Study Group AIDS therapy c/o Felix de Fries, Eglistr. 7 CH-8004 Zürich felix.defries@gmail.com

To people affected, their doctors and carers

To related institutions and to media

Zurich 1st June 2018

ref. **AIDS at HIV-viral load zero: The exhaustion of mucosa associated invariant T-cells by bacterial translocation after the intake of antibiotics or cART**

Dear Sir/Madam

As you may learn from various new studies carried out on PCR-testing, the antibodies against proteins which are considered to be products of the Hi-retroviruses, are expressed in mucosa at immune reactions to various bacterial, viral, fungible and parasitic agents (e.g. Mycoplasma Genitalum, Chlamydia trachomatis, Neisseria Gonorrhoea, Mycobacterium tuberculosis, Trichosomas vaginalis, Escheria Coli, Hepatitis A, B and C, Herpes 1 and 2.) This happens depending on the bacterial flora in the oral, genital and anal regions, the availability of the body's antigens and on the intake of medicaments and drugs, and in women, on the menstruation cycle, pregnancy and hormone-regulating contraceptives. These interactions have been demonstrated recently by animal trials on rhesus monkeys, identified as carriers of the so-called Simian Immunodeficiency Retrovirus (SIV), considered to act similar as the Human Immunodeficiency Retrovirus (HIV).

In the mucosa of the intestines, the sexual organs, the anal areas, the oral cavities and the lungs, where large numbers of bacterial, viral, fungal and parasitic agents, environmental toxins and micro-dust accumulate on mucosal epithelium, an ongoing activation of immune cells takes place, that leads to an increased release of oxygen and nitrogen radicals (ROS) normally produced in lower quantities in the mitochondria, which live as bacteria-like organelles with own DNA, inside of cells where they produce the energy-carrier molecule ATP used for various cellular functions. These interactions may lead to oxidation of proteins in the mitochondria which due to temporary shortage of nutrients, hypoxia or reperfusion, as well as after contact to products of bacteria, fungi, viruses and parasites, induce the mechanism of autophagy, that causes the dissolution of pathogens and of altered cellular materials for its reuse in the formation of newly built cells and for mechanisms of cell death and the maintenance of the oxidative equilibrium in cells.

After a while, continuously heightened levels of oxygen and nitrogen radicals (ROS) which cannot be bound by the anti-oxidative capacities of cells, cause via oxidation of proteins and lipids an increase in the permeability of the double membranes of mitochondria, resulting in a continuously heightened emission of oxygen and nitrogen radicals, leading to a lasting change in the PH-value and to oxidative damage to cellular elements such as the Golgi apparatus and the endoplasmic reticulum, with induction of the unfolded protein response (UPR), used for the defence in viral infections, and to changes in the cycle of autophagy that can no longer be fully completed, such as after the release of ATP, ROS and oxidised mitochondrial DNA the building of protein complexes, (inflammasomes) takes place, by which macrophages, enterocytes and dendrites dissolve intracellular viruses, fungi, bacteria and parasites. All of this may fascilate the living of viruses, bacteria (also in cell-wall deficient forms) and parasites inside of cells and their reproduction in newly formed cells and allows the emergence of pathogens, which are normally kept silent inside of cells, in the genital, anal, intestinal and pulmonary mucosa and the emergence of local infections there.

The building of inflammasomes such as NLRP3 or AIM2 takes place in two steps after the contact of parts of bacteria, viruses, fungi, parasites and its metabolites and of environmental toxins such as Asbestos or Aluminium-Chloride (from vaccine carrier substances) to pattern recognising receptors (PRR) such as toll-like receptors (TLR) , DAMPs (for bacteria) and PAMPS (for toxins), all of which induce after the contact to the endoplasmic reticulum the dissolution of these antigens. If the autophagy is inhibited and the functioning of mitochondria is disturbed, the building of inflammasomes is diminished so that the intracellular living of bacteria (also in cell-wall deficient forms) is facillated. Autophagy itself is regulated by inflammasomes after stimulation by bacterial products.

As demonstrated by numerous trials, antibiotics block the formation of glutathione molecules in the liver, which are used for the transportation of reduced oxygen into cells and effect thereby more oxygen-free energy production by anaerobic glycolysis. By direct damage to the unprotected mitochondrial DNA they cause DNA-mutations, which are transmitted to big numbers of its kind by division (fission) or by fusion, effecting thereby a mixture of intact and damaged mitochondria in cells, both of which finally heightens the formation of oxygen and nitrogen radicals in mitochondria, causing changes in the exchange of substances and signals with the cytosol and the cell nucleus, from where they continuously receive substances and signals. The building of inflammasomes, which is induced by the mitochondria at inflammatory reactions of any kind is changed under this conditions, with lasting effects on the cellular messengers for the activation of immune cells and for cell death. Already in the late 1980ies scientists proposed to block this oxidative stress by means of herbal antioxidants and the anti-oxidative acting sulphurous

compound N-azetyl-L-Cysteine, effective in the treatment of wasting in AIDS-patients.

The above mentioned actions also change the metabolism of bacteria, fungi, parasites and viruses living inside of cells, causing genetic mutations in their DNA, which are transmitted to other strains via plasmids, biofilms and transposomes, finally leading to a higher presence of genetically mutated, antibiotic resistant strains with changed metabolism, who themselves exert antibiotic activities against neighbouring strains, similar to those originally produced by the antibiotics themselves, inducing thereby lasting changes in the bacterial flora in various mucosa and its interactions with immune cells. The massive use of antibiotics in animal farming causes in this way the formation of antibiotic-resistant strains in nutrients, waters and grounds, which are transmittable to humans, who then may register a positive result in HIV testing.

Depending on the types of oxygen- and nitrogen- radicals, which are emitted in higher quantities after the intake of antibiotics and heavy metals or after wounding, the mechanism of programmed cell death, of autophagy induced cell death, or of autophagy without subsequent cell death is activated, during which bacteria, viruses, fungi and parasites as well as degenerated mitochondria and altered cellular parts are packed in closed membranes (called vesicles) to be broken down to their basic elements and be used for the building of new cells, or, if the autophagy cycle cannot be fully completed, are deposited on the cell membrane, from where they can trigger via the bloodstream reactions in distant cells to make these ready for the uptake of their ingredients; a mechanism which is used by intracellular living bacteria for their reproduction in newly built cells. Besides this action bacteria, parasites, viruses and fungi dispose of individual mechanisms to subvert destruction by autophagy, and use parts of its cycle for their entry into cells and their reproduction there.

The prolonged administration of antibiotics and the continuously repeated induction of autophagy may cause a shortage in substances, needed for a successful completion of the autophagy cycle. Such conditions, occurring together with higher oxidation of cellular parts and changes in signalling as also with cells, baring mixtures of intact and damaged mitochondria may lead to genomic instability and DNA mutations in the cell nucleus, which induce the formation of cancerous cells that receive their energy either from anaerobic glycolysis as also from aerobic glycolysis, coming from the surviving intact mitochondria.

Whereas many of the commonly administrated antibiotics induce autophagy, others, such as the widely used Macrolides, inhibit autophagy and thereby diminish the reproduction of intracellular bacteria in newly built cells, whilst at the same time allowing the growth of mycobacteria (such as mycobacterium Tuberculosis) and causing damage to the liver and kidneys. Efforts to treat intracellular infections that in time may

support the building of AIDS-defining degenerations such as Kaposi's sarcoma, by means of these antibiotics fail, as they block the autophagy needed for the breakdown of degenerated elements, whilst causing higher oxidation, which triggers cancerous degenerations. If immune reactions are directed continuously towards to the humoral antibody response (for example due to heavy metals in vaccine carrier substances or dental fillings), autophagy is inhibited by a continuous Th2 profile of messengers in T-cells. HIV test positive "non-progressors" who, without antiretroviral treatment, do not show AIDS-defining diseases for years, show a perfectly functioning autophagy.

As the ingredients of extracellular vesicles, which are made from parts of the cell, of mitochondria, bacteria or parasites, could only be partly characterised as agents of AIDS defining infections, their measurement in the plasma as "HIV-particles" by means of PCR-testing to determine the "HIV viral load" can only be understood as an intentional deceit. This because it is not known which parts of which bacteria, fungi, viruses or parasites define the "HIV viral load", as the producers of these tests declare this as part of their patent secret, and because the HIV could on to this day not be demonstrated as a retrovirus by centrifugation and budding, according to the established rules in retro-virology.

Lasting changes in the bacterial flora of the gut, due to repeated intake of antibiotics, herbicides insecticides and heavy metals in nutrients, as well as by nucleoside analogues, non-nucleoside analogues, protease inhibitors and fusion inhibitors in cART cause a diminished formation of bacterial short chain fatty acids (SCFA) needed for formation of the gut mucosa, with the result that parts of gut bacteria (LAS) can translocate to the lymphatic tissues of the gut (GALT), where they induce lasting inflammatory reactions which after time lead to lasting changes in T-4 and T-8 cells and in regulatory T-cells (Treg) that after time induce the immune deficiency syndrome AIDS.

The gut, where a multi-layered exchange between trillions of bacteria, viruses and fungi takes place, which is regulated by immune cells, becomes a breeding place for resistant strains from the environment, medical treatment and from the food chain. When the variety of resident bacteria and its common resistance to colonisation by foreign strains declines, strains from the environment may locate in the proximal gut, including the duodenum, esophagus and stomach, whilst the species of firmucites such as Lactobacillus Bifidobacterium are depleted and bacteroidetes, including pathogenic strains such as Escheria Coli, Camphilobacter Jeuni, Pseudonomas Aeguinosa or Desulfovibrio are enriched as also Prevotella in the oral cavities. Commensal bacteria in the intestinal lumen, the mucosal epithelium and lymphatic tissues of the lungs, the liver and the gut determine via metabolites and messengers the building of tissues and immune cells in the entire organism.

Whilst protease inhibitors by activating intestinal tissues promote bacterial translocation through the intestinal mucosa, and, depending on the used ingredients, heighten or diminish the building of inflammasomes, that play an important role in the defence activities in the intestines, nucleoside analogue and non-analogue drugs cause due to their bacteriostatic effects either the death of commensal bacteria, producing the materials for the intestinal mucosa and of bile acids, as also the death of pathogenic strains, which after passing the intestinal mucosa effect inflammation in the gut associated lymphatic tissues. Due to these opposite effects they may by diminishing inflammation temporarily heighten the rate of T-4 cells, whilst inducing after time “non-HIV/AIDS-related” disorders in the brain, the muscles, the bone marrow, the lungs and in the cardio-vascular system.

Protease inhibitors, that weaken the membrane of the cell nucleus, such as parts of nuclear DNA are released in the cytosol, induce thereby the building of special inflammasomes (AIM2), which then induce inflammatory reactions and thereby the blocking of autophagy in the cell. Under such conditions, occurring with changes in signalling and DNA-mutations in the cell nucleus, the building of cancerous cell takes place, which may get energy either from anaerobic glycolysis as also from OXPHOS by intact surviving mitochondria.

Antibiotics such as Vancomycin diminish already in low doses the number and quality of regulatory T-cells and the building of inflammasomes, whilst extending bacterial membrane and by changing the polarity the phagocytosis in macrophages. Beta Lactams exert via release of bacterial parts strong pro-inflammatory and by formation of multi resistant strains (MRSA) immunopathologic reactions.

The formation of regulatory T-cells after their release in the thymus is dependent on hormones and nutrients (vitamins A,D, B3 and B9) from dendrites in gut associated lymphatic tissues, who’s metabolism is directed by short-chain fatty acids produced by gut bacteria. They also provide the building of of lymphatic tissues and suppress pro-inflammatory messengers and the building of inflammasomes and regulate via conversion of neutral T-cells the building of tolerating antigen-presenting dendrites und support thereby the bacterial variety in the liver, diminishing damage to the liver and bacterial translocation through the intestinal mucosa. Antibiotics as also environmental toxins (Cadmium, Lead and Arsenic), pesticides, PCB, artificial sweeteners and emulators suppress via changes in the gut flora the building of regulatory T-Cells and of bile acids in the liver.

Invariant Killer T-cells (InkT) which represent 30-50% of all T-cells in the liver, get their organ-specific shape in the lungs, the small intestines and in the liver after activation by bacterial antigens from strains such as Heliobacter Pilori, Mycobacterium Tuberculosis and Escheria Coli, viruses as HPV, hepatitis B and C or HTLVI, as also from parasites such as Toxoplasma Gondi, Leishmania or Borrelia Rugdorfer and from fungal infestations inducing via IFNy and TNFa antibacterial activities in macrophages.

Mucosa associated invariant T-cells (MAIT) , which represent 1-10% of all circulating T-cells and the majority of T-8 cells, locating mainly in the mucosa of the intestines, the liver and the lungs, leave the thymus in quite immature form and are activated after the contact to metabolites of Escheria Coli, Salmonella, Enterobacteiracae, Staphilococcusand Candida Albicans as also by messengers (IL 12 and IL18) in viral infections to release pro-inflammatory cytokines (TNFy and TNFa and cytotoxic materials such as Perforin, Granolysin, Granzyme or Degranulate. Whilst circulating MAIT cells release IL17 after stimulation by messengers but not after contact to microbial metabolites. MAIT cells in the female genital tract release much more IL17 and IL22 and less TNFy and TNFa than freely circulating MAIT cells. MAIT cells which support macrophages in the control of intracellular strains and may induce the dissolution of infected cells, translocate at ongoing inflammatory reactions from the periphery to the affected organs. MAIT cells in the lungs can control infections already at early stages by the production of pro-inflammatory messengers and antimicrobial materials and orchestrate an optimal immune response. In the course of ongoing local infections they sustain a decline in their regulatory and cytotoxic abilities in single organs as after time a decline in the periphery of the organism and finally its exhaustion and dead. At ongoing viral infections such as Hepatitis B and C, MAIT cells can be activated by messengers from the response of toll-like receptors to viral RNA as also by bacterial translocation through the intestinal mucosa after administration of antibiotics, which induces their exhaustion despite effective treatment with or without Interferon. In the so-called HIV-infection, meaning after a positive result in HIV-testing, a fast decline in circulating MAIT cells in the periphery may be detected, which does not correlate with an increase in the “HI-viral load” or the decline of all circulating T-cells, whilst is not clear, how “Non-Progressors” can maintain their functionality.

In regard of recent studies on these topics, the question has to be raised, whether the HIV-infection, declared after a positive result in HIV-testing, is not simply the product of a decline in MAIT-cells and its functionality, after their exhaustion by an ongoing activation due to bacterial translocation induced by antibiotics and anti-retrovirals and their additional activation by bacterial, fungal, viral and parasitic infections. The ongoing translocation of bacterial products though the intestinal mucosa induces in any way their ongoing over-activation, exhaustion and decline in lymphatic tissues, the liver and the intestines. Its continuous activation correlates negatively with their frequency.

As it was demonstrated by various tests the bacterial translocation cannot be inhibited by the decline of the “viral load” below its detectability in cART treatment. The statement from promotors of cART that this would happen with an early or long lasting administration was disapproved by recent tests. Even as the bacterial translocation can be diminished by the administration of vitamin D, pro-biotics, antioxidants and sulphur

compounds, and the building of bone marrow cells and the activity of T-cells can be improved by it, the severe adverse effects of cART cannot be set off it.

As it has been demonstrated by animal trials with monkeys, showing a negative result in the Simian Immune Deficiency Retrovirus Antibody Test, the translocation of bacterial particles (LAS) into lymphatic tissues of the gut mucosa can be induced without the presence of the alleged SIV-retroviruses, by means of substances such as dextran sulfate sodium and be inhibited by substances such as Sevelamer, that bind bacterial liposaccharides. As the ingredients of extracellular vesicles, which are made from parts of the cell, of mitochondria, bacteria or parasites, could only be partly characterised as agents of AIDS defining infections, their measurement in the plasma as “HIV-particles” by means of PCR-testing to determine the “HIV viral load” can only be understood as an intentional deceit. This because it is not known which parts of which bacteria, fungi, viruses or parasites define the “HIV viral load”, as the producers of these tests declare this as part of their patent secret, and because the HIV could on to this day not be demonstrated as a retrovirus by centrifugation and budding, according to the established rules in retro-virology.

Vitamin D, which is produced under the skin by UV-radiation, or by the intake of nutrients, such as mackerel, sardines or shitake mushrooms, and is synthesized in the liver and kidneys, and the following production of Vitamin D receptors (VDR) in B-cells, T-cells, regulatory T-cells (Treg) and in cells of the gut, pancreas, prostate and peripheral tissues, is decisive for the formation and activity of T-cells in the thymus and for dendrites, macrophages and monocytes and their reactions after contact to antigens. Vitamin D activated by its receptors supports the production of antibacterial peptides (Cathelicidins) as well as the induction of autophagy and the destruction of mycobacteria such as the mycobacterium tuberculosis. Shortage in vitamin D and vitamin D-receptors, as it may occur at repeated intake of antibiotics, anti-retrovirals, hypotension lowering drugs, inflammation inhibitors, steroid preparations, anti-epileptics, contraceptives, anti-depressants, sedatives, insecticides and aflatoxins, induces a lessening of tolerance to antigens in immune cells of the gut, causing an increased production of TH17 cells and inducing thereby autoimmune reactions and an ongoing decline of T-4 cells. Isonazin and Rifampine, which are used for the treatment of tuberculosis, diminish the level of active Vitamin D and thereby, after some time, the antibacterial activities of immune cells and the induction of autophagy and loose in this way their bacteriostatic effects against the mycobacterium tuberculosis.

Nitric oxide gases (NO) are used in the organism for defence against the agents of AIDS defining diseases. At low quantities they support the formation of mitochondria and its functioning, the cohesion of mucosa, the inhibition of inflammatory signalling from neighbouring cells, the containment of bacteria, viruses and parasites inside of cells and their

breakdown by phagocytosis, as also pro-biotic acting bacteria in their attack on pathogenic strains, or activated immune cells in their attack on cells containing bacteria, viruses and parasites, whereas at high quantities they cause overextending immune reactions. The synthase of various types of NO-gases (iNOS, nNOS, bNOS and eNOS) in single cell systems and in bacteria and their emission under particular conditions (e.g. repeated antigen contact by polluted drinking water) is inhibited by antibiotics and bacteriostatic substances in cART, as well as by the uptake of lead, arsenic and aluminium from the environment and of mercury from vaccine carrier substances and dental fillings, all of which block the synthase of tetrahydrobiopterin used for NO-synthase. Due to this, the antibiotic treatment of AIDS defining diseases, such as tuberculosis, becomes more difficult in areas with high levels of environmental heavy metals.

As numerous epidemiologic studies carried out in the last 20 years, reveal, bacterial, fungal and parasitic infections, such as syphilis, tuberculosis and chlamydia, whose agents show nowadays resistance to antibiotics of various substance classes, and viral infections, such as hepatitis A, B and C, HPV and Herpes I and II, whose agents show resistance to various chemotherapeutics (including nucleoside analogue drugs), are pacemakers for the seroconversion to the test-result “HIV-positive” after which they are considered to be co-infections of the HIV-viruses, meant to be the cause of their severe course. According to these definitions attempts are made today to prevent this seroconversion by means of specific antibiotics and to treat infections occurring despite cART treatment (e.g. in hepatitis C) by the additional administration of specific antibiotics, which may cause high oxidative stress and consequently damage to the liver and kidneys.

By means of bacteriostatic drugs (protease inhibitors, fusion inhibitors, nucleoside analogues and non-nucleoside analogues) by which these infections are suppressed in the pre- and post-exposure treatment (PreP and PeP), the transmission of products of these infections, termed as HIV particles, should be inhibited at unprotected sex, whilst the transmission of the pathogenic agents itself, many of which show resistance to various sorts of antibiotics, cannot be inhibited by it, such as they have to be treated later by means of specific antibiotics. As Prep and PEP show the same severe adverse effects and growing resistance to the so called HIV as cART, it is improbable that the seroconversion to the test result “HIV-positive” can be averted by it over a longer period. That anyone who takes cART consequently cannot transmit HIV anymore and anyone, consequently taking PreP cannot take it up anymore at unprotected sex, as the promotors of cART and PreP state, cannot stop the transmission of infections that over time may induce a positive result in HIV-testing. High daily doses of cART respectively of PreP are necessary at unprotected sex to prevent the transmission of one pathogenic strain that may induce a positive result in HIV-testing. Genetically mutated, antibiotic resistant strains are transmitted nowadays in short time due to a high

intercontinental mobility. The continuous, preventive intake of cART in the form of PreP and PeP, as it was proposed by the WHO for African and Asian countries and for risk groups in Europe and the USA, cannot represent a substantial strategy against the emmergence of AIDS-defining diseases, due to their damaging side effects, the price of the pills and the necessary close laboratory monitoring, overrunning the annual health care budgets in the states of the developing world.

Politicians have to decide, whether PreP and PeP has to be refunded by the public health system to all HIV-Test-Positive persons and their friends doing unprotected sex. These can now buy cheaper generics of PreP if they get a prescription from an MD, even as they still do not know which Antibiotic resistant strains they are carrying and may transmit to others. If the public health systems refunds Prep and PeP, this would represent a new billion selling business for Big Pharma and the end of safer-sex for the prevention of the transmission of sexually transmittable infection. HIV-test-positive persons and their friends would again become guinea pigs in the global human trial for effective treatments for infections by antibiotic-resistant strains.

Due to its bacteriocidal, anti-viral, anti-parasitic and anti-fungible effects, cART, consisting of nucleoside analogues, non-nucleoside analogues, protease inhibitors and fusion inhibitors, reduces the number of various pathogens and suppresses thereby the emergence of AIDS-defining infections, as each of its ingredients does this to a certain group of pathogenic agents, allowing thereby a temporary increase of the T-4 cell counts, whilst causing resistance after longer time, which emerges fast in countries of the developing world.

Everyone who takes in cART continuously loses the ability to defend pathogenic strains by means of immunereactions, as this task is taken-over continuously by cART. Once interrupted new resistance and related immune reactions occur, that may cause severe complications. Therefore once started, cART has to be taken-in life-long, and could only be ended by specialists using immunity restitutive treatments, including transfusions. The promotors of cART know this already since some years and they also know, which antibiotics and formulas of cART actually work at what AIDS-defining infections in which groups of HIV-test positive persons in what areas of the world, and which of it don’t work anymore due to resistance developing.

By means of the administration of cART in pregnancy, birth by caesarean delivery, the avoidance of breastfeeding and the administration of cART to newly born babies, the transmission of gut bacteria from the mother to her child should be prevented, to achieve a negative test result in the newly born baby. As demonstrated by targeted tests, factors such as the bacterial flora of the mother and her intake of antibiotics before and during pregnancy are decisive for the result of HIV-testing in the newly born baby. All antiviral chemotherapies against HIV and Hepatitis C as well as

antibiotics, hyperlipidaemia drugs, Antiarrhytmics, Antimalarials, Antifungizides, Non-Steroidal Anti-inflammatory Drugs (NSAIDs) as well as alcohol and tobacco consumption cause damage to mitochondria in the pregnant mother and her new-born.

The blocking of mitochondrial functioning by cART damages the formation of the energy carrying molecule (ATP) in immune cells and causes thereby immune deficiencies occurring in infections of any kind, the translocation of fatty tissues and damage to the liver, the kidneys, the intestinal mucosa, the brain, the muscles, the bone density and the cardio-vascular system, all of which are termed as “non-HIV/AIDS related progeria”. In many patients, the intake of cART causes various types of non AIDS-defining cancer.

Neither the weakening of the mitochondria by antibiotics, toxins and medicaments, which damage its DNA and the transportation of oxygen into cells, and the following disturbance of autophagy and of inflammasomes, which supports the intracellular living of bacteria, fungi and parasites, nor the building of antibiotic-resistant bacteria and parasites, inducing changes in the gut flora and consequently in the gut mucosa, or the inhibition of the synthase of vitamin D, vitamin D-receptors and of nitric oxide in immune cells by antibiotics, as also not malnutrition in countries of the developing world, which weakens all immune reactions, should be the cause for the severe course of the AIDS defining infections and of the immune deficiency syndrome, but an infectiously transmittable “Human Immune Deficiency Retrovirus” who’s products detected by HIV-Antibody tests, induce under the above mentioned conditions the test result “HIV positive” at more than 60 AIDS defining infections and disease conditions. Why this retrovirus should not be an endogenous retrovirus, such as many others that represent 8 % of the human genome, which are activated under particular environmental conditions, couldn’t be explained by its promotors, who state that the HIV is an infectiously transmittable retrovirus, which came from monkeys into the human genome.

30 years after the postulation of an infectiously transmittable “Human immune deficiency retrovirus” by Françoise Barré Sinoussi, Luc Montagnier and Jean-Claude Cherman, the development of a test, meant to detect products of this retrovirus, by Robert Gallo, and a billion costing state financed AIDS research, it is still not clear, how this retrovirus should cause a lasting decline in T-4 and T-8 cells and the severe course of these infections. Yet it is quite clear today, that the signals and entities attributed to this retrovirus are not the cause of the severe course of AIDS defining diseases and of the acquired immune deficiency syndrome and that the so called Hi-Virus-particles, detected in plasma by means of PCR testing are more likely products of a disturbed autophagy, which occurs due to damaged mitochondrial function, malnutrition, environmental pollution, antibiotics and chemotherapy, and of a decline of mucosa associated invariant T-cells due to its over-activation by continuous bacterial translocation after intake of antibiotics or cART.

Neither the fact that patients with AIDS-defining diseases have done a positive result in HIV testing before getting ill, nor the presence of the increasing and decreasing “viral load” occurring due to the suppression of the AIDS defining infections by cART do change anything about these facts. The statement of the “HIV discoverers”, that the HIV could only be transmitted via blood or semen, has averted in the last thirty years an effective prevention of of droplet infections by transmission of antibiotic resistant agents, which can be transmitted in many ways and may result nowadays in severe, deadly infections, particularly in countries of the developing world, dealing with malnutrition, polluted drinking water and poor living conditions.

Up to this day HIV-test-positive persons and patients with AIDS-defining diseases, do not receive targeted treatment for a good bacterial flora in the gut, mitochondrial functioning or synthase of Vitamin D or NO. They also do not get anti-oxidative treatment against the adverse effects of antibiotics and cART, even as such additional treatment has shown a substantial decline in complications and fatality in clinical tests carried out 20 years ago. An effective treatment for AIDS-defining diseases is still only seen in chemotherapy. Measures to prevent antibiotic resistance, by means of its controlled and targeted administration and the ban of broad spectrum antibiotics in animal farming, or measures for the improvement of drinking water and the ban of insecticides and herbicides (such as Glyphosate) which heighten the release of heavy metals into the environment, have only been taken in beginnings.

That it became possible by means of HIV-testing to fade out all these factors for the emergence of endemic infections, declaring the products of infections by antibiotic resistant strains to be products of a transmittable retrovirus, responsible for the severe, deadly course of these infections, represents an unique case of crowd psychology manipulation. Many kinds of professionals (medical doctors, scientists, social pedagogues, psychologists, publicity specialists, journalists, and politicians) have played their role in the distribution of this double meaning model of a deadly, transmittable virus occurring at sexually transmittable infections, which has become a good business for many of them as shareholders as also for big pharma. The fact that over the last 30 years medical doctors have treated the HIV-test positives by means of harmful substances apparently without any therapeutic alternatives, whilst losing sight of a holistic understanding of these diseases and their treatment, will become a notation in the history of medicine. Many of them show no interest in the real causes of AIDS-defining diseases as this could mean responsibility and compensation for damages.

What the presence of the so-called HIV-antibodies and HIV-particles really means and how a non-damaging therapy for AIDS-defining diseases can be achieved has to become again the topic of a broad public discussion in regard of the now available research data. What mechanisms take place in the formation of AIDS-defining diseases and which therapies can efficiently

change it can be verified by means of new imaging processes of microscopy and quantum physics research at the x-ray laser- tunnel (http://www.xfel.eu/).

Study Group AIDS-therapy

Felix de Fries

AIDS at viral load zero: Studies and Links
www.ummafrapp.de/skandal/felix/zero/studies_and_links.pdf

Voir le SIDS autrement

Etienne de Harven Interview Video (2012)

https://www.dailymotion.com/video/xptfgh

Role of anaerobic flora in the translocation of aerobic and facultatively anaerobic intestinal bacteria.

Carol L. Wells et all, (1987)

Role of anaerobic flora in the translocation of aerobic and facultatively anaerobic intestinal bacteria.

Role of cysteine and glutathione in HIV infection and other diseases associated with muscle wasting and immunological dysfunction

Wulf Dröege und Eggbert Holm (1997)

Role of cysteine and glutathione in HIV infection and other diseases associated with muscle wasting and immunological dysfunction.

Mitochondrial toxicity of antiviral drugs

Lukinaos Dalakas (1995)

Mitochondrial toxicity of antiviral drugs

Gut barrier structure, mucosal immunity and intestinal microbiota in the pathogenesis and treatment of HIV infection

Tincati, Doucek and Marchetti (2016)

[HTML] Gut barrier structure, mucosal immunity and intestinal microbiota in the pathogenesis and treatment of HIV infection

Activation, exhaustion, and persistent decline of the antimicrobial MR1-restricted MAIT-cell population in chronic HIV-1 infection

Leansyah, Genesh Quigley et al.2013

Activation, exhaustion, and persistent decline of the antimicrobial MR1-restricted MAIT-cell population in chronic HIV-1 infection

Felix de Fries

Therapeutic Recommendations

http://www.ummafrapp.de/skandal/felix/therapeutic_recommendations.pdf

The „HIV-positive“ inducing mycobacteria

http://www.ummafrapp.de/skandal/felix/The_HIV-inducing_Mycobacteria.pdf

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

http://www.ummafrapp.de/skandal/felix/pro/AIDBSe.pdf

AIIDS: Antibiotic Induced Immune Deficiency Syndrome

http://www.ummafrapp.de/skandal/felix/antibiotics/aii-e.pdf

Gut flora, intestinal mucosa, antibiotics and AIDS
http://www.ummafrapp.de/skandal/felix/Darmflora/Gut_flora%20_intestinal_mucosa_antibiotics_and_AIDS.pdf

Dr. Gallo’s proof of Dr. Montagnier’s HIV retrovirus

http://www.ummafrapp.de/skandal/felix/Dr_Gallos_Proof.pdf

MD Heinrich Kremer:

The Lifesaving Knowledge of Healing

The Lifesaving Knowledge on Healing pdf 115 k

The Secret of Cancer: „Short Circuit“ in the Photon Switch

The Secret of Cancer: "Short-Circuit" in the Photon Switch

The Concept of Cellsymbiosis Therapy

The Concept of Cellsymbiosis Therapy pdf 24.5 k

Did Dr. Gallo and his colleagues manipulate the AIDS-test to order?

http://www.ummafrapp.de/skandal/test/did_gallo_manipulate.html

Acquired Iatrogenic Death Syndrome

http://www.ummafrapp.de/skandal/Acquired%20Iatrogenic%20Death%20Syndrome,%20H.%20Kremer%201996.pdf