Foley of LANL Dissociates from Moore of Cornell and Exposes Himself

(May 8, 2007) A few days ago, a passionate, although exactly what passion will consume him at any moment is not always predictable, contributor to AIDS dissidence “net-lore” learned the word “proteomics” (although not its meaning), and for reasons known (perhaps) only to himself began corresponding with Dr. Brian Foley of the Los Alamos National Laboratories, famous for building the bomb, and not much else.

In his first inquisitive missive to Dr. Foley, the naïve-sounding cyber-being asked if perhaps techniques of “proteomics” might be applied to the problem of HIV particle ‘isolation’.

Foley leaped at the chance to snow-job the borderline “denialist”, and wrote him back as follows:

*That would depend on the viral load in the blood. One milliliter of blood weighs 1 gram, of which roughly 83% is water. So we have say 0.17 gram (170 milligrams) of proteins, lipids, salts, nucleic acids and other components per milliliter of blood, to work with. A retrovirus has a mass of around 2.5 to 3 times ten to the eighth power Daltons per virion. So if we have 100,000 virus particles per ml, we have 3 time 10 to the 13th power Daltons of viral material (protein, RNA and lipids, assuming no water in the virion). Dividing by Avagadro’s number (the number of Daltons per gram) that means we have 4.982 times ten to the negative 11th power grams of viral material per ml of blood, or roughly 50 femtograms. So in a whole liter of blood, we could get 50 picograms of viral material, if there were 100,000 virions per ml. The liter of blood would have 170 grams of human proteins, lipids, DNA, salts, etc.*

This went on for many pages (with 4 pdfs attached) of similar waffle, double-speak deception and techno-babble designed to confuse and impress the kindergarten kid, who was indeed so confounded he even wrote me asking for help in dealing with the mass of material Foley sent and that he didn’t “quite understand completely”.

[As an aside, like Christine Maggiore wonders here - http://barnesworld.blogs.com/Commentongeiager.pdf - I wonder what this tax-payer paid scientist has to do besides spend hundreds of hours prowling the internet and writing at extravagant lengths to “refute the denialists” wherever they may be hiding and no matter how insignificant their emails might be.]

I suggested that he send him the pages from my biography of Duesberg that are reproduced with Foley’s interlinear below, which he did with some introductory lines that are, with Foley’s comments in black, also reproduced.

Foley begins (after the brief introduction) with some unexpected revelations about himself and his relationship with a self-styled general of AIDS Truth, John Moore (still) of Cornell Med’s faculty and not psychiatric teaching ward.

Brian, I’ve read carefully through what you wrote. It conflicts with some material I’ve read, which I’ll reproduce here. Yes. Even within the "rethinker" groups, there are many
conflicting ideas that cannot all be right.

Quite honestly, I don't know what to believe. I am not a biological scientist. This crap pisses me off and confuses me.

I am a biological scientist, with most of my training in infectious disease and cancer biology. And much of it still confuses me too, so don't feel bad about that.

So you know this is from Harvey's book I don't want to be like Christine and not understand what you're trying to say.

I have very different ideas than John Moore, about how to deal with information etc. I don't want to "push" stuff on anyone. I see a huge and significant difference between someone who is infected (or diagnosed as being infected) and who wants to live in peace and happiness, and people such as Harvey Bialy and Peter Duesberg who are not even at risk of infection and who know very well when they are being honest and dishonest.

I myself, am not a big fan of most doctors and "western medicine" etc. I don't like car mechanics either, for many similar reasons. I buy very used cars and work on them myself, as much as I can.

Quality of life is as important as quantity. It might be better to live 5 years in bliss than 10 with suffering. But that is an individual thing. Some people sort of like suffering, or at least tolerate it very well. Other people are never blissful at all. We also have to weigh costs as well as benefits. The best actions for a rich man with good insurance etc are different than the best actions for someone who has to pay for his own treatment and only has access to a local doctor.

Also, no matter what condition be it HIV infection or diabetes, we are all individuals whereas all medicine is based on statistics. Maybe 40% of people benefit from treatment Y, but there can be some ways of predicting in advance whether a given individual will be among that 40% or not. With HIV, most of our data was compiled from IV drug users and other risk groups which may not be indicative of others such as Christine or yourself.

I am willing to help you as much as I can, but I don't want to "push" too much. Tell me when I should back off.

So, where is he wrong and you're right? Can you break down what he has written? I do not understand enough to understand which one of you is telling me the truth.

I'll do what I can. With Bialy, it is often hard to tell what he is talking about. He is not straightforward at all.

And here begins the text from Chapter 3 of Oncogenes, Aneuploidy and AIDS. In this case prediction was more than confirmed as he wrote pure grade school waffle not much better than what Ken Witwer produced to “refute” Dr. Culshaw [http://barnesworld.blogs.com/barnes_world/2007/04/science_sold_ou.html] and hogwash that went on at greater length than the narrative he was sent. One gem is in bold italic. The scientific “sounding” stuff is of the same order of logical coherence.

However, the hogwash, which was also copied by the erstwhile cyber-being to David Crowe, steward of the Alberta Reappraising AIDS Society website, produced a VERY educational result that is located at the very end of this PDF, and which I commend to everyone’s attention as it is extremely instructive, and goes to prove (yet again) that “Bad Manners often produce “gossip” of the highest order…sometimes called truth. (OAA text in blue, Foley’s comment in black)
Some readers might discern a certain resonance with the NIH 3T3 cell and the lack of functional proof of cellular transforming activity associated with proto-oncogenes before its adventitious arrival. When standard assays fail to provide the required answers, new ones with which to keep a popular hypothesis afloat can always be devised. The references that Fauci provided a few days later prompted an editorial commentary for *Bio/Technology* entitled "Where is the Virus? And Where is the Press?" in which I pointed out an obvious and fatal flaw in these first-generation new techniques for measuring HIV by means of a surrogate biochemical marker, instead of the real biological parameter, infection.

In this case, the biochemical surrogate for infectious HIV is called "p24 antigenemia" and it was supposed to detect the number of HIV particles present in a patient's serum by measuring the amount of a particular virus protein with a molecular mass of 24 kiloDaltons that is at the core of HIV and every other infectious retrovirus.

Measuring the amount of p24 is one of several ways of determining "viral load" or number of particles, or mass, of HIV-1 virions per ml of blood. Looking at HIV-1 proteins, rather than HIV-1 proviral DNA, is needed because we don't want to measure inactive virus, just the ones that are actively replicating.

Today almost nobody, including AIDS' physicians even remembers the term, let alone uses the results of such assays in assessing the clinical status of their patients. Instead, they rely on the second-generation of assays that detect surrogate biochemical markers as the basis to administer potent, toxic 'anti-viral' chemicals.

Not all antiretrovirals are equally toxic. And different people tolerate different combinations differently. Some people get a lot of nausea from one combination, and others don't. Some get tingling fingertips and others don't. A few have severe reactions, even life threatening ones, and if they are ignored they can be fatal.

These tests, made familiar by David Ho and *Nature* in 1995 produce a number suggestively termed the "viral load", that has so clearly supplanted "p24 antigenemia" it outscored its predecessor by 2,250 to 82 in scientific publications in the scant five years between then and the end of the last century.

There were several problems with measuring p24 antigen levels in blood, which made newer methods better. The biggest one is that most HIV-infected people have very strong anti-HIV antibody responses, so more than 90% of the virions in the blood can be bound to antibodies. If we want to know how well a drug is working and not just how well the combination of drug plus antibody response is doing (and the antibody response can be highly variable, even within one patient as the virus evolves to evade some antibodies), we want to look at all virions, not just those that escape the antibodies. So you have to use heat and/or salt to free the virions from the antibodies before you measure the p24. Secondly, measuring the mass of a single protein, (not the 24 kiloDalton mass of a single molecule of p24, but the total mass of the millions of p24 molecules in a milliliter of blood) among a mix of all blood/serum proteins, is not easy. In fact it is very difficult. Reproducibility, testing multiple aliquots of the same well-mixed unit of blood, is low. It is just not that great of a test, and very expensive and time consuming to do.

But first generation surrogates first. The spate of papers Fauci sent claimed to have answered the central problem of so little virus and so much disease.

Actually, it is not "so much disease". HIV never kills anyone, it only weakens the immune system to certain types of infections. People with nearly zero CD4 count are not extra susceptible to influenza and most other viruses and bacteria that can be fought with antibodies. CD4 cells are helper cells, not antibody-making B cells. So the lack of CD4 only increases the problems caused by some fungi, and some viruses, that require a good helper response.

Also, it takes about 2 to 14 years or so for HIV to lower CD4 count below 200. Many other viruses kill people within 48 hours to 2 weeks of infection.
by demonstrating that in fact there was ample virus present in the blood of patients during the late stages of AIDS. But they made this claim based on an assay that detected a virus-specific protein, not the virus itself. Since viral protein is present in an infected person in different forms, e.g., in the blood, as part of an infectious virus or complexed with antibodies that effectively render it incapacitated, or inside phagocytic white blood cells where it is also non-infectious, the results of tests based on measuring this protein might be difficult to interpret correctly. In fact, the problem turns out to be severe enough to render the tests worthless. The amounts of protein reported in these papers imply a quantity of infectious virus of more than 100,000 particles per milliliter of blood, because the author's claim to detect upwards of 50 picograms (pg) of p24 protein per milliliter.

That 50 picograms per ml is off by like a factor of 1,000 or so (depending on what percentage of the mass of a virion is made up of p24). Harvey has not cited the source, so I am not sure where to find out if researchers really were talking about 50 picograms of p24 per ml equaling 100,000 virions/ml. I suck at math, so it could very well be an error I made this morning. But anyway, it is sort of a moot point. The important part of measuring things like this is not to know exactly how many infectious virus particles there are in a ml of blood, but only to know relatively how much there is. We want to know if patients who live long and healthy have less virus than those who get sick, for example (it could very well be that number of viruses means nothing, and other factors such as co-infection with herpes simplex is the key; or having a specific class of HLA alleles). We also want to know if the anti-retrovirals are working (can we reduce the amount of virus bay 90% or 99% or 99.999%? Regardless if it is reduced from 100 to 1, or 100,000 to 10,000 viruses per ml).

A picogram is one thousand billionth of a gram. Not much, but a retrovirus is one thousand times lighter, and only one half of its weight is p24. The appropriate long division shows that 50 pg correspond to 100,000 virus particles, an amount that if actually present would not require any special biochemical assay to detect. Such concentrations of virus would be easily seen by the electron microscope or by standard biological assays.

That is wrong. 100,000 sounds like a lot, but it is still just picogram quantities. Mixed in with a full 830 or so milligrams of water, and 170 milligrams of other proteins, it is sort of like a bunch of fleas in a haystack. It is easy to see viruses budding from T-cells in the blood, and we usually often also see several very near to the cell they just budded from. But once they float away into the ocean in between the infected cells it is difficult to see them.

Yet none of the papers showed anything resembling such a real viremia, and one even reported that no virus at all could be found in 31 "antigenemic" patients even after extensive in vitro cultivation of more than one million of their T cells, accompanied by the harsh treatments necessary to wake a sleepy retrovirus like HIV.

I have no doubt that Harvey's ref 26 reports some type of difficulty culturing virus. But without reading ref 26, I could only guess as to what they reported. Was this 31 of 31 patients? Or was it 31 out of 100 patients had virus that was impossible to culture using their methods? It turns out that some strains of HIV are easier to culture than others. None of the HIV strains are real simple to culture. That is why Gallo and Montagnier ended up with so many problems in the first 2 years of trying.

The authors of the various studies I questioned in the Bio/Technology piece never answered these objections in print: Perhaps because they "could not take time from their busy schedules to correct my simple minded, and erroneous calculation", as I was informed on a few occasions. Or maybe, because the basic error was not in the calculation but in the assay itself, they simply, "could not". In either case, the test never became particularly popular as the citation numbers above reflect. Even in its heydays between 1987 and 1995, "p24 antigenemia" was cited in only 144 publications. Nonetheless, viral antigenemia would become the basis for a remarkable exchange between Prof. Duesberg and one of his formidable scientific foes in Washington, DC in the spring of 1988. The occasion was a panel discussion sponsored by the American Foundation for AIDS Research (AmFAR), Elizabeth Taylor's favorite charity.

The event, to which 17 privileged journalists, of which I was one, were invited, was held on the campus of George Washington University, and was promoted by AmFAR as a "Scientific Forum on the Etiology of AIDS". It was, in the words of their fact sheet, "convened to
critically examine the evidence that human immunodeficiency virus (HIV) or other agents give rise to the disease complex known as AIDS. Data from laboratory, clinical, and epidemiological research will be presented and evaluated. The forum seeks no consensus, instead it is designed to permit discussion among experts on the conclusions the facts permit."

Similar to staging a "scientific debate" about creation vs. evolution, most serious scientists refuse to do so publicly.

In contrast to these noble objectives, the actual purpose of the event was more accurately described after the fact by Michael Specter, a mainstream AIDS journalist for the Washington Post. [Yes, this is the same Michael Specter featured here - http://barnesworld.blogs.com/barnes_world/2007/03/john_strausbaug.html. Evidently this event made such a powerful impression on him he still feels compelled to write about until today. The ONE thing that remains important about it so many years later is that this STAGED debate is the ONE and ONLY time Duesberg and his critics appeared on the same platform in front of a "serious" (or any for that matter) audience. If the "rebel without a cause (of aids)" is so utterly bankrupt of ideas why hasn't he been "set-up" again? HB 9.05.07]

who is no friend of Peter's, as follows: "Billed as a scientific forum on the cause of AIDS, it was really an attempt to put Duesberg's theories to rest." A different and detailed account of the AmFAR forum by John Lauritsen, a Harvard educated AIDS journalist who is Peter's friend, appeared in the New York Native, a gay newspaper critical of much of mainstream AIDS reporting. The part that pertains to antigenemia was riveting then and is even more powerful twelve years later. As it coincides perfectly with my own recollections, and has the dual advantages of being contemporaneous, and withstanding the tests of publication and time as to its accuracy, I quote from it at length. Lauritsen begins the relevant section by describing the demeanor of one of the high-ranking field officers in the new NIH war on AIDS.

William Haseltine, Chief of the Laboratory of Biochemical Pharmacology at the Dana Farber Cancer Center of Harvard Medical School, appeared to be an angry man. His presentation was devoted largely to personal attacks on Duesberg, in a manner which two of my colleagues described as "brutal" and "vicious". Haseltine's anger can probably be attributed to Celia Farber's interview with Duesberg in Spin Magazine (January 1988), in which Duesberg stated: "William Haseltine and Max Essex, who are two of the top five AIDS researchers in the country, have millions in stock in a company they founded that sells AIDS kits that test for HIV. How could they be objective?"

When Celia Farber contacted Haseltine, he confirmed his and Essex's business arrangement with Cambridge Bio-Science, a company that sells HIV testing kits. Said Haseltine: "I deeply resent the implication that my business investments have affected my work." Haseltine accused Duesberg of "serious confusion and misrepresentation of fact". He said that when rational arguments don't hold up, Duesberg "has resorted to personal attack; he has impugned the motivations of individuals and institutions."

OK. None of that surprises me. Neither the fact that it happened, nor that different people would report on the same fact in different ways.

So much for background, now to the theatre itself:

The most dramatic moment in the forum came when Haseltine began showing his slides; it deserves a separate section:

In presenting his first slide, Haseltine said: "This gives us a summary of the virology. Dr. Duesberg asserts that during the later phases of the disease one does not see free virus in circulation. That is not generally reflected in the patients". Pointing to his projected graph he continued, "the black line represents either virus titer or viral antigens directly detectable in the circulation. It rises later in the disease. That rise is concomitant with the period when T-cells fall. So it is not the case, the central assertion he has made in his arguments, that one does not have viremia."

At this point Duesberg asked, "Why are there no units on that slide?" Haseltine's response was, "Don't interrupt me; I didn't interrupt you." Duesberg replied, "I merely
asked why the slide has no units on it.” Haseltine angrily refused to answer the question, and the chairman intervened, saying that questions would have to wait until the presentation was finished.

Perhaps Duesberg ought to have waited, but one can understand his impatience. Witnessing a fast-flowing stream of propaganda, he spotted something that was obviously wrong, and wanted to confront it before the moment was lost. That his suspicions were more than justified became clear later.

In the question period following Haseltine’s presentation, Harry Rubin asked Haseltine if he could provide a reference for his statement that nude mice were capable of mounting a vigorous immune response (something he had casually asserted earlier, and which is completely untrue - HB). Haseltine said that there was a large literature on nude mice: “If you haven’t read it, how can I discuss it with you?”. Rubin gently replied that perhaps he had read it, but that he had only asked for a reference.

Duesberg then requested that the slide be shown on the screen again, and asked if it were an accident that the slide had no units on it. Haseltine was unable to answer the question himself, and asked Dr. Robert Redfield of the Walter Reed Army Research Institute, sitting in the audience, to explain how the slide was prepared. Redfield said something to the effect that “different measurements were used”, a grossly inadequate explanation. When Duesberg persisted, Haseltine became truculent, and said that Duesberg should read the literature, because there were different measures that could be used. With no satisfactory answer forthcoming, the chairman moved on.

The truth about the Slide Without Units came out in the evening, at a party at the home of Dr. Harris Coulter. In a relaxed and convivial mood, Redfield admitted, in the presence of Duesberg, Rubin, myself, and several other witnesses, that the graph had been prepared to illustrate a theoretical possibility. It had no units on it for the simple reason that it was not based on any data at all. In other words, the slide was a fake.

I’ve seen many such slides of real data, and also many such as Haseltine's which are merely "cartoon representations" of the average behavior of the real data. It is common in cartoons to not provide the units. And even when units are provided, it is technically wrong. For example some patients arrive at the "crisis" where CD4 count plummets and viral load rises, after 2 to 4 years of relatively stable numbers, and others arrive there 10 more years later. So putting a time line labeled with years 1 through 10 is wrong because it would look as if the cartoon plot were indicating that all patients reached the crisis at the same time.

So the slide was not exactly "a fake", it was just a simplified picture of the average condition, rather than a complex plot with 3 lines (one for viral load, one for CD4 count, one for antibody level) for each of 30 patients.

This pure invention of how HIV should behave if it were causing AIDS

Not a pure invention from no data. Just an averaging of many data points into one.

has, like other things untrue, found its way into textbooks and is the basis of official US government explanations of AIDS pathogenesis.

Well, not Haseltine's exact figure, but yes most textbooks etc, use that type of cartoon of the average behavior, to explain the average of the data.

Nonetheless, among most researchers the problem of massive cell killing by miniscule amounts of virus (David Ho's "new view", and the popular press notwithstanding)

100,000 viruses per ml, of which some 1% are infectious, is some 1,000 infectious viruses per ml. Typical CD4+ T-cell counts are typically 1,000 per microliter in healthy people. So that is only one infectious virion per 1,000 cells.

It is not all that surprising that it takes years for the CD4+ T-cell count to decline. If there were 100 infectious viruses per infectable cell, I might expect a rapid disease, with people dying within a few weeks of infection, depending on which type of cell was being infected. There really is not "massive cell killing". The CD4 count in most people declines very slowly
over many years. It's not like it goes from 1,000 down to zero in a week or two.

This sort of thing, with Haestline and Duesberg presenting an overview of the work of hundreds of other people, and then their presentation being "reported on" by a third party, is not really "science". Common sense is not always the best predictor of the way things really are. There is a lot in this world that is nearly impossible for humans to believe, but turns out to be very true. A lot of our experiences would make us think, for example, that heavy things fall to earth faster than light things. But given equivalent wind resistance forces, they fall at the same rate.

If you "rethink" or micro-dissect any field of research it can look absurd. For example all my training in physics and chemistry (not lot, but though college-level classes) teaches that it is ridiculous to claim that any force could "organize" air molecules to all fly around in the same circle to create a hurricane. So if the religious people wanted to, they could argue that God controls the weather, just as they argue that God (or another Intelligent Designer) creates life. It would be easy to sound like there was "scientific proof" and "plain common sense" that storms can't form just by themselves.

remains the central unanswered question about the HIV=AIDS equation, as a Pub. Med. search of the scientific literature using "HIV and", pathogenesis, indirect cell killing, mechanisms of cell killing, cytocidal effects, or similar terms will quickly verify.

Yes. It remains to this day, rather difficult to explain how a pathogen and host live together for many years in a rather stable relationship, and then things fall apart in the end. There are clearly many viral factors involved, and we are beginning to get enough data to see that different subtypes are more or less pathogenic than others (it is not fair to compare Ugandans with subtype A to Americans with subtype B, because there are many differences between Uganda and America that could influence the difference more than the virus strain does). But even then, it remains a mystery, with viral load not being the only answer:


But there are more human factors. We have many sets where the same unit of blood was split and infected several people with the exact same strain of virus, and each progresses differently. And with the monkeys, infected with a molecular clone so the virus is absolutely identical in all, each progresses at a different rate. We have very few identical twin infections to study.

The first reaction from some people is "Well, it's not the virus at all, then!". But with no virus at all, we humans typically live to be 40 to 85 years old and die from heart failure and cancer etc. whereas with infection the range is more like 2 to 20 years after infection with a large peak of deaths around 8 to 12 years after infection.

And even the 2 to 20 years is not the "full range" but just the range into which 95% of cases fall. There are many (a low percentage, but still many individuals) who are long term non-progressors. Contrary to what some people would like to believe, the long-term non-progressors are not easy to identify by "lifestyle". While smoking cigarettes and eating fatty/sugary foods is not going to really help anyone, and eating well and exercising is good, it is not simply a "healthy" vs "unhealthy" lifestyle that predicts disease progression rates.

EDUCATIONAL BONUS

From David Crowe
The Redford/Haseltine Invention (remember at the time of the presentation there was no cumulative data that could have been averaged as Foley suggested.

Today’s NIAID Figure

Today, on a NIAID website entitled “The Relationship between the Human Immunodeficiency Virus and the Acquired Immunodeficiency Syndrome” (http://www.niaid.nih.gov/publications/hivaids/all.htm) you will find the following figure (Figure 4) described as showing the “Typical course of HIV infection” and references Pantaleo, 1993a which turns out to be Pantaleo G, Graziosi C, Fauci AS. The immunopathogenesis of human immunodeficiency virus infection. N Engl J Med 1993a;328(5):327-35.
Pantaleo's 1993 Figure

If we go to the Pantaleo paper, we see something similar, but not identical:

It’s not clear why the NIAID referenced a paper for a figure but then made changes to it. That is certainly not honest science. If they had additional data they should have shown it, or included a reference to that also. The changes include:

- Addition of an HIV RNA axis (and data to match with the triangles).
• Replacement of the word “Possible” with the symbols +/- in front of “acute HIV syndrome”.
• Replacement of “Plasma Viremia Titer” with “Culturable Plasma Virema (dilutional titer).

The source of this figure is not data in this article, but another earlier paper, this time with the first author Fauci from 1991. But before we get to Fauci’s 1991 paper let’s look at Fauci’s 1996 summary of a conference which has the following figure:

Fauci’s 1996 Paper


Note that this figure is identical to the NIAID figure, even though both NIAID and Fauci reference the Pantaleo paper as the source of the figure. Clearly this is the true source of the figure, not Pantaleo’s 1993 paper. Although, rather confusingly, Fauci’s 1996 paper also references Pantaleo 1993 as the source of the figure.

Would it have been a bit much for a top bureaucrat to have his own figure having such a prominent place on a government website? Fauci was a co-author of the Pantaleo 1993 paper, but that would be lost on most readers. Did Fauci forget that when he included the figure in his 1996 paper he made a few adjustments?

Fauci’s 1991 Paper

Fauci’s 1991 paper is where the trail goes cold, it has no reference for the source of the data, but does title the figure “Typical course of HIV infection”, indicating that perhaps this is not constructed from data but merely from unproven ideas about the pathogenesis of HIV and AIDS.

The changes between this paper and the later versions are very significant. Differences in this original version are:

<table>
<thead>
<tr>
<th>Change</th>
<th>Fauci 1991</th>
<th>NIAID/Fauci 1996</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary HIV infection</td>
<td>Not mentioned</td>
<td>Shown at about week 6</td>
</tr>
<tr>
<td>Second axis</td>
<td>“p24 Antigenemia (pg/ml)”</td>
<td>“Culturable Plasma Viremia (dilutional titer)”</td>
</tr>
<tr>
<td>CD4 rebound</td>
<td>Returns to about 800 by week 9 after an initial drop</td>
<td>Only up to about 650 by week 12</td>
</tr>
<tr>
<td>Virus drop</td>
<td>Drops to negligible levels by week 9</td>
<td>This is shown at week 12.</td>
</tr>
<tr>
<td>Virus blips</td>
<td>Upward movements of CD4 counts at years 2 and 4 are shown (about 100/150)</td>
<td>Upward blips at years 3 and 5 are much smaller (much less than 100)</td>
</tr>
<tr>
<td>Final virus rise</td>
<td>The final rise in virus count is shown at year 6</td>
<td>The final rise is shown starting at year 7</td>
</tr>
<tr>
<td>Virus plateau</td>
<td>The virus count is shown leveling off at year 8</td>
<td>The virus count continues to rise until death at year 11.</td>
</tr>
<tr>
<td>Early Symptoms</td>
<td>Shown at year 5</td>
<td>“Constitutional symptoms” are shown at year 8</td>
</tr>
<tr>
<td>Late symptoms</td>
<td>PCP is shown at year 8</td>
<td>“Opportunistic diseases” is shown at year 9</td>
</tr>
<tr>
<td>Death</td>
<td>Shown at year 10</td>
<td>Shown at year 11</td>
</tr>
</tbody>
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It is very important to note that this paper by Fauci is not original research, it is a report on a conference. With no reference to a source, one can only assume that Fauci made the graph up or copied it from a slide presented at the conference. Given the later
changes one can only assume that this data is only illustrative and does not reflect real changes in CD4 counts and ‘viral load’ (however that is measured). It may reflect Fauci’s opinions more than reality.

Summary
The simplest story is that Fauci had a chart in 1991 and kept elaborating it as technology changed. p24 counts went out of vogue, so the chart was shown to indicate counting virus by culture dilution instead. There is no evidence that the chart is based on real numbers from real patients.