THE ROLE OF NUTRITION IN IMPROVING THE HEALTH OF PEOPLE LIVING WITH HIV/AIDS

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1. NUTRITIONAL IMMUNOLOGY.

The effects of malnutrition on lymphoid organs were first described during the middle of the 19th century (1). Lymphoid tissues are particularly vulnerable to the damaging effects of malnutrition and lymphoid atrophy is a prominent feature in nutritional deprivation (2-5). Cell division is a very singular characteristic of the functioning of immunocompetent cells. All types of immune cells and their products, such as interleukins, interferons, and complement, are known to depend on metabolic pathways that employ various nutrients as critical co-factors for their actions and activities (5,6). Most of the host defense mechanisms are altered in protein caloric malnutrition (PCM), as well as during deficiencies of trace elements and vitamins (2,4,7,8).

Patients with PCM have impaired delayed cutaneous hypersensitivity, poor lymphocyte proliferation response to mitogens, lower synthesis of lymphocyte DNA, reduced numbers of rosetting T lymphocytes, impaired maturation of lymphocytes seen through an increased deoxynucleotidyl transferasa activity, decreased serum thymic factor, fewer CD4+ cells, decreased CD4+/CD8+ ratio, impaired production of interferon gamma and interleukin 2, altered complement activity (especially reduction of C3, C5, factor B and total hemolytic activity), poor secondary antibody response to certain antigens, reduced antibody affinity, impaired secretory immunoglobulin A response, decreased antibody affinity, and phagocyte dysfunction (2-7).

Human malnutrition is usually a composite syndrome of multiple nutrient deficiencies. However, isolated micronutrient deficiencies do happen. Vitamin A deficiency results in reduction in the weight of the thymus, decreased lymphocyte proliferation, impaired natural killer cell and macrophage activities, and increased bacterial adherence to epithelial cells (8-11). Vitamin B6 deficiency produces failure of several components of both cell-mediated and humoral immune responses (2,4,7). Vitamin C deficiency impairs phagocytosis and cell-mediated immune reactions (12). Vitamin E deficiency also alters immune responsiveness (2,4,7). Zinc deficiency generates

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lymphoid atrophy, reduces lymphocyte responses and skin delayed hypersensitivity (2,4,7). Copper and selenium deficiencies impair T and B lymphocyte functions (2,4,7). Dietary deficiencies of selected amino acids such as glutamine and arginine also alter immunity (2,4,7).

Intrauterine malnutrition causes prolonged, even permanent, depression of immunity in offspring (13-14).

Considerable data implicate excess lipid intake in the impairment of immune responses (15). The potential for free radical damage is dependent in large part on the level of potentially oxidizable fatty acids, mainly polyunsaturated fatty acids (PUFAs) in the diet (15). High levels of dietary PUFAs have been shown to be immunodepressive. Dietary fats may undergo free radical-mediated oxidation prior to ingestion, as can occur when foods are fried (15). Animals fed oxidized lipids show marked atrophy of the thymus and T lymphocyte dysfunctions (15).

At the molecular level, the damage to immunocompetent cells by several nutritional deficiencies (PCM, Vitamin A, Vitamin C, Vitamin E, zinc, copper, zelenium deficiencies) is caused by increased free radicals through oxidative stress (8-11,15,16).

2. NUTRITIONAL DEFICIENCIES AND HIV/AIDS.

Since the beginning of the AIDS epidemic, researchers have provided scientific evidence that supports the possibility that AIDS can be effectively prevented, treated, and overcome by guaranteeing an optimal nutritional status to the individual or the patient (17,18). However, it seems that propaganda spread by pharmaceutical companies to commercialize antiretroviral medications has prevented these ideas from being widely accepted, despite the toxicity of these medications.

Early in the AIDS era, well recognized researchers in the field of nutrition and immunology, such as Dr. Ranjit Kumar Chandra, noticed that: “There is an uncanny similarity between the immunological findings in nutritional deficiencies and those seen in acquired immunodeficiency syndrome, AIDS” (17).

“There is a similarity between the immune deficiency, multiple infections, and severe weigh loss seen in AIDS patients, and the association of protein caloric malnutrition (PCM) with reduced resistance to infection observed in malnourished children, particularly in the Third World.” “It is also possible that nutritional deficiency may play a significant role in the clinical course of the immunodeficient state.” “These similarities between AIDS and PCM suggest that nutrition may contribute to the immunodeficient state. The immunodeficiency in children with PCM can be reversed by nutritional rehabilitation, which suggests that restoration of nutritional state may be a useful adjunct to therapy for AIDS patients” (19).

As described above, the immunological alterations found in PCM are practically identical to those of AIDS: impaired delayed cutaneous hypersensitivity, lymphocyte proliferation response to mitogens, complement activity and secondary response to antigens. There is also a reduced number of rosetting T lymphocytes, increased deoxynucleotidyl transferase activity, decreased serum thymic factor, fewer helper T cells, impaired production of interferon gamma and interleukins 1 and 2, reduced antibody affinity, impaired secretory immunoglobulin A (IgA) antibody response and phagocyte dysfunction. The proportion of helper/inducer T lymphocytes recognized by the presence of CD4 positive antigen on the cell surface is markedly decreased. The ratio CD4/CD8 is significantly decreased. Lymphoid atrophy is a prominent feature of nutritional deprivation. Serum antibody responses are generally intact in PCM. Most complement components are decreased, especially C3, C5, factor B and total hemolytic activity (20-26).

“Nutritional problems have been a part of the clinical aspects of AIDS from its earliest recognition as a new disease” (20,24). “In fact, in many AIDS patients, death seams to be determined more by the individual’s nutritional status than by any particular opportunistic infection. This is, when wasting of lean body mass approaches 55% of normal for age, sex, and height, death is imminent regardless of the forces resulting is such profound malnutrition” (20-24). Moreover, the severity

of the clinical manifestations of AIDS is proportional to the degree of the nutritional deficiencies (27-30).

In addition to supporting optimal function of the immune system, nutrition is especially critical in children, as it provides the best opportunity for normal growth and development (31,32).

“All persons with HIV infection should be screened for nutritional problems and concerns at the time of their first contact with a health care professional, and routine monitoring should be performed on an ongoing basis” (31).

Scientific evidence strongly suggests that nutritional and antioxidant deficiencies are a prior requisite to both reacting positively on the tests for HIV (ELISA, Western blot, Viral Load) (33-36) and progressing to AIDS (37,38).

3 NUTRITIONAL DEFICIENCIES AND THE PROGRESSION OF HIV-POSITIVE INDIVIDUALS TO AIDS.

An optimal nutritional status as well as adequate vitamin levels are known to be, by themselves, enough to prevent the development of AIDS in people who react positively on the tests for HIV (39-46).

For example, regarding vitamins in HIV disease progression and vertical transmission, researchers from the Harvard School of Public Health state: “The higher rates of HIV progression and vertical transmission in developing countries coincide with similarly higher rates of malnutrition and vitamin deficiencies, indicating that HIV infection, may be modified by nutritional status.” “Numerous observational studies report inverse association between vitamin status, measured bio-chemically or as levels of dietary intake, and the risk of disease progression or vertical transmission.” “Adequate vitamin status may also reduce vertical transmission through the intra-partum and breastfeeding routes by reducing HIV viral load in lower genital secretions and breast milk” and “vitamin supplements may be one of the few potential treatments that are inexpensive enough to be made available to HIV-infected persons in developing countries” (47).

Growing numbers of scientific trials implicate low serum vitamin A levels as a risk factor for HIV-positive individuals to progress to the clinical manifestations of AIDS (48-60).

“The risk of death among HIV-infected subjects with adequate serum vitamin A levels was 78% less, when compared with Vitamin A-deficient subjects” (47,52).

“In a study carried out among HIV-positive homosexual men, development of Vitamin A deficiency over an 18-month period was associated with a decline in CD4 cell count, widely used as a marker of HIV immune impairment. Normalization of vitamin A was associated with higher CD4 cell counts” (37, 47).

“Lower serum levels of vitamin A were associated with a faster rate of progression among men who participated in the Multicenter AIDS Cohort Study (MACS)” (42, 47).

On the other hand, “among well nourished HIV seropositive men who participated in the San Francisco Men’s Health Study, high energy-adjusted vitamin A intake at baseline was associated with higher CD4 cell counts at baseline, as well as with lower risk of developing AIDS during the 6 year period follow up” (44, 47).

“In a placebo-controlled trial in South Africa among children born to HIV-positive women, Vitamin A supplements resulted in approximately 50% reduction in diarrheal morbidity among HIV-infected children” (47,51).

Besides vitamin A, a growing number of studies show that “HIV-positive” individuals are at higher risk of deficiency of vitamins B1, B2, B6, B12, C, D, and E (47,61-68). Furthermore,

deficiencies of B-complex vitamins, vitamin C, vitamin E and vitamin D increment the risk of progression of “HIV-positive” individuals to AIDS (47,61-68).

4. NUTRITIONAL DEFICIENCIES AND THE “TRANSMISSION” OF HIV/AIDS.

Several studies show that vitamin A deficiency is more prevalent among HIV-positive persons compared with HIV-negative individuals (28,38,40,50,57).

Low levels of vitamin A and carotenoid were found to be a risk factor for reacting positively on HIV tests in Pune, India (69), for seroconversion among Kenyan men with genital ulcers (70), and for seroconversion among Rwandan women (71).

There are several trials investigating the role of vitamin A and carotenoid deficiencies in mother to child transmission of HIV/AIDS (MTCT) during pregnancy, delivery, and breastfeeding (72-89).

In Tanzania, for example: “Multivitamin supplementation is a low-cost way of substantially decreasing adverse pregnancy outcomes and increasing T-cell counts in HIV-1 infected women” (72,73).

“A growing body of data suggests that low serum levels of vitamin A among HIV-infected pregnant women is associated with higher risk of vertical transmission of HIV” (47).

“Mean vitamin A concentration in 74 mothers who transmitted HIV to their infants was lower than that in 264 mothers who did not transmit HIV to their infants” (77).

“In Malawi, higher serum retinol of HIV-infected pregnant women was associated with a reduced risk of vertical transmission” (47,77).

“Women who had increasing serum retinol levels over time, however, were at a lower risk, whereas women who had declining serum retinol were at a higher risk of transmitting the virus” (47,89).

“Vitamin A supplementation to a population of HIV-infected pregnant women, many of whom had low vitamin A levels, was associated with a decreased number of preterm births and with reduced mother-to-child transmission of HIV in preterm babies, but was not associated with a reduction in HIV transmission overall. Vitamin A decreased HIV transmission in the preterm babies by 47%” (80).

“Detection of vaginal HIV-1 DNA was associated with abnormal vaginal discharge, lower absolute CD4 cell count, and severe vitamin A deficiency” (85).

“Women with CD4 cell depletion, especially those with vitamin A deficiency, may be at increased risk of transmitting HIV-1 to their infants through breast milk” (88).

“Increased risk of maternal-infant transmission was associated with severe vitamin A deficiency among non-breastfeeding women” in the United States (76).

Scientific studies, therefore, support the contention that the use of vitamins by themselves, especially vitamin A, could be enough to avoid what is known as transmission of HIV (47,69-89). If this is the case, as many clinical trials and scientific papers contend, supplementation with vitamin A and carotenoids would constitute an effective, inexpensive and non-toxic practice for African countries.

5. OXIDATIVE STRESS AND HIV/AIDS.

Moreover, since the beginning of the AIDS epidemic, free radicals and, specifically, oxidizing agents, have been implicated in the pathogenesis of this new syndrome (90,91). There have been international meetings on the role of oxygen free radicals in HIV/AIDS (92,93).

There are currently increasing numbers of scientific publications demonstrating that oxidizing stress is an absolute requisite for both testing positive on the tests for HIV (94-100) and developing the clinical manifestations of AIDS (101-123).

Free radical reactions of special significance to immunological phenomena include, for example, the many oxidizing agents that can abstract a hydrogen atom from thiol groups to form thiol radicals (124-126). Thiol groups are important for enzyme activities, receptor functions, disulphite links in immunoglobulins, and T cell activation and proliferation. The super oxide anion radical can react with nitric oxide, resulting in a loss of endothelium-derived relaxing factor activity, which is important in the inflammation/disinflammation process. Methionine oxidation can cause protein damage with subsequent changes in immunogenicity. Proteolysis can be increased by free radical damage. The per oxidation of lipids by reactive free radicals produces many biological modulators, such as, for example, the 4-hydroxy-alkelans, which produces strong chemotactic activity for phagocytes, alters the adenyl cyclasa system, increases capillary permeability, and alters lymphocyte activation. Lipid hydroperoxides, also from per oxidation of lipids, alter lymphocyte activation. Conditions favoring lipid per oxidation may result in chemo taxis of leukocytes, protein modification, immune complex injury, and cell death (124-126).

Free radicals are produced throughout the regular immune system network. Despite the beneficial effects of the inflammation responses, it can also aggravate existing tissue damage by releasing free radicals. When uncontrolled, initiated by an abnormal stimulus, or occurring for prolonged periods of time, inflammation may become a disease process (124-126). It is critical for optimal immune responses that there be a balance between free radical generation and antioxidant protection. During phagocytosis by polymorphonuclear leukocytes, for example, super oxide anion radicals are released. These oxygen free radicals can oxidize thiol groups to thiol radicals and can stimulate lipid per oxidation with the formation of H_2O_2, which is highly significant in the mechanisms of cell injury. Oxygen free radicals produced during phagocytosis of immune complexes are associated with injury due to immune complexes (124-126).

It has been proposed many times that free radicals and, specifically, oxidizing species, play important roles in the pathogenesis of AIDS (91-93,127-129).

The above are the scientific fundamentals for the use of antioxidants such as vitamin A and carotenoids, vitamin C, vitamin E, selenium, n-acetyl cisteine, l-gluthamin, zinc, cooper, manganese, alphalipoic acid, coenzyme Q10, and flavonoids or vitamin P, as supplementation for the prevention and treatment of AIDS (90-129).

6. NUTRITIONAL AND ANTIOXIDANT DEFICIENCIES AND THE PATHOGENESIS OF HIV/AIDS.

African countries have a high incidence of malnutrition, vitamin deficiencies, anemia, bacterial, viral, fungal, and parasitic infections and infestations.

For any infectious or parasitic disease to begin, it is always requisite that the host suffers immunodeficiency (130). At the same time, infectious and parasitic diseases cause, by themselves, more immune suppression and more malnutrition (131,132). This immunesuppression is secondary to the accumulation of free radicals, especially oxidizing species, that occur during and after infectious and parasitic diseases (125,133).

Therefore, in African countries, a persistent cycle occurs: poverty, malnutrition, immunesuppression, infectious and parasitic diseases, more immunesuppression, and more malnutrition (134,135).

On the other hand, there is growing scientific data showing that many chronic diseases of adulthood have their origin at "*in utero* programming" (136-139). This includes illnesses such as coronary heart disease and stroke, hypertension, type II diabetes and other endocrine alterations (136-141), as well as several immunological disturbances (142-152). Therefore, it appears that whatever happens during embryonic and fetal times is remembered by cells, tissues, organs, and systems throughout the lifetime.

"Research in Gambia associated season of birth with infectious disease mortality after the age of 15 years, suggesting an association between prenatal undernutrition, immune function, and adult vulnerability to infectious disease" (148,152). Prenatal undernutrition has been found to impair antibody responses to vaccination with *Salmonella thyfi* that last at least up to adolescent times (146). The findings of these researchers "suggest a role for fetal and early infant experience in programming the immune system" which may accompany the individual during its entire life (145,146).

It has been scientifically demonstrated that prenatal undernutrition alters several aspects of cell-mediated immunity, causes involution of lymphoid tissues such as the thymus, and suppression of antibody responses to vaccination. These deficits persist for weeks or, in some cases, even years (142-152).

In addition, "murine models have documented impairments in immunity after maternal undernutrition that last through adulthood and into the next generation, despite ad libitum feeding of both F1 and F2 generations" (153). Also in mice, deprivation of zinc during pregnancy causes immunodeficiency that can last at least for three generations (154).

It is therefore very probable that in Africa the consequences of poverty and malnutrition are being transmitted from generation to generation with a cumulative effect and that AIDS in Africa may be the topmost consequence of these cumulative effects of poverty.

In this light, the crucial role of maternal undernutrition in the pathogenesis of pediatric AIDS must seriously be considered a reality in developing countries (155,156). This reasoning indicates that malnutrition constitutes the main risk factor for AIDS in adults in developing countries (155,156). Scientifically speaking, there is no rationale for indicting sexual promiscuity as the cause of AIDS in Africa, while underestimating the role of poverty, malnutrition, infections and parasites.

7. NUTRITIONAL AND ANTIOXIDANT THERAPY FOR THE PREVENTION AND TREATMENT OF AIDS.

"It is not surprising, therefore, that dietary therapy for AIDS has been proposed, debated, and, more importantly, surreptitiously or overtly used from the early days of the epidemic" (24).

Twenty years later, researchers insist: "Because nutrient deficiencies may play an important role in the pathogenesis of HIV disease, medical nutrition therapy and counseling are critical aspects of treatment" (31). Nutritional (157-179) and antioxidant (180-198) therapy is therefore requisite in preventing and treating AIDS.

Clinical trials have identified in detail the vitamin and mineral needs of HIV-positive persons and those with AIDS. These studies suggest the need for increasing the intake of the following micronutrients as supplementation for the prevention and treatment of AIDS: vitamin A and carotenoids, vitamin C, vitamin E, selenium, n-acetyl cisteine, l-gluthamin, zinc, cooper, manganese, alphalipoic acid, coenzyme Q10, flavonoids or vitamin P, and B-complex vitamins (17-38,90-129,157-198).

When providing Vitamin A as a supplement its potential teratogenic property should be kept in mind (199). In this regard, the World Health Organization recommends that pregnant women should not take more than 10,000 IU of Vitamin A per day (47).

If we really want to prevent and treat AIDS in Africa, it is absolutely requisite to provide at least the minimum food needs to HIV-positive individuals, to AIDS patients, as well as to all African communities.

A diet that provides adequate sources of vitamins, minerals, and antioxidants might have quantities of fruits, especially papaya, mango, kiwi, pineapple, avocado, bananas, and dry fruits, and vegetables, legumes, and alga. Use few animal products. Prefer fatty white fish, sheep and goat meat. Prefer sea salt. Use 60-80% fresh, whole, raw organic food. Use garlic, onions, asparagus, beets, cabbage, broccoli, cauliflower, Brussels sprouts, carrots, yeast, whet, pollen, as well as sprouts, legumes, and cereals. Prefer cool press oils (below 40 degrees Celcius) since this process preserves essential and polyunsaturated fatty acids needed in anti-inflammatory and regenerative processes. Carcamo, sunflower, and olive oils, in this order, are good sources of vitamin F or linoleic acid. Lino oil is a good source of alpha linoleic acid. Eat whole cereals in any preparation (rice, barley, wheat, oat). Decrease sugar and candies. Prefer raw organic vegetables and legumes. Drink lots of liquids: water (at least 1.5 litters per day), juices from fresh fruits and vegetables, especially carrots, vegetable broths, and green juices as a source of chlorophyll (for example, blend water, lettuce, spinach, celery, mint, parsley, coriander, and similar ingredients, and take without draining). It is also very convenient to use bifidogenic foods, for example yogurt and kumis better, if made with sheep or goat's milk, tofu, or miso. Coconut oil is a good source of louric and caprilic acids which are anti-candida (164,169,173-179,200).

Immune stimulating and/or antioxidant herbs include: Aloe (*Aloe vera)*, astragalus (*Astragalus membranaceus*), Siberian ginseng (*Eleutherococcus senticosus*), Fo-ti (*Polygonum multiforum*), turmeric (*Curuma longa*), echinacea (*Echinacea angustifolia y E. purpurea*), garlic (*Allium sativum*), licorice or liquorice (*Glycyrrhiza glabra*), golden seal (*Hydrastis Canadensis*), cat's claw (*Uncaria tomentosa*), ginkgo (*Ginkgo biloba*), grape fruit seeds (*Vitis vinifera*), zarzaparrilla or smilax (*Smilax officinalis y S. aspera*), Southerlandia (African herb); sedative and relaxing herbs include peace flower (*Passiflora incarnata*), valerian (*Valeriana officinalis*), chamomile (*Matricaria chamomilla*), mint (*Menta sativa*), lavender (*Lavanda officinalis*), and Siberian ginseng (*Eleuterococus senticosus*) (182,186,187,200-203).

A large number of scientific papers and books have been published reviewing the issue of nutritional and antioxidant therapy for the prevention and treatment of AIDS (157-206).

8. CONCLUSIONS

A) Nutritional and antioxidant deficiencies have been documented in all patients with AIDS.

B) Nutritional and antioxidant deficiencies are a requisite prior to reacting positively on "HIV tests."

C) Nutritional and antioxidant deficiencies are also a requisite of "HIV-positive" individuals prior to the development of the clinical manifestations of AIDS.

D) Nutritional and antioxidant deficiencies play a major role in the pathogenesis of AIDS.

E) Nutritional and antioxidant supplements are being used successfully in preventing and treating AIDS. An optimal nutritional and antioxidant status can guarantee success in preventing and treating AIDS.

F) Some of the nutritional and antioxidant supplements that have been used in the treatment and prevention of AIDS are: vitamin A and carotenoids, vitamin C, vitamin E, selenium, n-acetyl cisteine, l-gluthamin, zinc, cooper, manganese, alphalipoic acid, coenzyme Q10, B-complex vitamins, and flavonoids or vitamin P.

G) To prevent and treat AIDS in Africa, an absolute requisite is the provision of at least the minimum food needs to HIV-positive individuals, to AIDS patients, and to all African communities. Moreover, diets rich in fresh and organic fruits, vegetables, and cereals, as well as diets rich in bifidogenic foods (yogurt, kumis) are known to be immune stimulants.

REFERENCES

1. NUTRITIONAL IMMUNOLOGY.

1. Simon J. *A physiological essay on the thymus gland*. London: Ranshaw; 1845: 100.
2. Beisel WR. Single nutrients and immunity. *Am J Clin Nutr* 1982; 35: 417-468.
3. Beisel WR. The history of nutritional immunology. *J Nutr Immunol* 1991; 1: 62-78.
4. Chandra RK. Micronutrients and immune functions, an overview. *Ann NY Acad Sci* 1990; 587: 9-16.
5. Chandra RK. Nutrition and Immunity. In: Lachmann PJ et al. *Clinical aspects of immunology*. Boston: Scientific Publications; 1993: 1325-1338.
6. Chandra RK. Reduced secretory antibody response to live attenuated measles and poliovirus vaccines in malnourished children. *BMJ* 1975; 2: 583-585.
7. Bendich A, Chandra RK. Micronutrients and immune function. New York: New York Academy of Sciences; 1990.
8. Prabhala RH et al. Immunomodulation in humans caused by beta-carotene and vitamin A. *Nutr Res* 1990; 10: 1473.
9. Semba RD. Vitamin A, immunity, and infection. *Clin Inf Dis* 1994; 19: 489-499.
10. Semba RD. The role of vitamin A and related retinoids in immune function. *Nutr Rev* 1998; 56: S38-S48.
11. Chandra RK, Au B. Single nutrient deficiency and cell-mediated immune responses. III. Vitamin A. *Nutr Res* 1981; 1: 181-185.
12. Anderson R, et al. Vitamin C and cellular immune functions. In: Bendich A, Chandra RK. *Micronutrients and immune function*. New York: New York Academy of Sciences 1990: 34-48.
13. Chandra RK. Fetal malnutrition and postnatal inmunocompetence. *Am J Dis Chil* 1975; 125: 450-455.
14. Chandra RK. Interactions between early nutrition and the immune system. In: Barker DJL, Whelan J. *The childhood environment and adult disease*. Siba Foundation Symposium # 156. London: Wiley; 1991: 77-88.
15. Gurr MI. The role of lipids in the regulation of the immune system. *Prog Lip Res* 1983; 22: 257-287.
16. Jacob RA, et al. Immunocompetence and oxidant defense during ascarbate depletion of healthy men. *Am J Clin Nutr* 1991; 54: 1302s-1309s.

2. NUTRITIONAL DEFICIENCIES AND HIV/AIDS.

17. Jain VK, Chandra RD. Does nutritional deficiency predispose to acquired immunodeficiency syndrome? *Nutr Res* 1984; 4: 537.
18. Beach RS, Laura PF. Nutrition and the acquired immunodeficiency syndrome. *Ann Intern Med* 1983; 99: 565-566.
19. Gray RH. Similarities between AIDS and PCM (Protein Caloric Malnutrition). *Amer J Publ Health* 1983; 73: 1332.
20. Keusch GT, Farthing MJG. Nutritional aspects of AIDS. *Annu Rev Nutr* 1990; 10: 475-501.

21. Coodley GO. Nutritional deficiency and AIDS. *Ann Intern Med* 1990; 113: 809.

22. Bristol-Myers. *Malnutrition and the immune response: Focus on cancer and AIDS.* Princeton, NJ; 1994; 26.

23. Raiten DJ. *Nutrition and HIV infection: A review and evaluation of the extant knowledge of the relationship between nutrition and HIV infection.* FDA Contract No. 223-88-2124; 1990.

24. Keusch GT, Thea DM. Malnutrition in AIDS. *Med Clin NA* 1993; 77(4): 795-814.

25. Beisel WR. AIDS. In: Gershwin ME, German JB, Keen CL. *Nutrition and immunology: Principles and practice.* Totowa, NJ: Human Press; 2000; 389-402.

26. Watson RR. *Nutrition and AIDS.* 2nd Ed. Boca Raton: CRC Press; 2001: 231.

27. Chelluri L, Jastremski MS. Insidence of malnutrition in patients with acquired immunodeficiency syndrome. *Nutr Clin Pract* 1989; 4: 16-18.

28. Coodley GO *et al.* Micronutrient concentrations in the HIV wasting syndrome. *AIDS* 1993a; 7: 1595-1600.

29. Kotler DP et al. Body composition studies in patients with the acquired immunodeficiency syndrome. *Am J Clin Nutr* 1985; 42: 1255-1265.

30. Chlebowski RT. Significance of altered nutritional status in acquired immune deficiency syndrome (AIDS). *Nutr Cancer* 1985; 7: 85-91.

31. Mahan LK, Escott -Stump S. Medical nutritional therapy for human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS). In: *Food, nutrition, and diet therapy*. Philadelphia: W.B. Saunders Company. 2000; 889-911.

32. Heller L. Nutrition support for children with HIV/AIDS. *J Am Diet Assoc* 1997; 97: 473.

33. Melchior JC et al. Resting energy expenditure is increased in stable malnourished HIV-infected patients. *Am J Clin Nutr* 1991; 53: 437-441.

34. Ehrenpreis Ed *et al.* Malabsortion and deficiency of vitamin B12 in HIV-infected patients with chronic diarrhea. *Dig Dis sci* 1994; 39: 2159-2162.

35. Ward BJ et al. Vitamin A status in HIV infection. *Nutr Res* 1993; 13: 157-166.

36. Semba RD et al. Increased mortality associated with vitamin A deficiency during human immunodeficiency virus type 1 infection. *Arch Intern Med* 1993; 153: 2149-2154.

37. Semba RD et al. Vitamin A deficiency and wasting as predictors of mortality in human immunodeficiency virus-infected injection drug users. *JID* 1995; 171: 1196-1202.

38. Karter DL et al. Vitamin A deficiency in non-vitamin-supplemented patients with AIDS: a cross-sectional study. *J AIDS Hum Retrov*irol 1995; 8: 199-203.

3. NUTRITIONAL DEFICIENCIES AND THE PROGRESSION OF HIV-POSITIVE INDIVIDUALS TO AIDS.

39. Baum MK *et al.* Micronutrients and HIV-1 disease progression. *AIDS* 1995; 9: 1051-1056.

40. Beach RS *et al.* Specific nutrient abnormalities in asymptomatic HIV-1 infection. *AIDS* 1992; 6: 701-708.

41. Moseson M et al. The potential role of nutritional factors in the induction of immunologic abnormalities in HIV-positive homosexual men. *JAIDS* 1989; 2: 235-247.

42. Tang AM et al. Dietary micronutrient intake and risk progression to acquired immunodeficiency syndrome (AIDS) in human immunodeficiency virus type 1 (HIV-1)-infected homosexual men. *Am J Epidemiol* 1993; 138: 1-15.

43. Bogden JD et al. Micronutrients status and human immunodeficiency virus (HIV) infection. *Ann NY Acad Sci* 1990; 547: 189-195.

44. Abrams B et al. A prospective study of dietary intake and AIDS in HIV- seropositive homosexual men. *JAIDS* 1993; 6: 949-958.
45. Skurnick JH, et al. Micronutrients profiles in HIV-1-infected heterosexual adults. *J Acq Immundef Syndr Hum Retrov* 1996; 12: 75-83.
46. Periquet BA *et al.* Micronutrient level in HIV-1 infected children. *AIDS* 1995; 9: 887-893.
47. Fawzi WW, Hunter DJ. Vitamins in HIV disease progression and vertical transmission. *Epidemiology* 1998; 9: 457-466.
48. Fawzi WW et al. A randomized trial of vitamin A supplements in relation to mortality among human immunodeficiency virus-infected and uninfected children in Tanzania. *Pediatr Infect Dis* J 1999; 18: 127-133.
49. Tang AM *et al.* Association between serum vitamin A and E levels and HIV-1 disease progression. *AIDS* 1997; 11: 613-620.
50. Ullrich R *et al.* Serum carotene deficiency in HIV-infected patients. *AIDS* 1994; 8: 661-665.
51. Coutsoudis A et al. The effects of vitamin A supplementation on the morbidity of children born to HIV-infected women. *Am J Public Health* 1995; 85: 1076-1081.
52. Semba RD et al. Vitamin A deficiency and wasting as predictors of mortality in human immunodeficiency virus-infected injection drug users. *JIF* 1994; 171: 1196-1202.
53. Semba RD. Vitamin A and human immunodeficiency virus infection. *Proc Nutr Soc* 1997; 56: 1-11.
54. Landesman S. Vitamin A relationships to mortality in HIV disease and effects on HIV infection: recent and late breaking studies. Presented at forum, Lawton Chiles International House, *National Institutes of Health*, Bethesda, MD, May 16, 1996.
55. Tang AM et al. Association between serum vitamin A and E levels and HIV-1 disease progression. *AIDS* 1997; 11: 613-620.
56. Garewal HS et al. A preliminary trial of beta-carotene in subjects infected with the human immunodeficiency virus. *J Nutr* 1992; 122: 728-732.
57. Ullrich R et al. Serum carotene deficiency in HIV-1 infected patients. *AIDS* 1994; 8: 661-665.
58. Loya S et al. The carotenoid halocynthiaxanthin: a novel inhibitor of the reverse transcriptase of human immunodeficiency viruses type 1 and type 2. *Arch Biochem Biophys* 1992; 293: 208-212.
59. Watson RR et al. Enhanced survival by vitamin A supplentation during retrovirus infection causing murine AIDS. *Life Sci* 1988; 43: 13-18.
60. Yang Y et al. Retinoic acid inhibition of *ex vivo* human immunodeficiency virus-associated apoptosis of peripheral blood cells. *Proc Natl Acad Sci USA* 1995; 92: 3051-3055.
61. Harakeh S, Jariwalla RJ, Pauling L. Suppression of human immunodeficiency virus replication by ascarbate in chronically and acutely infected cell. *Proc Natl Acad Sci U.S.A.* 1990; 87: 7245-7249.
62. Harakeh S et al. Mechanistic aspects of ascarbate inhibition of human immunodeficiency virus. *Chemico-biological Interactions* 1994; 91: 207-215.
63. Tang AM et al. Association between serum vitamin A and E levels and HIV-1 disease progression. *AIDS* 1997; 11: 613-620.
64. Wang Y et al. Nutritional status and immune responses in mice with murine AIDS are normalized by vitamin E supplementation. *J Nutr* 1994; 124: 2024-2032.
65. Wang Y et al. Modulation of immune function and cytokine production by various levels of vitamin E supplementation during murine AIDS. *Immunopharmacol* 1995; 29: 225-233.
66. Tang AM et al. Low serum vitamin B12 concentrations are associated with faster human immunodeficiency virus type 1 (HIV-1) disease progression. *J Nutr* 1997; 127: 345-351.
67. Baum MK et al. Association of vitamin B6 status with parameters of immune function in early HIV-1 infection. *JAIDS* 1991; 4: 1122-1132.

68. Haug C *et al.* Subnormal serum concentration of 1 ,25 -vitamin D in HIV infection: Correlation with degree of immune deficiency and survival. *JID* 1994; 169: 889-893.

4. NUTRITIONAL DEFICIENCIES AND THE "TRANSMISSION" OF HIV/AIDS.

69. Mehendale SM et al. Low carotenoid concentration and the risk of HIV seroconversion in Pune, India. *JAIDS* 2001; 26: 352-359.
70. McDonald KS et al. Vitamin A and risk of HIV-1 seroconversion among Kenyan men with genital ulcers. *AIDS* 2001; 15: 635-639.
71. Moore PS et al. Role of nutritional status and weight loss in HIV seroconversion among Rwandan women. *JAIDS* 1993; 6: 611-616.
72. Fawzi WW et al. Randomized trial of vitamin supplements in relation to vertical transmission of HIV-1 in Tanzania. *JAIDS* 2000; 23: 246-254.
73. Fawzi WW et al. Randomized trial of effects of vitamin supplements on pregnancy outcomes and T cell counts in HIV-1-infected women in Tanzania. *Lancet* 1998; 351: 1477-1482.
74. Landers DV. *1995 Nutrition and immune function II*: Maternal factors influencing transmission. Nutrition in pediatric HIV infection: Setting the research agenda, Bethesda, MD, September 1995.
75. Stiehm RE. Newborn factors in maternal-infant transmission of pediatric HIV infection. In: *Nutrition in pediatric HIV infection*: Setting the Research Agenda. Bethesda, MD: NIH, September 1995.
76. Greenberg BL et al. Vitamin A deficiency and maternal-infant transmission of HIV in two metropolitan areas in the United States. *AIDS* 1997; 11: 325-332.
77. Semba RD et al. Maternal vitamin A deficiency and mother-to-child transmission of HIV-1. *Lancet* 1994; 343: 1593-1597.
78. Phuapradit W et al. Serum vitamin A and betha-carotene levels in pregnant women infected with human immunodeficiency virus-1. *Ostet Gynecol* 1996; 87: 564-567.
79. Semba RD et al. Infant mortality and maternal vitamin A deficiency during human immunodeficiency virus infection. *Clin Inf Dis* 1995; 21: 966-972.
80. Coutsoudis A et al. Ramdomized trial testing the effect of vitamin A supplementation on pregnancy outcomes and early mother-to-child HIV-1 transmission in Durban, South Africa. *AIDS* 1999; 13: 1517-1524.
81. Lan Y et al. Carotenoid status of pregnant women with and without HIV infection in Malawi. *East Afr Med J* 1999; 76: 133-137.
82. Semba RD *et al.* Maternal vitamin A deficiency and mother-to-child transmission of HIV-1. *Lancet* 1994a; 343: 1593-1597.
83. Semba RD et al. Maternal vitamin A deficiency and child growth failure during human immunodeficiency virus infection. *JAIDS* 1997; 14: 219-222.
84. Coutsoudis A et al. Effect of vitamin A supplementation on viral load in HIV-1 infected pregnant women. *JAIDS* 1997; 15: 86-87.
85. John GC et al. Genital shedding of human immunodeficiency virus type 1 DNA during pregnancy: association with immunosuppression, abnormal cervical or vaginal discharge, and severe vitamin A deficiency. *JID* 1997; 175: 57-62.
86. Mostand SB et al. Hormonal concentration, vitamin A deficiency, and other risk factors for shedding of HIV-1 infected cells from cervix and vagina. *Lancet* 1997; 350: 922-927.
87. Shah RS *et al.* Liver stores of vitamin A in human fetuses in relation to gestational age, fetal size and maternal nutritional status. *Br J Nutr* 1987; 58: 181-189.
88. Nduati RW et al. Human immunodeficieny virus type-1 infected cells in breast milk: association with immunosuppression and vitamin A deficiency. *JID* 1995; 172: 1461-1468.
89. Landesman S. *Vitamin A relationships to mortality in HIV disease and effects on HIV infection: recent and late breaking studies.* Presented at forum, Lawtom Chiles International House, National Institutes of Health, Bethesda, MD, May 16, 1996.

5. OXIDATIVE STRESS AND HIV/AIDS.

90. Dworkin BM, Rosenthal W, Wormser G, Weiss L. Selenium deficiency in the acquired immuno-deficiency syndrome. *J Parenteral Enteral Nutr* 1986; 10: 405.
91. Papadopulos-Eleopulos E. Reappraisal of AIDS – Is the oxidation induced by the risk factor the primary cause? *Medical Hypothesis* 1988; 25: 151-162.
92. Favier A. The place of oxygen free radicals in HIV infection. A collection of papers presented at a conference on "The place of oxygen free radicals in HIV infection", Les Deux Alpex, France, January 1993. *Chemico-Biological Interactions* 1994; 91: 77-224.
93. Piette et al. Molecular mechanisms of virus activation by free radicals. Collection of 5 articles presented at a conference on "The place of oxygen free radicals in HIV infection" Les Deux Alpes, France, January 1993. *Chemico-Biological Interactions* 1994; 91: 79-132.
94. Fuchs J et al. Oxidative imbalance in HIV infected patients. *Med Hypothesis* 1991; 36: 60-64.
95. Shallenberger F. Selective compartmental dominance: An explanation for a nonifectious, multifactorial etiology for acquired immune deficiency syndrome (AIDS), and a rationale for ozone therapy and other immune modulating therapies. *Med Hypothesis* 1998; 50:67-80.
96. Jarstrand C, Akerlund B. Oxygen radical release by neutrophils of HIV-infected patients. *Chemico-Biological Interactions* 1994; 91: 141-146.
97. Salvain B, Mark AW. The role of oxidative stress in disease progression in individuals infected by the human immunodeficiency virus. *J Leukocyte Biol* 1992; 52: 111.
98. Baruchel S, Wainberg MA. The role of oxidative stress in disease progression in individuals infected by the human immunodeficiency virus. *J Leukocyte Biol* 1992; 51: 111-114.
99. Polkyov VM et al. Superoxide anion production and enzymatic disbalance in peripheral blood cells isolated from HIV-infected children. *Free Radic Biol Med* 1994; 16: 15-21.
100. Hommes MJT et al. Resting energy expenditure and substrate oxidation in human immunodeficiency virus (HIV)-infected asymptomatic men: HIV affects host metabolism in the early asymptomatic stage. *Am J Clin Nutr* 1991; 54: 311-315.
101. Passi S. Progressive increase of oxidative stress in advanced human immunodeficiency. *Continuum* (London) 1998; 5(4): 20-26.
102. Favier A et al. Antioxidant status and lipid peroxidation in patients infected with HIV. *Chemico-Biological Interactions* 1994; 91: 165-180.
103. Fuchs J et al. Oxidative imbalance in HIV infected patients. *Med Hypothesis* 1991; 36: 60-64.
104. Lacey CJ et al. Antioxidant-micronutrients and HIV infection. *International J STD & AIDS* 1996; 7: 485-489.
105. Javier JJ et al. Antioxidant micronutrients and immune function in HIV-1 infection. *FASEB Proc* 1990; 4A: 940-945.
106. Allard VP et al. Oxidative stress and plasma antioxidant micronutrients in humans with HIV infection. *Am J Clin Nutr* 1998; 67: 143-147.
107. Revillard JP, Favier AE, Zittoun M. Lipid peroxidation in human immunodeficiency virus infection. *J AIDS* 1992; 5: 637-638.
108. Constants J et al. Fatty acids and plasma antioxidants in HIV-positive patients: correlation with nutritional and immunological status. *Clinical Biochemistry* 1995; 28: 421-426.
109. Dworkin BM. Selenium deficiency in HIV infection and the aquired immunodeficiency syndrome (AIDS). *Chemico-Biological Interactions* 1994; 91: 181-186.
110. Cirelli A et al. Serum selenium concentration and disease progress in patients with HIV infection. *Clin Biochem* 1991; 24: 211-214.
111. Schrauzer GN, Sacher J. Selenium in the maintenance and therapy of HIV-infected patients. *Chem Biol Inter* 1994; 91: 199.

112. Simon G et al. Effects of glutathione precursors on huma immunodeficiency virus replication. *Chemico-Biological Interactions* 1994; 91: 217-224.
113. Staal FJT et al. Intracellular glutathione levels in T-cells subsets decrease in the blood plasma of HIV-1 infected patients. *Biol Chem Hoppe Seyler* 1989; 370: 101-108.
114. Buhl R et al. Systemic glutathion deficiency in symptom-free HIV seropositive individuals. *Lancet* 1989; ii: 1294-1298.
115. Dorge W, Eck HL, Mihm S. HIV-induced cysteine deficiency and T-cell dysfunction: a rationale for treatment with n-acetyl-cysteine. *Immunol Today* 1992; 13: 211.
116. Passi S et al. Study on plasma polyunsaturated fatty acids and vitamin E, and on erythrocyte glutathion peroxidase in highrisk HIV infection categories and AIDS patients. *Clin Chem Enzym Comns* 1993; 5: 169-177.
117. Quey B et al. Glutathione depletion in HIV-infected patients: role of cysteine deficiency and effect of oral n-acetyl-cysteine. *AIDS* 1992; 5: 814.
118. Kalevic T et al. Suppression of human immunodeficiency virus expression in chronically infected monocytic cells by glutathione, glutathione ester and N-acetyl-cysteine. *Proc Natl Acad Sci U.S.A.* 1991; 88: 986
119. Fabris N et al. AIDS, zinc deficiency and thymic hormone failure. *JAMA* 1988; 259: 839.
120. Walter R et al. Zinc status in human immunodeficiency virus infection. *Life Sci* 1990; 47: 1579.
121. Falutz J et al. Zinc as a cofactor in HIV-induced immunosuppression. *JAMA* 1988; 259: 2850-2851.
122. Graham N et al. Relationship of serum cooper and zinc levels to HIV-1 seropositivity and progression to AIDS. *JAIDS* 1991; 4: 976-980.
123. Staal FJT et al. Antioxidants inhibit stimulation of HIV transcription. *AIDS Res Hum Retroviruses* 1993; 9: 299-306.
124. Kehrer JP. Free radicals as mediators of tissue injury and disease. *Crit Rev Toxicol* 1993; 23: 21-48.
125. Slater TF. Free radicals: formation, detection, reactivity, and citoxicity. In: Lachman PJ, Peters SK, Rosen FS, Walport MJ. *Clinical aspects of immunology*. Boston: Blackwell Scientific Publications; 1993: 377-393.
126. Reid L. Oxidative stress and antioxidants. A nutritional perspective. *Continuum* (London) 1998; 5(3): 52-54.
127. Papadopulos-Eleopulos E. Looking back on the oxidative stress theory of AIDS. *Continuum* (London) 1998/1999; 5(5): 30-35.
128. Papadopulos-Eleopulos E, et al. Oxidative stress, HIV and AIDS. *Res Immunol* 1992; 143: 145-148.
129. Giraldo RA. Role of free radicals in immunodeficiency. In: *Aids and Stressors.* Medellín: Impresos Begón; 1997: 72-75.

6. NUTRITIONAL AND ANTIOXIDANT DEFICIENCIES AND THE PATHOGENESIS OF AIDS.

130. Peterson PK. Host defense abnormalities predisposing the patient to infection. *Amer J Med* 1984; 72: 2.
131. Playfair JHL. Overview: parasitism and immunology. In: Llachmann PJ et al. *Clinical aspects of immunology*. Boston: Blackwell Scientific Publications; 1993: 1439-1454.
132. Ware RE, Kline MW. Immunodeficiency secondary to infectious disease. In: Rich RR et al. *Clinical immunology: principles & practice.* St. Louis: Mosby; 1996: 808-826.
133. Slater TF. Free radicals mechanisms in tissue injury. *Biochem J* 1984; 222: 1-15.

134. Chandra RK. Nutrition, immunity and infection: present knowledge and future directions. *Lancet* 1983; i: 688-691.

135. Giraldo RA. Papel de las enfermedades tropicales en el debilitamiento del sistema inmunolígico y en la fisiopatogénesis del sida. In: *Sida y agentes estresantes*. Medellín: Editorial Universidad de Antioquia; 2002: 37-46.

136. Barker DJ. In utero programming of chronic disease. *Clin Sci* (Colch) 1998a; 95: 115-128.

137. Barker DJ. *Fetal and infant origins of adult diseases*. London: BMJ Publishing Group; 1992; 343.

138. Barker DJ. *Mothers, babies and diseases in later life*. London: BMJ Publishing Group; 1994.

139. Barker DJ. *Mothers, babies & health in later life*. 2nd ed. Church Press 1998b; 217.

140. Naeye RL et al. Relation of poverty and race to birth weight and organ and cell structure in the newborn. *Pediat Res* 1971; 5: 17 -22.

141. Leon DA. Fetal growth and adult disease. *Eur J Clin Nutr* 1998; 52(suppl): S72-S82.

142. Chandra RK. Fetal malnutrition and postnatal immunocompetence. *Am J Dis Child* 1975b; 129: 450-454.

143. Chandra RK. Serum thymic hormone activity and cell-mediated immunity in healthy neonates, preterm infants, and small-for-gestational age infants. *Pediatrics* 1981; 67: 407-411.

144. Ferguson AC. Prolonged impairment of cellular immunity in children with intrauterine growth retardation. *J Pediatr* 1978; 93: 52-56.

145. McDade TW *et al.* Prenatal undernutrition is associated with reduced immune function in adolescence. *FASEB* 2000; 14: A792 (abs.).

146. McDade TW *et al.* Prenatal undernutrition, postnatal environments, and antibody response to vaccination in adolescence. *Am J Clin Nutr* 2001a; 74: 543-548.

147. McDade TW *et al.* Prenatal undernutrition and postnatal growth are associated with adolescent thymic function. *J Nutr* 2001b; 131: 1225-1231.

148. Moore SE *et al.* Prenatal or early postnatal events predict infectious deaths in young adulthood in rural Africa. *Int J Epidemiol* 1999; 28: 1088-1095.

149. Lewis D, Wilson C. Developmental immunology and the role of host defenses in neonatal susceptibility. In: Remington J, Klein J. *Infectious diseases of the fetus and newborn infant*. 4th ed. Philadelphia: W.B. Saunders; 1995; 108-139.

150. Moscatelli P et al. Defective immunocompetence in foetal undernutrition . *Helv Paediatr Act* 1976; 31: 241-247.

151. Hasselbachh H *et al.* Thymus size in infants from birth until 24 months of age evaluated by ultrasound. *Acta Radiol* 1999; 40: 41-44.

152. Moore SE *et al.* Season of birth predicts mortality in rural Gambia. *Nature* 1997; 338: 434.

153.Chandra RK. Antibody formation in first and second generation offspring of nutritional deprived rats. *Science* 1975c; 190: 289-290.

154.Beach RS *et al.* Gestational zinc deprivation in mice: persistence of immunodeficiency for three generations. *Science* 1982; 281: 469-471.

155.Giraldo RA. AIDS and Stressors II: A proposal for the pathogenesis of AIDS. In: *AIDS and Stressors*. Medellín , Colombia : Impresos Begón ; 1997; 57-96.

156.Giraldo RA. AIDS and Stressors III: A proposal for the natural history of AIDS. In: *AIDS and Stressors*. Medellín , Colombia : Impresos Begón ; 1997; 97-131.

7. NUTRITIONAL AND ANTIOXIDANT THERAPY FOR THE PREVENTION AND TREATMENT OF AIDS.

157.Steinbrook R. Battling HIV on many fronts. *NEJM* 1997; 337: 779.

158. Goldberg B. AIDS. In: *Alternative Medicine. The definitive guide*. Fife, Washington: Future Medicine Publishing Inc.; 1994: 494-509.

159.Abrams DI. Alternative therapies. In: Sande MA, Volberding PA. *The medical management of AIDS*. 6a ed. Filadelfia: W.B. Saunders Company; 1999: 601-612.

160. Null G. Alternative treatments. In: *AIDS: A second opinion*. New York: Seven Stories Press; 2002: 487-581.

161.Badgley L. *Healing AIDS naturally: natural therapies for the immune system*. Foster City, California: Human energy Press; 1990: 410.

162. Byrnes S. *Overcoming AIDS with natural medicine: A program for recovery*. Revised, 2nd edition. Honolulu, Hawaii: Ecclesia Life Mana; 2001: 183.

163. Chaitow L. *The natural way: HIV & AIDS. Your guide to complementary therapies, alternative techniques and conventional treatments*. Shaftesbury, UK: Element Books Limited; 1999: 150.

164. Passi S, De Luca C. Dietetic advice for immunodeficiency. *Continuum* (London) 1998-1999; 5(5): 43-52.

165.Ferguson A, Griffin GE. Nutrition and the immune system. In: Garrow JS, James WPT, Ralph A. *Human nutrition and dietetics*. Edinburgh: Churchill Livingstone; 2000: 747-766.

166. Embid A. Inmunidad aumentada por incremento de micronutrients. *Medicinas Complementarias* 1994; No. 35: 172.

167.Embid A. Inmunoterapia a dosis infinitesimales. *Medicinas Complementarias* 1995; No. 38: 170.

168. Gerrior J, Wanke C. Nutrition and Immunodeficiency Syndromes. In: Coulston AM, Rock CL, Monsen ER. *Nutrition in the prevention and treatment of disease*. San Diego: Academic Press; 2001; 741-750.

169. Life Sciences Research Office, FASEB. Nutritional therapy and nutrition education in the care and management of AIDS patients. Tentative report, Task Order 7. Washington, DC: Center for Food Safety and Nutrition, FDA, DHHS, 1990.

170. Tang AM et al. Effects of micronutrients intake on survival in human immunodeficiency virus type 1 infection. *Am J Epidemiol* 1996; 143: 1244-1256.

171. Romeyn M. *Nutrition and HIV: A new model for treatment*. San Francisco : Jossey -Bass Publishers; 1995: 353.

172.Young J. HIV and medical nutrition therapy. *J Amer Diet Assoc* 1997; 97: S161.

173.Collins CL. Nutrition care in AIDS. Diet Curr 1988; 15: 1.

174. Fenton M, Silverman E. Medical nutritional therapy for human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS). In: Mahan K, Escott-Stump S. *Krause's Food, nutrition and diet therapy*. Philadelphia: W.B. Saunders Company; 2000: 889-911.
175. Hickson JF. Diet and nutrition for optimal immune function. In: Bahl SM, Hickson JF. *Nutritional care for HIV-positive persons: a manual for individuals and their caregivers*. Boca Raton: CRC Press; 1995: 1-36.
176. Fenton M, Silverman E. Medical nutrition therapy for human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS). In: Mahan LK, Escott-Stump S. *Food, nutrition, & diet therapy*. Philadelphia: W.B. Saunders Company; 2000: 889-911.
177. American Dietetic Association (ADA). Position of the ADA and the Canadian Dietetic Association: Nutrition intervention in the care of persons with HIV infection. *J Am Diet Assoc* 1994; 94: 1042.
178. American Dietetic Association (ADA), HIV/AIDS medical nutrition therapy protocol. Medical nutrition therapy across the continuum of care, 2nd ed. Chicago: ADA, 1998.
179. Bahl SM, Hickson JF. *Nutritional care for HIV-positive persons: A manual for individuals and their caregivers.* Boca Raton: CRC Press; 1995: 190.
180. Bendich A. Role of antioxidants in the maintenance of immune function. In: Frei. *Natural antioxidants in human health and disease.* (Chapter IV, Immunity and Infection). San Diego: Academic Press; 1994: 447-467.
181. Bendich A. Antioxidant micronutrients and immunity. In: Roitt IM, Delves PJ. *Encyclopedia of immunology*. San Diego: Academic Press; 1992: 151-153.
182. Fishman RHB. Antioxidants and phytotherapy. *Lancet* 1994: 344: 1356.
183. Turner VF. Reducing agents and AIDS - Why are we waiting? *Med J Austr* 1990; 153:502.
184. Halliwell B, Cross C. Reactive oxygen species, antioxidants and acquired immunodeficiency syndrome. *Arch Intern Med* 1991; 151: 29-31.
185. Adam ES. Antioxidant supplementation in HIV/AIDS. *Nurse Practit* 1995; 20: 8.
186. Greenspan HC. The role of oxidative oxygen species, antioxidants and phytopharmaceuticals in human immunodeficiency virus activity. *Med Hypothesis* 1993; 40: 85.
187. Greenspan HC, Arouma O. Oxidative stress and apoptosis in HIV infection: a role for plant-derived metabolites with synergistic antioxidant activity. *Immunol Today* 1994; 15: 209.
188. Greenspan HC, Arouma O. Could oxidative stress initiate programmed cell death in HIV infection? A role for plant derived metabolites having synergistic antioxidant activity. *Chemico-Biological Interactions* 1994; 91: 187-197.
189. Tang AM et al. Improved antioxidant status among HIV-infected injecting drug users on potent antiretroviral therapy. *JAIDS* 2000; 23: 321-326.
190. Byrnes SC. Staying on top of oxidative stress. Healthy and Natural Journal, Millenium Wellness Guide 1999. sbyrnes@chaminade.edu. Available in http://www.powerhealth.net.
191. Zhang Z, Inserra PF, Watson RR. Antioxidants and AIDS. In: Garewal HS. *Antioxidants and disease prevention*. Boca Raton: CRC Press; 1997; 45-66.
192. Garewal HS. *Antioxidants and disease prevention*. Boca Raton: CRC Press; 1997: 186.
193. Sies H. *Oxidative stress: oxidants and antioxidants*. London: Academic Press; 1991: 507.
194. Frei B. *Natural antioxidants in human health and disease*. San Diego: Academic Press; 1994: 588.

195.CoodleyY GO. Beta-carotene therapy in human immunodeficiency virus infection (Abstract). *Clin Res* 1991; 29: 634A.

196. Coodley GO et al. Beta-carotene in HIV infection. *JAIDS* 1993; 272-276.

197.Coodley et al. Beta-carotene in HIV infection: an extended evaluation. *AIDS* 1996; 10: 967-973.

198. Cathart R. Vitamin C in the treatment of acquired immune deficiency syndrome (AIDS). *Med Hypothesis* 1984: 14: 423.

199. Rothman KJ et al. Teratogenicity of high vitamin A intake. *NEJM* 1995; 333: 1369-1373.

200. Rogers SA. *Man does not live by bread alone: enzymes, juicing, cleansing, flushing and brushing.* In: Wellness against all odds. Syracuse, NY: Prestige Publishing; 1994: 110-144.

201. Schultz V, Hansel R, Tyler VE. Agents that increase resistance to disease: adaptogens; immune stimulants; botanical antioxidants. In: *Rational phytotherapy. A physician guide to herbal medicine*. Springer; 1998: 269-273, 273-281, 282-286.

202. Yuan-kun L et al. Modulation of immune responses. In: *Handbook of probiotics.* New York: John Wiley & Sons, Inc.; 1999: 161-177.

203. Embid A. El yogur estimula la inmunidad. *Medicinas Complementarias* 1994; No. 35: 171.

204. Giraldo RA. An effective alternative for the prevention of AIDS. Internet Discussion during the South African Presidential AIDS Advisory Panel, 2000 <http://www.robertogiraldo.com/eng/papers/AnEeffectivePreventionForAIDS.html>

205. Giraldo RA. An effective alternative for the treatment of AIDS. Internet Discussion during the South African Presidential AIDS Advisory Panel, 2000. <http://www.robertogiraldo.com/eng/papers/AnEffectiveTreatmentForAIDS.html

206. Giraldo RA, Ródenas P, Flores JJ, Embid A. Tratamiento y prevención del sida: guía de principios básicos para una alternativa no tóxica, efectiva y barata. November 2002 http://www.robertogiraldo.com/esp/articulos/Tratamiento_y_Prevencion_2002.html

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