RESPONSE TO THE “THE HIV PUZZLE” COMMENTS

THE PERTH GROUP
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www.theperthgroup.com

We agree with Juliane Sacher, Felix de Fries and Michael Ellner that stress and antibiotics may have some contributory role in AIDS. In fact we have pointed out this fact for decades in our scientific publications and given good scientific reasons for it. However, they are not the main cause of AIDS, in any AIDS risk group. If stress were to be the cause, one would expect the beginning of the AIDS era to be about the time of the Stonewall riots when the gay men were oppressed and under considerable stress. In a short debate we had recently with Michael Ellner he agreed with us that sex and in particular semen play a key role. In his recent interview with Nicole Zwiren, Terry Michael also seems to agree that sex and gut trauma and flora play a role in AIDS. There is no doubt that gut flora plays a role in health and disease and that in “HIV”/AIDS patients the flora is abnormal. However, as we pointed out to Tony Lance some time ago, the question is what causes the abnormality? Unless one knows the cause and the mechanism, there will be only preventive measures and that will be abstinence. However, our view regarding the relationship between sex (homo/hetero) and pathology not only predicts ways of AIDS prevention and treatment but also in our view is the only way to lessen homophobia in general and avoid a backlash against gay men, if and when “HIV” is accepted not to be the cause of AIDS or, as Camille Paglia puts it, when the world economy destabilises, in particular. (See http://www.theperthgroup.com/PG_Statement.pdf).

Gay men face two conceptually different theories:

(i) the existence of “HIV” and its causative role in AIDS. However, since it is accepted that at present only blacks in sub-Saharan Africa and Papua New Guinea and gay men are affected by "HIV"/AIDS, it means that white gay men are different than white heterosexuals

or

(ii) semen is a strong biological oxidant and thus toxic. However,

(a) unlike “HIV”, a high exposure over time is necessary for semen toxicity to manifest;

(b) unlike “HIV”, semen does not discriminate. Everybody, gay, hetero, black, white, is equal, everybody is the same.

There is no doubt that “a conference that is dealing with this issue exclusively” (antibiotics and stress) as Torsten proposed will be very interesting. However, we cannot see such a meeting having a significant impact on the dissidents’ aim.

As far as we know the aim of all the dissidents, and thus what unites us all, is the scientific reappraisal of the “HIV” theory of AIDS, a theory accepted as fact since 1984.
There are some scientific principles which no scientist can afford to ignore. They include the following. Before a scientist puts forward a new theory, he/she must first address the failures of the old.

It goes without saying that any analysis of the “HIV” theory of AIDS will have to start with “HIV”, and that is exactly what the dissidents have done. Unfortunately the conclusion divided the dissidents in two. Some concluded that “HIV” has been proven to exist but is harmless, others that “HIV” has never been proven to exist.

In an email he sent to Torsten, copied to us, commenting on the “The HIV Puzzle”, Terry Michael wrote: “I have been trying for a long time to help end the divisive tone of the exchanges between those who believe “The Virus” has not been isolated and those who think there is some isolated genetic code, but it is just a harmless passenger.

To the world outside those camps, that difference of scientific opinion has little relevance for the health of human beings, who are my first concern. If “it” is not pathogenic, in the end why do we waste so much energy debating the difference of opinion? I respect both “sides” of the debate, and can understand their arguments, but I wish we could focus our energy on debating with the HIV=AIDS Cult instead of each other.”

However;

(1) The “harmless passenger” is a virus. That means it is a particle, not “some isolated genetic code”. So far nobody has claimed the cause of AIDS is a “genetic code”.

(2) In science what matters is facts, and the facts of “both ‘sides’ of the debate” cannot be correct. And the facts are of great “relevance for the health of human beings”.

(3) No debate has ever taken place regarding “the difference of opinion”. We have spent a lot of energy asking for one. All in vain.

The situation is not much different from twelve years ago when Fintan Dunne wrote: “Is the dissident movement aping the tactics of the orthodoxy by suppressing dissent among their own?...How ironic that the same exclusionary tactics that have dogged the dissident movement in their dealings with the orthodoxy may bedevil the movement itself”.

(4) We agree that we (the dissidents) should spend “our energy on debating with the HIV=AIDS Cult instead of each other”. However, the dissidents must have arguments which cannot be rebutted, otherwise the debate will have “little relevance for the health of human beings”, and may even end up being harmful. In this regard the “difference of scientific opinion” between the two groups is of great “relevance for the health of human beings”.

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From the very beginning it was obvious that the dissidents were not united in their scientific views. However, because we thought such division would be detrimental to the movement, we kept our own counsel. The members of the group for the scientific reappraisal of the “HIV”/AIDS hypothesis, especially Peter Duesberg were talking, and people including the late Sir John Maddox were listening and responding. However, it got us nowhere. By the mid 1990s, thanks to Continuum and to Peter, it became public knowledge that as far as the existence of “HIV” is concerned the dissidents were divided. At present there are three views on “HIV”:

(1) “HIV” has been proven to exist and to be the cause of AIDS;

“HIV” infection → decrease in T4 cells (immune deficiency) → diseases → death (at present, this view is shared by at least one “dissident” – Crowe). According to the "HIV" experts, including Montagnier, the best way to prevent and treat AIDS is with antiretroviral drugs. And this has been the case for 25 years.

(2) “HIV” has been proven to exist but is harmless.

There are three main problems with this claim:

(a) To date nobody has published evidence which proves the existence of “HIV” (see below);

(b) Once one concedes “HIV” isolation/purification and thus the existence of “HIV”, “HIV” proteins, antibodies, nucleic acid, sexual transmission and a correlation between “HIV” and AIDS, then it is very easy to rebut the harmless passenger virus arguments. And it has been done repeatedly by the “HIV” experts. We can do it. Eugene Semon stated recently: “I’ve come to the position where I can argue the HIV causes AIDS view as well as an HIV expert. If David’s flirted with that somehow, at this point I say – why not.” In fact Crowe made it RA policy;

(c) Commenting on “The HIV Puzzle” and the harmless passenger view, Fabio Franchi wrote: “Another fact against him is that many who followed Duesberg’s indications are dead (apart from inviting all to avoid drugs, Duesberg did not offer much)...I had remained almost disoriented in my profession for the discreet inability of forecast of Duesberg’s theory. A good theory must make good prediction (if not, where is its usefulness?)”.

Indeed, the proponents of the harmless virus view have never made any prediction regarding AIDS prevention (apart for advising not to take drugs) and treatment. Treating the AIDS indicator diseases in the same way as in non-AIDS patients does not solve the main problem, i.e. the underlying cause, “the terrain”.

(3) “HIV” has not been proven to exist.
This argument boils down to one electron micrograph. Yes, just one picture taken with the electron microscope. Tony Morrison, a boxer, realised it as soon as he became aware of the dissidents.

According to all virologists and retrovirologists, including Montagnier, Gallo and Barre-Sinoussi, to prove the existence of a retrovirus it is absolutely necessary but not sufficient to purify the virus particles. (See The Emperor’s New Virus? – In a nutshell. http://www.theperthgroup.com/OTHER/ENVCommentary.pdf).

Or as Montagnier put it, to prove the existence of a real virus one must purify the particles. No purification, no virus. They also agree that at least since the 1970s the method of choice for retroviral purification has been banding in density gradients. And the only way to prove purification to the rest of the world is by publishing a picture of the purified particles. Banding in density gradients is the method which Gallo used to prove the existence of the first human retrovirus, HTLV-1, as well as “HIV”. Likewise Montagnier to prove "HIV". Both claimed to have proven the existence of “HIV” by obtaining “purified” virus particles.

Gallo published an EM picture for HTLV-I but not for “HIV”. As Eugene Semon pointed out: “If anyone bothers to read all FOUR* papers that I’m recommending – there’s clearly an isolation standard covering all retroviruses. It’s my non court-qualified expert opinion of course but Gallo, starting with HTLV-I, clearly was following this standard. He had to cut corners after negative results, i.e. commit fraud in May 84 paper after Madame Secretary cast the die for him in April. And then redefine purification at Parenzee which looks like perjury to me”.

Fourteen years after Montagnier claimed to have proven the existence of “HIV” by purifying the particles he gave the reason why no pictures of the “purified” virus were published. When asked by Djamel Tahi why they did not publish an EM of the purified virus he said that in the “purified” virus they “saw some particles but they did not have the morphology typical of retroviruses. They were very different”. Charles Dauguet, the Pasteur Institute EM specialist, confirmed that all they had in what they claimed to be “purified” virus was nothing but cellular debris.

Since proof for the existence ≡ purification.

No proof for purification ≡ no proof for existence.

Terry says that stress plays a causative role in AIDS. Everybody agrees that a positive “HIV” test for infection with the virus is obviously stressful, not only to the patient, but relatives and friends as well. And this applies even to those who believe that “HIV” is not the cause of AIDS.

In 2008/09, in the Duesberg softspot debate, we repeatedly asked Crowe the following questions:
• Since the “HIV” experts, including Montagnier and Gallo, admit:
  
o  To prove the existence of a virus, it is necessary to purify the particles and to show that they have unique RNA;
  
o  To date no “HIV” experts, including Montagnier and Gallo, have proof of purification and admit that there is no unique RNA:

why should the dissidents give to the “HIV” experts that which they admit they do not have and debate with half-truths?

• Is it possible for the dissidents to be proven correct by debating with half-truths?

Crowe never replied to us. Recently, when Anthony Brink repeated the question, Crowe, who claims to lead the dissidents in their arguments with the “HIV” experts against the “HIV” = AIDS trap, responded: “They also imply that there is such a thing as absolute truth and even rethinkers don’t have access to that. It also implies that the mainstream are actively debating dissidents which has only very rarely been the case”. (Can anybody think of a better debate than the Parenzee case which Crowe sabotaged?)

The question is, do “HIV” and AIDS patients, their relatives and friends, think that the existence/non-existence of “HIV” affects their everyday lives no more than the existence/non-existence of the absolute truth? What reason do we have to accept the existence of “HIV” if no such proof exists? Would its acceptence benefit “the health of human beings”? If yes, how? If not, for whose benefit will the dissidents have to accept its existence? Is it scientifically and ethically justifiable?

In 1988 we (EPE) published a paper in Medical Hypotheses entitled: Reappraisal of AIDS: Is the oxidation induced by the risk factors the primary cause? In this paper we put forward the redox theory of “HIV”/AIDS and made Montagnier personally aware of it in 1991. According to this theory what is said to be “HIV” is nothing more than phenomena induced by the oxidation to which the cultures and the AIDS patients and those at risk are subjected. In this regard it is sufficient to mention:

(i) Both Montagnier and Gallo accept that neither the phenomena (“HIV”) nor the “HIV” effects can be detected unless the cultures are activated (stimulated). However, we have been presenting evidence since even before the AIDS era that activation (immune stimulation i.e. cellular division, cytokine production) is an oxidative phenomenon;

(ii) Our 1991 correspondence with Anthony Fauci (see Fauci’s letter attached and our comments below).

In his comment Liam Scheff said: “Stop the bloody navel gazing. Work to get rid of “HIV” tests”.
The only “‘HIV’ tests” routinely used are the antibody tests.

If there is “‘HIV’” there are “‘HIV’” antibodies. If there are antibodies, there are tests, no matter how non-specific they are. No antibody test is 100% specific. Furthermore, as Fabio Franchi pointed out, in his practice “Almost, if not all [AIDS patients] were HIV positive”.

So why should you get rid of a test which has clinical meaning, especially if you know it has nothing to do with “‘HIV’”? But as we pointed out in 1988 has everything to do with oxidation?

Over the years we have published extensively on the subject and have shown, as mentioned in “The HIV Puzzle”, that the “‘HIV’” antibodies are nothing more than autoantibodies and cross-reacting antibodies.

Liam also wrote: “Help people who have immune deficiency”.

By “immune deficiency” the “‘HIV’” experts, as well as nearly all dissidents including those who promote the harmless virus, mean decrease in T4 cells or even changes in the Th1/Th2 subset ratio.

In the 1988 paper we predicted, among other things, that the decrease in T4 cells may not be due to a killing by “‘HIV’” or any other agent but by decreased binding of the CD4 antibody to the cells resulting from changes to their surface by oxidation. Later on, no less an authority than Baltimore said that the decrease in binding resulted from “down regulation” of the CD4 receptor. In numerous subsequent publications we presented evidence which proved our prediction. There is a reciprocal change between T4 and T8 cells resulting from a decreased binding of the CD4 antibody and increased binding of the CD8 antibody, the sum T4 + T8 remaining constant.

In 1994 our prediction was proved experimentally by researchers from Germany when they showed that the number of T4 cells is defined by the redox. In fact as predicted by the redox theory of cellular structure and function they showed that both too much reduction or oxidation leads to decrease of T4 cells.

More importantly we have shown that the T4 cells, and thus their subsets, play no role in the development of the AIDS indicator diseases. At present the vast majority of “‘HIV’” experts, including Fauci accept that this indeed is the case, and claim that the syndrome is due to immune activation not immune suppression, something which we, and even Gallo and Montagnier, were saying at the beginning of the AIDS era. See:


As already mentioned, unlike them, we claimed that activation (immune stimulation) is caused by the oxidation to which the patients belonging to the AIDS risk groups are subjected.

Among the many other predictions in this paper (all of which have been fulfilled) the two most important and bold regarding “the health of human beings” are:

(i) the tissues of AIDS patients and those at risk will be oxidised in general, and in particular will have low sulphydryl (SH) groups levels.

In 1989, for some unknown reason, Eck, Dröge and their colleagues measured the redox state of AIDS patients. Since everybody, including Dröge, considered the redox was defined by GSH one would have expected them to have determined the GSH levels. Instead they measured the sulphhydryl (SH) level of the acid soluble proteins. Since in our view we were the only people until then (we will gladly accept correction) to claim that the cellular redox is defined by the SH level of the acid soluble proteins, and to consider them extremely clinical significant we found their findings very interesting. They reported that the level of the acid soluble SH was significantly decreased in AIDS patients and those at risk. In 1991 Dröge wrote in Lancet: “…we do agree with Papadopulos-Eleopulos and colleagues on the basic interpretation that a distorted balance of oxidants and antioxidants may play a key part in the immunopathology of HIV/SIV infection.”

(ii) AIDS can be prevented and treated by the use of antioxidants in general and SH groups containing compounds, in particular.

In 1992 in Research in Immunology, a Pasteur Institute publication, we wrote: “As long ago as 1983, one of us (E.P.-E.) proposed that oxidative mechanisms are of critical significance in the genesis of AIDS (acquired immune deficiency syndrome). A prediction of this hypothesis was that the mechanisms responsible for AIDS could be reversed by the administration of reducing agents, especially those containing sulphhydryl groups (SH groups). The discovery of HIV resulted in a broadening of this hypothesis in that it considered oxidative stress as a principal mechanism in both the development of AIDS and expression of HIV (Papadopulos-Eleopulos, 1988; Papadopulos-Eleopulos et al., 1989). However, the general acceptance of the HIV hypothesis of AIDS completely overshadowed this alternative hypothesis, and although many other scientists have questioned the role of HIV in the causation of AIDS (Duesberg, 1987; Root-Bernstein, 1990) Robert Gallo and most AIDS researchers consider HIV to be the sole “sine qua non” cause of AIDS….Because of the possible therapeutic implications of reducing agents in AIDS patients it is important to have a basic understanding as to why:

– reducing agents suppress the expression of HIV:
asymptomatic HIV-infected individuals and AIDS patients have decreased sulphydryl and total glutathione levels.

AIDS patients suffer from many opportunistic microorganisms. Like all cells, these microorganisms require reducing equivalents, including SH, for division and survival (Papadopulos-Eleopulos, 1982) which they obtain to the detriment of body tissues. In AIDS patients, a decrease in the level of SH may also result from malnutrition and diarrhoea. However, opportunistic infections, diarrhoea and malnutrition cannot account for the low level of GSH and acid-soluble SH found in HIV-positive, symptom-free, well-nourished homosexuals and haemophiliacs…At first sight it appears that there is no common factor, apart from HIV infection, linking the various AIDS risk groups. However, homosexuals are exposed to relatively high levels of nitrites and anally deposited sperm, drug abusers to opiates and nitrites, haemophiliacs to factor VIII. All these are known potent oxidising agents which oxidise many cellular reducing equivalents such as NADPH and all sulphydryl groups, including those of cysteine (acid soluble thiols) (Papadopulos-Eleopulos, 1988)…Malnutrition and diarrhoea may also lead to cysteine, magnesium acid ATP deficiency…The oxidative stress to which the AIDS patients are subjected would lead to cellular anomalies in many cells, including lymphocytes, resulting in opportunistic infection, immunological abnormalities and neoplasia.

All this argues in favour of oxidation as being a critical factor in the pathogenesis of AIDS and HIV expression”.

The 1988 Medical Hypotheses paper was first submitted to Nature in 1986, but rejected twice. The 10th of July 1986 letter of re-submission ended with the following: “If my paper does nothing other than draw attention to the oxidative nature of the risk factors and its biological importance, then it offers what is so far the only hope of treatment which will arrest and reverse the otherwise invariable fatal course of the disease. In my opinion this alone will more than justify its publication”.

To date there has been only one clinical trial where the authors determined the relationship between the level of SH groups and mortality as well as their replenishment by the use of the SH compound NAC. Although the trial was not ideally designed, the others found that “…by all measures, GSB [reduced glutathione, GSH] (in CD4T cells) emerges as a powerful yardstick for predicting survival in HIV infection”.

Let us repeat our comments about this trial from “Looking back on the oxidative stress theory of AIDS”, Continuum, Vol. 5, No. 5, 1998/99. “The best confirmation of this comes from researchers at Stanford University, USA. In 1997 discussing their results they wrote: ‘In essence, we have shown that GSH levels are lower in subjects with CD4 T cell counts below 200/ml (CD4 <200) than in subjects at earlier stages of HIV disease; that among subjects with CD4 <200 lower levels of GSB (a FACS
measure of GSH in CD4T cells) predict decreased survival; and that the probability of surviving 2-3 years increases dramatically as GSB levels approach normal range. In addition, we have presented preliminary evidence suggesting that oral administration of NAC, which supplies the cysteine required to replenish GSH, may be associated with improved survival of subjects with very low GSH levels (GSH reduced glutathione).

Last year they stated: ‘We have shown that GSH depletion is associated with impaired survival; the greater the depletion, the worse the prospects for survival…By replenishing GSH, NAC or other agents we may be able to modulate such adverse effects of GSH depletion.’ However, although the authors are most probably aware of our work (the publications of the Perth group were sent to them a few years ago and are indexed in Medline under oxidative stress), for some unknown reason, they state: ‘HIV-infected individuals would be better served if we could identify the mechanism that underlines the GSH depletion and intervene, if possible, to prevent its occurrence’. The best advice they can give in this regard is: ‘It may be prudent for those individuals to avoid excessive exposure to UV irradiation and unnecessary use of drugs that can deplete GSH – e.g. alcohol and prescription or over-the-counter formulations containing acetaminophen [paracetamol].’

Although nobody gives us credit the proponents of the “HIV” theory of AIDS, Montagnier in particular, took notice of our theory and tried to make a hybrid “HIV”/redox theory.

However, our work has been totally ignored by the dissidents. In fact Crowe went as far as to ascribe the oxidative “idea” to Montagnier. Even if Montagnier has become an apologist for our oxidative theory of AIDS, it has “little relevance for the health of human beings”. He advocates the use of antioxidants to combat the oxidation induced by antiretrovirals, and even then only for a few months then followed by ART as usual.

In his article “The AIDS war on breastfeeding” Crowe wrote: “It is depressing to think how much better off the world would have been, how many babies would be alive and how many mothers would have been spared the agony of watching their baby die in front of them, if only all those PhDs, MDs, RNs and other educated people who have swallowed the HIV=AIDS theory for all these years, had simply done nothing, if the world had paid them to stay at home rather than coming to work and promoting and managing the deadly formula feeding programs of the past decade.” (If one accepts that “HIV” causes AIDS, as Crowe does, the South African breastfeeding policy was a good evidence-based policy).

Can you, Terry and Liam, imagine how many babies, Africans, gay men, heterosexuals and comrades in arms, would be alive today if the dissidents, and in particular the leaders, accepted our theory, as the “HIV” experts have done, that:

(a) “oxidation and anti-oxidation may be critical factors in the control of virus expression” – Fauci;

(b) “distorted balance of oxidants and antioxidants may play a key part in the immunopathology of HIV/SIV infection” – Dröge;
(c) “GSH depletion is associated with impaired survival, the greater the depletion the worse the prospect for survival” – Herzenberg:

and made nutrition, antioxidants (administration under proper supervision, in collaboration with laboratories which can measure the redox) and reduced exposure to oxidising risk factors (drugs, recreational and prescribed, including antibiotics, semen, factor VIII) the official dissident policy for AIDS prevention and treatment?
Dear Dr. Fauci,


Although your findings were no surprise to us, we were suitably impressed by your well executed experimental work. You and your colleagues are apparently unaware of work published by us in which we, in addition to predicting your experimental results, advocate the use of antioxidants in the prevention and treatment of AIDS and also, based on well known facts, hypothesise:

(a) the mechanism which "produces a striking decrease in the level of a major cellular antioxidant" in AIDS patients and those at risk of developing it,

(b) the mechanism of retroviral induction.

Please find enclosed our published work and a letter, which my colleagues, Dr. V.F. Turner, Prof. J.M. Papadimitriou and I submitted for publication in Lancet, February 1990 which was rejected. In it we give the causes and a mechanism for the decrease in cellular sulphhydryl groups, GSH and total glutathione in AIDS patients and those at risk of AIDS.

We would be very grateful if, after reading our papers, you could find the extra time to give us your comments and criticism.

Yours sincerely,

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August 2, 1991

Dr. Eleni Eleopulos
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Dear Dr. Eleopulos:

I have read with interest your manuscript on the role of oxidation in the pathogenesis of HIV infection. I certainly agree with your general hypothesis that oxidation and anti-oxidation may be critical factors in the control of virus expression as well as in determining certain systemic dysfunctions associated with HIV infection. However, I suggest that you modify your penultimate sentence "... HIV cannot be detected unless strong oxidants ... are added to the culture ..." because it is an overstatement. First, it is not demonstrated that the mechanism through which 5-iodo-2-deoxyuridine and PHA are increasing the expression or replication of HIV is oxidation. At least for PHA, the most important functional explanation is its ability to induce IL-2 receptor expression on the target cells. Second, HIV expression or replication can be easily obtained in different cell types (for example, macrophages) in the absence of oxidants or PHA. In general, without underemphasizing the potential role of oxidation in HIV infection in vitro and in vivo, I think that much more needs to be understood in terms of the actual mechanism of action of these agents. As you have certainly noted, in our manuscript published in PNAS, we underscore the fact that NAC has a different, more profound suppressive effect than glutathione or glutathione monooester on HIV expression in our cell systems, suggesting that the most important component of NAC effect may not be, as postulated, just increasing the intracellular levels of glutathione.

I certainly appreciate your interest in our research and I will be available for further discussion on this important matter. Thank you and best regards.

Sincerely,

[Signature]

Anthony S. Fauci, M.D.
Director
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and Infectious Diseases
Comments on Anthony Fauci’s letter.

If oxidation is a “critical” factor in “virus expression” and system dysfunctions, then it follows that “HIV” infection and thus its effects should be treated with antioxidants, which are readily available and cheap;

(a) Both, 2-iodo-2-deoxyuridine and PHA are oxidising agents, the former a strong oxidant. As far back as 1998 a colleague of Peter Duesberg, Chandan Sen, has shown that “H2O2 [an oxidant] induced NF-kB activation and NF-kB directed IL-2 receptor expression”, in other words the IL-2 receptor expression is induced by oxidation;

(b) We have never said that GSH is the key cellular redox determinant. Even before the AIDS era we claimed that the cellular redox is determined by the redox of its proteins, namely the acid soluble proteins. The main role of GSH is to reduce these proteins.

Terry Michael’s request.

Terry sent his comments to Torsten and us. We asked him if we could make them public. He sent us the following response:

“You can make the response “public,” as long as there is no implication that I am taking “sides” in a debate with David Crowe and others. But frankly, I cannot follow the arguments you are making. I will just tell you what my research has led ME to believe, which is all I can take responsibility for. My view is that (1) numerous and repeated exposures to old pathogens; (2) historic levels of ingestion of toxins, including alcohol, poppers, controlled substances, anti-biotics and eventually the ARV’s; and (3) the extreme stress felt by gay men from religious right-wing prejudice in the U.S., social dislocation, gay ghetto-izing in urban enclaves, and the AIDS scare itself ALL created the “perfect storm” that comprise the MULTIFACTORIAL explanation for the breakdown of a subset of immune systems among gay men in the early 1980’s. I do not believe there is a single explanation, 19th Century germ theory or otherwise, for the syndrome, and the same is true for what is claimed as heterosexual AIDS in Africa.”