

How HIV Replication Causes AIDS: An Orthomolecular Model

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Introduction

Dr. David Ho's¹ mathematical model, purportedly able to explain how HIV replication leads to AIDS, is mortally wounded. Indeed, any moderately observant pathologist would undoubtedly declare it already dead. This is a major setback for the medical establishment because it is Ho's so-called "runaway hypothesis", an uncontrolled cycle of T cell activation, infection, HIV replication and cell destruction that eventually culminates in AIDS, which provides the base on which the hit-hard-hit-early current anti-retroviral protocol for the treatment of HIV infection rests. According to Ho, HIV replicates rapidly from the moment of initial infection at a rate that is just faster than the body's ability to mass produce the CD4 (+) T cells that are needed to defend against it. As a result, viral replication outpaces the immune system until the latter reaches the point of collapse. To prevent this destruction, Ho's¹ mathematical model calls for the immediate treatment of all newly identified HIV-positive patients with a continuous cocktail of anti-retroviral drugs.

Time and Timing

Ho, therefore, presents the route from initial HIV infection to AIDS as a positive feedback system. Such systems, for example avalanches and forest fires, expand their impacts very rapidly because their growth is exponential. For this fundamental reason, mathematicians tend to dislike Ho's model. To illustrate, Mark Craddock,² from the University of Technology, Sydney, Australia, has pointed out that if Ho were correct, over the ten years

AIDS usually takes to develop in untreated First World HIV-positive patients, they would each "produce more particles of HIV than there are atoms in the universe". More recently, Yates and colleagues,^{3,4} from Emory University in Atlanta and Imperial College London, produced a mathematical model of their own of the processes by which T cells are produced and destroyed. Similarly, they concluded that if Ho's model were correct, then the body's CD4 (+) T cell numbers would decline to very low levels in months, not years. That is, the positive feedback model, supported by Ho and used to justify early anti-retroviral treatment, predicts the development of AIDS within a few months after initial infection by HIV. However, even in Africa's poor, this process usually takes about five years. Professor Jaroslav Stark⁴, one of Yates' coworkers, is quoted as saying "scientists have never had a full understanding of the processes by which T helper cells are depleted in HIV, and therefore they've been unable to fully explain why HIV destroys the body's supply of these [T helper] cells at such a slow rate." Stark has also pointed out that "our new interdisciplinary research has thrown serious doubt on one popular theory [Ho's] of how HIV affects these cells, and means that further studies are required to understand the mechanism behind HIV's distinctive slow process of cellular destruction". This is a polite way of confirming Craddock's² position that the Ho¹ model, upon which current HIV/AIDS drug treatment rests, is "mathematical junk". In summary, Ho's positive feedback hypothesis predicts a collapse of the immune system in untreated HIV-positive patients in months rather than in the years that it actually takes to

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occur. What is needed, then, is a model that accommodates both the slow decline that really takes place and also the differences seen in the time taken to progress to AIDS in the developed and developing worlds. A hypothesis capable of achieving these two goals was first proposed by the current author,⁵ in 2002, in *What Really Causes AIDS*. This book points out that since HIV-1 encodes for an analogue of the human selenoenzyme glutathione peroxidase,⁶ as it is replicated, its genetic needs cause it to deprive seropositive individuals not only of this enzyme but of its four core components: selenium, cysteine, glutamine and tryptophan.⁷ Eventually, after a period of time, the length of which depends on the diet being eaten, this depletion process causes severe deficiencies of these nutrients, the symptoms of which we call AIDS. One consequence of the extreme selenium deficiency caused by this process,⁸ is a decline in CD4 (+) T cells, since the body cannot produce these without adequate quantities of this trace element.⁹

The widely held belief that HIV destroys most of the body's CD4 (+) T lymphocytes directly is incorrect. Most AIDS patient's CD4 (+) T cells are not killed off by the virus directly but are destroyed by so-called "bystander mechanisms". To quote Benigno Rodriguez¹⁰ "In an individual with advanced disease, if you look at the numbers of cells that are actually infected [with HIV], we are talking less than 1 percent. But in reality, the individual may have lost 20, 30 50 percent of his immune cells." This, of course, is because HIV replication has created a selenium deficiency that prevents the normal production of CD4 (+) T cells by such seropositive individuals. Virally created nutritional deficiencies are the "bystander mechanisms" that eventually so seriously damage the immune systems of patients who are HIV-positive.

This orthomolecular model, therefore, explains why the decline in CD4

(+) T cells is so relatively slow and why it takes longer to occur in the developed world, where diets generally contain more of these four key nutrients, than in the developing world.

Complexity

Another major weakness of the Ho model and of the very recently proposed Wodarz and Levy model^{10,11} is that they do not begin to explain the complexity of the symptoms involved in AIDS. While they seek to account, apparently incorrectly, for the decline in CD4 (+) T cells and associated collapse of the immune system, they make no effort to suggest causes for the other major symptoms. This generalization is also true of eight earlier models, described in *What Really Causes AIDS*.⁵ They too focused on the collapse of the immune system and ignored other key defining symptoms of AIDS, such as muscle wasting, depression, psychosis, dementia and diarrhea. Nor do such models explain why AIDS patients are particularly prone to Kaposi's sarcoma or to myocardial infarction. In contrast, all of these symptoms are easily explained by Foster's nutritional model.⁵ Muscle wasting and diarrhea, for example, are characteristics of glutamine deficiency,¹² while depression¹³ and myocardial infarction¹⁴ commonly accompany inadequate selenium. This is also true of Kaposi's sarcoma, which is caused by the human herpes virus 8 (HHV-8), a pathogen that only infects individuals who are very selenium depleted.⁵ Psychosis and dementia, in contrast, are characteristics of extreme tryptophan deficiency.¹⁵ AIDS, therefore, is clearly a complex nutritional deficiency illness cause by a virus.

Why Now?

Another major weakness of all the models of the HIV/AIDS process, put forward to date by virologists, biochemists and mathematicians is that they fail to explain why the first AIDS pandemic

is occurring now. The genetic evidence¹⁶ shows that HIV-1 originated as the chimpanzee (*Pan troglodytes*) virus SIV_{cpz} while SIV_{sm}, a sooty mangabey monkey virus, gave rise to HIV-2¹⁶. Such viruses probably jumped to humans as a consequence of the “bushmeat” trade. However, subsistence hunting and the “bushmeat trade” have provided countless opportunities for contact with simian body fluids and the associated cross-species exchange of viruses for hundreds of thousands of years. Why then is this HIV/AIDS pandemic the first? The answer to this question is provided by Foster’s orthomolecular model⁵ because it explains the link between viral diffusion and falling soil and dietary selenium levels. During the past century, acid rain, fertilizer use and animal husbandry¹⁷ have combined to virtually deplete many global soils of this essential trace element, setting the scene for several viral pandemics, including those of HIV/AIDS and Hepatitis B and C.¹⁸

Treatment Predictions

In his discussion with BBC News, Stark⁴ pointed out that “If the specific process by which HIV depletes this kind of white blood cell [CD4 (+) T cell] can be identified, it could pave the way for potential new approaches to treatment”. He was, of course, correct, but the orthomolecular model has already done more than that. Not only does it suggest new treatment protocols for HIV/AIDS,^{5,7} it also explains how the diffusion of HIV can be greatly reduced.^{19,20} To illustrate, it follows from the Foster model that, if the major symptoms of AIDS are caused by virus-created nutritional deficiencies, then supplementation with selenium, cysteine, glutamine and tryptophan ought to reverse them. The author^{21,22} has assisted in various open and one closed clinical trials in Africa to test this hypothesis. In Zambia, for example, the appropriate nutritional supplements were given to 15

HIV/AIDS patients in the care of a child care and adoption society. These children and guardians experienced dramatic improvement from this selenium-amino acid enriched nutrient mixture, most of them by the second or third week of receiving it. Their complexions, hair texture, energy levels and mobility improved. Some that had been bedridden began to walk. In Uganda, at the Mengo Hospital in Kampala, a 40 HIV/AIDS patient open trial also was set up. After one month, 77% of these patients reported noticeable health improvement. These results were better than they seemed at first glance since seven patients also had tuberculosis and four had syphilis. One patient who had been bedridden for four years was able to walk from his home to the hospital to ask for more nutrients when his month’s supply was exhausted.²¹ Furthermore, a nutritional mixture, called Nutramiracle®, thought to be ideal for the treatment of AIDS, was designed by this author and given to nine extremely ill patients in South Africa by a former nurse, Marnie Bradfield.²² None of the AIDS cases were receiving anti-retroviral drugs. On the basis of these trials, it was concluded that:

Firstly, it is possible to reverse all the symptoms of AIDS in dying patients using nutrition alone. Secondly, this requires selenium and the amino acids, cysteine, tryptophan and glutamine. Thirdly, while selenium alone can slow HIV replication, eventually HIV/AIDS patients also need amino acid supplements. These can be given temporarily until deficiencies are corrected. The patients can then return to selenium supplementation alone for several months, until the more complex nutritional mixture is again required for another month. There appear to be no adverse side effects from these nutritional treatments and patients are delighted with their greatly improved health status.

A much larger, year long 310 HIV-posi-

tive patient, double blinded clinical trial has recently been concluded at the Mengo Hospital in Kampala, Uganda. The nutritional hypothesis supporting, statistically significant results are described in detail elsewhere in this issue.

Not only does Foster's nutritional model for the origins of AIDS provide a simple and inexpensive treatment protocol for this disorder, it also suggests a very effective way of reducing the rate of diffusion of HIV^{19,20} and so halting, or at least slowing, the pandemic. On a global scale, it is obvious that HIV has spread much more rapidly in countries, regions and counties where soils, and in consequence diets, are selenium deficient.⁵ Zaire, Uganda, Tanzania, Kenya and South Africa, for example, where AIDS is now the chief cause of mortality, are all known to be selenium deficient.²³⁻²⁵ In contrast, despite widespread, unprotected promiscuity in Senegal, in 1998 only 0.5 percent of women attending Dakar's antenatal clinics tested positive for HIV. Outside the major urban centres, HIV-1 prevalence among such attendees ranged from 0 to 0.8 percent, in the years 1986 to 1998.²⁶ In contrast to most of the rest of selenium deficient subSaharan Africa, Senegal is essentially a desiccated Cretaceous and Early Eocene sea. As a result its rocks, soils and diets are enriched with this trace element.

Interestingly, all Nordic countries and Finland's eastern neighbours have both selenium deficient soils and relatively high HIV infection rates.²⁷ Finland, however, to reduce heart disease, mandated the addition of selenium to all its fertilizers in 1984, probably accidentally lowering that country's future HIV-prevalence rate by at least 50 percent and possibly much more.

Conclusions

The International AIDS Society 4th Conference was held in Sydney, Australia July 22 to July 25, 2007. As reported by the BBC News Service,²⁸ President George

W. Bush's senior advisor on HIV, Dr. Anthony Fauci admitted in an address to the attendees that the world is losing its fight against AIDS. The best evidence for this is that far more people are becoming infected with HIV than are being treated for its consequences. As Fauci pointed out, "For every person that you put in therapy [on anti-retroviral drugs], six new people get infected. So we're losing that game, the numbers game". Clearly, anti-retroviral drugs, condoms and demands for chastity will not halt the pandemic. We are still awaiting the often promised but never delivered vaccine against HIV. This author's orthomolecular model, that identifies how HIV causes AIDS, identifies prevention strategies that can be more effective than any currently available. These include the addition of selenium to fertilizers, the promotion of foods (including green algae), that are high in amino acids and the provision of specific nutritional supplements capable of increasing the body's production of glutathione peroxidase and so stimulating the activity of the immune system.²¹⁻²² Such orthomolecular approaches to slowing, or even reversing, the HIV/AIDS pandemic appear to be our only realistic remaining strategies. Others have already failed. Indeed, the addition of selenium to fertilizers and directly to table salt and foods have been successfully applied in China to greatly slow diffusion of the Hepatitis B and C viruses and the Coxsackie B virus.^{29,30} Interestingly, all these viruses that cannot diffuse in populations eating selenium enriched diets also, like HIV-1 and HIV-2, encode for the enzyme glutathione peroxidase.³¹ The reverse, however, is true. M.A. Beck³² has shown that the greater the selenium deficiency in infected mice, the more easily Coxsackie virus B₃ can mutate. It seems likely, therefore, that as HIV increases selenium deficiencies in a seropositive patient³³ an increasing number of viral mutations will occur, which in turn will

be more difficult for the patient's immune system to control. Beyond this, some of these mutations will be more virulent, threatening greater infection of local populations.

The US President's Fiscal Year 2007 federal budget request includes an estimated \$22.8 billion for domestic and global HIV/AIDS activities that will continue to be ineffective.³⁴ Yet this funding is more than enough to supply every HIV/AIDS patient on the planet with nutrients that would mitigate their symptoms. It could also pay for the global addition of selenium to fertilizers where necessary, so slowing new infection, not only by HIV-1 and HIV-2 but also by the Hepatitis B and C viruses, Coxsackie virus B, that together seriously threaten humanity.¹⁸

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