Questions for Prof. Duesberg Pertaining to his Area of Expertise

1) **Joyce Arthur states:** *the very fact that the South Africa estimates were inflated by more than double speaks volumes about the serious problems in HIV testing and identification - the very thing Duesberg et al. are pointing out!*

the obvious likely explanation for the differences between SA and Uganda is different practices in regards to testing, identification, prevalence estimating, etc. . . . Also, the very fact that there's such a large difference points again to the serious problems with HIV tests

What's incredible about this statement is Jensen's apparent ignorance of the common dissident observation that pregnancy causes a high rate of false positives. Henry Bauer had a recent good post explaining this in detail: http://hivskeptic.wordpress.com/2009/10/05/why-pregnant-women-tend-to-test-"hiv-positive"/ It may be because "Pregnancy brings a Th1→Th2 shift in the immune system [and] 'HIV-positive' is associated with a Th1→Th2 shift." Unfortunately for Jensen, this theory DOES discredit the tests and the statistics.

Question: Do you agree, then, that the HIV tests are wholly discredited and unreliable, and that they cannot be used to confirm any virus theory, be it passenger or pathogenic?

2) **Joyce Arthur states:** *Jensen says: "This is a powerful argument; it is also an old one, originally based on the understanding of HIV in the late 1980s. Since then it has been claimed that HIV infection is never completely suppressed, that HIV 'hides in secret places' in the body, and that tests such as 'viral load' are indirect proof of this." The operative word in that sentence is "claimed", as there isn't a shred of evidence for any of these claims as far as I know.*

Question: Do you agree that all the various HIV RNA and DNA tests that detect supposed viral activity and viral reservoirs have never been proven to do any such thing? In an interview from 1993, you state:

Bob Guccione: *What about this recent discovery that large quantities of HIV are in the lymph nodes?*

Duesberg: *What they're doing is using a bigger scope, the polymerase chain reaction, which amplifies a needle in a haystack to a haystack itself. So now you can all of a sudden see it. And they say, isn't it great what we can see with a new scope. Well, the problem is, you don't help the emperor a lot if you can see his clothes only with a microscope. All they're doing is applying bigger and bigger scopes. They magnify the needle, but they don't make more of it, they only see it better.*

Question: Since they were able to make a haystack of a single needle (or straw) pretty much from the first day PCR technology came into use, can we take it that by "using a bigger scope", you mean ever more sensitive PCR tests with increasingly "modified" primers - and again, that none of these can be used to prove viral presence or activity?

4) In the same interview you state:

Duesberg: *all viruses mutate, but in a very limited way. Actually, that was my claim to fame. In 1968 I found out why that is: They have different chromosomes. This [flu virus] is one of the rare viruses with multiple*

chromosomes, in fact it was the first time this was shown in a virus. And that gives it the additional ability to recombine.

Guccione: *Why can't HIV do the same thing? Why can't it recombine?*

Duesberg: *Because it doesn't have segmented chromosomes. And viruses with a single genome [genetic information] cannot recombine, they can only exchange in a very minor, very limited way.*

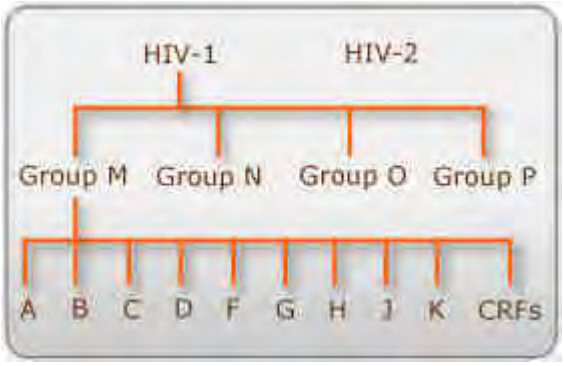
Guccione: *And HIV has only a single genome. So they've looked and discovered that HIV does not have the capacity to recombine.*

Duesberg: *It does have the capacity but it cannot change like flu because all HIVs are closely related. They are all one genome so if you recombine one genome with another, they are nearly identical and you don't get any new, different recombinant. There are chicken flus and swine flus and human flus and they can all recombine. But the HIVs are all from humans, and they are virtually all identical*

Question: Brian Foley of Los Alamos claims that HI-viruses are "as different as mice and elephants". What do you make of the various strains, groups, clades etc. of HIV-1, which can be almost as different from each other as HIV-1 and HIV-2? Here's a quick overview from Avert.org:

"How many subtypes of HIV-1 are there?

The strains of HIV-1 can be classified into four groups: the "major" group M, the "outlier" group O and two new groups, N and P. These four groups may represent four separate introductions of simian immunodeficiency virus into humans.



The different levels of HIV classification.

Group O appears to be restricted to west-central Africa and group N - a strain discovered in 1998 in Cameroon - is extremely rare. In 2009 a new strain closely relating to gorilla simian immunodeficiency virus was discovered in a Cameroonian woman. It was designated HIV-1 group P.[1] More than 90% of HIV-1 infections belong to HIV-1 group M and, unless specified, the rest of this page will relate to HIV-1 group M only.

Within group M there are known to be at least nine genetically distinct **subtypes** (or clades) of HIV-1. These are subtypes A, B, C, D, F, G, H, J and K.

Occasionally, two viruses of different subtypes can meet in the cell of an infected person and mix together their genetic material to create a new hybrid virus (a process similar to sexual reproduction, and sometimes called "viral sex").[2] Many of these new strains do not survive for long, but those that infect more than one person are known as "circulating recombinant forms" or **CRFs**. For example, the CRF A/B is a mixture of subtypes A and B.

The classification of HIV strains into subtypes and CRFs is a complex issue and the definitions are subject to change as new discoveries are made. Some scientists talk about subtypes A1, A2, A3, F1 and F2 instead of A and F, though others regard the former as sub-subtypes.

What about subtypes E and I?

One of the CRFs is called A/E because it is thought to have resulted from hybridization between subtype A and some other "parent" subtype E. However, no one has ever found a pure form of subtype E. Confusingly, many people still refer to the CRF A/E as "subtype E" (in fact it is most correctly called CRF01_AE).[3]

A virus isolated in Cyprus was originally placed in a new subtype I, before being reclassified as a recombinant form A/G/I. It is now thought that this virus represents an even more complex CRF comprised of subtypes A, G, H, K and unclassified regions. The designation "I" is no longer used."

5) In the same interview you further state:

Guccione: *The impression given is this virus is mutating like some kind of monster and there is no vaccinating against it.*

Duesberg: *That is the fantasy of an undergraduate science fiction writer. In a classroom, that's very possible, but in the laboratory of life it's ridiculous. Here's an example of just how restricted the range of HIV mutations really is: antibodies against all strains of HIV detected in all people all over the world were detected because they crossreacted with the same HIV strain Montagnier isolated in 1983*

Question: Although PCR is said to detect conserved regions in the HIV genome, they have had to develop various different PCR tests with different primers to pick up on the alleged different HIV strains. They have also had to modify the antibody tests to cover all the different subtypes now, but there are still uncertainties surrounding for instance variations of subtype O. How do you reconcile this fact with your claim?

Your interview was in 1993, sixteen years ago, and they still haven't managed to develop an HIV vaccine. They quickly develop vaccines against the various flu viruses you claim have a wider scope for recombination, and which, being RNA-viruses, are supposed to have a mutational rate similar to HIV. But they have yet to develop an HIV vaccine (note I am referring to an HIV vaccine, not an AIDS vaccine). How do you reconcile this fact with your claim?

They say the main obstacle is the virus's mutability and variability. They pick it up, they sequence it, and they find enormous differences. Are you saying that they don't actually find those differences?

In conclusion:

Guccione: *John Maddox wrote an editorial in the May 13 issue, saying that your questions are "unanswerable rhetorical questions" and "the stock-in trade of undergraduate debating societies."*

I hope you don't have the same opinion of my questions.

Regards

Claus Jensen

http://www.virusmyth.com/a