# The Failure of Medical Science To Prevent and To Adequately Treat HIV/AIDS: An Orthomolecular Opportunity

Harold D. Foster<sup>1</sup>

#### Introduction

The book *What Really Causes AIDS*¹ is dedicated to Foinavon, the horse that won the 1967 Grand National in the slowest time and at the highest odds (444/1) ever recorded. It did so because it was so far behind the field that its jockey could avoid the utter chaos that occurred at what is now called The Foinavon Fence, when twenty or so horses fell or threw their riders.

The chaos surrounding the medical science of HIV/AIDS provides orthomolecular medicine with a similar opportunity to provide society with simple but essential strategies to prevent and treat this deadly disease. In a recent issue of this journal, an orthomolecular model was put forward to explain how HIVreplication caused AIDS by removing the nutrients required to produce glutathione peroxidase.<sup>2</sup> This publication also proved that the conventional Ho3 model of how this virus causes AIDS is incorrect. Since this is the case, it is hardly surprising that treatment protocols, based on this model, are also faulty.

## Treatment Measuring Disease Progression Conventional Predictions

In 1996, Mellors and colleagues<sup>4</sup> published a paper in *Science* claiming that the numbers produced by the viral load test could be used to accurately predict the progression of HIV-positive patients into AIDS. Viral load numbers were soon used by doctors and research scientists as

1. University of Victoria, PO Box 3050, Victoria, BC. V8R 6H3

a method of persuading healthy HIV-positive patients with high numbers to "hit early and hard with the newly approved drugs [highly toxic protease inhibitors], while AIDS doctors throughout the world started using viral load for everything from diagnosing illness to confirming HIV infection."<sup>5</sup>

The truth is that viral loads are a very poor tool to predict anything. In the Journal of the American Medical Association (JAMA), a national team of orthodox AIDS researchers, led by Rodriguez and Lederman<sup>6</sup> of Case Western Reserve University in Cleveland, presented the results of studying 2,800 untreated HIV-positive patients. They concluded that viral load measures failed in over 90 percent of cases to either predict or explain immune status. In short, the viral load test is worthless. To cite Rodriguez and colleagues6 "HIV RNA level predicts the rate of CD4 cell decline only minimally in untreated persons. Other factors, as yet undefined, are likely to drive CD4 cell losses in HIV infection".

#### Orthomolecular Predictions

The orthomolecular model, put forward by Foster, 1.2 stresses that, as HIV-positive patients progress into AIDS, the virus depletes their bodies of selenium and the amino acids glutamine, cysteine and tryptophan, so causing a decline of serum levels of glutathione peroxidase. If this is the case, then declines in such nutrients would be useful predictors of disease progression. This is indeed the case. Numerous studies have shown selenium deficiency in the plasma of individuals with HIV/AIDS. 7-9 The worse the AIDS

symptoms become, the more depressed the plasma selenium levels. Indeed, Baum and coworkers7 have demonstrated that in both HIV-1-serum positive drug-using males and females, depressed selenium plasma levels are far more accurate predictor of mortality than falling CD4 T cell counts. This also was found to be true of HIV-infected children.10 Baum and coworkers' longitudinal study, for example, collected data on CD4 T lymphocyte count, antiretroviral treatment, and plasma levels of vitamins A, E, B<sub>6</sub> and B<sub>12</sub>, and selenium and zinc. A total of 21 of the 125 participants, adult drug-users, died of HIV-related causes during this 3.5 year study. Only CD4 T lymphocyte counts over time (RR=0.69, p<0.04) and selenium deficiency (RR=10.8, p<0.002) were significantly associated with mortality, with a lack of selenium being by far the most superior indicator of who was the most likely to die of AIDS. This was true also of selenium levels in infants.<sup>10</sup>

That adults and children who quickly died of AIDS had both depressed CD4 T lymphocyte counts and very depleted plasma selenium stores<sup>7,10</sup> is no coincidence. Rather, it seems much more likely that this viral decline provides evidence of a positive feedback system in which a fall in selenium causes a reduction of CD4 T cells because this trace element is essential for the production of T lyphocytes<sup>12</sup>. Such a drop in the efficiency of the immune system can also be documented by measuring the serum levels of glutathione peroxidase, the selenoenzyme that appears to protect against viral infection and to be a prime target of HIV. These declines in the efficiency of the immune system, caused by selenium inadequacy, then allows infection by other pathogens, resulting in a further decline in selenium. The "selenium-CDT cell tailspin" is beginning its downward spiral.11

It is clear, therefore, that the way to predict the future health of HIV-positive patients is not through measuring viral loads but by assessing serum levels of selenium and/or glutathione peroxidase. This is exactly what was done in the Mengo Nutritional Trial, recently conducted in Kampala, Uganda and reported on in this journal.<sup>12</sup> This clinical trial found that as glutathione peroxidase levels rose, so too did CD4 cell count, Karnofsky scores (measuring the patients quality of life) and body weights.

In summary, patient serum selenium and glutathione peroxidase levels are far better methods of predicting future health, or lack of it, than are the measurement of viral loads. To illustrate, at the beginning of the Mengo trial, the 160 HIV-positive patients in Group A had a median CD4 cell count of 347 mm3 and median serum glutathione peroxidase levels of 3628 u/L (international Units). One year later, after taking 37 nutrients daily, the median CD4 cell count of this group had risen to 388 cells per mm<sup>3</sup> and their median serum glutathione peroxidase levels had more than doubled to 8573 uL (p=0.0001). Improvements in weight and Karnofsky scores (showing quality of life) were also highly statistically significant.<sup>12</sup> The serum levels of the selenoenzyme glutathione peroxidase appear to be the optimum indicator of future immune function and survival rates in HIV-positive individuals.

## Conventional Side Effects

There is no doubt that antiretroviral drugs, often given as the HAART cocktail, can prolong life in patients who are receiving no other form of treatment. From the time of entering HIV care, the projected life expectancy for a patient is 24.2 years. <sup>13</sup> The question arises, however, "what is the quality of this life?" To quote from *Science's* News Focus, <sup>14</sup> "Confronting the Limits of Success" six years ago, new cocktails of anti-HIV drugs transformed prospects for infected people in industrialized countries. Now, serious limitations have become apparent. Indeed, two years after the

introduction of HAART, new side effects began to appear in treated patients. These included nausea and anemia, and odd distributions of fat known as lipodystrophy. Other metabolic abnormalities have since developed that lead to diabetes-like problems, heart disease and brittle bones. Research has appeared proving that a common mainstay of HAART, drug AZT (3'-Azido-3'-deoxythymidine), is likely to be carcinogenetic.<sup>15</sup> A European study also has shown that deaths from liver-related disease among HIV-positive patients with similar CD4 cell counts has increased since the introduction of HAART.<sup>16</sup> So too has HIV-associated renal disease.<sup>17</sup> Similarly, the use of combined antiretroviral therapy is a strong independent risk factor for subclinical carotid atherosclerosis in drug-treated HIV patients, clearly showing its cardiovascular toxicity.<sup>18</sup>

In addition to the physical decline associated with antiretrovirals, mental disorders also are unusually common among HIV-patients treated with them.<sup>19</sup> One of the reasons for this is that these drugs tend to be large molecules that cannot pass the blood-brain barrier. As a result, the brain acts as a sanctuary for HIV.<sup>20</sup> 3-D scans, for example, reveal tissue damage in the brains of many AIDS patients. In colour-coded images, researchers have shown that there may be as high as 15 percent tissue losses in the centres of the brain that regulate movement and co-ordination. Thinning also is seen in the reasoning and language centres.<sup>21</sup> In addition to these brain scans, the National Institute of Mental Health are funding a long-term brain function study of HIV positive patients that hopes to enroll about 1,600 drug treated individuals. Initial findings already suggest that about 50 percent of such patients have subnormal performance. To quote Clifford<sup>20</sup> "They might be slower on computer keyboards, working crossword puzzles or have difficulty keeping track of what's been said in a conversation. They may even begin to move more slowly".

In summary, antiretrovirals are keeping HIV-infected patients alive longer. However, as a consequence of the properties and side-effects of these drugs, patients are developing lipodystrophy, diabetes, liver and kidney problems, carotid atherosclerosis and brain thinning and associated subnormal performance. It is also likely that they are increasing their risk of subsequently developing cancer.

#### Orthomolecular Side Effects

The orthomolecular treatment of HIV/AIDS involves diets that are high in specific nutrients that are essential for all human survival. Nutritional intake must specifically provide elevated levels of those nutrients, selenium, cysteine, glutamine and tryptophan, that HIV replications removes from the body. There is no evidence that these nutrients, especially if provided in foods such as Brazil nuts, spirulina, desiccated liver or yogurt, cause any adverse side effects. Naturally, they should not be ingested at abnormally high levels.

The author has published several articles, describing open and closed trials, that have established that such nutrients can reverse the downward HIV/AIDS spiral, even when AIDS patients are very close to death.<sup>24</sup> Indeed, many of the orthomolecularly treated HIV/AIDS patients claim to be healthier than at any previous time in their lives. Selenium, of course, is known to extend lifespan and reduce the risk of death from cancer<sup>25</sup> and cardiovascular disease.<sup>26</sup>

#### Costs

## Conventional Expenses

According to Schackman and coworkers,<sup>27</sup> from the time of entering HIV care, the average patient life expectancy is an additional 24.2 years. Such a patient's medical care lifetime discounted costs,

in the United States, are \$385,200. Undiscounted cost is \$618,900 for adults who begin antiretroviral treatment with CD4 cell counts <350/muL. Seventy-three percent of these predicted costs are to pay for antiretroviral medications, 13 percent for inpatient care, 9 percent outpatient care and 5 percent other HIV-related medications and laboratory costs. If antiretroviral treatment begins in a patient with a CD4 cell count of <200/muL, projected survival time is 22.5 years, with a discounted lifetime cost of \$354,100 and an undiscounted cost of \$567,000.

## Orthomolecular Expenses

It is not yet possible to provide similar data for orthomolecular treatment. However, in South Africa, Uganda and Zambia, trials with HIV/AIDS patients have been successfully treated for a year or longer with nutrients costing between \$60 and \$300 annually.<sup>23,24</sup> These patients have, in many cases, proved so healthy after a few months that it is possible that over a lifetime their treatment cost will be effectively negative. That is, they will require less medical treatment than they would have received had they not been HIV-positive and, therefore, not receiving nutritional supplements. Since they are generally well enough to quickly return to work, the addition of their wages to the economic equation would certainly result in negative costs for the orthomolecular treatment of HIV/AIDS in such Third World patients.

#### Prevention

## Conventional Strategies: Vaccines

In April 1984, the HHS secretary Margaret Heckler held a press conference to announce that Dr. Robert Gallo of NCI had discovered the cause of AIDS, the retrovirus HTLV-III (later called HIV). She expressed hope that a vaccine against this virus would be produced within two years.<sup>28</sup>

Heckler was no Nostradamus, more than twenty-three years later there is no vaccine for HIV. To quote Horton,<sup>29</sup> writing in 2004:

"But contrary to the predictions and promises of most AIDS experts, the signs are that a vaccine to prevent HIV infection will not be found for, at the very least, several decades to come - if at all. Those responsible for carrying on the global fight against AIDS do not accept this grim outlook, at least publicly. Yet it is a conclusion, based on all the evidence gathered so far, which increasingly defies rebuttal. Until the gravity of this scientific failure is openly acknowledged, a serious debate about how to end HIV's lethal grip on some of the poorest and most vulnerable human populations in the world cannot take place."

Of course, this criticism by Horton is not new. In 1992, Albert Sabin, developer of the oral polio vaccine claimed: "...the available data provides no basis for testing any experimental vaccine in human beings or for expecting that any HIV vaccine could be effective in human beings." <sup>30</sup>

Nevertheless, the search goes on. In 2004, for example, world expenditure on AIDS vaccine research was between \$600 million to \$700 million, \$582 million being provided by the United States.<sup>31</sup>

What has medical science to show for the billions of dollars spent in AIDS vaccine research? Well, in 2007, the cream of the crop vaccine, being tested in the "STEP Study" failed spectacularly. Not only did the vaccine not prevent HIV infection, but those receiving it proved more likely to be infected with the virus than those volunteers receiving the placebo.<sup>32</sup> This study included 3,000 males and females, aged 18 to 45 who, because of their lifestyles, were considered at high risk of HIV infection. One hundred of these volunteers were from Seattle, the rest were drawn from 15 other US cities and from Peru, Brazil,

Canada, Australia, Jamaica, Haiti, Puerto Rico and the Dominican Republic.

Orthomolecular Strategies: Soil Remineralization

What are the reasons for the timing of the first AIDS pandemic, and indeed for the increased ability of viruses to cross the species barrier from animals to humans?<sup>33,34</sup> There seems to be a minimum daily dietary selenium intake above which, as seen in Senegal<sup>6</sup> and Finland,<sup>35</sup> HIV cannot be easily transmitted. This appears to be because the body's antioxidant defense system, especially the selenoenzyme, glutathione peroxidase, acts as an internal defense against viral infection, preceding the formation of antibodies. For this reason, HIV is having its greatest difficulty in infecting those with diets elevated, either naturally or by design, in the trace element selenium and in the amino acids cysteine, glutamine and tryptophan. Together these nutrients stimulate the body's production of glutathione peroxidase.36-37

As Foster wrote in the Well Being *Journal*:<sup>34</sup> If this is correct, any drop in selenium in the food chain would naturally encourage the diffusion of HIV and indeed many other viruses. In the second half of the twentieth century, coal combustion more than doubled, oil consumption increased by a factor of almost 8 and natural gas was used as a fuel at a rate of roughly 11 times that of 1950. Simultaneously, large parts of the earth were deforested and much of the wood burned. The resulting high levels of sulphur and nitrogen, emitted into the atmosphere, were largely converted into sulfuric and nitric acids, increasing the acidity of associated precipitation. Acid rain altered soil pH and so reduced selenium's bioavailability. Similarly, potassium, nitrogen and phosphorous in commercial fertilizers are further depressing the uptake of selenium in crops. These processes reduce the dietary intake of selenium by humans,

animals and insects, triggering viral mutation and promoting associated pandemics. Naturally, the effects of decreased selenium bioavailability have been most obvious in those unfortunate regions, like sub-Saharan Africa, where levels of this trace element are naturally depressed in the food chain. This is the fundamental reason why sub-Saharan Africa is so badly affected by the HIV/AIDS pandemic.

Field trials in China have shown that the addition of selenium to fertilizers, table salt and/or animal fodder can greatly reduce Keshan disease by slowing the diffusion of coxsackievirus B.<sup>38</sup> This is also true of the hepatitis B and C viruses and liver cancer.<sup>39,40</sup> The addition of selenium to fertilizers in Finland, mandated nationally in 1984, has apparently also significantly reduced the incidence of HIV infection.<sup>38</sup>

It seems that, for a fraction of the money spent with no return on HIV vaccine research, the AIDS and other viral pandemics could be halted by increasing the global dietary intake of selenium. Such a strategy would be particularly effective if combined with efforts to increase protein consumption worldwide.

## Conclusion

As William A. Hasteltine<sup>41</sup> pointed out in a 1992 lecture to the French Academy of Sciences, "The future of AIDS is the future of humanity". Hasteltine, the then chief retrovirologist at Harvard's Dana-Farber Cancer Institute, went on to add that "unless the epidemic of AIDS is controlled, there is no predictable future for our species". Soon afterward he testified to a US Senate hearing,<sup>42</sup> pointing out that by the year 2000 we might see 50 million people who had been infected by HIV. In his opinion, by 2015 the total number dead or dying from this cause could reach one billion, that is about one sixth of the current global population. Hasteltine may have been a little optimistic: the AIDS

pandemic has not been controlled and by the end of 2000, 57.9 million people had been infected with HIV, 21.8 million of whom were already dead.<sup>43</sup> We are at, or near, the tipping point.<sup>44</sup> If the orthomolecular strategies described in this article are not applied globally, very soon there will be, as Hasteltine suggested, no predictable future for our species.

#### References

- 1. Foster HD: *What really causes AIDS*. Victoria: Trafford Publishing, 2002.
- Foster HD: How HIV replication causes AIDS: an orthomolecular model. *J Orthomol Med*, 2007; 22(3): 123-128.
- 3. Ho DD: Viral counts in HIV infection. *Science*, 1996; 272(5265): 1124-1125.
- Mellors JW, Rinaldo CR Jr., Gupta P, White RM, Todd JA, Kingsley LA: Prognosis in HIV-1 infection predicted by the quantity of virus in plasma. *Science*, 1996; 272(5265): 1167-1170.
- Alive and Well AIDS Alternatives. The Failure of Viral Load Tests. JAMA Study Shakes AIDS Science, Angers HIV Advocates. http://www. aliveandwell.org/
- Rodriguez B, Sethi AK, Cheruvu VK, Mackay W, Bosch JR et al: Predictive value of plasma HIV RNA level on rate of CD4 T-cell decline in untreated HIV infection. *JAMA*, 2006; 296(12): 149-1506
- 7. Baum MK, Shor-Posner G, Lai S, Zhang G, Lai H et al: High risk of HIV-related mortality is associated with selenium deficiency. *J Acquired Immune Deficiency Syndromes and Human Retrovirology*, 1997; 15(5): 370-374.
- Dworkin BM: Selenium deficiency in HIV infection and the Acquired Immunodeficiency Syndrome (AIDS). Chemico-Biological Interactions 1994; 91(2-3): 181-186.
- Schrauzer GN, Sacher J: Selenium in the maintenance and therapy of HIV-infected patients. *Chemico-Biological Interactions*, 1994; 91(2-3): 199-205.
- Campa A, Shor-Posner G, Indacochea F, Zhang G, Lai H, et al: High risk of HIV-related mortality is associated with selenium deficiency. *J Acquired Immune Deficiency Syndromes and Human Retrovirology*, 1997; 15(5): 508-513.
- 11. Foster HD: AIDS and the "Selenium CD4T cell Tailspin". The geography of a pandemic. *Townsend Lett Doctors Patients* 2000: 290: 94-99.
- 12. Namulemia E, Sparling J, Foster HD: Nutri-

- tional Supplements can delay the progression of AIDS in HIV-infected patients: results from a double-blinded, clinical trial at Mengo Hospital, Kampala, Uganda. *J Orthomol Med*, 2007; 22(3): 129-136.
- Schackman BR, Gebo KA, Walensky RP, Losina E, Muccio T et al: The lifetime cost of current human immunodeficiency virus care in the United States. *Med Care* 2006; 44(11): 990-997.
- 14. Jon Cohen. Confronting the limits of success. *Science*, 2002; 296(5577): 2320-2324.
- 15. National Toxicology Program. NTP Toxicology and Carcinogenesis studies of AZT (CAS No. 30516-87-1) and AZT/alpha-Interferon A/D B6C3F1 Mice (Gavage Studies) *Natl Toxicol Program Tech Rep Ser* 1999; 469: 1-361.
- Harding A: European HIV study finds rise in liver-related deaths. Reuters January 2002. http://www.medscape.com/viewarticle/ 520754?src=mp.
- 17. Röling J, Schmid H, Fischereder M, Draenert R, Goebel FD: HIV-associated renal diseases in highly active antiretroviral therapy-induced nephropathy. Clin Infect Dis, 2006; 42(10): 1488-1495.
- Jeric, Knobel H, Calvo N, Sorli ML, Guelar A: Subclinical carotid atherosclerosis in HIV-infected patients: role of combination antiretroviral therapy. Stroke, 2006; 37(3): 812-817.
- Pence BW, Miller WC, Gaynew BN, Eron JJ Jr: Psychiatric illness and virologic response in patients in initiating highly active antiretroviral therapy. J Acquir Immune Defic Syndr 2007; 44(2): 159-166.
- 20. Brink S: Brain escapes benefits of antiretrovirals. *Guardian Weekly*, November 25-December 1, 2005: 5.
- 21. Thompson PM, Dutton RA, Hayashi KM, Lu A, Lee SE: 3D mapping of ventricular and corpus callosum abnormalities in HIV/AIDS. *Neuroimage*, 2006; 31(1): 12-23.
- 22. Clifford cited by Brink op.cit.
- 23. Foster HD: HIV/AIDS a nutritional deficiency disease. *J Orthomol Med*, 2005, 20(2):67-69.
- Bradfield M, Foster HD: The successful orthomolecular treatment of AIDS: accumulating evidence from Africa. *J Orthomol Med*, 2006, 21(4):193-196.
- Foster HD: The geography of disease family trees: the case of selenium in Bobrowsky PT (ed). Geoenvironmental Mapping: Methods, Theory and Practice. Rotterdam: Balkema: 497-529.
- 26. Foster HD: Coxsackie B virus and myocardial infarction. *Lancet*, 2002; 359(9308): 804.

- 27. Schackman BR, Gebo KA, Walensky RP, Losina E, Muccio T et al: The lifetime cost of current human immunodeficiency virus care in the United States. *Med Care*, 2006, 44(11): 990-997.
- 28. In Their Own Words .... NIH Researchers Recall the Early Year of AIDS. Timeline, 1981-1988. http://72.14.253.104/search?q=cache: Ko8kWXqcaQQJ:aidshistory.nih.gov/timeline/ index.html+vaccine+ALOS+prediction+histor y&hl=en&ct=clnk&cd=2&gl=ca
- 29. Horton R: AIDS: The Elusive vaccine. The New York Review of Books 2004; 51(14): 1-7. http://www.nvbooks.com/articles/17400.
- Sabin A: cited in Miller SK. HIV strategy will fail says vaccine veteran. New Scientist, September 1992: 8.
- Associated Press. Foxnews.com. AIDS vaccine funding shrinking. http://www.foxnews.com/story/0,2933,148281,00.html.
- 32. Song KM, Ostrom CM: Failure of AIDS vaccine punctures soaring hopes. *The Seattle Times*, November 8, 2007.
- 33. Henderson M: Times in Line. August 15, 2006. Deadly viruses mutating to infect humans at rates never seen before. www.timesonline. co.uk/article/0.3-2049697.html.
- 34. Foster HD. A nutritional solution to AIDS and other viral pandemics. *Well Being Journal*, November/December 2006, 15(6): 1-10.
- 35. Foster HD. AIDS in Finland. *J Orthomol Med*, 2005; 20(3): 221-222.
- 36. Foster HD. The role of the antioxidant defense system in preventing the transmission of HIV. *Med Hypoth*, 2007; 69(6):1277-1280.
- Foster HD: Host-pathogen evolution: implications for the prevention and treatment of malaria, myocardial infarction and AIDS. *Med Hypoth*, 2008; 70(1): 21-25.
- 38. Cheng YY: Selenium and Keshan disease in Sechuan Province, China. In: Combs GF Jr, Spallholz JE, Levander OA, Oldfield JE (eds). Selenium in Biology and Medicine. New York: Van Nostrand Reinhold, 1987: 877-891.
- 39. Yu SY, Li WG, Zhu YJ, Yu WC et al: Chemoprevention trials of human hepatitis with selenium supplementation in China. *Biol Trace Elem Res*, 1989; 20: 15-22.
- 40. Yu SY, Hu YJ, Li WG, Huang QS, Huang CZ et al: A preliminary report on the intervention trials of primary liver cancer in high-risk populations with nutritional supplementation of selenium in China. *Biol Trace Elem Res*, 1991; 29: 289-294.
- 41. Hasteltine WA: cited in More cases, same old question. The Philadelphia Inquirer June 6,

- 1993. Review and Opinion D1.
- 42. Hasteltine WA: cited in Large AIDS increases predicted by early 2005. *The Vancouver Sun* December 15, 1992. A12.
- 43. Worldwatch Institute. *Vital Signs 2001. The Trends that are Shaping our Future*. New York: WW Norton, 2001.
- 44. Gladwell M. The Tipping Point: *How Little Things Can Make a Big Difference*. New York: Little, Brown and Company, 2002.