Alberta Reappraising AIDS Society

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David Crowe, President
Phone: +1-403-289-6609
Fax: +1-403-289-6658
Email:
David.Crowe@aras.ab.ca

Box 61037, Kensington Postal Outlet
Calgary, Alberta T2N 4S6
Canada

Office
Phone: +1-403-220-0129
Email: aras@aras.ab.ca
Web: aras.ab.ca

Kathleen Newell, Treasurer

Are Malnutrition and Oxidative Stress the Cause of gp41, gp120 and gp160 in Robert Gallo's HIV Isolate?

Beldeu Singh
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This page consists of three parts:

- Beldeu Singh's original article.
- A challenge from Deepak of AIDSMythExposed.com.
- Beldeu's response.

Original Article

Undernutrition can prevent optimal cellular function of cells while the lack of specific minerals and vitamins in the diet and depletion of antioxidants through excess free radicals generated by toxic drugs, drug abuse, smoking and alcohol abuse.

Malnutrition impairs the body's natural defenses and its natural defense mechanisms. When selenium intake is low, the production of selenoproteins, including glutathione that form the selenium based antioxidant enzymes falls and in those cases where the intake of vitamins from natural sources, the antioxidant defense biochemistry is weakened while their cellular function may be rendered below optimal. Over time, these may be impaired as oxidative stress and oxidative injury may alter the integrity of cell membranes and the metabolic pathways in cells while the ability to repair and rejuvenate is compromised.

Prolonged malnutrition may be accompanied by immunological deficiencies and abnormalities and it is natural to expect a fall in blood cell count of macrophages, red blood cells, natural killer (NK) cells and the total lymphocyte count is depressed. Any drug that generates free radicals in the body, such as heroin, cigarette tars, benzene (whether from petrol where it is now used as an additive to replace lead

or from cigarettes), chemo-medications, AZT etc., or heavy metal ions such as the mercury ion can produce oxidative stress on the Krebs cycle or mitochondrial metabolic activity or microsomes or the sodium pump and lower or inhibit energy production or mitosis or protein synthesis.

When white blood cells encounter a bacteria, they engulf it and move the bacteria deeper into the cell, and it fuses with a lysosome, forming a phago-lysosome. These contain hydrogen peroxide, active oxygen species (free radicals), peroxidase, lysozyme and hydrolytic enzymes. A sudden burst of free radicals towards the bacteria will kill it and it leads to digestion by enzymes, after which they are eliminated by exocytosis into the bloodstream.

The degradation of protein molecules takes place through a multienzymatic process that requires ATP from the energy pathway. Oxidative stress and injury to lysosomes could compromise the killing of bacteria and the ingestion of infective agents by polymorphs and macrophages.

When an infective agent enters the body, the antigen is picked up by a monocyte or macrophage and the antigen, which may be a protein molecule, is degraded and presented to both the B cells and T lymphocytes.

B cells form about 25% of lymphocytes in the blood. When the B cell is presented with an antigen, it is activated. It divides to increase the number its population and they differentiate to become plasma cells that produce large amounts of antibody specific to that antigen. When it has concluded its antibody producing function, it dies leaving only a small number of memory cells that can very quickly respond to future infections.

The antibody may bind to the antigen and neutralize it. If the antibody binds to the virus, it triggers the complement system which uses neutrophils and activated macrophages to identify those viruses to which the antigen has attached and to attack and eliminate the virus.

The body may produce T cells if the antigens, such as viruses, are presented directly to B cells and a cytotoxic response results in killing the virus or inactivating it. Additionally, there NK cells involved in a process that is not antigen specific. The role of NK cells is to eliminate abnormal cells, tumour cells and virus-infected cells. Hence, the NK cells are an important part of the constant surveillance of the immune system to protect itself from infections and abnormal cells that may be formed in the body.

Acute oxidative stress on B cells will not only cause their cell to fall but the amount of antibody they produce may also be lowered resulting in antibody deficiency. Simultaneous oxidative stress on the cell membranes of cells of the immune system may compromise their optimal and normal function and add to the immunodeficiency.

The mammalian immune system is a remarkable and complex system that evolved over 65 million years. A combination of specific immunity and general defense that includes killing by phagocytosis, killing of tumour and virus-infected cells by lysis, the use of macrophage NO as a cytotoxic agent to kill microbial cells and tumor cells and the use of free radicals to kill pathogens surrounded by white blood cells and specific neutralization of certain antibodies while keeping memory of previous antigen infection is that hallmark of the human immune system. The special mechanism of altering DNA encodes for multiple rearrangements of germline DNA in the T lymphocytes enables it to create a trillion different types of antibodies that allows for providing specific antibodies for the antigens that could possibly be produced by mutations of viruses. Hence it is difficult for viruses to target the immune system but immunotoxic drugs and narcotics abuse when there is immunodeficiency due to acute oxidative stress on it enables viral toxins to produce disease conditions rather easily. Natural selection favored the vast genetic diversity in its response to antigen challenges from mutating viruses.

In such a complex system, there is a great extent of coordination that involves communication between cells of different types. It can also communicate and transfer information to the brain and other centers

in the system about foreign bodies that have invaded the body and it has the ability to recognize cells of self and non-self cells that will be targeted for destruction. Oxidative damage can compromise or impair the amazing communication in the system and its role. Excess free radicals in the brain, as in the case of heroin addicts, can impair the pituitary and adrenal gland secretions producing endocrine complications. Excess free radicals in other parts of the brain could produce respiratory complications. Oxidative damage by excess nitric oxide could be the cause of impairment in the recognition of self and non-self by the white blood cells.

A number of drugs can weaken the immune system including AZT, chemo-drugs and barbiturates. A number of antiviral drugs are also immunosuppressive. This presents a catch 22 situation: When there is a viral infection, the immune system must be in optimal cellular function to reproduce cells and produce antibodies to kill the virus-infected cells but the antiviral drug suppresses it or weakens it. Naturally, prolonged use of immunosuppressive or immunotoxic drugs will lead to progressive destruction of the immune system. The administration of such drugs, in the long term, increases the rate of destruction of neutrophils and macrophages. This is called acquired neutropenia or acute phagocyte deficiency. In adults, „the risk of infection rises steeply once the neutrophil count falls below 0.5 x 109/L regardless of the cause" (Clinical Medicine, 4th Ed, WB Saunders, p 2) and when the monocyte count also begins to fall, caused by free radical damage, the immune system may become sufficiently impaired to allow opportunistic infections to take root and when the NK cell count falls its surveillance for cancer cells weakens along with its ability to destroy them. In such a state, it can be said that the person has acquired immune deficiency. If there is sufficient oxidative stress on the mitochondria resulting in mDNA depletion, chronic fatigue will be one of the symptoms.

In severe or chronic malnutrition there is detectable depression in both circulating immunity and antibody production showing a direct link between malnutrition and the effects of the accompanying oxidative stress on the immune system.

The liver contains many immunological cells that trap bacteria and other antigens. These are called Kupfer cells which are actually macrophages attached to the endothelium that phagocytocise and degrade antigens. Many drugs also impair liver function and may impair the immunological function of the liver.

Oxidative stress on the liver may not only impair its immunological function but also reduce its enzyme production leading to cirrhosis. Depletion of the antioxidant system in the liver cells slows down the glutathione biochemical pathways that metabolize (breakdown) drugs.

In the liver, enzymes are used to oxidize the drugs which are conjugated with glucuronide or sulphate and secreted through the bile and urine. The conjugation prevents their reabsorption in the kidney and bile duct. A large portion of the toxic drugs undergo conjugation with glucuronide and sulphate and the remaining portion is metabolized by microsomal enzymes to produce derivatives that generate free radicals and must be conjugated with glutathione. Prolonged use of toxic drugs depletes glutathione and upon depletion of glutathione, the toxicity accumulates and causes more damage to cells.

In persons with low intake of selenium, the glutathione levels are low. Glutathione is an antioxidant that effectively neutralizes free radicals in mitochondria by donating an electron to the free radicals and oxygen reactive species and neutralizes them. The depletion of glutathione by microsomal derivatives during the breakdown process only means the exponential increase of free radicals. When glutathione levels in cells fall below 50%, it is not effective in scavenging free radicals to maintain optimal cellular function and signs of aging and degenerative disease begin to appear. When glutathione level in cells, falls even further, oxidative damage to organs and the immune system progresses more rapidly. When glutathione level in cells fall below 80%, the cell dies from acute oxidative stress.

Glutathione is an antioxidant. It readily donates an electron to free radicals in mitochondria. The spent glutathione and coenzyme Q10 can be recharged by natural vitamin A and C, alpha lipoic acid and

anthocyanins. Selenium, from natural sources is used the body to produce glutathione. The selenium ion is itself an antioxidant and can be recharged by possibly all the vitamins, anthocyanins and alpha lipoic acid. When glutathione begins to deplete and there is a corresponding increase in the free radical population, the depletion rate of natural vitamins in the body also increases as more and more of the vitamins are spent in scavenging the increasing free radical populations which means their role to recharge glutathione is diminished and that aggravates oxidative stress and oxidative damage thereby accelerating the development of disease states in the body.

The recharge process in the antioxidant network that makes the natural vitamins and anthocyanins work as an integral part of the natural antioxidant network, emphasizes the importance of intake of natural antioxidants through the diet or supplements for optimal cellular function and emphasizes their combined use in treating disease conditions fostered by malnutrition and oxidative stress or oxidative damage in order to restore optimal cellular function and restore healthy functioning of the body.

An oxidative stress situation faced by cells in the immune system compromises their role in immune function and immune response and the cell counts fall leading to immunodeficiency. Macrophages under oxidative stress caused by free radicals and/or in chronic malnutrition or in people recovering from oxidative stress caused by influenza viral toxins or parasitic infections such as malaria where the white blood cells destroy the plasmodium parasite by free radicals (hydroxyl ions), do not function at optimal levels to ingest and degrade antigen proteins.

Most of the drugs used in the treatment of malaria patients, like chemo-drugs are toxic. They generate large amounts of hydroxyl ions that kill the parasite in the bloodstream and these highly reactive free radicals also create oxidative stress on the cells of the immune system. Prolonged use of drugs that generate the hydroxyl ions in the body will result in a fall in cell counts and later depress the monocyte count as well. Prolonged exposure to hydroxyl ions will ravage the immune system and lead to AIDS and the person becomes at risk to opportunistic infections.

Lymphocytes form about 50% of circulating white blood cells. Cellular immunity is important and therefore about 80% of circulating lymphocytes are T lymphocytes. Under oxidative stress, especially in malnourished people, the lymphocute count drops and a low count is also found in people with chronic infections, TB, toxoplasmosis, in viral infections and in some cancers. Toxic medication in people with oxidative stress is likely to depress the cell count further while elevating the risk of mitochondrial disease or causing the cell to transform from aerobic metabolism to one dependent on anaerobic metabolism. Sufficient oxidative stress that reduces the availability of energy (ATP) for the sodium pump can cause its failure resulting from sodium retention in cells producing oedema and later on cell death by apoptosis (by the release of enzymes in the lysosomes). Heart failures can also be caused by free radical stress on the sodium pump. It is now clear that oxidants play a major role in brain damage in cerebrovascular diseases (Pak H. Chan, Departments of Neurological Surgery and Neurology, University of California, School of Medicine, San Francisco; Role of Oxidants in Ischemic Brain Damage, Stroke,1996;27:1124-1129.) There is evidence to demonstrate the role of oxidative and nitrosative stress, leading to peroxynitrite formation in renal damage (Eisei Noiri1 et al, Oxidative and nitrosative stress in acute renal ischemia, Am J Physiol Renal Physiol 281: F948-F957, 2001.)

Oxidative stress in the bone marrow by hydroxyl free radicals can damage the DNA and cause disruptions to cell metabolic pathways or hydrogen peroxide inactivation of enzymes involved in Krebs cycle and lead to bone marrow failures and leukemias while oxidative stress on the immune system is the underlying cause of abnormal B or T cells and lymphomas. On the other hand, lupus and multiple organ failures as in multiple scelerosis may be due to excess nitric oxide caused by dysfunction of the endothelium of the major arteries under prolonged oxidative stress.

Abnormalities in white blood cells may be due to its special cytotoxic role against invading bacteria. The phagocyte undergoes anaerobic glycolysis producing lactic acid and a pH of about 4.0, myeloperoxidase, singlet oxygen, H2O2, and other factors that are toxic to bacteria. Large amounts of glutathione and vitamin C(above 85%) enable white blood cells (lymphocytes such as T cells, B cells,

and natural killer cells() to function at optimal levels and reproduce in order to make antibodies. If the glutathione and vitamin C in these cells falls (ie below 50%), there is insufficient antioxidant (free radical scavenging) activity to revert the anaerobic metabolic pathway to aerobic metabolism and it is at a higher risk of becoming abnormal when the immune system is under acute oxidative stress especially in malnourished people. Oxidative damage in young cells (blasts) prevent them from maturing. Hence, oxidative stress to the bone marrow can be devastating as it can lead to the development of leukemias. Also survival of the white blood cell that is low in antioxidant enzymes and vitamin C after phagocytosis is low.

Coenzyme Q10, like glutathione, is made naturally by the human body. Coenzyme Q10 is used by cells of the body in aerobic respiration or aerobic metabolism. Through this process, energy for cell growth and maintenance is created inside cells in the mitochondria. It helps cells to produce energy and it acts as an antioxidant. Coenzyme Q10 has shown an ability to stimulate the immune system and to protect the heart from damage caused by certain chemotherapy drugs. Low blood levels of coenzyme Q10 have been detected in patients with some types of cancer (see Coenzyme Q10 (PDQ®),Last Modified: 01/11/2005, Health Professional Version; National Cancer Institute, US National Institutes of Health, www.cancer.gov). Depletion of mitochondrial enzymes by oxidative stress can lead to a broad range of disease conditions, including some cancers. Oxidative stress or a chronic lack of minerals that disrupt the Krebs cycle or energy output in mitochodria is likely to result in a transformation that switches to anaerobic metabolism from aerobic metabolism and that leads to the formation of cancer cells.

It would be a sound hypothesis in clinical biochemistry to propose an antioxidant stress, whether temporary or short term depending on the oxidative state that has produced the disease condition, in order to attempt to restore optimal cellular function or to attempt to reverse the anaerobic metabolism in cancer cells to aerobic metabolism. Apart from aconitase, other enzymes in the Krebs cycle may also suffer inactivation through oxidative stress and it may shut down the aerobic metabolism and the cell may switch to anaerobic metabolism. It is therefore useful to research into potential mechanisms of enzyme inactivation and reactivation which can then be incorporated into processes to regain optimal cellular function based on aerobic metabolism and arrest or slow down or reverse disease progression and reverse the damage caused by oxidative stress. Excess free radicals and radicals cause metabolic enzyme inactivation and antioxidant stress may activate them and improve biological activity in cells. Also, in the absence of minerals in organic form hinders the metabolic pathways and results in a decline in cell function.

However, ROS and free radicals have a very useful role to play in the body. The super oxide radical, hydrogen peroxide and the hydroxyl radical (HO·) can produce additional secondary reactive oxygen metabolites, which are products of lipid peroxidation (hydroperoxides, alkoxyl and peroxyl radicals, epoxides, or aldehydes) and peroxynitrite (ONOO-), a product of the reaction between superoxide with nitrogen monoxide (Beckman, J.S. and Koppenol, W.H. (1996). Nitric oxide, superoxide, and peroxynitrite: the good, the bad, and ugly. Am. J. Physiol. 271: C1424-C1437: Sigler, K., Chaloupka, J., Brozmanova, J., Stadler, N. and Hofer, M. (1999). Oxidative stress in microorganisms - I. Microbial vs higher cells - damage and defenses in relation to cell aging and death. Folia Microbiol. 44: 587-624). Oxidative or nitrosative stress depolarizes the plasma membranes of some microorganisms, indicating plasma-membrane perturbation by reactive oxygen species and nitrsative stress that may lead to cytotoxicity and eventually to cell death by necrotic or apoptotic pathways, (Lloyd et al (2000), The microaerophilic flagellate Giardia intestinalis: oxygen and its reaction products collapse membrane potential and cause cytotoxicity. Microbiology 146, 3109–3118; Lloyd et al,(2003b), Respiratory oscillations in yeast: mitochondrial reactive oxygen species, apoptosis and time; a hypothesis. FEMS Yeast Res 3, 333–339: D. Lloyd et al, The plasma membrane of microaerophilic protists: oxidative and nitrosative stress, Microbiology 150 (2004), 1183-1190, DOI 10.1099). The microbicidal capability of macrophages relates to reactive oxygen and nitrogen intermediates production, with nitric oxide (NO) being one of the most potent cytotoxic agents (MacMicking, J., Xie, Q.W. and Nathan, C. (1997). Nitric oxide and macrophage function. Annu. Rev. Immunol. 15:

323-350). Macrophages phagocytize invading bacteria and kill them by a number of mechanisms, including production of antimicrobial peptides, lysosomal enzymes, reactive oxygen species (ROS), and reactive nitrogen species (RNS), (Tânia M. Stevanin et al, Flavohemoglobin Hmp Protects Salmonella enterica Serovar Typhimurium from Nitric Oxide-Related Killing by Human Macrophages, Infection and Immunity, August 2002, p. 4399-4405, Vol. 70, No. 8). Neutrophils and mavrophages use the highly reactive free radicals (superoxide, hydrogen peroxide, hydroxyl) to kill bacteria. Additionally, the neutrophil also kills engulfed pathogens by using the enzyme myeloperoxidase which catalyzes the reaction of hydrogen peroxide (made from superoxide anions) with chloride ions to produce the microbicidal hypochlorite ion. White blood cells may also form a large number of superoxide (ROS - an oxygen molecule with an extra unpaired electron) to kill the engulfed bacteria and possibly inactivate viruses.

These same reactive species that are used by white blood cells to kill bacteria in a controlled manner can damage lipids, proteins, and nucleic acids and can cause abnormal function or mutations in DNA, which contribute to tumor formation in mamalian tissues and organs.

Oxidative stress precedes the cytotoxic death of microorganisms and since it also precedes cell and tissue damage (called oxidative damage) in higher organisms, pharmaceutical research has branched out to identify and patent compounds that reduce, inhibit or prevent oxidative stress or oxidative damage that will be used to treat oxidative stress-related disorders and diseases.

Consistent with the free radical theory of AIDS, there is a broad spectrum of symptoms and illnesses associated with AIDS, including neuropathies, endocrine complications, diarrhea, accelerated aging, chronic fatigue, respiratory complications and cardiac complications. Sone of the symptoms observed in AIDS patients are muscle wasting, chronic tiredness and mDNA depletion. This means that the genetic material in motochondria (the power house of the cell), is destroyed or depleted or its multiplication is inhibited and the energy output drops and the affected person feels muscle pains and fatigue. As the oxidative damage progresses under toxic medication especially in malnourished people, CD4 count and other cell counts fall further. The immunotoxicity and the resultant immunodeficiency allows the development of opportunistic infections, including cerebral abscess, TB, pneumonia, viral infections such as cytomegalovirus, herpes, fungal infections, Kaposi’s sarcoma, lymphomas and chronic inflammations.

Depletion of CD4 lymphocytes has been found to be a common event that appears to be a non-specific response by the body to any type of physical or psychological stress and is often found to be below that required to diagnose AIDS in someone who is HIV-positive (Carney, 1981; des Jarlais, 1987; Feeney, 1995; Kennedy, 1988; Kiecolt-Glaser, 1984, 1991, 1992; Pariante, 1997; Williams, 1983) as would be expected in oxidative damage.

A general conclusion is that drug abuse and toxic ‘medication’ that generate free radicals in the body interfere with the Krebs cycle and mitochondrial activity or cause damage to DNA in cells or cause excess nitric oxide or lower its secretion in certain tissues and in all these cases there is disruption in cellular function or lowering of cellular function below its optimal levels. In malnourished people, oxidative stress leads to the development of disease states more easily.

Over 75 years ago, Otto Warburg was awarded two Nobel prizes for his theories that cancer is caused by weakened cell respiration due to lack of oxygen at the cellular level. A normal healthy cell burns oxygen and glucose (blood sugar) for energy and releases carbon dioxide and water. The normal and healthy cell functions aerobically - in the presence of sufficient oxygen. Anaerobic diseases (cancer, Epstein Barr, herpes, mono, genital warts, etc.) cannot naturally flourish in this oxygen rich environment in the cell.

An unhealthy or diseased cell lacks sufficient oxygen but will continue to produce energy by using mainly glucose in a different process. This energy burning process (sugar fermentation) releases mainly lactic acid and carbon monoxide instead of carbon- dioxide. Under anaerobic growth

conditions, pyruvate is primarily reduced to lactic acid, while it is oxidized to acetate and CO2 under aerobic growth conditions. This is a very inefficient pathway and a severe drain on the body. The cancer cell functions anaerobically – without any oxygen present and without using any oxygen.

Oxidative stress is a crtical mechanism in the progression of AIDS, symptoms like chronic fatigue, abonormal function of cells and cancer formation.

As long ago as 1983, it was proposed that oxidative mechanisms are of critical significance in the genesis of AIDS (acquired immune deficiency syndrome). A prediction of this hypothesis was that the mechanisms responsible for AIDS could be reversed by the administration of reducing agents, especially those containing sulphydryl groups (SH groups). The discovery of HIV resulted in a broadening of this hypothesis in that it considered oxidative stress as a principal mechanism in both the development of AIDS and expression of HIV (Papadopulos-Eleopulos, 1988; Papadopulos-Eleopulos et al., 1989), (see Oxidative Stress, HIV and AIDS, E. Papadopulos-Eleopulos, V.F. Turner, and J.M. Papadimitriou, Res. Immunol. 1992, 143, 145-148).

Peter H. Duesberg (Ph.D. Professor of Molecular and Cell Biology at the University of California, Berkeley. Based on his research and experience with retroviruses, Duesberg challenged the virus-AIDS hypothesis in the pages of such journals as Cancer Research, Lancet, Proceedings of the National Academy of Sciences, Science, Nature, Journal of AIDS, AIDS Forschung, Biomedicine and Pharmacotherapeutics, New England Journal of Medicine and Research in Immunology. He has instead proposed the hypothesis that the various American/European AIDS diseases are brought on by the long-term consumption of recreational drugs and/or AZT itself, which is prescribed to prevent or treat AIDS.

The common denominator in the long term use of recreational drugs and AZT is their capacity to generate free radicals in the body and cause oxidative damage which presetnts itself in a broad range of symptoms and illnesses associated with immune deficiencies.

AZT has been found in five studies to be toxic to T-cells, the very cells whose absence is blamed on HIV. This is not surprising since T-cells are produced in the bone marrow and all the other cells produced there are depleted by AZT. AZT may cause an initial increase in T-cells as the body's immune system responds to the toxic stress being placed on it by AZT, but in relatively short time the T-cells, neutrophils, and other immune system cells begin to decline. AZT generates a large number of hydroxyl ions that cause oxidative stress and oxidative damage which explains why cell counts decline and opportunistic infections appear in people taking AZT.

A study on cardiovascular toxicology reports "AZT treatment increases superoxide (free radical) production" and "the effects of AZT on endothelium-dependent relaxation are eliminated by pretreatment with a free radical scavenger" (ie an anti-oxidant).

There is evidence of alcoholic toxicity being mediated via the generation of free radical species. Ethanol also induces free radical formation that damages mitochondria and alters metabolism in mitochondria. The consumption of alcohol results in the formation of two very toxic compounds; acetaldehyde and malondialhyde which generate massive amounts of free radicals throughout the body. This type of free radical damage is both to the cell wall and the mitochondria.

The oxidative damage initiated by AZT also produces a broad range of symptoms and illnesses. "It produces excruciating headaches; severe nausea; muscular pain; wasting of the muscles; damage to kidneys and nerves; excruciating pains in the legs; encephalitis; severe anemia requiring transfusions to stay alive; lymphoma (cancer); cancer in 49% of cases, versus 2% incidence in non AZT group; liver damage; nail dyschromia (fingernails turn black); insomnia; impotence; dementia; mania; ataxia (failure of muscular coordination); seizures; alopecia (hair falls out). It is a fairly well established fact that AZT was designed to kill the bone marrow. It causes neutropenia or leukopenia (loss of white blood cells) or bone marrow aplasia and bone marrow toxicity. White blood cells are the basis of the

immune system. T cells, granulocytes, those are all parts of the immune system. You kill those with AZT and the immune system is gone," Harvey Bialy, Research editor Bio/Technology Science Journal.

There are also very well documented investigations showing that AZT has carcinogenic properties with respect to fast growing human and animal immune and other cells. In humans, AZT magnifies the risk of lymphomas by 50. AZT has also been confirmed to be carcinogenic in mice. AZT can also cause anemia, lymphocytopenia, hepatitis, pancreatitis, myositis, muscle atrophy, wasting disease, dementia, lactic acidosis, severe hepatomegalia with steatosis, vasculitis, and it prevents mitochondrial DNA synthesis. Prolonged use of AZT decreases white blood cells by killing young CD4 lymphocites. It causes anemia, vomiting, lactic acidosis, fatigue, muscles wasting and lymphocytopenia and it stimulates leukemias – all the classic symptoms of AIDS! (The Role Of Selenium In Reversing AIDS; Detoxamin, TOX NEWS) and coincides with the broad range of symptoms caused by oxidative damage.

Early AIDS coincided with the cumulative effects of unprecedented, intense use of volatile nitrite (poppers) as an aphrodisiac marketed almost exclusively to homosexuals. The vast majority of African "AIDS patients" tested HIV negative. AIDS occurs in the absence of "HIV" and a new medical definition (Idiopathic CD4+ T-cell lymphocytopenia) was created but recreational drugs (heroin, poppers, crystal met, ecstasy, cocaine) also reduce CD4 cell numbers while anti-HIV drugs, including protease inhibitors, destroy T-cells. Additionally, anti-HIV drugs inhibit human enzymes and septrin (also called Septra, Bactrim, Co-trimoxazole) and anti- HIV drugs destroy mitochondria in cells. Since anti-HIV drugs cause AIDS-defining diseases, it is not surprising to note reports of HIV positive patients recovering after they stop taking drugs (The Role Of Selenium In Reversing AIDS; Detoxamin, TOX NEWS).

Unfortunately, "practically every single medicament from the following groups have been found to have immunotoxic properties: antibiotics; antifungal, antiviral, and antiparasitic agents; tranquilizers, antiepileptics, antiparkinson, and anesthetics; antihypertensive, anti-anginal, and antiarrhythmic drugs; gastrointestinal medications; antidiabetics, antithyroid drugs, and sex hormones including oral contraceptives; antiallergics; bronchodilating agents; anticoagulants, drugs acting on fibrinolysis, blood expanders, clotting factors, and inhibitors of platelet aggregation; non?steroidal anti? inflammatory drugs, corticosteroids, antirheumatismal, and anti gout drugs; and immunodepressive and immunomodulating drugs such as antitumoral drugs and medications to avoid graft rejection," (Roberto Giraldo MD: Dale MM, Foreman JC & Fan TD Eds. Texbook of Immunopharmacology. Third Edition. Blackwell Scientific Publications, Oxford , 1994; Dean JH, Luster MI, Munson AE & Kimber I Eds. Immunotoxicology and Immunopharmacology. Second Edition. Raven Press, New York , 1994; Descotes J. Immunotoxicology of Drugs and Chemicals, Second Updated Edition. Elsevier, Amsterdam , 1988). So, almost everyone has been exposed to pharmaceutical drug induced oxidative stress.

Though, as early as 1983-88, it was proposed that oxidative damage progressed into developing the AIDS condition, it was overtaken by a media scare created at an official press conference. HIV was announced to be the "probable cause" of AIDS by Robert Gallo at a government press conference. It was popularized as the cause of AIDS because HIV is found in virtually all AIDS patients (90%). And tests were created to test the HIV antibody in "HIV infected persons".

At that time, no one questioned the Gallo Isolate but it became apparent to scientists and virologists that Dr. Gallo had not followed nor complied with the gold standard and the established rules in isolating, purifying and later reinfecting cells to show the specific infecting agent produced the condition or disease. Proof that the particles are infectious, that is when pure particles are introduced into an uninfected culture or animal, the identical particle is obtained as shown by repeating steps in the established procedure. Analysis of the particles' proteins and RNA and proof that these are unique is of critical importance. But, there is no validation of the Gallo Isolate and the viral infective theory of AIDS.

HIV as the pathogenic factor in AIDS cannot explain the hundreds of thousands of healthy people who tested HIV-positive and live as long as HIV-negative people and there are many people with non-HIV AIDS who respond to proper nutrition. That alone is sufficient to debunk the HIV viral cause of AIDS.

No particle of 'HIV' has ever been obtained pure, free of contaminants; nor has a complete piece of 'HIV' RNA (or the transcribed DNA) ever been proved to exist. Moreover, Dr David Ho admits that 99.8 per cent of putative 'HIV particles' are non-infectious; the remaining 0.2 per cent of 'viral particles', being defective, are not capable of replication. The phenomena collectively known as 'HIV' are non-specific: reverse transcriptase is non-specific; PCR is non-specific; Viral Load is non-specific that should never have been classed as a virus.

Dr John Papadimitriou states that the proper controls have never been done: "They have not proven that they actually have detected a unique, exogenous retrovirus. The critical data to support that idea have not been presented. You have to be absolutely certain that what you have detected is unique and exogenous, and a single molecular species...('Aids: The failure of contemporary science', Neville Hodgkinson, Fourth Estate, 1996, page 375). Since 1989, detection of a 24,000 molecular weight protein (p24) in cell cultures, (T cells from persons presumed to be infected), or co-cultures, (of T cells from persons presumed to be infected, with T cells from normal individuals), has been used to quantify HIV in cells, "cellular viremia" (Masquelier et al., 1992). Detection of p24 in cultures of T cells from normal individuals with plasma from those presumed to be infected has been used to quantify HIV in plasma, "plasma viremia" (Coombs et al., 1989; Ho et al., 1989; Clark et al., 1991). There are many reasons why p24 cannot be used to quantitate or even detect the presence of "HIV infectious particles". There is ample evidence that the p24 protein is not HIV specific (Papadopulos-Eleopulos et al., 1993a).

Gallo claimed that the interaction of gp41 with antibodies found in AIDS patient sera is proof that gp41 is coded by the "HIV genome", and that both gp41 and the antibodies are specific to a retrovirus. In contrast, when Gallo and his colleagues reported the presence in humans of antibodies, in 1981, to what he now calls the first human retrovirus, HTLV-I, they described the finding of antibodies to a "major internal structural protein (p24) of HTLVCR" and claimed that such antibodies were "specifically directed at HTLVCR proteins and not at cell-specific determinants. This means that the immunological reactions are not those reported in human sera against animal virus glycoproteins which, lacking virus specificity, are directed against the carbohydrate residues of the glycoprotein" (see VirusMyth;Eleni Papadopulos-Eleopulos, THE ISOLATION OF HIV – HAS IT REALLY BEEN ACHIEVED? THE CASE AGAINST, Continuum Vol.4 No.3 Sept./Oct. 1996).

In 1980, two research groups, one from the Laboratory of Cellular and Molecular Biology, National Cancer Institute and the other from the Laboratory of Viral Oncology, Memorial Sloan-Kettering Cancer Center, using the "viral glycoproteins", found that the antibodies present in human sera which reacted with these proteins were "directed against carbohydrate structures" and concluded that "The results are consistent with the idea that the antibodies in question are elicited as a result of exposure to many natural substances possessing widely cross-reacting antigens and are not a result of widespread infection of man with replication competent oncoviruses".

Lacking specificity that viral antigens display, the finding by other scientists that these are nothing more than viral glycoproteins that are elicited as a response to many natural substances or the proteins that are used in the 'HIV' test are merely the biological outcome of stressed white blood cells used in the lab and in 'Bio/Technology', June 1993, 'Aids' analyst, Dr Eleni Papadopulos-Eleopulos exposed the non-specificity and unreliability of the 'HIV' 'antibody test'. Obviously, if that is true one would expect a non-specific molecule to a virus to be a common factor in other conditions especially in people recovering from illnesses or infections with associated oxidative stress and in people with chronic malnutrition and the so called viral glycoproteins in the Gallo Isolate are degraded molecules of white blood cells functioning below optimal levels commonly found under oxidative stress and chronic malnutrition.

Christine Johnson, a researcher and author, compiled a long list of conditions documented in scientific literature to cause positive test results on HIV tests, and provides references for each condition. He cites 63 research papers by over 100 scientists. The list - Anti-carbohydrate antibodies; Naturally-occurring antibodies; Passive immunization: receipt of gamma globulin or immune globulin (as prophylaxis against infection which contains antibodies); Leprosy; Tuberculosis; Mycobacterium avium; Systemic lupus erythematosus; Renal (kidney) failure; Hemodialysis/renal failure; Alpha interferon therapy in hemodialysis patients; flu vaccination; Herpes simplex I; Herpes simplex II; upper respiratory tract infection (cold or flu); Recent viral infection or exposure to viral vaccines; Pregnancy in multiparous women; Malaria; High levels of circulating immune complexes; Hypergammaglobulinemia (high levels of antibodies); False positives on other tests, including RPR (rapid plasma reagent) test for syphilis; Rheumatoid arthritis; Hepatitis B vaccination; Tetanus vaccination; Organ transplantation; Renal transplantation; Anti-lymphocyte antibodies; Anti-collagen antibodies (found in gay men, haemophiliacs, Africans of both sexes and people with leprosy); Serum-positive for rheumatoid factor, antinuclear antibody (both found in rheumatoid arthritis and other autoantibodies); Autoimmune diseases; Systemic lupus erythematosus, scleroderma, connective tissue disease, dermatomyositis Acute viral infections, DNA viral infections; Malignant neoplasms (cancers); alcoholic hepatitis/alcoholic liver disease; Primary sclerosing cholangitis; Hepatitis; “Sticky” blood (in Africans); Antibodies with a high affinity for polystyrene (used in the test kits); Blood transfusions, multiple blood transfusions; Multiple myeloma; HLA antibodies (to Class I and II leukocyte antigens); Anti-smooth muscle antibody; Anti-parietal cell antibody; Anti-hepatitis A IgM (antibody); Anti-Hbc IgM; Administration of human immunoglobulin preparations pooled before 1985; Haemophilia; Haematologic malignant disorders/lymphoma; Primary biliary cirrhosis; Stevens-Johnson syndrome; Q-fever with associated hepatitis; Heat-treated specimens; Lipemic serum (blood with high levels of fat or lipids); Haemolyzed serum (blood where haemoglobin is separated from the red cells); Hyperbilirubinemia; Globulins produced during polyclonal gammopathies (which are seen in AIDS risk groups); Healthy individuals as a result of poorly-understood cross-reactions; Normal human ribonucleoproteins; Other retroviruses; Anti-mitochondrial antibodies; Anti-nuclear antibodies; Anti-microsomal antibodies; T-cell leukocyte antigen antibodies; Proteins on the filter paper ; Epstein-Barr virus; Visceral leishmaniasis and Receptive anal sex.

These “antibodies”, lack virus specificity and are commonly found in many people with different conditions as well as healthy individuals and during pregnancy. It is found in conditions common in underprivileged and impoverished communities that are known to cause false positive results such as tuberculosis, malaria, hepatitis and leprosy. False-positive ELISA [antibody] test results can be caused by alloantibodies resulting from transfusions, transplantation, or pregnancy, autoimmune disorders, malignancies and alcoholic liver disease. The common factor in impoverished communities and these disease states or pregnancy is oxidative stress.

The “HIV glycoproteins”, gp160, gp120 and gp41 are not viral but some ubiquitous cellular protein or macrophage polymer debris.

Ethanol-exposed macrophage exhibited impaired phagocytosis and increased apoptosis in experiments. But glutathione supplementation during and after ethanol exposure improved fetal macrophage function and viability. A growing body of clinical and experimental evidence has demonstrated that the chronic oxidative stress of alcohol exposure decreases the availability of the antioxidant GSH in the adult lung and independently increases the risk and severity of acute respiratory distress syndrome. The increased macrophage apoptosis demonstrated after chronic in utero ethanol parallels the oxidant-induced injury and apoptosis described in other organs models of fetal alcohol syndrome, such as the developing brain and liver (see Theresa et al, Fetal Alcohol Exposure Impairs Alveolar Macrophage Function via Decreased Glutathione Availability, Pediatric Research 57:76-81 (2005). Such studies show that oxidative stress caused by alcohol metabolites can lead to ethanol-induced dysfunction of macrophages and increased apoptosis. Ethanol-induced decreases in glutathione availability is the key link in ethanol toxicity to macrophage function and viability.

There is more evidence of macrophage impaired function due to oxidative stress and increase in energy production from anaerobic metabolism when under oxidative stress.

The process of phagocytosis requires the polymerization of actin in a directional fashion to surround and internalize the target. Actin polymerization is dependent on adequate supplies of ATP. Exposure to hyperoxia can impair the activity of cellular enzymes important in oxidative metabolism, which could lead to increased anaerobic glycolysis, depletion of glucose in the cell growth medium, and subsequent depletion of ATP. However, ATP levels in RAW 264.7 cells cultured in hyperoxia were not reduced. Glucose content in the cell growth medium was higher in the hyperoxic cultures, likely as a result of inhibition of cell proliferation by hyperoxia. Examination of the actin cytoskeleton of RAW 264.7 cells cultured in hyperoxia, by immunofluorescence microscopy, revealed marked changes, including an increase in the degree of actin polymerization, loss of cortical actin, and the formation of prominent stress fibers and actin aggregates (see Philip J et al, Hyperoxia Impairs Antibacterial Function of Macrophages Through Effects on Actin, American Journal of Respiratory Cell and Molecular Biology. Vol. 28, pp. 443-450, 2003).

Quantitative Western blotting revealed that the amount of actin in RAW 264.7 cells exposed to hyperoxia (ROS) was increased compared with normoxic controls, implying that *de novo* synthesis of actin occurred, perhaps secondary to the depletion of actin monomers. Exposure of RAW 264.7 macrophages to JP, a cell permeant analog of phalloidin that increases and stabilizes polymerized actin in living cells, reduced the ability of RAW 264.7 macrophages to phagocytose fluorescent Klebsiella by 50%. This indicates that increased actin polymerization is a potential mechanism explaining impairment of phagocytosis by oxidative stress and since AIDS is a condition caused by excess free radicals (in malnourished people) (see Philip J et al, Hyperoxia Impairs Antibacterial Function of Macrophages Through Effects on Actin, American Journal of Respiratory Cell and Molecular Biology. Vol. 28, pp. 443-450, 2003).

This clearly proves that oxidative stress on macrophages leads to increased actin polymerisation and formation of prominent stress fibers and actin aggregates which could occur in people recovering from malaria, influenza or in people sufferring from chronic fatigue due to mitochodrial oxidative stress or ethanol toxicity or drug induced oxidative stress and even in people with some forms of cancers including leukemia and since chemo-drugs, barbiturates, antiviral drugs and AZT increase oxidative stress, there is increased actin polymerization and formatiom of prominent stress fibres and actin aggregates. And that amounts to proof of increase of oxidative stress and not the presence or activity of a fictitious HIV virus.

Interestingly, the cell line most often used in AIDS research is the leukaemic cell line H9. It is known that H9 is a clone of HUT78, which was derived from a patient with adult T-cell leukaemia. The proteins considered to represent HIV antigens are obtained from mitogenically stimulated cultures in which tissues from AIDS patients are co-cultured with cells derived from non-AIDS patients-usually established leukaemic cell lines. Following the detection of the enzyme reverse transcriptase (RT) in the cultures, the supernatant, and more often the cell lysates, are spun in density gradients. Material which bands at 1.16 gm/ml is considered to represent “pure HIV” and consequently the proteins found at that density are considered to be HIV antigens. The immunogenic HIV proteins are thought to be coded by three genes, namely gag, pol and env. The gag gene codes a precursor p53/55, which is then cleaved to p24/25 and p17/18. The pol gene codes for p31/32, and the env gene codes the precursor protein p160 which is cleaved to p120 and p41/p45, (Ratner, L., Haseltine, W., Patarca, R.P. et al. 1985. Complete nucleotide sequence of the AIDS virus, HTLV-III. Nature 313:277-284; cf, Eleni Papadopulos-Eleopulos et al, Is a Positive Western Blot Proof of HIV Infection? Bio/Technology Vol.11 June 1993).

Actin is an ubiquitous protein which is found in all cells as well as bacteria and several viruses. Well known retroviruses such as the mouse mammary tumour virus and Rous sarcoma virus have also been shown to contain actin of cellular origin and it has been postulated that this protein plays a key role in both retroviral assembly and budding. It is also known that oxidation of cellular sulphydryl groups, as

is the case in AIDS patients, is correlated with assembly of polymerised actin, and that the level of actin antibody binding to cells is determined by the physiological state of the cells. For this reason actin antibody binding to cells has been proposed "as a sensitive marker for activated lymphocytes"(ref; Eleni Papadopulos-Eleopulos et al, Is a Positive Western Blot Proof of HIV Infection? Bio/Technology Vol.11 June 1993) but it is nothing more than the polymer actin and its aggregates produced by cells or macrophages under oxidative stress, cleaving at different points to show up as p160, p120, p80, p45, p41 and p24. It has been shown that a precursor p53/55, which is then cleaved to p24/25 and p17/18 and the precursor protein p160 which is cleaved to p120 and p41/p45 (Ratner, L., Haseltine, W., Patarca, R.P. et al. 1985. Complete nucleotide sequence of the AIDS virus, HTLV-III. Nature 313:277-284). These polymers being not the normal biomolecules produced during healthy metabolism and healthy cellular function, the body recognizes them as non-self and produced auto-antibodies called actin auto-antibodies.

Many scientists have reported anti-actin autobodies.(see,15 years of AIDS; Hassig A et al, The continuous failure in the prevention and treatment of AIDS is rooted in the misinterpretation of an inflammatory autoimmune process as a lethal, viral venereal disease First published in Continuum Magazine, London). JOHNSON et al., in 1965, were the first to report on anti-actin-autoantibodies. They described autoantibodies directed against smooth muscle cells. In 1994 BERMAS et al. showed that both sera from patients with lupus erythematosus and from mice suffering from the same illness react with glycoprotein 120 and peptides of the postulated HIV-1 envelope. They further proved that control sera of healthy individuals and patients with other autoimmune diseases contain small amounts of the same autoantibodies. Last but not least, they showed that autoantibodies reacting with glycoprotein 120 do not possess antinuclear specificity (ref; Eleni Papadopulos-Eleopulos et al, Is a Positive Western Blot Proof of HIV Infection? Bio/Technology Vol.11 June 1993).

The serological diagnosis of HIV infection is usually made on the basis of the detection of circulating antibodies specific for viral antigens gp41, gp120 and gp160. A confirmed positive test (i.e. one or two ELISA tests, followed by a Western Blot) indicates that a person has been exposed to the virus and has mounted an immunologic response (serum antibodies). gp41, gp120 and gp160 but the evidence points to the fact that the claim that such antibodies were "specifically directed at HTLVCR proteins" is false. It is consistent with the observation supported by scientific tests the viral antigens in the Gallo Isolate are actin polymers produced by cells or white blood cells in oxidative stress and consistent with the free radical theory of AIDS that oxidative stress produces a broad range of symptoms and illnesses and that includes suppression of the immune system or immunodeficiency.

The Challenge…

In this article, Beldeu Singh seems to believe that the protein actin codes for 'HIV' core protein p24. It is not so, it 'could' be another ubiquitous cellular protein myosin which has two weights, 24 and 18 Kilo-daltons.

Beldeu's Response

The research certainly points in the direction of a "ubiquitous cellular protein" and specifically to the polymer produced by cells and white blood cells under oxidative stress. Oxidative stress is the key in the AIDS condition and if the cellular protein myosin which has two weights, 24 and 18 Kilo-daltons, is to be included as one of the possible proteins in the Gallo isolate, it logically must be one that is produced under oxidative stress which the body recognizes as non-self and responds by producing antibodies against it.

It would be interesting to research that possibility for myosin. In any case, it is to be noted, it proves that there is no HIV-virus that targets and ranges the immune system but that oxidative injury to the cell wall would allow viruses to enter the cell and destroy it.

Currently, as the research stands, it leads to the conclusion that the ubiquitous proteins produced by white blood cells under oxidative stress, against which the body produces antibodies, can be transmitted through the semen and "infect" the woman. They pass through the vaginal walls and into her blood stream. Once in her bloodstream, her body recognizes them as non-self and the body responds by producing antibodies which may be picked up by the ELISSA or other such tests.

So, myosin must also be present in semen before it can be accepted as one of the possible proteins in the Gallo isolate. I understand that "molecular mass standards (BioRad Labs, Hercules, CA, USA) were run and included myosin (200 kDa), ß-galactosidase (116 kDa), phosphorylase b (97 kDa), bovine serum albumin (66 kDa), ovalbumin (45 kDa), carbonic anhydrase (31 kDa), soybean trypsin inhibitor (21.5 kDa), lysozyme (14.4 kDa) and aprotinin (6.5 kDa). Western blotting indicates that seminal fluid fucosidase contains a major protein band with a molecular mass ratio (Mr) of ~56 kDa while sperm fucosidase contains a major protein band of ~51 kDa. The overall results indicate the presence of a low-abundance, plasma membrane-associated human sperm -L-fucosidase, which is different in its properties from human seminal fluid -L-fucosidase(s), and whose function is not yet known" (see;Characterization of human semen -L-fucosidases , Molecular Human Reproduction, Vol. 5, No. 9, 809-815, September 1999).

A variety of autoantibodies against cardiac cellular proteins have been identified in dilated cardiomyopathy and the list includes G-protein-linked receptors (such as the ß1-adrenergic and muscarinic cholinergic receptors), myosin, mitochondrial proteins (such as the adenine nucleotide translocator and keto–acid dehydrogenase), actin, tubulin, heat shock proteins, and the sarcoplasmic reticulum ATPase.

A "relationship between modifications in the redox state of the actin-myosin system and other key biological processes (e.g. transport, muscle function, metabolism...)" (see; Looking back on the Oxidative Stress theory of Aids, Eleni Papadopulos-Eleopulos; Department of Medical Physics, Royal Perth Hospital, Perth, Western Australia) was also proposed. Considering other studies that show that "if we immunize animals with myosin we can stimulate the production of anti-specific myosin antibodies that are capable of producing injury in experimental animals" (Guillermo Torre-Amione, MD, PhD Immune Activation in Heart Failure), it appears that myosin antibodies cause injury directly to cells. Evidence that autoantibodies can directly damage the myocardium and initiate the sequence of events that lead to dilated cardiomyopathy comes exclusively from experimental models. However, increased actin polymerization is a potential mechanism explaining impairment of phagocytosis by oxidative stress and since AIDS is a condition caused by excess free radicals (in malnouurished people), consequently oxidative stress on macrophages leads to increased actin polymerisation and since it is this polymer actin that is in the Gallo isolate, the antibody tested is antibody against the polymer actin produced under oxidative stress."

Children with "malaria, tuberculosis or malnutrition" in communities where they are present in high frequency will have "shorter and poorer quality lives" compared to the healthy and well nourished children in the same community. It is also a fact that malnutrition (weight loss), malaria and TB are associated with a higher rate of positive "HIV" antibody tests (VIRUSMYTH hom page, "Perth Group Response to Rasnick", 15 March 2000). Such a correlation only proves that oxidative stress, whether on account of disease conditions or malnutrition will show a higher incidence of polymer actin produced by white blood cells under oxidative stress and these polymer proteins are picked up "HIV" antibody tests and explains the 'higher' rate of 'HIV' rather than a specific virus that targets the white blod cells or the immune system. This association means that antibodies looked for by the test were to cellular proteins such as actin, released under conditions of immune system stress and that a reactive test is a measure of the degree of one's exposure to stressor or oxidizing agents. (see; AIDS: Scientific or Viral Catastrophe? Neville Hodgkinson; The Journal of Scientific Exploration, Vol. 17, No. 1, pp. 87-120, 2003).