Analysis of Alexandra Levine's December Declaration In Support of GlaxoSmithKline LLC's Motion For Summary Judgment.

Impressions and responses of paragraphs 2-15 of Dr. Levine's declaration:

Dr. Levine's credentials appear to be extremely impressive. Her experience as a "clinical researcher" with both AIDS and cancer are made to appear vast. Yet in the view of someone who has also been involved in the fight against these same two diseases for a similar length of time and with similar intensity and commitment, I must confess that the impressive list of political accomplishments and career advancements with numerous policy-forming task forces or affiliations and organizations listed in Dr. Levine's extensive autobiography in paragraphs 2-14 are inconsistent with the actual contributions made by those organizations and efforts she identifies herself with, and with which she has been associated. Her Curriculum Vitae as it is presented in the first 14 paragraphs are presented in such a way, in addition, so as to give the impression that far more information is known about cancer and AIDS than is actually known, and that near universal consensus has been reached about the science and medicine of both cancer and AIDS than actually exists, and that more progress has been made regarding the identification, treatment, or reversal of these syndromes than has ever been accomplished anywhere on earth. While normally, such extensive affiliations with so many politically powerful connections and organizations for a long period of time would qualify someone such as Dr. Levine more than most to be able to point out the known shortcomings of all these agencies and their efforts to quell or reverse AIDS or cancers, she chooses not to do this, but instead paints a picture of certainty where in fact little if any certainty exists. This criticism comes to light through consideration of the following subtleties and wording in Dr. Levine's claims regarding her credentials:

In paragraph 2:

For instance, the statement and wording claiming:

"I am the Chief Medical Officer, and Professor of Hemotology at City of Hope National Medical Center which one of only 40 National Cancer Institute (NCI) designated Comprehensive Cancer Centers in the US, and known for the research and treatment of cancer…."

This emphasis creates an impression to the laymen, but not to those experienced with clinical trails or comprehensive cancer centers, or those who work or teach at institutions that either are or are not "NCI-designated," that such places have a special knowledge or success rate that other non-NCI-designated institutions don't possess. For example, there is nothing particularly unique or special about NCI designation that qualifies these places as anything special, or more capable of treating two largely incurable diseases such as malignant cancer or profound immune suppression, as Dr. Levine would have us believe by her emphasis of the fact that she works at an "NCI-designated" place. Both cancer and AIDS patients are still dying in these places as well as everywhere else.

In paragraphs 5, 6:

While Dr. Levine continues to present her credentials in paragraphs 5 and 6 regarding her experiences with cancer and AIDS and her other affiliations and experiences for some 9 additional paragraphs, again, much of it appears to be misleading. For instance, in paragraph 5 Dr. Levine claims that:

"I have treated thousands of patients with those illnesses,"

and, in paragraph 6,

"I have diagnosed and treated hundreds of patients with HIV/AIDS, and have prescribed AZT many hundreds of times,"

Such statements give no indication regarding how those hundreds or thousands of patients responded to Dr. Levine's treatments, theories, or drug experimentation on them, and lacking this kind of information, these statements have no meaning, and are stated to perhaps give the [false] impression that either the hundreds or thousands of patients she variously claimed to have treated, have somehow objectively benefited by her diagnoses and treatments (through the criteria of evidence-based medicine). Similarly, in paragraph 7, which recounts her appointment(s) during the Clinton years, her FDA and NCI advisory appointments, and the rest she mentions, it is now widely appreciated that these efforts were often surrounded by secrecy during the Clinton Presidency when HIV/AIDS was declared to be a "Issue of National Security." The peer-reviewed scientific and medical literature testifies as to why these lofty appointments and advisory roles have meant little in either the "war on cancer" or "the war on AIDS," and simply to mention them as evidence of some special knowledge about these syndromes is misleading, without pointing out their utter failure to reverse either syndrome in advanced stages. The same criticisms can be summoned regarding her claims regarding her vast experience with clinical trials in the context of AIDS.

In contrast to Dr. Levine's claims regarding how her credentials and experiences uniquely provide her with special knowledge regarding cancer or AIDS, most researchers actively engaged in research and development of new diagnostics and treatments for these syndromes are painfully aware of information such as the recent position statement of innovators such as Dr. Richard Ablin, the discoverer of PSA (prostate specific antigen), who, just this year, wrote an op-ed article in the New York Times pointing to the true state of knowledge regarding this cancer, with such information as:

"*The* [PSA*] test's popularity has led to a hugely expensive public health disaster. It's an issue I am painfully familiar with — I discovered P.S.A. in 1970. As Congress searches for ways to cut costs in our health care system, a significant savings could come from changing the way the antigen is used to screen for prostate cancer. Americans spend an enormous amount testing for prostate cancer. The annual bill for P.S.A. screening is at least $3 billion, with much of it paid for by Medicare and the Veterans Administration."*

"…the test is hardly more effective than a coin toss. As I've been trying to make clear for many years now, P.S.A. testing can't detect prostate cancer and, more important, it can't distinguish between the two types of prostate cancer — the one that will kill you and the one that won't…"

… The medical community is slowly turning against P.S.A. screening. Last year, The New England Journal of Medicine published results from the two largest studies of the screening procedure, one in Europe and one in the United States. The results from the American study show that over a period of 7 to 10 years, screening did not reduce the death rate in men 55 and over. The European study showed a small decline in death rates, but also found that 48 men would need to be treated to save one life. That's 47 men who, in all likelihood, can no longer function sexually or stay out of the bathroom for long."

"Numerous early screening proponents, including Thomas Stamey, a well-known Stanford University urologist, have come out against routine testing; last month, the American Cancer Society urged more caution in using the test. The American College of Preventive Medicine also concluded that there was insufficient evidence to recommend routine screening. So why is it still used? Because drug companies continue peddling the tests and advocacy groups push "prostate cancer awareness" by encouraging men to get screened. Shamefully, the American Urological Association still recommends screening, while the National Cancer Institute is vague on the issue, stating that the evidence is unclear. The federal panel empowered to evaluate cancer screening tests, the Preventive Services Task Force, recently recommended against P.S.A. screening for men aged 75 or older."

"I never dreamed that my discovery four decades ago would lead to such a profit-driven public health disaster. The medical community must confront reality and stop the inappropriate use of P.S.A. screening. Doing so would save billions of dollars and rescue millions of men from unnecessary, debilitating treatments."

Richard J. Ablin is a research professor of immunobiology and pathology at the University of Arizona College of Medicine and the president of the Robert Benjamin Ablin Foundation for Cancer Research. Dr. Levine also has been associated with similar groups that Dr. Ablin identifies as "advocacy groups" [that] push "prostate cancer awareness," such as her membership on the Board of Directors of AIDS Project Los Angeles, cited in paragraph 10. Advocating wrong or incomplete information does not help patients who are in desperate need, and often serves to contribute significant morbidity, as pointed out by Dr. Ablin.

The results for clinical trails that include other cancers such as melanoma and lymphomas are far more concerning than Ablin's recent New York Times admissions, which have largely been submerged by those very organizations Dr. Levine lists as her affiliations and experience.

Instead of conveying the impression that Levine's "NCI-designated" institutions in which she works and where she has gained her credentials, experience, and special knowledge of either cancer or AIDS, and with wording which suggests some kind of special knowledge for diagnosing and treating cancers or advanced immune suppression, other researchers typically present their credentials to provide somewhat of an opposite impression, and as slightly more humble assessments regarding their observations during their "practice of medicine" on patients. For instance, it is more widely and generally acknowledged in the cancer community that directed, and often-aggressive chemotherapies, radiation therapies, or immune therapies constitute irrational assaults on the cancer patient. Not only do these "rational," target-directed approaches not increase life expectancy in most cancer patients, they cause significant harm in the form of myelosuppression, immune dysfunction, epithelial cell destruction, nervous system stress or destruction, loss of salivation and taste in head and neck radiotherapy, burns of the skin, massive infections, and gastrointestinal collapse, castration, cachexia, and consequent mal-absorption of food, and other side-effects leading to morbidity and death. Yet this kind of assault became the model upon which "HIV" anti-retrovirals is based. According to a New England Journal of Medicine meta-analysis of Phase 1 Oncology Trials (where toxicity is typically assessed) between 1991 and 2002:

*"In a survey of **460** Phase I trials of standard toxic cancer chemotherapy agents given to slightly less than **12,000** patients, the partial and complete response rates were reported to have changed from **4-5% to 10%** during 1991-2002, with **3%** showing a complete response, and **7%** showing a partial response."*

3% complete response does not mean a 3% "cure" rate, but simply, a measure of the rate of tumor regression, as measured by the best current methods of tumor detection during the period studied. These kinds of numbers need to be appreciated along side Dr. Levine's high credentials, and along with the contributions she has made according to official statistics regarding curing cancer. Although the meta-analysis claimed that as many as 44.7% of patients showed **some** "benefit" from their therapy, and that there was a 0.5% death rate attributable to Phase I dose escalation itself, suggesting minimal overall toxicity, the "benefits" they measured were not defined and included surrogate endpoints.

The data they present in this large meta-analysis also must be qualified regarding the fact that a host of different cancer types were assessed, in which blood-borne cancers (like leukemia and lymphoma) that are now more responsive than ever before to targeted therapies, heavily weighted their analysis toward the positive value of the ***3% complete response rate*** they reported.

This overall success rate of complete response provided by this meta-analysis of 12,000 or more patients is not encouraging, not to mention the fact that a cure rate is not even considered, discussed, or mentioned. When discussion of "cure" does occur, it is typically about the successes in treating early or responsive cancers, as shown recently by a new target, the abl receptor, targeted by Gleevec (imatnib mesylate). However it should be borne in mind that this drug combats a "free-swimming" blood-borne population of tumor cells, instead of solid tumors, and many issues regarding abl inhibitors have been raised, including those related to toxicity. A survey of oncology reviews about the toxicity and lack of efficacy of current Phase I,II, and III trials for specific cancers treated with traditional chemotherapeutic agents, radiation, and targeted immunotherapy, such as those employed by Dr. Levine with her either hundreds or thousands of cancer and AIDS patients, can be obtained on a daily basis at the website of the peer-review institute at: ntkwatch@peerview-institute.org.

The following 20 clinical trial assessments indicate much less optimism than Dr. Levine suggests with her NCI-designated credentials, or her FDA and NCI or Clinton appointments. With solid tumors from trials aimed at specific types of cancer, the picture is far from certain regarding the value of these treatments for these syndromes. For instance:

1) The role of adjuvant therapy in melanoma management.[1]

*This article underlines that two decade of research in melanoma treatment **failed** to demonstrate a relapse-free and overall survival advantage in patients with stage II and stage III melanoma treated with adjuvant chemotherapy or levamisole, compared to those treated with surgery only. **One trial that studied the efficacy of interferon-gamma was interrupted after patients in the treatment group demonstrated higher mortality rates that the control group.***

2) Cutaneous malignant melanoma in Scotland: incidence, survival, and mortality, 1979-94.[2]

*The results of this study show that overall mortality rates in patients with melanoma decreased by 12% from 1984 to 1990, and this decrease seems attributable to earlier detection and other unknown factors, **but not to treatment.** The study was conducted on 6288 patients who had been diagnosed with melanoma between 1979 and 1990. During this time frame, the incidence of melanoma approximately doubled in both men and women (from 3.5 to 7.8 new cases for 100,000 men per year, and from 6.8 to 12.3 new cases for 100,000 women per year). Mortality rates remained steady from 1979 to 1984, then decreased by 10% in men and by 6% in women during 1985-1987, due to an increased detection of early stage cancers, and again decreased slightly from 1987 to 1990. **The reasons for this latter decline in mortality rates is unknown, but it is not attributed to treatment, since the only change in treatment modalities that occurred during this time was the introduction of a more conservative surgical approach to tumor removal.***

3) Interferon alfa-2a and **interleukin-2** with or without cisplatin in metastatic melanoma: a randomized trial of the European Organization for Research and Treatment of Cancer Melanoma Cooperative Group. Keilholz U; et al. J Clin Oncol, 15(7):2579-88 1997 Jul.

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[1] Barth A; Morton DL. Cancer, 75(2 Suppl):726-34 1995 Jan 15

[2] The Scottish Melanoma Group. MacKie RM, et al. BMJ 1997 Nov 1;315(7116):1117-21.

The results of this study show that chemotherapy treatment with cisplatin does not prolong survival in patients with metastatic melanoma. The study was conducted on 138 patients with advanced melanoma who were divided in two groups: one group received interferon and interleukin-2 plus cisplatin, and the other received interferon and interleukin only. ***No differences in survival were detected between the two groups.***

4) Adjuvant treatment in stage I and II malignant melanoma: a randomized trial between chemoimmunotherapy and immunotherapy. Castel T; et al. Dermatologica, 183(1):25-30 1991.

The results of this study show that chemotherapy does not prolong survival in patients with early stage melanoma. Eighty-two patients were randomized to receive immunotherapy (with the bacillus Calmette-Gu´erin) only, or immunotherapy and chemotherapy. ***No differences in survival were observed between the two groups.***

5) Recombinant interleukin-2-based treatments for advanced melanoma: the experience of the European Organization for Research and Treatment of Cancer Melanoma Cooperative Group.Keilholz U; Stoter G; Punt CJ; Scheibenbogen C; Lejeune F; Eggermont AM Cancer J. Sci Am, 3 Suppl 1():S22-8 1997 Dec.

This article presents current evidence on the role of chemotherapy in the management of patients with advanced melanoma. Single-agent or combination chemotherapy in patients with stage IV melanoma has shown to produce high rates of tumor responses (tumor shrinkage), ***but no improvement in overall survival. It has not yet been determined whether the toxicity of these regimens outweighs their potential (and yet to be proven) benefits.***

6) Phase II trial of topotecan in malignant melanoma. Kraut EH; Walker MJ; Staubus A; Gochnour D; Balcerzak SP. Cancer Invest, 15(4):318-20 1997.

This study assessed the effects of the anticancer drug topotecan in patients with advanced melanoma. Sixteen patients were enrolled in the trial. ***No tumor responses were observed. Severe toxicity occurred in 70% of patients.***

7) Prospective randomized trial of the treatment of patients with metastatic melanoma using chemotherapy with cisplatin, dacarbazine, and tamoxifen alone or in combination with **interleukin-2** and interferon alfa-2b. Rosenberg SA, et al. J Clin Oncol, 17(3):968-75 1999 Mar.

This was a randomized study to determine whether the addition of immunotherapy to chemotherapy results in better tumor control in patients with advanced melanoma. One hundred-two patients were enrolled; 52 patients received chemotherapy only, and 50 patients received chemotherapy plus immunotherapy (interferon alpha and interleukin-2). Although tumor responses were observed more frequently in the chemo-immunotherapy group (44% vs. 27%), this group also experienced higher treatment-related toxicity and showed a trend of decreased survival. ***Both regimens produced tumor responses that were only partial and short lasting.***

8) Randomized phase II trial of BCDT [carmustine (BCNU), cisplatin, dacarbazine (DTIC) and tamoxifen] with or without interferon alpha (IFN-alpha) and **interleukin** (IL-2) in patients with metastatic melanoma. Johnston SR; et al. Br J Cancer, 77(8):1280-6 1998 Apr.

The results of this randomized trial show that the addition of interleukin 2 and interferon 2 alpha to chemotherapy in patients with advanced melanoma does not result in prolonged relapse-free and overall survival, ***and is associated with a twofold increased rate of toxic reactions.***

9) Randomized, double-blind, placebo-controlled trial comparing the response rates of carmustine, dacarbazine, and cisplatin with and without tamoxifen in patients with metastatic melanoma National Cancer Institute of Canada Clinical Trials Group. Rusthoven JJ; et al. J Clin Oncol, 14(7):2083-90 1996 Jul.

The results of this double-blind placebo-controlled, randomized trial show that the addition of tamoxifen to chemotherapy ***does not improve*** *rate of tumor response in patients with advanced melanoma.*

10) Phase II trial of interleukin 1 alpha and indomethacin in treatment of metastatic melanoma. Janik JE; et al. J Natl Cancer Inst, 88(1):44-9 1996 Jan 3.

The results of this study show that combination treatment with interleukin 1 alpha and indomethacin in patients with melanoma is associated with ***minimal tumor response (10%) and significant adverse effects.***

11) Phase II trial of recombinant human interleukin-4 in patients with disseminated malignant melanoma: a Southwest Oncology Group study. Whitehead RP; et al. J Immunother, 21(6):440-6 1998 Nov.

The results of this study show that ***interleukin 4*** *is* ***not*** *effective in the management of patients with advanced melanoma. Thirty-four patients were enrolled in the study. Tumor response was observed in only* ***one*** *patient (3%). Average survival was 6 months. Adverse effects included liver toxicity, nausea and vomiting, diarrhea, headache, fatigue, muscular and joint pains, edema, fever and chills.*

12) Eastern cooperative group trial of interferon gamma in metastatic melanoma: an innovative study design. Schiller JH; Pugh M; Kirkwood JM; Karp D; Larson M; Borden E. Clin Cancer Res, 2(1):29-36 1996 Jan.

The results of this study show that treatment with interferon gamma is ineffective in the management of patients with metastatic melanoma. Ninety-eight patients were enrolled in the study. ***Tumor responses were observed in 5% of patients and were of short duration****. Toxicity included liver toxicity, fever and chills.*

13) Dacarbazine-vindesine versus dacarbazine-vindesine-cisplatin in disseminated malignant melanoma. A randomised phase III trial. Jungnelius U; et al. Eur J Cancer, 34(9):1368-74 1998 Aug.

The results of this study show that the addition of cisplatin to a chemotherapy regimen consisting of dacarbazine and vindesine ***does not*** *result in improved survival and adds significant toxicity in patients with advanced melanoma.*

14) Phase II clinical trial of recombinant alpha 2b interferon and 13 cis retinoic acid in patients with metastatic melanoma. Rosenthal MA; Oratz R. Am J Clin Oncol, 21(4):352-4 1998 Aug.

The results of this study show that treatment with interferon alpha and retinoic acid ***does not improve survival and causes significant toxicity*** *in patients with metastatic melanoma. Thirteen patients were enrolled in the study. Tumor shrinkage was observed in* ***one case****. All patients experienced substantial fatigue, muscle pains, loss of appetite, and inflammation of the oral lining. Severe toxicity required 50% dose reduction in 7 patients, and interruption of treatment in another one.*

15) Phase III trial of dacarbazine versus dacarbazine with interferon alpha-2b versus dacarbazine with tamoxifen versus dacarbazine with interferon alpha-2b and tamoxifen in patients with metastatic malignant melanoma An Eastern Cooperative Oncology Group study.Falkson CI; Ibrahim J; Kirkwood JM; Coates AS; Atkins MB; Blum RH. J Clin Oncol, 16(5):1743-51 1998 May.

The results of this study show that tamoxifen and interferon ***are ineffective*** *in the treatment of patients with advanced melanoma. Two hundred fifty-eight patients were randomized to receive the anticancer drug dacarbazine in one of the following four regimens: dacarbazine only, dacarbazine plus tamoxifen, dacarbazine plus interferon, or dacarbazine plus both tamoxifen and interferon****. No differences*** *in survival were observed between the four groups****, but patients receiving interferon experienced significantly more toxicity.***

16) Interferon-alpha and chemohormonal therapy for patients with advanced melanoma: Final results of a phase I-II study of the Cancer Biotherapy Research Group and the Mid-Atlantic Oncology Program. Stark JJ; et al. Cancer, 82(9):1677-81 1998 May 1.

The results of this study show that the addition of interferon alpha to combination chemotherapy in patients with advanced melanoma ***does not improve survival and is associated with severe toxicity.***

17) A phase II study of carboplatin, cisplatin, interferon-alpha, and tamoxifen for patients with metastatic melanoma. Gause BL; et al. Cancer Invest, 16(6):374-80 1998.

*The results of this study show that combination treatment consisting of cisplatin, carboplatin, tamoxifen, and interferon-alpha in patients with advanced melanoma is associated with an 18% tumor response and with **unacceptable toxicity**.*

18) The role of interleukin-2 in the management of stage IV melanoma The EORTC melanoma cooperative group program. Keilholz U, Eggermont AM. Cancer J Sci Am 2000 Feb;6 Suppl 1:S99-103.

*This study reviewed the results of **27** trials conducted on 631 patients with advanced stage melanoma receiving combination treatment with **interleukin** (IL)-containing regimens. Administration of chemotherapy **was not associated with improved outcome**. The effects of IL2 on survival are still being evaluated in a trial that is currently under way.*

19) Combined treatment with dacarbazine, cisplatin, fotemustine and tamoxifen in metastatic malignant melanoma. Richard MA, et al. Melanoma Res, 8(2):170-4 1998 Apr.

*The results of this trial, conducted on 20 patients with advanced stage melanoma, show that treatment with a combination chemotherapy regimen consisting of dacarbazine, cisplatin, fotemustine, and tamoxifen **does not improve survival and causes significant toxicity**, and is therefore not recommended in the management of this disease.*

20) Phase II study of combined levamisole with recombinant **interleukin-2** in patients with advanced malignant melanoma. Creagan ET, et al. Am J Clin Oncol, 20(5):490-2 1997 Oct.

*This study presents the results of a trial conducted on 19 patients with advanced melanoma enrolled to receive an experimental protocol consisting of levamisole and interleukin-2. No tumor responses were observed. Severe toxicity was observed in 5 patients. **The authors conclude that this regimen should not be further tested on patients with malignant melanoma.***

Perhaps I belabor the point here by giving 20 examples, but these randomly selected outcomes for only 20 trials in the context of only one cancer are representative of a much larger effort that Dr. Levine dismisses by touting her NCI-institution designations without pointing out the wasted efforts of these vast human experiments. And, although such results are bleak for the cancer patient, they are as problematic for the occasional cancer survivor. An Institute of Medicine report was just released calling for a paradigm shift regarding how cancer patients are managed over the long term, and asking for ways to reduce the toxicity and often morbid long-term side effects of conventional chemotherapies and radiation. (Institute of Medicine Report: Cancer Survivorship: Improving Care and Quality of Life**,** November 7, 2005; http://www.iom.edu/report.asp?id=30869).

"Some 10 million Americans are now cancer survivors. Large numbers are living longer than ever because of remarkable advances in early detection and treatment. But many survivors receive less than optimal follow-up, and improvements in care are necessary, the Institute of Medicine, part of the National Academies of Science, advised in a major report released Monday, November 7, 2005".

As presented in the Chicago Tribune:

"The negative consequences of cancer and its treatments are substantial and under-appreciated," said Dr. Sheldon Greenfield, panel chairman and director of the center for health policy research at the University of California, Irvine. "Many [patients] suffer permanent and disabling symptoms that impair normal functioning ... [but] there is much that can be done to avoid, ameliorate or arrest these late effects."

*"The Institute of Medicine study, which focuses on adult cancer survivors, highlights a profound shift in thinking about this **once-deadly disease**. Until recently, researchers and clinicians had one goal: saving more lives. With improved survival rates, however, cancer increasingly is being viewed as a chronic illness like diabetes or hypertension, presenting a new set of challenges."*

*"Some are medical. The very toxic therapies that assault tumors and **help** save lives put patients at risk of new problems down the road, including second cancers, heart disease, sexual dysfunction, cognitive impairment, infertility, and chronic inflammation, research shows. For any given patient, experts note, the risk of long-term complications depends on the type and location of the cancer, the nature and duration of treatment and other factors."*

"For instance, women with breast cancer who receive chest radiation therapy are at risk of developing lung cancer later, according to research cited in the report. Chemotherapy using agents known as anthracyclines increases the odds of contracting leukemia. And tamoxifen, a commonly used therapy for women with estrogen receptor-positive tumors, increases the risk of stroke, blood clots, and endometrial cancer."

Nevertheless, a survey of the FDA's list of approved drugs entering mainstream cancer chemotherapy clearly reveals a tendency to repeat the failures of the past. The FDA granted marketing applications to 71 oncology applications between January 1, 1990, and November 1, 2002. New additions to the FDA lists include cytotoxic drugs, monoclonal antibodies that have no efficacy and significant toxicity, immune-modulating drugs that oxidize cells and cause severe morbidity, and, a plethora of accessory drugs to boost erythrocyte production or T-cell production, anti-diarrhea medications, or medications to correct the myriad of complications due to the current toxic regimens the patient experiences.

It is truly surprising that despite these kinds of results from studies that directly target tumors (and these examples are representative of hundreds of similar trials not discussed here), new ideas or strategies that could potentially combat cancer more effectively and less toxically, are seldom given a chance, or are suppressed, and they are simply glossed over by an air of Papal infallibility and misleading descriptions or trumped credentials that suggest vast experience along with credentials and/or associations with various institutions and cancer and AIDS diagnosis and treatment efforts that qualify those directing them as having some kind of unique insights into the actual diagnoses or cures of these diseases.

In reference to AIDS, Dr. Levine's proclamations are even more misleading in paragraphs 11-14, as shown by the kinds of information that often are discussed even at International AIDS conferences, in reports in the New England Journal of Medicine, and in a plethora of scientific journals such as AIDS, AIDS Clinical Care, and in many others. Despite her service (as described in paragraphs 2-12) on various medical boards, among professional societies, among Program Committees, among Advisory Committees, Working Groups, etc., it is nothing if not misleading, if not once does Dr. Levine reveal in her December statement the fact that the general consensus and feeling in both the cancer and AIDS contexts as well as in both the scientific-medical biomedical communities, is that these efforts are regarded as totally non-productive efforts generated by the very kinds of political bodies as those she has been associated with and lists as her credentials, in terms of either understanding or quelling cancer or AIDS.

This Levine-promulgated credential list, exists behind her name without any scientific foundation or basis, or correct assessment of the state of affairs regarding our knowledge of cancer or AIDS. At some point, such information should be at least mentioned by Dr. Levine, if not emphasized. To do otherwise is irresponsible. For instance, she should have, at some point during those 15 or so credential-touting paragraphs, at least provided at least of the few following widely acknowledged track records of non-production of the types of initiatives she has been involved with for more than 25 years, and at least some mention of the often enormous harm of that this medical and scientific non-production has resulted in, due these same kinds of "advocacy groups" and organizations Dr. Levine claims to advise, or participate with, as shown by the following track records of failure in multiple contexts of recent AIDS research and treatment:

"Vaccine Failure Is Setback in AIDS Fight-Test Subjects May Have Been Put at Extra Risk Of Contracting HIV." *By David Brown Washington Post Staff Writer, Friday, March 21, 2008; Page A01.*

"The two-decade search for an AIDS vaccine is in crisis after two field tests of the most promising contender not only did not protect people from the virus but may actually have put them at increased risk of becoming ***infected****."*
" The results of the trials, which enrolled volunteers on four continents, have spurred intense ***scientific*** *inquiry and unprecedented* ***soul-searching*** *as researchers try to make sense of what happened and assess whether they should have seen it coming."*
"Both field tests were halted last September, and seven other trials of similarly designed AIDS vaccines have either been stopped or put off indefinitely. Some may be modified and others canceled outright. ***Numerous experts*** *are questioning both the scientific premises and the overall strategy of the nearly* ***$500 million in AIDS vaccine research funded annually*** *by the U.S. government."*

"This is on the same level of catastrophe as the Challenger disaster that destroyed a NASA space shuttle…" *said Robert Gallo,* ***co****-discoverer* ***of the human immunodeficiency virus (HIV), which causes AIDS****, and head of the Institute for Human Virology in Baltimore."*

This is only the tip of the ice burg of failure regarding any vaccine for "HIV," as there have been 64 failed trials that were abruptly halted in many cases, including AIDSVAX and many more, and currently, there are 170 in the vaccine pipeline for "HIV" as none of them appear to evoke T-cell activation in many cases, or anticipated seroconversion, at a price tag of $500 M/year (**http://aidsinfo.nih.gov/vaccines/MainSearch.aspx?strSponsor=All&strRecruiting=on&status=1&strNoRecruiting=on&NRstatus=1**).

The even larger Thailand-US military vaccine trial on some 16,000 human beings also was halted, and the PAVE trial was cancelled before it was begun. There have been no less than 16 halted microbicide trials on Africans because the microbicides increased rather than decreased the "HIV+" detection rate in the genitally smeared, there have been halted breast feeding-dissuasion campaigns because hundreds of infants died due to the advice of physicians such as Dr. Levine regarding breast feeding dissuasion, and numerous (too many to count) failed drug trials, including Concorde, the Veteran's affairs, and the Fischl trial of AZT, in which all the patients "prescribed AZT hundreds of times," as described by Dr. Levine, died within several years after receiving the drug, while many of the few survivors went on to develop cancers.

In light of how front line researchers in cancer and AIDS typically present their credentials, including what they emphasize or not, it is my sincere and honest opinion that these kinds of disclosures are glossed over, or are misleading in the way the experiences and credentials of Dr. Levine are presented in paragraphs **2-14**. Especially as in paragraph 13, where Dr. Levine discloses she has more than 300 publications or book chapters, some of which relate to "HIV." In my experience, it isn't the quantity, but the quality of work that is noted by the scientific and medical communities. For instance, among her 300+ publications, could Dr. Levine submit at least one of them that discloses how "HIV" kills T-cells as she claims it does? One such paper would be worth 100,000 papers that assume biological mechanisms that have no basis in experimental reality: instead of revealing such dismal progress in AIDS research (or cancer research and "treatment"), the numerous committees, professional attachments, and claims of experience with hundreds or thousands of AIDS patients as claimed by Dr. Levine gives the impression that such experiences have led to some "special knowledge" that only those in her position or with her history can comprehend, regarding what is best for these patients. Nothing could be further from the truth, as this challenge to Dr. Levine's God-like certainties are provided by the following information that reveals typically how scientifically trained, and as I would argue, less self-impressed researchers of AIDS, describe their research, the state of their science, clinical trails, or new treatments and their outcomes (I will give only several examples instead of 20 as I did for melanoma above). For instance, instead of claiming, as does Dr. Levine, that "I have been involved in the study, care and treatment of patients with HIV/AIDS and associated malignancies since the earliest recognition of the epidemic, before it had a name," other researchers show slightly more scientific caution, and what may be described as the kind of scientific approach that comes with true experience involving the information associated with such fatal syndromes as cancer and AIDS, as opposed to rather glib reassurances that long lists of credentials can somehow lend special insights not acquired by the rest of the scientific and medical community.

Dr. Abigail Zugar stated it in most clearly in words that, although slightly technical, are easy to understand, and the implications are clear:

"The Puzzle of CD4-Cell Depletion Despite Good Viral Suppression. *In some patients, CD4-cell counts fail to rise as expected. Could extensive lymph node fibrosis be responsible?"*

In other words, instead of Dr. Levine's glib reassurances that "the sciences" of cancer and AIDS are clear and transparent to all, in the most recent journals of AIDS Clinical Care, the "AIDS" syndrome is unexpectedly progressing in front of doctor's eyes, and despite treatments with "life saving anti-retrovirals;" and in many cases without "HIV" being detected at all. Even more concerning statements followed:

"In a recent study, NIH researchers sought evidence to support ***any*** *of several* ***hypothetical explanations*** *for the aberrant CD4-cell responses seen in four patients on combination ART whose CD4 counts had fallen from a median of 719 cells/mm^3 to a median of 227 cells/mm^3* ***despite persistently undetectable plasma viral loads****."1qa*[2] AIDS Clincial Care, June 1, 2009.

These patients were taking a double or triple AIDS-drug cocktail regimen, no "viral load" could be detected, yet their T-cells were plunging from relatively normal levels to the worrisome and CDC-defined level reached when doctors suggest that drug therapy should begin, at around 200-300 CD4+ cells/mm. Drug "resistance" was "checked" and found not to be an issue. However, what is most troubling is that Dr. Zugar then writes that:

"Residual ***replicating*** *HIV did not seem to be the problem: Results of* ***ultrasensitive*** *PCR and assays for peripheral blood mononuclear* ***cell–associated*** *HIV RNA and proviral HIV DNA — and of assays for cell-associated HIV RNA and proviral DNA in mononuclear cells from inguinal lymph nodes — were similar to those obtained in other,* ***successfully treated*** *patients."*

Dr. Levine's credentials and touted experience also are of concern because of the following types of admissions by federally funded research groups throughout America:

"A nationwide team of orthodox AIDS researchers led by doctors Benigno Rodriguez and Michael Lederman of Case Western Reserve University in Cleveland ***are disputing the value of viral load tests****-a standard used since 1996 to assess health, predict*

progression to disease, and grant approval to new AIDS drugs after their study of 2,800 HIV positives concluded viral load measures ***failed in more than 90% of cases*** *to predict or explain immune status…" "Viral load is only able to predict progression to disease in 4% to 6% of HIV-positives studied, challenging much of the basis for current AIDS science and treatment policy* [Rodriquez B, Sethi AK, Cheruvu VK, et al. Predictive value of plasma HIV RNA level on rate of CD4 T-cell decline in untreated HIV infection. JAMA 296(12):1498-506, 2006; Cohen J. Study says HIV blood levels don't predict immune decline. Science 313(5795):1868, 2006].

Most horrifying, regarding Levine's use of her "credentials" to justify her point of view on this issue, is the fact that universal testing for "HIV" "infection" is known to increase morbidity and death amongst those designated as "HIV/AIDS" patients, rather than decrease their morbidity and death. For example, de Martino *et al.* concluded that children born to ZDV-treated mothers (ZVD is AZT, or the AIDS drug, Azidothymidine):

"*are* ***more likely*** *to have a rapid course of HIV-1 infection compared with children born to untreated mothers, as disease progression and immunological deterioration are significantly more rapid* ***and the risk of death is actually increased during the first 3 years of life****"* [de Martino et al., Rapid disease progression in HIV-1 perinatally infected children born to mothers receiving zidovudine monotherapy during pregnancy. AIDS. 13(8):927-933, May 28, 1999. The Italian Register for HIV Infection in Children AIDS, 13:927-933, 1999].

In the journal Pediatrics, Antoni Noguera et al reported that:*"Almost* ***half*** *of the children (63 of 127) who were exposed to nucleoside analogues developed benign and self-limited hyperlactatemia when symptomatic, nucleoside analogue–induced toxicity affected* ***neurologic development*** *"* [Antoni Noguera et al. Pediatrics, Vol. 114 No. 5 November, pp 598-603, 2004].

In 1992, The Veterans Affairs Co-operative Study Group reported that AZT disproportionately harmed Blacks and Hispanics, and provided no benefit to the quelling of advancing immune suppression in Caucasians, and harmed healthier subjects (early treated) more than persons considered to exhibit clinical symptoms of AIDS [JD Hamilton et. al. and the Veterans Affairs Cooperative Study Group. A controlled trial of early versus late treatment with zidovudine in symptomatic human immunodifficiency virus infection." New England Journal of Medicine, 326: 437-434, 1992].

The Concorde trial, which was published without endorsement by Burroughs Wellcome's Coordinating Committee who declined to endorse the final report, and which was the largest, longest, and best controlled adult AZT trial concluded:

"The results of Concorde ***do not*** *encourage the early use of zidovudine in symptom-free HIV-infected adults. They also call into question the uncritical* ***use of CD4 cell counts as a surrogate endpoint*** *for assessment of benefit from long-term antiretroviral therapy"* [Seligmann et al., Concorde: MRC/ANRS randomised double-blind controlled trial of immediate and deferred zidovudine in symptom-free HIV infection. Concorde Coordinating Committee. Lancet, Apr 9;343(8902):871-81, 1994].

Perhaps this sad legacy of drugging experiments on human subjects is nowhere made so obvious as in the report issued after the first decade of HAART, where it was claimed that improvements in "viral load" measurements were obtained but there were NO improvements in mortality. In fact, Dr. Levine lists the kinds of committees as her credentials that recommend these kinds of vast an uninformed human experiments-in some cases, they are the very same ones, that Dr. Levine lists as evidence of her vast experience with AIDS:

Methods: *We analyzed data from* ***22,217 treatment-naïve HIV-1-infected*** *adults who had started HAART and were followed in one of* ***12*** *cohort studies. The probablility of reaching 500 or less HIV-1 RNA copies per mL by 6 months, and the change in CD4 cell counts, were analyzed for patients starting HAART in 1995-96, 1997, 1998, 1999, 2000, 2001, and 2002-03. The primary endpoints were the hazard ratios for AIDS and for death from all causes in the first year of HAART, which were estimated using Cox regression.*

Interpretation: *Virological response after starting HAART* ***improved*** *over calander years,* ***but such improvement has not translated into a decrease in mortality.*** [The Antiretroviral Therapy (ART) cohort Collaboration-www.thelancet.com Vol 368, 451-58, August 5, 2006].

Thus, for those on the front lines, rather than the layman perhaps, the impressive credentials of Dr. Levine that are associated with these types and other ongoing human experiments which might be more aptly described as challenger-sized drugging disasters, are inconsistent with the true state of both AIDS and cancer research and treatment. Long lists of impressive credentials, such as Dr. Levine's, cannot ever substitute for actual scientific or medical progress or achievement in the directions that might actually make a difference in patient care. The blurring of actual scientific or medical achievement toward any possibly curative directions, waged against such syndromes as cancer or AIDS with such inflated descriptions of political achievement and academic/medical

advancement in these contexts, can only be written to be misleading as to the true nature of our ignorance as either scientists or front-line physicians regarding these syndromes, as any review of the information would reveal.

Paragraph 16:

Again, in this paragraph, I find Dr. Levine's word choice misleading. She carefully refers to and claims that [paraphrase] "NHL is not **fully** understood, [and] it has long been appreciated that the disease occurs at greater rates in the setting of chronic immunosuppression." In my experience, not only is any cancer "not fully understood," but those who study it intensively would agree that very little, if anything is known about the syndromes called cancer. Moreover, the word-choice "in the setting of chronic immunosuppression" belies the fact that in biology, as well as in medicine, immune suppression itself has never been linked to the cause of any cancer(s)…(please see my analysis of this issue in my report, "Is AZT a carcinogen").

Paragraph 17:

Dr. Levine's misinformation and distortions continue in this paragraph: *"The human immune system protects against infection (true enough) and other diseases including cancer* (this is a biologically indefensible claim). While it is true that the immune system protects against infection, one of the ways it does this (either through natural infection or vaccination), is that antibodies are produced by the immune system against a universe of pathogens, and a positive antibody test against molecules of those antigens that that immune system produces are thereafter protective of future infections by those same antigens (either through natural acquisition or vaccine challenges). While generally true, and with the admission that natural infection always provides better and longer protection than any vaccine introduction of antigens, the AIDS construct stands as a principal exception to this general immunological hypothesis. Instead of being protective against "HIV-infection," a positive antibody test signals to an incorrectly interpreted phenomenology, that an "HIV-positive" individual is more likely to develop disease (AIDS) than a non-"HIV-positive "carrier" of "HIV" antibodies. To correct this biologically contrary and indefensible view of the facts known to immunology that contradicts an entire biotech industry that provides antibodies to biological laboratories like mine against actin, or tubulin, for instance, the Nobelist, and discoverer of "HIV," Professor Luc Montagnier, has tried repeatedly and recently on public record to correct this absurdity. In a recent interview, for instance, Luc Montagnier tried to correct this misapprehension and misapplication of his Nobel-worthy finding of "HIV." Luc Montagnier in a recent documentary shown all over the world said in this regard that: "you can catch it ("HIV") many times and if your immune system is strong you can get rid of it" no problem! (This specific interview with the Nobelist, Luc Montagnier, discoverer of "HIV," [in exception to Dr. Levine, can be watched at http://www.youtube.com/watch?v=WQoNW7lOnT4l, and the entire film, "House of numbers," in which this interview takes place, can be seen in its entirety at http://www.facebook.com/l.php?u=http%3A%2F%2Fhivdissidents.taba.ru%2Fvideo%2Ff%2F26508_Dom_chisel.html&h=f6d5eYwjJV-GDsjmQI3gQp8u_Vg:

Luc Montagnier:

"…I believe HIV, we can be exposed to HIV many times without being chronically infected. ***Our immune system will get rid of the virus in a few weeks****, if you have* ***a good*** ***immune system****; and this is also* ***the problem*** ***with African people****;* ***their nutrition*** *is not very equilibrated, they are in* ***oxidative stress****,* ***even*** *if they* ***are not*** *infected with HIV, so their immune system doesn't work well already, so it is prone, you know, to allow HIV to get in and persist. So there are many ways,* ***not the vaccine****, many ways to decrease* ***the transmission****, just by simple measures of* ***nutrition, giving anti-oxidants, proper anti-oxidants-hygiene measures, fighting the other infections****."*

Interviewer:

"If you have a good immune system, then your body can naturally get rid of HIV?"

Luc Montagnier:

"Yes."

This recent internationally-presented correction and readjustment of the irrational "HIV"=AIDS=Death false construct by the Nobelist and discoverer of "HIV" brings into focus what is known more generally about how antibodies against an infectious agent are protective of future infection, in the setting of a normal immune system, and should stand as a testimonial regarding Dr. Levine's understanding of debunked immunological hypotheses regarding "the immune system's protective role" more generally.

Regarding putting a cart before the horse regarding the causal association between immune suppression and cancer, or cancer causing immune suppression, the preponderance of evidence that cancer causes immunosuppression is more frequently or predictably encountered than the fact that low or no immunity or immune systems, as is found in embryos and newborns, is a favorable condition for cancer development, which they are not. For instance, Ryungsa Kim, Manabu Emi, and Kazuaki Tanabe published that, "Cancer immunosuppression and autoimmune disease: beyond immunosuppressive networks for tumour immunity wrote (Immunology. 2006 October; 119(2): 254–264. doi: 10.1111/j.1365-2567.2006.02430.x. International Radiation Information Centre, Hiroshima University, Hiroshima, Japan):

Abstract
Cancer immunosuppression evolves by constitution of an immunosuppressive network ***extending from a primary tumour site*** *to secondary lymphoid organs and peripheral vessels and is mediated by several tumour-derived soluble factors (TDSFs) such as interleukin-10 (IL-10), transforming growth factor-β (TGF-β) and vascular endothelial growth factor (VEGF). TDSFs induce immature myeloid cells and regulatory T cells in accordance with tumour progression, resulting in the inhibition of dendritic cell maturation and T-cell activation in a tumour-specific immune response. Tumour cells grow by exploiting a pro-inflammatory situation in the tumour microenvironment, whereas immune cells are regulated by TDSFs during anti-inflammatory situations—mediated by impaired clearance of apoptotic cells—that cause the release of IL-10, TGF-β, and prostaglandin E2 (PGE2) by macrophages. Accumulation of impaired apoptotic cells induces anti-DNA antibodies directed against self antigens, which resembles a pseudo-autoimmune status. Systemic lupus erythematosus is a prototype of autoimmune disease that is characterized by defective tolerance of self antigens, the presence of anti-DNA antibodies and a pro-inflammatory response. The anti-DNA antibodies can be produced by impaired clearance of apoptotic cells, which is the result of a hereditary deficiency of complements C1q, C3 and C4, which are involved in the recognition of phagocytosis by macrophages. Thus, it is likely that impaired clearance of apoptotic cells is able to provoke different types of immune dysfunction in cancer and autoimmune disease in which some are similar and others are critically different. This review discusses a comparison of immunological dysfunctions in cancer and autoimmune disease with the aim of exploring new insights beyond cancer immunosuppression in tumour immunity.*

In other words, it is more generally accepted in the scientific and medical communities that tumors and cancer cause immune suppression, rather than the opposite, and that the undeveloped or injured immune system does not cause cancers, but will under suppressed circumstances, allow them to flourish without rejection. The transplant literature also is consistent with this view, as immune suppressive drug regimens are themselves carcinogenic (see my treatment of this subject in "Is AZT a carcinogen"). Indeed, recently, the medical community has now become aware that such drugs as cyclosporines cause cancer directly, rather than allowing the cause of cancer to arise because of these drugs abilities to suppress certain elements of the immune system.

Paragraph 18: Dr. Levine's discusses how NHL and Kaposi's sarcoma became first recognized as AIDS-defining illnesses. Her argument regarding how NHL was known to be statistically increased and associated with HIV well before the advent of AZT neglects to mention, for instance, that Kaposi's sarcoma was first named by Moritz Kaposi more than a century ago, and can occur in the complete absence of any T-cell defect (Flosie Wong-Staal & Robert C. Gallo. Nature Vol 317, 3 Oct 1985).

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"The association of Kaposi's sarcoma with AIDS deserves special mention. This otherwise ***extremely*** *rare malignancy occurs predominantly in a restricted group, that is, the homosexuals, and can occur* ***in the absence*** *of any T-cell defect in the patients."*

Following Wong-Staal's and Gallo's construct above this means: A (Homosexuals)=B (no decline or detectable defect in T-cells)=C (AIDS-Kaposi's).

This kind of simplistic textbook description of Dr. Levine regarding complex associations known to exist between various risk groups and so-called AIDS-defining illnesses is presented again here without proper discussion of the complexities inherent in these associations/syndromes, in order to propagate these tired, long-debunked, and woefully inaccurate hypotheses regarding the immune system and cancers or cancer-like syndromes. The fact that even the so-called co-discoverer of "HIV," Robert Gallo recognized and published this disconnect between T-cell defects and Kaposi's as far back as 1985 illustrates that Dr. Levine is not current on the evolving picture of "HIV/AIDS." In fact, illicit drug usage, as well as nitrosamines were identified as being significant risk factors for the initial development of Kaposi's sarcoma before the advent of AZT, and it may have escaped her memory that it was Robert Gallo, who consulted with the Late Judah Folkman of Harvard (my former mentor), to explain why Kaposi's itself, as one of the first two AIDS-indicator illnesses, could not possibly have any thing to do with "HIV," as it is presented in Dr. Gallo's book, "Virus Hunting and Cancer" (page 267), and that even more concerning, that Kaposi's spindle-shaped cells have characteristics other than endothelial cells, as my mentor educated him about. Thus eventually, after the tat-transgene experiments of Jay failed to show any "HIV-tat" protein among Kaposi cell lesions, another virus was soon blamed (HHV) for Kaposi's on the basis of equally tenuous experimental evidence.

Paragraph 19:

Again, I find it disconcerting that Dr. Levine provides us with increasingly inaccurate and wrong information in this paragraph, where she describes how AZT "kills" viruses, and that AZT was the first effective treatment against "HIV." Please see my description of the 1987 fraudulent Fischl trial, after which AZT became "FDA approved" to kill AIDS patients at those dosages used then, within about 3 years, which was not long enough to assess its carcinogenic potential. The collaborative European and American trail of HAART cited above that describes the outcome of more than 22,700 HAART-prescribed patients addresses Dr. Levine's cheerful, if incorrect recollections regarding how *"The introduction of AZT followed by other effective antiretroviral medications used in combination radically changed the course of the AIDS epidemic,"* and where she goes on to admit that *"However, the antiretroviral treatments cannot kill the HIV completely, and thus are not a cure."* Many patients that do not die from liver or kidney failure, or profound

anemia, or cancer due to continuous AZT or HAART, are still continuously drugged for as long as they can tolerate the regimens of the same ineffective and toxic drugs used to nearly kill cancer patients, which is why they are cycled on and off drugs with similar mechanistic profiles. This paragraph is riddled with inaccuracies, distortions, and biologically wrong information, while it at the same time, it demonstrates how propaganda continues to be advanced regarding the value of HAART. For instance, nobody has ever demonstrated that AZT "kills" "HIV" In Vivo, as too few particles of "HIV" are ever indirectly found in an "infected person" to begin with (which is why PCR is used today against its inventor's warnings that it doesn't detect "HIV"). Also in this context, the language used, "AZT kills the virus" betrays an extremely naïve if not uninformed view of viruses existing as living things in the first place, which they are not of course. AZT doesn't "kill" any virus…it interferes with the normal metabolic functioning of nucleic acid synthesis, leading to mitochondrial damage, mistakes in DNA replication, and a plethora of other damage exerted on normal replicating cells of the body, which will eventually lead to death as would any chemotherapeutic regimen with a similar mechanism of action.

Paragraphs 20-37:

In paragraphs 20-37 Dr. Levine gives us a series of short discussions exhibiting what she knows about the association of NHL with AZT. I have extensively dissected these data in my previously admitted position statement, "Is AZT a carcinogen," and so therefore I will not cover this here. However, to summarize the principal point made in the "Is AZT a carcinogen analysis," it should be emphasized here that one cannot demonstrate causality using epidemiological studies, surrogate markers (T-cell numbers), other syndromes associated with cancer such as immune suppression, or indeed with any of the arguments presented by Dr. Levine in paragraphs 20-37. Suffice it to say, to do so would be akin to claiming that fire trucks cause fires because they frequently are seen where there are fires occurring, or that skid marks cause auto accidents because they too are often frequently associated. Similarly, epidemiological arguments cannot be used to demonstrate causality or in many cases, to even exclude a causal relationship because, absurdities and unrelated information can never be held as independent or dependent variables. Let me give one example. Lets assume that the consumption of alcohol is involved in 20% of auto accidents. This does not mean that the remaining 80% of auto accidents are caused by the consumption of water, fruit juice, coffee, or other beverages. One must set up experiments using one independent variable, to compare the outcome that variable exerts in the system being tested. When Pluda examined the NHL rate(s) of men treated with AZT, Dr. Levine is fundamentally correct that this did not prove a causal connection between AZT use and NHL; when Moore examined his cohorts for a connection between AZT usage and NHL and found a much higher odds ratio for the development of lymphoma, again this does not prove causality. However, when the carefully designed rodent studies with AZT were performed, using organisms whose entire life-span could be followed while on the drug, it was demonstrated by a number of groups that the scientific conditions to demonstrate causality were satisfied, and that AZT was associated with a significant rate of cancer in various rodents. When Pluda examined his surviving cohorts at 36 months who had consumed AZT and found a 46% rate of lymphoma, this information, along with all other information should have signaled to the medical community, a possibly dangerous association between AZT and NHL, more than a decade or more before California decided to place AZT on its list of carcinogens.

Dr. Levine can choose to ignore the numerical associations in the very same studies she cites to conclude no association between AZT and NHL, but none of these studies can show causality, as they all measure indirect markers of immune deficiency which in many cases don't reflect immune deficiency at all (as Dr. Gallo warned regarding the fact that Kaposi's sarcoma can occur without any evidence of T-cell defect, or as the Cleveland study cited above pointed out that these markers only could predict disease progression in 4% to 6% of patients). Often, if not always, these "epidemiological studies" used indirect or disputed markers of viral infection, or in one case (Grulick's study), the abundance of immunoglobulins as being the highest risk factor for the development of cancer as the antibodies are said to "stimulate" B-cells" (that supposedly "HIV" leaves alone) to generate cancers (as has been known also with other blood markers such as SED rates before these studies were even conducted), etc. Yet these associations are inherently contradictory to begin with. For instance, how could an immune suppressive illness cause an up-regulation in the immune system too, and in doing so, how could an epidemiologist isolate a single independent variable to demonstrate causality? With all of these variables existing as a blur as epidemiological markers, like fire trucks causing fires, the only cogent conclusion possible with Pluda's, Moore's, Munoz, and others incomplete epidemiological assessments, is that when rates of NHL or other cancers are encountered frequently in persons who are highly antigenically stimulated, drugged, or for other reasons, it should serve as a danger signal that drugs, with mechanisms of action known to causally generate cancers in test organisms such as mice at rates sometimes exceeding 20%, that such a substance also might be responsible for the generation of cancers in other mammals such as humans, and which, as California wisely recently decided in 2009, should be banned for use in humans as potential human carcinogens instead of fed to human experimental test subjects for life with warnings of compliance, as Dr. Levine advocates doing in paragraph 37.

Paragraph 38:

Most disturbing of all, perhaps, of Dr. Levine's assessments or opinions are encountered by her treatment and explanation of the Robert Zapata records. If her assessment in her declaration is representative of her diagnosis, treatments, and assessments of thousands or hundreds (whatever the real number is is not clear as she claims both), then the extent of the damage she might have

caused is beyond estimation. The repeated lymph node biopsies unequivocally rule out a possible AIDS diagnosis, and because they were checked so many times for lymphoma, while ignoring the bone findings, it is almost a certainty that Mr. Zapata harbored a diffuse B-cell lymphoma in his bone marrow.

Perhaps the most egregious error of certainty she makes is in the definition of cancer being advanced by Dr. Levine, which is at odds with the Robert Zapata's records, the certainty he is an "HIV" patient in the setting of a negative "HIV" test being discovered before he developed and was treated for a herpes infection, and the fact that not even NHL experts" or the textbooks are in agreement about many of the diagnostic features of various NHL's or lymphomas more generally. The "patchy" highly metabolic labeling and crushed lymphoid cells are consistent with NHL cancer, that Dr. Levine apparently is not aware of despite her claimed expertise and NHL research focus. The following information can rule out AIDS, but can only suggest that Mr. Zapata harbored an NHL cancer at the time of his death. It strongly suggests that his tonsil lymphoma in 2005 was caused by his consumption of AZT.

TIMELINE OF MEDICAL INFORMATION PROVIDED FOR ROBERT ZAPATA.

Treatment for "HIV" and herpes.

1. 1999 (October). Mr. Zapata, a 33 year-old Hispanic male, is tested for "HIV." **His test is negative**. He has recently married, and has had 2 children with his wife.

2. 2000 (March). Five months later, Mr. Zapata visits his doctor. His doctor's written notes describe that Mr. Zapata is suffering from fatigue, night sweats, skin rashes, and herpes. Mr. Zapata is tested for "HIV" again and the test indicates to his doctor that he is positive for "HIV." There is no discussion noted that it had been widely acknowledged since 1992, even by the CDC, that herpes causes "HIV" tests to react positive:

Langedijk J, Vos W, Doornum G, et al. 1992. Identification of cross-reactive epitopes recognized by HIV-1 false-positive sera. *AIDS*. 6:1547-1548.

Challakere K, Rapaport M. 1993. False-positive human immunodeficiency virus type 1 ELISA results in low-risk subjects. *West. J. Med*. 159(2):214-215.

3. 2000 (April). A second visit is made a month later in April, and the doctor's notes describe bronchitis, a rash on buttocks, night sweats, a new rash on his arm with red blisters, leukoplakia of the mouth and throat, and "herpes zoster." At this visit, six months after testing "HIV-negative," a confirmatory test is not given, and Mr. Zapata is prescribed full dosages of **combivir, sustiva, septrim, chlortrimazole, and acyclovir**. Combivir is a combination AIDS medication made by GlaxoSmithKline, and contains zidovudine (AZT), and lamivudine (3TC). One of the many major and frequent adverse reactions to AZT is the development of non-Hodgkin's lymphoma in some 40-70% of patients (see Pluda et al.). This life threatening adverse reaction is stated on the package insert of the AZT, as well as a long list of other life-threatening and life-ending side effects of AZT:

*"…anemia, dementia, diarrhea, muscle wasting, candidiasis, non-specific oral lesions, severe fatigue, **enlarged liver and liver failure, heart failure**, diabetes, unmasking of opportunistic infections including CMV retinitis, spontaneous bleeding in hemophiliacs, **lymphoma…**"*

AZT (Zidovudine) has been placed on California's list of known carcinogens: (http://www.oehha.ca.gov/prop65/prop65_list/files/P65single100810.pdf**)**. Lamivudine is a nucleoside analog whose chemical adverse effects are known to be similar to those of AZT's. Sustiva is an addictive drug now known to be smoked by children and adults in South Africa to get high. All warnings that can be found in medical databases claim that zidovudine and lamivudine is not a cure and may not decrease the number of HIV-related illnesses. Yet, none of the potential adverse effects of any of these drugs were disclosed to Mr. Zapata. There also are known warning **contraindications against combining combivir with acyclovir**, a drug given to herpes patients, which a month earlier was prescribed to Mr. Zapata at a dose of 800 mg/5X day (almost a gram), for 10 days. The CDC recommends for first time herpes infections either acyclovir 400 mg orally three times a day for 7–10 days or acyclovir 200 mg orally five times a day for 7–10 days. In recurrent infections, the recommended dosages are: acyclovir 400 mg orally three times a day for 5 days, acyclovir 800 mg orally twice a day for 5 days or acyclovir 800 mg orally three times a day for 2 days. The quantities given of combivir is not noted on the records, but the addictive drug sustiva is prescribed at 200mg 3X/day.

4. 2000 (October). Seven months after initially testing "HIV-positive in March," Mr. Zapata's CD-4 T-cells and "viral load" are measured (T-cells=164, and his VL= 147). These measurements generally should suggest to modern medical standard of care

thinking that either: **1**. Mr. Zapata has had AIDS for a long time which is contra-indicated by his October 1999 negative "HIV" test, as 164 CD-4 represents a low reading, typically thought to represent a very long-time AIDS condition of more than several years duration, yet he tested negative only 12 months before. **2**. In light of the negative "HIV" test provided in 1999 in October, and although a "confirmatory "HIV" WESTERN blot test was not obtained, Mr. Zapata is extremely likely to be a false positive "HIV" tester due to his herpes infection, or for 70 other known reasons. Mr. Zapata presents at this visit with dermatitis, a rash on his buttocks, fatigue, hairy leukoplakia in his mouth and throat, rhinitis, cough, bronchitis, and genital herpes. He is offered **a pneumococcal vaccine, flu-medicine, septra, combivir, sustiva** (200mg/3X day), and **lotrimin** for diarrhea.
5. 2000 (December). Mr. Zapata again visits his doctor and presents with a cough. His T-cells and viral load are measured again: T-cells=247, VL=1233. Clearly, according to the diagnostic information and expected therapeutic outcomes available on medical databases, these numbers signal that he is receiving no benefit from his ARV cocktail because his so-called viral load has increased to 1233 while his T-cells are becoming only slightly more numerous. HAART should depress viral load, not increase it.

Cancer diagnosis and treatment:

6. 2005 (November). Five years later, Mr. Zapata again visits his doctor, and presents with peritonsilitis (no mention of hairy leukoplakia), and a golf-ball sized tumor is found growing on his left tonsil in his throat. The lesion is biopsied, stained, it reacts positive for B20, and negatively for cytokeratin, AE1/AE/3, and other diagnostic indicators of a **B-cell** non-Hodgkin's lymphoma (NHL). The tumor tissue is necrotic, and there are "sheets" of large lymphoid cells with irregular nuclear borders, one to multiple conspicuous nucleoli, and an even chromatin pattern. The tumor is surgically removed. A discussion is recorded on the medical record about his compliance/non-compliance with HAART (combivir, sustiva, septrin, and other medications since he had been prescribed them 5-years ealier, in 2000). **A monoclonal gammopathy to rule out systemic involvement of the lymphoma is not performed, but discussed among his doctors**. Cysts in his left kidney are discovered on CT scans.

7. 2005 (December). A bone marrow study is performed for staging purposes. CBC from 12/12/05 reveals RBC=5.0 million/microliter, HGB=15.2 gm/dl, HCT=43.7%, MCV=87, platelets=245,000/ml, **WBC=5.0 million/microliter**, with an automated differential of 74% neutrophils, 22% lymphocytes, and 4% monocytes. Despite his admitted non-compliance with HAART preceding the removal of the tumor, his blood work, including his RBC's and white count are in the normal range. R-CHOP therapy is initiated. At this time it is noted that he has adequate marrow iron stores, mild mature plasmacytosis (4%), **slight peripheral leucopenia**, but he is deemed negative for marrow involvement by malignant non-Hodgkin's lymphoma.

8. 2008 (July). Mr. Zapata, now 43 years old, presents at the hospital with debilitating back pain, involving both lumbar and thoracic spinal vertebra. His pain is unresponsive to vicadin. His abdomen is soft and tender. His spleen in CT scans is described at the upper limits of normal size at 13 cm, increased from a previous scan, **with a low density focus posteriorly**. Mild scarring is seen at the right lung base, along with some dependent atlectasis. There appeared to be more vasculature in these regions **in mosaic patterns**. His pancreas, gallbladder, and adrenals are unremarkable. Although his lymph nodes appear unremarkable, he is given a lymph node biopsy of a slightly enlarged left axillary lymph node with negative findings for non-Hodgkin's B cell lymphoma. **However, what the biopsy results do indicate about this left axillary lymph node is surprising from the perspective of an 8-year "HIV/AIDS" diagnosis. Sections of his enlarged node demonstrate no evidence of effacement. The lymph node is composed predominantly of primary follicles with occasional secondary follicles formation. The secondary follicles have polarized germinal centers. The interfollicular population is mixed and includes plasma cells. No Reed-Sternberg cells are present, and there are no granulomas. With this careful pathological analysis, no fibrosis of this axillary lymph node of any kind is noted, despite the imagined ravages of an 8-year "HIV" infection and admitted non-compliance to HAART.**

On scans two renal calculi are visualized and **metabolic labeling of his bone marrow is greater than that normally seen without stimulation.** The finding is explained as a result of "**individual variation**." Punctate sclerotic foci are seen in both femoral heads, and degenerative changes are seen in his lumbar spine. A bone marrow apirate smear and core biopsy shows **extensive necrosis (80% necrotic)**, with the only viable portion of the core biopsy showing **dense fibrosis**, with occasional mixed hematopoietic elements and **crushed lymphoid cells (crushed lymphoid cells have been established as a diagnostic criteria for primary bone lymphomas at least since 1996-see below)**. **Reticulin fibrosis** is markedly increased (3-4+) **even in the large area of necrosis**. Rare bone marrow elements including maturing myeloid cells, erythroid cells, and **rare** megakaryocytes are entrapped within fibrious material and scattered **small lymphocytes** are also noted. Immunophenotypic analysis that is performed by US Labs (#AFTO8016225) shows decreased CD4+ T-cells but no evidence of non-Hodgkin's lymphoma. **85% of the lymphocytes are T-cells with a reduced CD4: CD8 ratio and normal pan-T-cell antigen expression**, 4% are polyclonal B cells, and the remainder are NK cells. He is diagnosed with mild bilateral axillary adenopathy with larger and more numerous nodes than are normally seen, that have increased since the last exam. His lungs are clear. **His white blood cell count is in the normal range (5.3)**, suggesting he no longer (or ever did) have AIDS. **Finally, and significantly, it is noted that bone marrow**

activity is greater than usually seen without recent bone marrow stimulation, and a diffuse hypermetabolic infiltrating process in the marrow cannot be excluded. The diffuse marrow infiltrative process is present throughout the thoracic spine. It is also noted that the marrow of the lumbosacral spine involving the length of the spinal axis is diffusely heterogeneous, consistent with diffuse marrow infiltration, and consistent with a bone marrow lymphoma.

8. 2008 (August). Mr. Zapata returned to the hospital on 8/2/2008 presenting with dyspnea, chest pain, and vomiting. He submits to a stomach GE biopsy, and he is diagnosed as having a benign squamous mucosa with mild chronic change, contiguous gastric mucosa with moderate chronic inflammation, and he appears negative for intestinal metaplasia or dysplasia. Some new small bilateral pleural effusions are noted on a scan associated with atelectasis or consolidation, which have increased from the previous exam. **There is an abnormal leukocyte accumulation on his lung's lower quadrant, which is described as "abnormal."** His monocytes measure at 10.6 which is elevated. His total lymphocyte count is measured at 1.7. He stays in the hospital for 18 days and is discharged on 8/20/2008. Although it is stated that there is no evidence of malignancy, Mr. Zapata presents with severe back pain and severe osteonecrosis of his vertebra and spine, and with a worrisome high-metabolic signal discovered throughout his bone marrow suggestive of "a diffuse infiltrative process;" a grade 3 esophagitis and mild gastropathy, and fever are reported, all cultures are negative for microorganisms, he has mixed respiratory and metabolic alkadosis, he is hyperventilating, is diaphoretic, and shows a potassium and magnesium deficit. He is given but cannot tolerate NSIDs (Non Steroidal Inflammatory Drugs). He is given a fentanyl patch, and methadone for pain. His monocyte count is normal, and his bone marrow is "hyper-reactive" on PET. His medical records say that he is provided with and restarted on HAART, although it is not clear in Mr. Zapata resumed HAART at this point or not.

9. 2008 (September). Mr. Zapata indicates profound anemia and normal total T-cell counts.

10. 2008 (October). Mr. Zapata presents in the morgue with jaundice, hepatospenomegaly with necrosis of both organs, hemorrhages through his body, a yeast infection of the lungs but not bone marrow, profound osteonecrosis and bone-marrow hemorrhages, a calcified pancreas, he is emaciated, his intestines have hemorrhages on their outsides (serosal layer), and he has a new organ in his chest known as extramedullary hematopoiesis, suggesting that the body has tried to compensate for his lack of red blood cell production since his bone marrow was wiped out, by providing this extra organ located outside the bone marrow. Mr. Zapata died with profound bone marrow necrosis, hepatosplenomegaly and necrosis of these organs. The official death certificate states that he presented with aspiration pneumonia, an upper GI bleed, DIC, HIV, hypoxemia, hypoglycemia, sepsis, bone marrow hemorrhage, and jaundice. He is deemed an AIDS case.

Although pathology is a science when differential diagnoses are correctly performed and interpreted, it can first and foremost definitively eliminate causes. In this context, it can be decisively stated that the autopsy, pathology, and the clinical data and observations described in this report show clearly that Robert did not suffer from AIDS during the years prior to his death or at the time of his death:

A) Mr. Zapata did not die due to complications of "HIV," "AIDS," or an infectious cause. At 34 years of age, he had just been married and had two children, and tested "HIV" negative 5 months before he was diagnosed with herpes, which causes "HIV" tests to react positive. He was not given a confirmatory WESTERN blot test.
B) Mr. Zapata's T-cell numbers were normal 5 years later although it was claimed he wasn't compliant with HAART near the time immediately preceding his lymphoma diagnosis and treatment.
C) Mr. Zapata's T-cell numbers were normal in July the year he died and again in September they were near normal only a month before he died.
D) Although Mr. Zapata's CD4/CD8 was low in July (3 months before his death, his total WBC count was in the normal range). According to the WHO, total lymphocyte counts are as predictive if not more predictive than CD4/CD8 ratios in predicting demise and death due to AIDS.
E) Sections of Mr. Zapata's enlarged node demonstrate no evidence of effacement, and it was composed predominantly of primary follicles with occasional secondary follicles. The secondary follicles had normal, polarized germinal centers. According to the most recent studies published and discussed in such journals as The Journal of Infectious Diseases and AIDS Clinical Care, the most diagnostic hallmark of long term AIDS progression is seen as fibrosis in the lymph nodes. (Evaluation of the pathogenesis of decreasing $CD4^+$ T cell counts in human immunodeficiency virus type 1–infected patients, receiving successfully suppressive antiretroviral therapy. Nies-Kraske E. et al. , *J Infect Dis*. Jun 1; 199:1648, 2009). No fibrosis was noted, and instead, only normal primary follicles and occasional secondary follicles **with polarized germinal centers** were discovered, making any claim that Mr. Zapata had had AIDS for 8 years or at all a proposition that ignores all of his data and records. Mr. Zapata was intermittently falsely diagnosed and treated with highly toxic medications, each of which have been shown to cause many, if not all of the very

symptoms he presented with. It is very likely that his demise was initiated by these medications, including his lymphoma. To identify the most likely cause(s) of Mr. Zapata's demise and death, it can be decisively stated that:

A) Mr. Zapata died from a cause that was clearly noted in July and August preceding his death as "a diffuse infiltrative process" occurring in his bone marrow, that involved his entire vertebral column, his ribs, and perhaps other bones. The high "patchy" metabolic labeling along with the extensive fibrosis indicates that a cancer, leaving signs that it was a malignant one, ravaged and killed him. This is a rare syndrome, but there have been extensive reviews on the subject at least since 1996 in textbooks and scientific journals (From: Diagnostic Cytopathology Volume 15, Issue 5, pages 421–426, December 1996):

*Fine-needle aspirate of primary lymphoma of bone. Winston M. Htwe M.D., David R. Lucas M.D., Carlos W. M. Bedrossian M.D., James R. Ryan M.D. Section of Cytopathology, Department of Pathology, and Department of Orthopaedic Surgery, Wayne State University School of Medicine, Hutzel Hospital, Detroit, MI. Keywords: * malignant lymphoma * bone neoplasms;* **** fine-needle aspirations (FNA specimens).***

Abstract
Primary lymphoma of bone (PLB) is a rare bone tumor. Fine-needle aspirates (FNA) were done on large destructive bone tumors from 2 elderly men, and both were initially read as inconclusive for malignancy ***because of scant cellularity****. On retrospective study of the* ***FNA slides*** *after examining tissue histology, l****ow numbers of diagnostic cells for lymphoma were recognized on the smears. There was extensive crush artifact, and most intact cells were stripped of their cytoplasm. In neither case was enough material harvested to make cell blocks or to perform special studies. Tissue histology disclosed abundant fibroconnective tissue stroma*** *which probably made it difficult to acquire adequate* ***FNA specimens****.* ***Another FNA done on a postoperative PLB tissue specimen disclosed similar features.*** *Our experience in the 3 cases is consistent with the view that even though smears show scant cellularity, the diagnosis of PLB can* ***at least be suggested*** *by FNA. It is therefore important not to undercall the specimen because of low cellularity, and recommendation for tissue diagnosis can be given. This process* ***is facilitated by a high index of suspicion based on clinical and radiographic findings****. Diagn Cytopathol 1996;15:421–426. © 1996 Wiley-Liss, Inc.*

Other oncologists from the world's largest comprehensive cancer centers have called attention to the importance of looking for this neoplasm in bone marrow, and have described how frequently they encounter it. A group from M.D. Anderson Comprehensive Cancer center reported in Diagnostic Cytopathology (Lin, F., Staerkel, G. and Fanning, T. V. (2003), Cytodiagnosis of primary lymphoma of bone on fine-needle aspiration cytology specimens: Review of 25 cases. Diagnostic Cytopathology, 28: 205–211. doi: 10.1002/dc.10266: 1 Department of Laboratory Medicine, Geisinger Medical Center, Danville, Pennsylvania 2 Department of Pathology, University of Texas MD Anderson Cancer Center, Houston, Texas):

Abstract
Diagnosis of nodal lymphomas on fine-needle aspiration (FNA) cytologic specimens has been well established. However, cytodiagnosis of primary lymphoma of bone has not been well documented because of its rarity. We undertook a retrospective study of 25 cases of FNA cytologic specimens of primary lymphoma of bone. The slides were available for review in 20 cases; each case was evaluated with 15 cytologic features in conjunction with immunophenotyping and available surgical materials. Three diagnostic categories were assigned, including nondiagnostic (4/16%), suspicious (3/12%), and malignant (18/72%). Among the 18 malignant lymphoma, all were diagnosed on the basis of cytologic materials together with immunocytochemistry, except that two cases also relied on the cell blocks. The nondiagnostic and suspicious cases were subsequently confirmed to be malignant lymphoma on the surgical core biopsies. ***Of the 25 cases, 23 cases were large B-cell lymphoma****, one follicular lymphoma large cell type, and one small lymphocytic lymphoma. False-positive or false-negative cases were not present in this study series.* ***In conclusion, the vast majority of primary lymphoma of bone can be accurately diagnosed and classified on FNA cytologic specimens in conjunction with immunocytochemistry.*** *The nondiagnostic and suspicious categories can be further reduced or eliminated by improving FNA techniques or by recommendation of surgical core biopsies together with other techniques such as flow cytometry and molecular analysis. Diagn. Cytopathol. 2003;28:205–211.*

It is important to point out that Mr. Zapata had endured fine needle aspiration and cytological examination, yet the determination of primary bone lymphoma was not made. This potential oversight on the part of the diagnostic team, because they considered him an "AIDS" case, can be appreciated by the fact that in NHL, both of the B and T-cell types, have been noted in approximately ½ of cases studied. Even physicians in countries with a less sophisticated health care systems have appreciated that nearly ½ of patients they studied will present with this tumor, as this following study of 49 patients shows, and that it should be considered especially important to properly assess bone marrow for this cancer because 50% or more patients will show the cancer there if it is looked

for carefully (From The Indian Journal of Pathology and Microbiology, 2009, Volume 52, Issue 3, Page : 332-338 Bone marrow biopsy in non-Hodgkin lymphoma: A morphological study):

"The overall incidence of marrow involvement by NHL was 55.1%."

"Conclusions: Critical examination of BM biopsies can increase the diagnostic accuracy, thereby contributing to the prognosis and appropriate treatment modalities."

Thus the cancer is not so rare or unknown that it isn't now medically recognized world-wide. '

The hypothesis that Mr. Zapata may have long been suffering from a primary bone NHL, as emphasized by the emboldened words in the first abstract presented above from the Wayne State group, also is supported by the observation that fibrosis is a common, if not diagnostic hallmark of many if not most malignant neoplasms. When the observation of severe fibrosis becomes coupled to the detection of "high metabolic labeling," these data strongly suggest, that his bone marrow only could have come to his current state because 1) both the fibrosis and high metabolism detected was due to populations of tumor cells growing in his bones, and because 2) long-term usage of myelosuppressive drugs such as AIDS drugs, together with the toxic effects of R-CHOP cancer chemotherapy, together with possible synergism with such toxic compounds as acyclovir that we are informed in his records was administered for his herpes, all destroy or damage the bone marrow, often irreversibly, and often even after only treatments of short duration. The extramedullary hematopoiesis noted at autopsy indicated that the bone marrow had been under assault, either from the cancer, his drug regimens, or from both, to the point that it could not sustain life, or generate the needed cells (predominantly megakaryocytes, and red blood cell precursors, but not white cells) required to sustain life.
B) The contrast areas noted on his lungs and spleen earlier that year of his death also suggests strongly, that metastasis had begun at least 3 months before his death. Also suggestive of malignant cancer, are the mosaic patterns detected in his lung lesions, which again suggest malignant patterns of tumor perfusion .

Yet the textbooks would suggest that Mr. Zapata's cancer was first manifested as his tonsil tumor, and then spread to his spine's bone marrow. Here is how it is usually presented to medical students:

*Diffuse Large B-cell Lymphoma: Cells are large, with prominent nucleoli and abundant cytoplasm and many mitoses. Most are B-cell, but 20% are T-cell phenotype. CD19, 20, 79a; some have t(14;18); some have Bcl-2 and Bcl-6 expression; linked to EBV infection; negative TdT. Though often localized, **they tend to be aggressive extranodal masses**; seen in adults and children, can be seen in HIV infection.*

Therefore, both Mr. Zapata's rapid demise and his death were the result of "the diffuse infiltrative process" consistent with cancer in his bone marrow, that was first noted three months before his death, which spread to his spleen, lungs, and elsewhere rapidly, and to his tonsil 2 years earlier, as much of B-cell production first occurs in bone marrow. **Therefore it can be said with some degree of certainty that Mr. Zapata's death was most likely caused by a recurrence of lymphoma, or due to a smoldering primary lymphoma in his bone marrow that was missed in 2005.** It also can be stated with some certainty, that due to presence of extramedullary hematopoesis discovered at autopsy, that the bone marrow had been under assault for some time before symptoms of severe debilitating back pain began in July, 2008. It also can be stated with some degree of certainty that the lymphoma originated in his bone marrow but was never detected, and metastasized to his left tonsil in 2005. This series of events, in turn, could have been the result of the mutagen AZT, that was in his HAART cocktail, as it has been reported that some 40%-70% of AZT-treated patients develop this cancer.

Finally, the organ failure and hemorrhaging and DIC observed at the end of his illness was most likely due both to metastatic cancer and to the multiple assaults on his system of nucleoside analogs, R-CHOP cancer chemotherapy, and possibly acyclovir, that he was apparently over-prescribed beginning in 2000. As it is clear in this case because of the blood work results and lymph node biopsies that were normal, that Mr. Zapata could not have been an AIDS patient. It stands to reason and is most likely that the HAART medications, particularly the AZT, had suppressed his hematopoietic system for some years, beginning in March of 2000, when he was falsely diagnosed with "HIV," because he had a herpes infection that has been long known to cross-react with "HIV" tests. What makes this interpretation even more likely is a survey of the medical literature on Medline, where the terms "bone-marrow necrosis, and the constellation of conditions and toxins given to Mr. Zapata are cross-checked. In such an analysis, it appears indeed most likely that the diffuse infiltrate and bone marrow necrosis seen in August in his entire vertebral column's bone marrow, in the lungs, and on his growing spleen indicated that his ultimate cause of death was most likely NHL cancer (either a

primary bone marrow NHL, or primary tonsil-associated NHL, that in turn, was likely induced by AZT, and/ or synergism of the many toxic regimens he was prescribed along with a recurrent or smoldering bone marrow NHL cancer).

The Medline medical database associates the following reported bone marrow necrosis in association with the following syndromes or drugs: Summary of Medline Hits: October 2010 connecting various conditions or drugs to bone marrow necrosis **(not mutagenesis)**:

Bone Marrow Necrosis	**Condition or drug**	**# of studies**
Bone marrow necrosis only		= 615
	+Cancer	**= 1786**
	+Neoplasms	**= 1437**
	+Chemotherapy	**= 1105**
	+Non-Hodgkin's lymphoma	**= 142**
	+NSAIDs (drug)	= 116 (Mr. Zapata couldn't tolerate)
	+HIV	**= 3**
	+ AIDS	= 69 (**don't indicate if drugs were used**)
	+ Nucleoside analogs	**= 19 (cancer,transplant,methylprednazone,chemo)**
	+ acyclovir	= 9* (**valacyclovir**, experimental Feline HerpesVirus infection)
	+R-CHOP	**= 8**
	+Septrim	= 4 (not relevant) (Prednizone, GVHD)
	+ ceftrioxone	= 0
	+ PPI (Proton Pump Inhbitors	= 0
	+ combivir	= 0
	+ sustiva	= 0
	+ chlortrimazole	= 0
	+ pneumococcal vaccine	= 0
	+ fentanyl	= 0
	+ dilaudid	= 0
	+ methadone	= 1 (Not relevant)

- **It is interesting to note that alacyclovir has been given to both people and cats, and in cats is associated with severe bone marrow depression and hepatic necrosis, but CDC says it is OK to use in humans.**

Am J Vet Res. 1997 Oct;58(10):1141-4. Effects of valacyclovir in cats infected with feline herpesvirus 1. Nasisse MP, Dorman DC, Jamison KC, Weigler BJ, Hawkins EC, Stevens JB.

PROCEDURE: Cats were infected with FHV-1 strain 87-727 (300 microliters, 10 (7) plaque-forming units/ml) by ocular and nasal inoculations, and were treated every 6 hours with dextrose (controls) or valacyclovir (60 mg/kg of body weight, PO). RESULTS: All cats developed acute conjunctivitis and rhinitis typical of FHV-1 infection. Beginning between days 6 and 9, valacyclovir-treated cats became noticeably more lethargic and dehydrated than did cats of the control group. Total WBC and neutrophil counts were significantly lower in cats of the valacyclovir group. The experiment was terminated on day 12 **for humane reasons.** Histologic changes attributable to FHV-1 infection were similar in all cats. Additional histologic abnormalities seen only in the valacyclovir-treated cats were **coagulative necrosis of the renal tubular epithelium, centrilobular atrophy and hepatic necrosis, and severe bone marrow depression. CONCLUSIONS:** Cats appear to be uniquely sensitive to the toxic effects of valacyclovir, and even high doses appear not to suppress FHV-1 replication in acutely infected cats.
CLINICAL RELEVANCE: Use of valacyclovir **is of questionable value in cats** with acute FHV-1 infection and, at high doses, the drug may be toxic.

In 2006, this was the CDC's recommended dosages for people with herpes. http://www.cdc.gov/std/treatment/2006/genital-ulcers.htm First Clinical Episode of Genital Herpes, Valacyclovir 1 g orally twice a day for 7–10 day Suppressive Therapy for Recurrent Genital Herpes: Valacyclovir 500 mg orally once a day or Valacyclovir 1.0 g orally once a day.

The numerous normal biopsies obtained from Mr. Zapata's lymph nodes further suggests that primary bone cancer could have ended his life, or a re-emerging NHL that behaved as a diffuse patchy high metabolic labeling syndrome in his bone marrow, and that he couldn't have possibly had an illness like AIDS which is characterized best when fibrosis is found in the lymph nodes, that his doctors so carefully, again and again analyzed without simply reviewing the literature on primary NHL.