Response to Jeanne Bergman re: "House of Numbers" Lies about Research Findings on T Cells Destruction and AIDS*

Eleni Papadopulos-Eleopulos
Valendar F. Turner
John M Papadimitriou
David Causer

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There are several issues raised in Jeanne Bergman's ""House of Numbers" Lies about Research Findings on T Cells Destruction and AIDS". They include the following:

1. The relevance to human AIDS of research done in non-human primates.
2. The role of T4-cells in immunity.
3. The role of T4 cells in the causation of the clinical syndrome, that is, the diseases from which "HIV" positive people die.

Bergman states: "The lynchpin of Brent Leung's argument in "House of Numbers" that HIV does not cause AIDS is the headline of a 2007 [September 26] article on *ScienceDaily.com* that read, "Sudden Loss Of T Cells Is Not Trigger For AIDS, New Study Suggests."…Leung, in a voice-over, intones, "In late 2007, *ScienceDaily* reported that three prominent research teams had published papers in the Journal of Immunology, challenging the theory that the sudden loss of T-cells triggers disease and AIDS." Since T cell destruction is understood to be the primary mechanism by which HIV destroys the immune system, this seems to seriously challenge the HIV/AIDS paradigm…If the sudden loss of T-cells in HIV positive individuals can't explain why people get disease, then there must be co-factors that cause people to get sick and die. Or, factors that have absolutely nothing to do with HIV". According to Bergman *House of Numbers* "did not accurately represent the research: *notably, it failed to mention that the research was done with non-human primates*" (emphasis in original).

If Bergman thinks findings in non-human primates cannot be extrapolated to humans then why have "HIV" experts spent decades doing such research and making their extrapolations? Why have the "HIV" experts been using non-human primates as a model for human AIDS? Why do "HIV" experts spend millions of dollars on an AIDS animal model if the findings cannot be extrapolated to humans? Is their mission to save the non-human primates? If so, from what? They know better than all of us that the non-human primates which are the natural host of SIV do not develop SAIDS while infected either with SIV or "HIV".

Perhaps we can rely on Jeanne Bergman advising Donald Sodora, the senior author of one of the three 2007 *Journal of Immunology* papers, that findings in non-human primates cannot be extrapolated to humans. She might also ask why researchers continue to experiment on scarce non-human primates but ignore the non-infectious model involving repeated, allogeneic immune stimulation reported by Israeli scientists in 1997. "Here we describe a new condition in mice that closely resembles human AIDS, namely, chronic lymphoproliferation with dramatic depletion of CD4-positive cells, progressive impairment of the immune responses, and Kaposi's sarcoma-like tumors or terminal B-lymphomas".[1]

Bergman claims Leung was wrong because the non-human primates "rebound from the T cell destruction caused by the infecting virus, whereas humans generally don't when they are infected

with HIV. Leung also ignored the actual *Journal of Immunology* articles that *Science Daily* linked to – which is remarkable since his entire case against HIV's causality rests on them".

Two weeks earlier, on September 11, *Science Daily* published an article entitled "SIV Infection of Natural Hosts Provides New Insight Into HIV Disease Complexity". In this article Sodora was quoted as saying: "Our assessment of these natural hosts like mangabeys offers insight into the disease and shows us that progression to AIDS likely results from the cumulative effects of HIV/SIV replication, CD4 T-cell depletion, generalized immune activation and non-CD4 T-cells depletion or dysfunction". Obviously Bergman and whoever she is in collaboration with did not read the *Journal of Immunology* papers or the earlier *Science Daily* article. In the latter, Jeffery Milush, the senior author of the paper, was quoted saying: "When we first observed the dramatic CD4 depletion in all the tissues we examined in these monkeys, we were concerned that they might begin to exhibit clinical signs of AIDS…But after more than six years, we are sure that CD4 depletion by itself does not necessarily result in progression to AIDS". Their graphical data show that for the duration of their study, up to 250 weeks, the "dramatic decline" in CD4 cells was permanent. There was no "rebound" – "rebound" is an invention of Bergman's. In their paper Milush, Sodora and their colleagues wrote: "Therefore, these data provide a rationale for investigating multifaceted therapeutic strategies to prevent progression to AIDS, even following dramatic CD4 depletion, such that HIV^+ humans can survive normal life spans analogous to what occurs naturally in SIV^+ mangabeys.[2] Hence, despite Bergman's protestations, the authors did extrapolate their findings to humans. Which means the original *Science Daily* September 26 title and commentary was not at all unreasonable.

At Bergman's request the editorial staff at *Science Daily* revised their September 26 article and also changed the title from "Sudden Loss Of T Cells Is Not Trigger For AIDS, New Study Suggests" to "Progression Of SIV Infection In Monkeys Points To Differences Between Human And Simian Forms Of AIDS." According to Bergman, the revised "summary of the research clarifies the distinction between the virus in humans and simians". What is Bergman talking about? "…the virus" is not the same virus—one is "HIV" and the other is "SIV". And it is not possible to compare AIDS in humans and mangabeys for the simple reason the latter do not develop AIDS.

Before Bergman and her fellow inquisitors rush to also censor the researchers whose work was discussed in *Science Daily* let us remind them that the "evidence-based science" from humans shows that a decrease in T4 cells "does not necessarily result in progression to AIDS", that is, the clinical syndrome. Lymphocyte activation (T4 and T8) is more predictive for the development of AIDS and death than are plasma "HIV" RNA levels or CD4+ lymphocytes.[3 4] "Although Ethiopians not infected with HIV-1 do show signs of persistent immune activation and have lower numbers of naïve cells compared with healthy individuals [and AIDS patients] from the developed world, they do not develop AIDS-like symptoms".[5] Neither do T4 cells protect from the clinical syndrome.[6-10] In fact increase in CD4 cells leads to the clinical AIDS syndrome. "Approximately 10–40% of patients beginning ART with advanced immunodeficiency experience immune restoration disease (IRD)", that is, they develop AIDS indicator diseases. "The timing of these events coincides with increases in CD4 T-cell counts on ART, suggesting that restored immune responses against antigens of viable or non-viable pathogens can be **immunopathological** rather than protective"[11] (emphasis added).

The reasons are simple and were known to both Gallo and Montagnier at the beginning of the AIDS era. Ten years after the discovery of the existence of T4 and T8 cells it became obvious these cells do not have unique immunological functions. In 1983 Zagury (one of Gallo's collaborators) and his colleagues wrote: "Testing functional properties we found that NK activity was mediated not only by T10+ cells but also, in some cases, by T4+ and T8+ cells. Moreover, TCGF production, which may reflect helper activity, was mediated not only by T4+ cells. Only the cytotoxic (CTL) activity seems to be confined to the T8 phenotype. Thus, it appears that T antigens, which seemed to be molecular markers of differentiation, are not markers for terminal differentiation and do not always reflect defined functional properties".[12] Both Gallo and Montagnier were fully aware of Zagury's work.

In 1988 Göran Möller (an immunologist from the University of Stockholm) wrote: "There are three good and several not so good reasons for questioning the existence of suppressor T cells as a separate T cell subpopulation".[13] Commenting on Möller's editorial, researchers from the Pasteur Institute wrote: "It follows that the difference between these two cell populations concerns their *repertoires* and, in consequence, their maturative or activation stages, possibly their differential mechanisms of activation…As discussed here, even primary populations of lymphocytes may follow functional rules in vitro that depart substantially from those operating in vivo, and cells may look and function differently simply because they are either connected or isolated. In essence, and this is both more interesting and difficult to approach, it seems unavoidable that systems (such as the immune) are more than the sum of isolated clonal activities"[14] (emphasis in original). In 2007 "HIV"/AIDS experts from the National Centre in HIV Epidemiology and Clinical Research, University of New South Wales, Australia wrote: "We believe that CD4 count at AIDS diagnosis could be an insensitive indicator of association with immune deficiency".[15]

In a 1981 commentary in JAMA entitled: "OKT3, OKT4, and all that", one reads: "The T- and B-cell measurers – having run through the sick, the elderly, the young, the pregnant, the bereaved – had finally run out of diseases. Each condition was the subject of many reports; so that now, to give but one example, we can conclude with some assurance that T-cell numbers are up, down, or unchanged in old folks….And now it's starting all over again, this time with T-cell subsets. Think, dear reader, and grieve, dear editor, about how many investigators are at this very moment measuring T-cell subsets in systemic lupus erythematosus, in rheumatoid arthritis, in solid tumours (all different sorts – one article for each), in lymphomas, in pneumonia, after surgery, after burns, after trauma, in asthma, in cirrhosis, in Crohn's disease, in glomerulonephritis, in myositis, in familial Mediterranean fever, in leprosy, in Dengue fever, after cardiac transplants, and so on. Meanwhile others will be out measuring blacks, whites, Orientals, native Americans, men, women, children, babies, old folk, astronauts, and laboratory technicians. Cells will be garnered and measured from blood, from lungs, from kidneys, from liver, and from CSF and ascitic fluid…What can be done to staunch the anticipated outflow?…We might legitimately ask, why fight? Why not let us unimaginative immunologists publish to our heart's content? I will ignore the obvious economic arguments for fear that they might be taken seriously. My strongest argument is this: Measurement of T and B cells and their subsets in diseases has no clinical meaning…There is a feeling about that T- and B-cell numbers mean something an immunologic equivalent of an SGOT level or creatinine clearance…Non-immunologists have naturally assume that any subject occupying so much journal space must be relevant in some way – a logical but incorrect assumption".[16] The evidence in the last 25 years, including that from "HIV"/AIDS, amply confirms Goodwin's claim that measurement of T cells and their subsets in AIDS "has no clinical meaning".[17]

After a quarter of a century of the slogan <u>"HIV" infection = T4 destruction (AID) = deadly diseases</u> the top "HIV" experts (the protagonist "foot soldiers", to paraphrase John Moore, are still to wake

up to this), realised that the "evidence-based science" shows that T4 decrease (Acquired Immune Deficiency) does not equal deadly diseases (S), that is, T4 destruction ≠ S. To the contrary, the cause of the deadly diseases(s) is immune activation (stimulation) not immune suppression. In other words, in the history of medicine there has been no other more harmful misnomer than Acquired Immune Deficiency Syndrome (S).

Now the "HIV" experts claim that "HIV" causes immune activation (stimulation) and the equation "HIV" infection = T4 destruction (AID) = deadly diseases has become "HIV" infection = immune stimulation (activation) = deadly diseases. However, despite the development of this new "evidence-based science" the "HIV"/AIDS experts have been reluctant to change the name AIDS to Acquired Immune Stimulation Syndrome (AISS). Why? Nonetheless, they advocate treating these patients with immunosuppressant agents including steroids and cyclosporin A. [18]

The problem for those who still want to promote the notion that "HIV" has been proven to exist and is the causative agent of AIDS/AISS, is that at present there is no "evidence-based science" to prove their claim. To the contrary.

1. At the very beginning of the AIDS/AISS era Montagnier and Gallo accepted that the phenomenon which they claimed signified the existence of "HIV" cannot be detected unless the cells are stimulated. "HIV" cannot be both the cause and the effect of immune activation.
2. Patients belonging to the AIDS risk groups are exposed to a plethora of stimulating agents regardless of "HIV". As far back as 1986 Gallo wrote: "the results revealed a cytopathogenic [cell killing] mechanism that may account for T4 cell depletion in AIDS patients and suggest how repeated antigenic stimulation by infectious agents such as malaria in Africa or by allogenic blood or semen, may be important determinants of the latency period in AIDS".[19] Is it possible that without such antigenic stimulation the latency period may well be infinite in "HIV" "infected" individuals?
3. In 1995 Gallo stated: "The first thing I can tell you is that we've been able to regularly culture from Kaposi's tumors what pathologists say is a tumor cell. We asked: What is the role of HIV in all this? And we found that inflammatory cytokines...were the very likely initiatory events in creating this cell. We said, "Oh, the role of HIV is likely to be in increasing these inflammatory cytokines." But we have learned – this should be of interest to everybody that isn't completely married to HIV – that the inflammatory cytokines are reportedly increased in gay men even without HIV infection. Inflammatory cytokines are usually promoted by immune activation, not by immune suppression. So here was a paradox....So the inflammatory cytokines may be increased by HIV, but I wish I knew what else was increasing them before a gay man was ever infected with HIV. Maybe it's nitric oxide, maybe it's a sexually transmitted virus, maybe it's all of them, maybe it has to do with rimming because it's immune stimulation with non-specific infections".[20]
4. "This study demonstrated for the first time that low preseroconversion numbers of CD4 T cells and increased levels of immune activation were associated with an increased risk to develop AIDS after seroconversion...In conclusion, our data show that chronic immune activation and the size of the CD4 T cell pool are critical factors in HIV-1 pathogenesis, even when measured before seroconversion" [21] (emphasis added).

*www.houseofnumbers.org/HoN_Lies_about_T_Cells.html

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