



# What really causes AIDS

HAROLD D. FOSTER



a series  
outside  
the box

# What Really Causes AIDS

HAROLD D. FOSTER

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*Dedicated to Foinavon 444/1*

## AUTHOR'S NOTE

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*Whoever wishes to investigate medicine properly, should proceed thus: in the first place to consider the seasons of the year, and what effects each of them produces ... Then the winds, the hot and the cold, especially such as are common to all countries, and then such as are peculiar to each locality. We must also consider the qualities. In the same manner, when one comes into a city to which he is a stranger, he ought to consider its situation, how it lies as to the winds and the rising of the sun: for its influence is not the same whether it lies to the north or the south, to the rising or to the setting sun. These things one ought to consider most attentively, and concerning the water which the inhabitants use, whether they be marshy and soft, or hard, and running from elevated and rocky situations, and then if saltish and unfit for cooking, and the ground, whether it be naked and deficient in water, or wooded and well watered, and whether it lies in a hollow, confined situation, or is elevated and cold: and the mode in which the inhabitants live, and what are their pursuits, whether they are fond of drinking and eating to excess, and given to indolence, or are fond of exercise and labour, and not given to excess in eating and drinking.*

Francis Adams, *The Genuine Works of Hippocrates*, 1849  
(on Airs, Waters, and Places), vol. 1, p. 190

# WHAT REALLY CAUSES AIDS: AN EXECUTIVE SUMMARY

The AIDS pandemic is likely to become the greatest catastrophe in human history. Unless a safe, effective vaccine is quickly developed, or the preventive strategies outlined in this book are widely applied, by 2015 one sixth of the world's population will be infected by HIV-1 and some 250 million people will have died from AIDS. Its associated losses by then will be more than those of the Black Death and World War II combined, the equivalent of eight World War Is.<sup>1</sup>

This pandemic is only one of several ongoing catastrophes involving viruses that encode the selenoenzyme glutathione peroxidase.<sup>2</sup> Indeed, the world is experiencing simultaneous pandemics caused by Hepatitis B and C viruses, Coxsackie B virus and HIV-1 and HIV-2. As these viruses replicate, because their genetic codes include a gene that is virtually identical to that of the human enzyme glutathione peroxidase, they rob their hosts of selenium. Paradoxically, however, they diffuse most easily in populations that are very selenium deficient,<sup>3</sup> possibly because their members have depressed immune systems. It is no coincidence that such viruses are causing havoc at the beginning of the 21<sup>st</sup> century. The last 50 years have seen enormous expansions in the use of fossil fuels and deforestation by fire. The resulting pollutants have greatly increased the acidity of global precipitation, reducing selenium's ability to enter the food chain. This situation is being made worse by the widespread use of commercial fertilizers since their sulphates, nitrogen, and phosphorus all depress the uptake of selenium by crops. Deficiencies in this essential trace element are being felt most acutely in areas, such as sub-Saharan Africa, where soil selenium levels are naturally very low. Acid rain is making a bad situation worse, so increasing vulnerability to those viruses that encode glutathione peroxidase. Many populations are also being exposed to a thinning ozone layer, heavy metals such as mercury and cadmium, pesticides, and drug,

tobacco, and alcohol abuse, all of which depress the human immune system, increasing vulnerability to viruses, including HIV-1 and HIV-2.

In July 2000, physicians and scientists from around the world met in Durban, South Africa for the XIII International AIDS Conference. In a declaration, named after the city, 5,018 of them proclaimed that "HIV is the sole cause of AIDS."<sup>4</sup> There are, however, at least seven anomalies that strongly suggest that this conventional wisdom is incorrect and that belief in it is blocking progress in the development of new treatments for AIDS and of novel ways of preventing its spread. To illustrate, despite widespread unprotected promiscuous sexual activity in Senegal, HIV-1 is diffusing very slowly, if at all, amongst the Senegalese.<sup>5</sup> It is very apparent that in Africa, differences in soil selenium levels are greatly influencing who becomes infected with HIV-1 and who does not. Indeed, the recently published *Selenium World Atlas* used the incidence of HIV-1 as a surrogate measure of soil selenium levels because actual levels are, as yet, poorly established in sub-Saharan Africa. A similar relationship has been documented in the United States<sup>6</sup> where there has been an inverse relationship, especially in the Black population, between mortality from AIDS and local soil selenium levels.

It is well established that individuals who are HIV-positive gradually become more and more selenium deficient.<sup>7</sup> This decline, which is known to undermine immune functions, is not unique to HIV-infection but is seen in almost all infectious pathogens.<sup>8</sup> However, under normal circumstances, where death does not occur, selenium levels rebound soon after recovery. HIV-1, however, can effectively elude the defense mechanisms of the immune system, and can continue to replicate indefinitely, endlessly depressing serum selenium. As a result, the immune system is compromised, allowing infection by other pathogens that continue to deplete the host of selenium, allowing HIV-1 to replicate more easily, further undermining immunity. Therefore, this relationship between selenium and the immune system is one of positive feedback, in which a decline in either of these two variables

causes further depression in the other. Termed the “selenium-CD4 T cell tailspin” by the author,<sup>9</sup> it is the reason that serum selenium levels are a better predictor of AIDS mortality than CD4 T cell counts. Like other positive feedback systems, such as avalanches and forest fires, it is extremely difficult to control and gains momentum as it progresses.

HIV-1, however, encodes the entire selenoenzyme, glutathione peroxidase. As it replicates, therefore, it depletes its host not only of selenium but also of the other three components of this enzyme: namely, cysteine, glutamine, and tryptophan.<sup>10</sup> AIDS, therefore, is a nutritional deficiency illness caused by a virus. Its victims suffer from extreme deficiencies of all four of these nutrients which are responsible for such symptoms as depressed CD4T lymphocyte count, vulnerability to cancers (including Kaposi’s sarcoma), depression, psoriasis, diarrhea, muscle wasting, and dementia. Associated infections cause their own unique symptoms and increased risk of death.

HIV-1 alone, therefore, does not cause AIDS. It involves a multiplicity of co-factors, specifically anything that either depletes serum selenium levels or depresses the immune system enough to permit viral replication. Manipulating the “selenium-CD4T cell tailspin” by adding this trace element to fertilizers and food stuffs opens new avenues for both prevention and treatment. This strategy has been shown to work on other viruses that encode glutathione peroxidase, such as Hepatitis B and C and the Coxsackievirus. The logical treatment of AIDS patients involves supplementation with selenium, cysteine, glutamine, and tryptophan, at least to levels at which deficiency symptoms associated with a lack of these nutrients disappear. While this can be most easily achieved by supplements, certain foods contain elevated levels of those four nutrients. Strangely enough, one of the ideal meals for anyone who is HIV-seropositive would include a cheeseburger to which Brazilnut flour had been added to the bun.



Brazil nuts contain the highest levels of selenium found in any human food.

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*There are causes for all human suffering, and there is a way by which they may be ended, because everything in the world is the result of a vast concurrence of causes and conditions and everything disappears as these causes and conditions change and pass away.*

[The teachings of Buddha by  
Bukkyo Dendo Kyokai,  
112th revised edition]

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*The evil that is in the world always comes of ignorance, and good intentions may do as much harm as malevolence, if they lack understanding. On the whole, men are more good than bad; that, however, isn't the real point, but they are more or less ignorant, and it is this that we call vice or virtue; the most incorrigible vice being that of ignorance that fancies it knows everything and therefore claims for itself the right to kill. The soul of the murderer is blind; and there can be no true goodness nor true love without the utmost clear-sightedness.*

Albert Camus, *The Plague*

# AIDS: THE CONVENTIONAL WISDOM

# 1

*Truth is not determined by majority vote.*

Doug Gwyn

In July 2000, physicians and scientists from around the world met in Durban, South Africa for the XIII International AIDS Conference. In a declaration, named after the city, 5,018 of them proclaimed that “HIV [human immunodeficiency virus] is the sole cause of AIDS.” This highly unusual document, published in *Nature*,<sup>1</sup> was more political than scientific, targeting a small group of maverick researchers, most of whom supported the views of Dr. Peter Duesberg,<sup>2</sup> a microbiologist from the University of California at Berkeley. Duesberg has argued consistently that HIV is merely a harmless passenger virus and that AIDS is the result of destruction of the immune system by long-term cumulative use of intravenous, recreational, and pharmaceutical drugs, including AZT, cocaine, amphetamines, and nitrite inhalants. He believes that noninfectious immune-suppressant factors in blood transfusions can lead to AIDS, as can factor 8 taken by hemophiliacs.

Duesberg<sup>3</sup> further points out that there is little evidence that HIV is particularly active in the cells of those dying of AIDS and that it does not infect enough lymphocytes to seriously depress the immune system. He also believes that the current method of testing for HIV is in error. Traditionally, the presence of antibodies to any infectious agent indicated that the threat of serious disease had passed. The immune system has recognized and can attack and control the invader. In the case

of HIV, antibodies are thought by conventional medicine to show the worst is yet to come, that AIDS is eventually inevitable.

The epidemiology and geography<sup>4</sup> of the AIDS pandemic both clearly illustrate that Duesberg and his supporters are wrong. One of the first individuals in North America known to have developed AIDS was Gaetan Dugas,<sup>5</sup> sometimes referred to as Patient Zero, an airline steward. Gay, highly active sexually, careless of the welfare of others and, above all, extremely mobile because of his employment, Dugas frequented bathhouses throughout Canada and the United States. When interviewed, in July 1981 by Dr. Mary Guinan, a researcher at the Centers for Disease Control's Venereal Disease Division, Dugas admitted to 250 sexual contacts a year, a total of some 2,500 gay sexual partners.<sup>6</sup> By April 12, 1982, 248 US gay men had been diagnosed with AIDS (known at that time as GRID, Gay-Related Immune Deficiency). At least 40 of these had sex either with Gaetan Dugas, or with someone who had. Dugas could be linked to 9 of the first 19 cases of AIDS in Los Angeles, 22 cases in New York City and nine patients in eight other North American cities. To quote Shilts,<sup>7</sup> "A CDC statistician calculated the odds on whether it could be coincidental that 40 of the first 248 gay men to get GRID [later renamed AIDS] might all have had sex either with the same man or with men sexually linked to him. The statistician figured that the chance did not approach zero—it was zero."

Further evidence that AIDS is caused by a pathogen(s) was provided by its diffusion in West Germany.<sup>8</sup> All 44 of the initial cases of AIDS reported there, on or before March 31, 1983, had occurred in people who had either travelled to Haiti or Africa or were amongst gay men who had vacationed recently in California, Florida, or New York. This is hardly surprising since each one of these locations was by then an AIDS hot spot, where all sexual encounters were high risk.

A social science experiment to prove, once and for all, whether HIV is essential for the development of AIDS would involve testing the inhabitants of one island regularly for HIV infection. Those found to be positive would be immediately, permanently quarantined. After several years the prevalence of AIDS would be compared with that on neighbouring islands, where no such HIV detection scheme had been in force. If the AIDS prevalence on the island undergoing such testing and quarantine was significantly lower than that in neighbouring islands, the evidence of a key role for HIV in AIDS would be overwhelming and undeniable.

In 1983, Cuba began repeatedly to test its population for HIV.<sup>9</sup> Since 1986, all those found positive have been quarantined in sanatoriums. While Cuba's response to the AIDS pandemic may be viewed by some as an assault on personal freedom, it has been exceptionally successful in preventing the spread of AIDS into its 10 million population.<sup>10</sup> By 2000, the cumulative number of patients in Cuba<sup>11</sup> to have developed AIDS was only 889, roughly 8.9 per 100,000 over the time span of the pandemic. In contrast, in neighbouring Jamaica,<sup>12</sup> with a population of only some 2.56 million, there have been 2,963 cases of AIDS. This is roughly 115 per 100,000 over the same time period. Clearly, AIDS has been about 12 times more common in Jamaica than in Cuba. Indeed, Cuba has one of the lowest prevalence rates of HIV infection in the world<sup>13</sup> and its experience appears to prove beyond any reasonable doubt that HIV is involved in the development of AIDS.

The geography of AIDS continues to support the position that at least one of its causes is an infectious pathogen. In New Jersey, for example, the majority of the early AIDS cases did not occur in gay men but in intravenous drug users, many of whom shared contaminated needles. In the early 1980s, when

AIDS began to appear in New York State,<sup>14</sup> it spread rapidly, in ever expanding concentric circles, focused on the centre of Manhattan. Since this date, AIDS has tended to occur first in the major cities of the Developed World, in gays, intravenous drug users, and blood and blood product recipients, gradually diffusing into evermore remote rural areas. Such a spatial distribution pattern is much more typical of an infectious agent than a toxin.<sup>15</sup>

Nevertheless, Duesberg has supporters in high places, including the South African government.<sup>16</sup> This became apparent in April 2000, 3 months or so before the XIII International AIDS Conference was to be held in Durban when South African President Thabo Mbeki appointed Duesberg to a government task force on AIDS, designed to disprove its links to HIV-1. Mbeki also sent a letter to US President Clinton and other world leaders defending the right of maverick AIDS theorists to be heard. Indeed, South Africa's Deputy President, Jacob Zuma<sup>17-18</sup> declared that all sides of the debate had the right to free speech and drew parallels between arguments about HIV's role in AIDS and the 17<sup>th</sup> century controversy surrounding Galileo's belief that the Earth orbited around the Sun. In a statement released by the Office of the South African Presidency, Zuma is quoted as saying "As we all know today, he was right and they were wrong." "Suppose we discover, as Galileo did, that the so-called mainstream scientific view is incorrect," said Zuma. "Suppose there was even a one percent chance that the solution lay elsewhere. As a country we cannot afford to overlook this possibility."

On May 7<sup>th</sup>, 2000, Duesberg and nine associated AIDS dissidents issued a *Minority Statement and Recommendations to the Government of South Africa*.<sup>19</sup> In it they claimed HIV did not cause AIDS and that AIDS was neither contagious nor sexually transmitted. They also stated that anti-HIV drugs proved fatal

to many patients and caused side effects that could not be distinguished from AIDS itself. Five recommendations were made to the South African government as a result of these beliefs. The first of these was that South Africa and indeed all African countries should devote the bulk of their national and international biomedical and other resources to the eradication and treatment of the predominant AIDS-defining diseases (such as tuberculosis, malaria, and enteric infections); and to the improvement of nutrition and the provision of clean water and better sanitation. They also suggested the complete rejection of anti-HIV drugs; the promotion of sex education to prevent the spread of sexually transmitted diseases and unwanted pregnancies; stopping dissemination of the false message that HIV infection was invariably fatal and the suspension of HIV testing. As a consequence of Duesberg's influence,<sup>20</sup> the South African government refused to make AZT available in public clinics and discontinued the drug's use by its military. The Durban Declaration,<sup>21</sup> which promotes the conventional wisdom that HIV alone causes AIDS, was the medical establishment's reaction to this public relations coup by the anti-HIV mavericks.

Interestingly, the *Minority Statement and Recommendations to the Government of South Africa* did not just upset the medical establishment. It also drew fire from another group of AIDS dissidents<sup>22</sup> headed by John Scythes and Colman Jones, long-term advocates of undetected syphilis as the major causal variable in AIDS. Scythes and Jones quickly issued a statement of their own which provided a point-by-point response to Duesberg and his colleagues' document. They argued that historically syphilis often dispatched its victims by opportunistic infections rather than through the classical direct effects of late syphilis. Scythes and Jones provided evidence which they felt documented a key role for undiagnosed syphilis in AIDS. This idea was not new. Dr. Stephen Caiazza, a Manhattan internist,

treated AIDS patients in the 1980s with 20 million units of IV penicillin by constant infusion in the belief that AIDS was due to infection by the undetected bacterium *Treponema pallidum* (the cause of syphilis) acting in concert with HIV and other pathogens.<sup>23</sup>

Despite his reputation as a radical, Duesberg is not the most extreme of the AIDS dissidents. In 1994, Dr. Robert E. Willner stunned the Spanish public by inoculating himself with the blood of Pedro Tocino, an HIV-positive hemophiliac.<sup>24</sup> This demonstration of contempt for the conventional wisdom seemed designed to promote his book *Deadly Deception: The Proof That Sex and HIV Absolutely Do Not Cause AIDS*. Willner<sup>25</sup> argued that “most of the medically supervised AIDS deaths were either caused or contributed to by the deadly drug AZT . . . that was shelved more than 20 years ago because it was found to be too toxic to give to terminally ill cancer patients.”

Naturally, such anti-establishment views are poorly tolerated. Those who believe them are widely thought to be obstacles to halting the AIDS pandemic. Or, as the Durban Declaration<sup>26</sup> puts it, “HIV causes AIDS. It is unfortunate that a few vocal people continue to deny the evidence. This position will cost countless lives.” This pre-conference document was just one of a series of attacks against Duesberg and his supporters by the medical establishment.

They are not the only AIDS mavericks to feel its wrath. It has also been directed against Nicolas Regush,<sup>27</sup> author of *The Virus Within*. In this book, Regush describes research conducted by Drs. Donald Carrigan and Konnie Knox that suggests that HHV-6 (Human Herpes Virus-6) may be much more damaging in AIDS than HIV. Reacting to this publication, Dr. Mark Weinberg,<sup>28</sup> a professor of medicine at McGill University and President of the International AIDS Society compared those

researchers who refused to toe the official line that HIV alone causes AIDS to Holocaust deniers. Furthermore, he suggested that their erroneous views might interfere with efforts to reduce the spread of HIV, such as blood screening, condom use, abstinence, and the public acceptance of valuable drugs such as AZT.

In the Durban Declaration the claim that “HIV is the sole cause of AIDS” is backed by the following eight lines of evidence.<sup>29</sup> Firstly, AIDS patients, regardless of where they live, are infected with HIV. If they are not treated, most people testing HIV positive will begin to show signs of AIDS within 5 to 10 years. Indeed, in Africa, HIV-infected individuals are 11 times more likely to die within 5 years than those in North America. Thirdly, such HIV infection can be identified not only by detecting antibodies but also by gene sequences and viral isolation tests thought to be as reliable as any used for detecting other viral infections. Furthermore, people who receive HIV-contaminated blood or blood products develop AIDS while those given untainted or screened blood do not. Similarly, most children who develop AIDS are born to mothers who are HIV-infected. The higher the mother’s viral load, the greater the risk to the infant. Furthermore, in the laboratory, HIV infects CD4 T lymphocytes, the same type of white blood cells depleted in AIDS victims. In addition, drug combinations that block the replication of HIV in the test tube can reduce viral load and slow progression to AIDS. In the short term, at least, where this treatment has been available it has reduced AIDS mortality. Finally, monkeys inoculated with cloned SIV (simian immunodeficiency virus) DNA become infected and subsequently develop AIDS-like symptoms. While these eight lines of evidence appear, to this author at least, to prove a role for HIV in AIDS, they do *not* establish that there are no other essential co-factors involved in immune system collapse.

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*No lesson seems to be so deeply inculcated by the experience of life as that you never should trust experts.*

Lord Salisbury  
Letter to Lord Lytton, 1877

## TERMITES IN THE FOUNDATIONS

*The evolution is this: a premature explanation passes into tentative theory, then into an adopted theory, and then into ruling theory.*

*When the last stage has been reached, unless the theory happens, perchance, to be the true one, all hope for the best results is gone. To be sure, truth may be brought forth by an investigator dominated by a false ruling idea. His very errors may indeed stimulate investigation on the part of others. But the condition is an unfortunate one. Dust and chaff are mingled with the grain in what should be a winnowing process.*

T.C. Chamberlin<sup>1</sup>

*The Method of Multiple Working Hypotheses*

Scientific theories resemble architectural wonders. They are interesting to visit and prestigious to be associated with. All too often, however, while they appear sound to casual observation, termites are feasting deep within their foundations. Anomalies, facts that the ruling theory and its supporters cannot explain, are the termites of science. As they multiply, the infected theory weakens until eventually it collapses. While the “HIV alone causes AIDS” theory still dominates the scientific skyline, termites are hard at work within it. Here are seven anomalies that suggest it is incorrect and will eventually fall.

### **ANOMALY ONE: DEATH BY BREAST FEEDING**

Breast feeding has long been a very controversial issue in the AIDS debate. Epidemiologists estimate that worldwide there

are some ten million children who are infected with HIV. The overwhelming majority of these have been exposed to the virus through their mother's breast milk. The rate of HIV transmission by breast-feeding seems to vary from about 14 to 26 percent, depending on the timing of maternal infection and viral load. As a result of this high risk of HIV transmission to the infant, many researchers have suggested that formula feed should be used instead. Unfortunately, in Kenya<sup>2</sup> where this issue was studied by randomly assigning 425 HIV-infected women and their infants to either breast feeding or formula, both child groups had similar high death rates after 2 years. This seemed to be because although the rate of HIV transmission was cut by substituting formula for breast milk, polluted water used to mix it often seems to increase the probability of death from other causes.

Far more relevant to the current discussion was the observation that breast-feeding seemed to cause a higher AIDS death rate amongst nursing mothers.<sup>3</sup> In the 2 years of observation, mortality among mothers was three times as high in the breast-feeding group as it was in the formula group (18 compared to 6 deaths, log rank test  $p=0.009$ ). The cumulative probability of maternal death at 24 months after delivery in HIV-positive women was 10.5 percent in the breast-feeding group compared to 3.8 percent in those using formula ( $p=0.02$ ). The Kenyan study demonstrated, therefore, that the relative risk of death for breast-feeding mothers versus formula-feeding mothers was 3.2 (95 percent CI 1.3-8.1,  $p=0.01$ ). Indeed, the attributable risk of maternal death due to breast feeding was 69 percent. Even stranger, there was an association between later death of the infant, even after its HIV-1 infection status had been controlled for (relative risk 7.9, 95 percent CI 3.3-18.6,  $p<001$ ). Cutting through the jargon, this study showed that mothers in Kenya who were HIV-positive were about three times as likely to die within 2 years of diagnosis if they breast fed their babies.

Beyond this, infants were about eight times more likely to die if their mothers failed to survive the first 2 years after their delivery. Simply put, breast feeding by an HIV-infected woman shortens her life expectancy, speeding her death from AIDS. It may also increase the probability of the death of her infant. If AIDS is caused by HIV alone, why is this the case? Why does breast feeding accelerate HIV-1's negative impact on the immune system?

## **ANOMALY TWO: SEX IN SENEGAL**

Senegal's government has been registering prostitutes since 1966, long before AIDS was first identified, in an attempt to combat rampant syphilis, gonorrhea, and other sexually transmitted diseases. Every day, women crowd the clinic in downtown Dakar to register, yet Michael Tardy<sup>4</sup> who runs it admits that only about a quarter of the city's professional sex trade workers register. There is little doubt that Dakar, Senegal's capital, is one of the largest commercial sex centres of Africa, with prostitutes visiting it from all over Western Africa and the Middle East.

At the same time, polygamy is common and Senegalese men can legally marry up to four wives. When married men are searching for second or third wives they almost invariably "try out" lots of other women before making a final selection.<sup>5</sup> Indeed, during a survey carried out in 1997, 33 percent of Senegalese males aged 15 to 49 admitted to recently having sex with non-regular partners and a third of those who did so were not using a condom.<sup>6</sup>

Given this high level of unprotected promiscuity, it might be expected that AIDS would be very common in Senegal. After all, this country is located in sub-Saharan Africa, close to the

region where SIV<sub>cpz</sub> (simian immunodeficiency virus) is believed to have been transmitted from chimpanzees to humans<sup>7</sup> on several occasions and where it subsequently evolved into HIV-1. However, in Dakar,<sup>8</sup> Senegal's major urban centre, HIV-1 prevalence among women attending antenatal clinics has remained at one percent or less since the time surveillance began in the mid-1980s until 1998. The latter date is for the most current data provided in the *UNAIDS Epidemiological Fact Sheets* at the time of writing. Indeed, in 1998, only 0.5 percent of antenatal clinic women in Dakar tested positive for HIV. Outside Senegal's major urban areas, HIV-1 prevalence amongst antenatal clinic attendees also has remained low, ranging from zero to 0.8 percent in the years 1986 to 1998. In total, 7,800 adults and children are thought to have died of AIDS in Senegal in 1999. This is less than one per thousand of the population of 9,240,000.

It is of interest to compare these figures with those of other countries in sub-Saharan Africa. Information on HIV prevalence among antenatal clinic attendees, for example, also has been available from Uganda since the mid-1980s.<sup>9</sup> In Kampala, the prevalence rate amongst such pregnant women increased from 11 percent in 1985 to 31 percent in 1990. Since 1993, HIV prevalence has been declining, dropping to 14 percent by 1998. Outside Uganda's major urban centres the median HIV prevalence in clients of antenatal clinics reached a high of 13 percent in 1992, falling to 8 percent in 1998. Some 110,000 individuals were thought to have died of AIDS in Uganda during 1999, out of a population of 21,143,000.

Rather than burden the reader with endless statistics, it will simply be pointed out that in South Africa,<sup>10</sup> HIV prevalence amongst pregnant women in urban areas, such as Johannesburg, Pretoria, Durban, and Port Elizabeth, increased from one percent in 1990 to a median 19 percent in 1998. In the

more rural areas it rose from one percent in 1990 to 21 percent in 1998. Deaths from AIDS in South Africa reached 250,000 during 1999, out of a total population of 39,900,000.

There is a major anomaly illustrated by these African figures. It is clear that HIV-1 is much easier to transmit, and AIDS much more likely to develop, in some countries than in others. The Senegalese are a highly sexually active society.<sup>11</sup> Many of its members are promiscuous and commonly practise unprotected sex with a variety of partners.<sup>12</sup> Nevertheless, unlike South Africa, Uganda, and many other countries in Southern Africa, HIV-1 is spreading very slowly, if at all, in its population. The most logical explanation for this geographical anomaly is that HIV-1 needs a co-factor or co-factors to infect and to subsequently cause the immune system collapse associated with AIDS.

### **ANOMALY THREE: SELENIUM DEFICIENCY**

A healthy person usually has between 800 and 1,200 CD4 T lymphocytes (T-helper cells) in each cubic millimetre of their blood. These lymphocytes play an essential role, signalling other cells in the immune system to carry out their specific functions. In the original conventional view of AIDS, it was believed that HIV-1 directly killed CD4 T lymphocytes, so undermining the immune system's ability to function effectively. It is further believed that when the CD4 T lymphocyte count falls below 200 per cubic millimetre, HIV-1 positive individuals become particularly vulnerable to the opportunistic infections such as tuberculosis, pneumocystis carinii pneumonia, and toxoplasmosis and to the cancers that typify and are used to define AIDS.<sup>13</sup>

When HIV-1 infection has been confirmed and AIDS is suspected, the number of CD4 T lymphocytes per cubic millimetre

of a patient's blood is regularly assessed as a guide to how well the immune system is continuing to function. If the conventional model is correct and HIV is the sole cause of AIDS, it would be logical to expect that this CD4 T lymphocyte count would be the most accurate predictor of patient survival.<sup>14</sup> It is not. Plasma selenium levels are a far better indicator of the probability of death from AIDS than are CD4 T lymphocyte counts.

To illustrate, numerous studies have documented the presence of declining plasma selenium levels and an associated decrease in glutathione peroxidase activity in individuals with HIV/AIDS.<sup>15-17</sup> More recently, Baum and co-workers<sup>18</sup> have reported monitoring immunologic and nutritional factors at 6 month intervals, over 3.5 years, in 125 HIV-1-seropositive drug-using men and women in Miami, Florida. This longitudinal study collected data on CD4 T lymphocyte count, antiretroviral treatment, and plasma levels of vitamins A, E, B<sub>6</sub>, B<sub>12</sub>, and selenium and zinc. A total of 21 of the participants died of HIV-related causes in the course of the 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 selenium deficiency being the superior indicator of it. Similarly, 24 HIV-infected children were monitored for 5 years,<sup>19</sup> during which time, 50 percent of them died from HIV-related causes. In pediatric HIV-1 infection, as in adults, low plasma level of selenium was found to be an independent predictor of mortality. Indeed, the lower the serum selenium, the more rapidly death occurred, indicating that it was associated with faster disease progression. In short, if one needs to predict whether or not a patient with AIDS is likely to die, the best indicator of survival probability is their plasma selenium level not their CD4 T lymphocyte count. Why?

## ANOMALY FOUR: KAPOSI'S SARCOMA

It had been a standing joke that whenever Gaetan Dugas (later known as Patient Zero) entered a gay bar he would quickly scan the crowd and announce to his friends, "I'm the prettiest one."<sup>20</sup> Dugas was both handsome and vain and it was not surprising that, in the early stages of his AIDS, what worried him the most was a small purplish spot on his face.<sup>21</sup> This he soon discovered was Kaposi's sarcoma, an AIDS-defining cancer that can develop on the skin or in the mouth, lymph nodes, or in organs that include the liver, lungs, and bowel.

Kaposi's sarcoma is named after the physician who first described it, Moritz Kaposi, an Austro-Hungarian dermatologist who reported it in 1872. Before the AIDS pandemic began, Kaposi's sarcoma was a rare tumour in the Developed World.<sup>22</sup> In North America and Europe, it was occasionally seen in elderly men of Italian or Eastern European Jewish ancestry, pre-pubescent children and patients receiving immunosuppressive therapies.<sup>23</sup> However, in the 1950s, Kaposi's sarcoma was found to be relatively common in the native population of equatorial Africa.<sup>24</sup> In Uganda it accounted for 9 percent of all cancers. Unlike Europeans and North Americans, Africans developing this cancer tended to be relatively young.

Kaposi's sarcoma has become the commonest form of neoplasm in AIDS patients and has long been recognized as a marker for the disease.<sup>25</sup> In the United States, the first official report of a rapid rise in the incidence of this formerly rare cancer appeared in a newsletter, published by the Centres for Disease Control on July 4, 1981. This particular issue of the *Morbidity and Mortality Weekly Report* carried an article entitled "Kaposi's sarcoma and Pneumocystis Pneumonia Among Homosexual Men—New York and California".<sup>26</sup> This outlined the strange symptoms that had developed in 20 gay patients in New York

City and in six more in California. These patients displayed various combinations of Kaposi's sarcoma, pneumocystis, severe herpes, candidiasis, cryptococcal meningitis, and toxoplasmosis.

The great rise in the incidence of Kaposi's sarcoma which accompanied the AIDS pandemic led to extensive research into this cancer. It has been discovered that Kaposi's sarcoma lesions regularly harbour the DNA of a novel herpes virus, now called either human herpes virus 8 (HHV8) or KS-associated herpes virus (KSHV).<sup>27-28</sup> Infection with HHV8 is tightly linked to Kaposi's sarcoma risk and it precedes tumour development. It is now generally agreed that the presence of HHV8 infection is necessary before this cancer will occur. Nevertheless, some researchers consider that HIV may be a co-factor in the disease. Ganem<sup>29</sup> points out, for example, that "Given the magnitude of the increase in Kaposi's sarcoma risk conferred by HIV infection, especially compared to that conferred by other forms of immunosuppression, an important co-factor role for HIV in this disease still seems likely."

When two diseases commonly occur together in the same patient, it is possible that one may be a co-factor for the other, or indeed the relationship might be symbiotic, with two pathogens acting as co-factors for one another. Clearly, since Kaposi's sarcoma was first described in 1872, it cannot be argued that HIV is an *essential* co-factor for this cancer. The elderly men of Italian or East European Jewish ancestry who traditionally developed Kaposi's sarcoma were not HIV-positive. Neither, of course, was the patient described by Moritz Kaposi in 1872. It is also very unlikely that Ugandan Africans who developed this cancer in the 1950s or earlier were HIV positive. The evidence, therefore, is conclusive that Kaposi's sarcoma can develop in the absence of HIV. Similarly, although Kaposi's sarcoma is the commonest cancer in AIDS patients, many HIV-positive

individuals who progress to AIDS never develop it. Clearly, therefore, HHV8 is not an *essential* co-factor for AIDS.

There is a third possibility. When two diseases commonly occur together in the same individual, it is likely that both are members of a common family tree.<sup>30</sup> That is, both diseases require one or more of the same co-factor(s) before they can develop. It is argued here that this explains the frequency with which HIV and HHV8 occur in the same individual. Both require the same co-factor(s) before they become virulent.

### **ANOMALY FIVE: WHY NOW?**

In West Central Africa, monkeys and chimpanzees are hunted by the “bushmeat” trade. Others are caught for pets or research purposes. Butchers who slaughter such animals for sale in local markets work splattered in their blood.<sup>31</sup> This is not simply an animal rights issue, since evidence of simian immunodeficiency virus (SIV) infection has been collected from 26 different species of African nonhuman primates. Two of these viruses gave rise to HIV-1 and HIV-2 in humans.<sup>32</sup> That is, humans are not the natural hosts for either HIV-1 or HIV-2. These viruses have evolved from simian viruses that have entered the population as a result of zoonotic, cross-species transmission. The genetic evidence shows that HIV-1 originated as the chimpanzee (*Pan troglodytes*) virus SIV<sub>cpz</sub>, while SIV<sub>sm</sub>, a sooty mangabey (*Cercocebus atys*), that is monkey virus, gave rise to HIV-2.<sup>33</sup>

Exactly how these two viruses made the jump from chimpanzees and sooty mangabeys to humans is unclear, but subsistence hunting and the bushmeat trade have provided countless opportunities for contact with simian body fluids and the associated cross-species exchange of viruses. Such exchanges

have occurred not just twice but numerous times, as is shown by variations in the genomes of both HIV-1 and HIV-2. To illustrate, current evidence indicates that HIV-1 has three distinct viral groups (which are termed M, N, and O) and that the dominant M group consists of 11 clades denoted as subtypes A through K. Similarly, HIV-2 strains infecting humans consist of six distinct evolutionary lineages, subtypes A through F. What this genetic evidence shows is that the simian counterparts of HIV-1 and HIV-2 have been introduced into the human population on at least seven occasions, possibly more.<sup>34</sup>

In Africa, mankind has been in close contact with chimpanzees, sooty mangabeys, and other non-human primates for hundreds of thousands of years. It seems extremely likely that during these eons, humans have been exposed to simian body fluids on countless occasions; providing frequent opportunity for the cross-species transmission of viruses. Why then did the *first* AIDS pandemic only begin in the late 20<sup>th</sup> century? It might be argued that it has been driven by the transport revolution which allowed greater mobility for HIV-positive individuals, so spreading the disease. Certainly, transportation innovations have always favoured the diffusion of infectious pathogens. But the 16<sup>th</sup> to the 19<sup>th</sup> centuries saw the rise of the Slave Trade, with the movement of millions of West Africans to Europe, North America, and elsewhere.<sup>35</sup> Had HIV-1 or HIV-2 been endemic in Western Africa at the time, they would certainly have been diffused around the globe by sailing ships, more slowly but just as certainly by both slave and slavers. Slavers were often exposed to dangerous tropical pathogens. Many of the resulting diseases were fatal, as illustrated by the slaver's jingle<sup>36</sup> "Beware the Bight of Benin, of the one that comes out there are forty that go in." Indeed, exotic diseases certainly were spread by the Slave Trade from Africa to the Americas and elsewhere. While these included yellow fever, they did not include AIDS.<sup>37</sup>

Why then, after hundreds of thousands of years of opportunities for simian to human viral transmissions was there no evidence of an AIDS pandemic until the late 20<sup>th</sup> century, even though simian immunodeficiency viruses have been reported for 26 different species of nonhuman African primates? The most logical answer to this question is that cross-species transmission occurred many times but, when it did, evolving human SIV equivalents did not have the co-factor(s) they require to cause AIDS.

### **ANOMALY SIX: PATERNAL NEGLECT**

As Chamberlin<sup>38</sup> pointed out while discussing his method of multiple working hypotheses, the originator of a theory tends to have a maternal or paternal relationship with it. As a consequence, they tend to continue to defend it long after it becomes obvious that it is incorrect. However, this is not true of the “HIV as the sole cause of AIDS theory.” HIV was first identified in 1983, at the Pasteur Institute in Paris by a research team headed by Dr. Luc Montagnier. However, in March 1990, approximately 7 years later, Montagnier published the results of a simple study which clearly showed that HIV could not be the sole cause of AIDS. Montagnier<sup>39</sup> demonstrated that although cultured cells infected with HIV usually died rapidly, if they were given the antibiotic tetracycline, they flourished.

Since antibiotics (such as tetracycline) do not kill viruses, Montagnier had to accept that some other undetected co-factor, probably a bacterium, must be needed before cell death can occur. That is, HIV cannot, in and by itself, kill CD4 T lymphocytes. It needs assistance from one or more co-factors. Montagnier believed that the other pathogen involved was probably some form of mycoplasma.<sup>40</sup> This was a reasonable suggestion as tetracycline is a well known treatment for “walking

pneumonia” (Mycoplasma infection). This respiratory illness is caused by *Mycoplasma pneumoniae*, a microscopic organism related to bacteria but lacking a cell wall. Such mycoplasmas are larger than a virus but often smaller than a bacterium. They can only live inside a body cell and are also known to cause conjunctivitis (Pink eye) and otitis media (middle ear infection). Montagnier has been looking for the mycoplasma that he feels acts as a co-factor for HIV for over a decade but has yet to find it. Indeed, as the following quotation<sup>41</sup> explains, he is receptive to the possibility of multiple co-factors:

*We must know whether a virus acts alone or with an accomplice as we try to stop its spread. The body fights germs, parasites and bacteria at times; the immune system is always on alert. I have found that HIV on its own does not always cause disease, but in people who have already been infected with one of a family of mycoplasmas, HIV becomes deadly. Of course, we don't know yet if there is one or many co-factors. I think it is possible another virus could be the culprit; we seek the answer to that.*

It is clear from Montagnier's book *Virus*<sup>42</sup> and from the March 3, 2000 interview quoted above that the discoverer of HIV is far from convinced that this pathogen alone causes AIDS. It would appear, therefore, that the “HIV is the sole cause of AIDS” theory has been abandoned by its father. As a result, Montagnier has been denied funds to support his research into possible co-factors. Consequently, he co-founded (along with the director-general of UNESCO, Frederico Mayor<sup>43</sup>), the *World Foundation for Research and Prevention of AIDS*. This foundation is designed to fund basic and clinical AIDS research and humanitarian projects. One of its main aims is to provide Montagnier's laboratory at the Pasteur Institute in Paris with the resources it needs to conduct mycoplasma research, a field of endeavour seen by the scientific community as “too risky for public money.”<sup>44</sup>

## **ANOMALY SEVEN: HEMOPHILIACS AND SPOUSAL HIV SEROPOSITIVITY**

The widespread use of blood derived from prisoners and other high risk donors, long after HIV was known to have entered the supply, was one of the greatest medical scandals of the 20<sup>th</sup> century.<sup>45</sup> It caused the transmission of HIV to thousands of hemophiliacs who relied on blood and blood products to maintain normal health. Since hemophiliacs were generally unaware that they had become HIV-positive, many continued to have unprotected sexual intercourse with their spouses. These relationships provided the opportunity to study the risk of transmission of HIV in sexually active couples, one of whom was seropositive.

Andes and colleagues,<sup>46</sup> for example, surveyed a population of 30 monogamous hemophiliacs who had been treated with potentially-infected coagulation factor concentrates during the period 1980 to 1984. Of these, 24 were found to be HIV-positive. The duration of spousal HIV exposure from unprotected sexual intercourse ranged from more than 12 to 78 months. Interestingly, despite repeated opportunities to become infected, only 17 percent of spouses had become HIV-positive. That is, despite repeated unprotected sexual intercourse with a hemophiliac who carried HIV, 83 percent of their spouses had not been infected by the virus. In a comparable study Goedert and co-workers<sup>47</sup> concluded that the heterosexual transmission of HIV can occur during routine vaginal intercourse but it usually does not do so until the hemophiliac has severe immune deficiency late in the course of HIV infection. Why did HIV have so much trouble infecting the spouses of seropositive hemophiliacs? What was protecting the latter from the virus?

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*I don't try to describe the future. I try to prevent it.*

Ray Bradbury

As William A. Hasteltine pointed out in a 1992 lecture to the French Academy of Sciences, “The future of AIDS is the future of humanity.” Hasteltine,<sup>1</sup> then chief retro-virologist 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 afterwards he testified before a US Senate hearing,<sup>2</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 could reach one billion, that is about one sixth of the current global population. The passage of time has proved Hasteltine to have been a little optimistic. In *Vital Signs 2001*, Worldwatch<sup>3</sup> estimated that 57.9 million people had been infected with HIV by the end of the year 2000, 21.8 million of whom were already dead.

On September 11, 2001, some 4,000 innocent people died when terrorists crashed four commercial airliners, two of them into the towers of the World Trade Center. This event was international news for weeks, changed American foreign policy, and led to the bombing of Afghanistan. On September 11, 2001 over 5,000 people also died of AIDS.<sup>4</sup> The world’s media hardly noticed. After all, more than 5,000 people died of AIDS on September 10, and as many were killed by it on September 12, 2001. Given the number of HIV-positive individuals in Africa, Asia, and elsewhere, we can expect a mounting daily death toll for years, probably decades to come.

In 1976, this author<sup>5</sup> described a novel method of quantifying disasters, which has since become known as the Foster Scale. A new methodology was required to allow comparison of the significance of such events because there is so much variety in the types of negative impacts they create. Some disasters, such as pandemics, kill people but do little physical damage. Others can be responsible for immense loss to crops, livestock, fisheries, and other economic activities without directly causing any human casualties. A third group of disasters, such as earthquakes and floods, can be responsible for major damage, life loss, and injury. The Foster Scale attempts to accommodate this diversity of impacts by quantifying the one thing all disasters have in common: their capacity to generate stress. Its use involves combining the stress caused by the deaths, injuries, and infrastructural damage, linked to an adverse event, to obtain the total stress it creates. To illustrate, using the appropriate formulae, the volcanic eruption of Mt. Pelée that destroyed St. Pierre, Martinique was calculated to have caused 25,022,000 units of stress, while World War I was responsible for approximately 53 billion. A calamity magnitude scale was also developed so that disasters could be easily compared. This scale was logarithmic, so that an event registering 8 in the scale was 10 times as large as one with a magnitude of 7.

At the time of writing (October, 2001) the AIDS pandemic has been responsible for approximately 26.8 billion units of stress. While this qualifies it as a major catastrophe, it still ranks behind World War I in magnitude (Figure 1). However, if Hasteltine<sup>6</sup> is correct, roughly one billion people will have become HIV-positive by 2015, and of these perhaps as many as 250 million will already have died from AIDS. In this case, the AIDS pandemic will have generated some 450 billion units of stress, giving it a magnitude of almost 11.5 on the Foster Scale. This compares with 10.9 for the Black Death of the 14<sup>th</sup> century, 10.5 for World War I, and 11.1 for World War II. Simply



put, if there are no significant breakthroughs in the prevention and treatment of AIDS, by 2015 this pandemic will have become by far the worst catastrophe in human history.

Terrible though this prediction is, it underestimates the potential impact of the AIDS pandemic. The misuse of drugs by the livestock industry to fatten pigs, cattle, and poultry and their overuse by the medical profession has driven old pathogens to evolve into drug resistant strains.<sup>7</sup> Microbes are forced by drugs to adapt or die out. This “selective pressure” has inevitably stimulated the rapid evolution of microbes carrying genes for resistance to antimicrobial agents. Bacteria are particularly capable of developing such resistance for two reasons. They multiply rapidly and can transfer resistant genes from one strain to another.<sup>8</sup> Formerly effective drugs then no longer work against them.

Whenever the sick and susceptible congregate, resistant bacteria, such as *Staphylococcus*, can quickly spread. Overcrowding, inadequate sanitation and hygiene in slums and prisons have encouraged the diffusion of resistant pathogens that cause typhoid, tuberculosis, respiratory infections, and pneumonia.<sup>9</sup> Many of these diseases are growing more difficult, if not impossible, to effectively treat. At the same time, rapid transportation ensures that newly evolving resistant microbial strains are quickly spread. Resistant strains of gonorrhea that developed in Africa and Asia, for example, are now found globally.<sup>10</sup>

Organizations such as the WHO that are monitoring antimicrobial resistance<sup>11</sup> plead for reductions in the overuse and inappropriate use of antibiotics and related products. While this might slow the development of antimicrobial resistance, how realistic is it? The key characteristic of AIDS is the collapse of immune function and associated infections by pathogens, including but not limited to bacterial skin infections, shingles,

thrush, athlete's foot, oral hairy leukoplakia, tuberculosis, pneumocystis carinii, and toxoplasmosis. In the Developed World, those AIDS patients infected by such pathogens are given a wide variety of antibiotics.<sup>12</sup> Patients in the Developing World are demanding similar medications.<sup>13</sup> In consequence, unless major improvements occur in the prevention and treatment of AIDS, hundreds of millions of patients will be using antibiotics and related products to control AIDS-defining infections by 2015. This trend will inevitably increase the speed with which antimicrobial resistance is developing in such pathogens, creating strains that are ever harder to treat. Already, parallel pandemics of resistant tuberculosis and other diseases<sup>14</sup> are underway which, while most likely to kill HIV-positive individuals, are quite capable of leading to the deaths of others who are not infected by this virus.

This process is occurring in the former USSR, as Laurie Garrett<sup>15</sup> describes in her book *Betrayal of Trust: The Collapse of Global Public Health*.

*...with the collapse of the Communist State no one had the power to impose TB [tuberculosis] incarceration upon Russian or Georgian citizens ... so by 1997, officials said, Russia's primary resistance rate was 23.4 percent; 21 percent to two drugs; 19.4 percent to three drugs; 6.4 percent to more than four antibiotics.*

*In the jails, incidence of TB and resistant tuberculosis continued to rise. At the Tomsk central prison, for example, the TB incidence was 7,000 per 100,000 [7 percent] ... Estimated rates in other Russian prisons ranged from 2,481 per 100,000 [2.5 percent] to more than 7,000 per 100,000 [+7 percent].*

And the prison system was every day feeding costly, multi-drug-resistant, TB-infected patients into the beleaguered state public health network. A 1997 Ministry of Interior memo<sup>16</sup> estimated that by the year 2000 there would be 1.75 million

tuberculosis deaths in Russia, compared with 1.5 million from cancer and 1 million from heart disease.

HIV mutates rapidly and can also develop drug resistance.<sup>17</sup> Nucleoside analog reverse transcriptase inhibitors (NRTI), nonnucleoside analog reverse transcriptase inhibitors, and the HIV protease inhibitors make up the currently available arsenal of drugs for the conventional treatment of HIV infection.<sup>18</sup> These three classes of drugs aim to inhibit the replication of the virus. This they appeared to do quite effectively initially, so reducing death rates from AIDS.<sup>19</sup> However, all too often these treatments begin to fail because HIV is developing resistance to such therapeutic agents.

Unfortunately, the current treatment regime ultimately accelerates viral evolution. One result of this process has been the development of dangerous drug-resistant strains of HIV that are now rapidly spreading among sexually active individuals in North America, Europe, and elsewhere. For example, Dr. Martin Markowitz<sup>20</sup> of the Aaron Diamond AIDS Research Center in Manhattan led a team of New York and Los Angeles researchers who analyzed HIV in three women and 77 males who had been infected, on average, for less than 2 months. This meant that it was still possible to examine the original viral strain. It was found that HIV in 13 individuals (16.3 percent) had genotypes associated with drug resistance to some antiretroviral agent. Viruses with resistance to any nucleoside reverse transcriptase inhibitors were found in 10 patients, and to any non-nucleoside reverse transcriptase inhibitors in 6 subjects. Two individuals had viruses resistant to protease inhibitors. Multi-drug resistant virus was found in three of the newly infected sample. It is quite clear, therefore, that individuals being treated with highly active antiretroviral chemotherapy (HAART) are still transmitting HIV to their sexual partners in New York and Los Angeles. The virus strain passed on is often drug-resistant.

Such findings present “a serious threat to the management of antiretroviral therapy.”<sup>21</sup> Indeed, a new HIV ‘superbug’ that cannot be treated effectively with any existing AIDS drugs has begun to spread in Vancouver, British Columbia.<sup>22</sup> This strain is resistant to all three classes of drugs. The two patients infected by it have gone from being totally asymptomatic to having fully developed AIDS in a few months.

The current evidence, therefore, points to a basic weakness in the conventional treatment of HIV-positive individuals which is speeding up the evolution of not just the virus, but also a host of other AIDS-defining pathogens. This evolutionary process is leading to the emergence of far more deadly strains, not just of HIV but also of the microbes responsible for tuberculosis, typhoid, syphilis, pneumonia, and various other AIDS-related diseases. The situation will get far worse as such drug regimes become widely used in Africa and elsewhere in the Developing World.<sup>23</sup> Tuberculosis is already the principal cause of death worldwide in individuals who are HIV-positive<sup>24</sup> and it is becoming a major threat in its own right.

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*The retroviral map is like a map of the world in the fourteenth century. Some coastline is well-defined, other portions are blank and infested with dragons.*

Jonathan Mann, before resigning as  
Director of the WHO AIDS Program,  
cited by Peter Gould, *The Slow Plague*,  
1993, Oxford: Blackwell

*The Sixth no sooner had begun  
About the beast to grope  
Than, seizing on the swinging tail  
That fell within his scope,  
“I see,” quoth he, “the Elephant  
Is very like a rope!”*

John Godfrey Saxe  
*The Blind Men and the Elephant*

As I write, it is October 2001. There are 14 years remaining until 2015, just as 14 years have passed since 1987. During that year, then Soviet leader Mikhail Gorbachev called for more democracy. Andy Warhol died. The Herald of Free Enterprise capsized in the cold waters off Zeebrugge, with great loss of life. The presidential hopes of former Senator Gary Hart disappeared after his relationship with Donna Rice, a 29-year old model, was exposed by the media. Mrs. Thatcher swept into power for a third term in Britain. In Rome, Canada’s Ben Johnson surged past American Carl Lewis to set a new world record for the 100 metres of 9.83 seconds. On Black Monday, October 19<sup>th</sup>, the bottom fell out of the Stock Market, yet Van Gogh’s “Sunflowers” still sold in the same year for £24 million at Christie’s.<sup>1</sup> All of these events are still fresh in my memory. 1987 does not seem so far away, but neither does 2015.

There has been only one past. Human history can be modelled as a single line, with the present as its moving, leading edge. In contrast, there are endless potential futures, possibilities that radiate outwards from the present in every conceivable direction. Nevertheless, some are much more probable than

others. The future just described appears to be our most likely if no effective vaccine(s) is found for HIV and if conventional medicine continues its inevitably self-defeating drug-dominated battle against the virus. Under these circumstances, by 2015 we can expect the AIDS pandemic to have caused eight times more stress than World War I, with no end to the carnage in sight. HIV will never submit to a cease fire. Hasteltine<sup>2</sup> was right: unless the AIDS pandemic is controlled, there is no predictable future for our species.

The first rule of disaster planning is to identify and understand the hazards. Despite almost two decades of research, virologists still do not know how HIV causes AIDS.<sup>3</sup> Between infection and the onset of AIDS lies an unexplained disease process. As Brown<sup>4</sup> points out, it is not yet clear whether HIV directly destroys the immune system or provokes it into self-destruction. Or do both of these processes occur? This is not an unusual situation. There are many diseases, including tuberculosis, leprosy, and syphilis, for which the disease process is incompletely understood.<sup>5</sup> However, despite this, vaccines and therapies have been more or less successfully developed for these illnesses so that this lack of knowledge is of more academic than medical interest. This cannot be said of HIV. There is little doubt amongst scientists that, if real progress is to be made, HIV's tactics must be understood. Here, however, the consensus ends and gives way to a multiplicity of conflicting theories. Eight will now be briefly described. None has been confirmed, nor need they be mutually exclusive.

It was originally thought that AIDS results from the direct destruction of the CD4 T lymphocytes by HIV.<sup>6</sup> The virus certainly infects and destroys such immune cells but not at levels that seem particularly significant. The lowest estimate suggests that HIV infects only 1 in 10,000 such cells in the bloodstream at any one time; the very highest estimate is 1 in 100.

Research also indicates that long before their numbers drop significantly, the CD4 T lymphocytes of HIV-positive individuals steadily lose their ability to respond to foreign antigens. Even though HIV may be “thriving” in large reservoirs elsewhere in the body, such as in lymph nodes, few researchers still believe that the direct destruction of CD4 T lymphocytes by HIV is a sufficient explanation for AIDS.

In contrast, Knight and Patterson<sup>7</sup> believe that the destruction of the immune system by HIV comes from its ability to infect ‘sentinel’ dendritic cells. Such dendritic cells are vital for alerting CD4 T lymphocytes to foreign antigens and for stimulating specific responses to them. Without such dendritic cells, new CD4 T cells are not recruited and the infected individual becomes increasingly dependent on “memory” immune cells when recognizing and reacting to incoming pathogens. This loss of the ability of the immune system to recognize and react vigorously to infectious agents on the next encounter is thought to lead to a slow decline in its efficiency, culminating in AIDS.

A third possibility is that HIV can avoid the control of the immune system by very rapid mutation.<sup>8</sup> It is known that when compared with most other human viruses, HIV is exceptionally variable. This seems to be because it lacks the ability to “proofread” its genetic sequences during replication. As a result, it makes an average of one or two errors with each replication cycle. This means, of course, that the longer an individual has been HIV-positive the greater the number of variants of the virus they will carry. Some researchers believe that this variation itself may eventually overwhelm the immune system, leading to AIDS. As Brown<sup>9</sup> described, a team from Oxford University developed a mathematical model to examine this possibility. This simulation treats the virus as a multiplying prey and the immune system as its natural predator. Initially, the predator keeps the prey in check but, so the model

predicts, after a critical threshold of prey diversity is reached, the immune system (the predator) cannot keep up and the variants begin to multiply unchecked. Such constant change created by “errors” in viral replication is, of course, why even more dangerous strains of HIV have developed so rapidly. It is also one of the main reasons why it has proved so difficult to develop an effective vaccine.

Some researchers<sup>10</sup> believe that HIV infection may trigger programmed cell death, known as apoptosis, in CD4 T lymphocytes. There are certain normal circumstances under which healthy cells receive and obey signals to destroy themselves. This suicide occurs during the development of an embryo, that must not be attacked by the body. Another example of normal apoptosis takes place in the thymus where T cells mature. Certain subgroups of T cells recognize some proteins in the tissue of the body as “foreign.” If they were to multiply, such T cells would attack the host’s own body. To prevent this, they are programmed to destroy themselves on receiving certain signals, so preventing autoimmune diseases associated with inflammation of, or damage to, other cells around them.

It is thought by some research workers that HIV primes cells for such suicide and so destroys large numbers of CD4 T lymphocytes. Ameisen, Capron, and Groux,<sup>11</sup> of the Pasteur Institute in Lille, have suggested that HIV induces apoptosis by interfering with the signalling mechanisms of CD4 T lymphocytes. While many scientists believe that apoptosis may occur in HIV infection, they are unsure how important it is in the development of AIDS. Nevertheless, animal studies<sup>12</sup> have shown that when attempts are made to infect chimpanzees, rhesus monkeys, and African green monkeys with either HIV or SIV (simian immunodeficiency virus) apoptosis happens only in the animals that become ill and not in those like the African green monkey that can tolerate infection.

A fifth hypothesis which attempts to explain how HIV causes AIDS rests on the belief that the virus mimics crucial human defence proteins and so confuses the immune system into attacking itself. Proponents, such as Habeshaw and Dalgleish,<sup>13</sup> believe that HIV is responsible for a chronic autoimmune disease because it persistently activates certain groups of T cells. They argue that the way to prevent AIDS is to find a method of turning those T cells off. A group of human proteins known as MHC molecules (classes I and II) occur on the surfaces of cells that present foreign antigens to CD4 T lymphocytes, making them recognizable to these lymphocytes. Habeshaw and Dalgleish<sup>14</sup> argue that HIV's envelop protein (gp120) mimics MHC Class II molecules. As a result, this viral protein is recognized by certain T cells with specific receptors as a "foreign MHC," that is as "foreign human protein." Once such cells are alerted, there is a chain reaction of CD4 T lymphocyte activation which never stops, since HIV is always present in the body after infection. Eventually, this constant CD4 T lymphocyte activation is thought to result in the immune system collapse, as seen in AIDS.

Another alternative hypothesis is that T helper cells are thrown out of balance by HIV infection and so produce too few killer cells and too many antibodies. Clerici and Shearer,<sup>15</sup> for example, have emphasized the division of labour between two categories of T helper cells, which are known as TH1 and TH2. It is believed that TH1 cells are most involved in encouraging the production of killer T cells, while TH2 cells trigger antibody production by B cells. These researchers postulate that in HIV infection, TH2 cells become more common and the number of TH1 cells declines. Individuals with HIV infection show evidence that their B cells are overstimulated and, therefore, produce a large numbers of antibodies. A decline in TH1 cells, on the other hand, seems linked to a drop in the production of killer T cells that appear to be needed to control HIV.

Certainly, individuals with more TH2 cells than TH1 cells may be at greater risk of rapidly progressing to AIDS.

In 1999, Habeshaw and colleagues<sup>16</sup> suggested that HIV-associated immune deficiency had the characteristics of chronic graft versus host disease (GVHD), caused by human leukocyte antigen class 2 incompatibility. That is, AIDS was essentially caused by the mechanisms seen in transplants when organs are rejected. A year later, Cloyd and co-workers<sup>17</sup> postulated that HIV binding to resting CD4 T lymphocytes unregulates L-selectin, causing such cells to home from the blood into lymph nodes at an accelerated rate. The disappearance of CD4 T lymphocytes from the blood would then be the result of them leaving it to enter lymphoid tissues. Furthermore, secondary signals through homing receptors received during such homing may induce many of these cells into apoptosis, that is to “commit suicide.”

So where does all this disagreement leave us? It demonstrates just how illogical the conventional wisdom is. How is it possible to freely admit that it is still not clear, after some 20 years of research, how HIV causes AIDS,<sup>18</sup> yet to simultaneously argue, as the Durban Declaration does, that “HIV is the sole cause of AIDS.”<sup>19</sup> Simply put, if the 5,018 people who signed the Durban Declaration do not know how HIV causes AIDS they cannot possibly be certain that no co-factor(s) is involved.<sup>20</sup> What competent detective would argue that while they had no idea how the victim died, they were certain that their prime suspect had no accomplice(s)?

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*We knew that HIV infection or AIDS weakens the immune system and makes people at least 800 times more likely to have their latent TB infection activated. It wasn't realized until later that the reverse is also true: Active tuberculosis further suppresses the immune system of AIDS patients; curing tuberculosis actually improves the immune system.*

Lee B. Reichman,  
*Timebomb: The Global Epidemic of  
Multi-Drug-Resistant Tuberculosis,*  
2002, New York: McGraw Hill

## HIV: THE ACHILLES HEEL

*First a new theory is attacked as absurd; then it is admitted to be true, but obvious and insignificant; finally it is seen to be so important that its adversaries claim they themselves discovered it.*

William James<sup>1</sup>, *Pragmatism*, 1907

During World War II the Allies analysed Germany's war machine and decided that its greatest weakness was its reliance on industrial plants in Schweinfurt to produce all required ball-bearings. 122 American B-17s were shot out of the sky during two raids designed to destroy this centre of ball-bearing production and so cripple Germany's war effort. While the plan did not work, the basic principle was sound. Look for your enemy's weakest link and attack it. In this manner, defeat can be achieved with the least effort and greatest reliability. This approach worked far better when "The Dam Busters" attacked the Moehne and Eder dams, cutting off hydroelectricity to much of the Ruhr, Germany's industrial heartland.

Unfortunately, HIV is very resilient, with few weak links. It belongs to a viral class known as retroviruses, which have genes composed of ribonucleic acid (RNA) molecules.<sup>2</sup> Most other organisms, including humans, have genes made of a related molecule, deoxyribonucleic acid (DNA). All viruses can only replicate within cells after they have commandeered the cell's machinery to reproduce, but only retroviruses such as HIV use an enzyme called reverse transcriptase to convert their own RNA into DNA and incorporate it into the host cell's genes. HIV is a member of a subgroup of retroviruses called

lentiviruses which are often called slow viruses. Other members include the feline immunodeficiency virus (FIV) and the simian immunodeficiency virus (SIV). These infect cats and monkeys and other nonhuman primates respectively.<sup>3</sup> Such animal viruses, like HIV, focus their attacks on immune system cells and are associated with immunodeficiency and AIDS-like symptoms.<sup>4</sup>

HIV exhibits at least two characteristics that make it particularly difficult to eradicate. Firstly, as discussed already in the preceding chapter, HIV is exceptionally variable because it lacks the ability to “proofread” its genetic sequences during replication. The errors made result in a large number of variants being produced,<sup>5</sup> some of which will be immune to the antiretroviral agents being used in treatment. Secondly, within days of initial infection, HIV enters “resting” T-cells.<sup>6</sup> These cells are especially good places to hide because they are inactive and, therefore, they are not noticed by the immune system. Similarly, “resting” T-cells cannot be targeted by drugs which also require some form of activity, by either the infected cell or the virus, in order to work. Since “resting” T-cells can exist in the body for many years, even decades, without showing activity, HIV can remain undetected for a similar length of time.

HIV is not unique in being virtually impossible to eradicate. Many other viruses have a similar longevity inside the body. They include herpes simplex virus, cytomegalovirus, varicella zoster virus, and Epstein-Barr virus.<sup>7</sup> Typically, however, such viruses do not cause progressive diseases that gradually worsen over time. Rather they are generally held in check by a protective immune response. However, disease outbreaks can occur when the immune system is depressed. This is seen in intermittent herpes simplex infections and in the shingles suffered by the elderly who had chickenpox as children. The situation,

however, is very different with HIV. In the cytoplasm of the cell, HIV reverse transcriptase, as described, is used to convert viral RNA into DNA, the nucleic acid form used by the cell to carry its own genes. Conventional medicine has identified this process as a major Achilles heel of HIV and many of the anti-viral drugs used for the treatment of individuals who are HIV-positive or have AIDS are designed to interfere with this stage in the life cycle of the virus.<sup>8</sup> Known as nucleoside analog reverse transcriptase inhibitors and nonnucleoside analog reverse transcriptase inhibitors these drugs include AZT, ddC, 3TC, ddI, and d4T. As their names imply, they act by blocking reverse transcriptase activity.

During a process known as assembly and budding, newly produced HIV core proteins, enzymes, and RNA congregate inside the membrane of the infected cell, while the viral envelope proteins aggregate within the membrane itself. An immature viral particle is created and pinches off from the cell, acquiring both cellular and HIV protein from its membrane. At this stage of its life cycle, the virus is still immature and not yet infectious.<sup>9</sup> However, the long chains of enzymes and proteins that make up the immature viral core are then cleaved into many smaller pieces by a viral enzyme called protease. This process produces infectious viral particles and is seen as a second point at which HIV is most vulnerable to attack. Drugs called protease inhibitors are used to interfere with this step in viral replication. They include saquinavir, ritonavir, and indinavir.<sup>10</sup>

Unfortunately, as has been described previously, the use of inhibitors of reverse transcriptase and protease is gradually resulting in the evolution of drug-resistant strains of HIV that are now spreading rapidly in the Developed World. At least one of these strains is resistant to all three classes of drugs. Patients infected by it have gone from being totally asymptomatic to having fully developed AIDS in a few months.

The failure of most spouses of HIV-positive hemophiliacs to develop AIDS, despite years of unprotected sexual intercourse,<sup>11</sup> and the inability of HIV to spread in Senegal,<sup>12</sup> however, both suggest a viral Achilles heel that has yet to be exploited.

In an earlier chapter, the author pointed out that HIV appears to have been much easier to transmit in some countries than others. The Senegalese commonly practise unprotected sex with a variety of partners, but are still proving very resistant to infection with HIV-1 and therefore, for sub-Saharan Africa, have an extremely low AIDS mortality rate. This abnormality is of particular interest because in an earlier book, *Reducing Cancer Mortality: A Geographical Perspective*, this author<sup>13</sup> presented evidence which suggested that, after the effects of smoking and alcohol consumption had been accounted for, global cancer mortality seemed to be largely determined by environmental levels of protective selenium and calcium and cancer-promoting mercury and road salt. Subsequent research in China and elsewhere<sup>14-15</sup> confirmed that environments promoting human longevity also had elevated selenium and calcium and depressed mercury in their food chains.

Senegal is essentially a dried-up Cretaceous and early Eocene sea. When this dessication took place, sedimentary rocks were formed from the dissolved minerals in the evaporating sea water. As a result, calcium phosphates now mined for use in fertilizers are one of Senegal's chief mineral products. They are derived from phosphorite, a rock type that is always selenium-enriched.<sup>16</sup>

As a result of its equatorial climate, only some 2 percent of rainfall runs off in Senegal's rivers, so the population is almost entirely dependent on groundwater. Furon<sup>17</sup> has described this drinking water as having more calcium and magnesium in it than water drunk anywhere else on the planet. Conversely,

the environment contains little mercury, normally associated with igneous rocks, or road salt which is used in colder climates to de-ice highways. Senegal, therefore, has the ideal environment to protect against cancer and it is not surprising that its cancer incidence rates are easily the lowest on earth. In summary, the author believes that Senegal is fortunate to have the ideal environment for the support of the human immune system. Its inhabitants, therefore, rarely develop cancer<sup>18</sup> and have an abnormally low infection rate for HIV.<sup>19</sup>

Given the fact that AIDS is extremely common in those African countries, such as Zaire, that are known to have very selenium deficient soils,<sup>20-22</sup> the reverse also appears true. Individuals who are selenium deficient are easily infected by HIV, which quickly progresses to AIDS. This would explain why in HIV-seropositive drug addicts<sup>23</sup> and maternally infected infants,<sup>24</sup> low plasma selenium levels are a better indicator of probable future mortality than depressed CD4 T lymphocyte levels. It would also explain why so many of the spouses of HIV-seropositive hemophiliacs failed to become HIV-infected,<sup>25</sup> even after years of unprotected sexual intercourse. Such partners were likely to be eating diets containing adequate selenium and, therefore, were resistant to HIV transmission. This hypothesis would also account for the high frequency of Kaposi's sarcoma in AIDS patients. Kaposi's sarcoma is known to be linked to the human herpesvirus 8 (HHV-8) and has been endemic in Uganda and other selenium deficient regions of sub-Saharan African for many years.<sup>26</sup> Myxoedematous cretinism, a developmental abnormality associated with inadequate selenium and iodine also occurs in this region.<sup>27</sup> It is obvious, therefore, why HIV and HHV-8 are found together in so many AIDS patients. Both viruses are members of the deficiency branch of the selenium family tree. That is they only occur in individuals who are deficient in selenium. This is why AIDS patients, who are known to be both selenium deficient and

infected with HIV are also highly vulnerable to developing Kaposi's sarcoma. Both HIV and HHV-8 have the same co-factor, selenium deficiency.

This hypothesis also explains why breast feeding encourages the development of AIDS in HIV-seropositive nursing mothers.<sup>28</sup> Elsewhere, the author has shown that iodine deficient mothers have infants that are likely to die of SIDS.<sup>29</sup> Such women also develop goiter during pregnancy. This is because essential nutrients including iodine and selenium are passed by the mother to her child during breast feeding. If the woman is HIV-seropositive, she is already likely to be selenium deficient. Any further loss of this mineral to her child will simply increase this problem. If, as suggested here, HIV can replicate more easily in selenium deficient individuals, the passage of selenium from a mother to her nursing infant will accelerate the rate at which HIV replicates in her body and encourages the development of AIDS. The infant is likely to die subsequently because a selenium deficient mother is a poor source of this essential trace element. If, as suggested here, HIV has difficulty infecting individuals who are not selenium deficient and can only progress into AIDS in the absence of adequate serum levels of this trace element, then this is clearly another viral Achilles heel. Selenium is cheap, simple to acquire, and can be added to fertilizers, fodder crops, and various foods.<sup>30</sup> It is also easy to take in tablet form.

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*The next speaker, Dr. Tom Spira of the CDC, presented some options. One thing that struck epidemiologists, he said, was the correlation between AIDS and Hepatitis B—in fact, nearly 90 percent of the known AIDS sufferers in America had been exposed to Hepatitis B. With such a tight correlation between hepatitis and AIDS, Hepatitis Could serve as a “surrogate marker” for AIDS—an indication of those who might harbor the disease.*

Douglas Starr,  
*Blood: An Epic History of Medicine and Commerce*,  
1998, New York: Harper Collins.

## SELENE: GODDESS OF THE MOON

*It's time to stop ignoring the facts! If you want to maintain your good health, increase your resistance to disease and ensure a long and energetic life, it is vitally important that you increase your intake of selenium.*

Richard A. Passwater<sup>1</sup>

Will Taylor, from the University of Georgia, has been a driving force behind the hypothesis that selenium deficiency plays a significant role in AIDS.<sup>2</sup> Taylor and his co-workers<sup>3</sup> have demonstrated that HIV-1 encodes a selenium-containing protein, specifically the enzyme glutathione peroxidase. This means that the virus cannot replicate without competing with its host for this trace element. To confirm that this is happening, Baum and colleagues<sup>4,5</sup> have shown that not only are AIDS patients selenium deficient, but the lower the level of this trace element is in the blood, the more likely they are to die. Indeed, numerous other studies have confirmed that HIV patients are always selenium deficient and that as AIDS progresses, this deficiency relentlessly increases.<sup>6</sup>

There seems to be more to the HIV-selenium relationship, however, than just the depletion of this trace element by the virus. Differences in environmental selenium levels appear to be influencing who is infected by HIV and where. This viewpoint is now more accepted by the geochemists who study selenium. Indeed, the recently published *Selenium World Atlas* used the incidence of HIV-positive populations as a surrogate measure of selenium deficiency in Africa, since knowledge of actual soil levels of this trace element is quite scarce.<sup>7</sup> This argument by

analogy was made on the advice of E.W. Taylor, who was the first to recognize that the diffusion of HIV-1 in Africa was occurring most rapidly in selenium deficient regions.<sup>8-9</sup>

A link between elevated mortality from AIDS and depressed environmental selenium has been confirmed in the continental United States by Cowgill.<sup>10</sup> Selenium concentrations in US soils previously had been established by Kubota and associates<sup>11</sup> on the basis of this element's level in local alfalfa. Cowgill used analysis of variance to search for possible statistically significant relationships between age-specific and age-adjusted AIDS mortality data for 1990 and environmental selenium, established using alfalfa concentrations. He found that in the US:

*... a pattern exists between the geographical distribution of Se [selenium] using alfalfa as a dietary guide and AIDS mortality such that an inverse relationship persists between Se quantity in an area and AIDS mortality in the same area.<sup>12</sup>*

This inverse relationship between the death rates from AIDS and local selenium levels was particularly obvious amongst Blacks. Cowgill<sup>13</sup> believed that this was because they tended to be less mobile and more likely to eat locally grown foods. It seems clear then that, as in Africa, AIDS deaths are higher in the United States where local foods contain less selenium.

It has been known for many years that poor nutrition increases susceptibility to viral infection. It was generally believed that an inadequate diet compromised the immune system so that it was less able to protect against viruses. However, recent work by Beck and her co-workers<sup>14-15</sup> has shown that the relationship is not so simple. Their laboratory work demonstrated that a normally benign Cocksackievirus caused significant heart damage in selenium deficient mice. These researchers further showed that the virulent strain of this virus (recovered from the selenium deficient host) differed significantly from the original

virus. Mutation had occurred to produce a much more virulent phenotype displaying six nucleotide changes. Furthermore, if this new, more virulent strain of Coxsackievirus was used to inoculate selenium-adequate mice it caused significant heart disease. It is not clear whether such an increase in virulence caused by selenium deficiency is occurring in HIV, but it is certain that AIDS patients become progressively selenium deficient and, as they do so, are infected by an ever increasing number of variants of HIV.

This relationship between virulence in the Coxsackievirus and heart disease in mice is not simply of academic interest. Keshan disease is widespread and endemic in selenium deficient areas of China.<sup>16</sup> It presents as a non-obstructive cardiomyopathy and occurs in people who are both deficient in selenium and infected by the Coxsackievirus. Keshan disease, therefore, can be viewed as a selenium-deficiency disease with a viral co-factor, or vice versa.<sup>17</sup> Interestingly, it may be only the tip of the iceberg. Nicholls and Thomas,<sup>18</sup> for example, showed that 10 of 38 patients with acute myocardial infarction admitted over a 2 month period to King Edward VII Hospital, Midhurst, Sussex had serological evidence of very recent Coxsackie B virus infection. That is, roughly 25 percent of these British heart attack patients had suffered from an influenza-like illness caused by Coxsackie B virus within 7 days prior to admission.

There seem to be several other viruses that require selenium-deficiency as a co-factor before they become virulent. One of these is Hepatitis B. In certain low selenium regions of China, for example, both liver cancer and Hepatitis B viral infection are endemic. A major 5-year trial involving selenium supplementation was able to significantly reduce not just deficiencies of this trace element, but also the incidence of both viral Hepatitis and liver cancer.<sup>19-20</sup> It seems likely, therefore, that

selenium-deficiency is a co-factor for Hepatitis B and the liver cancer commonly associated with it. Indeed, Taylor<sup>21</sup> has suggested that a variety of viruses carry selenoprotein genes, which means they must deplete the body's selenium stores as they replicate. Included amongst these are Coxsackie B3, Ebola Zaire, Molloscum contagiosum, Hepatitis C virus, and HIV. The highly pathogenic Zaire strain of Ebola, for example, is one such virus.<sup>22</sup>

*... where one such potential gene has 16 UGA selenocysteine codons, as well as structural features necessary to express this selenoprotein, which would require 16 Se atoms per molecule. This suggests that infection with Ebola Zaire may place an unprecedented demand for selenium on the host, potentially causing more drastic Se depletion in a matter of days than HIV infection can accomplish in 10 years.*

Simply put, there appears to be a fairly large group of viruses that encode selenoproteins. As they replicate, therefore, they rob their hosts of selenium, causing immune system dysfunction that includes depression of CD4 T lymphocytes (which also require selenium). Such a decline in serum selenium is very well documented in AIDS patients and gets progressively greater over time as the disease worsens.<sup>23-24</sup> Look and colleagues,<sup>25</sup> for example, write that “the progressive deprivation of serum selenium in HIV-infection is associated with loss of CD(4+)- cells and with increased levels of markers of disease progression and inflammatory response.” As HIV-seropositive individuals progress into AIDS they become increasingly selenium deficient. Selenium is an essential trace element with several key functions in the body, so it must follow that the selenium deficiency that accompanies AIDS is responsible for some of the associated disease symptoms. This is clearly the case.<sup>26</sup>

Selenium, for example, plays an essential role in thyroid function. Without it thyroxine (T4) cannot be converted to triiodothyronine (T3) because the process requires a de-iodinase enzyme

that includes selenium.<sup>27</sup> It is not surprising, therefore, that a low T3 syndrome accompanies AIDS and may be a factor in AIDS wasting syndrome.<sup>28</sup> This makes sense because human growth hormone,<sup>29</sup> used to treat wasting, stimulates conversion of T4 to T3. This process is bound to be ineffective in the presence of depressed selenium because this shortage will inevitably result in inadequate levels of the de-iodinase enzyme that is essential for this hormone conversion.<sup>30</sup>

As described previously, the Coxsackievirus causes Keshan disease<sup>31</sup> in selenium deficient areas of China and may be linked to many of the cases of acute myocardial infarction seen in the Developed World.<sup>32</sup> If this is the case, such heart attacks ought to be common in those with AIDS, who are both selenium deficient and obvious targets for Coxsackie infection. As might be expected, cardiac-related deaths are indeed quite common in AIDS patients<sup>33-34</sup> and it seems likely that selenium deficiency is a co-factor in many of those mortalities.

One of the body's key defences against the ubiquitous and destructive process known as lipid peroxidation is the selenoenzyme glutathione peroxidase. Another antioxidant protein called selenoprotein 8 is the chief type of selenium found in human plasma.<sup>35</sup> When lipid peroxidation is not reversed, it causes cell membrane destruction. It is also known that when T cells are short of the selenoenzyme glutathione peroxidase they accumulate lipid hydroperoxides which stimulate programmed cell death (apoptosis).<sup>36</sup>

In short, selenium deficiency reduces the availability of glutathione peroxidase leading to the depletion of CD4 T lymphocytes seen in HIV-seropositive patients. Selenium deficiency, therefore, is a direct cause of the immune system decline that is associated with AIDS. The reverse is true. Selenium supplementation increases the cytotoxicity of killer T cells and

encourages the proliferation of T cells<sup>37</sup> when challenged with mitogens and antigens. It follows, therefore, that without adequate selenium, CD4 T cell counts will fall and with them the efficiency of the immune system.

Selenium deficiency is also known to be associated with greatly increased cancer risk.<sup>38</sup> Conversely, supplementation has been shown to reduce the incidence of a wide range of cancers, including those of the lung, prostate, and digestive tract.<sup>39</sup>

The evidence presented so far shows that the genome of HIV-1 includes a homologue for glutathione peroxidase. This is an enzyme that is crucially important to human health and which contains selenium. As HIV-1 replicates it must compete with its host for the minerals needed to make this essential enzyme. HIV-seropositive individuals and AIDS patients, therefore, have depressed blood selenium levels that continue to fall as the virus replicates and the immune system declines. Selenium deficiency itself is known to be linked with thyroid hormone malfunction, cardiomyopathy, depressed serum CD4 T lymphocyte levels, and various cancers. It follows that whatever the role of selenium in the development of AIDS, supplementation with this trace element is essential in those who are HIV-seropositive or have AIDS. Taylor,<sup>40</sup> in his excellent review of the role of selenium in viral diseases, suggests that “a dose of 400 mcg [micrograms] seems reasonable for HIV-infected individuals, if they do have impaired absorption.”<sup>41</sup> This seems to be a reasonable suggestion. I have been taking 200 micrograms daily since 1984 with no obvious adverse side-effects. If I was HIV-seropositive, which thankfully I am not, this dose would have been doubled.

There remains one interesting enigma concerning not only HIV but also all of the other viruses, including Hepatitis B and C, Coxsackievirus B3, and Ebola Zaire, that have selenium-

utilizing genes. The geographical, laboratory, and clinical trial evidence suggests that all of these viruses are much more liable to cause disease in low selenium environments where the populations are typically selenium deficient. As Taylor<sup>42</sup> writes:

*... if the virus requires Se, why is it that a deficiency of Se appears to be associated with increased viral replication, and Se supplementation inhibits the virus, rather than "feeding" the virus?*

There are various possible answers to this question. Taylor<sup>43</sup> suggests that it "most likely involves some sort of repressor type of mechanism, analogous to known situations in bacteria, like the famous tryptophan repressor ... it seems quite possible that viruses like HIV and Coxsackie B3 may respond to Se deficiency by a mechanism analogous to the involved in this arginine effect." However, it is also possible that since oxidative stress stimulates HIV replication and the selenoenzyme glutathione peroxidase reduces oxidative stress, any shortage of selenium encourages HIV replication.<sup>44</sup> It is also possible that HIV is simply unable to infect any individual with adequate serum selenium and a well functioning immune system. This certainly appears to be one possible explanation for the low AIDS mortality in Senegal and the inefficient transmission of HIV-1 to the spouses of hemophiliacs.

What happens when AIDS patients are given selenium supplements? As yet, few results are available from clinical trials. However, as early as August 1987, the *Positive Health Centre*, Harley Street, London<sup>45</sup> reported treating three AIDS patients with vitamins and mineral supplements that included 100 micrograms of selenium taken three times daily. All of these individuals markedly improved and, still in good health, had exceeded the then life expectancy for people who were HIV positive.

In addition, Schrauzer and Sacher<sup>46</sup> reported on a 70 day study that involved giving 400 micrograms of selenium to HIV-seropositive men. Serum selenium levels increased and 74 percent of the 19 subjects reported improvements in their appetites and gastrointestinal function as well as neurological and psychological benefits. Batterham and co-workers<sup>47</sup> also have shown that daily antioxidant supplementation, including 100 micrograms of selenium, significantly improved some measures of oxidative defence in men who are HIV-seropositive. Clearly, a great deal more work has to be done to assess the value of selenium supplementation in HIV/AIDS patients. Initial results seem very promising. Indeed, many other individuals may benefit from selenium supplements. Peretz and co-workers<sup>48</sup> have shown that even in elderly institutionalized patients who were not HIV-seropositive, supplementation with selenium-enriched yeast improved lymphocyte response, demonstrating its immunostimulatory properties.

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*AIDS is a watershed in the study of human disease, and when you keep your antennae turned in the right direction, you can hear something happening off stage as the shimmering crystals begin to coalesce and form new patterns in science and in society. Tomorrow another collection of molecules will drive the world of medicine. Today is the day of the virus.*

Ann Giudici Fetter,  
*The Science of Viruses*,  
1990, New York: McGraw-Hill

## THE “SELENIUM-CD4 T CELL TAILSPIN”

*Perhaps it is better to be irresponsible and right than to be responsible and wrong.*

Winston S. Churchill<sup>1</sup>

AIDS is a disorder defined by a committee. The term was first used in 1982 in a *Morbidity and Mortality Weekly Report*, published by the Centers for Disease Control.<sup>2</sup> In this article, AIDS was defined as “... a disease, at least moderately predictive of a defect in cell-mediated immunity, occurring with no known cause for diminished resistance to that disease.” The initial list of AIDS-defining conditions included amongst others *Pneumocystis carinii* pneumonia, *Mycobacterium avium* complex, and Kaposi’s sarcoma. Since 1982, the definition of AIDS has been revised on several occasions. The most significant of these revisions termed the *1993 Revised Classification System for HIV Infection and Expanded Surveillance Case Definition for AIDS Among Adolescents and Adults* was published in the December 18, 1992 edition of the *Morbidity and Mortality Weekly Report*.<sup>3</sup> Here AIDS was defined as the presence of at least one of 25 AIDS-indicator conditions, in patients with HIV infection, who had a CD4 T lymphocyte count of less than 200 cells per cubic millimetre (mm<sup>3</sup>) of blood.

There seems to be no other infectious disease defined in such a curious manner. If one has cholera they have been infected by the bacterial strain *Vibrio comma*; scarlet fever by streptococcus bacteria; smallpox by *Variola* and so on. No other infectious agent is needed to develop or define these diseases. The supporters of the conventional wisdom that “HIV is the

sole cause of AIDS” see the 25 AIDS-indicator conditions as opportunistic infections that attack AIDS patients because their immune systems have been seriously weakened by HIV. However, there is a much more logical explanation why AIDS never occurs (by definition) without the presence of at least one other infectious disease. Each one of these AIDS-indicator conditions acts as a co-factor because they cause the decline in selenium that is necessary for HIV to replicate.

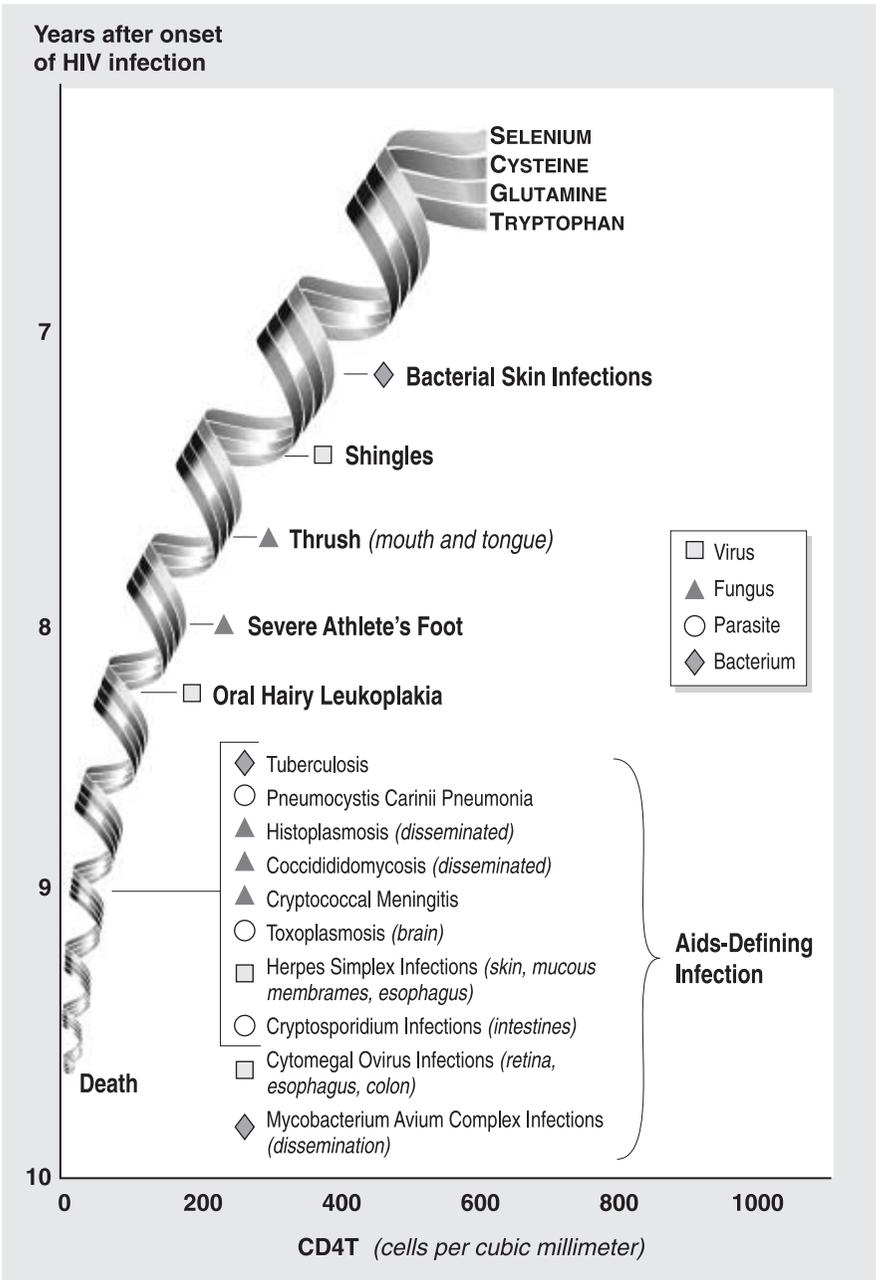
This relationship between infectious diseases and selenium depression was documented first by Sammalkorpi and colleagues.<sup>5</sup> These researchers described measuring serum selenium and iron concentrations in 64 patients with uncomplicated viral (n=33) or bacterial (n=31) infections, during the acute stage of infection, the early convalescent phase and after a minimum recovery of 3 weeks. Both serum selenium and iron showed highly significant depression during the acute stage of infection but rebounded after recovery. Sammalkorpi and co-workers<sup>6</sup> not surprisingly concluded that any acute infection would decrease serum selenium levels. It has been shown repeatedly that serum selenium levels are linked directly to immune system function, including CD4 T lymphocyte count.<sup>7-8</sup> It follows, therefore, that the decline in serum selenium levels that accompanies viral or bacterial infection will be paralleled by some degree of immunological depression. However, as Sammalkorpi and co-workers<sup>9</sup> have shown, under normal circumstances where death does not occur, serum selenium levels rebound soon after recovery from bacterial or viral infection.

However some pathogens, including certain retroviruses, effectively elude the immune system’s defence mechanisms and can continue to replicate indefinitely. Of particular significance are those viruses such as Coxsackievirus B3, Hepatitis C virus, and HIV-1 which encode glutathione peroxidase and, therefore, continue to deplete their hosts’ selenium.<sup>10</sup> It appears, there-

fore, that while HIV-1 is sensitive to high levels of serum selenium, sequestered within CD4 T lymphocytes and elsewhere, it can slowly deplete its hosts' selenium stores. The associated decline in available selenocysteine must have a negative impact in the body's ability to produce CD4 T lymphocytes which require this compound.<sup>11</sup> This drop in CD4 T lymphocytes, however, permits the so-called opportunistic infections to thrive, further depressing serum selenium levels and allowing high replication rates for HIV-1. As can be seen, once the "selenium-CD4 T cell tailspin"<sup>12</sup> has begun, it accelerates; not only because AIDS-defining infections increase selenium depletion, but also because HIV itself encodes not just for this trace element but also for the components of glutathione (Figure 2). As a result, because glutathione inhibits reverse transcriptase activity, as serum levels of glutathione drop, it becomes easier for HIV to replicate.<sup>13-14</sup> This is because, as described previously, retroviruses such as HIV use reverse transcriptase to convert their own RNA into DNA<sup>15</sup> and incorporate it into their host cell's genes. The removal of an inhibitor, glutathione, from the system facilitates this process.

The "selenium-CD4 T cell tailspin," therefore, is a positive feedback system which, like an avalanche or forest fire, becomes more and more dangerous over time. It does so because once other infectious agents have begun to lower serum selenium levels, HIV can replicate with increasing speed, removing from the host's blood the two substances that naturally inhibit its spread—selenium and glutathione.

This positive feedback system has caused considerable confusion in both conventional and unconventional AIDS theory. Such misinterpretation perhaps can be explained best by analogy. Consider a gang of thieves that specializes in cracking safes, but has only one member with the ability to actually open one. When the safe to be cracked is in a high-rise building, he



**Figure 2** Depletion of four essential nutrients by the replication of HIV-1 and its associated infections. (Infection information is based on Mills and Masur, 1990.<sup>16</sup>)

will be accompanied to the crime scene by a cat burglar, who gains them both entry. He may on other occasions need the assistance of a driver for a fast getaway, "muscle" for protection, or a fence to sell stolen antiques. Simply put, while this expert safe cracker [HIV] cannot commit the crime unaided, he is always active during the robbery, accompanied at different times by a wide variety of accomplices [AIDS-defining conditions and anything else capable of depressing serum selenium].

Detectives trying to identify the criminals involved have generally taken one of two erroneous stances. Some, like those who signed the Durban Declaration<sup>17</sup> and members of the Centres for Disease Control,<sup>18</sup> have argued that since all of the potential accomplices for the known safecracker [HIV] have alibis for some of the crimes, they cannot have been involved in any of them:

*However, extensive epidemiologic and laboratory studies of HIV-infected persons have failed to identify any consistent factor, including drug use, malnutrition, or co-infections with other organisms, that affects the rate of progression to AIDS.<sup>19</sup>*

In contrast, most AIDS mavericks (with the notable exception of Montagnier) have identified various gang members (co-factors capable of independently lowering serum selenium levels) that are known to be present at some of the crime scenes (AIDS) and have claimed that they are solely responsible for all of the robberies. Such gang members include drugs,<sup>20</sup> HHV-6 (Human Herpes Virus-6)<sup>21</sup> and syphilis.<sup>22</sup> To quote Duesberg:

*Based on the lifestyle hypothesis of the early 1980s and my own research I have proposed that in IAV drugs cause AIDS. The drug hypothesis holds that AIDS is caused either by recreational drugs, or by DNA chain terminators such as AZT prescribed as anti-HIV drugs, or by a combination of both.*

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*The emerging viruses are surfacing from ecologically damaged parts of the earth. Many of them come from the tattered edges of tropical rain forest, or they come from tropical savanna that is being settled rapidly by people. The tropical rain forests are the deep reservoirs of life on the planet, containing most of the world's plant and animal species. The rain forests are also its largest reservoirs of viruses, since all living things carry viruses. When viruses come out of an ecosystem, they tend to spread in waves through the human population, like echoes from the dying biosphere. Here are the names of some emerging viruses: Lassa; Rift Valley; Oropouche; Rocio; Q. Guanarito; VEE; Monkeypox; Dengue; Chikungunya; The hantaviruses; Machupo; Junin; The rabieslike strains Mokola and Duvenhage; LeDantec; The Kyasanur Forest brain virus; HIV — which is very much an emerging virus, because its penetration of the human species is increasing rapidly, with no end in sight.*

Richard Preston,  
*The Hot Zone*,  
1994, New York: Random House

## WHY NOW?

*Ring a-ring a-roses  
A pocketful of posies  
'Tishoo, 'tishoo,  
We all fall down*

L.W. Cowie<sup>1</sup>

17<sup>th</sup> Century Bubonic

Plague-related nursery rhyme

Throughout human history pandemics have swept through the known world.<sup>2</sup> Typically, millions have died from a particular pathogen which then retreated, only to return later as community immunity to it declined. Cholera, influenza, typhoid, smallpox, and bubonic plague, for example, have taken repeated heavy tolls.<sup>3</sup> There is, however, no convincing evidence of repetitive AIDS pandemics. The current scourge appears to be the first. Why then did HIV only begin to infect the human population on a global scale, for the first time, in the last two decades of the 20<sup>th</sup> century?<sup>4</sup> As discussed in this book's second chapter, there must have been hundreds if not thousands of past opportunities for the necessary simian to human viral transmission to occur. Indeed, we are aware of at least seven such recent viral introductions into the human population.<sup>6</sup>

The AIDS establishment has no answer to the “why now?” question. The reasons for the timing of the current pandemic seem obvious, however, if the validity of the “selenium-CD4 T cell tailspin” is accepted. There appears to be a minimum daily dietary selenium intake above which, as seen in Senegal,<sup>7</sup> HIV cannot be easily transmitted. If this is correct, any widespread

drop in selenium in the food chain would encourage the spread of HIV from source areas and, as a result, greatly increase mortality from AIDS. Rapid diffusion of HIV could also occur if other factors were weakening human immune systems, encouraging a diversity of pathogens, which in turn lowered serum selenium levels.<sup>8</sup> Both of these processes could occur simultaneously.

If global serum selenium levels declined in the 20<sup>th</sup> century, one would have expected to see increases in other diseases and disorders that flourish in selenium deficient environments. That is, the AIDS pandemic would have been paralleled, for example, by an increase in cancer,<sup>9</sup> heart disease,<sup>10</sup> and Hepatitis B and C,<sup>11</sup> all of which are caused, at least in part, by inadequate selenium intake.

There can be little doubt that age adjusted cancer mortality has risen rapidly during the 20<sup>th</sup> century<sup>12</sup> and there is a wealth of evidence from animal laboratory studies, geographical and epidemiological research, and field trials that selenium plays a major prophylactic role in cancer.<sup>13-15</sup> One field trial in the US,<sup>16</sup> for example, involved 1,312 patients, with a mean age of 63, who had a previous history of skin cancer. Fifty percent of these received daily 200 microgram selenium supplements, while the remainder were given a placebo. While selenium had no impact on skin cancer, the incidence of cancer in total fell 41 percent in those patients who received selenium. The declines in prostate cancer, colorectal cancer, and lung cancer incidences, 69, 64, and 46 percent respectively, were particularly impressive. It is clear, therefore, that selenium is protective against many cancers and that these have been increasing in frequency during the 20<sup>th</sup> century, a trend that is consistent with declining serum selenium in the general population. Cardiovascular disease, that is disease of the heart and blood-supply vessels, is now the major cause of death in the US and

accounts for almost 50 percent of all mortality.<sup>17</sup> There is nothing exceptional about this preeminence since cardiovascular disease is also the chief cause of death in almost all Developed countries.<sup>18</sup> While many risk factors are involved in cardiovascular disease, there appears to be a close negative association between mortality from it and high soil selenium levels.<sup>19</sup> Indeed, Kok and colleagues<sup>20</sup> clearly demonstrated significantly low blood and toenail selenium concentrations in 84 Dutch patients with acute myocardial infarction, as compared to a similar number of controls. This suggests low selenium status prior to infarction. Interestingly, as mentioned previously, Nicholls and Thomas<sup>21</sup> found that roughly 25 percent of patients with acute myocardial infarction admitted to King Edward VII Hospital in Midhurst, Sussex had suffered a Coxsackie B viral infection in the previous week. This is not too surprising as this virus encodes selenium and is a major cause of Keshan disease, an endemic cardiomyopathy in the regions of China where soil selenium levels are particularly depressed.<sup>22</sup> It would appear, therefore, that the extremely high cardiovascular disease mortality seen in the Developed World is partially a reflection of declining environmental selenium levels.

Hepatitis B and C, also members of the deficiency branch of selenium's family tree,<sup>23</sup> are spreading rapidly as would be expected if this trace element is becoming scarcer in the food chain. This is true of Eastern Europe and Russia, for example, as described by Garrett:<sup>24</sup>

*While some other countries in the region had better blood banking systems, only a handful had resources for universal screening of donors for Hepatitis B and C, HIV, or any other dangerous viruses. Given the extraordinary explosion of these viruses occurring in the IV drug-using population, and the local practice of paying donors for providing blood or plasma, this seemed an extraordinary regional public health time bomb.*

*Nowhere was that possibility as scary as in Russia. Across the entire eleven-time-zone length of the vast nation, Hepatitis, in particular, was emerging from obscurity into a full-fledged epidemic.*

According to the World Health Organization,<sup>25</sup> Hepatitis B is now one of the major diseases of mankind. Of the 2 billion people who have been infected by the Hepatitis B virus (HBV), more than 350 million have developed chronic infections. Such lifelong infected individuals are at high risk of death from cirrhosis of the liver and liver cancer, which together are killing about one million people annually. The Hepatitis C virus (HCV) is also a major cause of cirrhosis and liver cancer.<sup>26</sup> Globally, an estimated 170 million individuals are chronically infected with it and 3 to 4 million are newly infected each year. Like HIV, HCV seems to be very easily spread by direct contact with human blood.

It is obvious from this brief summary that the Hepatitis B and C viruses, both members of the deficiency branch of selenium's disease family tree, have also diffused globally during the late 20<sup>th</sup> century. Interestingly, cancer and Hepatitis B and C commonly occur together in AIDS patients, who of course are both HIV-seropositive and selenium deficient. Indeed, as Shilts<sup>27</sup> pointed out, in the late 1970s before the AIDS pandemic was recognized in San Francisco, cases of Hepatitis B quadrupled in gay men. Looked at from this perspective, there is nothing particularly unusual about the rapid diffusion of HIV in the closing decades of the 20<sup>th</sup> century. AIDS is only one of several diseases that include cancer, myocardial infarction, and Hepatitis B and C that reached pandemic proportions as the millennium approached. All of these diseases are members of the deficiency branch of the selenium family tree and so occur most often in individuals who are deficient in this essential trace element. While this rapid increase in the prevalence of such diseases does not prove that serum selenium depression

occurred in the human population at this time, it is virtually impossible to envisage such parallel pandemics occurring unless this was indeed the case.

In 1950, coal, oil, and natural gas consumption (known as total global fossil fuel use) reached 1,666 million tons of oil equivalent.<sup>28</sup> This figure rose steadily throughout the remainder of the 20<sup>th</sup> century, so by 1998 fossil fuel consumption had reached 7,869 million tons of oil equivalent.<sup>29</sup> That is, in slightly less than 50 years mankind had increased its annual fossil fuel use by a factor of almost 5. Coal combustion had more than doubled, oil consumption had increased by a factor of almost 8, and natural gas had been employed as a fuel at a rate of roughly 11 times that of 1950.

Throughout the 20<sup>th</sup> century, as a result of the growing fossil fuel consumption, ever increasing quantities of sulphur and nitrogen were emitted into the atmosphere, where they were converted into sulphuric and nitric acids,<sup>31</sup> increasing the acidity of subsequent precipitation. Rainfall and snow was also often polluted with mercury and other heavy metals. Pesticides and assorted persistent organic pollutants were spread by the global wind systems. Much of this pollution came from industry, agriculture, transportation systems, and residences, but was added to by fires used for deforestation and the burning of chaff. Such acid and heavy metal rains have caused extensive damage to the environment at local, regional, and even global scales. They have been particularly problematic in Northern and Central Europe, Eastern North America, and Eastern China where they are associated with many indirect health effects.<sup>32</sup> Problems associated with pollutants generated by deforestation using fire are now commonplace in the Developing World.<sup>33-34</sup>

Such acid precipitation has a major impact on the capacity of the soil to bind elements, which reaches its maximum under

neutral or slightly alkaline conditions. There are, however, exceptions to this general rule. Arsenic, selenium, molybdenum, and some valency states of chromium are more mobile in alkaline or calcareous soils. The net result of acid precipitation, then, is that it alters soil pH and, as it does so, increases the bioavailability of some elements and lowers that of others, including selenium.<sup>35</sup> As the global acidification of precipitation has intensified with the increasing use of fossil fuels and biomass for energy purposes and fire-based deforestation practices, it has reduced selenium's relative abundance in the food chain.<sup>36</sup> This process has been exacerbated by the presence of mercury and other heavy metals in rainfall. The latter, for example, combines with selenium to form highly insoluble mercury selenide<sup>37-38</sup> which is unavailable to plants and, therefore, does not enter the food chain.

Since sulphates, nitrogen, and phosphorus all depress the uptake of selenium by crops, the widespread use of commercial fertilizers also is causing a decline in the availability of this element in the human food chain.<sup>39</sup> In addition, domestic cattle and other animal livestock reduce selenium bioavailability because the form of selenium excreted in their urine, trimethylselenonium, is largely unavailable to plants.<sup>40</sup> As a result, in the US, "selenium deficiency is a major problem in all classes of livestock. Seventy percent of domestic corn and soybeans do not have adequate selenium to meet animal needs. Forty states are now classified as selenium deficient and 50 percent of the land is marginal or deficient."<sup>41</sup> Naturally, this process of selenium depletion is not limited to the US.

In summary, during the latter half of the 20<sup>th</sup> century, precipitation became increasingly acidic, soil pH fell, and heavy metal and fertilizer contamination increased. As a consequence, selenium bioavailability declined and levels of this element in the food chain fell.<sup>42</sup> This depressed human selenium intake

is likely to have been the most significant in those regions of the planet where soil levels were naturally very low, including much of sub-Saharan Africa and in the Chinese “Disease Belt,” a huge region of selenium deficiency that crosses China from northeast to southwest.<sup>43</sup> As discussed previously, this decline in selenium in the human food chain appears to have made it much easier for those viruses that encode selenium to spread. It has also encouraged an increase in the prevalence of chronic degenerative diseases that are associated with selenium deficiency. The former include Coxsackievirus (implicated in Keshan disease and acute myocardial infarction) Herpes virus 8 (Kaposi’s sarcoma), Hepatitis B and C (Hepatitis) and HIV (AIDS). Together, these viruses have already infected more than one third of the global human population and show no sign of halting their rapid diffusions. Similarly, cancer and cardiovascular diseases are now the major causes of death through the Developed World.

It must be admitted, however, that the widespread recycling of contaminated syringes, needles, IV solution bags, and other medical supplies in China and elsewhere in the Developing World is giving added impetus to the Hepatitis B and C and AIDS pandemics.<sup>44</sup> Such equipment is often collected from garbage dumps and reused. As a consequence, the World Health Organization has estimated that worldwide the multiple use of unsterile needles is now annually responsible for 8 to 16 million cases of Hepatitis B, 2.3 to 4.7 million cases of Hepatitis C, and an additional 80,000 to 160,000 HIV-1 infections.<sup>45-46</sup>

Interestingly, many of the current medical tests for pathogens do not work well on HIV-seropositive individuals. This is because such tests require an immune response to challenge and this is not possible when the immune system is badly damaged. As a result, it would seem that numerous cases of syphilis and tuberculosis go undiagnosed in such patients.<sup>47</sup>

In *Disaster planning: The Preservation of Life and Property*,<sup>48</sup> I wrote “Communities, like individuals, may often work toward their own destruction through neglect, ignorance, or a deliberate emphasis on fulfilling superficially advantageous short-term goals. Incrementally, in doing so, they magnify risk and eventually suffer the disasters they deserve.” Although I was thinking of the bubonic plague that struck London in 1665 and the Great Fire that consumed 80 percent of it in the following year, this generalization applies just as well to the many ongoing health pandemics that are consequences of our unwillingness to take environmentalism seriously.

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*A sounder approach to distinguishing normal from abnormal is to call abnormal those observations that are regularly associated with disease, disability, or death—that is, any clinically meaningful departure from good health. The disease may be expressed directly by symptoms, or predicted by characteristics that are themselves strongly associated with poor health, such as “risk factors” or clinically important physical signs.*

Robert H. Fletcher, Suzanne W. Fletcher,  
and Edward H. Wagner,  
*Clinical Epidemiology: The Essentials*,  
1988, Baltimore: Williams & Wilkins

## WHY NOT NOW?

*We have, I hope, abandoned the convenient but restricted and artificial two-foci correlations linking single factors in causality of disease processes. Hence we need no longer argue whether a disease is caused by heredity, constitution, disordered chemistry or physiology, infection, trauma, or repressed pathogenic emotions. We are no longer concerned with either-or-single-factor polarities but currently attempt to map out the widest possible ranges of conditions, all of which in some way and at some time, seem to be implicated in a dynamic chain of causes and effects.*

Roy Grinker<sup>1</sup>

If the “selenium-CD4 T cell tailspin” hypothesis<sup>2</sup> is correct, it follows that there must be some recently introduced widespread variable(s) (such as acid precipitation and associated heavy metal rains) that are reducing selenium levels in the food chain and/or depressing the efficiency of human immune systems. Either or both of these possibilities could initiate the “tailspin” and make infection by, and replication of, HIV-1 and HIV-2 more likely. Evidence has just been put forward to show that selenium bioavailability has been dropping because of the increasing acidity of precipitation.<sup>3</sup> It will now be demonstrated that several other factors have been simultaneously depressing the functioning of human immune systems.

During the 20<sup>th</sup> century, the domestic and industrial use of stable chlorine-containing compounds, such as chlorofluorocarbons, halon gases, and carbon tetrachloride, polluted the earth’s stratosphere. Such gases are dissociated by solar radiation in the upper atmosphere to produce atoms of chlorine

that can destroy ozone by way of a series of complex chemical reactions.<sup>4</sup> As this process continues, the earth's ozone layer is thinning and a large hole has been appearing in it above the Antarctic. A similar hole is developing in the ozone of the Arctic stratosphere.<sup>5</sup> Despite political initiatives such as the Montreal Protocol, which pledged a 50 percent reduction of the use of chlorofluorocarbons by 1999, and a subsequent amendment calling for a total ban on this gas, the earth's ozone continues to thin.<sup>6</sup> It appears that the resulting excess ultraviolet B radiation that can now reach the earth is depressing human immune system responses.<sup>7-8</sup> Patients with multiple non-melanoma skin cancers, for example, who have had previous heavy ultraviolet light exposure, show an abnormally low helper T-lymphocytes/suppressor T-lymphocytes ratio.<sup>9</sup> This unusual characteristic is associated with an absolute increase in the number of suppressor T-lymphocytes, suggesting that strong sun exposure can result in a permanent rise in suppressor T-lymphocytes in some individuals. This, of course, increases the probability that they will develop cancer, and makes them more susceptible to the "selenium-CD4 T cell tailspin."<sup>10</sup>

During the 1950s, there was a very rapid rise in the production of pesticides, such as DDT, which were claimed to be essential for agriculture. The Green Revolution that followed saw the global diffusion of commercial agriculture and with it pesticide use. Annual worldwide pesticide production is now approximately 1.8 to 2.3 billion kilograms. The World Health Organization has estimated that such chemicals cause 20,000 human deaths and 500,000 pesticide-related illnesses each year.<sup>11</sup> However, the true toll may be much higher than this because there is a large body of evidence that suggests chronic exposure to pesticides, and indeed other environmental chemicals, can lead to altered immune function and lowered host pathogen resistance.<sup>12</sup> Many of these chemicals are stable and environmentally persistent and may, like organochlorine

compounds, cross the placental barrier giving chronic exposure to both mother and fetus.<sup>13</sup> Aldicarb, for example, can pollute groundwater and cause a decreased CD4/CD8 cell ratio in those who drink such water.<sup>14</sup> Similarly, many of the thousands of people poisoned by methyl isocyanate during the Bhopal, India disaster developed numerous abnormal immune responses, including an increase in the number of T-helper and total T-cells and a decline in lymphocyte metagenesis.<sup>15</sup>

Animal experiments also suggest that exposure to TCDD (2,3,7,8 - tetrachlorodibenzo-p-dioxin) can cause a reduction in CD4 T cell count.<sup>16</sup> In addition, a Japanese study<sup>17</sup> of 36 breast-fed infants showed that such organochlorine compounds lower levels of CD4 T cells in the human neonatal immune system. Furthermore, B[a]P (benzo [a] pyrene) is known to induce pre-B lymphocyte apoptosis.<sup>18</sup> PCBs may also depress glutathione peroxidase activity in the plasma of animals.<sup>19</sup> It is clear, therefore, that a variety of widely used chemicals, many of them pesticides, are adversely affecting the integrity of human immune systems on a global scale. This often involves a depression of CD4 T cell counts.

Other political and social factors may also be encouraging immune system malfunction. In Europe, selenium food levels are in rapid decline, only partly due to acid precipitation. The major cause of this sharp drop has been changes in the origin of much of the consumed food. In general, European soils contain low levels of selenium.<sup>20</sup> However, European Economic Community trade barriers have forced the increased use of locally grown crops, preventing the importation of more seleniferous North American wheat. Thus, the average daily intake of selenium in the UK fell from 42.8 micrograms in 1991, through 38.5 micrograms in 1994, to 29 to 39 micrograms in 1995.<sup>21</sup> Similar unmonitored declines have almost certainly taken place over most of Europe for the same trade-related reasons.

In North America, Europe, and elsewhere in the Developed World, AIDS affected some groups disproportionately, suggesting that their members were more likely to have compromised immune systems prior to HIV infection.<sup>22</sup> Evidence is now presented to show that homosexuals, intravenous drug users, and hemophiliacs typically had either serum selenium and/or CD4 T cell counts which were abnormally depressed prior to HIV infection. It is argued that, as a consequence, this virus diffused more easily in these groups than in the general population, and that because of such abnormalities the subsequent HIV-seropositivity rapidly progressed to AIDS.

Shilts<sup>23</sup> graphically described the homosexual lifestyle in the United States during the 1980s and early 1980s in *And The Band Played On*. This lifestyle often involved very promiscuous sexual activity, accompanied by excessive alcohol and drug consumption. Drugs of choice were cocaine, amphetamines, nitrite inhalants, and heroin. In addition, homosexuality was associated with frequent infection by a wide variety of bacteria and viruses. To illustrate, a Denver study<sup>24</sup> discovered that, in the 1970s, an average bathhouse patron had 2.7 sexual contacts a night and so was exposed to a 33 percent chance of developing either syphilis or gonorrhea, because one in eight of those wandering the hallways had asymptomatic cases of the diseases. Many of those infected with HIV by Gaetan Dugas (Patient Zero)<sup>25</sup> met him for the first and only time in one of North America's gay bathhouses.

Excessive alcohol consumption and drug use also increased immunosuppression in many gays. It has been established, for example, that depressed selenium levels occur frequently in alcoholics.<sup>26-27</sup> In addition, human and animal studies have shown that cocaine interferes with the function of natural killer (NK) cells, T cells, neutrophils, and macrophages, altering the ability of such cells to secrete immunoregulatory cytokines.

Cocaine also enhances the infectivity and/or replication of HIV-1 of human cells in vitro.<sup>28</sup> Furthermore, rodent studies have proved that inhaled isobutyl nitrite inhibits both specific T cell mediated cytotoxicity and the tumoricidal activity of activated macrophages.<sup>29</sup> These immune system abnormalities suggest that exposure to abuser levels of nitrite inhalant by homosexuals, who often used this drug to enhance sexual pleasure, probably caused compromised tumour surveillance mechanisms.

Some prescription drugs, including acetaminophen,<sup>30-31</sup> depress glutathione production, so encouraging replication of HIV-1. This is because glutathione naturally inhibits reverse transcriptase which the virus needs to convert its own RNA into DNA and incorporate it into its host's cell genes.<sup>32</sup> It is also possible that smoking depresses serum selenium. Certainly, smokers develop more cancers. This seems to be partly because tobacco contains cadmium, a major selenium antagonist, which probably reduces the availability<sup>33</sup> of this essential trace element.

Intravenous drug users were naturally exposed to all the immunosuppressive effects of the drugs used by gays. In addition, because they often shared blood contaminated needles, a wide range of bacteria and viruses, including but not limited to HIV-1, were quickly spread amongst them.

Hemophiliacs were a third group who displayed depressed immune systems prior to infection with HIV.<sup>34</sup> Blood and blood products were badly screened for pathogens. As a result, all too often, hemophiliacs quickly became infected with the Hepatitis C virus<sup>35</sup> and the human T lymphotropic virus type III<sup>36-37</sup> as well as other viruses. Beyond this, the provision of HIV-1 contaminated blood and blood products in the 1970s and early 1980s ensured widespread infection by this virus in hemophiliacs. Regardless of its origin, several deficits in immune

response have been seen in hemophiliacs, even if they are not HIV-seropositive. These include a low CD4+/CD8+ ratio and a profound impairment of leukocyte inhibiting factor. These immune abnormalities are most apparent in hemophiliacs who have received factor 8 concentrates.<sup>38-40</sup>

To summarize, HIV-1 transmission and replication is inhibited in regions such as Senegal where soils and local food chains contain high levels of selenium. Conversely, in selenium depleted regions, such as those of most of the countries of sub-Saharan Africa, HIV-1 can easily replicate and infect host CD4 T cells.

HIV-1 encodes Se-dependent Gpx (glutathione peroxidase) modules, therefore its replication involves depleting its hosts of both glutathione and selenium. The former process naturally diminishes the availability of glutathione, an inhibitor of reverse transcriptase,<sup>41</sup> an enzyme which is essential for HIV-1 replication. This glutathione depletion, therefore, permits faster HIV-1 infection. Declining host selenium levels also simultaneously inhibit the production of CD4 T cells, which need this trace element. This immunosuppression permits further infection by opportunistic pathogens which lead to increased depression of serum selenium levels, magnifying the decline in CD4 T cells. The “selenium-CD4 T cell tailspin” is now operating in earnest and typically continues downwards until some virulent pathogen provides the coup de grâce. It is at this later stage that HIV-positive individuals are their most infectious and, therefore, are most likely to transmit the virus to others.<sup>42</sup> Outside the earth’s major selenium deficiency belts, HIV-1 has more difficulty diffusing. It can, however, spread rapidly in groups such as gays, intravenous drug users, and hemophiliacs whose lifestyles make them more likely to have either a serum selenium deficiency or a compromised immune system.

The author's "selenium-CD4 T cell tailspin"<sup>43</sup> hypothesis suggests that signatories to the Durban Declaration<sup>44</sup> and most of the AIDS mavericks are wrong.<sup>45</sup> There is no single cause for AIDS. For HIV-1 to cause AIDS all that is needed is a continuous depression of serum selenium. This in turn results in declining CD4 T cell counts, accompanied by a multitude of infectious pathogens, each of which is capable of depressing serum selenium still further and with it the CD4 T cell count. If any of these opportunistic pathogens simultaneously kills CD4 T cells, or impairs other aspects of the function of the immune system, it will accelerate the "tailspin" and further reduce life expectancy. This model suggests that HIV-1 does not have a single co-factor but rather a multitude of them.

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*Stop it at the start; it's late for medicine to be prepared when  
disease has grown strong through long delays.*

*Ovid, Remedii Amoris*

*Your body naturally produces glutathione in abundance—it is essential to life itself. Glutathione circulates constantly throughout your body, neutralizing free radicals and removing dangerous waste products and toxins from your system. When your glutathione level is high, your overall health is high. You feel good and you look good. You fight off minor illnesses quickly, you have plenty of energy, and you feel mentally and physically alert.*

*If your glutathione level is high, you're at an optimal level of good health, for now and for the future. Glutathione naturally protects you from the long-term dangers of free radicals, metabolic wastes, and environmental poisons. Since those dangers include cancer, heart disease, premature aging, autoimmune diseases, and chronic illnesses such as asthma, keeping your glutathione level high helps you stay healthy and active.*

Alan H. Pressman<sup>1</sup>

On November 20<sup>th</sup>, 1820 an enraged sperm whale struck the whaleship Essex and sank it. The captain and crew had to take to lifeboats in the central Pacific, 1,500 nautical miles west of the Galapagos Islands.<sup>2</sup> Imagine that you and a close friend had to abandon your yacht in mid Pacific, after it had been sunk by some similar mischance. Your lifeboat contains a minimum of food and water. There had been no time to broadcast an SOS before the yacht sank. You have no EPIRB (Emergency Position Indicating Radio Beacon). Your only hope for survival rests on chance winds and currents and the slight possibility that the lifeboat will drift close enough to a passing ship to be seen and for you to be rescued. My question is this. “Will you kill your friend, or will your friend kill you?” In all

probability you will, in fact, kill one another because simply by drinking half the water and consuming fifty percent of the food, each of you will greatly reduce the survival time of the other. This means that the probability of a chance encounter with a passing ship while either of you is still alive is greatly diminished. When there is strong competition for scarce resources, everybody's life expectancy tends to fall.

What relevance does this principle have here? It is fundamental since it is how HIV causes AIDS and why infection by it typically leads to an early demise. HIV competes with its host for certain essential nutrients.<sup>3</sup> Initially, most of these are available to the host, but as the "selenium-CD4 T cell tailspin"<sup>4</sup> picks up speed and HIV replication accelerates, more and more of these essential nutrients are utilized by the virus. Severe deficiencies develop in the host and are responsible for most of the symptoms of AIDS, including immune system collapse, muscle wasting, dermatitis, diarrhea, and dementia. Associated infectious co-factors are also responsible for a variety of their own unique symptoms. Eventually the host is so weakened that almost any pathogen can provide the coup de grâce. It does not really matter where HIV is in the body. As long as it is replicating, it will compete for these nutrients and weaken the host.

As Taylor and co-workers<sup>5</sup> have shown, HIV-1 encodes for glutathione peroxidase. This genetic characteristic ensures that as the virus replicates it competes with its host for the four basic components of this essential selenoenzyme:<sup>6</sup> specifically selenium, cysteine, glutamine, and tryptophan (Figure 2). It follows that, unless individuals who are HIV-positive add supplements of these nutrients to their diet, or at the very least eat specific foods that contain unusually high levels of them, this process will inevitably culminate in extreme shortages of these four substances; each of which will be accompanied by

its own deficiency symptoms. AIDS is the end product of this multiple depletion process. As a consequence, the disease can be most effectively treated not by drugs, but by reversing all four of these nutrient deficiency states. This depletion process is why nursing mothers in Kenya<sup>7</sup> died faster than HIV-positive mothers who gave formula to their infants. In nursing mothers, selenium, cysteine, glutamine, and tryptophan stores are shared not merely by the woman and the replicating virus but also by her child. Naturally, she becomes nutrient deficient more quickly. The “selenium-CD4 T cell tail-spin,” therefore, accelerates and death comes more rapidly. Such nutrient depletion also explains why HIV-positive individuals die more quickly in Southern Africa than in most other regions.<sup>8</sup> The soils of sub-Saharan Africa are generally very selenium deficient.<sup>9</sup> As a result, diets there are typically low in this nutrient. It is easier for HIV to replicate and to cause rapid depletion of this trace element and so speed up the downward spiral into AIDS. People living in poverty almost invariably eat poor quality diets and can, therefore, be infected by HIV more easily. Short of nutrients, they then progress to AIDS more quickly.

If this hypothesis is correct, HIV/AIDS patients will be found to be deficient in glutathione peroxidase, and its four basic components: specifically selenium, cysteine, glutathione, and tryptophan. Evidence to show that this is indeed the case is now provided. Selenium deficiency in HIV/AIDS patients has been described already at some length in earlier chapters. Rather than repeat the evidence discussed previously, only the main points will be reviewed briefly. Numerous studies have shown selenium deficiency in the plasma of individuals with HIV/AIDS.<sup>10-12</sup> The worse the AIDS symptoms become, the more depressed the plasma selenium levels. Indeed, Baum and co-workers<sup>13</sup> have demonstrated that in both HIV-1-seropositive drug-using men and women, depressed selenium

plasma levels are a more accurate predictor of mortality than falling CD4 T cell counts. This was also found to be true of HIV-infected children.<sup>14</sup> That adults and children dying of AIDS display both depressed CD4 T lymphocyte counts and very depleted plasma selenium stores is no coincidence. Rather, it seems much more likely that this dual depression provides evidence of a positive feedback system in which a fall in selenium causes a reduction in CD4 T cells, because this trace element is essential for the production of T lymphocytes. Such a drop in the efficiency of the immune system, caused by selenium induced inadequate lymphocyte production, then allows infection by other pathogens, resulting in a further decline in selenium. The “selenium -CD4 T cell tailspin” is beginning its downward spiral.

As previously described, a hypothyroid or low T3 (triiodothyronine) syndrome is well known in AIDS patients.<sup>16-17</sup> This occurs because selenium deficiency causes a reduction in deiodinase, the enzyme required to convert T4 (thyroxine) to T3. It has been further suggested that such a selenium-deficiency abnormality of the thyroid may be a significant factor in the AIDS wasting process.<sup>18</sup>

Cysteine is a water-soluble amino acid. Its structure is chemically simple: the amino acid group is combined with a sulphur-containing thiol group.<sup>19</sup> Cysteine helps to determine the form and mechanical properties of a variety of proteins. In addition, it plays a role in energy metabolism and it can be used as fuel if necessary. Cysteine is a very important component of the enzyme system that synthesizes fatty acids whenever they are required by body cells. Coenzyme A and fatty acid synthase use cysteine's thiol grouping to help transfer molecular segments. This very reactive thiol helps attach carbon atoms, two at a time, onto the lengthening chains that constitute each fatty acid.<sup>20</sup>

Cysteine has been shown to have many clinical uses.<sup>21</sup> It increases hair shaft diameter and hair growth density in some cases of hair loss and human baldness. It also plays a key role in wound and skin healing and has been successfully used to treat psoriasis. Cysteine also helps to protect lung alveoli against tobacco smoke. The toxic chemicals in cigarette smoke impair the ability of alveolis' macrophages to kill bacteria. Cysteine, however, improves their bactericidal effectiveness; often reducing smoker's cough. The cysteine derivative N-acetyl-cysteine in aerosol form can liquify mucus in the lungs and bronchial tubes and so is prescribed in chronic bronchitis, asthma, cystic fibrosis, emphysema, lung abscess, and chronic obstructive pulmonary disease.<sup>22</sup> N-acetyl-cysteine has also been shown to be a useful adjunct in bacterial infection and can alleviate both cobalt and molybdenum toxicity. In addition, there are dozens of reports of cysteine's value in diabetes and as an adjunct in treating cancer.<sup>23</sup>

It is clear, therefore, that chronic cysteine deficiency will cause a broad range of disease symptoms. Numerous clinical and laboratory studies have established beyond a doubt that HIV-infected patients do, indeed, display decreased plasma cysteine concentrations at all disease stages.<sup>24-25</sup> One of the chief results of this deficiency is an inability to produce adequate glutathione. This is because, while glutathione is a peptide consisting of three amino acids, it is usually the availability of the latter that controls its production. Since HIV-infected patients are deficient in cysteine, they are also typically short of glutathione.<sup>26</sup>

Such cysteine inadequacy and associated low blood glutathione levels have proven to be excellent indicators of the probability of AIDS-related death.<sup>27</sup> To illustrate, AIDS patients who have very low glutathione blood levels were found to have a 3-year survival probability of about 20 percent. In contrast, those

with more normal glutathione levels have a 60 to 80 percent survival rate over the same time period. This is not surprising because glutathione acts as a nucleophilic scavenger and as an antioxidant in the event of tissue injury. It has a key role, therefore, as a protector of biological structures and functions. Depletion, as in paracetamol intoxication, is extremely hazardous.<sup>28</sup> Beyond this, intracellular glutathione has a powerful impact on how well T- and B- lymphocyte cells function and its availability also affects the production of macrophages, monocytes, and neutrophils. It is clear, therefore, that any cysteine deficiency leading to a decline in glutathione, will further damage the immune system in HIV-1 seropositive patients. This may be why HIV patients with depressed glutathione levels develop more secondary infections and cancers and have an elevated mortality rate.<sup>29</sup> Indeed, there is evidence which suggests that glutathione supplementation can slow HIV-1 replication by inhibiting reverse transcriptase activity.<sup>30-31</sup> Several studies<sup>32-33</sup> have also demonstrated that cysteine supplementation (usually given as N-acetyl-cysteine) can replenish low glutathione in CD4 T cells and other immune system components, presumably improving the ability to resist secondary pathogens and cancers.

In 1981, Bunk and Combs<sup>34</sup> described an experiment demonstrating that, in chickens, selenium deficiency impaired the conversion of the S-amino acid methionine into cysteine. It is highly likely that this is true of humans. If it is, by encoding for the selenoenzyme glutathione peroxidase, HIV-1 causes a deficiency of cysteine in infected individuals in two distinct ways. Firstly, the virus removes cysteine directly from the body as it replicates. Secondly, it creates a selenium deficiency which impairs the conversion of methionine to cysteine, so reducing the availability of the latter. Simply put, HIV-1 both increases the demand for and reduces the supply of cysteine in patients who are HIV-1 positive.

Glutamine, a metabolite of glutamic acid,<sup>35</sup> plays a major role in DNA synthesis. Glutamine is also found in the cerebral cortex and is abundant in the substantia nigra, the thalamus, and other regions of the brain. This is not surprising since it is a prolific neurotransmitter.<sup>36</sup> Beyond this, it is known to be important in the glands controlled by the sympathetic nervous system such as the thymus, pancreas, and duodenum.

Braverman and Pfeiffer,<sup>37</sup> writing in 1987, reported that glutamine deficiencies were very rare because this nutrient can be synthesized in several different ways. However, inadequate glutamine is now recognized as a characteristic of AIDS.<sup>38-39</sup> Since it is a major nutrient for rapidly proliferating cells, glutamine is of particular significance in the digestive tract where it is essential for intestinal cell proliferation, intestinal fluid/electrolyte absorption, and mitogenic response to growth factors. Glutamine deficiency also produces apoptosis.<sup>40</sup> It is not surprising, therefore, that many glutamine deficient AIDS patients have abnormal intestine permeability associated with digestive malfunction,<sup>41</sup> and that Noyer and co-workers<sup>42</sup> have demonstrated that a daily glutamine supplementation of 8 grams can stabilize the intestinal permeability of patients with AIDS.

Muscle protein wasting is a symptom of HIV-infected individuals and is frequently an early indication of AIDS. Several studies<sup>43-45</sup> have shown a great benefit from glutamine supplementation. Indeed, Shabert and colleagues<sup>46</sup> were able to rehabilitate HIV-positive patients with such weight loss by glutamine-antioxidant supplementation during a 12 week trial. Clark and colleagues<sup>47</sup> also conducted a randomized, double-blind, placebo controlled study involving 68 HIV-infected patients which clearly showed that hydroxy beta-methylbutyrate, glutamine, and arginine, given as a mixture, could markedly reduce lean tissue loss in patients suffering from AIDS-associated wasting.

The fourth nutrient for which HIV competes with its host is the essential amino acid tryptophan. This is needed by the body for the biosynthesis of niacin, serotonin and certain proteins.<sup>48</sup> The relationship between tryptophan and niacin is especially interesting because niacin can be made from dietary tryptophan and, strictly speaking, niacin is not a vitamin, just a metabolite of the vitamin tryptophan.<sup>49</sup> There is no doubt that tryptophan is depressed in the serum and cerebrospinal fluid of patients with HIV-1 infection.<sup>50-51</sup> Werner and co-workers,<sup>52</sup> for example, showed that in the sera of 11 male patients with advanced HIV infection, tryptophan levels were less than 50 percent of those in gender and age matched controls. As would be expected, niacin and serotonin levels in the serum of HIV/AIDS patients are also very depressed.<sup>53-54</sup>

Tryptophan is the least abundant essential amino acid in foods,<sup>55</sup> a characteristic that, in the past, has led to major health problems. One of these was pellagra, which typically developed in children eating a diet that was very rich in corn. Maize is deficient in tryptophan, so that such children quickly developed pellagra, which is thought to be due to a codeficiency of both tryptophan and its metabolite niacin.<sup>56</sup> As a consequence of these two deficiencies, such individuals could not produce adequate nicotinamide adenine dinucleotide and so developed pellagra. The symptoms of this disease were known as the four Ds, namely dermatitis, diarrhea, dementia, and ultimately, if not treated effectively, death.<sup>57</sup> AIDS patients commonly experience such symptoms and also display inadequate levels of nicotinamide adenine dinucleotide. This can be reversed, at least in vitro, by the administration of nicotinamide.<sup>58</sup> Fuch and colleagues<sup>59</sup> have shown further that there is a close association between depressed serum tryptophan and polyneuropathy and dementia in HIV-1 seropositive patients. This relationship suggests that many of the neurologic/psychiatric symptoms seen in AIDS may be caused by the same nutrient

deficiencies that cause pellagra, namely of tryptophan and niacin.

Since tryptophan is also a necessary precursor of serotonin, the latter is always depressed when the former is deficient. As might be expected, whole blood serotonin levels are abnormally low in AIDS patients,<sup>60</sup> especially so in those with neuropsychiatric symptoms. Indeed, there is an inverse relationship between serotonin levels in whole blood and AIDS severity.<sup>61</sup> This is not surprising since high doses of serotonin have been shown to inhibit the multiplication of HIV-1 in T4 lymphocytic cell lines.<sup>62</sup> As Fawkes<sup>63</sup> has explained, tryptophan plays a unique role in defence against infection because it is normally relatively scarce. When infection takes place, the body induces tryptophan-catabolizing enzymes in an attempt to starve invading pathogens by increasing tryptophan's scarcity. However, in unresolved infections, like HIV-1, such tryptophan deficiency seems to be linked to neuroendocrine dysregulation, cognitive deficits and immune incompetence. It is not clear when, or if, tryptophan supplementation would be of benefit to HIV/AIDS patients. One of the beneficial effects of zidovudine therapy in patients with HIV infection, however, is the gradual normalization of tryptophan in their serum and cerebrospinal fluid,<sup>64</sup> implying that tryptophan supplementation is likely to be of benefit to HIV/AIDS patients. It has been demonstrated also that the tryptophan derivative niacin (together with vitamins B1 and C) significantly depresses the progression of HIV-1 infection to AIDS,<sup>65</sup> while serotonin may be able to inhibit HIV-1 in T4 lymphocytic cell lines.<sup>66</sup> This suggests that not only tryptophan but its two metabolites niacin and serotonin should also be of value in the treatment of AIDS.

AIDS then is an accidental death. The genetic code of HIV includes a homologue for the essential selenoenzyme glutathione peroxidase. To replicate, HIV must compete with its host

for the four nutrients that make up this enzyme, namely: selenium, cysteine, glutamine and tryptophan. As a consequence, the HIV-seropositive individual tends to gradually become more and more deficient in these four essential nutrients, their metabolites and glutathione peroxidase itself. AIDS is the end product of these declines and the majority of its symptoms are caused by such deficiencies (Table 1). A lack of adequate selenium and cysteine, for example, undermines the immune system in a process that is accelerated by other infectious pathogens. A deficiency of glutamine encourages muscle wasting and digestive malfunction, while a lack of tryptophan and the compounds it biosynthesizes (such as niacin and serotonin) results in dermatitis, diarrhea, and various neurologic and psychiatric symptoms including dementia. Glutathione peroxidase is one of the most significant antioxidants in the body. A deficiency of this enzyme is associated with extensive free radical damage and oxidative stress. As Pressman<sup>67</sup> states in the quotation that begins this chapter, “When your glutathione level is high, your overall health is high.” Conversely, when it is in a constant downward spiral, so too is your life expectancy.

**Table 1** AIDS: The Nutrient Deficiency Disease

<i>Nutrient</i>	<i>Deficiency Symptoms (Examples)</i>
<b>Selenium</b>	Depressed glutathione peroxidase Oxidative stress Depressed CD4 T lymphocytes Depressed triiodothyronine (hypothyroidism) Cancers of lung, colon, etc. Myocardial infarction Kaposi's sarcoma (with HHV-8) Depression
<b>Cysteine</b>	Depressed glutathione Poor wound and skin healing Psoriasis Abnormal immune function Secondary infections and cancers
<b>Glutamine</b>	Depression Abornmal intestine permeability Diarrhea Muscle wasting
<b>Tryptophan</b>	Depressed niacin and serotonin levels Immune incompetence Neuroendocrine disregulation Polyneuropathy Dementia Dermatitis Diarrhea

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*And now was acknowledged the presence of the Red Death. He had come like a thief in the night. And one by one dropped the revellers in the blood-bedewed halls of their revel, and died each in the despairing posture of his fall. And the life of the ebony clock went out with that of the last of the gay. And the flames of the tripods expired. And Darkness and Decay and the Red Death held illimitable dominion over all.*

Edgar Allan Poe<sup>1</sup>

*The Masque of the Red Death*

Edgar Allan Poe published *The Masque of the Red Death* in 1842. The gay he referred to were not homosexuals but individuals whose reaction to impending disaster was to “party on.”<sup>2</sup> This is not an uncommon response to threat. Nero was Emperor and for 2 weeks there had been serious rioting in Rome.<sup>3</sup> The economy of the world’s greatest empire was disintegrating since the cost of maintaining the gigantic armed forces, fully equipped with fast war galleys, catapults, and ballistae, was bleeding the coffers dry. The impoverished government had neither funds nor power to stop the rioting. In this crisis, a key decision had to be made. The merchant fleet was in Egypt awaiting loading. Its holds could carry either corn for the starving masses or special sand needed on the track for the chariot races at the Circus Maximus. Three hundred pairs of gladiators were soon to fight to the death, while 1200 condemned criminals were to be eaten by lions. Twenty beautiful girls were to be raped by jackasses. It was no contest. The sand was loaded. Those games would keep the minds of the masses off their troubles. On a smaller scale, Shilts<sup>4</sup> described the same phenomenon

“partying on” in the United States during the early stages of its AIDS pandemic.

Medicine is dominated by a biomedical view of the body as a machine and disease as an internal malfunction.<sup>5</sup> Breakdown can generally be traced to a substandard component because machines usually operate in a linear function, so that D causes E which results in F. The body machine in this biomedical model is viewed as a system of separate but interacting parts. The physician, that is the body mechanic, is expected to identify which component is causing the disorder or disease and advise on how to repair or replace it.

Health has become the end product of applied science in which answers to every human illness can be discovered ultimately by laboratory research. Thomas<sup>6</sup> best expressed belief in this biomedical paradigm when he claimed: “For every disease there is a single key mechanism that dominates all others. If one can find it and then think one’s way around it, one can control the disorder.” In truth, the situation is so simple, whether the ultimate agent of mortality is an earthquake, automobile, or virus.<sup>7</sup> To illustrate, if asked the question “Why did Marie Antoinette die?” one could reasonably reply, “She had her head cut off by a guillotine.” However, a more comprehensive list of causes of her early demise would have to include every event that led to the invention and development of this instrument of execution, all of the factors that provoked the French Revolution and the Reign of Terror associated with it, each step in the evolution of humanity that gave our species the brains and bodies capable of designing and constructing the guillotine, and so on *ad infinitum*. In truth, as pointed out by Bohm,<sup>8</sup> while we usually choose to ignore the vast cascade of variables that stand behind any event, it is still important to accept that no single cause and effect relationship can really be separated from the universe as a whole. Like the strands that make up a

rope, events converge and combine to create an evermore binding set of circumstances, that, in total, produce some eventual consequence, whether a pandemic or an avalanche.

Growing acceptance of this interconnectedness of reality has had important repercussions in disaster planning.<sup>9</sup> As this discipline matured, emphasis tended to move away from the development of optimum methods of trying to deal with the destruction, deaths, and injuries caused by hazards towards greater emphasis on preventing disaster. Ultimately, this trend will have to lead to the design of more resilient systems, capable of withstanding stress with grace.<sup>10</sup>

We are losing the “War on AIDS.” By the year 2015, if we continue along our current path, stress caused by this pandemic will be the equivalent of eight times that of World War I and far greater than that of World War II. A new approach is obviously essential; one which recognizes the holistic nature of the problem and, therefore, of necessity our answers to it. If, for example, we ask the question “Why did John Doe die of AIDS?”, one could reasonably answer that “He became infected with HIV-1.” A more realistic list of the causes of his fate, however, would have to include the hedonistic lifestyle he led with its relentless promiscuity and associated constant infection by selenium-depressing, sexually transmitted pathogens. In addition, John Doe lowered his resistance to disease by drinking alcohol and taking drugs. His diet was poor, eating foods too low in selenium and other nutrients. However, the inadequacy of his diet was due, at least in part, to the Green Revolution and its overuse of fertilizers and to the impact of acid rain and heavy metal pollutants. But didn’t John Doe die because of a medical profession that failed to accept the obvious: that HIV alone does not cause AIDS? This outdated paradigm has led to misdirected efforts to block the ravages of HIV by the use of drugs, which have in fact produced more

virulent strains, not just of HIV, but also of its accompanying pathogens. Then again, John Doe was killed by political complacency that allowed the use of contaminated blood supplies,<sup>11</sup> ignored environmental decay, and underfunded early attempts to block the spread of HIV. The list of reasons that John Doe died of AIDS is almost endless. In truth, he died because we live in a virtual reality world, and so did he, until reality came crashing through his illusions.<sup>13</sup>

In 1665, bubonic plague struck London, killing 500 of the city's inhabitants a week at its peak.<sup>14</sup> Since no one knew what was causing this disease, a wide variety of strategies was applied in vain efforts to halt its spread. These included bonfires, ordered by the Lord Mayor of London to purify the air of infection. Cats and dogs were thought to be implicated in the spread of the plague, so they were exterminated in large numbers, increasing the rat population which, through their fleas, stimulated the plague's diffusion. Such desperate times clearly demanded desperate remedies. The College of Physicians claimed that the best way to treat the bubonic plague was to apply hot onion, fig, and treacle poultices to buboes.<sup>15</sup> Less prestigious authorities, like the author of *The Plague's Approved Physitian*, advocated the use of a live, partially plucked pigeon to draw off the poisons from these swollen lymph nodes.<sup>16</sup> Abandoning such science, the more superstitious wore charms, often engraved with the word *Abracadabra*, arranged in a triangle. Many carried herbs in their pockets. Of course, none of these remedies worked. By the time the outbreak was over, approximately 100,000 Londoners had died from the plague. Typically, during such medieval pandemics, the bubonic plague killed between 30 and 75 percent of its victims, pneumonic plague 95 percent, while septicaemic plague was almost always fatal.<sup>17</sup> Treatments of the day had little, if any, positive effects. Some, like the extermination of cats and dogs, were even counterproductive.

History, of course, does not repeat itself. It is perhaps not too cynical to point out, however, that if the preventive strategies, based on the belief that HIV alone causes AIDS, were really working there would not have been 3 million deaths from it in 2000.<sup>18</sup> If sub-Saharan Africa and many other regions of the planet are not to become largely depopulated in the next 20 years, more effective strategies are needed to protect those who are not yet HIV-seropositive. Three of these: sexual abstinence, vaccination, and increasing the selenium content of foods, will now be discussed and evaluated.

## **SEXUAL ABSTINENCE**

The Kenyan president, Daniel arap Moi, was embarrassed by the number of condoms his east African country imported and by their cost.<sup>19</sup> As a result, he urged Kenyans to refrain from sex “even for only 2 years.” According to newspaper reports, the president’s case for abstinence did not reach a receptive audience. Felix Githingi, a computer technician, is quoted as saying, “How can I do that? Am I not a Kenyan man? There are three important things for a Kenyan man: to drink beer, to eat meat of the goat, and to have sex.”<sup>20</sup> However, President Moi’s espousal of sexual abstinence did gain support from the Roman Catholic Church and the Islamic community, both of which still oppose the use of condoms. In the meantime, some 700 Kenyans are dying each day from AIDS.<sup>21</sup>

Soon after, Swaziland’s King Mswati III introduced a 5 year ban on sexual intercourse for unmarried women.<sup>22</sup> They are also prohibited from wearing pants and shaking hands with men. “Virgins” have been instructed to signal their support for these new rules by sporting “chastity tassels” of blue and yellow wool, while women in relationships are to don pompoms of red and black. The tassels, called *umchwasho*, hang at the back of the

head and are designed to ward off potential sexual partners. These royal rules are to be policed by traditional chiefs. Girls who fail to wear such tassels will be fined a cow. Young men who touch a chaste girl will be ostracized and also fined.

The reaction to enforced chastity in Swaziland has been similar to that seen in Kenya. In Mbabane, the country's capital, a woman in tassels cannot be found. "That is totally old-fashioned" laughed Emilia Nkosi,<sup>23</sup> a 15 year old who lives in the capital's suburbs. "It won't stop anyone from having sex. We won't bother. People would laugh!" Dozens of other "virgins" have expressed similar views in local newspapers. Education officials in Swaziland also blamed girls in miniskirts for the spread of AIDS and so introduced a new school knee-length skirt rule.

On the other hand, AIDS workers claim that polygamy<sup>24</sup> is one of the major reasons for Swaziland's high incidence of HIV. King Mswati's reaction to such suggestions was to announce his engagement to a 17-year old girl, days after his new sexual intercourse ban for unmarried women was introduced. She is to be his ninth wife.<sup>25</sup> As of March 19, 2001 Swaziland's HIV prevalence rate was 34.2 percent, slightly more than one third of the total population.<sup>26</sup> In those aged between 20 and 24, the rate was higher, estimated at 42.5 percent. And the band played on.

Drug resistant strains of HIV are showing up in newly diagnosed seropositive individuals.<sup>27</sup> This establishes beyond doubt that some AIDS patients, who are receiving various antiviral drugs, are still engaging in unsafe sexual activities. This being the case, the probability that millions of sub-Saharan Africans, many of whom show no obvious signs of infection with HIV, will willingly abstain from sexual activity for years lies somewhere between zero and nil. There is little point in assessing the

impact of this strategy further because it is less likely to be successful than wearing Abracadabra charms was during the 17<sup>th</sup> century bubonic plague pandemic.

## VACCINATION

Funding for research into HIV vaccine strategies has increased 6-fold since 1990, to an estimated \$356 million for fiscal year 2002.<sup>28</sup> As a result, work on the development of new potential vaccines for HIV has rapidly expanded in the last few years. Several recent successful animal challenges with live viruses, after SIV and HIV-specific vaccinations, have increased the belief that a protective vaccine for HIV may soon be on the horizon.<sup>29-30</sup> As Dr. A. Fauci<sup>31</sup> pointed out at the *AIDS Vaccine 2001 Conference*, “Historically, vaccines have provided safe, cost-effective and efficient means of preventing the illness, disability, and death from infectious diseases. The development of a safe and effective vaccine for HIV infection is an essential goal of AIDS research and a necessary tool to bring the HIV epidemic under control.” Nevertheless, because of differences between humans and other primates and between SIV and HIV, extrapolation for such early animal studies should be viewed with caution.

It is not my purpose to discuss, in any detail, the progress made in the search for potential HIV vaccines. Within the last 2 years, many vaccine candidates have been developed and are now in various stages of preclinical and early clinical evaluation. Some 25 of these were discussed at the *AIDS Vaccine 2001 Conference*,<sup>32</sup> but so far only one, the VaxGen rgp 120 vaccine is being tested in phase 3 clinical trials.<sup>33-34</sup> These studies are currently going ahead in Canada, the United States, the Netherlands, and Thailand. They are a consequence of earlier research that showed the production of neutralizing

antibody responses to HIV gp 120 and involve giving the vaccine to volunteers. Another prime/boost vaccine strategy test will soon begin in Thailand.

While, as Fauci<sup>36</sup> pointed out, a safe and effective vaccine against HIV would be an enormous breakthrough in the “War Against AIDS,” there are several reasons to question whether this goal realistically can be achieved. One such doubter is Dr. Veljko Veljkovic<sup>37</sup> of the Institute of Nuclear Sciences in Belgrade, who claims that current vaccine methods are unsafe and may produce a mutated version of HIV, that could go undetected by available test methods. Veljkovic argues that: “despite the urgent need for preventive AIDS vaccines, it would be wise to introduce a moratorium on clinical trials until there is a serious reexamination of the current concepts for their development. Premature testing, without complete knowledge of the biological and immunological properties of HIV, could produce irreparable and irreversible long term consequences.” In this case such vaccines could become the source of potentially new infectious diseases rather than an effective instrument for AIDS prevention. Anyone who believes that the rush to create a new vaccine, and the corners that are cut in the process, cannot produce new hazards, is urged to read Edward Hooper’s<sup>38</sup> *The River*. In this volume, the author provides considerable evidence, from more than 600 interviews and analyses of 4,000 scientific texts, to support his hypothesis that the AIDS pandemic was the result of the use of chimpanzee kidneys to produce early oral polio vaccines. While Hooper<sup>39</sup> may be wrong, there is no doubt that millions of humans now carry simian virus as the result of their previous vaccinations.<sup>40</sup>

Certainly there are several reasons to be extremely cautious about HIV vaccines. As described previously, this virus has the ability to produce a virtually endless source of new strains.<sup>41</sup> Antiviral drug use, for example, while giving temporary benefit

to the recipient, is driving the creation of more virulent and increasingly dangerous new types of HIV. This constant viral change increases the difficulty of developing a safe and effective vaccine. In addition, HIV-1 has the ability to enter “resting” T cells.<sup>42</sup> These cells are particularly good places to hide since they remain inactive and, therefore, are not noticed by the host’s immune system. Since such “resting” T cells can exist for years, if not decades, it is possible that a vaccine may appear to have given protection against HIV when it has, in fact, not done so. The virus is simply dormant.

One critic who pointed this out was Albert Sabin,<sup>43</sup> developer of the oral polio vaccine. In 1992, he claimed that “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.” The successful vaccines that were developed for polio and measles are effective because these diseases are transmitted only by viruses that are free and not attached to cells. HIV, as just pointed out, hides inside cells, embedding its genetic material in the DNA of its host. It is clear that any HIV vaccine will have to encourage the immune system to target and in some way kill all cells that are under attack by the virus.

Even if a vaccine that appears to be absolutely safe and effective is developed, numerous social issues need to be addressed before it can be used on a large scale. It will be extremely difficult, for example, to ensure that it is only given to individuals who are not already HIV-positive. In sub-Saharan Africa, where a vaccine is most urgently needed, an estimated 25.3 million people are now infected with HIV.<sup>44</sup> For the region as a whole, the average national prevalence of HIV infection amongst individuals in the 15 to 49 year age group is 8.8 percent. In seven countries in southern and eastern Africa, about 20 percent of this age group is HIV-seropositive, while in Botswana,<sup>45</sup> the

nation with the highest prevalence, 36 percent of the adult population has been infected. In South Africa, AIDS deaths are now thought to number 4,800 each week.<sup>46</sup> This means that if the vaccine is given to as many individuals in sub-Saharan Africa as possible, millions who receive it will already be HIV-positive and will probably die from AIDS, regardless of the vaccine's efficiency. However, in all probability the vaccine, not the sexual behaviours of those dying, will then be blamed for this disease. Even an effective vaccine, therefore, is likely to come under enormous criticism for its inability to save those already infected and its use may be seen as a deliberate attempt to exterminate certain societies.

An adjunct of this problem is that many people receiving such a vaccine will probably believe it gives them immunity from AIDS, even if they are, in fact, already HIV-positive. Such a belief is likely to reduce their willingness to use condoms, and to practice safe sex, encouraging the spread of HIV to others who have not been vaccinated.

In truth, reliance on a newly developed vaccine to halt the AIDS pandemic will be the greatest gamble ever undertaken by our species. Billions of individuals will need to be vaccinated quickly using a technique that has been developed in a great hurry. Yet the strategy is lacking in resilience as it is irreversible.<sup>47</sup> There is no way an individual can be unvaccinated. If errors are made, they will be permanent and global in scale. It is a "crap shoot" for the survival of the species.

## **INCREASING THE SELENIUM CONTENT OF DIET**

The identification of the most desirable strategy for reducing risk is best achieved by learning from the experience of others. Few hazards are unique and valuable insights can be gained

by reviewing hazard-related literature, and contacts with those involved elsewhere in mitigating similar risk. Simply put, the best ways to decide on the optimum strategy to handle risk is to see what has worked or failed elsewhere.<sup>48</sup> This is just as true for HIV as for any other hazard.

It has been argued at length, earlier in this book, that Senegal has the world's lowest cancer incidence and an extremely depressed HIV prevalence rate. This is not because of particularly effective disease prevention strategies, but rather is the result of a geological accident that ensures a highly alkaline, selenium-enriched environment. As a consequence, selenium easily moves into the Senegalese food chain. Diseases caused by members of the deficiency branch of the selenium disease family tree, such as AIDS and many cancers, are extremely rare in Senegal. Would adding selenium and calcium to fertilizers give protection against HIV in other countries? Can a virus that encodes glutathione peroxidase be prevented from infecting a population by increasing selenium in the local food chain? The answer to these questions can be found in China.

As has been pointed out previously, Keshan disease is an endemic cardiomyopathy,<sup>49</sup> a human heart disease that is limited to specific types of environment. It occurs in 309 counties of 15 provinces and autonomous regions of China, that together form a fairly broad belt which crosses the country from northeast to southwest.<sup>50</sup> Although environments vary within this belt, they consist mainly of those within temperate and warm temperate forest and forest-steppe soil types. Keshan disease has been recognized also in similar areas in the north of Korea. What unites such regions is their very low soil selenium levels. Rural peasants, eating locally grown foods, are at the greatest risk of developing Keshan disease, with children under 10 years of age and women of childbearing age being the most susceptible.<sup>51</sup> The Chinese average annual incidence of acute and

sub-acute Keshan disease, for the period 1959 to 1982, was 9.3 per million.<sup>52</sup> This figure is deceptive, however, since the disease is limited to the selenium-deficiency belt of China and occurs nowhere else in the country.

Despite its known association with inadequate dietary selenium, Keshan disease incidence fluctuates annually. It is particularly high in some years and low in others. Whether the annual incidence rate will be high or low appears linked to meteorological factors, such as temperature, precipitation, humidity, early or late frost, and sunshine hours. This is not surprising as it has recently been discovered that Keshan disease is caused by Coxsackievirus B3.<sup>54</sup> As Taylor and his colleagues<sup>55-57</sup> have pointed out, this virus, like HIV-1, encodes glutathione peroxidase and therefore depletes its host of selenium as it replicates. However, within China's "Disease Belt" selenium is already a rare commodity. Soils typically contain less than 0.13 ppm<sup>58</sup> and, as a result, the average daily dietary intake of this trace element is generally below 10 micrograms. In the most selenium deficient areas, intakes can be less than half this figure.<sup>59</sup> For comparison, estimated daily intake in Canada and the US is some 170 micrograms.<sup>60</sup>

It stands to reason then that because source dietary selenium intake in the Chinese Keshan disease regions is already amongst the lowest on earth, a virus capable of depleting the trace element further soon creates an extreme deficiency. In its most acute form this viral-trace element deficiency combination leads to serious arrhythmia associated with acute cardiac failure.<sup>61</sup>

Fortunately, Keshan disease incidence and mortality rates have been declining recently in China.<sup>62-63</sup> This is because concerted efforts have been made to keep the Coxsackievirus in check by increasing dietary selenium levels. A variety of strategies have been used to achieve this goal.<sup>64-65</sup> They have included

selenium supplements, increasing the consumption of grain grown outside the deficiency belt, spraying selenium-enriched fertilizers onto soils and crops and adding this trace element to the feed of domestic livestock and to table salt. As the level of selenium in local diets has risen, the incidence and mortality from Keshan disease has fallen.<sup>66</sup>

To illustrate, numerous field studies have been conducted in Sichuan Province in an attempt to clearly establish the effects of selenium supplementation on Keshan disease. Since 1974, 620,000 people in 24 counties have been provided with sodium selenite, either in tablet form or as fortified table salt. One such study<sup>67</sup> compared the effect of such tablets on the incidence of Keshan disease in children 10 or younger. The incidence rate for this disease was established at 7.1 per thousand for a group of 24,225 children during the period 1972 to 1973. All children in these tested communes were then provided with daily sodium selenite tablets. By 1983, a total of 88,373 had received such supplements and their Keshan disease incidence rate had fallen to 0.12 per thousand during the time period. In contrast, in neighbouring communes used as untreated controls, Keshan disease incidence amongst such children was 8-10 in the period 1972-1973 and had averaged 9.39 during the field test. The difference between the children in treated and untreated areas was statistically significant ( $p < 0.1$ ). A further field trial using sodium-selenite fortified salt showed a decrease in the average annual incidence rate from 1974 to 1976 from 3.19 to 0.195 per thousand, compared with a drop of 1.11 to 0.86 per thousand in an untreated control population ( $p < 0.1$ ). There can be no doubt then that adding selenium to diet greatly reduces the frequency with which Keshan disease is reported in China.

Whether the daily intake of selenium in the Chinese “Disease Belt” is as yet sufficient to protect simultaneously against both

the Coxsackievirus and HIV-1 still remains to be seen. If not, once HIV-1 becomes firmly established in this region, it is to be expected that very elevated mortality from AIDS will rapidly occur, on a scale at least comparable to that of sub-Saharan Africa. Nevertheless, the Chinese clearly have demonstrated that it is possible to reverse mortality from a disease caused by a virus that encodes glutathione peroxidase by increasing dietary selenium intake. This principle is of extreme importance when considering strategies to combat the spread of HIV-1 and associated AIDS.

Interestingly, the Chinese have shown that this principle probably applies to all such viruses. Hepatitis B viral infections tend to be most common in China where local grains contain depressed selenium. As with other viruses that encode glutathione peroxidase, such as HIV-1 and the Coxsackievirus, a deficiency of selenium appears to encourage infection. Chinese research, however, has demonstrated that selenium supplementation can greatly reduce the incidence of Hepatitis B. In 1989, for example, Yu and colleagues<sup>68</sup> published details of a 3 year research project conducted in Qidong County, Jiangsu Province. The 20,847 inhabitants of one township were provided with table salt fortified with 15 ppm anhydrous sodium selenite. In contrast, residents in six surrounding townships were encouraged to use normal table salt. Prior to and during the first year of the study, there was no statistically significant difference in hepatitis infection between the selenium supplementation and control populations. However, by the third year, a drop in the incidence of hepatitis had occurred in the selenium supplied township (4.52 per 1,000) compared with those communities using normal salt (10.48 per 1,000; 56.8% reduction,  $p < 0.002$ ). A similar study in the same county, also conducted by Yu and colleagues<sup>69</sup> further established that daily selenium-yeast (200 micrograms of selenium) supplementation could significantly reduce the primary liver cancer often

associated with Hepatitis B and C infection. Interestingly, Berkson<sup>70</sup> has demonstrated that the liver damage caused by Hepatitis C can be reversed by a combination of alpha lipoic acid, silymarin, and selenium, often negating the need for expensive liver transplantation.

It is apparent, therefore, that the Coxsackievirus is not the only glutathione peroxidase encoding virus that can be greatly inhibited by selenium supplementation. Indeed, the available evidence suggests that it is probably a characteristic that is common to all such viruses, including HIV-1 and HIV-2.

Acid rain is increasing mortality from a wide range of diseases, some of which are associated with selenium deficiency. Others such as Alzheimer's disease reflect the greater solubility of aluminum caused by lowering soil water pH.<sup>71-72</sup> Included in the former group are those caused by viruses such as Hepatitis B and C, Coxsackievirus, and HIV-1 and HIV-2 that encode selenoproteins but, paradoxically, appear to replicate best when their host is selenium deficient.<sup>73</sup> As a consequence, as acid rain reduces the bioavailability of selenium it increases the virulence of such viruses. It is too much to expect that this link will be accepted by those who benefit most from the pollution of the atmosphere. As an interim measure, however, it is possible to quickly increase selenium levels in diet by, as the Chinese have been doing in severely deficient regions, adding it to salt, animal fodder, and fertilizers.<sup>74-75</sup> Such programs are also already underway in New Zealand<sup>76</sup> and Finland<sup>77</sup> for other health reasons. This strategy, if employed on a global scale, seems the most likely to halt or at least slow the diffusion of HIV and of other pathogens such as Hepatitis B and C. It has many advantages. It can be applied immediately and its benefits will be virtually instantaneous. It is relatively cheap and easy to administer. Its beneficial effects will continue to be felt as long as trace element supplementation continues. The

strategy is also compatible with, and indeed will improve the outcome of any large scale vaccination program against HIV. While excess selenium can have an adverse effect on the environment, in areas where it is inadequate in soils and vegetation its addition to fertilizers is likely to improve health in local fauna. The biggest objection against selenium supplementation will come from those who insist on keeping the Recommended Daily Allowances for this trace element at clearly inadequate levels. Several trials in China<sup>78</sup> and the US<sup>79-80</sup> have shown that increasing daily selenium intake greatly reduces cancer incidence and mortality. In a major US<sup>81</sup> study, those obtaining these benefits were given 200 micrograms daily for several years. No adverse effects were seen and cancer mortality dropped markedly in the large group receiving the supplements. The toxicity of selenium in humans seems to have been greatly exaggerated. In northern Greenland,<sup>82</sup> for example, aboriginals have been ingesting as much as 1,000 micrograms daily for extended periods of time without any sign of ill effects. Yang<sup>83</sup> has described an outbreak of selenium intoxication that occurred during the period 1961 to 1964 in Enshi County, Hubei Province, Southern China. Exceptionally high selenium levels were measured in the hair, blood, urine, and, of course, food of the inhabitants. The original source of the element was a stony coal, that contained as much as 8.2 percent selenium. The actual outbreak of intoxication was triggered by the decision to use lime and plant ash as fertilizers, increasing alkalinity of the soil and with it selenium solubility. A drought reduced available food supplies, forcing villagers to rely on seleniferous vegetables and maize, containing up to 44 ppm selenium. As a result, villagers consumed about 38 milligrams of selenium every day, often for protracted periods of time. While the long term consumption of elevated levels of selenium killed local livestock, its human impacts were less dramatic. Those affected lost hair and nails, suffered skin lesions and abnormalities to the nervous system, together with disturbances of

the digestive tract and possibly tooth decay. There is, however, no evidence that such enormous selenium intakes, far in excess of those suggested here, have reduced the life spans of the elderly. Indeed, when affected individuals left the area, their nails and hair grew back, although in some cases, their thumbnails remained thickened 20 years later. The evidence from Greenland and from China demonstrates that it is relatively difficult to kill humans with excess selenium. Certainly, classic selenium toxicity from massive overdosing is very easy to diagnose—garlic odour of breath and sweat, metallic taste in the mouth, brittle nails and hair. It is also easy to reverse by lowering intake.<sup>84</sup> Indeed, massive doses of selenium for short periods of time appear to have great therapeutic potential in the treatment of haemorrhagic fever, caused by Hantaan virus infection.<sup>85</sup> In one Chinese outbreak involving 80 patients, oral sodium selenite was given at 2 milligrams a day for 9 days to a treatment group. Mortality in these patients was only 7 percent, compared with the untreated control group which had a 38 percent death rate. This clearly demonstrates that in cases of certain viral infection, selenium supplementation at higher than normally acceptable doses can be very beneficial. Since selenium deficiency is a key co-factor in the diffusion of HIV, supplementation at every level from the global to the personal seems an excellent approach to reducing the scale of the AIDS pandemic. As will be shown in the following chapter, seeking to increase selenium intake to slow the AIDS pandemic will have one advantage over all other prevention strategies. It will be of great benefit to those who already are HIV-seropositive.

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*A hot virus from the rain forest lives within a twenty-four hour plane flight from every city on earth. All the earth's cities are connected by a web of airline routes. The web is a network. Once a virus hits the net, it can shoot anywhere in a day — Paris, Tokyo, New York, Los Angeles, wherever planes fly.*

Richard Preston,  
*The Hot Zone*,  
1994, New York: Random House

*All men dream, but not equally. Those who dream by night in the dusty recesses of their minds wake in the day to find that it was vanity; but the dreamers of the day are dangerous men, for they may act their dreams with open eyes, to make it possible.*

T.E. Lawrence<sup>1</sup>  
*The Seven Pillars of Wisdom*

Testing positive for HIV is not a death sentence, one without hope of clemency or appeal. Rather, it is an opportunity to change and even to increase one's life expectancy. As has been demonstrated in this volume, AIDS is the end product of a positive feedback system in which HIV-1 competes with its host for the four basic components of the enzyme glutathione peroxidase, which this virus encodes and so must obtain if it is to replicate. In the early stages of HIV infection these four nutrients—selenium, cysteine, glutamine, and tryptophan—are normally still adequately available to the host. However, as their levels fall, HIV-1 replicates more rapidly, depressing them further, until severe deficiencies of each start to occur. These host deficiencies are responsible for most of the symptoms of AIDS, such as immune system collapse, muscle wasting, dermatitis, diarrhea, dementia, and acute myocardial infarction. The remaining AIDS symptoms are caused by accompanying symbiotic pathogens or degenerative diseases, all of which may act as selenium-depressing co-factors for HIV-1.

## STAMPING OUT TERMITES

Earlier in this volume, the author described seven anomalies that could not be adequately explained by the ruling paradigm that HIV alone causes AIDS. All these anomalies can be easily accounted for by the 'selenium-CD4 T cell tailspin' hypothesis and its associated nutrient deficiencies. To illustrate, breast-feeding leads to early maternal death because it increases selenium, cysteine, glutamine, and tryptophan deficiencies in the nursing mother, so speeding the "tailspin" and encouraging the development of AIDS. The AIDS pandemic is largely sparing Senegal because of a geological accident which created an environment that is both alkaline and selenium-enriched and so provides protection for its inhabitants against viruses which, although they encode glutathione peroxidase, cannot infect individuals who are not deficient in selenium. Selenium deficiency is a better indicator of mortality in AIDS patients than CD4 T lymphocyte counts because it is the lack of this essential trace element that causes much of the depression of such lymphocytes. The more deficient the patient is in selenium, the faster HIV-1 will replicate and the sooner the individual will die. Kaposi's sarcoma frequently occurs in AIDS patients because it is caused by the human herpes virus 8 (HHV-8) which only replicates in individuals who are extremely selenium deficient. All AIDS patients have depressed serum selenium levels and so additionally are prime targets for HHV-8. The AIDS, Hepatitis B and C, and cancer pandemics are occurring now because acid rain is reducing selenium bioavailability and is lowering its levels in the food chain. Pesticides, drugs, alcohol, and tobacco use are also damaging human immune systems. In Montagnier's experiment, tetracycline stopped HIV destroying CD4 T lymphocytes because this antibiotic killed the bacteria and/or mycoplasma that were acting as selenium-depleting co-factors for the virus. There is no reason for the spouses of hemophiliacs to be selenium deficient.

As a consequence, most of them could not be infected by HIV-1, despite long periods of unprotected sexual activity with seropositive wives or husbands.

## UNCONVENTIONAL TREATMENTS

It is not yet known how to kill HIV-1 in the body. This, however, does not mean that there can be no fully effective treatment for this virus. There are many viruses that can never be eradicated after infection. Typically, they initially cause a disease, then an uneasy equilibrium is reached in which the infected individual's immune system, perhaps with the help of medication, gains the upper hand and prevents further outbreaks. The virus is controlled, but never totally eradicated. Unfortunately, the reverse is true of HIV. Its initial symptoms are mild but its impact increases over time, eventually culminating in AIDS. However, it is clear from other viruses, such as herpes simplex virus, cytomegalavirus, and varicella zoster virus, that even if eradication is impossible, successful treatment can stop or reduce viral replication and prevent the development of serious disease states. In the case of HIV-1, such a treatment should involve keeping co-factors under control and preventing the development of selenium, cysteine, glutamine, and tryptophan deficiencies.

In answer to the obvious question "Will it work?" I can provide only the following information. I am a geographer, without funding from any AIDS-related organization. I have no patients on which to test a nutritional treatment for AIDS because I am not a medical doctor. I work alone; any original ideas in this book are mine and so are the errors. I have no funding, laboratory, research team, or formal support network to lobby for my ideas or to work towards testing them. Although I have written or edited some 190 books and articles, mostly

on disaster planning or on the causes of chronic degenerative diseases, I chose to pay for the publication of this book myself. There was only one reason for this. It was the only way I could retain copyright and so put the entire book on the Internet for free downloading by those who need the information it contains and any hope it provides. As a consequence of all these limitations, I cannot say that I know **for sure** that the treatment I am about to describe works for AIDS. However, there is no reason I am aware of that it cannot be added to the regimen of patients receiving conventional treatment. Indeed, while writing this book I have been approached on behalf of two such individuals whose conventional treatments were failing and who were too well to die but too sick to really live. Since I am not a physician I could not prescribe, but I carefully described what I would do if I were HIV-seropositive and dying of AIDS. Thankfully, I am not. Both of these patients are now in good health and one is back at work. Two swallows do not make a summer, nor two more healthy former AIDS patients a cure, but they are encouraging. What follows is what I would do if I needed to treat myself for HIV-1 or AIDS. It is not a suggestion that any reader do the same. That is their decision alone. It should be pointed out, however, that such treatment aims to normalize patient levels of selenium, cysteine, glutamine, and tryptophan. How much of each of these nutrients needs to be added to diet through eating specific foods, or taking supplements, will depend on their current body levels, which in turn will reflect previous diet and the current replication rate of HIV-1. There is, therefore, no ideal dosage of each nutrient for every patient. This will vary from individual to individual and over time in the same patient as their condition improves. One final note on the topic of proof: I would be happy to cooperate with any organization with the facilities to test this nutritional approach to reversing AIDS and/or halting the decline of HIV-seropositive individuals into this disease state.

## EXPERIMENTAL SUPPORT

There is considerable experimental evidence suggesting that this approach to treatment may work. Meister and colleagues<sup>2</sup> have shown, for example, that two forms of glutathione could both greatly reduce HIV replication rates. In addition, several small studies have demonstrated that selenium supplements are beneficial in the treatment of HIV-seropositive individuals. Cirelli and co-workers,<sup>3</sup> for example, evaluated the serum selenium concentrations in 67 patients with HIV infection in four different stages. Such selenium levels were found to be normal in HIV antibody positive symptom-free individuals, but depressed in the other three groups. For two months, daily doses of 80 micrograms of sodium selenite and 25 mg of vitamin E were given. Symptomatic improvement occurred, along with elevation of serum selenium levels. Similarly, Schrauzer and Sacher<sup>4</sup> report that 19 symptomatic HIV-seropositive men were given 400 micrograms of selenium daily for 70 days. Of these individuals, 74 percent reported an improvement in appetite and gastrointestinal function as well as beneficial psychological and neurological changes. Reading also described the benefits of selenium supplementation in AIDS.<sup>5</sup> Beyond this, Look and co-workers<sup>6</sup> described giving a combination of 600 mg N-acetyl-cysteine, three times a day, together with 500 micrograms of selenium daily, for 24 weeks, to 12 HIV-infected patients in Stages I and II. There was a trend towards an increase in CD4 lymphocytes after 6 weeks, a rise in the CD4/CD8 ratio after 6 and 12 weeks, and a decrease in the absolute CD8/CD38 count and percentage of lymphocytes after 6 and 12 weeks, as compared with a similar untreated control group. Shabert and Winslow<sup>7</sup> described a study in which 11 male and 1 female AIDS patients were given 40 grams daily, in divided doses, of an L-glutamine-antioxidant supplement. This contained, in addition to glutamine, 800 mg/day of Vitamin C, 500 IU/day of vitamin E, 27,000 IU/day of betacarotene, 280

micrograms/day of selenium and 2,400 mg/day of N-acetylcysteine. It was found that over 3 months the L-glutamine-antioxidant group gained 2.2 kilograms in body weight, while the control group only added 0.3 kilograms. This gain represented a 3.2 percent increase in weight for the treated group. In addition, it has been shown that nicotinamide, the amine form of niacin, the tryptophan derivative, inhibits both acute and chronic HIV-1 infection in cell culture.<sup>8</sup> There is considerable evidence, therefore, to show that nutritional supplements can result in significant improvements in HIV/AIDS patients.

## **TREATMENT BY DIETARY CONTROL**

### **Optimum Nutrient Levels**

The aim of a dietary treatment for AIDS is to reverse the four nutrient deficiencies invariably linked with the condition (Table 1). In addition, the depressed levels of associated derivations of these substances should be addressed. What then are the ideal levels of selenium, cysteine, glutamine, and tryptophan in the human body? A study by Shamberger and Willis<sup>9</sup> discovered that healthy US individuals between 50 and 71 years old averaged 21.7 micrograms of selenium per 100 millilitres of blood. This compared with cancer patients of the same age range who were found to have only 16.2 micrograms per 100 millilitres. Furthermore, in a subsequent paper, the same authors<sup>10</sup> described a comparison of selenium levels in the blood of people from 19 US cities. These were highest in Rapid City, South Dakota and lowest in Lima, Ohio; 25.6 and 15.7 micrograms per 100 millilitres respectively. Interestingly, Shamberger and Willis<sup>11</sup> found a clear inverse relationship between cancer death rates in these cities and blood selenium levels. The higher the selenium, the lower the cancer mortality. To illustrate, in Rapid City, where blood selenium levels were highest, the annual cancer death rate per 100,000 people, during the period

1962 to 1966, was 94.0. In contrast, in Lima, Ohio the cancer mortality rate for this time period was exactly double this at 188.0. This strongly suggests that it is far better to have a blood selenium level of 25.6 micrograms per 100 millilitres than one of 15.7. Such US regional differences in blood selenium levels were almost certainly a reflection of variations in the local soil content of this mineral.

Braverman and Pfeiffer<sup>12</sup> have published data on various blood amino acid levels in both healthy adult males and females. For cysteine such levels range from 6 to 14 micromoles per 100 millilitres for men and 5 to 13 for women. Blood levels for glutamine given by these authors vary from 45 to 105 micromoles per 100 millilitres for adult males and from 40 to 90 for adult females. Similarly, the figures quoted for tryptophan in blood for both men and women vary from 4 to 25 micromoles per 100 millilitres. It would seem logical to attempt to achieve the higher ends of these ranges.

### **Ideal Foods**

The Brazil nut tree (*Bertholletia excelsa*) is found growing in many Amazonian states of Brazil, Peru, Colombia, Ecuador, and Venezuela.<sup>13</sup> It can be enormous, often reaching a height of 160 feet or more. This tree carries its fruit in a largely spherical woody pod that averages 6 inches in diameter and may weigh up to 5 pounds (Plates 1 and 2). These pods fall from the tree between January and June. Inside each one are some 12 to 25 Brazil nuts, in their own shells. A single tree can produce 300 or more pods, that is about 3,600 to 7,500 nuts. These three-sided nuts are composed of 70 percent fat or oil and 17 percent protein. For centuries, the indigenous tribes of the Amazon rainforest have used Brazil nuts as a significant staple of their diet and have even accepted them as a trade commodity, rather like money. Indigenous people eat the nuts raw, or grate them to be mixed with gruels.

So why is this biogeography lesson of significance here? To quote from *Herbal Secrets of the Rainforest*:

*Brazil Nut Oil is a clear yellowish oil which has a pleasant and sweet smell and taste. In addition to protein and fat, Brazil Nuts are a substantial source of selenium, an important antioxidant. One single Brazil nut exceeds the US Recommended Daily Allowance of selenium. The proteins found in Brazil nuts are very high in sulphur-containing amino acids like cysteine (8%) and methionine (18%) and are also extremely rich in glutamine, glutamic acid, and arginine.<sup>14</sup>*

Brazil nuts, therefore, contain large quantities of three of the four basic nutrients that HIV-1 depletes in its hosts, and so are the ideal food for anyone infected with the virus.

Since the soils of the Amazon Basin show local variations in selenium content, so too do the Brazil nuts grown there. It is probably best, therefore, to use defatted Brazil nut flour. Since Brazil nuts from numerous locations are combined and mixed in the manufacture of this product, the flour tends to have a relatively constant selenium content, 200 micrograms per 2.3 grams.<sup>15</sup> Such selenium is very bioavailable.<sup>16</sup> There are other advantages associated with using this product. One hundred grams of it also contain 1.02 grams of cystine, 9.20 grams of glutamic acid and 0.76 grams of tryptophan.<sup>17</sup> Brazil nut flour has a pleasant taste when eaten alone but can also be added to a variety of other dishes without substantially altering their overall flavour or texture.<sup>18</sup> In summary, ideally Brazil nut flour should be added to the diet of anyone who is HIV-1 positive and not allergic to such nuts. Unfortunately, the Brazil nut trees in the Amazon Basin could never meet such a huge demand.

Cysteine is also common in certain other foods beyond Brazil nuts. These include yoghurt, wheat germ, duck, turkey, and pork. Similarly, glutamine can be found at elevated levels in

wild game, luncheon and sausage meats, ham, bacon, wheat germ, and cottage and ricotta cheeses.<sup>19</sup>

Tryptophan, the fourth essential nutrient that HIV-1 removes from its host, is critical for the formation of structural proteins, enzymes and the neurotransmitters melatonin and serotonin.<sup>20</sup> It is not as widely available in diet as many other nutrients and plasma amino-acid profiles of hundreds of patients have demonstrated that it is often the amino acid that is most lacking in the blood of Americans. Such a deficiency, since it reduces the body's ability to produce serotonin, affects mood. L-tryptophan given to 98 volunteers, for example, made them more agreeable and less quarrelsome.<sup>21</sup> Need I say more! Ham and beef extract contain large amounts of tryptophan, as do eggs, almonds, salted anchovies, Parmesan and Swiss cheeses, and nutritional yeast.<sup>22</sup>

Rather than go into great length about the selenium, cysteine, glutamine, and tryptophan content of specific foods, this information is shown in Table 2 which follows. It is provided as a guide to individuals who wish to raise their dietary intake of these nutrients. Far more details are available in Appendices 1-5. The data in these appendices was derived from Nutricircles, a computer program designed to analyse the nutrient content of some 3,000 foods.

**Table 2** Examples of Foods Usually Elevated in at Least One of the Nutrients, Selenium, Cysteine, Glutamine, and Tryptophan

	<i>Selenium</i> (depends on soil where produced)	<i>Cysteine</i>	<i>Glutamine</i>	<i>Tryptophan</i>
NUTS	Brazil nuts	Brazil nuts	Brazil nuts	Almonds
VEGETABLE	Garlic Mushrooms	Garlic Onions Broccoli Brussel sprouts		
MEATS	Liver Round steak	Pork Duck Chicken Turkey	Ham Bacon Luncheon/ sausage meat Wild game	Ham Beef extract Pork Luncheon meat Chicken Turkey
SEAFOOD	Lobster Shrimp Cod Crab Herring Oysters Tuna			Anchovies
GRAINS	Barley Whole Wheat	Wheat germ	Granola Wheat germ Rolled oats	Granola Wheat germ Rolled oats
OTHER FOODSTUFFS	Soybean meal Brewer's yeast Egg noodles	Yoghurt Egg yolks	Cottage cheese Ricotta	Eggs Cottage cheese Unripened cheese (Swiss/Parmesan) Brewer's yeast Pineapple Yoghurt Bananas

Amazingly, and I cannot quite believe I am writing this, one of the best food choices is the cheeseburger which is high in cysteine, glutamine, and tryptophan. Indeed, if one were to make a cheeseburger that had 2.3 grams of Brazil nut flour added to its bun, one would have the perfect food for preventing the downward spiral of HIV infection into AIDS (Figure 2).

It has been shown by numerous animal studies that a high polyunsaturated fat intake combined with selenium deficiency greatly increases carcinogenesis.<sup>23-24</sup> In addition, polyunsaturated fats have been shown to inhibit the lymphoblastic transformation of certain T lymphocytes in AIDS patients.<sup>25</sup> Since many of the polyunsaturated fats found in margarine are not essential fatty acids, it would seem logical for HIV-seropositive individuals to eat butter.<sup>26</sup> It is also of interest that although the essential fatty acids (linoleic and linolenic acids) can be immunosuppressive, this does not seem to be the case if they are eaten in balance, with an n-3 to n-6 ratio of 1:2.1.<sup>27</sup>

## **TREATMENT BY SUPPLEMENTS**

### **Selenium**

There has been considerable debate over which form of selenium should be taken as a supplement. While minerals are single elements, they are rarely eaten in elemental forms. Typically, we eat an organic form or 'salt' of the mineral. This is important because every mineral form varies in digestibility, absorption rate and toxicity. As Passwater<sup>28</sup> points out, because selenium is so essential to human life, any form of selenium that can be assimilated is better than none, as long as it is not taken in toxic amounts. However, some mineral forms are much less toxic and better utilized than others. Natural organically-bound selenium in brewer's yeast is considerably more effective in raising blood concentrations of this trace

element than is sodium selenite, and has been taken by the author at a dose of 200 micrograms per day for almost 20 years without ill effects.

How much selenium should be taken daily by HIV-positive individuals is subject to discussion. Perhaps the most logical comments have come from Taylor:<sup>29</sup>

*. . . research has shown that there are problems in nutrient absorption even in asymptomatic HIV+ individuals, the suggestion has been made that HIV patients need to take larger amounts of vitamins than uninfected individuals to attain the same blood levels. Since the USDA states that nutritional supplementation in the range of 50-200 mcg of Se daily is safe and effective for healthy individuals, **a dose of 400 mcg seems reasonable for HIV-infected individuals**, if they do have impaired absorption. For an AIDS patient who is demonstrably deficient in Se, an even higher daily dose (up to 800 mcg) for a brief period of time (say several weeks) to get their blood levels up, followed by a decrease to 400 mcg is an effective strategy that was used in one published clinical study involving AIDS patients. This question of dose level naturally arouses concerns, because in the past so much has been made of the potential toxicity of Se. I believe that the danger of serious toxicity with Se supplementation has been exaggerated. The threat of serious acute toxicity with supplementation is in my opinion nonexistent at doses less than 1000 mcg per day in some individuals. Thus, doses in the 400 mcg range are undoubtedly safe. In any case, the signs of chronic Se toxicity—garlic odour of breath and sweat, metallic taste in mouth, brittle hair and fingernails—are very distinctive, and easily reversed by lowering the dose.*

I have nothing further to add. Dr. Taylor seems to be correct as usual.

## Cysteine

Cysteine supplements have been used for many years by orthomolecular physicians to treat diseases as diverse as stroke, manic depression, asthma, and schizophrenic psychosis.<sup>30</sup> Cysteine, however, is a poorly absorbed amino acid and has to be given in fairly large doses. To quote Braverman and Pfeiffer:<sup>31</sup>

*When we determine that cysteine supplementation is necessary, we usually begin with a dose of 500 mg/day. (Starting with a larger amount can lead to indigestion). Gradually, we may increase the dose to 3 or 4 g per day. Meanwhile, we keep an eye on serum cystine values. We find that, as cystine levels return to normal, low plasma levels of zinc, folic acid and taurine also return to normal. Some researchers have used as much as 7 g per day of cysteine.*

*It should be noted that extremely high doses of cysteine, probably greater than 7 g daily, can be harmful. Patients with cystinuria, an hereditary disorder characterized by excretion of large amounts of cystine and other amino acids in the urine, are at increased risk of forming cystine gallstones. We would suggest a limit of 500 mg of cysteine twice per day except under medical supervision. Vitamin C may prevent cysteine toxicity.*

Indeed cysteine should always be taken with high dose vitamin C and vitamins B1, B6, and E which improve its efficacy.<sup>32-33</sup> Other recommended supplements are magnesium and zinc, deficiencies of which are detrimental to glutathione metabolism. In magnesium deficiency, for example, one of the enzymes that is required in glutathione synthesis, gamma glutamyl transpeptidase, is lowered. Zinc and magnesium supplements, therefore, may enhance glutathione synthesis under specific conditions.<sup>34</sup> However, it is known that diabetics should avoid cysteine supplementation because it can block the effects of insulin by altering its chemical structure.<sup>35</sup> That is, cysteine breaks some S-S cross-link bonds, changing insulin's molecular shape.

Some orthomolecular physicians prefer to prescribe N-acetylcysteine for the treatment of AIDS.<sup>36</sup> This is because AIDS patients usually have digestive absorption problems. It is easier for them to take N-acetyl-cysteine than either cysteine or glutathione. Physicians treating AIDS patients generally recommend fairly high daily doses of N-acetyl-cysteine, in the range of 1,800 to 2,400 milligrams, taken at regular intervals, in three or four divided doses.<sup>37</sup>

## **Glutamine**

Glutamic acid is normally manufactured by the body, which also converts it into the amino acids glutamine and gamma-aminobutyric acid. Glutamic acid and its two metabolites glutamine and gamma-aminobutyric acid are necessary for energy production and brain function. They have been found to have significant therapeutic value in the treatment of hypertension, schizophrenia, ulcers, diarrhea, chorea, dyskinesia, epilepsy, Parkinson's disease, and alcoholism.<sup>39-40</sup>

Glutamic acid is normally referred to as a nonessential amino acid. This may be confusing because it does not mean it is unnecessary for health. Rather it implies that it need not be taken directly, because it can usually be synthesized by the body from a variety of substances, including ornithine, arginine and proline.<sup>41</sup> Glutamine supplements, normally in the form of 500 milligram tablets, are readily available in health food stores.

AIDS patients are known to be very deficient in glutamine. In a Harvard study<sup>42</sup> of HIV-seropositive individuals who were largely asymptomatic, glutamine serum levels were found to be very depressed, even though they showed no sign of AIDS. Despite the fact that a subgroup was given 20 grams (20,000 milligrams, that is 5 teaspoons) of glutamine daily in small doses over 24 hours for one month, glutamine blood levels remained

depressed. Patients<sup>43</sup> were then given 40 grams of glutamine per day, an amount usually reserved for bone-marrow transplant patients fighting off infection.

Pressman and Buff<sup>44</sup> probably provided the best advice on the therapeutic use of glutamine as a supplement when they wrote:

*Glutamine may also help treat serious diarrhea caused by AIDS or by other intestinal problems such as ulcerative colitis. Fairly large doses of glutamine, as high as 40,000 milligrams, may be needed. The glutamine improves the absorption of water through the colon, which helps relieve the diarrhea. Dosages that high should be taken under supervision, and only by those with diarrhea caused by a serious medical problem. Don't treat minor diarrhea from indigestion or a 24-hour stomach virus with glutamine.*

As Shabert and Ehrlich<sup>45</sup> point out:

*There are very specific instances in which giving glutamine to a sick individual would not be indicated. Individuals who have severe cirrhosis of the liver, Reye's syndrome, or another metabolic disorder that can lead to an accumulation of ammonia in the blood are at an increased risk for encephalopathy or coma. The basic problem is an inability to clear the body of excess nitrogen, which is converted to ammonia and ultimately causes brain swelling and brain-cell death. When the liver is severely damaged or when hepatic coma is imminent, glutamine is not effective and would cause only further damage to the brain.*

## **Tryptophan**

Tryptophan is the least abundant essential amino acid in foods. Deficiencies of it are known to be linked with a wide range of health problems including Hartnup's disease, pellagra, depression, hypertension, anorexia, insomnia, and overly aggressive

behaviour.<sup>46</sup> Tryptophan supplements of up to 3 grams daily have been used also to control intractable pain.<sup>47</sup> This amino acid is used by the body for the biosynthesis of niacin, serotonin, and various proteins. As a result, deficiencies of tryptophan, seen in individuals who are HIV-seropositive, seem to result in a variety of symptoms including those associated with pellagra; namely dermatitis, diarrhea, and dementia.<sup>48-49</sup>

L-tryptophan is the most desirable supplement form since all other metabolites of tryptophan, with the exception of niacin, have significant side effects. Braverman and Pfeiffer<sup>50</sup> explain that:

*Infusions of tryptophan can raise serum tryptophan six to ten times in normal persons without apparent side effects. Oral loading (4 grams) to normal controls can increase plasma levels up to four times normal within two hours. Twelve grams daily to manic patients can maintain plasma levels at three times normal.*

They further describe giving seven patients 2 grams of tryptophan daily for 6 weeks. Their plasma tryptophan levels were by then nearly double those of a control group of 96 patients.

Unlike selenium, cysteine, and glutamine, tryptophan is not readily available in health food stores. In the fall of 1989, the FDA recalled all L-tryptophan, stating it caused the rare and deadly condition Eosinophilia-Myalgia Syndrome (EMS).<sup>51</sup> On March 22, 1990 the FDA completely banned the public sale of L-tryptophan.<sup>52</sup> This is interesting considering that on February 9, 1993 a US government patent (#518157) was issued to use L-tryptophan to treat Eosinophilia-Myalgia Syndrome.<sup>53</sup>

The truth appears to be that one faulty batch of tryptophan probably caused the death of 37 people, and permanently disabled 1,500 more.<sup>54</sup> It is clear, however, that this was due not to the amino acid itself but to a contaminant in it, produced as

a result of the use of genetically engineered bacteria in its production.<sup>55-56</sup> Banning the sale of tryptophan, because of the world's first genetic engineering disaster, was like banning the sale of whisky because of deaths due to a bad batch of moonshine. However, it is still possible to buy 5-Hydroxy Tryptophan (5-HTP), derived from the seeds of *Griffonia simplicifolia*, a medicinal plant traditionally used in Ghana, Cameroon, and Côte d'Ivoire.<sup>57</sup> This supplement should not be taken by anyone using SSRI (serotonin reuptake) or MAO (monoamine oxidase) inhibitor prescription medications.<sup>58</sup> 5-Hydroxy Tryptophan is formed by the addition of a hydroxyl group (OH) to tryptophan, by the enzyme tryptophan hydroxylase and is the intermediate in the natural synthesis of tryptophan to serotonin.<sup>59-60</sup> In addition, another tryptophan metabolite, niacin, is available in any health food store. It should also be recalled that there are 400 milligrams of tryptophan in a cup of wheat germ, while low fat cottage cheese contains 300 milligrams per cup. There are also some 600 milligrams of tryptophan in a pound of turkey or chicken.<sup>61</sup> In most countries, if not available in health food stores, tryptophan can be prescribed by a physician. Indeed, it is strongly suggested that anyone attempting to reverse selenium, cysteine, glutamine, and tryptophan deficiencies caused by HIV infection should do so under the supervision of a doctor who is well trained in nutrition.

## **IN CONCLUSION**

If the glutathione peroxidase-encoding Hepatitis B virus can infect one third of the planet's population, it seems likely that so too can HIV. Since both viruses lower their host's serum selenium levels, the increasing prevalence of either one makes subsequent infection by the other more likely. The scientific establishment has spent some 20 years and over \$40 billion trying to establish that HIV is the sole cause of AIDS. As a

consequence of this misconception, not only has nobody been cured of HIV infection, but this virus and its bacterial co-factors are now more virulent than they were when the pandemic began. It is definitely time for a change of direction.

Either one can seek to accommodate change by developing greater resilience, or one can fight against it. In the war against change, two alternative methods of resistance are commonplace: fanaticism and inertia.<sup>62</sup> The fanatic, certain of a monopoly of truth, displays a perverse unwillingness to accept reality. An enormous desire to move in a particular direction, often in the face of all logic, may in the short term insulate him or her from the consequences of change, or even momentarily alter its dominant direction. This is where the AIDS scientific establishment is now. The greater the consequence of its failure to halt the pandemic, the more money it demands to continue its research and the more viciously it attacks those who point out that the emperor has no clothes.

Inertia, so commonplace in institutions, is a much more passive form of resistance to change. In a futile effort to preserve the *status quo*, countless obstacles are placed in the path of those who seek to deal with the new reality. Tomorrow is faced by seeking to repeat yesterday. "The Book," no matter how obsolete, must still be followed and tradition, however irrational in the face of radically new threats, has to be upheld. Such inertia gripped the US gay community in the early 1980s and is still apparent in the condemnation by the Roman Catholic Church of the use of condoms. It is also very evident in our unwillingness to switch from an energy economy based on coal, oil, and natural gas to one driven by renewables, such a hydro-electricity, solar, and wind. Unfortunately, the sad reality is becoming more and more obvious. There is no room for either fanaticism or inertia in the face of the AIDS pandemic. The sand is running too rapidly through the hourglass.

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## Appendices

The five tables that follow were created using *NutriCircles for Windows*, Version 4.21. This software is produced by Drs. E.H. Strickland and Donald R. Davis, Strickland Computer Consulting. It was used to search 3,000 foods for levels of selenium, cystine (the oxidized form of cysteine), glutamate (the salt form of glutamic acid), and tryptophan. The foods that are highest in each of these nutrients are displayed in the five tables which illustrate the number of times the Recommended Dietary Allowance is provided by 100 grams of each particular food.

**SELENIUM**

<i>Food</i>	<i>High Selenium RDA</i>
BRAZIL NUT, dry -----	50.20
MIXED NUTS, with peanuts, oil roast,salt -----	7.14
MIXED NUT, no peanuts, oil roast, salted -----	7.14
KIDNEY, pork, braised -----	5.28
KIDNEY, beef, simmered -----	4.76
TURKEY, giblets, simmered -----	3.76
KIDNEY, lamb, braised -----	3.71
YEAST, brewers, LewisLab -----	3.39
OYSTER, Pacific, steamed -----	2.61
COD, Atlantic, dried, salted -----	2.51
YEAST, Vegetarian Support, Red Star -----	2.37
YEAST, Red Star NBC600 -----	2.37
YEAST, KAL flakes -----	2.37
CHICKEN, skin, roasted -----	2.32
MUSHROOM, shiitake, dried -----	2.30
MUSTARD SEED, yellow -----	2.26
TURKEY, skin, roasted -----	2.11
PUFFED WHEAT, enriched -----	2.09
PUFFED WHEAT, Quaker -----	2.09
SWEET PUFFS,Quaker -----	2.08
LAMB LIVER, pan-fried -----	1.97
LAMB LIVER, braised -----	1.89
TURKEY, liver, simmered -----	1.71
CHICKEN LIVER, simmered -----	1.70
KIDNEY, veal, braised -----	1.69
THYMUS, veal, braised -----	1.66
CHICKEN, gizzard, simmer -----	1.60
TURKEY, gizzard, simmered -----	1.58
SPLEEN, beef, braised -----	1.55
OCTOPUS, boiled/steamed -----	1.52
CUTTLEFISH, cooked -----	1.52
MUSSEL, blue, boiled/steamed -----	1.52

WHELK, cooked -----	1.52
SEMOLINA, enriched -----	1.52
WHEAT, durum, grain -----	1.52
SPLEEN, veal, braised -----	1.37
TUNA, light, canned in water, drained -----	1.36
TUNA, light, canned in water, drain, salt -----	1.36
SUNFLOWER SEED KERNELS, dry roasted -----	1.34
SUNFLOWER SEED KERNELS, dry roast, salt ----	1.34
WHEAT GERM, raw -----	1.34
SUNFLOWER SEED KERNELS, oil roasted -----	1.33
SUNFLOWER SEED KERNELS, oil roast, salt ----	1.33
WHEAT BRAN, raw -----	1.32
OYSTER, eastern, farmed, cooked -----	1.31
OYSTER, Pacific, raw -----	1.31
TUNA, light, canned in oil, drained, salt -----	1.29
TUNA, light, canned in oil, drained -----	1.29
MATZO, whole wheat -----	1.27
SPAGHETTI, whole wheat, dry -----	1.24
PASTA, whole wheat, dry -----	1.24
PORK, pancreas, braised -----	1.23
OYSTER, eastern, wild, baked/broiled -----	1.22
OYSTER, eastern, wild, steamed -----	1.21
FLOUR, whole wheat -----	1.20
WHEAT, hard, red spring -----	1.20
WHEAT, hard, red winter -----	1.20
ANCHOVY, canned, drained -----	1.15
PORK LIVER, braised -----	1.14
OYSTER, eastern, breaded, fried -----	1.13
OYSTER, breaded, fried, fast food -----	1.12
TUNA, white, canned in water, drained -----	1.11
TUNA, white, canned in water, drain, salt -----	1.11
CAVIAR, black or red -----	1.11
WHEAT GERM, toasted -----	1.10
LAMB, pancreas, braised -----	1.10
CLAM, steamed -----	1.08

OYSTER, eastern, wild, raw-----	1.08
SPAGHETTI, spinach, dry -----	1.08
PASTA, spinach, dry -----	1.08
SUNFLOWER SEED KERNELS, toasted, salted ---	1.06
PASTA, enriched, dry -----	1.05
SPAGHETTI, enriched, dry -----	1.05
MACARONI, dry, enriched -----	1.05
SWORDFISH, baked/broiled -----	1.04
CALF LIVER, broiled -----	1.04
TUNA, white, canned in oil, drained, salt -----	1.02
TUNA, white, canned in oil, drained -----	1.02
SUNFLOWER SEED KERNELS, dried -----	1.01
PASTA, egg noodle, enriched, dry -----	1.00

**CYSTINE**

<i>Food</i>	<i>High Cystine RDA</i>
GLUTEN, commercial -----	1.15
COTTONSEED FLOUR, 1.4%fat, raw -----	0.87
COTTONSEED MEAL, 4.8%fat, raw -----	0.86
SOY, protein isolate, Supro, Protein Tech -----	0.77
SOYiprotein isolate, ProPlus, ProteinTec -----	0.73
COTTONSEED FLOUR, 4.2%fat, raw -----	0.72
SOY, protein isolate, potassium type -----	0.70
SOY, protein isolate -----	0.70
SESAME FLOUR, 1.8% fat -----	0.68
SUNFLOWER SEED KERNELS, flour(1.6%fat)-----	0.63
SOY,protein concentrate, by alcohol-----	0.59
SOY, protein concentrate, by acid -----	0.59
SOY, protein concentrate, acid wash -----	0.59
COTTONSEED, kernel, roasted-----	0.57
FLAX SEED, raw-----	0.56
FLAX SEED, milled, raw -----	0.56
FLAX SEED, ground, stabilized, raw -----	0.56
SESAME FLOUR, 12% fat -----	0.54
SOY FLOUR, defatted, raw -----	0.50
SOY FLOUR,low fat (6.7%), raw -----	0.50
SPLEEN, beef, braised -----	0.48
SOY MEAL, defatted, raw -----	0.48
YEAST, brewers LewisLabw -----	0.47
SAFFLOWER SEED HEAL, partially defatted -----	0.46
COD, Atlantic, dried, salted-----	0.45
PEANUT FLOUR, defatted -----	0.45
TOFU, freeze-dried -----	0.44
KOYADOFU -----	0.44
SPIRULINA, dried -----	0.44
SEA VEG, spirulina, dried -----	0.44
SOYBEANS, dry roasted -----	0.42
BEANS, soy, dry roasted -----	0.42

MEAT EXTENDER -----	0.42
SESAME FLOUR, 37%fat -----	0.41
SOYBEANS, raw -----	0.39
BEANS, soy, mature, raw -----	0.39
MUSTARD SEED, yellow -----	0.39
KAMUT, grain -----	0.39
WHEAT GERM, toasted -----	0.38
OAT BRAN, raw -----	0.38
SOYBEANS, oil roasted, salted -----	0.38
SOYBEANS, mature, roasted -----	0.38
SOY FLOUR, full fat(22%), roasted -----	0.37
PORK, kidney, braised -----	0.37
SOY FLOUR,full fat(21%), raw -----	0.37
BEANS, winged,raw -----	0.36
CALF LIVER, broiled -----	0.35
PORK SKINS, plain -----	0.35
SESAME SEED, kernel, dried -----	0.35
PORK SKINS, BBQ-flavor -----	0.35
YEAST, baker's, dry -----	0.34
YEAST, Red Star NBC600 -----	0.33
YEAST, Vegetarian Support, Red Star -----	0.33
YEAST, KAL flakes -----	0.33
FISH ROE, baked/broiled -----	0.33
PORK LIVER, braised -----	0.33
BUTTERNUT, dry -----	0.32
WALNUTS, black -----	0.31
WHEAT GERM, raw -----	0.31
POPPY SEED -----	0.30
SUNFLOWER SEED KERNELS, dried -----	0.30
CAVIAR, black or red -----	0.30
LUPIN, raw -----	0.30
GOOSE, domesticated, skinless, roasted -----	0.30
DUCK, domestic, leg minus skin, braised -----	0.29
WATERMELON SEED, kernel, dried -----	0.29
PINE NUTS, pignolia, dried -----	0.29

PEANUT FLOUR, low fat(22%fat)-----	0.29
WATER BUFFALO, lean, roasted -----	0.29
CHICKEN, breast, boneless, skinless, fried -----	0.29
CUTTLEFISH, cooked -----	0.28
PORK, heart, cooked -----	0.28
SUNFLOWER SEED KERNELS, oil roasted -----	0.28
SUNFLOIDER SEED KERNELS, oil roasted, salt --	0.28
DUCK, domestic, breast minus skin, broil -----	0.28
RABBIT, wild, stewed -----	0.28
BEEF LIVER, pan-fried -----	0.27
DUCK, domestic, leg with skin, roasted -----	0.27
SQUASH SEED, kernel, roasted -----	0.27
PUMPKIN SEED, kernel, roasted, salted-----	0.27
SQUASH SEED, kernel, roasted, salted-----	0.27
PUMPKIN SEED, kernel, roasted-----	0.27
WHEAT,hard, red spring -----	0.27
CHICKEN, breast, boneless, skinless, broiled-----	0.26
QUAIL, breast, lean, raw -----	0.26
HORSE, lean, roasted -----	0.26
OCTOPUS, boiled/steamed -----	0.26
SUNFLOWER SEED KERNELS, butter, salted ----	0.26
SUNFLOWER SEED KERNELS, butter-----	0.26
FISH ROE,raw -----	0.26

**GLUTAMATE**

<i>Food</i>	<i>High Glutamate RDA</i>
GLUTEN, commercial -----	0.98
SOY, protein isolate, potassium type -----	0.58
SOY,protein isolate -----	0.58
SOY, protein isolate, Supro, Protein Tech -----	0.56
SOY, protein isolate, ProPlus, ProteinTec -----	0.55
COTTONSEED FLOUR, 1.4%fat, raw -----	0.42
COTTONSEED MEAL, 4.8%fat, raw -----	0.41
SOY PROTEIN CONCENTRATE, acid wash -----	0.40
SOY, protein concentrate, by alcohol -----	0.40
SOY, protein concentrate, by acid -----	0.40
SUNFLOWER SEED KERNELS, flour(1.6%fat) -----	0.39
SESAME FLOUR, 1.6%fat -----	0.37
PEANUT FLOUR, defatted -----	0.36
COTTONSEED FLOUR, 6.2%fat, raw -----	0.34
CHEESE, parmesan, grated -----	0.32
COD, Atlantic, dried, salted -----	0.31
YEAST, Red Star NBC600 -----	0.30
YEAST, Vegetarian Support, Red Star -----	0.30
YEAST, KAL flakes -----	0.30
SOY FLOUR, defatted, raw -----	0.30
SOY FLOUR, low fat(6.7%), raw -----	0.30
SESAME FLOUR, 12% fat -----	0.30
GELATIN, powder, unsweetened -----	0.29
SOY MEAL, defatted, raw -----	0.29
LUPIN, raw -----	0.29
MEAT EXTENDER -----	0.29
SPIRULINA, dried -----	0.28
SEA VEG, spirulina, dried -----	0.28
TOFU, freeze-dried -----	0.28
KOYADOFU -----	0.26
CHEESE, parmesan, hard -----	0.27
COTTONSEED, kernel, roasted -----	0.27

SAFFLOWER SEED MEAL, partially defatted -----	0.27
SOYBEANS, dry roasted -----	0.26
BEANS, soy, dry roasted -----	0.26
PORK SKINS, plain -----	0.25
MILK POWDER, nonfat, +Vit. A&D -----	0.25
MILK, dry, nonfat, +Vit. A, instant -----	0.24
MILK POWDER, nonfat, +Vit, A,instant -----	0.24
WHELK, cooked -----	0.24
CHEESE, Romano -----	0.24
PORK SKINS, BBQ-flavor -----	0.24
SOYBEANS, raw -----	0.24
BEANS, soy, mature, raw -----	0.24
PEANUT FLOUR, low fat(22%fat)-----	0.24
SESAME FLOUR, 37%fat-----	0.23
SOYSEANS, mature, roasted -----	0.23
SOYBEANS, oil roasted, salted -----	0.23
SOY FLOUR, full fat(22%), roasted -----	0.22
SOY FLOUR, full fat(21%), raw -----	0.22
CHEESE, mozzarella, skim, 17% water -----	0.21
CHEESE, provolone -----	0.21
CHEESE, caraway -----	0.21
CHEESE, Edam -----	0.20
CHEESE, Gouda -----	0.20
CHEESE, cheddar(33%fat) -----	0.20
BUTTERNUT, dry -----	0.20
CHEESE, Monterey -----	0.20
CHEESE, Gruyere -----	0.20
PEANUT, Spanish, oil roasted, salted -----	0.19
CHEESE, Colby -----	0.19
SQUASH SEED,kernel, roasted, salted -----	0.19
SQUASH SEED, kernel, roasted -----	0.19
PUMPKIN SEED, kernel, roasted,salted-----	0.19
PUMPKIN SEED, kernel, roasted-----	0.19
FLAX SEED, raw-----	0.19
FLAX SEED, milled, raw -----	0.19

FLAX SEED, ground, stabilized, raw -----	0.19
CHEESE, Cheshire -----	0.19
CHEESE, Swiss -----	0.19
WATERMELON SEED, kernel, dried -----	0.19
CHEESE, goat, 36% water -----	0.19
CHEESE, mozzarella, skim, 16% water -----	0.19
PEANUT,Valencia, oil roasted, salted -----	0.19
YEAST, baker's, dry -----	0.19
SUNFLOWER SEED KERNELS, dried -----	0.19
CHEESE, Muenster -----	0.19
MILK POWDER, whole -----	0.18
CHEESE, lagerkaese -----	0.18
CHEESE, brick -----	0.18
PEANUT, oil roasted, salted -----	0.18
CHEESE, Tilsit -----	0.18
CHEESE, cheddar(7%fat) -----	0.18
CHEESE, cheddar(33%fat), low salt -----	0.18
PEANUT, Virginia, oil roasted, salted -----	0.18
PEANUT, raw -----	0.18
ALMOND, dry roasted, salted -----	0.18
ALMOND, dry roasted -----	0.18
CHEESE, Port du Salut -----	0.18
ALMOND, blanched -----	0.18
RABBIT, wild, stewed -----	0.18
PEANUT BUTTER, salted -----	0.18
SUNFLOWER SEED KERNELS, oil roasted -----	0.17
SUNFLOWER SEED KERNELS, oil roast, salt -----	0.17
WALNUTS, black -----	0.17
BREADNUT SEED, dried -----	0.17

**TRYPTOPHAN**

<i>Food</i>	<i>High Tryptophan RDA</i>
SOY, protein isolate, potassium type -----	4.47
SOY, protein isolate -----	4.47
SOY, protein isolate, Supro, Protein Tech -----	4.40
SOY,protein isolate, ProPlus, ProteinTec -----	4.40
SESAME FLOUR, 1.8%fat -----	4.39
YEAST, brewers, LewisLab -----	4.04
SPIRULINA, dried -----	3.72
SEA VEG, spirulina, dried -----	3.72
SESAME FLOUR, 12%fat -----	3.52
GLUTEN, commercial -----	3.43
SOY,protein concentrate, by alcohol -----	3.34
SOY,protein concentrate, by acid -----	3.34
SOY, protein concentrate, acid wash -----	3.34
BEANS, winged, raw -----	3.05
COTTONSEED FLOUR, 1.4%fat, raw -----	3.01
TOFU, freeze-dried -----	2.99
KOYADOFU -----	2.99
COTTONSEED MEAL, 4.8%fat, raw -----	2.96
SUNFLOWER SEED KERNELS,flour(1.6%fat) -----	2.94
COD, Atlantic, dried, salted -----	2.81
SOY FLOUR, defatted, raw -----	2.73
SOY FLOUR, low fat(6.7%), raw -----	2.70
SESAME FLOUR, 37%fat -----	2.70
SOY MEAL, defatted, raw -----	2.61
PORK, pancreas, braised -----	2.50
WHELK, cooked -----	2.47
COTTONSEED FLOUR, 6.2%fat, raw -----	2.47
SQUASH SEED, kernel, roasted,salted -----	2.31
SQUASH SEED, kernel, roasted -----	2.31
PUMPKIN SEED, kernel, roasted, salted -----	2.31
PUMPKIN SEED,kernel,roasted -----	2.31
SOYBEANS, dry roasted -----	2.30

MEAT EXTENDER -----	2.29
CHEESE, parmesan, grated -----	2.24
ELK, lean, roasted -----	2.18
SOYBEANS, raw -----	2.12
BEANS, soy, mature, raw -----	2.12
MUSTARD SEED, yellow -----	2.10
SOYBEANS, oil roasted, salted -----	2.05
SOYBEANS, mature, roasted -----	2.05
MILK POWDER, nonfat, +Vit A&D -----	2.04
PEANUT FLOUR, defatted -----	2.03
SOY FLOUR, full fat(22%), roasted -----	2.03
SOY FLOUR, full fat(21%), raw -----	2.01
YEAST, Red Star NBC600 -----	2.00
YEAST, Vegetarian Support, Red Star -----	2.00
YEAST, KAL flakes -----	2.00
MILK,dry,nonfat, +Vit A, instant -----	1.98
COTTONSEED, kernel, roasted -----	1.97
YEAST, baker's, dry -----	1.94
CHEESE, parmesan, hard -----	1.93
SESAME SEED, kernel, dried -----	1.89
YEAST, brewers PlusProd -----	1.88
FLAX SEED, raw -----	1.84
FLAX SEED, milled, raw -----	1.84
FLAX SEED, ground, stabilized, raw -----	1.84
CARIBOU, lean, roasted -----	1.83
RABBIT, wild, stewed -----	1.74
SQUASH SEED, kernel, dried -----	1.72
PUMPKIN SEED, kernel dried -----	1.72
CHEESE, Romano -----	1.72
CHEESE, Gruyere -----	1.68
GOOSE, domesticated, skinless, roasted -----	1.61
GOAT, lean, roasted -----	1.61
SAFFLOWER SEED MEAL, partially defatted -----	1.61
CHEESE, Swiss -----	1.60
RABBIT, domestic, stewed -----	1.60

DUCK, domestic, leg minus skin, braised -----	1.60
WHEAT GERM, toasted -----	1.59
SESAME BUTTER -----	1.58
TAHINI, from raw kernels -----	1.57
FENUGREEK SEED -----	1.56
TAHINI, from raw kernels, treated -----	1.56
WATERMELON SEED, kernel, dried -----	1.56
CHICKEN, breast, boneless, skinless, fried -----	1.56
SESAME SEED, whole, dried -----	1.55
BEEF LIVER, pan-fried -----	1.54
RABBIT, domestic, roasted -----	1.54
CHEESE, mozzarella, skim, 17% water -----	1.54
PORK, loin, chop, lean, broiled -----	1.53
DUCK, domestic, breast minus skin, broil -----	1.52
BOAR, lean, roasted -----	1.52
FISH ROE, baked/broiled -----	1.50
PORK, lean, cooked -----	1.50
PORK, leg, lean, roasted -----	1.50
HAM, fresh, lean, roasted -----	1.50
TAHINI, from roasted kernels, treated -----	1.49
PORK, lean, roasted -----	1.49
SESAME SEED, kernel, toasted -----	1.48
SESAME SEED, kernel, toasted, salted -----	1.48
SESAME MEAL 48%fat -----	1.48

**FOODS CONTAINING HIGH LEVELS OF ALL FOUR NUTRIENTS**

<i>Food</i>	<i>High Glutamate RDA</i>	<i>High Tryptophan RDA</i>	<i>High Cystine RDA</i>	<i>High Selenium RDA</i>
BRAZIL NUT, dry -----	0.10---	1.04---	0.23-	50.20
COD, Atlantic, dried, salted -----	0.31---	2.81---	0.45---	2.51
Sunflower Seed Kernals, flour(1.6% fat) -----	0.39---	2.94---	0.63---	0.99
YEAST, Vegetarian Support, Red Star -----	0.30---	2.00---	0.33---	2.37
YEAST, Red Star NBC600-----	0.30---	2.00---	0.33---	2.37
YEAST, KAL flakes -----	0.30---	2.00---	0.33---	2.37
TOFU, freeze-dried -----	0.28---	2.99---	0.44---	0.92
KOYADOFU-----	0.28---	2.99---	0.44---	0.92
MUSTARD SEED, yellow -----	0.17---	2.10---	0.39---	2.26
PORK, kidney, braised-----	0.10---	1.32---	0.37---	5.28
WHELK, cooked-----	0.24---	2.47---	0.25---	1.52
NUTS, mixed, with peanuts,oil roast, salted --	0.14---	0.99---	0.20---	7.14
NUTS, mixed, no peanuts,oil roast, salted ----	0.13---	1.01---	0.20---	7.14
TURKEY, giblets, simmered-----	0.13---	1.23---	0.24---	3.76
WHEAT GERM, toasted-----	0.17---	1.59---	0.38---	1.10
CUTTLEFISH, cooked-----	0.15---	1.45---	0.28---	1.52
SUNFLOWER SEED KERNELS, oil roasted ---	0.17---	1.30---	0.28---	1.33
SUNFLOWER SEED KERNELS, oil roast, salt	0.17---	1.30---	0.28---	1.33
SOYBEANS, dry roasted-----	0.26---	2.30---	0.42---	0.33
SUNFLOWER SEED KERNELS.dried -----	0.19---	1.39---	0.30---	1.01
LAMB, kidney, braised -----	0.09---	1.28---	0.18---	3.71
TURKEY, gizzard, simmered -----	0.17---	1.05---	0.26---	1.58
OCTOPUS, boiled/steamed -----	0.14---	1.34---	0.26---	1.52
WHEAT GERM, raw -----	0.13---	1.27---	0.31---	1.34
Sunflower Seed Kernals, dry roasted-----	0.16---	1.18---	0.26--	1.34
Sunflower Seed Kernals, dry roast, salt ----	0.16---	1.18---	0.26--	1.34
SOY FLOUR, low fat(6.7%), raw -----	0.30---	2.70---	0.50--	0.16
LIVER, lamb, braised -----	0.11---	1.42---	0.21--	1.89
LIVER, pork, braised -----	0.11---	1.46---	0.33--	1.14
PUFFED WHEAT, enriched -----	0.17---	0.90---	0.19--	2.09
SPLEEN, beef, braised -----	0.08---	1.04---	0.48--	1.55
SOYBEANS, raw -----	0.24---	2.12---	0.39--	0.30

BEANS, soy, mature, raw -----	0.24	2.12	0.39	0.30
PORK, pancrease, braised -----	0.08	2.50	0.24	1.23
CHEESE, parmesan, grated -----	0.32	2.24	0.18	0.44
COTTONSEED FLOUR, 6.2%fat, raw -----	0.34	2.47	0.72	0.10
CHICKEN, gizzard, simmer -----	0.15	0.97	0.24	1.60
SOYBEANS, mature, roasted -----	0.23	2.05	0.38	0.32
BEANS, soy, oil roasted, salted -----	0.23	2.05	0.38	0.32
SOYBEANS, oil roasted, salted -----	0.23	2.05	0.38	0.32
BEANS, soy, mature, roasted -----	0.23	2.05	0.38	0.32
SPIRULINA.dried -----	0.28	3.72	0.44	0.12
CHICKEN LIVER, simmered -----	0.11	1.37	0.22	1.70
MILK POWDER, nonfat.+Vit. A&D -----	0.25	2.04	0.22	0.46
CAVIAR, black or red -----	0.12	1.29	0.30	1.11
TURKEY, liver, simmered -----	0.10	1.35	0.21	1.71
TUNA, light, canned in oil, drained, salt --	0.15	1.31	0.21	1.29
TUNA, light, canned in oil, drained -----	0.15	1.31	0.21	1.29
YEAST, baker's, dry -----	0.19	1.94	0.34	0.41
FISH ROE, baked/broiled -----	0.11	1.50	0.33	0.88
PORK, loin, chop, lean, broiled -----	0.16	1.53	0.26	0.80
OAT BRAN, raw -----	0.13	1.34	0.38	0.77
BEEF LIVER, pan-fried -----	0.12	1.54	0.27	0.97
MILK POWDER, nonfat, +Vit A, instant --	0.24	1.98	0.22	0.46
PORK, leg, lean, roasted -----	0.15	1.50	0.25	0.85
HAM, fresh, lean, roasted -----	0.15	1.50	0.25	0.85
PORK, lean, cooked -----	0.15	1.50	0.25	0.77
ANCHOVY, canned, drained -----	0.14	1.30	0.21	1.15
LOBSTER, spiny, cooked -----	0.15	1.47	0.20	1.00
RABBIT, domestic, stewed -----	0.16	1.60	0.25	0.65
PORK, lean, roasted -----	0.15	1.49	0.25	0.76
WHEAT, hard, red spring -----	0.16	0.78	0.27	1.20
PEANUT FLOUR, defatted -----	0.36	2.03	0.45	0.12
LIVER, lamb, pan-fried -----	0.09	1.18	0.18	1.97
RABBIT, domestic, roasted -----	0.16	1.54	0.24	0.65
LIVER, beef, braised -----	0.11	1.40	0.25	0.95
MUSSEL, blue, boiled/steamed -----	0.11	1.07	0.21	1.52

KIDNEY, veal, braised -----	0.08---	1.35---	0.19--	1.69
TUNA, light, canned in water, drained ---	0.13---	1.14---	0.18--	1.36
TUNA, light, canned in water, drain, salt --	0.13---	1.14---	0.18--	1.36
SEMOLINA, enriched -----	0.15---	0.65---	0.24--	1.52
Sunflower Seed Kernals, toasted, salted ----	0.14---	1.05---	0.23--	1.06
WHEAT BRAN, raw -----	0.10---	1.13---	0.25--	1.32
PORK, spareribs, lean+fat, braised -----	0.15---	1.48---	0.25--	0.63
MEAT EXTENDER -----	0.29---	2.29---	0.42--	0.13
TUNA, yellowfin, baked/broiled -----	0.15---	1.34---	0.21--	0.79
TUNA, bluefin, baked/broiled -----	0.15---	1.34---	0.21--	0.79
PORK, lean+fat, cooked -----	0.14---	1.40---	0.24--	0.70
KIDNEY, beef, simmered -----	0.10---	1.39---	0.05--	4.76
CHICKEN, breast, boneless, skinless, fried	0.17---	1.56---	0.29--	0.44

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Zuma, Jacob 4

## The Author

The author lives with his wife Sarah, stepson Dan, and cat McNuff in Victoria, British Columbia. A Canadian by choice, he was born in Tunstall, Yorkshire, England where he was educated at the Hull Grammar School and University College London. While at university, he specialized in geology and geography, earning a B.Sc. in 1964 and Ph.D. in 1968 from London University.

He has been a faculty member in the Department of Geography, University of Victoria since 1967. A tenured professor, he has authored or edited some 190 publications, the majority of which focus on reducing disaster losses or identifying the causes of chronic disease or longevity. He has published hypotheses on the origins of numerous diseases including myocardial infarction, AIDS, cancer, diabetes, schizophrenia, multiple sclerosis, amyotrophic lateral sclerosis, Alzheimer's and Parkinson's diseases, and stroke.

His numerous books include *Disaster planning: The Preservation of Life and Property*, Springer Verlag, New York; *Reducing Cancer Mortality: A Geographical Perspective*, Western Geographical Press, Victoria; and *The Ozymandias Principles*, Southdowne Press, Victoria.

He is a member of the Explorers Club and several academic organizations including The New York Academy of Sciences, The Royal Geographical Society, and The Royal Society of Literature. He is also the editor of both the International and Canadian Western Geographical Series and is a member of the boards of the Journal of Orthomolecular Medicine and the Canadian Schizophrenia Foundation. He has been a consultant to numerous organizations, including the United Nations, NATO, and the governments of Canada, Ontario, and British

Columbia. Every day he takes at least the recommended daily allowance of the known essential nutrients, in the belief that this will slow the aging process. As a consequence, most of his salary is spent in health food stores. His other bad habits include providing treats to all the neighbourhood dogs; losing at chess to his computer; being regularly beaten by Dan at video games; and, with the assistance of @Derby and various computer models, failing to correctly predict the outcomes of horse races. For a more complete curriculum vitae visit <http://www.geocities.com/fosterhd/>.

The man who discovers a new scientific truth has previously had to smash to atoms almost everything he had learned, and arrives at the new truth with hands blood-stained from the slaughter of a thousand platitudes.

José Ortega y Gasset  
*The Revolt of the Masses*, 1930