The Perth Group's open letter to Professor Jean Umber concerning his article 'What if HIV was simply a natural sign of cellular death (apoptosis)' posted by Crowe on his ARAS website, '(copyright 2007 – Jean Umber and the Alberta Reappraising AIDS Society)' – which is to say, these ideas belong to Jean Umber and David Crowe

May 24th 2010

Dear Professor Umber,

Firstly, we would like to thank you for your offer to financially contribute to the Parenzee hearing. Also to point out that, regrettably, our exchange of information regarding antiretrovirals was very brief. There are two reasons for this.

(1) Our interest in antiretrovirals is secondary. AZT and nevirapine were exceptions. We performed a detailed analysis of these drugs at the request of President Mbeki.

(2) Lately we have been necessarily distracted by dissident politics at the expense of science.

Recently our attention was drawn to a posting of yours entitled "What if HIV was simply a natural signal of cellular death (apoptosis)?" on Crowe's website, " (copyright 2007 – Jean Umber and the Alberta Reappraising AIDS Society)".

Judging by an earlier contribution of yours: "Why did HAART improve the prognosis of AIDS" (translated from the French by you and "edited by David Crowe", it is obvious that you are fully aware of our work. Since Crowe:

(a) professes to be our leader;

(b) not long before your posting we sent him a summary of our work and asked him to post it on the RA website. Although he accepted that our

summary was factual, he declined to publish it on the grounds we were seeking priority.

This being the case you must have known that we:

(1) were the first to put forward the hypothesis and present supporting scientific evidence that the ""HIV" particles and proteins are nothing more than "non-viral material altogether" induced by agents to which the AIDS patients and cultures are exposed…" And that both apoptosis and the "HIV" particles are induced by the same mechanism: oxidation in general and myosin SHs in particular. The latter will lead to myosin-actin interaction, budding and ultimately to particles ("HIV") release. Excessive oxidation, including "endogenous forming of peroxynitrates" is the result of the many oxidising agents, including nitrites, to which the patients belonging to the AIDS risk groups are exposed.[1-3]

(2) were the first to show that both the viral load and the antibodies have nothing to do either with "HIV" or even the "famous microvesicles" released by lymphocytes. [4-8]

(3) were the first to put a unifying drug theory of AIDS (Michael Cullen and John Lauritsen preceded us in regard to AIDS in gay men) and to show "that the real cause of AIDS" is the cellular oxidation induced by drugs and semen to which these patients are exposed.[7 9-12]

(4) have shown that AZT cannot act as a DNA chain terminator. To perform such a role the drug must enter the cell and be triphosphorylated in the presence of reducing equivalents. However the cells of the AIDS patients are oxidised and in addition the drug itself is oxidising, which means it cannot be

triphosphorylated and this is exactly what the evidence shows.

We have shown that AZT toxicity, including inhibition of DNA synthesis, is the result of its oxidative nature: We wrote: "At present, evidence also exists which shows that AZT is rapidly reduced by compounds containing sulphydryl (-SH); that is, AZT oxidises the –SH groups. Ample evidence also exists which shows that oxidation in general (and of –SH in particular) and decreased levels of ATP may lead to many laboratory and clinical abnormalities, including wasting, muscular atrophy, anaemia, damage to the liver and kidney, decreased cellular proliferation, cancer and immunodeficiency. Since patients who are at risk of AIDS are exposed to many oxidising agents and are known to have low –SH levels one would expect AZT to have particularly toxic effects in these individuals – and the sicker the patient the more toxic the drug. That this is the case was accepted by researchers from the National Cancer Institute, Wellcome Laboratories and Abbott Laboratories as far back as 1988: 'Azidothymidine, however, is associated with toxicities that can limit its use…These toxicities are particularly troublesome in patients with established AIDS; the use of azidothymidine is often limited in this population'. Despite these caveats it is possible that, if a thymidine analogue is to be administered to patients with AIDS or to those at risk, at least part of its toxicity may be eliminated by substituting the 3'-OH group with a –SH-group instead of an azido (ºN) group. Yuzhakov et al have performed such experiments and shown that the resulting compound inhibits 'HIV RT'".[6] In other words, in our opinion, to decrease the toxicity of the AZT types of drugs they should be modified and transformed from oxidising to reducing agents. It appears, as you have shown in your elegant work, that the "HIV" experts may have done exactly this with lamivudine.

We were therefore unpleasantly surprised that none of our original work was mentioned, even once, let alone that it was attributed to others.

Regards,

Eleni, Val and the Perth Group

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