transcription activator (HIV-1-Tat) and its products, which from a once set value on define a positive result in antibody HIV-tests. Bacterial

lipposacharides and DNA transmitted directly by blood and of blood preparations can induce the activation of the HIV-transcription attractor, leading to a positive result in HIV-antibody tests. Receivers of blood preparations produced from the blood of antibiotic treated donators in this way may induce a positive result in HIV-antibody tests.

When under ART-treatment bacteria in the gut are decimated day by day due to its bacteriostatic effects, in many patients a decrease in the activation of immune cells and of circulating bacterial parts is occurring, resulting in an increase of CD-4-T-cell counts. These values do not increase in so called ART non-responders and never increase to the values measured in sane persons in all receivers of ART. Even after the complete elimination of the so-called HIV-viruses by ART ("viral load 0") the amount of bacterial liposacharides and bacterial DNA remain elevated with the consequence that after an increase in the defence capacities and in the immune border in the gut for some time, a progression of AIDS-defining diseases takes place. The massive daily elimination of potentially pathogenic bacteria by ART is apparently faced by an ongoing damage of the commensally acting bacteria producing materials for the building of the gut mucosa and the protecting film on it. The suppression of bacterial growth by protease inhibitors in ART is faced by the suppression of the growth of proteases in cells of the gut mucosa, which need a high degree of cell division to maintain the inpermeability of the gut. Even at a total elimination of the so-called HIV-retroviruses by ART (viral load 0), which are the product of the activation of immune cells by bacterial lipposacharides after bacterial translocation, bacterial lipposacharides remain elevated. (**E7**) (**E8**)

Antibiotic induced damage to the mitochondria, which as organelles inside of cells produce the energy carrier molecule ATP, and antibiotic induced damage to the transportation of reduced oxygen in to cells due to blocking of the production of glutathione in the liver, diminishes the activity of cells in the intestinal mucosa and the bacteria producing ATP for the renewal of intestinal tissues. Diminished ATP-productions by genetically damaged mitochondria enhances the progression of AIDS-defining diseases.
The damage to mitochondria by protease-inhibitors and nucleoside analogue drugs in ART causes over time via enhanced production of oxygen radicals dysfunctions in the brain, the inner organs, the muscles and the arteries and the translocation of lipids from the face and the arms. These effects of ART have been termed.as "early aging", which has nothing to do with the administration of ART by the promoters of ART. (**E9**)

In regard of the central role of bacterial translocation for the development of AIDS-defining diseases representatives of the antiviral AIDS-treatment now take in consideration the administration of pro-biotics and pre-biotics as a principal AIDS treatment. Doing this they put in question for the first time the theory of Anthony Fauci from 1993, that all AIDS-defining disorders in various organs are caused directly by the Hi-retroviruses, which after lodging in lymphatic tissues for six weeks and a following latency of up to 10 years, during which they cause a continuous damage to CD-4-T-cells, induce the breakout of opportunistic infections and neoplasia, at which co-infections (in particular hepatitis, herpes simplex, herpes virus 6, cytomegalo virus, human lymphotropic virus type 1 and non defined mycoplasma may induce the the expression of HIV and the transition from the latency phase to the chronic phase, which in singular organs may be influenced by the presence of immune cells.

With this intentionally blurred theory, which became fast pointing the way for the entire AIDS-research and AIDS therapy, Dr. Fauci, who apparently knew a lot on the antibiotic induced dysfunctions in immune cells and other cells, that had been demonstrated in numerous trials since the 1970ies, faded out at once, that all the dysfunctions that he traced

back to a newly discovered retrovirus could also be the result of antibiotic resistant strains and antibiotic induced disorders in bacteria, immune cells and other cells. The fact that receivers of blood preparations, made from the blood of donators, being intravenous drug users, frequently receiving antibiotics, showed a positive result in HIV-antibody tests helped to establish the model of an independently pathogenic and deadly virus. The fact that a positive test result in receivers could be avoided by the processing of the plasma, which changes the form of bacterial liposacharides including bacterial DNA did not bring them to doubt their HIV theory.

With their model of a transmittable deadly virus, whose transmission could be avoided by respecting safer-sex rules and the use of preservatives and whose deadly effects could only be prevented by the lifelong intake of cell-killing bacteriostatic drugs, Fauci and his colleagues caused in millions of earth inhabitants a mortal agony in relation to sexuality and a billion selling business. With the tests, that detected the products of antibiotic induced disorders in immune cells and other cells and in bacteria, fungi, parasites and mycoplasma as the product of a newly discovered deadly virus they suppressed successfully for many years the knowledge on non-retroviral causes of AIDS defining disease and a corresponding research.

By means of world wide publicity campaigns for the prevention of the transmission of the newly discovered retrovirus via blood and semen by means of preservatives and the regard of safer-sex rules, they could repeat and deepen around annual World AIDS-Conferences their effective mass-psychology message on the transmittable, deadly retrovirus, while the epidemics of the diseases went on, which were meant to define the AIDS syndrome Despite many very expensive safer-sex campaigns in the last 30 years, on to this day not even the members of risk groups (such as men making sex with men) do know clearly, that oral contact with the anus is the most direct way for the transmission of sexually transmittable infections (STI) caused by bacteria, viruses and parasites such as Shigella, Hepatitis A, B und C, Human Papillomavirus (HPV), Herpes simplex Virus, Chlamydia, all of which can induce a positive result in HIV-testing. Even as medical doctors already in 1984 have stated the importance of this transmission path for infections in their prevention recommendations, this is on to this day no central point in the prevention message of AIDS-help groups. A fact that leads day by day to new infections inducing cellular “HIV”-signalling and new positive results in HIV-testing. (**E10**)

As consequence of the retrovirus AIDS-model from tests to detect the so-called Hi-viral load it cannot be taken today whether a rise in this “viral load” comes from an enhanced growth of antibiotic- and ART-resistant strains, from enhanced bacterial translocation into the intestinal mucosa, from damage to the gut flora and the gut mucosa, from co-infections or from enhanced oxidative stress due to ART and antibiotics.

That resistance to ART, can be treated successfully by changing the formula of ART, or the additional administration of specific antibiotics against particular strains, which brings down the “viral load” shows, that resistance of particular strains, occurring due to the bacteriostatic action of ART and additionally administrated antibiotics plays a crucial role in the development of the resistance of the “continuously transcribing” Hi-retrovirus to ART. The fact, that antibodies against active, strains (such as for example syphilis bacteria) can be detected in every ART-treated person by means of specific tests, is faded out by measuring the “HIV viral load”. The general public and those affected are kept in the believe, that the “Hi-virus” cannot be transmitted to others anymore if ART is taken in continuously and that consequently AIDS-defining diseases can be eliminated everywhere.

The great success of the ART treatment, declared in 1997 substantiated on a decrease in “viral load”, a decrease in “viral transmission” a diminished progression of AIDS defining diseases and lower mortality in patients treated was faced from the beginning on by ongoing infections and the sever adverse effects of ART such us the damage to the mitochondrial DNA, which is transmitted from the antiretroviral treated mother to her new-born child and induces preterm aging. ART receivers and the general public were kept then in the believe, that with a consequent intake of ART the virus cannot be transmitted to others anymore and that HIV-can eliminated world-wide, so that AIDS-defining disease diminish.

According to leading specialists for antiretroviral treatment, symptom free HIV-test-positive persons, in whom sexually transmittable infections have been treated successfully by means of specific antibiotics, so that they do not show enhanced titters of antibodies against the causing agents anymore, as also members of risk-groups, such as men having sex with men, should take ART preventively to stop the emergence of the human immune deficiency retrovirus. Not being able to transmit or receive the HI-retrovirus anymore, if they take in ART continuously, they should not be obliged anymore to respect the safer sex rules strictly. This new safer-sex-doctrine is now propagated by AIDS-help groups, who go on thereby playing a major role in the promotion for ART which allows its producers a further extension of the market for this expensive treatment that needs to be controlled by continuous labour analysis.

The fact that by means of pre-biotics and pro-biotics an equilibrated bacterial gut flora can be re-established in HIV-Test-positives and AIDS-patients, so that the building of the intestinal mucosa and the protecting gel on is activated again, with the effect that the bacterial translocation into the intestinal mucosa and the following inflammation in the lymphatic tissues of the gut (GALT) with the subsequent over activation of antigen-presenting dendrite cells (DC), which finally leads to a decline in CD-4 T-cells, Th17 T-cells, regulatory T-cells (Treg) and T-8 cells, can be halted, does not make ART specialists to put in question their HIV-theory and their treatment by means of cytostatic, nucleoside analogue substances, protease inhibitors and fusion inhibitors, which by its mitochondrial toxicity causes severe damage in various organs and finally the so-called resistance of the Hi-retrovirus, which can only be treated by new ART-formulas and the additional administration of specific antibiotics, both of which cause again new adverse effects and interactions with other preparations administrated to diminish these adverse effects.The fact that the so-called viral load can be substantially reduced and the disease progression can be halted by the administration of amino acids, vitamins, trace elements and unsaturated fatty acids does not motivate them to administrate these substances to HIV-test positives and AIDS-patients. **(E11)**

After they had traced back the products of antibiotic induced dysfunctions in persons, who had been treated continuously with antibiotics, distributed often in big packages for self-medication, and the occurrence of many antibiotic resistant strains they could see an effective therapy only in the administration of a continuous antibiotic treatment with nucleoside analogue substances such as AZT. The reduction of the toxic effects of these drugs, which could only be achieved by the reduction of its dose and the co-administration of protease inhibitors, resulted in the belief that an effective treatment had been found and that therefore there was no need for a research on non-viral causes of AIDS and on treatments for immune deficiencies occurring with genetically mutated, antibiotic resistant strains

On to this day neither measures to limit the uncontrolled administration of antibiotics in animal farming, where it produces multi-resistant strains transmitted to humans via meat and

by vegetables growing in their surroundings, nor measures for a limited administration of antibiotics to humans by means of a world wide registration of its administration or a ban on mitochondria damaging antibiotics have been brought under way by the WHO although antibiotic resistance could be detected nowadays world-wide by means of PCR tests.

The perfect, self-referring HIV=AIDS model, that by means of so-called HIV-tests, which detect the products of cellular signalling, occurring after the contact of cells with parts of oxidised, genetically mutated and often antibiotic resistant strains or with environmental toxins as products of a transmittable human immune deficiency retrovirus, does not allow any changes.

The uncontrolled administration of antibiotics and the lacking administration of a pro-biotic, anti-oxidative, mitochondria supporting treatment after antibiotic administration produces ay by day new patients carrying multi-resistant strains, later doing a positive result in HIV-tests, and therefore ready to receive the ART-treatment.

The emergence of antibiotic resistant strains of any kind and of antibiotic induced disorders puts in question nowadays medical care anywhere in the world. Singular persons, self-aid groups and medical institutions have to find ways out of this dead end, produced by the reckless administration of antibiotics in the last 40 years, whilst the billion selling business with antibiotics and “anti-retroviral treatments” goes on.

Study Group AIDS-therapy

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