Hypothesis

Examining the causes of AIDS

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Abstract

Review of the medical literature on the causes and the pathogeneses of Acquired Immune Deficiency Syndrome (AIDS) revealed the following facts: (1) AIDS in drug users and homosexuals in the USA and Europe is actually caused by the heavy ancillary use of glucocorticoids and other immunosuppressive agents to medically treat the wide range of the chronic serious illnesses caused by drugs and medications and infections. (2) AIDS in hemophiliacs is related to the use of corticosteroids and other immunosuppressive agents to prevent the development of antibodies for factors VIII and IX and other clotting factors and to treat chronic illnesses. (3) AIDS in people receiving blood and/or tissue is related to the use of glucocorticoids to prevent reactions of transfusion and tissue rejection and other illnesses. (4) AIDS in infants and children is caused by their exposure to drugs and corticosteroids in utero and their exposure to corticosteroids after birth used to treat infectious and noninfectious illnesses. (5) AIDS in Africa is caused by malnutrition, release of endogenous cortisol, and by opportunistic diseases. (6) HIV is a harmless virus both in the *in vivo* and the *in vitro* settings and the HIV-hypothesis needs to be reevaluated. (7) The use of glucocorticoids, Zidovudine (AZT), and protease inhibitors to treat AIDS patients are contraindicated.

Keywords: AIDS, HIV, glucocorticoids, corticosteroids, immunosuppressive agents, Epivir, Zidovudine (AZT), Protease inhibitors, malnutrition, thymic atrophy, Tuberculosis, Pneumocystis carinii pneumonia (PCP), Kaposi Sarcoma, drug users, hemophiliacs, homosexual men, children with AIDS, viral load

1. Introduction

In 1984, the United States Secretary of Health and Human Services announced that the Acquired Immunodeficiency Syndrome (AIDS) is an infectious disease, caused by a sexually and parentally transmitted retrovirus. This virus was called Human Immunodeficiency Virus (HIV). The U.S. Centers for Disease Control and Prevention (CDC) and the AIDS establishment claimed that epidemiological evidence indicated that HIV is the primary cause of AIDS in the USA and the rest of the world [1-3].

For the last twenty-five-year, billions of dollars have been spent worldwide on HIV research and the treatment of HIV-positive people and AIDS patients with antiretroviral drugs. However, these huge global efforts and the sum of money have led to zero progress in the treatment and the prevention of AIDS. In addition, the medical evidence shows that the treatment of AIDS patients and the HIV-positive individuals with Zidovudine (AZT), protease inhibitors, and other toxic antiviral drugs have led to the poisoning and death of people worldwide [1-7].

My review of the published literature on the causes and the pathogeneses of AIDS worldwide indicates that (1) The U.S. CDC and the AIDS establishment did not perform differential diagnoses to rule out the involvement of non-infectious causes of AIDS prior to declaring HIV causes AIDS; (2) the CDC and the AIDS establishment have ignored the true biomarkers of AIDS that show the heavy use of illicit drugs, corticosteroids and other immunosuppressant drugs, and severe malnutrition are the true causes of AIDS; (3) the HIV-hypothesis is false and should be examined; (4) the CDC and the Director of the AIDS program at the US National Institute of Health have called pathological lesions induced by the use of illicit drugs, corticosteroids and other medications, and severe starvation as HIV-indicator diseases. Their unscientific approaches have misled physicians worldwide to think that HIV is a killer virus and treated their patients with toxic and expensive drugs.

In this report, I described the factual causes and the true biomarkers of AIDS in all risk groups. I also gave scientific evidence that indicate HIV does not cause AIDS and the HIV-hypothesis is false. Furthermore, I presented recommendations that will help healthcare providers, governments, and the private sectors in their efforts to successfully treat and control the AIDS epidemic worldwide.

2. Causes and pathogeneses of AIDS

The U.S. CDC and the AIDS establishment have claimed that AIDS is a new disease and the Human Immunodeficiency Virus (HIV) is the primary cause of AIDS. Their theory states that HIV causes AIDS by killing the CD4+ T cells directly or indirectly and after long incubation times (about 10 years), the number of these cells will reach very low levels, which leads to severe immune deficiency. Patients with CD4+ T cells < 200/μL usually suffer from opportunistic infections and/or certain form of cancer such Kaposi's sarcoma and lymphoma. The HIV-hypothesis prescribes that treatment of patients with antiviral drugs such as Zidovudine (AZT) and/or protease inhibitors

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can delay the progression of AIDS by preventing the HIV replication in the cells [1-3].

My research on the pathogeneses and causes of AIDS worldwide show that AIDS is not a new disease and it is not caused by HIV. The spread of AIDS in the USA and Europe in drug users and homosexuals in the late 1970's and early 1980's coincided with the synergistic actions of several events. These include: (1) the spread of illicit drug use by inhalation (especially smoking crack cocaine) in 1970's; (2) the approval of glucocorticoids aerosol by the US FDA in 1976 to treat respiratory illnesses caused by inhaling illicit drug; (3) the wide use of alkyl nitrites by some homosexuals to facilitate anal sex in 1970's; (4) the use of corticosteroids to treat chronic gastrointestinal tract illnesses in homosexuals.

Furthermore, AIDS in hemophiliacs, people receiving blood and or organ transplant, and infants is caused by the use of immunosuppressant and cytotoxic drugs. In Africa, the primary cause of AIDS is severe malnutrition. The approval of the antiviral drugs (AZT and protease inhibitors) and the corticosteroids by the U.S. FDA to treat patients with AIDS has made the problem worse**.** Below are the descriptions of the medical data that outline the true causes and biomarkers of AIDS in risk groups**.**

2.1 Drug users

My review of the epidemiology of AIDS revealed that approximately 30% of the AIDS cases in the USA and Europe have been observed in drug users [1, 2]. In the USA, the use of crack cocaine among drug users became very popular in the 1970s and the inhalation of crack cocaine has caused severe respiratory illnesses [1]. These illnesses have been treated with high doses of corticosteroids and other immunosuppressant drugs. For example, the inhalation of crack cocaine caused nasal septal perforation, necrosis, and granulation; chronic rhinitis; laryngeal edema; bronchial asthma; bronchiolitis obliterans; pulmonary edema; diffuse alveolar damage and hemorrhage; pneumonitis; eosinophilic pneumonia; interstitial lung diseases; and foreign body granuloma of the lungs. These illnesses are treated with high doses of corticosteroids that cause AIDS [1, 2, 3, 8, 9, 10].

For example, Tuberculosis, an AIDS-indicator illness, was induced by the treatment of a cocaine user with immune suppressant drugs. A 33-year-old previously healthy female developed acute bilateral pulmonary infiltrates after 18 hours of intense rock cocaine (crack) smoking. Ten months later, she developed progressive dyspnea and interstitial pneumonia. She was unsuccessfully treated with high doses of prednisone (1 mg/kg/day for eight weeks) followed by a trial of cyclophosphamide. She died due to respiratory failure with a superimposed mycobacterial infection. The time between her first admission to the hospital with interstitial pneumonia and her death with AIDS-indicator illness was about 21 months [11].

The followings are additional clinical examples that show the treatment of individuals with high therapeutic doses of corttiticosteroids and other immunosuppressant agents causes AIDS. These individuals suffered from severe reduction in their CD4+ T cell counts and the development of AIDS-indicator illnesses such as Tuberculosis, Pneumocystis carinii pneumonia (PCP), fungal infections, and/or Kaposi's sarcoma (KS).

1) Gluck *et al.* determined the CD4+ and CD8+ T cells lymphocyte count in seven HIV-negative patients who developed Pneumocystis carinii pneumonia (PCP) as a complication of immunosuppressive treatment. CD4+ T-lymphocyte counts (T-helper phenotype) were less than 200/μL in all seven patients (mean 90.6/μL) [12].
2) Godeau *et al.* conducted retrospective analysis of 34 HIV-negative who suffered from connective tissue diseases (CTD). These individuals developed Pneumocystis carinii pneumonia (PCP). The majority of patients (25/34 patients; 74%) presented with PCP during the first 8 months following the diagnosis of CTD. At the time of diagnosis with PCP, most patients (32/34; 94%) were receiving corticosteroids (mean prednisone equivalent dose: 1.2 mg/kg/day) associated in 24 cases with cytotoxic agents (cyclophosphamide, n = 19; methotrexate, n = 5). Most patients were lymphocytopenic at the onset of PCP: 91% (31/34) of patients had fewer than 1500 circulating lymphocytes per μL of blood and 76% (26/34) had fewer than 800/ μL [13].
3) Arend *et al.* evaluated the charts of 78 patients with Pneumocystis carinii pneumonia (PCP). They found that these patients were previously treated with immunosuppressive medication consisting of prednisone or other corticosteroids in 72 (92%) of 78 patients, cytotoxic drugs in 55 (71%) of 78 patients, and both in 50 (64%) of 78 patients. The overall mortality rate for patients was 35% (27/78). PCP occurred at all levels of immunosuppression; no threshold level could be defined. A trend toward a higher mortality in patients with previous corticosteroid use was detected ($p = 0.06$). They concluded that PCP might complicate a variety of immunocompromised states, with considerable mortality [14].
4) Yale and Limper retrospectively analyzed a consecutive series of 116 patients who developed Pneumocystis carinii pneumonia (PCP). Regardless of the associated underlying disease, corticosteroids had been administered systemically in 105 patients (90.5%) within one month preceeding the diagnosis of PCP. The median daily corticosteroid dose was equivalent to 30 mg of prednisone; however, 25% of patients had received as little as 16 mg of prednisone daily. The median duration of corticosteroid therapy was 12 weeks before the development of pneumonia; however, PCP developed after eight weeks or less of corticosteroid therapy in 25% of these patients [15].
5) Roblot *et al.* evaluated retrospectively, 103 HIV-negative patients diagnosed with Pneumocystis carinii pneumonia (PCP). Seventy-one patients (69%) received cytotoxic drugs, 57 (55%) were treated with long-term corticotherapy, and 15 (14.7%) underwent bone marrow transplantation [16].
6) Saksasithon *et al.* conducted a retrospective study of Pneumocystis carinii pneumonia (PCP) in 19 HIV-negative male and female adult patients. They found that all patients had underlying immunocompromised diseases and 94.7% of the cases received immunosuppressive drugs. PCP occurred at a mean duration of 26.4 months after the diagnosis and treatment of underlying diseases [17].
7) Sepkowitz *et al.* conducted a retrospective review of 140 patients with morphologically proved Pneumocystis carinii pneumonia (PCP). These patients had hematologic malignancy (47%), solid tumor (31%), or bone marrow transplan-

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tation (18%). All but 7 patients had previously established predisposing factors for PCP, including corticosteroid use in 87% [18].

8) Gerrard reported the development of Pneumocystis carinii pneumonia in 28 HIV-negative individuals who received corticosteroids combined with other immunosuppressive agents before the onset of PCP symptoms. The symptoms appeared within six months of immunosuppression [19].
9) A 66-year old man developed Kaposi's sarcoma (KS) after 5 years of receiving prednisone (10 to 50 mg daily or on alternate days for about 5 years) to treat bronchial asthma [20]. Cocaine is a very powerful asthma-inducing agent and drug users who developed asthma, were treated with corticosteroid [1-3].
10) Kaposi's sarcoma (KS) developed eight months after initiation of prednisone treatment (40 mg per day for three months) in a 58-year-old man with systemic rheumatoid disease. He also had lymphocytopenia (896/μL), a CD4+ T cell count of 215/μL of blood and CD4+ T cell and CD8+ T cell of 0.7. This man was HIV-negative as tested by western blot [21].
11) Drug use, alcohol, and some medications such as aspirin can induce thrombocytopenia. Individuals suffering from thrombocytopenia are treated with high therapeutic doses of immunosuppressive agents that induce the development of Kaposi's sarcoma (KS) [1-3]. For example, an 18-year-old woman with thrombocytopenia was treated with corticosteroid for 42 months. She subsequently developed Kaposi's sarcoma that spread to the spleen [22].

Furthermore, the chronic use of medications containing glucocorticoids at high doses by inhalation and other routes causes severe impairment of the immune defenses of the lungs and the immune system in general. It has led to the infection of the lungs and other organs with opportunistic microorganisms and the development of cancer. For example, the treatment of a patient with prednisone at 60 mg per day for about three months or more can actually cause severe reduction in CD4+T cell counts and AIDS. This dose is usually given to patients suffering from lung fibrosis, thrombocytopenia, and other chemically induced chronic illnesses [1, 2, 3, 8, 9, 10]. The impact of drug use and alcohol on health and the treatment with immunosuppressant drugs and other medications should be considered in the pathogeneses of AIDS. Below is a list of adverse reactions of corticosteroids and Table 1 contains a list of illnesses caused by drugs use and alcohol.

Adverse reactions of corticosteroid compounds reported in children and adults may include: (1) reduced size and function of the immune system (reduced numbers of B and T cell lymphocytes in circulation, altered migration patterns of lymphocytes, suppressed cutaneous delayed-type hypersensitivity reaction to antigens and reduced expression of lymphocyte function, decreased lymphoid cell access to antigens in inflammatory sites and antigen blastogenesis, and lymphoid atrophy); (2) increased susceptibility of the individual to infections (viral, bacterial, fungal, yeast, and parasitic); (3) gastrointestinal problems (peptic ulcer, pancreatitis, and ulcerative esophagitis); (4) fluid and electrolyte disturbances (sodium and fluid retention, potassium loss, hypokalemic alkalosis, and hypertension); (5) musculoskeletal problems (muscle weakness, loss of muscle mass, steroid myopathy, osteoporosis, bone necrosis, and fracture of long bones); (6) dermatological [impaired wound healing, thin fragile skin, facial erythema, and bleeding (petechiae and ecchymoses)]; (7) neurological (convulsions, increased intracranial pressure, vertigo, and headache); (8) endocrine (suppression of growth in children, decreased carbohydrate tolerance, manifestations of latent diabetes mellitus, development of Cushingoid state, and atrophy of adrenal glands); and (9) Ophthalmic (posterior subcapsular catracts, increased intraocular pressure, glaucoma, and exophthalmos) [1-3, 8-10].

My investigation revealed that Anthony Fauci, the Director of the AIDS program at US National Institute of Health and other proponents of the HIV-hypothesis have full knowledge of the prevalence of drug abuse in the United States and other industrial countries and the impact of the drug use on health. However, they have failed to consider the contribution of drug use in the pathogenesis of AIDS. For example, Fauci *et al.* stated that the prevalence of drug abuse in the United States remained at epidemic levels during the mid-1990s and is believed to exceed that of other industrial nations. During 1993, The National Household Survey on Drug Abuse revealed that 4.5 million persons used cocaine. Approximately 1.3 million persons stated that they self-administered cocaine on a frequently, monthly basis [2].

Furthermore, the Drug Abuse Warning Network reported in 1993 that 30,900 cocaine-related emergency room visits occurred during the later part of 1992. Illnesses that prompted room visits included cocaine-induced gastrointestinal, cardiovascular, and cerebrovascular disorders. In 1995, the National Institute on Drug Abuse's report highlighted that cocaine (including crack) remains the nation's most serious drug problem [2].

Fauci *et al.* also indicated that smoking cocaine causes severe pulmonary disease. They stated although cocaine is commonly self-administered by inhalation (snorting), there has been an increase in inhalation of pyrolyzed material via smoking. Inhalation of pyrolyzed materials includes smoking coca paste, a product produced by extracting cocaine preparations with flammable solvents, and cocaine free-base smoking. Coca paste is frequently contaminated with toxic solvents used in its preparation. Severe pulmonary diseases may develop in individuals who smoke coca paste; this effect is attributed both to the direct effects of cocaine and to residual solvent contaminants in the smoked material [2].

My investigation also revealed that Anthony Fauci and other proponents of the HIV-hypothesis have full knowledge of the impact of corticosteroids and other immunosuppressive agents on the structure and the functions on the immune system. However, they have not considered the contributory role that immunosuppressant drugs has on the pathogeneses of AIDS. In addition, they recommended the use of high therapeutic doses of corticosteroids and other immunosuppressive agents that cause AIDS to treat illnesses caused by drug use such as lung fibrosis, thrombocytopenia, and others.

For example, the treatment described on page 1463 of Fauci *et al.*'s book for patients suffering from lung fibrosis (LF) can cause AIDS [2]. They stated, "A trial of oral prednisone is begun at a dose of 1mg/kg daily and continued for about 8 weeks. Should the disease not respond or be progressive, additional immunosuppression with cyclophosphomide should be consid-

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ered. The objective is to reduce the white blood cell count to approximately half the normal baseline value, causing a distinct drop in the total lymphocyte count. However, a minimum count of 1000 PMNs/μL should be maintained." At these dose levels, the CD4+ T cells count in the peripheral blood of the treated individual is expected to be <300/μL which meets the definition for AIDS set by the United States Centers for Disease Control and Prevention (CDC) [1, 2].

I evaluated the medical records and the case history of HIV-negative white male adult who had lung fibrosis and treated with prednisone (60 mg per day) and azathioprine (50-100 mg per day). He developed AIDS following treatment with a two month course of prednisone and a two week course of azathioprine. His blood analysis showed that he had severe lymphocytopeniha, CD4+ T cell count of 255/μL, and CD4+ T cells /CD8+ T cells ratio of 0.6. He also suffered from pneumonia and severe fungal infection in his mouth and skin. Cessation of the treatment with prednisone and azathioprine led to the reversal of the damage in his immune system. He recovered from pneumonia and the fungal infection after a short course of antibiotics and the use of antifungal lotion. Twenty-two days after the last dose of prednisone, his CD4+ T cell count was 657 cells/μL [1].

The chronic use of cocaine, heroin, and alcohol has also caused brain damage, peripheral neuropathy, thrombocytopenia, renal problems, and other systemic illnesses. The majority of the drugs and alcohol induced health problems are usually treated with high doses of glucocorticoids and/or cytotoxic drugs. Since the 1970s, the prescriptions containing glucocorticoids have increased tremendously to treat more than forty medical conditions in AIDS risk groups [1-3, 8-10].

Fauci has called thrombocytopenea, peripheral neuropathy, glomerulonephritis, and other illnesses induced by drugs and medications as HIV diseases. For example, Fauci *et al.* stated on page 1,842 of their book [2]. HIV-associated nephropathy closely resembles the heroin-associated nephropathy seen in intravenous drug users (IDUs). It is now recognized as a true direct complication of HIV infection. The prototypic lesion of HIV-associated nephropathy is a focal segmental glomerulosclerosis, which is seen in approximately 80% of patients with this complication and occurs predominately in IDUs (heroin) blacks. However; on page 1,550 of the same book, they reported that intravenous heroin use is associated with an increased incidence of focal and segmental glomerulosclerosis (heroin-associated nephropathy) and occurs predominantly in blacks [2].

It seems reasonable to conclude that heroin, impurities, and infectious agents other than HIV present in dirty needles are the causes of the renal problem in the heroin drug users and not HIV. Gross examined biopsies from the kidneys of 14 drug users found that 11 (79%) showed focal segmental glomerulosclerosis [23].

Fauci's assumption of including glomeruloseclerosis as HIV disease is not supported by medical facts. In addition, Fauci *et al.* reported that "at least 25% of the opiate abusers are likely to die within 10 to 20 years of active abuse." The same period (10-20 years) was also given by Anthony Fauci and the proponents of the HIV-hypothesis as the incubation period for HIV in the drug users [2].

Table 1. Pathological changes in tissues of people induced by alcohol and illicit drugs abuse [1]

Health conditions	Causative agents
Cerebral vasculitis	Cocaine, heroin, ephedrine
Cerebral ischemia	Cocaine
Subarachnoid & intraparenchymal hemorrhage, brain	Cocaine
Cerebellar and cerebral atrophy	Alcohol
Hepatic encephalopathy	Alcohol
Leukoencephalopathy	Heroin
Optic neuritis, panophthalmitis	Cocaine
Peripheral neuropathy	Alcohol
Nasal septal perforation, necrosis, granulation	Crack cocaine
Chronic rhinitis and sinusitis	Crack cocaine
Bronchial asthma	Crack cocaine
Bronchiolitis obliterans	Crack cocaine
Pulmonary edema	Crack cocaine, heroin
Diffuse alveolar damage and hemorrhage	Crack cocaine
Pneumonitis	Crack cocaine, heroin
Pneumonia, bronchopneumonia	Cocaine, heroin
Interstitial lung diseases	Crack cocaine, heroin
Foreign body granuloma/lung	Cocaine, heroin
Aspiration pneumonia	Narcotic, alcohol
Pneumomediastinum and pneumothorax	Crack cocaine
Endocarditis	Cocaine, heroin
Cardiomyopathy	Cocaine
Peripheral vein, thrombosis, granuloma, fibrosis	I.V. drug use
Lymph node, enlargement	Cocaine, heroin
Splenomegaly	Cocaine, heroin
Thymus, enlargement	Cocaine, heroin
Ischemia of the GI tract	Cocaine
Chronic hepatitis	Narcotic, alcohol
Pancreatitis and malabsorption	Alcohol, opiates
Hyperthermia	Cocaine
Glomerumonephritis, nephrotic syndrome	Heroin
Osteomylitis, septic arthritis, & systemic sepsis	I.V. use of narcotic
Spontaneous abortion	Cocaine
Abruptio placentae	Cocaine
Severe respiratory difficulties in neonates	Cocaine
Intrauterine growth retardation	Cocaine, heroin, alcohol
low birth weight infant	Cocaine, heroin, alcohol
Malnutrition, anorexia	Cocaine, alcohol, amphetamine

2.2 Homosexual men

My review of the epidemiology of AIDS revealed that approximately 60% of the AIDS cases in the USA are observed in homosexual men [1, 2]. It appears that the incidence of AIDS among homosexual men is about twice that found among drug users and this can be explained by the chronic use of corticosteroids and/or other immunosuppressive agents by some homosexual men to treat (1) respiratory and other systemic illnesses induced by drugs; (2) anal and rectal illnesses caused by practicing anal sex and infections; (3) adverse reactions to medications such as antiviral drugs (AZT and protease inhibitors), antibiotics, and analgesic; (4) infectious illnesses such as Pneumocystis carinii pneumonia, and Tuberculosis [1-3].

The following studies cited by Duesberg demonstrate the degree of the alkyl nitrites and illicit drugs use among homosexual men: (1) 86.4% of 420 homosexual men attending clinics for sexually transmitted diseases in New York, Atlanta and San Francisco reported that they frequently used amyl-and butyl

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nitrites as sexual stimulants and the frequency of nitrite use was proportional to the number of sexual partners; (2) a total of 170 male homosexuals from sexual disease clinics, including 50 with KS and pneumonia, and 120 without AIDS were surveyed showing 50-60% had used cocaine, 50-70% amphetamines, 40% marijuana, 10% heroin, over 50% had also used prescription drugs; (3) a study of a group of 359 homosexual men in San Francisco reported in 1987 that 84% had used cocaine, 82% alkyl nitrites, 64% amphetamines, 51% methaqualone and 41% barbiturates; (4) a total of 3916 self-identified American homosexual men were surveyed, among which 83% had used one, and about 60% of them used two or more drugs with sexual activities during the previous six months (similar drug use has been reported from European homosexuals at risk); and (5) survey of homosexual men from Boston, conducted between 1985 and 1988, documented that among 206 HIV-positives, 92% had used nitrite inhalants, 73% cocaine, 39% amphetamines, 29% lysergic acid in addition to six other psychoactive drugs as sexual stimulants [24, 25].

As previously described in this report, the use of illicit drugs causes respiratory and other systemic illnesses and these illnesses are treated with high therapeutic doses of corticosteroids that cause AIDS. For example, a 38-year-old homosexual man with a history of drug abuse, presented with acute bronchitis and focal organizing chronic pneumonia with granulomatous reaction. He was treated with prednisone at 90 mg per day. After three weeks of prednisone treatment, he developed Kaposi's sarcoma (KS) on the foot, trunk, and upper and lower extremities. KS was regressed after the cessation of treatment with corticosteroid [26].

In addition, the use of alkyl nitrites (poppers) to facilitate anal sex became popular in the1970's among homosexuals. In addition, the inhalation of "poppers" at sufficient amounts causes methemoglobinemia and severe headache, which was then treated with aspirin. The heavy use of aspirin and alcohol causes thrombocytopenia. As well as, AZT and proteases inhibitors cause bone marrow depression, thrombocytopenia, and peripheral neuropathy. Thrombocytopenia and peripheral neuropathy are classified by the CDC as AIDS indicator diseases, which are also treated with high doses of glucocorticoids that cause AIDS [1-3, 6].

Fauci *et al.* described the treatment for thrombocytopenia as follows: 60 mg of prednisone is administered for 4 to 6 weeks and then decreased slowly for over another few weeks. Cyclophosphamide, azathioprine, and AZT are also among the drugs recommended for the treatment of thrombocytopenia [2]. This treatment for thrombocytopenia can cause AIDS as shown in the following case. For example, an individual with thrombocytopenia was treated with corticosteroid for 42 months and subsequently developed Kaposi's sarcoma that spread to the spleen [22].

Furthermore, some homosexual men suffer from acute and chronic rectal and gastrointestinal diseases and have been treated with rectal and oral corticosteroids and other immunosuppressive agents at high therapeutic doses that cause AIDS. Below are the findings of seven selected studies that included 736 patients who suffered from extensive rectal and gastrointestinal problems.

1) Goldberg *et al.* evaluated 163 HIV–positive homosexuals male (mean age of 33 years) and found that they complained from anorectal pain (79%); pus per anum (28%); blood per anum (26%); perianal tenderness (60%); condyloma (38%); perianal ulcers (38%); anal fissures (34%); and infections with herpes virus (50%), cytomegalovirus (25%), *Neisseria gonorrhoeae* (16%), and *Chlamydia* (16%) [27].
2) Yuhan *et al.* studied a population of 180 HIV–positive individuals (94% male with mean age of 34 years). These individuals have an average blood CD4+ T cells count of 160 cells/μL. They also suffered from anorectal pain (57%); lumps or warts (28%); rectal bleeding (12%), discharge (11%), and pruitus (6%); and 24% of patients had multiple complaints [28].
3) Orkin and Smith evaluated 40 HIV–positive homosexual men (mean age of 32 years) who had anorectal symptoms for 6 months. They suffered from condylomata, fistulas and/or abscesses, and a 63% had more than one anorectal conditions [29].
4) Barrett *et al.* studied the anorectal problem among 260 HIV–positive adult individuals (96% homosexual males) who had an average CD4+ T cells count of 175 cells/μL. Forty different perianal disorders were identified and 66% had more than one disorder [30].
5) Puy-Montbrun *et al.* evaluated 148 HIV–positive adult individuals (97% homosexual male) for anorectal diseases and 141 patients (95.2%) were found to have anorectal diseases [31].
6) Schmitt *et al.* evaluated 26 HIV–positive homosexual men and found that 15 of them had perianal ulcerations [32].
7) Lenhard *et al.* evaluated 19 HIV–positive males and found that 7 cases had hemorrhagic proctitis and 3 cases had purulent cryptitis with abscess formation and fistulation. All patients showed CD4+T cells/CD8+ T cells ratio <1 [33].

My investigation also revealed that Anthony Fauci and other proponents of the HIV-hypothesis are aware of the types and the prevalence of rectal and gastrointestinal problems among homosexual men. However, they have not considered the contribution of these illnesses and the adverse reactions of medications used in the pathogeneses of AIDS. For example; Fauci *et al.* stated, "anal douching and sexual practices such as insertion of hard objects or a clenched fist into the rectum (fisting), traumatizes the rectal mucosa and increasing the likelihood of infection during receptive anal intercourse. Sexually acquired proctitis, or inflammation limited to the rectal mucosa, results from direct rectal inoculation of typically sexually transmitted diseases (STD) pathogens. In contrast, inflammation extending from the rectum of the colon (proctocolitis), involving both the small and the large bowel (enterocolitis), or involving the small bowel alone (enteritis) can result from ingestion of typical intestinal pathogens through oral fecal exposure during sexual contact" [2].

Fauci *et al.* also reported that most infectious proctitis is due to the acquisition of *N. gonorrhoeae*, herpes simplex virus (HSV), or *C. trachomatis* during receptive anorectal intercourse. Primary and secondary syphilis also can produce anal or anorectal lesions, with or without symptoms. Primary proctitis due to HSV and proctocolitis due to the strain of *C. trachomatis* that cause lymphogranuloma venerum (LGV) usually produce severe anorectal pain and often cause fever. With LGV, a biopsy typically shows crypt abscesses, garnulomas, and giant cells; findings resembling those of Crohn's disease. Syphilis also can produce rectal granulomas, usually in association with infiltration by plasma cells or other mononuclear cells. Perianal

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ulcers and inguinal lymphadenopathy, most commonly due to HSV, can also occur in LGV or syphilis. Infectious agents may cause acute proctitis indistinguishable from idiopathic ulcerative proctitis. Such infections, often seen in homosexuals, may represent herpes simplex virus infection, gonorrhea, lymphogranuloma venerum (LGV), cytomegalovirus infection, *Isospora* infection, or *Treponema pallidum* infection, as well as amebiasis. In homosexual men, non-LGV strains of Chlamydia have been shown to produce a granulomatous proctitis closely resembles Crohn's diseases of the rectum [2].

The following clinical cases demonstrate that the severe reductions of CD4+ T cell counts in HIV-positive homosexuals who suffered from rectal and gastrointestinal problems are caused by the use of corticosteroids and not by HIV. Sharpstone *et al.* reported that eight HIV-positive males with inflammatory bowel disease who used rectal corticosteroid preparation had a decline in their CD4+ T cell at a rate of 85 cells/μL per year. Four of them underwent coloectomy that eliminated the need for the corticosteroids and their CD4+ T cell increased 4 cells/μL per year. Eight HIV-positive homosexual men who did not have surgery were used as match control. They continued to have a decline of 47 cells/μL per year as the result of the use of rectal corticosteroids [34].

Furthermore, investigators from George Washington University and the National Institutes of Health reported a case of an HIV-positive homosexual man with ulcerative colitis who developed a severe reduction in his CD4+ T cell counts following a 9 day treatment with corticosteroid. The depletion in CD4+ T cell number was reversed following the cessation of the treatment with the corticosteroid. Briefly, approximately 3 weeks prior to surgery for ulcerative colitis that was unresponsive to corticosteroid, the patient's CD4+ T cell count was 930 cells/μL of blood and the count fell to 313 cells/μL within 10 days of treatment with corticosteroid. Five days postoperatively, the patient became asymptomatic and was discharged on tapering prednisone without the use of antiretroviral agents.

After surgery, the patient's CD4+ T cell counts progressively increased. The CD4+ T cell counts were 622 cells/μL and 843 cells/μL at three and six weeks following the operation, respectively. In addition, the viral load dropped from 31,300 RNA copies/mL to 11,400 RNA copies/mL within a few weeks following the cessation of the glucocorticoid treatment and without the use of the antiviral therapy [35].

The results of these studies described above clearly show that the reductions in CD4+ T cell counts in homosexual patients have resulted from their treatment with glucocorticoid and not as the result of their HIV-infection. They also show that the viral load count is highly influenced by the glucocorticoid treatment. These studies provided clinical proof that HIV is a harmless virus and the HIV tests are worthless.

2.3 Hemophiliacs

AIDS has been reported in HIV-negative and HIV-positive hemophiliac patients. For example, Duseberg presented the result of 17 studies showing that a total of 717 hemophiliac patients had CD4+ T cell/CD8+ T cell ratios ≤ 1; 329 patients (46%) were HIV-negative [24]. My review of the medical evidence clearly shows that the likely cause of AIDS in hemophiliacs is the treatment with immunosuppressive agents (cyclophosphamide and glucocorticoids).

These medications have been used to prevent the development of antibodies to factors VIII and IX and others in patients with hemophilia. Furthermore, patients with severe hemophilia usually suffer from serious chronic joint problems resulting from bleeding inside the joints, which are treated with glucocorticoids and other immunosuppressive agents. Below are clinical studies that support my conclusions.

1) Nilsson reported that the number of hemophiliacs in Sweden in 1976 was 557 (436 with A and 121 with B). Inhibitors were demonstrated in 8% of the patients with severe hemophilia A and in 10% of those with severe hemophilia B [36].
2) Brettler stated that inhibitor formation is a serious complication, occurring in 24-52% of patients with hemophilia A and in 1.5-3% of patients with hemophilia B [37].
3) Colvin *et al.* evaluated reports of all factor VIII inhibitors arising in the United Kingdom in patients with hemophilia A during the years 1990-93 that were collected by the United Kingdom Hemophilia Center Directors Organization. A total of 32 new inhibitors were reported during this period, giving an average incidence of new inhibitors of 1.5 per 1000 patients registered per year [38].
4) Rosendaal *et al.* surveyed 935 Dutch hemophiliacs and found that 41% and 18% had severe or moderately severe hemophilia, respectively. Of these, 180 cases had more than five bleedings per year [39].
5) Ikkala *et al.* analyzed the data of all known 163 patients with severe hemophilia A living in Finland and found that the prognosis of patients with inhibitors was poor [40].
6) Nilsson and Hender reported 9 patients with severe hemophilia A and inhibitors and 3 patients with severe hemophilia B and inhibitors (inhibitor levels between 0.1 to 11 U/ml). These patients were treated on a total of 16 and 13 occasions, respectively, with a large dose of antigen (factor VIII or factor IX) and cyclophosphamide (10-15 mg/kg b.w. i.v. initially and then 2-3 mg/kg b.w. orally for 7-10 days) in connection with severe bleeding and surgery [41].
7) Allain *et al.* stated that prevention of a secondary response to factor IX by cyclophosphamide was attempted in an 11-year-old patient with severe Christmas disease. An antibody to factor IX had been present for 4 years before immunosuppressive therapy (cyclophosphamide) was tried. Despite profound lymphocytopenia, synthesis of factor IX antibody was not depressed. The difficulties of modifying the anamnestic response to factor IX by chemical immunosuppression may be as real as has been reported for factor VIII in classical hemophilia [42].
8) Syrbe and Linde analyzed reports of 223 hemophiliacs for bleeding events in 1986. Altogether, 1,802 bleeding episodes had been registered with the following locations: elbow joint 21.0%, knee joint 17.9%, ankle 14.2%, and shoulder 4.3%. In 5% of all cases, multiple joints were affected at the same time. Soft tissue bleeding occurred in 16.7% [43].
9) Aledort *et al.* stated that arthropathy is the major cause of morbidity in hemophilia. They conducted a longitudinal study to determine bleeding episodes in 673 patients at 21 international hemophilia centers to follow patients over a 6-year period. Severe (<1%) factor VIII deficient patients under the age of 25 without inhibitors were recruited into the study. The status of the six major joints of these patients was measured annually for morbidity. X-ray evaluation of these joints was carried out at the beginning and end of the study. Physical and x-ray examination scores increased significantly with age, and the number of joint

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bleeds were significant. Approximately 10% of patients began the study with all six joints normal. Of these, 50% remained normal. The average total hemorrhage or joint hemarthroses per year were 23.3 and 15, respectively [44].

My investigation revealed that Anthony Fauci and other proponents of the HIV-hypothesis have knowledge about the health problems in hemophilia patients, such as the formation inhibitors for factors VIII and IX, the joint problems, and the use of immunosuppressive agents in the treatment regimen of these patients. Yet, they have ignored all these facts and claimed that the problems in these patients are caused by HIV, thus resulting in the treatment of these very sick people with extremely toxic drugs (AZT and protease inhibitors).

For example, Fauci *et al.* stated that hemophilia A, is characterized by bleeding into soft tissue, muscles, and weight-bearing joints. Symptomatic patients usually have factor VIII levels below 5%, with a close correlation between the clinical severity of hemophilia and plasma antihemophilic factor (AHF) level. Patients with <1% of normal factor VIII have severe diseases. Following multiple transfusions, between 10 and 20% of patients with severe hemophilia develop inhibitors to factor VIII. Inhibitors are, generally, IgG antibodies that rapidly neutralize factor VIII activity. Chronic immunosuppressive regimens have been sometimes employed [2].

Fauci *et al.* also reported that typically, a hemophiliac patient presents with pain followed by swelling in a weight-bearing joint, such as the hip, knee, or ankle. Patients with congenital plasma coagulation defects characteristically bleed into muscles, joints, body cavities hours or days after an injury. The presence of blood in the joint (hemarthrosis) causes synovial inflammation and repetitive bleeding erodes articular cartilage and causes osteoarthritis, articular fibrosis, joint ankylosis, and eventually muscle atrophy. Although bleeding may occur into any joint, after a joint has been damaged, it may become a site for subsequent bleeding episodes [2].

The large body of medical evidence stated above clearly shows that the likely cause of AIDS in hemophiliacs is the use of immunosuppressive agents to treat a wide variety of chronic health problems. The functions of the immune system should be monitored during the treatment of hemophiliacs with immunosuppressant drugs by measuring blood T cell and lymphocyte counts. In addition, hemophiliacs should not be treated with AZT and protease inhibitors.

2.4 Patients received blood transfusion

Fauci *et al.* reported that from the late 1970s until the spring of 1985, over 10,000 individuals in the United States were infected with HIV through blood transfusion of blood or blood products. Approximately 7,250 individuals who survived the illness for which the transfusion was administered have developed AIDS. The risk of infection with HIV from blood transfusion was estimated to be 1/490,000 [2].

My investigation revealed that instead of HIV, the treatment with corticosteroids and other immunosuppressant agents is the likely cause of AIDS in individuals who received either a blood transfusion or blood products. Some of the patients who received blood transfusions and products developed allergic reactions that were treated with immunosuppressent drugs. In addition, some of the patients who required a blood transfusion also suffered from illnesses that are treated with immunosuppressant agents. The treatment with immunosuppressant agents should be taken into consideration prior to declaring that HIV is the cause of immunosuppression in these patients.

The list of adverse reactions to blood transfusions is described in Fauci *et al.* and the standard treatment used to prevent or cure these reactions is glucocorticoid. For example, the risk of getting an allergic reaction from a blood transfusion is 1-4 per 100. The risk for delayed hemolytic reaction is 1 per 1,500 [2]. The risk of death in a patient requiring transfusion as a result of their illness that initiated the need for blood transfusion is many thousand folds greater than the risk of infection with HIV from transfusion (1/490,000). Yet, the proponents of the HIV-hypothesis overlook these facts.

2.5 Patients received organ transplant

It has been known that patients who receive organ transplants are treated with very high therapeutic doses of glucocorticoids and other imunosuppressant drugs to prevent organ rejections. These treatments cause severe depression in the functions of the immune systems and infections (viral, bacterial, fungal, yeast, and parasitic). The CDC and the AIDS establishment call these illnesses AIDS-indicator illnesses.

My investigation revealed that the complications resulting from the immunosuppressant drug treatments of patients receiving organ transplants have been also described by Fauci *et al.* [2]. However, Fauci and other proponents of the HIV-hypothesis have ignored these medical facts. For example, Fauci *et al.* (1976) stated that they have reviewed many aspects of the host defenses that are altered by corticosteroids, and the combined effects of these changes must be considered in trying to understand the relation between corticosteroids and infections. Since the defect with corticosteroids is broad, it is not surprising that many types of infections seem to occur more often in patients treated with corticosteroids. Of the bacterial infections, staphylococcal and gram-negative infections, as well as tuberculosis and *Listeria* infections, probably occur most often. Certain types of viral, fungal, and parasitic infections also occur often [45].

In addition, Fauci *et al.*, reported that cytomegalovirus (CMV) appears to be the most common and important viral pathogen complicating organ transplantation. Syndromes produced by CMV in the immunocompromised host often begin with prolonged fever, malaise, anorexia, fatigue, night sweats, and arthralgias or myalgias. Liver function abnormalities, leukopenia, thrombocytopenia, and atypical lymphocytosis may be observed during these episodes [2].

2.6 Children

In the U.S., most infants who developed AIDS usually have mothers who are drug users [2]. The prevalence of cocaine use among pregnant women in the U.S. is relatively high as shown by countless studies. For example, cocaine-positive urine was found in 15.3% of 411 pregnant women surveyed in hospitals at the time of delivery. The impact of illicit drug and alcohol

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abuse during pregnancy on infant's health is extremely serious. Nine studies that included 1,295 drug-using mothers and 4,293 nonusers showed that cocaine use during pregnancy has led to a high prevalence of premature births and low birth weights [1].

O'shea *et al.* evaluated the outcome of pregnancy of 95 HIV-positive pregnant women and found that there was little variation in the plasma viral load that occurred during pregnancy. However, there was an association between the viral load and prematurity; the mean gestation at delivery decreasing by 1.3 weeks for every 10-fold increase in maternal HIV RNA [46]. We know that HIV does not induce labor but premature delivery is a good indicator for drug use.

Mothers expected to have premature birth are usually treated with glucocorticoids prior to delivery to facilitate the development of the lungs in the premature infants. Premature infants are also treated with glucocorticoids after birth to reduce the incidence of chronic respiratory disease. In addition, drug exposed infants usually have serious health problems that are treated with glucocorticoids [1, 2].

The thymuses of HIV-infected infants and children with AIDS usually show atrophy of the lymphoid and connective tissue. These changes are consistent with those observed in the lymphoid organs of HIV-negative children suffering from severe malnutrition or treated with high doses of corticosteroids [1-3].

2.7 AIDS in Africa

My investigation revealed that severe malnutrition is the likely cause of AIDS in Africa. Chevalier *et al.* stated that in developing countries, more than 12 million children die each year from the combined effects of malnutrition and infection. Malnourished children have impaired cellular immunity and are particularly sensitive to opportunistic infections [47]. The finding of atrophy of lymphoid tissue in people suffering from malnutrition was observed as early as 1925. For example, Jackson's review on this topic in 1925 noted that many investigators had found a pronounced tendency of atrophy of lymphoid tissue in all conditions of malnutrition. Thymus weight was exquisitely sensitive to malnutrition and was earlier designated as the "barometer of nutrition" [48].

The functions of the immune system, especially the cellular immunity, are impaired in malnutrition cases and the severity of the impairment is dependent on the degree of malnutrition. For example, the size of the thymus of 42 malnourished children was reduced by 90% as compared with those of a case-match normal control [49]. In a second study involving 110 malnourished children, the thymic area was found to be 20% of the size in healthy children [50].

The reduction in the thymus and the lymphoid tissue size and the reduction in the function of the immune system of malnourished children and adults were reversed after proper feeding [1, 50]. For example, the size of the thymus increased from 20% of normal in a malnourished child to 107% of normal following 9 weeks of proper feeding [50].

Furthermore, in Tanzania, Fawzi *et al.* studied the influence of multivitamin supplements in the diet on T cells counts in peripheral blood of 1,075 HIV-infected pregnant women who had poor nutritional status. These women received regular diet (n=267), vitamin A (n=269), multivitamins excluding vitamin A (n=269), or multivitamins including vitamin A (n=270) in a randomized, double-blind, placebo-controlled trial between 12 and 27 weeks of gestation. The T cells (CD4+, CD8+, and CD3+) increased in all groups between baseline (mean 18 weeks gestation) and 6 weeks postpartum. The average CD4+ T cells at baseline were 423 and 424/μL for the placebo and multivitamins group, respectively. At 6 weeks postpartum, the average CD4+ T cells count were 520/μL (123% of baseline) for the placebo and 596/μL (141% of baseline) for the multivitamins group [51].

The incidence of starvation, parasitic diseases, septicemia, and low birth weight are very high in Africa and other developing countries [1-3]. For example, the mortality among 299 severely malnourished children in Zambia was 25.8%. Pneumonia and diarrhea were the major causes of death [52]. In addition, Sibanda and Stanczuk reviewed all lymph node histopathology reports of lymph node biopsy submitted to the Histopathology unit in Harare, Zimbabwe in the period of January 1988 to June 1990. The commonest diseases in the 2,194 lymph node specimens submitted were: non specific hyperplasia (33%), tuberculous lymphadenitis (27%); metastases (12%), Kaposi's sarcoma (9%); and lymphomas (7%). Kaposi's sarcoma involving the lymph nodes was reported in 176 (9%) of the lymph nodes. In children, the prevalence was higher in children under 5 years than in 6-15 year bracket. Approximately two thirds (65%) of all patients with KS were aged between 20 and 40 years [53].

The high prevalence of malnutrition and disease in Africa and the impact of malnutrition on the functions of the immune systems were also reported by Fauci and other proponents of the HIV-hypothesis. However, they have not considered malnutrition in the pathogeneses of AIDS. For example, Fauci *et al.* stated that insufficient consumption of protein and energy causes loss of both body mass and adipose tissue, although one or the other loss may predominate in a given individual. Protein energy malnutrition (PEM) occurs primarily under two circumstances: in developing nations it may be present in endemic form, and under famine conditions the prevalence may approach 25%. In children of developing nations two syndromes of PEM have been distinguished: (1) maramus, manifested by stunted growth, loss of adipose tissue, generalized wasting of protein mass; and (2) kwashiorkor, manifested by growth failure, edema, and hypoalbuminemia, fatty liver, and preservation of subcutaneous. Mixed forms are common in both children and adults [2].

Furthermore, Fauci *et al.* reported that the magnitude of malnutrition problem worldwide is immense**.** In 1983 the World Health Organization estimated that 300 million children had growth retardation secondary to malnutrition. Gastrointestinal infections frequently precipitate clinical PEM because of the associated diarrhea, associated anorexia, vomiting, increased metabolic needs, and decreased intestinal absorption. Parasitic infections play a major role in many parts of the world. Cell-mediated immunity is impaired as indicated by all standard tests. Common infections and opportunistic infections can lead to increased morbidity and mortality. Pneumonia is common. All wounds and incisions heal more slowly in PEM. Wound dehiscence is common. Nearly every aspect of reproduction is impaired in the woman with PEM, including implantation, fetal

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growth, lactation, and parturition. The infants are stunted in size and may have cognitive impairment if they survive [2].

3. Biomarkers that show the HIV-hypothesis is not valid

The HIV-hypothesis states that HIV causes AIDS by killing the CD4+ T cells directly or indirectly and after long incubation times (about 10 years), the number of these cells will reach very low levels which lead to severe immune deficiency. Patients with CD4+ T cells < 200/μL usually suffer from opportunistic infections and certain form of cancer such Kaposi's sarcoma and lymphoma and others. The HIV-hypothesis prescribes that treatment of patients with antiviral drugs such as reverse transcriptase inhibitors (AZT) or protease inhibitors can delay the progression of AIDS by preventing the HIV replication in the cells [1-3]. My investigation revealed medical facts that show the HIV-hypothesis is invalid and HIV does not cause AIDS. Below is a list of biomarkers that indicate Anthony Fauci and the CDC have misinterpreted medical facts to favor the HIV-hypothesis:

1) The HIV-hypothesis states that HIV causes AIDS by selective killing of the CD4+ T cells because these cells have a special receptor on their membrane that binds with HIV [2]. I have found no truth to support these assumptions. People with AIDS usually suffer from severe loss of CD4+ T cell, CD8+ T cell, and other white blood cells in the peripheral blood and lymphatic tissues. The lymph nodes of AIDS patients show atrophy and the loss of all components that include T cell, B cell, and connective tissues. These abnormalities resemble those found in patients treated with high doses of corticosteroids and people suffering from severe malnutrition [1, 2, 54].

   For example, I reviewed published reports that describe the changes in the lymph nodes of 117 HIV-positive patients with AIDS [1]. These lymph nodes showed atrophy of lymphoid tissues and stroma. Fauci and his colleagues also examined the lymph nodes from HIV-positive AIDS patients and they found that all types of lymphocytes were depleted. They stated that apoptosis was not restricted only to CD4+ T cell; both B cell and CD8+ T cell were found to undergo apoptosis [54].

   It has been known that corticosteroids cause T cell apoptosis. For example**,** Cohen stated that destruction of thymus cells was one of the earliest observed properties of adrenal glucocorticoids. The cells affected are primarily immature, CD4/CD8 double-positive lymphocytes. This process has been clearly shown in vivo and in vitro to be apoptosis, as characterized by cell shrinkage, membrane alterations, nuclear collapse and chromatin fragmentation into oligonucleosomes. Glucocorticoid-induced thymocyte death requires new mRNA and protein synthesis. A beginning has been made in identifying the genes involved in thymocyte apoptosis. A case is made for the death of unselected thymocytes in vivo being regulated by endogenous glucocorticoids [55].
2) HIV has been found in CD4+ T cell, CD8+ T cell, and B cell lymphocytes in the lymph nodes of some people. It's ability to infect cells is not restricted to cells that have CD4 receptors as stated by the HIV-hypothesis [1, 2].
3) In vitro and in vivo studies show that HIV does not kill CD4 T cells. In 1985, Hoxie *et al.* observed no evidence of death in T cells infected with HIV in tissue culture. These cells continued to produce virus particles for more than four months after inoculation with the virus [56]. Fauci and his colleagues examined the lymph nodes from HIV-positive AIDS patients and they observed apoptosis of CD4+ T cells, CD8 T cells, and B lymphocytes. However, they found that the increased intensity of the apoptotic phenomenon in lymphocytes is independent of the progression of HIV activities and the levels of viral load [54].
4) The following clinical examples show that the reductions in the T cell counts observed in HIV-positive patients treated with corticosteroids were reversed following the cessation of the treatment. Briefly, eight HIV-positive males with inflammatory bowel disease who used rectal corticosteroid preparation had a decline in their CD4+ T cells at a rate of 85 cells/μL per year. Four of them underwent coloectomy that eliminated the need for the corticosteroids and their CD4+ T cells increased 4 cells/μL per year [34].

   In addition, an HIV-positive homosexual man with ulcerative colitis developed a severe reduction in his CD4+ T cell counts following nine days of treatment with corticosteroid. The depletion in CD4+ T cell counts was reversed following the cessation of the treatment. Approximately 3 weeks prior to surgery for ulcerative colitis that was unresponsive to corticosteroid, the patient's CD4+ T cell count was 930 cells/μL of blood and the count fell to 313 cells/μL within 10 days of treatment with corticosteroid. Six weeks following the operation, his CD4+ T cells increased to 843 cells/μL [35]. These data indicate that the reduction of T cells was caused by the corticosteroid and not by HIV.
5) The reversal of the reduction in CD4+T cell count in 1,075 HIV+ pregnant women following proper feeding was also reported. The CD4+ T cell counts of the women who received multivitamins increased from 424/μL to 596/μL during six months of proper feeding [51]. These data indicate that the reduction in T cells was caused by malnutrition and not by HIV.
6) I reviewed published reports describing the changes in the lymph nodes of 505 HIV infected patients who were asymptomatic or had AIDS. Three distinct stages of changes in lymph nodes are evident. These are hyperplasia (245 patients), atrophy (117 patients), and mixed stage (172 patients) [1]. The presence of hyperplasia in the infected lymph nodes contradicts the HIV-hypothesis that states that HIV destroys infected T cells. My conclusions are also supported by the findings reported by Fauci and his colleagues. They examined 29 HIV+ lymph nodes and found twelve of these lymph nodes with follicular hyperplasia and extensive germinal centers, five with follicular hyperplasia mixed with follicular involution, twelve lymph nodes with a mixture of follicular involution and lymphocyte depletion, and five lymph nodes with lymphocyte depletion [54].
7) My review of the medical literature revealed that a large portion of AIDS patients suffer from metabolic and endocrine abnormalities [1]. The high prevalence of adrenal insufficiency among AIDS patients provides very strong evidence that AIDS in these patients is caused by the use of corticosteroids. My conclusion is also supported by Fauci and other proponents of the HIV-hypothesis. Fauci *et al.* stated that endocrine and metabolic abnormalities are frequently seen

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in HIV-infected individuals and most HIV-infected individuals studied at autopsy had involvement of adrenal glands. The most common abnormality seen in HIV infected individuals is hyponatremia, seen in up to 30% of patients. They also stated that the presence of a low sodium level combined with a high serum potassium level in a patient should alert one to the possibility of adrenal and adrenocortical insufficiency—as seen following prolonged administration of excess glucocorticoids [2]. However; the use of corticosteroids by AIDS patients was not considered by Fauci and his colleagues.

8) Physicians reported to the CDC many cases of individuals with AIDS but were not infected with HIV. Fauci and the CDC have not investigated the cause(s) of AIDS in these people but rather called this condition as "idiopathic CD4+ T cell lymphocytopenia" (ICL). They stated that ICL is different from AIDS because the ICL patients also have low CD8+ T cell and B cell counts in addition to low CD4+ T cell counts [2]. However, Fauci *et al.* also stated that people with AIDS have low B cell and CD8+ T cell counts in addition to CD4+ T cell [2, 54]. It seems that Fauci's has made contradictory statements.
9) There are thousands of healthy people who have been infected with HIV for more than 10 years. However, they remained asymptomatic. Fauci and the CDC have referred to these people as "long-term non-progressors" [2]. They have not explained why people are living in perfect health for 10 years or more with HIV, if HIV is supposed to be a killer virus. The logical explanation for this mystery is that these people are not using drugs and/or taking toxic medications.

4. Toxicities of the antiretroviral drugs prescribed for AIDS patients

AIDS patients, HIV-positive pregnant women, and HIV-positive malnourished people have been treated with toxic and expensive drugs (AZT, protease inhibitors, and other antiviral drugs) based on a false assumption that HIV causes AIDS. AZT causes severe bone marrow depression and reduces white blood cell counts including T cells. It is very toxic to the stem cells in bone marrow (the source of T and B lymphocytes) and to fast growing tissues such as embryonic and fetal tissues. It also causes other systemic toxicities. In addition, protease inhibitors and other toxic antiviral agents cause wide spread systemic damage in liver, kidneys, pancreas, and other organs. [1-10].

The AIDS establishment claimed that protease inhibitors helped AIDS patients by increasing CD4+ T cells. My investigation also revealed that the protease inhibitors increase T cell count in some patients [57, 58]. However, this increase in T cell number is likely to be caused by inflammation in tissues caused by protease inhibitors and/or as nonspecific response similar to those occurred in some people with cancer who were treated with chemotherapy. Below is a list of studies that shows thymic hyperplasia was induced by chemotherapy.

1) Kissin *et al.* reported that 14 patients out of 120 patients developed thymic hyperplasia at 3-14 months after initiation of treatment with chemotherapy to treat cancer of the testes. Histologic examination of the thymus revealed that the thymic enlargement represented true hyperplasia [59].
2) Serial thoracic CT scans of 100 patients suffering from testicular cancer revealed that the thymus appears to atrophy temporarily during administration of cytostatic agents. About two months after cessation of chemotherapy rebound enlargement of the thymus occurs and persists for about two years followed by a slow involution [60].
3) Twenty-two patients 2-35 years old underwent serial thoracic CT evaluations for metastatic disease. Thymic volumes were determined for each patient during cycles of chemotherapy and were compared with the patient's clinical status. These patients showed cyclic thymic volume changes in response to chemotherapy or its discontinuance. During the first course of chemotherapy, the thymic volume decreased by an average of 43% in 20 of 22 patients. Between the first and second course, regrowth was observed in all 20 of these patients [61].
4) Hansen *et al.* stated that 10% of patients treated for germ cell tumors develop a benign, reversible hyperplasia of the thymus within two years after treatment [62].
5) Carmosino *et al.* reported two patients who developed thymic hyperplasia following combination chemotherapy for malignant disease [63].
6) Murphy *et al.* reported two cases who developed thymic hyperplasia after systemic chemotherapy for cancer [64].

I hope that the information described below concerning the serious toxicities of the antireteroviral drugs given to HIV-positive individuals and AIDS patients will alert physicians and people about the suffering of these patients. As shown below, these medications cause bone marrow depression, reduction in B and T cell counts and the functions of the immune system, thrombocytopenia, peripheral neuropathy, infections, and cancer. Unfortunately, the CDC organized the adverse reactions of these medications in a list and called them AIDS-indicator diseases.

4.1 Zidovudine (AZT or azidothymidine)

The following are medical data that demonstrate the toxicity of AZT (brand name Retrovir® or Retrovis®) and the invalidity of the AIDS establishment's claim that AZT has helped people with AIDS.

Fischl *et al.* gave AZT to 524 subjects who had a first episode of Pneumocystis carinii pneumonia [5]. These subjects received AZT in either a dose of 250 mg taken orally every four hours (n=262) or a dose of 200 mg taken orally every four hours for four weeks and thereafter 100 mg taken every four hours (n=262).

In this study, additional AIDS-defining opportunistic infections developed in 429 subjects (82%) in the AZT treated groups. Furthermore, the neutrophil counts declined to less than 34% of baseline in 230 subjects; the hemoglobin levels declined to less than 66% of baseline in 178 subjects; and 134 subjects received red-cell transfusions. 183 subjects (35%) were withdrawn from AZT therapy because of toxic reactions such as severe anemia and neutropenia. At 24 months of treatment, the mortality rates were 66% and 73% in the low and standard AZT doses, respectively [5].

Furthermore, the following is a list of some of the serious adverse reactions to AZT that have been reported in some chil-

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dren and adults: (1) cardiovascular problems (neutropenia, granulocytopenia, anemia, thrombocytopenia, vasculitis, and vasodilatation); (2) digestive system problems (edema of the lip, bleeding of the gum, edema of the tongue, mouth ulcer, pharyngitis, constipation, diarrhea, rectal hemorrhage, hepatomegaly with steatosis, hepatitis, hyperbilirubinemia, and pancreatitis); (3) neurological problems (tremor, twitch, anxiety, confusion, depression, dizziness, emotional problems, loss of mental acuity, nervousness, paresthesia, hyperalgesia, somnolence, and vertigo); (4) muscle and joint problems (myopathy and myositis, muscle spasm, and arthralgia); (5) respiratory system problems (flu syndrome, cough, dyspnea, epistaxis, hoarseness, rhinitis, and sinusitis); (6) skin problems (photophobia, sensitization reactions, acne, changes in skin and nail pigmentation, pruritus, rash, sweat, and urticaria); (7) urinary system problems (dysuria, polyuria, urinary frequency, and urinary hesitancy); (8) other systemic reactions (lactic acidosis, abdominal pain, back pain, body odor, chest pain, chills, fever, syncope, lymphadenopathy, and hearing loss). [1-10].

4.2 Epivir® (lamivudine)

Epivir® is a synthetic nucleoside analogue, which is given singly or in combination with other antiviral drugs to AIDS patients. The adverse reactions of Epivir may include (1) body as a whole (headache, malaise and fatigue, and fever or chills); (2) digestive system (nausea and vomiting, diarrhea, anorexia and/or decreased appetite, abdominal pain, abdominal cramps, pancreatitis and dyspepsia); (3) nervous system (neuropathy, insomnia and other sleep disorders, dizziness and depression); (4) respiratory system (nasal signs and symptoms, and cough); (5) skin rash, muscles, and joints (skin rash, musculoskeletal pain, myalgia, and arthralgia); and (6) bone marrow (neutropenia, anemia, and thrombocytopenia) [8-10].

4.3 Viread® (tenofovir disoproxil fumarate)

Viread® is the brand name for tenofovir disoproxil fumarate, which is converted in vivo to tenofovir, an acyclic nucleosise phosphonate. The adverse reactions of Viread may include asthenia, hypophosphatemia, lactic acidosis, dizziness, dyspenia, rash, renal insufficiency, kidney failure, and Fanconi syndrome. [8-10].

4.4 Sustiva® (efavirenz)

Sustiva® is an HIV-1 specific, non-nucleoside, reverse transcriptase inhibitor (NRTI). It has been given to AIDS patients alone and with AZT and other antiviral agents. The most significant adverse events associated with this drug observed in a study that included 2215 adult patients were nervous system symptoms and rash. Fifty-two percent of patients receiving Sustiva® reported central nervous system and psychiatric symptoms (dizziness, somnolence, insomnia, abnormal dreaming, confusion, abnormal thinking, impaired concentration, amnesia, agitation, depersonalization, hallucinations, and euphoria).

Other adverse reactions to this drug may include alcohol intolerance, allergic reaction, asthenia, fever, hot flashes, malaise, pain, peripheral edema, syncope, ataxia, confusion, convulsions, impaired coordination, migraine headaches, neuralgia, paresthesia, peripheral neuropathy; speech disorder, tremor, vertigo, dry mouth, pancreatitis, tinnitus, flushing, palpitations, tachycardia, thrombophlebitis, hepatitis, arthralgia, myalgia, asthma, alopecia, eczema, folliculitis, skin exfoliation, urticaria, abnormal vision, diplopia, parosmia, and taste perversion. [8-10].

4.5 Kaletra® (combination of lopinavir and ritonavir)

Kaletra® is a co-formulation of lopinavir (inhibits HIV protease) and ritonavir (inhibits the CYP3A-mediated metabolism of lopinavir). The adverse reactions of Kaletra may include the following: (1) body as a whole (abdominal pain and enlargement, headache, fever or chills, allergic reaction, back pain, chest pain, face edema, flu syndrome, bacterial and/or viral infection, malaise, and asthenia); (2) cardiovascular system (atrial fibrillation, deep vein thrombosis, hypertension, migraine, palpitation, thrombophelobitis, varicose vein, and vasculitis); (3) digestive system (dry mouth, constipation, nausea and vomiting, diarrhea, dyspepsia, dysphagia, flatulence, increased appetite or anorexia, ulcerative stomatis, sialadenitis, esophagitis, fecal incontinence, gastritis, gastroenteritis, hemorrhage, enterocolitis, colitis, cholangitis, cholecystitis, jaundice, and pancreatitis); (4) hemic and lymphatic system (anemia, leukopenia, and lymphadenopathy); (5) endocrine system (Cushing's syndrome, diabetes mellitus, and hypothyroidism); (6) metabolic and nutritional disorders (avitaminosis, dehydration, edema, decreased glucose tolerance, lactic acidosis, obesity, peripheral edema, weight gain, and weight loss); (7) nervous system (abnormal dream, agitation, amnesia, anxiety, apathy, ataxia, confusion, convulsion, dizziness, dyskinesia, emotional lability, encepahalopathy, facial paralysis, hypertonia, decreased libido, neuropathy, paresthesia, peripheral neuritis, somnolence, and tremor); (8) respiratory system (asthma, bronchitis, dyspnea, lung edema, pharangitis, rhinitis, and sinusitis); (9) urogenital system (abnormal ejaculation, gynecomastia, hypogonaddism in male, kidney calculus, and urine abnormality); (10) skin and appendages (acne, alopecia, dry skin, eczema, exofoliative dermatitis, furunculosis, maculopapular rash, nail disorder, pruritis, seborrhea, skin benign neoplasm, skin discoloration, skin ulcer, and sweating); and (11) special senses (abnormal vision, eye disorder, otitis media, taste perversion, and sweating). [8-10].

4.6 Fortovase® (saquinavir)

Fortovase® is an inhibitor of HIV-protease. The treatment of patients with Fortovase® alone or in combination with other antiviral drugs may lead to the following adverse reactions: night sweats, increased sweating, allergic reaction, anorexia, appetite reduction, appetite disturbances, asthenia, chest pain, edema, fever, intoxication, malaise, olfactory disorder, general body pain, pelvic pain, retrosternal pain, shivering, wasting syndrome, generalized weakness, weight loss, cyanosis, heart murmur, heart rate disorder, heart valve disorder, hypertension, hypotension, stroke, syncope, vein distension, ataxia, cerebral

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hemorrhage, confusion, convulsions, dizziness, dyserthria, hyperesthesia, hyperreflexia, hyporeflexia, light-headed feeling, myelopolyradiculoneuritis, neuropathy, face numbness, paresis, paresthesis, peripheral neuropathy, poliomyelitis, prickly sensation, progressive multifocal leukoencephalopathy, spasms, tremor, unconsciousness, acne, alopecia, dermatitis, erythema, folliculitis, furunculosis, hair changes, hot flashes, nail disorder, papillomatosis, papular rash, photosensitivity reaction, pigment changes, pruritus, psoriasis, maculopapular and pruritic rash, red face, skin ulceration, urticaria, verruca, xeroderma, and dehydration.

The treatment of patients with protease inhibitors also can lead to development of diabetes, hyperglycemia, hypoglycemia, hypothyroidism, thirst, elevation in the triglyceride level, weight gain, abdominal distention, bowel movements frequent, buccal mucosa ulceration, oral canker sores, cheilitis, abdominal colic, dysphagia, esophageal ulcration, esophagitis, fecal incontinence, fecal blood stained, fecal discoloration, gastralgia, gastritis, gastroesophageal reflux, gastrointestinal inflammation, glossitis, rectal hemorrhage, hemorrhoids, infectious diarrhea, melena, painful defecation, parotid disorder, pruritis ani, pyrosis, salivary glands disorder, stomach upset, stomatitis, unpleasant taste, toothache, tooth disorder, and gastrointestinal ulcer.

Also, protease inhibitors can cause anemia, neutropnia, pancytopenia, splenomegaly, cholangitis sclerosing, cholelithiasis, hepatitis, hepatomegaly, hepatosplenomegaly, jaundice, liver enzyme disorder, pancreatitis, arthralgia, arthritis, back pain, leg cramps, muscle cramps, lumbago, musculoskeletal disorders, myalgia, myopathy, facial and jaw pain, leg and musculoskeletal pain, stiffness, Kaposi's sarcoma, dermal bleeding, hemorrhage, microhemorrhages, thrombocytopenia, agitation, amnesia, anxiety attack, behavior disturbances, excessive dreaming, euphoria, hallucination, reduced intellectual ability, irritability, lethargy, psychic disorder, psychosis, somnolence, speech disorder, male reproductive problems (epididymitis, erectile impotence, penis disorder, and prostate enlargement), female reproductive problems (menstrual disorder, menstrual irregularity, and vaginal discharge), bronchial asthma, bronchitis, cough, dyspnea, epistaxis, hemoptysis, laryngitis, pharyngitis, pneumonia, pulmonary disease, respiratory disorder, rhinitis, sinusitis, upper respiratory tract infection, blepharitis, conjunctivitis, cytomegalovirus retinitis, dry eye syndrome, earache, ear pressure, eye irritation, hearing loss, otitis, unpleasant taste, tinnitus, visual disturbance, xerophthalmia, micturition disorder, nocturia, renal calculus, renal colic, urinary tract bleeding, and urinary tract infection. [8-10].

5. Conclusions and recommendations

The United States Centers for Disease Control and Prevention (CDC) and Anthony Fauci, the Director of the AIDS program at the U.S. National Institute of Health have overlooked crucial medical evidence that indicates HIV does not cause AIDS. The medical evidence presented in this report and the references cited clearly show that AIDS is not a new disease and HIV is a harmless virus. The HIV-hypothesis has misled physicians from all over the world to prescribe toxic medications to healthy HIV-positive people and people with AIDS. It has also influenced physicians to overlook the health problems associated with the use of illicit drugs, alcohol and medications.

I urge the medical community, scientists, and governments to evaluate the true causes of AIDS to save lives and vital resources. Differential diagnoses should be used that consider all factors involved in causing immune suppression in each case. The synergistic actions among agents in causing immune suppression should also be considered. In this report, I presented many examples of agents that cause immune suppression and illnesses listed by the CDC as AIDS indicator illnesses. There are also many other agents that cause bone marrow depression and immune suppression such as antibiotics, antivirual, and anti-fungal drugs and these agents should also be considered in the pathogeneses of AIDS.

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