generated from a linear model could be a function of both the baseline CD4 level and viral loads. Further studies with larger sample sizes are needed to resolve these discrepancies.

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HIV an illusion

SIR — In an editorial[1] in the 19 January issue of *Nature*, John Maddox invited "Duesberg and his associates" to comment on the "HIV-1 dynamics" papers published the previous week, indicating that these new results should prove an embarrassment to us. Although we do not think that a scientist should be embarrassed for pointing out inconsistencies and paradoxes in a hypothesis that have only been reportedly resolved 10 years later, we nonetheless prepared a fully referenced, approximately 2,000-word critique of the Ho *et al.*[2] and Wei *et al.*[3] papers that we believed met the criteria of "not being longer than it needs to be, and pertaining to the papers at hand" that Maddox set out in his widely read challenge.

Unfortunately, he did not share our view and agreed to publish only a radically shortened version, and only after he had personally "gone over it with a fine-tooth comb" to remove our perceived misrepresentations of the issues. We found these new conditions so totally at variance with the spirit of free and fair scientific debate that we could not agree to them.

Readers of *Nature* who are interested in these questions, and feel that they do not need to be protected by Maddox from our ill-conceived logic, can find the complete text of our commentary in the monograph supplement to the most recent issue of *Genetica*[4]. Here we would point out only that the central claim of the Ho *et al.*[2] and Wei *et al.*[3] papers — that 10^5 HIV virions per ml plasma can be detected in AIDS patients with various nucleic-acid amplification assays — is misleading. The senior author of the Wei *et al.* paper has previously claimed that the PCR method they used overestimates by at least 60,000 times the real titre of infectious HIV[5]: 100,000/60,000 is 1.7 infectious HIVs per ml, hardly the "virological mayhem" alluded to by Wain-Hobson[6]. Further, Ho and a different group of collaborators have just shown[7] that more than 10,000 "plasma virions", detected by the branched-DNA amplification assay used in their *Nature* paper, correspond to less than one (!) infectious virus per ml. And infectious units, after all, are the only clinically relevant criteria for a viral pathogen.

Finally, in view of Wain-Hobson's statement[6] that "the concordance of their [Wei and Ho's] data is remarkable", note that Loveday *et al.*[8] report the use of a PCR-based assay and find only 200 HIV "virion RNAs" per ml of serum of AIDS patients — 1,000 times less than Ho and Wei. So much for the "remarkable concordance".

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■ Peter Duesberg was offered space in Scientific Correspondence for 500 words of his own choice, but declined. — Editor, Scientific Correspondence.

SIR — The most recent AIDS press releases published as two "scientific articles" in your magazine[1,2] indeed are the most clever and, if possible, the most deadly yet! Since March 1987, when Duesberg first published his critique of retrovirology[3] and his proof that the "AIDS virus" is not sufficient to cause AIDS, the AIDS/HIV entrepreneurial gaggle have railed against him both professionally and financially. Rather than test Duesberg's hypothesis or for that matter fund him to test his own hypothesis, you change your model, which has not saved a single life, to suit your own purposes, from HIV kills CD4 cells directly, to HIV kills cells indirectly, and now back to the latest direct kill model of Wei *et al.*[1] and Ho *et al.*[2].

Wei *et al.*[1] and Ho *et al.*[2] describe a viraemia that on administration of certain drugs drops exponentially and raises CD4 counts exponentially, seemingly without a trace of "mayhem". What is this viraemia of billions of RNA particles that can only be seen with an undocumented branch-PCR or PCR but not with a functional infectivity test? How do the authors know that these RNA particles are infectious and not defective? Is it reverse transcriptase activity? Is it some protein that is unique to the quasispecies HIV? The tests for HIV are all flawed. What is this free virus? How does one have free virus after immune response? Are not symptomatic people with AIDS HIV-positive, that is, don't they have antibodies against HIV? So what is free virus?

The Wei *et al.* and Ho *et al.* models do not account for other probable immune activity and the uniqueness of each individual. Haynes and Fauci showed in 1978 that hydrocortisone selectively caused CD4 cells to hide in tissue. Indeed, what effect do multiple drugs have on CD4 cells? The notion of clearance of one billion CD4 cells per day versus one billion virions per day is not defined or addressed, nor can it be! No one else has access to either the unapproved drugs or the branch PCR technology! What is the benchmark rate for the turnover of CD4 cells in the general population?

To counter the 42 case studies of Wei *et al.*[1] and Ho *et al.*[2], we at HEAL (Health Education AIDS Liason) can provide at least 42 people who are western-blot-positive for 'HIV', have low T4 cells, who are not using orthodox procedures, and have been healthy for years! On the other hand, we can also provide you with hundreds of 'HIV$^+$' people with high T4 cells, who were then hospitalized with opportunistic diseases! Not to mention the thousands of AIDS cases without HIV which were conveniently renamed idiopathic CD lymphocytopenia.

We at HEAL maintain that what is called AIDS is no more complicated than a recreational drug–fear–medical drug disease syndrome which, with the exception of acute medical care, is out of the purview of orthodox medicine.

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Toxic shock

SIR — The recovery rate of CD4$^+$ T cells reported by Wei *et al.*[1] and Ho *et al.*[2] in HIV patients following therapy is remarkably similar to that observed in another acute clinical condition. We have documented lymphocyte counts and proportions of CD4 cells double-staining for CD45 isoforms in three patients during episodes of toxic shock syndrome and one following toxic shock-like syndrome.

In these clinically defined conditions, bacterial superantigen initially induces massive T-cell and monocyte activation. There is subsequent apoptosis of the activated CD4$^+$ cells, during which patients demonstrate a lymphopenia[3], followed by the return to normality of the lymphocyte count as patients recuperate. The mean rate of change in the counts in the first 2 weeks of recovery was 2.1, which compares with 2.0 (ref. 1) and 1.8 (ref. 2).

The similarity in rates of recovery suggest that this rate has some value as a common measure of maximal CD4 T-cell output to the circulation in adults. Slower rates may be observed, for instance, in patients following radiotherapy or chemotherapy[4]. This rate decreases with time; it took one patient 14 months to reach a final CD4$^+$ lymphocyte count of 1.4×10^9 cells cm^{-3}. The nature or mechanisms of the brake on this rapid prolifer-