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To people affected,
their doctors and caretakes
To related institutions and to media

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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 on PCR-testing, antibodies against proteins considered to be products of the Hi-retroviruses are expressed in mucosa in 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 antibodies 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 .particulate matter 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 an own DNA inside of cells where they produce the energy-carrier molecule ATP used for all 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.

In this process, continuously heightened levels of oxygen and nitrogen radicals (ROS), which cannot be bound by the antioxidative capacities of cells, cause via oxidation of proteins and lipids an increase in the permeability of the double membrane 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 of viral infections, and to changes in the cycle of autophagy, that cannot be fully completed anymore, and after the release of ATP, ROS and oxidised mitochondrial DNA in the cytosol to the building of protein complexes, called inflammasomes, by which macrophages, enterocytes and dendrites dissolve parts of intracellular viruses, fungi, bacteria and parasites. All of this may facilitate 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, of the genital, anal, intestinal and pulmonary mucosa and the emergence of local inflammation there. Protease inhibitors, nucleoside analogue drugs and non-nucleoside analogue transcriptase inhibitors, cancer medicaments and analgesics (such as Diclophenac) may heighten the endoplasmic reticulum stress in these cells and the accumulation of lipids, inflammatory reactions, cell death and cytosis and disturb in this way the protein synthase in these cells.

The building of inflammasomes such as NLRP3 or AIM2 takes place in two steps after the contact to parts of bacteria, viruses, fungi, parasites and their metabolites and to 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 facilitated. Autophagy itself is regulated by inflammasomes after stimulation by bacterial products. An early damage to the mitochondria leads in AIDS-patients to a

big number of dead lymphocytes in lymphatic tissues, which show mitochondrial swelling, typical for necrosis, representing a particular form of cell death.

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 a higher oxygen-free energy production by anaerobic glycolysis. By direct damage to the weakly protected mitochondrial DNA they cause mitochondrial 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 these 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 causing 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 antibiotics, inducing thereby lasting changes in the bacterial flora in various mucosa and in 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 inflammasomes and use parts of this cycle for their entry into cells and their reproduction there. This also goes for DNA-viruses, such as the Human Papilloma virus 16 (HPV16), which plays a role in the building of cervical cancer, ano-genital tumours, head and neck-cancers and skin cancer, as also for the Epstein Barr virus (EBV), which plays a role in the building of tumours such as Burkitt Lymphoma, or the HTLV-1 virus, which induces T-cell leukaemia, the Kaposi Sarcoma Herpes virus (KSHV) inducing Kaposi's Sarcoma, as also Hepatitis B and C viruses, all of which may settle more easily under these conditions in tissues and later inhibit autophagy for their reproduction which finally causes the emergence and the progression of the related diseases. The transmission of these viruses can be inhibited by avoidance of small injuries, their activation and pathogenic action party by means of vaccines or specific antibiotics.

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 in 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 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 dieases such as Kaposi's sarcoma, by means of such antibiotics fail, as they block the autophagy needed for the breakdown of degenerated elements, whilst causing a 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 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 analogue drugs, non-nucleoside analogues, protease inhibitors and fusion inhibitors in cART cause a decline in the 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 may induce dysfunction defining the immune deficiency syndrome AIDS.

In this situation the gut, where a multi-layered exchange between trillions of bacteria, viruses and fungi takes place, which is regulated by immune cells, may become 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 environmental origin may locate in the proximal gut, including the duodenum, oesophagus and stomach, whilst the species of firmicutes such as Lactobacillus Bifidobacterium are depleted and Bacteroidetes, including pathogenic strains such as Escheria Coli, Camphilobacter Jeuni, Pseudomonas Aeruginosa 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 its 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 materials for the intestinal mucosa and of bile acids, as also the death of pathogenic strains, which after passing the intestinal mucosa may cause inflammation in 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, so that 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 receive energy either from anaerobic glycolysis as also from OXPHOS produced 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 changing the polarity of phagocytosis in macrophages. Beta Lactams exert via release of bacterial parts strong proinflammatory and by formation of multi resistant strains (MRSA) immunopathologic reactions. The composition of the gut flora is decisive in HIV-test-positives for their body's defences to infections, respectively for the building of an immune deficiency. Multi-resistant strains are often accumulated at international travelling.

The formation of regulatory T-cells after their release in the thymus is dependent on hormones and nutrients (vitamins A, D, B3 and B9) released 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 lymphatic tissues and suppress inflammatory messengers, the building of inflammasomes and regulate via conversion of neutral T-cells the building of tolerating antigen-presenting dendrites and support thereby the bacterial variety in the liver, diminishing thereby 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 bacteria 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 Bugdorfer 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 the 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, Enterobacteriaceae, Staphylococcus and Candida Albicans, as also by messengers (IL 12 and IL18) in viral infections to release 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 in men. 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 also 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 may be activated either 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 it 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 so-called HIV-infection, declared after a positive result in HIV-testing, is not simply the product of a decline in MAIT-cells and their functionality in the defence of bacterial, viral, fungible and parasitic infections, and whether the inflammatory "HIV entities" measured under such conditions in various organs, which can be mimicked today easily by laboratory conditions in cell-cultures and animal trials, are nothing, but cellular products of this decline. This would explain, why after their exhaustion by an ongoing activation due to bacterial translocation, induced by antibiotics and chemotherapy, and their additional activation by bacterial, fungal, viral and parasitic infections, the test result "HIV-positive" occurs. The ongoing translocation of bacterial products through 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 have been 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 by 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 sulphate 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 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 Bcells, 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, antiretrovirals, 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 qut, causing an increased production of TH17 cells and 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 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. 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 its 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 may cause overextending immune reactions. Repeated infections and inflammation due to local wounding may cause a lasting overproduction of NO in cells, which over time uses up the pool of anti-oxidative thiols in cells, inducing thereby a lasting switch in messengers (Th-1-Th-2 switch) and finally the blocking of NO-synthase in cells. A process which can also be induced by the inhalation of nitrites termed "Poppers", used for the relaxation of the smooth muscle, facilitating the opening of the anal sphincter for anal intercourse, causing vasodilation of the penile blood vessels, resulting in stronger erection, and brining an euphoric high by increase of the intracranial pressure, a feeling of warmth, a reduced pain

threshold in the receptive partner, and an enhancement of sexual pleasure through prolonged orgasm leading to a reduction of social an sexual inhibitions.

The synthase of various types of NO-gases (iNOS, nNOS, bNOS and eNOS) in certain cell systems and in bacteria and its 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 today 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 "HIVpositive", after which they are considered to be co-infections of the HIV-viruses, meant to be the cause of its 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 higher 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 postexposure 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 by means of specific antibiotics additionally. The fact, that PreP-medicaments such as Tenofovir-Emtricitabine, which inhibit infections by Syphilis, Chlamydia Trachomatis and Gonorrhoea being, as admitted now, the big drivers of the seroconversion to the HIV-positive Test result in sexually active persons, shows once more that bacterial, viral, parasitic and fungal infections are the cause of all symptoms attributed to the "Human Immune Deficiency Retrovirus" - as the promotors of the HIV-AIDS-theory

apparently know since many years. After declaring persons with Syphilis, Gonorrhoea and Chlamydia Trachomatis infections by means of HIV-testing to be carriers of an infectiously transmittable, deadly acting Hi-retrovirus in the last 30 years and treating them by means of toxic substances such as AZT, they now want to stop the transmission of the alleged-Hi-retrovirus by means of substances, which inhibit these three infections. The now stated success of PreP, which occurs due to frequent laboratory testing for detection and treatment of sexually transmittable infections, should now be enhanced by safer-sex measures, such as condom use, not applied anymore after implementation of PreP by many people. All of this should now reinsure the belief in the HIV-AIDS-model and in the deadly acting retrovirus, which allows a billion selling business to the AIDS-medicine and big pharma.

As PreP and PEP show the same severe adverse effects and growing resistance to the so called HIV as cART does, it is improbable that the seroconversion to the test result "HIVpositive" 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. Due to its influence on the gut flora and the gut mucosa and the following bacterial translocation into the gut associated lymphatic tissues, that after time may induces immune dysfunctions, HIV-test-negative partners of HIV-test-positives, who are able to defend continuously the test positive inducing infections of their partners, might lose this ability, such as they would sustain themselves the sero-conversion to the test-result "HIVpositive".

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 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 emergence of AIDS-defining diseases, due to its damaging effects on mitochondria, causing lasting damage to the kidneys, the liver, the bones and the neuronal system, the price of the pills and the necessary close laboratory monitoring, overrunning the annual health care budgets of states in the developing world. Whether chemical protease inhibitors causing

lasting damage to cells can now be replaced by natural protease inhibitors from sea algae, is still not know.

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. They 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, even as this could be measured nowadays easily by means of PCR-testing. If the public health systems refund 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. The denial of the role of nitrite inhalations for the emergence of the most frequent AIDSdefining diseases in homosexual men (PCP, Lymphoma and HPV, followed by Kaposi 's sarcoma by AIDS-specialists at the university clinics of big cities in the last 30 years, who treated their Poppers-consuming patients, showing repeated venereal infections by means of TMPSMX and other fast working antibiotics, has founded a remaining complicity with his clientele, who consequently reduce homosexual life often to fast sex in the darkroom of a gay club. Despite million-costing prevention campaigns they are apparently not able to inform the affected gay community, that Poppers acts immunosuppressive and that anilingus ("rimming") is a direct way for the transmission of these infections. Not regarding the Safer-Sex rules after poppers consummation still causes repeated infections, followed by a positive result in HIV-testing and finally by AIDSdefining diseases. Almost no research has been carried out to this day on the effects of the simultaneous intake of Prep and Poppers respectively of cART and Poppers even as this happens many folds every day.

Due to its bacteriostatic, anti-viral, anti-parasitic and antifungible 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 immune reactions, as this task is taken over continuously by cART. Once interrupted new resistance and related immune reactions occur immediately, 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 what formulas of cART actually work at what AIDS-defining infections in which groups of HIV-test positive persons, and which of it don't work anymore due to resistance developing so that they have to be treated by continuously changing to new formulas. The irreversible side-effects of the combi-therapies of the first and second generation (DDI, DDC, 3TC, d4T etc.), now occurring in persons treated -from the translocation of fatty tissues over polyneuropathy, brain impairment, impairment, kidney impairment, cardio-vascular diseases on to the decline in bone density- are talked down by medical doctors, who once again state, that new combi-therapies would not show such adverse side effects anymore.

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, Antiarrhythmics, 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, 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 which are activated under particular environmental conditions, such as many others that represent 8 % of the human genome, couldn't be explained by its promotors, who state that the HIV is an infectiously ransmittable 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 of 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 all 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.

That the signals and cellular proteins, which are measured as products of Hi-retroviruses, occur at various kinds of infections, when autophagy and inflammasomes are blocked due to damage to mitochondria by antibiotics, environmental toxins and medicaments does not make them revise their HIV-AIDS-hypothesis or change their AIDS-therapy. That infections by resistant strains can only be ended if the inhibition of autophagy and inflammasomes can be stopped by supporting the mitochondrial functioning by means of detoxification of mitochondria-toxic substances and the administration of antioxidants, trace elements and amino-acids, does not make them doubt their

antiretroviral treatment concept, to block inflammatory acting inflammation by products by means of synthesized protease inhibitors, nucleoside-analogue and non-nucleoside analogue drugs and integrase inhibitors, all of which cause damage to the mitochondria and in this way irreversible dysfunction in various organs, which are passed on from mothers to their children. How should such treatments, which are nowadays administrated all over the world, diminish AIDS-defining diseases? How should the substances of the so-called Preexposure prophylaxis and the post-exposure prophylaxis—treatment prevent infections in persons with blocked mitochondrial functioning?

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 droplet infections of antibiotic resistant agents, which can be transmitted in many ways and may result 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 to support 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 broadspectrum 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 a 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 entire AIDS-research is still directed by the unknown hand of the market focussing on patentable preparations that promise high benefits, administrated in public hospitals that get a 30 percent margin from each of it, charged to the health insurance.

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.

In regard of the fact that the Hi-Retrovirus has not been isolated on to this day according to the established rules and that persons with various types of infections or disease conditions (including receivers of blood preparations) show a positive result in HIV-testing, we demand the application of specific testing to find the agents of these infections, and its treatment by means of specific antibiotics. Furthermore, we demand, that everyone treated with antibiotics receives at the end of the administration a labour-controlled treatment to reestablish the intestinal flora and mitochondrial functioning. Furthermore we ask for the free choice of treatment and of doctors for anyone treated including the cART non responders. The forced treatment of mothers and their children and of internees and dependants by cART, represents a violation of their human rights and has to be stopped immediately due to its various severe adverse effects.

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 public discussion. What mechanisms take place in the building of AIDS-defining diseases and which therapies can efficiently inhibit it can be verified by means of the new imaging processes of kryo electron microscopy and quantum physics research at the x-ray laser- tunnel (http://www.xfel.eu/). Then we will see, what is left of the so-called Hi-retrovirus, which has been postulated by Françoise Barré-Sinoussi, Luc Montagnier, Jean Claude Cherman and others in 1984 first as a "lymphadenopathy associated virus" in patients with "unexplainable lymphadenopathy", in homosexual

AIDS-patients with (non- defined) opportunistic infections, in promiscuous male homosexuals having had (non- defined) venereal infections, treated by (non-defined) antibiotics, but without visible (meaning intravenous) drug use and without considering poppers inhalations as a possible cause of lymphadenopathy.

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

Attachments:

DETECTION OF IGG ANTIBODIES TO LYMPHADENOPATHY-ASSOCIATED VIRUS IN PATIENTS WITH AIDS OR LYMPHADENOPATHY SYNDROME Françoise Barrée Sinoussi et al. (1984)

https://docs.ufpr.br/~microgeral/arquivos/
2013FUNDAMENTOSBarreSinoussi.pdf

Impact of the 1987 Revision of the Case Definition of Acquired Immune Deficiency Syndrome in the United States

https://journals.lww.com/jaids/Abstract/1990/01000/ Impact of the 1987 Revision of the Case Definition.12.aspx

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

Death of Bystander Cells by a Novel Pathway Involving Early Mitochondrial Damage in Human Immunodeficiency Virus-Related Lymphadenopathy

Mauricio Carbonari et al.

Death of Bystander Cells by a Novel Pathway Involving Early
Mitochondrial Damage in Human Immunodeficiency Virus-Related
Lymphadenopathy

Gut barrier structure, mucosal immunity and intestinal microbiota in the pathogenesis and treatment of HIV infection Tincati, Doucek and Marchetti (2016)

[HTML] <u>Gut barrier structure</u>, <u>mucosal immunity and intestinal</u> 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

ROS, mitochondria and the regulation of autophagy ROS, mitochondria and the regulation of autophagy

Mitochondrial DNA in the regulation of innate immune responses
[HTML] Mitochondrial DNA in the regulation of innate immune responses

Role of endoplasmic reticulum stress in drug-induced toxicity

Role of endoplasmic reticulum stress in drug-induced toxicity

Human Papilloma Virus and Autophagy

[HTML] Human Papilloma Virus and Autophagy

Off-target effects of drugs that disrupt human mitochondrial DNA maintenance

[HTML] Off-target effects of drugs that disrupt human mitochondrial DNA maintenance

A double-edged sword: does highly active antiretroviral therapy contribute to syphilis incidence by impairing immunity to Treponema pallidum?

[HTML] A double-edged sword: does highly active antiretroviral therapy contribute to syphilis incidence by impairing immunity to Treponema pallidum?

Human Immuno-Deficiency Virus Drug Resistance, Nuclesoide Reverse Transcriptase Inhibitors, Non-nuclesoide Reverse Transcriptase Inhibitors And ...

[HTML] <u>Human Immuno-Deficiency Virus Drug Resistance,</u>
<u>Nuclesoide Reverse Transcriptase Inhibitors, Non-nuclesoide</u>
<u>Reverse Transcriptase Inhibitors And ...</u>

Mitochondrial DNA mutations in ageing and disease: implications for HIV? [HTML] Mitochondrial DNA mutations in ageing and disease: implications for HIV?

HIV Pre-Exposure Prophylaxis: Are we "PrEPing" for a new epidemic?

[PDF] <u>HIV Pre-Exposure Prophylaxis: Are we "PrEPing" for a new epidemic?</u>

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

Acquired Iatrogenic Death Syndrome

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

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Therapeutic Recommendations

http://www.ummafrapp.de/skandal/felix/
therapeutic recommendations.pdf

The HIV-positive inducing mycobacteria

http://www.ummafrapp.de/skandal/felix/The_HIVinducing 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

Various authors :

Voir le SIDA autrement

Etienne de Harven Interview Video (2012)

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

15 years of AIDS

Alfred Hässig, Wen-Xi Liang, Heinrich Kremer und Stefan Lanka

http://www.ummafrapp.de/skandal/continuum/15 years of AIDS.pdf

Questioning the HIV-AIDS hypothesis: 30 years of dissent

[HTML] Questioning the HIV-AIDS hypothesis: 30 years of dissent