

Natural Health Strategies Against AIDS

With Extrapolation to other Oxidative Disorders

Including Infectious / Auto-immune / Degenerative Conditions

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This author does not believe that AIDS is caused by HIV, a mere immune cell passenger virus, if indeed a human immuno-virus exists and is present at all and is not a mere likely artefact, even in cases of test confirmed, but likely false seropositivity, attributable rather to cross-reacting antibodies in response to multiple organisms in a compromised cellular and resultantly hyper-activated humoral immune response. AIDS is caused by all chronic multiple opportunistic macro- and/or micro-organism infections, where immune systems are already compromised by socio-economic, behavioural or environmental related... a) nutritional deficiency, and/or b) foreign protein factor and/or c) chemically induced... "**oxidative stress**". Accordingly, correction of these factors and appropriate reduction of the hosted organisms can reverse AIDS.

The correlated scientific information in this report is not to be construed to constitute medical advice, nor the active promotion of any substance or device for any medicinal purposes. It represents the author's research over several years in the field of free radicals, which are at the cellular basis of all pathologies, and even the aging process itself. Arising from this fact, is the logical conclusion that free radical scavengers/quenchers, ie antioxidants/reducing agents, are critical to health, especially considering that the primary immune response utilizes oxidation as its defensive mechanism against pathogens and malignant cells. If a normally constituent dietary substance is deficient and its optimal provision in basic form as provided by nature is able to reverse any pathological condition, then that substance cannot be deemed to be medicinal, but rather nutritional. Medicines are synthesised substances or even natural products, which are not a normal part of a natural (early) human diet. This is the entire focus of this paper, coupled with the fundamental behavioural changes necessary to remove any other precipitating factors and accommodate the innate natural spontaneous healing response. The focus of this paper is "health optimisation" as an alternative to allopathic and natural medicine.

Immune function is highly dependent on nutritional status because the large mass and high rate of cellular turnover of the immune system make it a major user of nutrients. Furthermore, nutrient requirements may be increased during and chronic infections, including HIV. Research has found lower concentrations of **magnesium** beginning early in the course of HIV infection, with significant univariate associations between CD4(+) T-lymphocytes and plasma magnesium, **choline** and **zinc** concentrations, which factors together explained 43% of variability in CD4(+) cell counts. Compromised nutritional and anti-oxidant status begin early in HIV infection and may contribute to disease progression. (Bogden J, et al, Am J Clin Nutr, 72(3): 809, 2000) Several other key nutrients, in particular, trace elements (Cu, Zn, Mn, Se) will be detailed herein.

Nutritional deficiencies are the primary cause of AIDS in the Third World, coupled with parasites (foreign protein) as a result of unhygienic conditions and specific susceptibility due to malnutrition. Nutritional deficiencies are also common in the First World. Universally, foreign protein factors include receptive anal semen, hemophilic Factor VIII and other blood products (irrespective of the presence of hypothetical HIV), multiple behavioral and environmental microorganism exposures, macro-parasitic infections and also vaccinations. Pharmacological factors, primarily, but not limited to the First World, include recreational and medical drugs including eg anti-retroviral drugs, amyl nitrate vasodilators, vaccines and inoculations, non-steroidal anti-inflammatories, analgesics, antibiotics, corticosteroids, progesterones and estrogens, anti-depressants, and sedatives. Several environmental contaminants may also lead to serious immune damage.

Benzene exposure can lead to myelotoxicity, expressed as leukopenia, pancytopenia, anemia, aplastic or hypoplastic bone marrow, lymphocytopenia, granulocytopenia and thrombocytopenia, the resultant immunosuppression leading to increased susceptibility to tuberculosis and pneumonia, which are the most common causes of death in AIDS. The introduction of lead-free petrol will lead to increased environmental exposure to benzene which is used as a replacement lubricant for lead and which if used contrary to design, without a functional catalytic converter, as is now commonplace because of a cheaper price differential, will build up, in particular in our cities. Some other chemicals of concern are the ubiquitous PCB's (T-helper/T-suppressor balance), PBB's (decreased peripheral T-cells), TCDD's (thymic epithelial cytotoxicity and thymic atrophy), asbestos (reduced T-cell proliferation and circulation), mercury (depressed lymphocyte responses to T-cell mitogens) and pesticides, all the major classes of which latter negatively impact on T-lymphocyte mediated cellular immunity and are progressively being implicated as a conveniently neglected far more serious and more direct cause of immunodeficiency in AIDS than HIV, which is a very lucrative modern scapegoat.

Hundreds of eminent and well-credentialed progressive scientists and academics, understanding **AIDS as a multi-factorial condition**, have questioned and competently scientifically criticised the HIV-AIDS hypothesis, including three Nobel laureates, Walter Gilbert, Barbara McClintock and Kary Mullis and (restricted to a few "professors" of the following disciplines/institutions by way of illustration): Addy, clinical microbiology, University Sci & Tech, Kumasi, Ghana; Bradford, biology, Kansas Univ; Canton, medical ethics, Griffith Univ, Australia; Duesberg, molecular / cell biology, Univ Calif, Berkeley; Gesheker, African History, Calif State Univ; Griffin, virology, Royal Med School, London; Johnson, law, Univ Calif, Berkeley; Loman, biophysical chemistry, Free Univ Amsterdam; Papadimitriou, pathology, Univ W. Australia; Papadopoulos-Eleopoulos, biophysics, Univ W Australia; Pinching, immunology, St. Bartholomew's Hospital, London; Root Bernstein, physiology, Michigan, State Univ; Rubin, molecular biology, Univ Calif Berkeley; Stewart, public health, Univ Glasgow; and Thomas, biochemistry, Harvard University.

This alternative, more consistent paradigm is rapidly gaining scientific ground internationally amongst progressive free-thinking scientists, with two core groups forming, the Berkeley Group, led by Professor Peter Duesberg, and the Perth Group, led by Professor Eleni Papadopoulos-Eleopoulos. Some key papers arguing against HIV-AIDS are: (Duesberg P, *Science*, 241: 1988), (Duesberg P, *Natl Acad Sci, USA*, 86: 755, 1989 & 88: 1575, 1991), (Papadopoulos-Eleopoulos E, et al, *Res Immunol*, 143, 1992), (Duesberg P, *Pharmacol & Therap*, 55: 201, 1992), (*AIDS: Virus or Drug Induced? Contemporary Issues in Genetics and Evolution*, Vol 5, Edited by Peter Duesberg, Kluwer Acad Publ, 1996), (Shallenberger F, *Med Hypothesis*, 50(1): 67, 1998), (Duesberg P, *Genetica*, 104: 85, 1998), (Papadopoulos-Eleopoulos E, et al, *Current Med Res*

Opinion, Vol 15: Suppl, 1999). Medical politics however, biased towards the germ theory (rather than the milieu in which they are predisposed to proliferate), mitigates strongly against the fair publication of alternative scientific arguments to the HIV thesis, despite their high merits.

Since HIV is currently still widely held to be the causative agent of AIDS and HIV is considered to be a retrovirus, most chemotherapeutic approaches have been anti-retroviral, with the molecular targets usually being reverse transcriptase inhibitors and associated protease inhibitors, since both are essential for retroviral replication and hence assumed pathogenesis. The HIV-AIDS hypothesis is however steadily eroding under the sheer weight of well-argued contradictory evidence. Various dideoxynucleoside analogs and only the triphosphoralated form of the highly toxic AZT, have demonstrated inhibition of assumed pro-viral DNA synthesis by HIV associated reverse transcriptase, yet have failed to prevent AIDS progression via (erroneously) postulated HIV-induced cytopathogenicity. AIDS science as practiced by the medical establishment is in fact not science at all, just a predominant theory which has too many financially and legally vested interests to undergo the vigorous evolution which usually accompanies true objective science.

Various serious immuno-suppressive and other debilitating side effects and the appearance of drug resistant virus strains are associated with anti-retroviral drugs, further limiting the scope of this approach. Recently, a US Government study established clear carcinogenicity for AZT at realistic clinical doses, but the shocking results of this official study, authored anonymously, languishes unpublished behind obscure access codes on the US National Toxicology Program database. Only a fortuitous personal exchange between the author of this article before you and Dr Richard Beltz, the government scientist who first synthesised AZT, has led to the discovery of this suppressed landmark document, published for the first time in abstract form in association with this article. (Toxicology and Carcinogenesis Studies of AZT. Natl Toxicol Program Tech Rep Ser, 1999, (469), 1-357) (Access the Gaia Research website for this previously unpublished paper)

From a natural health perspective, the new anti-AIDS frontier is that of the free radical paradigm, comprising natural and benign antimicrobial agents – botanicals, colloidal silver and oxygen – plus antioxidants and copper, zinc, manganese & selenium trace mineral-dependent endogenous antioxidant enzyme induction. For die-hard sceptics, a protocol including HIV remains appropriate, since it can still serve as a control for other parasites, pathogens and opportunistic organisms which are responsible for the real clinical disease progression and death in AIDS. Conversely, if HIV does in fact play a significant role in AIDS, the alternative strategy to be presented here will remain equally effective, the best of both worlds. Two critical, vastly under-considered, indeed scientifically ignored, if not suppressed phenomenon / mechanisms, because of the germ theory obsession with HIV, yet absolutely essential to understanding AIDS will be introduced here, namely “Selective Compartmental Dominance” and “Apoptosis”.

Selective Compartmental Dominance

Selective Compartmental Dominance (SCD) is a term coined by progressive American physician, Dr Frank Shallenberger MD, to describe a holistic concept of the real causes and immunological phenomenon of AIDS, which recent studies involving cellular mediated immunity and cytokine modulation can explain without the need to invoke infectious causation, but rather via functional characteristics and feedback loops in the immune system. This model predicts that even HIV negative members of the risk groups are susceptible to AIDS, assigns no special causal role for HIV and suggests a rational course of non-toxic therapy that can potentially reverse earlier AIDS cases.

The primary etiological “event” in the development of AIDS is a failure of an adequate cellular mediated immune response, attributed to the summation of various clinical and lifestyle factors. This results in an increase in extracellular antigen, which will in turn activate a humoral response, which induces cytokines that exert a negative feedback to the cellular mediated response, resulting in even further suppression of the cellular system. This creates a vicious cycle, which results in the progressive deterioration of the cellular system in the face of a hyper-activated humoral system, a hallmark of AIDS. Progressive loss of cell-mediated immunity (CMI) occurs as a consequence of the inhibitory effect of cytokines released when antibody mediated immunity (AMI) is chronically activated. SCD explains how HIV seropositivity may be nothing more than an excellent but arbitrary marker for individuals, who for reasons below, are in a state of chronic AMI activation. **The real causes of AIDS** according to Shallenberger’s SCD model are as follows:

Cell mediated immunity (CMI) suppressors: Male-male sperm allergens; Recreational drugs; Corticosteroids; Transfusions; Hemophiliac blood clotting factor VIII; Histiocytic & lymphoreticular cancer; Thymic damage; Pharmacological agents – anaesthetics, antibiotics, antivirals, tranquilisers; Malnutrition; Malabsorption; Sickle cell disease; Age, advanced & premature infants; Stress; Heavy metals (esp. Hg, Ni, Pb); Viral infections.

Antibody mediated AMI stimulators: Male-male sperm; Intravenous drugs; Root canals & other occult infections; Blood-clotting factor VIII; Multiple Infections; Vaccines; Parasites; Mycotoxins; Transfusions; Toxic bowel. (Shallenberger F, Med Hypothesis, 50(1): 67, 1998)

Apoptosis (Programmed Cell Death)

Apoptosis, programmed cell death, as opposed to cell proliferation, is distinct from degenerative necrosis. In the blood, lymphocytes are, after neutrophils, the most numerous white blood cells, representing from 20% to 45% of all leucocytes. Only a small fraction of the 10-12 have relative permanence, with under normal conditions, some 10-9 lymphocytes generated by the bone-marrow and about the same number dying every day, largely in the lymphoid tissues. The T-lymphocytic lineage is conceived in the bone marrow and the T-lymphocytes are differentiated in the thymus where the rest of the development, all the way to the mature T lymphocyte, and where they are stimulated to divide repeatedly to proliferate. Each effector T-cell cell arising from activation is programmed to die by apoptosis after the infection has been warded off. (Jan Klein and Vaclav Horejsi, Immunology, Blackwell Science, Oxford, 1997)

Oxidising agents can induce reversible cellular changes, including death by apoptosis. The ultimate outcome depends on the concentration of the agent, its rate of application, the initial state of the cells and the cellular milieu. Since both AIDS cultures and patients are exposed to activating agents (all of which are oxidising agents), both apoptosis and the phenomena upon which the presence of HIV is based (viral-like particles, antigen/antibody reactions), may be the direct result of oxidative stress and therefore their specificity is questionable. Activation is induced by oxidation. (Papadopulos-Eleopulos, E, *Med Hypotheses* 25: 151, 1988), (Turner V, *Med J Australia*. 153: 502, 1990), (Papadopulos-Eleopulos E, et al, *Med Hypotheses*, 39: 22, 1992), (Papadopulos-Eleopulos E, 'Oxidative Stress, HIV and AIDS', *Res Immunol*, 143: 145, 1992) This is supported by Montagnier's group's finding that apoptosis can be inhibited by reducing agents (René O, et al, Volume 2, VIIIth International Conference on AIDS, Amsterdam, 1992). Montagnier agrees with the Perth group that anti-oxidants should be used for treatment of AIDS patients (Gougeon M & Montagnier L, *Science*; 260: 1269, 1993), (Papadopulos-Eleopulos E, et al, *Genetica*, 95: 5, 1995)

A large body of evidence indicates that AIDS may be the consequence of a virus-induced antioxidant deficiency and implicates reactive oxygen species (ROS) in the pathogenesis of HIV and related infection. The high level of antigenic acid and cytokines activities in AIDS results in the production of superoxides (O₂⁻), hydrogen peroxide (H₂O₂) and hydroxyl radicals (OH). HIV-infected T-cells display low levels of superoxide dismutase, catalase, thioredoxin and glutathione peroxidase, rendering them susceptible to undergo apoptosis. Antioxidants may present potential interest as antiviral agents or as adjuvant therapy in AIDS. (Edeas M, et al, *CR Seances Soc Biol Fil* 189(3): 376, 1995) Intracellular oxidation is an obligate, early component of thymocyte apoptosis. Recent findings suggest that intracellular oxidants are involved in the induction of apoptosis, and that this type of cell death can be inhibited by various antioxidants (*See green tea, copper, zinc, manganese & selenium*). (Bustamante J, *Free Radic Biol Med* 19(3): 339, 1995)

Apoptosis of thymocytes plays a crucial role in shaping the repertoire of T-cell receptor specificities during T-cell development in the thymus. Mature CD4⁺ T-cells of asymptomatic patients have undergone apoptosis when stimulated *in vitro* and this process plays an important role in the regulation of normal T-cell responses to antigens *in vivo*. Heightened susceptibility to apoptosis might be responsible for loss of CD4⁺ T-cells and helper cells following HIV infection. (Owen J et al, Ch 18, in 'Programmed Cell Death', M Lavin and D Watters, Eds, Harwood Academic Publishers, GmbH, 1995) There is evidence that the CD4⁺ T lymphocyte deletion that is responsible for the development of immunodeficiency in patients with HIV infection is mediated by apoptosis and that infection of these lymphocytes by the virus is 'not' necessary for the triggering of their death. Much of the lymphocyte apoptosis is a result of defective support by cytokines or an inappropriate response to activation. Understanding the process may make it possible to prevent or delay the development of immunodeficiency without it even being necessary to eliminate the virus from the body. (J Kerr, Forward to 'Programmed Cell Death, Harwood Academic Publishers, 1995)

Apoptotic cell death significantly contributes to the depletion and dysfunction of CD4⁺ lymphocytes in AIDS, including cells uninfected with the HIV. (Pitrak D, *Oncologist*, 2(2): 121, 1997) In HIV infected patients, the increase of the concentration of free radicals is related to a depletion of endogenous antioxidative enzyme protective systems consecutive to the activation of lymphocytes and phagocytosing cells and direct or indirect effect of several pathologic agents. This free radical excess could impair cell membranes and generate apoptosis, the main cause of lymphocytes CD4⁺ depletion. (Rabaud C, et al, *Ann Biol Clin (Paris)*, 55(6): 565, 1997) Apoptosis of T lymphocytes in HIV infected individuals can be linked to oxidative stress. In view of the diminished oxidative resistance of HIV-infected individuals, research results

suggest that ROS-mediated apoptosis might contribute to the deletion of lymphocytes and to the pathogenesis of the disease. (Dobmeyer T, et al, *Free Radical Biol Med*, 22(5): 775, 1997)

Studies suggest that the killing of *Mycobacterium tuberculosis* in human monocytes in vitro by the addition of exogenous H₂O₂, is dependent on the susceptibility to a peroxide-induced killing pathway (Laochumroonvorapong P, et al, *Infect Immunol*, 65(11): 4850, 1997). Unlike antibacterial defence, when ROS and their derivatives act as biological weapons killing pathogens, the antiviral defence is assumed to be mediated by apoptosis. Cells activate generation of superoxide and hydrogen peroxide by xanthine oxidase and intracellular NADPH-oxidase in response to appearance of a virus in its cytoplasm. Increase in ROS level turns on the process of programmed cell death in the infected cells. Moreover, H₂O₂ diffuses into the adjacent cells (due to its high membrane permeability), also inducing apoptosis (of bystander cells), so that the infected cell and its neighbours (which are the most likely to be infected) are eliminated, blocking the spreading of the viral infection. (Skulachev V, *Biochem (Mosc)*, 63(12): 1438, 1998)

Apoptosis is the 'main' cause of CD4+ T-lymphocyte depletion in AIDS. Various chemical and biological agents trigger apoptosis in CD4+ Tcells. Oxidative stress induces apoptosis and participates in the CD4+ T cell apoptosis observed in AIDS patients, who present low levels of antioxidants (manganese superoxide dismutase (Mn-SOD), selenium and glutathione), due to inappropriate nutrition. Anti-apoptotic/antioxidant strategies should be considered alongside antiviral strategies for efficient therapy for AIDS. (Romero-Alvira D & Roche E, *Med Hypothesis* 51(2): 169, 1998) Common denominators in HIV-positive patients are an increase in oxidative stress and a weakened antioxidant defence system (Allard J, *Am J Clin Nutr*, 67(1): 143, 1998). Improved apoptosis inhibition with zinc in HIV+ individuals is documented (Neves I, *Clin Exp Immunol* 111(2): 264, 1998). Mn SOD protects T-cells from cell death in apoptosis and peripheral T-cell deletion (Hildeman D et al, *Immunity*, 10(6): 735, 1999).

Apoptosis is related to the ability of the cell to maintain an appropriate oxidant-antioxidant balance (Wedi B, et al, *Blood*, 94(7): 2365, 1999). It is a mechanism activated as a suicidal event to get rid of excess, damaged, or infected cells (Wang E, et al, *J Cell Biochem*. S32: 95, 1999). Apoptosis is the fate of most thymocytes (Yang Y, & Ashwell J, *J Clin Immunol* 19(6): 337, 1999). In the thymus, 95-98% of all thymocytes die by apoptosis (Guevara Patino J, et al, *J Immunol*, 164(4): 1689, 2000). Long-term activation of the immune system, weaker in HIV and related infections, significantly contributes to T-cell deletion and disease evolution (Michel P, et al, *J Infect Dis*, 181(1): 64, 2000). HIV infection is associated with increased cell death by apoptosis. in infected and 'uninfected' cells (Blanco J, et al *Antimicrob Agents Chemother*, 44(1): 51, 2000). Antioxidants can prevent apoptotic cell death, the protective mechanisms being their scavenging of oxygen free radicals (Shen J, et al, *Biochem Biophys Acta*. 1500(2): 217, 2000)

Colloidal Silver & AIDS

Another non-patentable natural product in increasingly wide-spread use in AIDS is electro-colloidal silver, positively-charged ultra-microscopic silver clusters suspended in water, as with the bio-colloids of the vital

fluids of all living organisms. These highly motile microclusters are naturally microbicidal, are as potent as the most powerful anti-microbials, yet are safe to higher life-forms by disabling only the metabolic enzymes of anaerobic micro-organisms and imparting disabling electrical charges to viruses. (Thomson, *Comprehensive Inorganic Chemistry*, Pergamon, 1973), (Mentel R, et al, *Vopr Virusol*, (6): 731, 1977), (Myers et al, *Review of Medical Pharmacology*, Lange Med Publ, 1978 (Williams D, *The Biocompatibility of Silver*, 1st Intl Confer on Gold and Silver in Medicine, Silver Institute, Washington, 1989), (Martin & Bustamante, *Physical Pharmacy*, Lea & Febiger, 1993), (Several references to follow)

Many researchers are of the opinion that silver is in fact an essential element, not because it is required for any mammalian enzyme system, but conversely, because since it is an anti-bacterial, anti-viral, anti fungal metabolite that disables specific enzymes that pathogenic and parasitic anaerobic micro-organisms use for respiration, colloidal/ionic silver functions as a systemic anti-anaerobic microbial and immune system supporter, which may be impaired by a silver deficiency (Drs J Wallach, DVM and M Lan MD, *Rare Earths: Forbidden Cures*, Double Happiness Publishing, 1995). Dr Robert Becker MD, identified a relationship between low levels of tissue and dietary silver and infection, stating that silver did more than kill disease-causing organisms, since silver was responsible for improper functioning of the immune system and its presence promoted and accelerated bone and tissue healing by over 50% (Becker R, *J Bone Joint Surg*, American Volume, 60: (7), 1978), (R Becker & G Seldon, *The Body Electric*, Morrow, 1985), (Becker R, *The Effect of Electrically Generated Silver Ions on Human Cells*. Proceedings of the 1st International Conference on Gold and Silver in Medicine, Silver Institute, Washington, 1989)

Among a number of metal ions tested at the Biochemistry of Upjohn Laboratories, zinc, copper and silver were found to be the most effective inhibitors of HIV protease (Unknown, *Biochemistry*, Sept 10, 1991). Colloidal silver kills HIV, according to Daryl Tichy, an administrator at Brigham Young University, who determined in independent testing at two different labs that colloidal silver killed a variety of pathogens, including HIV (*The Daily Herald*, Provo, Utah, February 13, 1992). Once the virus has invaded a cell in the body, the cell will revert back to the primitive type structure and use an enzyme as its chemical lung, which is promptly crippled by the presence of colloidal silver, the cell suffocates and dies, thus denying the virus an opportunity to replicate. Colloidal silver kills not only present viruses, but future forms as well, because no matter how the virus mutates, it cannot change the way human cells respond to invasion and because of its catalytic nature, colloidal silver is not affected in the reaction, continuing to kill other single celled pathogens nearby. (*The Colloidal Silver Handbook*, Silver Education Coalition, Utah Silver Institute, 1995)

Professor E Henderson at Temple University reported that colloidal silver completely eliminated latently infectious HIV and at lower doses significantly reduced HIV infectivity (Report, Temple Univ, School of Medicine, Dept of Microbiology and Immunology, Philadelphia, March 20, 1995). Dr M P Farber PhD, of the Colonel Leonard Farber Mild Silver Protein Foundation for Research and Development, cites a 1992 study at the University Medical Centre, Geneva, Switzerland, one of the most prestigious medical research facilities in the world, as confirming that colloidal silver kills all viruses, including HIV, via suffocation. Eight people recovered from HIV-AIDS in a scientifically documented study and an additional seven AIDS patients recovered as verified by anecdotal reports. (Dr M P Farber PhD, *The Micro Silver Bullet: A Scientifically Documented Answer to the Three Largest Epidemics in the World*, Myca Inc, 1997)

Electrochemical colloidal silver ions have potent microbicidal effects in water (Metodiev V & Bozhilova N, Probl Khig 15: 26, 1990). Research evidences that colloidal silver kills HIV and inhibits its replication and latent formation. It attacks the HIV and co-factor viruses and then wards off other infectious health problems that the immune system has not been able to handle. Research has proven under laboratory conditions that colloidal silver destroys HIV within 34 minutes after coming into contact with it. (Dr Keith Courtney ULC, Colloidal Silver: The Hidden Truths, 1997) Colloidal / ionic silver solutions exhibit better anti-microbial effectiveness than conventional silver solutions, due to the particularly potent and stable characteristics of electrochemical Ag⁺ (Simonetti N, et al, Appl Environ Microbiol, 58(12): 3834, 1992). New technologies give technical insight into the physics involved eg “completely non-toxic colloidal silver ions, triggered by pathogens, fire electrons, electrocuting HIV, pathogens and immunity suppressing moieties, destroying them” (US Patent No 5676977, Antelman Technologies Ltd, 14 October 1997).

Several studies now strongly suggest that colloidal silver has a stimulating effect on the immune system and there is considerable evidence that silver works as an antibiotic, thereby renewing interest in electro-colloidal silver, with companies developing new silver compounds for a wide variety of applications, including protection against the spread of the HIV (Dr Hill, Colloidal Silver: A Literature Review, Clear Lake Press, 1997). Tichy, at Brigham, now a scientific advisor to company planning clinical trials of a proprietary ionic colloidal silver formula, sent samples to Dr Larry Ford MD at the University of California, Los Angeles Medical Centre for testing. Ford reported that it killed every bacteria, fungus and virus tested, including HIV (Press Release, Invision International, Fort Lauderdale, FL, July 17, 1998).

Hydrogen Peroxide (H₂O₂) & AIDS

Hydrogen peroxide is an adjunctive source of oxygen, protecting against infections without significant systemic toxicity (Urschel H, Diseases of the Chest, 51(2): 180, 1967). Interferons, in addition to antiviral action, activate natural killer cells and macrophages, and modulate phagocytosis. Activated macrophages produce H₂O₂, which is responsible for the sterilising action against micro-organisms. (Das U, et al, J Free Radic Biol Med 2(3): 183, 1986) Some researchers believe that AIDS can be treated and even cured in some cases with hydrogen peroxide, the theory being that HIV and opportunistic pathogens are anaerobic and do not thrive when exposed to singlet oxygen supplied by the hydrogen peroxide on breakdown to water and oxygen in a reaction as follows: H₂O₂ ---> H₂O + O⁻. Singlet oxygen is the active microbicidal agent. It kills, or severely inhibits anaerobic organisms (pathogens using carbon dioxide for energy and leaving oxygen as a by-product). (Ed McCabe, Oxygen Therapies, Energy Publ, 1988)

Dr William Douglas MD, states that: “No other chemical comes even close to hydrogen peroxide in its importance to life. The cells of the body that fight infection, called granulocytes, produce H₂O₂ as a first line of defence against every type of invading organism: parasites, viruses, bacteria, and yeast. H₂O₂ must be present for the immune response to function properly”. Dr Douglas conducted an extensive search of the medical literature and identified several indispensable anti-infectious immunological attributes of hydrogen peroxide, including: a) Stimulating oxidative enzyme systems via metabolic pathways; b) Altering T-4/T-8 ratio, increasing the T-4 helper cells; c) Stimulating monocytes; d) Stimulating T-helper cells; e) Stimulating gamma - interferon production; and f) Responsibility for immuno-regulation. (Dr W Douglas, Hydrogen

Peroxide: Medical Miracle, Second Opinion Publ, 1992) Hydrogen peroxide is confirmed as a potent activator of T-lymphocyte functions and to significantly increase T-cell proliferation when applied for short periods under reducing (antioxidant) conditions (Los M, et al, Eur J Immunol, 25(1): 159, 1995).

Hydrogen peroxide is active against a wide range of organisms: bacteria, bacterial spores, yeasts, fungi and viruses (Block S, Peroxygen Compounds, in S Block, Ed, Disinfection, Sterilisation and Preservation, Lea & Febiger, 1991), (Heckert R, et al, Appl Environ Microbiol, 63(10): 3916, 1997). HIV is rapidly inactivated by exposure to peroxidase and H₂O₂ (Klebanoff S & Kazazi F, J Clin Microbiol, 33(8): 2054, 1995). Hydrogen peroxide is known to potentiate the virucidal effects of copper ions (Sagripanti J, et al (Appl Environ Microbiol 59: 4374, 1993), including against HIV (Sagripanti J & Bonifacino A, Appl Environ Microbiol 62(2): 545, 1996). ***[Precautionary Note. It is imperative to avoid the indiscriminate use of oxidising agents in the presence of inadequate anti-oxidative cellular defence, since such circumstances may have the opposite of the intended effect, actually stimulating viral replication, following resultant oxidative stress.]*** Ozone inactivates extra-cellular HIV at non-cytotoxic concentrations and has been proposed as a treatment for AIDS (Wells K et al, Blood, 78: 1882, 1991), (Carpendale M & Freeberg J, Antiviral Res, 16(199): 281, 1991), (Carpendale M & Griffis J, Proc 11th Ozone World Congr, 1993), (Shallenberger F, Med Hypothesis, 50(1): 67, 1998). However, when ozone is introduced into the blood, it reacts with red cells, producing hydrogen peroxide, of which the presence of pharmacological concentrations in the blood is clearly a double-edged sword, easily causing as much harm as good (Green S, Scientific Rev Alternative Medicine, Spring/Summer, 1998).

CD8⁺ T-lymphocytes from HIV⁺ individuals is functionally defective in the biochemical indices related to cell proliferation. In HIV⁺ but not HIV⁻ individuals, constitutively generated hydrogen peroxide levels are significantly lower in CD8⁺ T-cells compared with CD4⁺ T-cells. Importantly, activated effector CD8⁺CD28⁻ cells show remarkably low H₂O₂ levels compared with CD8⁺CD28⁺ cells, and the latter in HIV⁺ individuals also show low levels. Catalase content is lower in CD8⁺ cells compared with CD4⁺ cells only in HIV⁺ individuals. These results suggest that CD8⁺ T-lymphocytes are functionally defective with constitutively generated levels of H₂O₂ and the corresponding scavenger (catalase). Diminished immunocompetence of HIV⁺ individuals may be caused, in part, by this functional suppression of intracellular H₂O₂ defect of CD8⁺ T-cells. (Yano S, et al, Free Radic Biol Med 15; 24(2): 349, 1998)

Significantly, progressing HIV infection below 0.01mM hydrogen peroxide concentrations safely declines with increasing directly administered H₂O₂ concentrations from 0.05 to 0.1mM, with 5mM, resulting in significant HIV suppression (Ranjbar S & Holmes H, Free Radic Biol Med, 20(4): 573, 1996), (Kurata S, J Biol Chem, 271(36): 21798, 1996). Appropriate H₂O₂ disinfection guarantees destruction of HIV and opportunistic infections, including lipid and non-lipid viruses (Heckert R, et al, Appl Environ Microbiol 63(10): 3916, 1997), (Roberts C & Antonoplos P, Amer J Infect Control, 26(2): 94, 1998), (Vickery K, et al, J Hosp Infect, 41(4): 317, 1999). H₂O₂ is acknowledged to be highly toxic to bacteria and to viruses, including HIV (Stephenson J, J Am Med Assoc, 283(14), 12 April 2000).

Colloidal Silver / H₂O₂ Synergy & AIDS

A new strategy, bearing phenomenal potential, synergises colloidal silver with hydrogen peroxide and is accepted as a safe drinking water alternative to toxic chlorine disinfection, by health authorities in Switzerland, Germany, Israel, Australia and elsewhere. Trace microbicidal hydrogen peroxide and especially silver residues remaining after primary virus decontamination of drinking water further sustains critical instability of viruses. (Mahnel H, & Schmidt M, Zentralbl Bakteriell Hyg [B] 182(4): 381, 1986), (Thurman R & Gerba C, CRC Critical Rev Environ Contr 18(4): 295, 1989), (Moyasar T, et al, Canadian J Microbiol, Natl Res Council of Canada, 109-116, 1990), (Drinking Water Treatment Chemical – Silver / Hydrogen Peroxide, National Health and Medical Research Council (Australia), NHMRC Water Quality Panel, 10/11/93), (Pedahzur R, et al, - Health-related Water Microbiology. Select Proc Intl Symp Water Quality, July 1994, Budapest, Hungary, - Water Sci Tech, 31(5-6): 123, 1995), (Pandya M, Chemical Engineering World, Vol XXXII, No 10, 1997), (Report on the Laboratory Evaluation of a Biocide Intended for Water Treatment, D Peterson, Water Examination Laboratory, Perth, Australia, March 1998)

The spectrum of pathogenic organisms susceptible to even low concentrations of this combination is truly remarkable: bacteriophages, gram-negative, positive and spore-bearing bacteria, yeasts, fungi, mycodermis, amoeba, meningococci and viruses, including HIV (Expert Report on HIV effectiveness of SS-25, Prof Dr med, Gert Frosner, Max von Pettenkofer Institut für Hygiene und Medizinische Mikrobiologie, Universität München, BRD, November 23, 1987), (Pandya M, Chem Eng World, XXXII (10), 1997) and AIDS related pathogens: M-R Staphylococcus aureus, Tuberculosis, Hepatitis-B, Herpes, etc (Expert Opinion on the Anti-Viral Effect of SS-25, National Institute of Hygiene, Budapest, Hungary, June 1988), (Effect of SS-25 on Mycobacterium Tuberculosis, Microbiological Laboratory, Zagreb, Croatia, November 1998). HIV actually bottoms the list in terms of resistance to anti-microbials (Mc Donnell G & Russell A, Clin Microbiol Rev, 12(1): 147, 1999). Medicines regulatory authorities ironically are attempting to ban colloidal silver / H₂O₂ as medicines, in spite of it paradoxically being approved for national drinking supplies, albeit less effective at time of use, due to problems with the distribution networks and quality of the bulk water treated. (See associated definitive rebuttal titled: ***“The Colloidal Silver Ban Scam”*** <http://www.gaia.research.co.za>)

Independent developments in South Africa take the colloidal silver / hydrogen peroxide synergy potential even further, to concurrently address the actual micro-nutritional causes of the breakdown of the cellular immune response itself. The Gaia protocol involves an affordable kit comprising a hand-sized portable electro-Colloidal Silver Generator - water steriliser and fortification unit, incorporating both silver as well as alloy electrodes of copper (Cu), zinc (Zn) and manganese (Mn), specially compounded in the ratio required by the body's own cellular immuno-enzymes. Selenium (Se), a non-metallic element unable to be electro-colloidalised, is best assimilated when processed through a plant by adding as a micronised powder to sprouting seed soak water to be biologically colloidalised by the seedling, not feasible with the metals, which are too hard to micronise mechanically. Selenium enriched water, preserved with colloidal silver, can be used repeatedly as a drench after initial soaking, ensuring continual uptake of the selenium. Tableted or powdered dietary supplements, whilst better than nothing, are not efficient vehicles, unless chelated. (For more protocol information, contact the author at ph/fax 044-532-7765/7695 or email <gaia.research@pixie.co.za> or access the Gaia Research website at: <http://www.gaiaresearch.co.za>)

Trace Elements & AIDS

Nutrition is a critical determinant of immune responses and malnutrition and even over-nutrition and obesity are the most common causes of immunodeficiency worldwide. Even relatively moderate deficiency of protein, vitamins A, C, E, B-spectrum, folate and the abovementioned micronutrients may result in negatively altered immune responses. (Chandra R, Amer J Clin Nutr, 66(2): 460S, 1997) Whilst the former macro-nutrients are more readily assured by a healthy diet, the critical micro-nutrients (Cu Zn, Mn, Se etc) are only available to food plants if the soil in which they grow have these in bio-available form, optimised only in organically husbanded soil bearing a diversity of beneficial organisms and their by-products (microbial and humic chelating acids), needed to colloidalise and chelate the elements (Schatz A, Teaching Science with Soil, Rodale Press, 1972), (Tompkins P & Bird C, Secrets of the Soil, Arkana, 1992), which natural process at least doubles the mineral content of such plants (Smith B, J Appl Nutr, 45(1): 35, 1993).

These nutrients are also significantly decreased in infants with malnutrition and infection increases the risk of deficiency (and visa versa) (Khaldi F, et al, Arch Pediatr, 2(9): 854:1995). Among the trace elements, copper, zinc, manganese and selenium are essential for the integrity and optimal functioning of the immune system. Although each element has different functions, the deficiencies mainly cause dysfunction of the cell-mediated immunity, which can be improved by supplementation. An excess of an element also impairs immunity and a proper balance of elements is essential for immunocompetence. (Kodama H, Nippo Rinsho, 54(1): 46,1996) The frequent occurrence of abnormal nutrition found in AIDS subjects contributes to disease pathogenesis. **Magnesium** is an essential nutrient required for many biological functions in the body, including over 300 enzymes (A Schauss, Minerals, Trace Elements & Human Health, Life Science Press, 1995). Magnesium deficiency may be partially relevant to AIDS symptoms of fatigue, lethargy and impaired mentation. (Skurnick J, et al, J Acquir Immune Defic Syndr Hum Retrovirol, 12(1): 75, 1996)

Whatever the nutritional potential of foods or supplements, their contribution is non-existent if they do not pass the test of absorption (R Pike & M Brown, Nutrition: An Integrated Approach, John Wiley and Sons, 1984). Four of the eight essential minerals known to be absolutely required in ionic form at the point of intraluminal absorption are the abovementioned critical anti-infectious antioxidants, namely Cu, Zn, Mn and Se. In foods or supplements these specific minerals must first be freed from whatever matrix they are bound up in, a liberating process relying on stomachic hydrochloric acid (Alexander Schauss, PhD, Minerals, Trace Elements & Human Health, Life Science Press, Tacoma, WA, 1995), which in ill-health contributes to oxygen antagonistic acidosis and interfere with available colloidal ions. Electro-colloids are able to by-pass this process since they are inherently highly ionic. When only slightly soluble solid ionic substances dissolve in water, they break up into individual ions and are liberated. Significantly, colloidal/ionic silver (& Cu, Zn & Mn) ions are rendered relatively insoluble by hydrochloric acid and hence contemporary dietary sources are rarely able to meet requirements. (Steven Zumdahl, PhD, Chemical Principles, DC Heath & Co, 1992).

HIV-infected cells exhibit reduced levels of antioxidant enzymes (Sandstrom P, et al, Free Radical Biol Med, 24(9): 1485, 1998). Sufficient essential nutrients such as methionine, cysteine, copper, zinc, manganese and selenium are indispensable for the maintenance of optimal immune cell functions. The way in which the right amount of Cu and Zn ions and cysteine/glutathione (GSH) are made available in the right place at the right time and in the right form, can prevent an unchecked multiplication of HIV and AIDS pathogens in a more passive or active way. Zinc and copper ions stimulate/inhibit/block in a concentration-dependent way the intracellular activation of essential protein-splitting enzymes such as HIV proteases. Zinc and copper ions act

as 'passive' virus inhibitors. Ions that remain available in sufficient amounts via cysteine/GSH are effective natural inhibitors/combaters of AIDS viruses and prevent the development of chronic virus diseases that can lead to AIDS. (Sprietsma J, *Med Hypotheses*, 52(6): 529, 1999)

Beneficial aerobic (oxygen using) organisms possess antioxidant defence systems that deal with reactive oxygen species produced as a consequence of aerobic respiration and immuno-defence. Reactive oxygen is related to growth and cell differentiation. Low concentrations of reactive oxygen intermediates may be beneficial or even indispensable in processes such as intracellular messaging and defence against anaerobic (oxygen shunning) pathogenic micro-organisms, but higher amounts of active oxygen may be harmful to cells, especially those of the immune system and even beneficial organisms. A wide array of endogenous enzymatic antioxidant defences exists, namely copper, zinc and manganese dependent superoxide dismutase (SOD), selenium dependent glutathione peroxidase (GPX) and copper dependent catalase (CAT). (Mates J, & Sanchez-Jimenez F, *Frontiers in Bioscience*, 4: d339, 1999)

Antioxidant Enzymes & AIDS

HIV and AIDS related infection mediated modification of host antioxidant enzymes are important components in mediating ongoing infections and the ultimate progression to severe immunodeficiency, altered by the presence of opportunistic pathogens. Advances in understanding have prompted investigations into the use of antioxidant therapy for AIDS. (Miller R & Britigan B, *Clin Microbiol Rev*, 10(1): 1, 1997) Small deviations from the physiological values of these antioxidant enzymes, which work synergistically together, may have a dramatic effect on the essential resistance of cells to oxidative damage. Uncontrolled toxic oxygen plays a role in the ageing process as well as in a number of human diseases and an unbalanced production of reactive oxygen intermediates has established its relationship with specific pathologies, including HIV infection and AIDS. When these systems are overwhelmed, pathologic conditions, including AIDS may result. (Banki K, et al, *J Biol Chem* 273: 11944, 1998)

It is expected that understanding the contribution of oxidant-antioxidant imbalance to diseases may develop a new strategy of 'antioxidant' therapies (Takahashi K, et al, *Nippon Rinsho*, 57(9): 1988, 1999). Cellular regulation and expression of antioxidant enzymes and their scavenging of active oxygen intermediates has been proposed as one of the mechanism to promote immunity (Mates J, & Sanchez-Jimenez F, *Frontiers in Bioscience*, 4: d339, 1999). Dr Neil Graham and colleagues of Johns Hopkins School of Hygiene and Public Health think that a change in mineral levels in the blood may be a better predictor of HIV progression than CD4 cell counts (*Discover* 11(11): 14, 1990). The functions of the endogenous (internally produced) trace element dependent anti-oxidant enzyme systems are briefly summarised as follows:

Manganese - dependent superoxide dismutase is essential for the survival of beneficial aerobic life and the development of cellular resistance to oxygen radical-mediated toxicity. Copper/zinc - dependent intracellular superoxide dismutase plays a major role in the first line of antioxidant defence by catalysing the dismutation

of superoxide anion radicals to form hydrogen peroxide and molecular oxygen. Copper/Zinc - dependent extracellular SOD is found in the interstitial spaces of tissues and in extracellular fluids and is similarly responsible for the majority of the antioxidant SOD activity in plasma, lymph, and synovial fluid. Copper - dependent catalase is one of the most efficient enzymes known. It cannot be saturated by H₂O₂ at any concentration. Catalase reacts with H₂O₂ to form water and molecular oxygen and protects cells from hydrogen peroxide generated within them, playing an important role in the acquisition of tolerance to oxidative stress in adaptive cellular responses. (Mates J, & Sanchez-Jimenez F, *Frontiers in Bioscience*, 4: d339, 1999) An immunological synopsis of the key endogenous antioxidant enzyme nutrients follows:

Copper

Diets, especially in Western countries, provide copper below or in the low range of the estimated adequate daily dietary intake (*Sci News*, Vol 148, Aug 12, 1995), (Uauy R, et al, *Am J Clin Nutr*, 67(5): 952S, 1998). Copper deficiency results in increased infection rates due to immune abnormalities, such as reduced cellular immune response, reduced activity of white blood cells and reduced thymus hormone (Elson Haas, M.D, *Staying Healthy With Nutrition*, Celestial Arts, 1992). High fibre diets inhibit especially copper absorption (Knudson E, et al, *J Trace Elem Med Biol*, 10(2): 68, 1996) contributing to suboptimal copper status (Wapnir R, *Am J Clin Nutr*, 67(5 Suppl): 1054S, 1998). High iron and zinc intakes also interfere with copper absorption, yet none of 40 fortified high fibre breakfast cereals examined were fortified and many supplements contained iron and zinc, but no copper, or only in poorly absorbed form, with prenatal and infant formulas actually faring the worst (Johnson M, et al, *Am J Clin Nutr*, 67(5 Suppl): 1035S, 1998)

Premature infants have high copper requirements as a result of low perinatal stores and infants in general constitute a risk group because milk is low in copper (Lonnerdal B, *Am J Clin Nutr*, 63(5): 821S, 1996). If diets low in copper are consumed during pregnancy, maternal stores will be depleted (Klevay L & Medeiros D, *J Nutr*, 126(9 Suppl): 2419S, 1996). Copper is involved in the function of several enzymes and especially for infant growth and development and for host immune defence mechanisms. "Acquired" deficiency of copper is mainly a pathology of infants, but also of malnourished children. Clinical deficiency manifestations are anemia, neutropenia, altered phagocytic capacity of neutrophils and as a direct result of these, increased incidence of infections. (Olivares M & Uauy R, *Am J Clin Nutr*, 63(5): 791S, 1996)

Copper is an essential nutrient having no adverse effects except in rare chronic use and Wilson's disease (Olivares M, et al, *J Pediatr Gastroenterol Nutr*, 26(3): 251, 1998), (Barceloux D, *J Toxicol Clin Toxicol*, 37(2): 217, 1999). When dietary copper is high and more is absorbed, endogenous excretion increases, protecting against excess accumulation of copper in the body (Turnlund J, *Am J Clin Nutr*, 67(5 Suppl): 960S, 1998). Micromolar concentrations of copper can inhibit HIV-1 protease, the enzyme that duplicates the virus (Karlstrom A & Levine R, *J Am Med Assoc*, 26(9): 1185, 1991) Serum from individuals with AIDS have more **catalase** activity, which increases progressively with advancing HIV infection. Increases in serum copper – dependent catalase activity correlates with increases in serum hydrogen peroxide scavenging without altering the bactericidal activity of neutrophils or mononuclear cell cytotoxicity in vitro. Increases in

serum catalase activity may reflect and/or compensate for systemic **glutathione** and other antioxidant deficiencies in HIV-infected individuals. (Leff J, et al, Free Radic Biol Med, 13(2): 143, 1992)

Copper, ascorbate and sublethal amounts of hydrogen peroxide are powerfully synergistic in destroying HIV (WHO AIDS Series 2, World Health Organisation, Geneva, 1992). Copper ions inactivate HIV (Sagripart J & Lightfoote M, AIDS Res Hum Retroviruses, 12: 333, 1996). Copper inhibits intracellular HIV, offering real prospects against AIDS (Sprietsma J, Med Hypotheses, 49(1): 1, 1997). The immune system requires copper to perform several functions. Interleukin-2 is reduced in copper deficiency and is likely the mechanism by which T-cell proliferation is reduced, even in marginal deficiency. The number of neutrophils in human peripheral blood is reduced in severe copper deficiency and their ability to generate superoxide anion and kill ingested micro-organisms is reduced in even marginal copper deficiency (Hopkins R, Failla M, J Nutr, 127(2): 257, 1997), (Percival S, Am J Clin Nutr, 67(5): 1064S, 1998).

Cupric (copper) chloride inhibits HIV protease (PR), a pre-requisite for viral replication, representing a promising chemotherapy of AIDS, by acting on non-active-site cysteines and suggesting that copper chelates could be useful inhibitors of HIV-PR (Karlstrom A, et al, Proc Natl Acad Sci, USA, 88: 5552, 1991; Dettorre C & Levine R, Arch Biochem Biophys, 313: 71, 1996; Davis D, et al, Biochemistry, 35: 2482, 1996). Several copper compounds were potent inhibitors of this enzyme. An inhibitory effect of 82% and 93% was observed for 0.4 micro-M copper chelate and copper chloride respectively. Stoichiometric concentrations of copper ions inhibited HIV-PR by acting on the cysteine residue(s) outside of the active site. Cupric chloride has been shown to inhibit HIV-PR, including mutant protease in the presence of ascorbic acid (Davis D, et al, Arch Biochem Biophys, 322: 127, 1995), leading to postulation that the so-formed copper complexes could fit into the active site of the enzyme and inhibit its activity. (Lebon F, et al, Eur J Med Chem, 33: 733, 1998)

In association with reverse transcriptase inhibitors, protease inhibitors are used for the inhibition of viral replication. **The use of copper complexes is a potentially fruitful approach to the development of a new family of HIV-PR inhibitors, which could provide future alternatives to multi-drug AIDS treatment.** (Lebon F, et al, J Chem Soc, Perkins Trans, 2: 795, 1999) With AZT, as with many nucleoside analogues, toxicity is a problem. The peptide-like nature and size of most HIV-PR inhibitors limit oral bioavailability and half-life in humans, making high blood levels difficult to achieve and sustain. New copper co-ordination compounds, having geometry favourable for the orientation of their interacting substituents within the protease sub-sites have inhibited HIV-PR protease in the micro-molar range (Lebon F, et al, Perkin Trans, 2: 795, 1999). An additional target has been identified that affects protease activity, as cupric ions lead to the inhibition of the HIV protease enzyme. (Lebon F & Ledecq M, Current Medicinal Chemistry, 7: 455, 2000)

Zinc

Insufficient zinc has multiple effects on the immune system, particularly proliferation of T-lymphocytes, depression in number and activity of killer cells, and impaired antibody production (Alexander Schauss, PhD, Trace Elements and Human Health, Life Science Press, 1995). In particular, zinc confers biological activity to the thymic peptide, thymulin, responsible for cell-mediated immunity. In deep deficiencies, low thymulin levels are due to reduced peripheral saturation of thymic hormones by zinc ions (Mocchegiani E, et al, Int J

Immunopharmacol, 17(9): 703, 1995). Zinc is essential for the biological activity of thymulin, important in its zinc-bound form for the maturation and differentiation of T-cells and is also relevant for the liver extrathymic T-cell pathway (Mocchegiani E, et al, Mech Ageing Dev, 106(1-2): 183, 1998).

Functional deficiencies of zinc can change immune functions prematurely from predominantly cellular Th1 responses to humoral Th2 responses. T-helper (Th1) cells produce cytokines such as interleukin-2 and gamma - interferon, thereby controlling viral infections and other intracellular pathogens more effectively than Th2 responses. The shift adversely influences the course of AIDS. HIV does not replicate in Th1 cells, which contain more zinc, because zinc ions are known to inhibit intracellular HIV replication. Real prospects are offered by zinc against AIDS. (Sprietsma J, Med Hypotheses, 49(1): 1, 1997) Zinc regulates, via the zinc finger protein molecular structures, the activities of virus-combating Th-1 cells such as cytotoxic T-cells (Sprietsma J, Med Hypothesis, 52(6): 529, 1999).

Manganese

Manganese can function exert a pro-oxidant antimicrobial effect, inhibited by cellular protective catalase and can also act as an antioxidant and scavenge superoxide anions and hydrogen peroxide. These findings suggest that manganese or manganese superoxide dismutase, by increasing the conversion of superoxide to H₂O₂, can increase the activity of the antimicrobial system released by stimulated polymorphonuclear leukocytes. (Klebanoff S, et al, J Leukoc Biol 53(6): 666, 1993) The growth of Mycoplasma, an AIDS related pathogen (Montagnier's proposed co-factor), is also inhibited by manganese (Watanabe T, J Clin Microbiol, 32(5): 1343, 1994). Manganese also inhibits the binding of an AIDS related pathogen Cryptosporidium parvum sporozoite membrane antigens to immune cells and also affects sporozoite penetration of live immune cells, resulting in a dose-dependent inhibition of parasite development and in some cases, elimination of the intestinally derived oocysts (Nesterenko M, et al, Biol Trace Element Res, 56(3): 243, 1997).

The mitogenic activation of T-lymphocytes from human immunodeficiency virus-positive subjects involves perturbation of redox balance, as indicated by manganese **superoxide dismutase** (Mn-SOD) adaptive induction (Piedimonte G, et al, J Infect Dis, 176(3): 655, 1997). HIV infection induces a down-regulation of Mn-SOD transcription in CD4+ lymphocytes. Conversely, macrophages over-express the MN-SOD gene in response to infections and viral replication (Raoul H, et al, AIDS Res Hum Retroviruses, 14(5): 427, 1998). Some nutritionists recommend manganese supplements with AZT (AEGIS HIV/AIDS Database, 1998). Mn-SOD protects T-cells from superoxide generation and cell death in apoptosis and peripheral T-cell deletion (Hildeman D et al, Immunity, 10(6): 735, 1999). Manganese protects cells against HIV-1 protease-induced cytotoxicity. HIV-1 protease may contribute to brain atrophy seen in neuro-AIDS patients. Manganese may be an AIDS neuroprotective cation in the brain. (Pinkrah J, Howard Hughes Medical Institute, First Quarter, 2000) Electrocolloidal manganese supplementation aside, the content of manganese in **green tea** is 670-1850 micrograms/g in the tea leaf and 1.75-6.67 micrograms/ml in the tea beverage, a high dietary contribution (Matsushima F, et al, Nippon Eiseigaku Zasshi, 48(4): 864, 1993) that in some countries may be the most important dietary source (Proc Intl Symp Tea Sci, Shizuoka, Japan, 1991).

Selenium

[Important Precautionary Note for Colloidal Silver Protocols Hepatic necrosis and ultrastructural changes of the liver have been induced by silver administration to selenium deficient rats (Bunyan J, et al, Br J Nutr 22(2): 165, 1968). Investigators have hypothesised that this toxicity is related to a silver-induced selenium deficiency that inhibits the synthesis of the seleno-enzyme glutathione peroxidase. In animals supplemented with selenium, exposures of silver as high as 140 mg/kg/day (100 mg Ag/L drinking water) were well tolerated. (USEPA Integrated Risk Information System. Silver CASRN 7440-22-4, May 1998)]

Selenium is required for activity of the enzyme glutathione peroxidase, and selenium deficiency may be associated with myopathy, cardiomyopathy and immune dysfunction including oral candidiasis, impaired phagocytic function and decreased CD4 Tcells (Dworkin B, Chem. Biol. Interact, 91 (2-3), 1994). Due to its antiviral effects and its importance for all immunological functions, the administration of selenium is suggested as a supportive measure in early as well as in advanced stages of HIV-induced disease. Initial observations on the effects of selenium supplementation in HIV-infected patients indicate that selenium causes symptomatic improvements and possibly slows the course of the disease. An adequate supply of selenium and of antioxidant vitamins is also proposed as a measure to reduce the probability of the placental transmission of HIV in pregnancy. HIV and related pathogen infected patients are under chronic oxidative stress. Perturbations to the antioxidant defence system, including changes in levels of selenium have been observed to be indicative of oxidative stress during HIV and related pathogen infection in asymptomatic patients early in the course of the disease and may contribute to several aspects of HIV and related disease pathogenesis, including viral replication, decreased immune cell proliferation, loss of immune function, apoptosis and chronic weight loss. (Pace G & Leaf C, Free Radical Biol Med, 19(4): 532, 1995)

Selenium is an essential trace element in humans and supplementation in the prevention and treatment of AIDS-related pathology has been considered (Badmaev V, et al, Altern Ther Health Med, 2(4): 59, 1996). Stages I-III of HIV-disease are characterised by significant impairments of antioxidative defences provided by selenium dependent glutathione peroxidase (Look M, Eur J Clin Nutr, 51(4): 266, 1997). Selenium inhibits the expression and induction of HIV and related pathogen replication and supplementation also increases the activities of the cellular antioxidant, glutathione peroxidase, and hence may prove beneficial as an adjuvant therapy for AIDS. (Hori K, et al, AIDS Res Hum Retroviruses, 13(15): 1325, 1997) Declining plasma selenium levels and decreased glutathione peroxidase activity in AIDS is of particular concern in the light of selenium's influence on immune function, viral replication, and survival. Recent investigations indicate that selenium supplementation helps to increase the enzymatic defence systems in HIV and related pathogen infected patients. (Baum M & Shor-Posner G, Nutr Rev, 56(1Pt 2): S135, 1998)

The primary function of glutathione peroxidase enzymes, which are efficient extracellular antioxidants, is counteracting oxidative attack. In plasma it is directed to extracellular compartments and is expressed in tissues in contact with body fluids. (Brigelius-Flohe R, Free Radic Biol Med, 27(9-10): 951, 1999) Selenium deficiency, one of the most common nutritional deficiencies, is important in AIDS due to its dual function as a necessary immune modulation nutrient and antioxidant. Selenium deficiency is highly significant in predicting AIDS-related mortality. HIV and related pathogens themselves manufacture selenoproteins involved in the regulation of viral replication, further depleting marginal selenium levels. (Patrick L, Altern Med Rev 4(6): 403, 1999) The effects of HIV-1 infection are exacerbated in individuals who are selenium

deficient (Proc. Natl. Acad. Sci. USA 97(12), 2000) When all nutrient factors that are associated with survival are considered together, only selenium deficiency is a significant predictor of mortality. The profound effect of selenium on disease progression may reflect selenium's action in antioxidant defense systems, as well as gene regulation. (Baum M, J Acquir Immune Defic Syndr, 25, Suppl 1, 2000)

Selenium appears to be a key nutrient in counteracting the development of virulence and inhibiting HIV progression to AIDS (Rayman M, Lancet, 356(9225), 2000). An important role for selenium in AIDS has been proposed. Decreased selenium levels, as found in persons with HIV infection or AIDS, are sensitive markers of disease progression. Selenium deficiency, an independent predictor of mortality in both HIV-1-infected adults and children, is an essential micronutrient that is associated with an improvement of T cell function and reduced apoptosis in animal models. In addition, adequate selenium may enhance resistance to infections through modulation of interleukin (IL) production and subsequently the Th1/Th2 response. Selenium supplementation up-regulates IL-2 and increases activation, proliferation, differentiation, and programmed cell death of T helper cells. Moreover, selenium supplementation may down-regulate the abnormally high levels of IL-8 and tumor necrosis factor-alpha observed in HIV disease, which has been associated with neurologic damage, Kaposi's sarcoma, wasting syndrome, and increased viral replication. Together, these findings suggest a new mechanism through which selenium may affect HIV-1 disease progression. (Baum M, et al, J Infect Dis Sep, 182 Suppl 1, 2000)

Green Tea & AIDS

Green tea has long been scientifically established as a near miraculous natural health prophylactic and therapeutic substance, partially because of its powerful antioxidant status, with an incredible unprecedented 12,000 peer reviewed published scientific papers listed in a Medline electronic search as of mid-2000. Reuters, New York, reported on 12 September 1999 that Dr. L Mitscher, distinguished professor of medicinal chemistry at the University of Kansas, presented findings at the American Chemical Society National Meeting, stating that: "Green tea contains the strongest of all antioxidants". Green tea, notably both water and oil soluble and heat stable, also actively stimulates induction of the body's own anti-oxidative enzymes and is furthermore astonishingly broadly anti-microbial, yet sparing, even encouraging, of beneficial aerobic intestinal organisms (Proc Intl Symp on Tea Sci, Shizuoka, Japan, Aug 1991), (Amer Chem Soc Symp Ser 506 / 507, 1992 and 546 / 547, 1994), (Proc Soc Exp Biol Med 220(4): 255, 1999).

Green tea components have been reported to be strongly inhibitory or lethal to every opportunistic and pathogenic virus, mycoplasma, bacteria, yeast and fungus tested to date (Kubo I, ACS Symp Ser 525: 57, 1993), (Hara Y, ACS Symp Ser 547: 34, 1994), (Kono K, et al, J Japanese Assoc Infectious Disease, 68, 2:1518, 1994), (Yam T, et al, FEMS Microbiol Lett, 152(1): 169, 1997). Healing plants have relatively high concentrations of the powerful immune system stimulant, organic germanium. Green tea is the richest source of **organic germanium** (Chem Pharm Bull, 28: 2687, 1980). Germanium is able to repair and boost immune response, in particular T-lymphocytes, macrophages and natural killer cells, precisely the components compromised in AIDS (Dr Kuzihiko Asai, Miracle Cure: Organic Germanium, Japan Publ, 1980), (Intl Arch Allergy, 63, 1980), (J Interferon Res, 4, 1984), (Gan ToKagaku Ryoho, 12, 1985). Green tea is also a rich

source of organic minerals and has been determined to significantly increase the zinc, copper, manganese, and magnesium concentrations in the total blood (Hamdaoui M, et al, Ann Nutr Metab, 41(3): 196, 1997)

Green tea, like AZT (but without the horrendous toxic effects), is also a potent natural retroviral reverse transcriptase inhibitor, including against HIV (Kakiuchi et al, J Nat Prod, 48: 14, 1985), (Hatano et al, Chem Pharm Bull, 36: 2286, 1988), (Asanka et al, 4th Intl Confer Immunopharmacol, Osaka, Japan, May 1988), (Ono et al, Biochem Biophys Res Comm, 160, 1989), (Nakane & Ono, Nucl Acids Symp Ser, 21: 115, 1989), (Nakane & Ono, Biochem, 29(11): 2841, 1990), (Chung Kuo I Ko Hsueh Yuan Hsueh, 14(5): 334, 1992), (Moore & Pizza, Biochem J, 288 (Pt 3): 717, 1992), (Tao, Chung Kuo I Ko Hsueh, 14(5): 334, 1992), (Hara, Intl Symp on Tea Sci & Human Health, Calcutta, India, January 1993), (Nakane et al, 204th Natl Meeting of the Amer Chem Soc, Washington DC, Amer Chem Soc Symp Ser 547, 1994).

McCarty reported that the long-term efficacy of new combination drug therapies for HIV and related infections are limited by the tendency of transfected virus to mutate to drug-resistant forms and argues for the use of safe antimutagenic measures. He specifically stated that selenium and green-tea can suppress mutagenesis and can be expected to prolong the efficacy of therapy in HIV infection (Med Hypothesis, 48(3): 215, 1997). Dr A Hassig, Professor of Immunology at the University of Bern, Switzerland, in reply to a query as to his recommendations for HIV positive individuals and those with AIDS, stated that in treating immune dysfunction he recommended natural anti-oxidants like green tea and spices like curry, effective and of no danger, unlike the insufficiently tested chemical RT inhibitors (Continuum, June/July 1997). Although unpatentable, the exceptional health potentials of green tea to play a significant immunological role in AIDS conditions continue to excite scientists (Mathe G, Biomed Pharmacother, 53(4): 165, 1999).

*[Important note for Green Tea use. Not all types and grades are alike. The “qualities” to look for in green tea for health optimisation are not optimised in most commercial teas, which may have a much lower polyphenol content, due to breeding lineages to suit other agronomic, taste and aesthetic criteria. These antioxidants are not optimally induced in irrigation, fertiliser and pesticide pampered commercial crops. Besides the lack of toxic agrichemical residues, “peasant grown” plants struggle against harsh environmental stress, which optimises their synthesis of complex survival chemistry, of which the potent polyphenol catechins are the most important for health. **[Precautionary Note. An unappreciated significant risk relates to the chemical bio-accumulation characteristics of the tea plants themselves, whereby heavy nitrogen and superphosphate fertilisation, besides pampering the plants, also radically increases the uptake of aluminium, fluoride and cadmium respectively to potentially toxic levels, of particular concern given that at such levels, fluoride and cadmium have the potential to poison enzymes.]** (For more information on this topic, read the associated literature or contact the author of this paper)]*

This concludes my synopsis of the premier (safe, effective & affordable) natural anti-AIDS strategies. **Key to top 140:** The premier items are discussed above are bold underlined hereunder and expanded upon below, all weighted according to the scientific quality of the motivations, the remaining bold and underlined items being my priority and possible adjuvants. The remaining non-bolded items are listed as a research resource of substances bearing varying degrees of potential in managing AIDS generally, over and above nutritional /dietary strategies, which should ideally exclude all animal products other than unpasteurised dairy products, if use of reliable Bifidobacterium and Lactobacilli probiotics are not feasible to restore healthy colon

ecology. Foods of choice should be fresh fruits, salads, vegetables, seeds, nuts and sprouted legumes, all as raw as possible, mechanically blended/juiced if necessary for optimum biological integrity and assimilation.

See the “GAIA DIET” for details re optimal nutrition to balance pH, sodium-potassium & oxi-antioxidation.

All of the mechanisms and strategies outlined here (admittedly by no means exhaustive, just the absolute essentials) are applicable also to most other conditions having their genesis in damage to / deficits of the cellular immune response, including cancers and allergies, with the exceptions on the following list being the more exotic items, which are mainly antiretrovirals, requiring advanced application knowledge and professional monitoring, especially of the toxicological parameters. Other botanicals will relate and apply to the specific condition at hand, eg for heart disease and cancer, but will not offer much over and above the protocol outlined in this report, and may in fact likely be unnecessary if the protocol is strictly adhered to.

THE AIDS TOP 140

(Key two paragraphs back)

Acacia nilotica, Acemannan polysacharrides, Achyrocline flaccida, Aesculus chinensis, Agastache rugosa, **Alpha-lipoic acid**, Altemanthera philoxeroides, Andrographis paniculata, Arctium lappa, Aspalathus linearis, **Astragalus membranaceus**, Azadirachta indica, **Bee Propolis**, Betulinic acid, **Bifidobacterium logum**, Boswellia carterii, **Brazil nuts**, Buxus sempervirens (SPV-30), Calendula officinalis, Calophyllum cerasiferum, Calophyllum cordato, **Calophyllum lanigerum (calanolide)**, Carrisyn-rich Aloe vera, **Castanospermum australe**, Cayenne, Chamaesyce hyssopifolia, Co-enzyme Q10, Coix lachryma-jobi, **Colloidal minerals, especially Copper, Zinc and Manganese**, **Colloidal silver**, Colostrum, Cordia spinescens, Coriolus versicolor, Croton tiglium, Curcumin, Curry spices, Cynomorium songaricum, DHEA, DMG, DNCB, Echinaceae, Elderberry, Epimedium grandiflorum, Eucommia ulmoides, Euphorbia granulata, Esterized glutathione superoxide dismutases (Cu/Zn or Mn), Eupatorium buniifolium, **Flaxseed (complete lignan oil)**, Fomitella supine, Fructo-Oligo-Saccharides (FOS), Gamochaeta simplicaulis, Ganoderma lucidum, **Garlic**, Organic Germanium, Ginseng, **Glycyrrhizin**, Glycyrrhiza uralensis, **Green tea**, **Hydrogen Peroxide**, Hypoxis rooperi, Hyptis lantanifolia, **Hyssopus officinalis**, Inulin, Jatropha curcas, **Kelp**, **Green tea- Kombucha**, **Lactobacilli acidophilus**, casei & plantarum, L-Arginine L-Carnitine, **L-Cysteine**, L-Cystine, L-Glutamine, **L-Glutathione**, L-Lysine, **L-Methionine**, L-Ornithine, L-Threonine, L-Tryptophan, Lithospermum erythrorhizon, Lonicera japonica, **Lemon balm** (Melissa officinalis), **Licorice** (Glycyrrhiza glabra), Maitake mushroom, **Magnesium peroxide**, Marila laxiflora, Maytenus senegalensis, **Mentha piperata var crispa**, Methyl-Sulfonyl-Methane (MSM), Milk-thistle extract (Silymarin), Momordica charantia, **N-acetyl-cysteine (NAC)**, Ocimum basilicum cv 'cinnamon', Oleanolic acid, **Olive leaf extract**, **Oregano**, Ozone (stringent antioxidative enzyme preparation & extreme caution required), PADMA28, Papaverine alkaloids, Paprika, Perilla frutescens var crispa f. viridis, Phellinus rhabarbarinus, Phyllanthus myrtifolius, Phyllanthus sellowianus, Platanic acid, Pomolic acid, Proanthocyanidins, **Prunella vulgaris subsp asiatica**, Pumpkin seed oil, Quercetin, Rhizophora mucronata, Rodiola rosea, Rosemary, **Savory** (Satureja Montana), Scutellaria baicalensis (Baicalin), **Selenomethionine**, Shitake mushroom, **Beta-Sitosterols & Beta-Sterolin glycosides**, Super-Oxygenated Water, Staphage lysate, St Johns wort, Syzgium claviformum, Tetrapteris macrocarpa, Thiamine disulfide, Tofu, Trametes cubensis, Trichaptum perrotteti, Trichosanthes kirilowii, Tumeric, Urine, Viola yedoensis, Viscum album, **Whey protein**.

The above research has been condensed into a “how to” **“Immunological Optimization Protocol”**.

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