

## AFFIDAVIT OF NANCY BANKS MD, MBA

1 I, NANCY T. BANKS, MD, do hereby make the following sworn declaration.  
2 All matters contained herein are of my own personal knowledge unless stated as based  
3 upon information and belief:

- 4 1. I am a resident of Guadalajara, Jalisco, Mexico.
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- 6 2. I am an expert as defined by Kansas law. No previous opinions rendered by me have  
7 been disqualified by any court of law. I have never been found guilty of fraud or  
8 perjury. A copy of my curriculum vitae is attached hereto as Exhibit A, which outlines  
9 my education, training and background.
- 10 3. In 1974 I received a Bachelor of Arts degree with honors with a double major in  
11 Biology and Psychology from Hunter College, a division of the City University of  
12 New York. In 1978, I received an M.D. degree from Harvard University. I completed  
13 an internship in general surgery at the St. Luke's-Columbia Presbyterian Hospital in  
14 1979 and a residency in obstetrics and gynecology at Mt. Sinai Hospital in 1983, both  
15 in New York City. After completing my residency I was appointed as Director of  
16 Outpatient Gynecology at North General Hospital and developed a thriving solo  
17 private practice in obstetrics and gynecology in Nyack, New York. I am board-  
18 certified by the College of Obstetricians and Gynecologists. As a board certified  
19 gynecologist, I am an expert in the care and treatment of sexually transmitted diseases.  
20 I held staff positions at Nyack Hospital, Mt. Sinai Hospital, North General Hospital  
21 and Columbia Presbyterian. The positions at North General and Mt. Sinai involved  
22 teaching and training residents.
- 23 4. In 1996, I completed a Master's degree in Business Administration and Finance in  
24 from Pace University, White Plains Campus in White Plains, New York. With this  
25 degree, I completed a business model for a women's total wellness center that  
26 combined multiple medical disciplines including alternative care modalities in a  
27 coordinated manner under one roof – a model that is now being developed and  
28 launched around the globe.

1 5. In 2010 – after six years of research – I published a book, *AIDS, Opium, Diamonds*  
2 *and Empire, the Deadly Virus of International Greed*". I have lectured about the  
3 history, science and political aspects of the HIV and AIDS crisis in Europe, Mexico  
4 and throughout the United States.

5 6. I have actively reviewed the medical and scientific literature concerning HIV science  
6 and related areas with an emphasis on problems associated with claimed sexual  
7 transmission said virus and HIV diagnostic testing and diagnosis. To date, the  
8 majority of diagnostic tests approved by the FDA for medical use related to HIV  
9 neither claim nor attempt to confirm the actual presence of HIV in a patient's sample.  
10 None of the 35 HIV-related diagnostic tests or devices currently listed on the FDA's  
11 website claim to confirm the actual presence of HIV in any sample with any degree of  
12 stated accuracy.

13 7. Currently, tests for HIV infection are designed to detect serum antibodies produced by  
14 the immune system in response to what are claimed to be protein components of the  
15 virus. To date, there is no practical test for screening that definitely indicates whether  
16 a person has the virus and is infectious. A direct reliable method for determining  
17 infection status of an individual would be to culture the virus from the blood or other  
18 body tissue. This has never been accomplished. Therefore the HIV tests are an  
19 assumption, but not direct proof, that the bands identified on these tests are from a  
20 unique HIV virus. The only way to distinguish between real reactions and cross  
21 reactions is to use HIV isolation. All claims of HIV isolation are based on a set of  
22 phenomena detected in tissue culture, none of which are isolation and none of which  
23 are specific for retroviruses.<sup>1</sup>

24 8. Antibodies are proteins that the body's immune system produces in response to the  
25 presence of germs (i.e. bacteria and viruses). Antibodies do not represent the germ  
26 itself. In fact, these antibodies assist in the elimination of the germ by binding to it,  
27 thereby interfering with its ability to replicate and marking it for digestion by other

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28 <sup>1</sup> Eleni Ppadopoulos-Eleopoulos, Valender F. Turner, John M. Papadimitriou, "Is a Positive Western Blot  
Proof of HIV Infection, *Nature Biotechnology*; 11, 696-707 (1993).

1 cells in the immune system. Furthermore, germ-specific antibodies produced in this  
2 way remain at detectable levels in the body for several months to several years, even  
3 after the complete elimination of the infectious agent from the body. Antibodies to a  
4 germ do not mean the germ is present. Indeed, in the vast majority of cases (such as  
5 polio, small pox, measles, hepatitis, chicken pox), it means the germ has been  
6 neutralized and eliminated from the body.

7 9. The “HIV” antibodies are notoriously non-specific. The scientific literature reveals  
8 numerous studies demonstrating false-positive HIV-antibody reactions on ELISA and  
9 WB, the two main types of HIV antibody tests. A false-positive reaction means that  
10 antibodies to other germs, or naturally circulating antibodies in persons, coincidentally  
11 bind to the purported HIV proteins in test kits. HIV antibody tests have been found to  
12 be positive in Amazonian Indians who had no contact with the outside world, in dogs  
13 at the University of California Davis Veterinary School and in mice of certain  
14 autoimmune strains. The tests have been shown to cross-react with at least 70 other  
15 factors including flu, hepatitis and tetanus vaccines, acute viral infections, other  
16 retroviruses, auto-immune conditions, anti-mitochondria and anti-nuclear antibodies,  
17 among others.<sup>2</sup>

18 10. In addition to these observations, some persons have an overabundance of a broad  
19 spectrum of antibodies due to a condition called hypergammaglobulinemia [HGG].  
20 One would logically expect persons with HGG to have a greater rate of false positive  
21 reactions on any antibody laboratory test, including HIV antibody tests. Numerous  
22 conditions can lead to HGG, including liver disease, connective tissue disease,  
23 lymphoproliferative disorders, malignancy, viral and bacterial infections, autoimmune  
24 conditions, and thyroid disease.<sup>3</sup>

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26  
27 <sup>2</sup> Christine Johnson, “Whose Antibodies are they anyway”, *Continuum*, Sept./Oct., 1996

<sup>3</sup> A. Dispenziere, et al, *Mayo Clin Proc* 2001; 76:476-87.

1 11. It is also well known that people of African descent make more antibodies than  
2 Caucasians to a given stimuli and that HGG is more often found in this population.<sup>4</sup>  
3 Given that HIV tests are antibody tests that measure the level of antibodies in the  
4 blood, it has been found that these tests discriminate by race – that given the same  
5 social and economic factors, the test is 3X as likely to be positive in Hispanics and 5X  
6 as likely to positive in Blacks as in Whites.<sup>5</sup> In a series of studies, Henry Bauer PhD  
7 demonstrated that the frequency of finding a positive HIV test was a non-specific  
8 indicator of physiological stress and in no way reflected the epidemiology of sexually  
9 transmitted diseases. Racial ancestry as an independent variable determines the  
10 relative level of F (HIV) – at all ages, in both sexes, and in groups presumed to be at  
11 low risk for AIDS or HIV infection as well as in those judged to be at high risk.  
12 According to Bauer’s well-researched demographic studies, this explains the variation  
13 of the frequency of positivity between social groups and with age, sex and population  
14 density. These variations were incompatible with the behavior of a sexually  
15 transmitted infection and were more evidence that the HIV test is a non-specific  
16 indicator of physiological stress.

17 12. Bauer found that the correlation between the frequency of HIV positivity in the  
18 United States reflects genomic polymorphisms like those used to track the course of  
19 human migration over the last few hundred thousand years: Genomic patterns that  
20 influence skin color are linked to genomic patterns that modify physiological  
21 responses to certain stresses. People of African ancestry display the strongest response  
22 and people of Asian ancestry the weakest.

23 13. From the beginning, HIV tests were designed to support the assumption of  
24 immune deficiency in high risk populations and were never a direct measure of an

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25 <sup>4</sup> Greidinger EL, Flaherty KT, White B, Rosen A, Wigley FM, Wise RA. African-American race and  
26 antibodies to topoisomerase I are associated with increased severity of scleroderma lung disease. *Chest*  
1998;114:801-7

27 <sup>5</sup> Henry H. Bauer, “The Origin, Persistence and Failings of the HIV/AIDS Theory,” McFarland, Jefferson,  
28 N.C. 2007

1 infectious agent. The tests are notoriously inaccurate, especially in what are  
2 considered to be low-risk populations.<sup>6</sup>

3 14. This then approaches the issue of sexual transmission. From the beginning of the  
4 AIDS era, it was assumed that the “virus” was sexually transmitted (and through blood  
5 products) because early on the disease was confined to men who had sex with men, IV  
6 drug users and hemophiliacs. However, later long term studies on discordant partners  
7 (one who was HIV+ and the other HIV-) have not shown that the transmission has the  
8 characteristics of a sexually transmitted disease. Before the AIDS era, over 20 STDs  
9 had been identified. These diseases have a high infectivity rate that varies from 30 to  
10 60% infectivity after one contact. The long term studies on discordant HIV positive  
11 partners have found that the possibility of male to female transmission is less than 1 in  
12 1000 contacts and the female to male transmission is less than 1 in 9000 contacts.<sup>7</sup>  
13 This means that if discordant partners engaged in sex an average frequency of every  
14 three days it would take from six to twenty-four years for male-to-female transmission  
15 and from 51 to 222 years for female-to-male transmission.

16 15. I have reviewed the testimony of Donna Sweet MD, which is found on pages 160-  
17 187 and 238-246 of the trial transcript. Throughout my years of medical practice, I  
18 have never heard of a physician who voluntarily testified against her own patient for  
19 prosecutors and defense, who relied upon those statements to convict. Generally,  
20 Federal HIPAA rules prevent medical doctors from disclosing patient information to  
21 anyone without a court order. Dr. Sweet’s testimony raises serious questions with  
22 regard not only her competence as a physician, but also of her professional integrity  
23 and duty to her patients.

24 16. Throughout her testimony, Dr. Sweet never referred to Gutierrez as her patient –  
25 although the medical record clearly indicates that she was his primary care physician  
(PCP) for the treatment of HIV. Despite the 68 pages of medical records that her

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26 <sup>6</sup> Michael J. Barry, Paul D. Cleary, and Harvey V. Fineberg, “Screening for HIV Infection: Risks, Benefits,  
27 and the Burden of Proof”, *J Law Medicine & Ethics*; Vol. 14, Issue 5-6 5 June 2007

<sup>7</sup> Nancy S. Padian, et al., “Female to Male Transmission of the Human Immunodeficiency Virus”, *JAMA*,  
September 25, 1991, Vol. 266, 12

1 office faxed to USAF prosecutors on 4 January 2011, those records provided no  
2 evidence that anything more than a few blood tests were taken by a nurse-practitioner.

3 17. After alluding to some unknown credentials from unknown agencies as an HIV  
4 expert, Dr. Sweet claims (p161 7-9) that she saw her first HIV patient in 1983 and that  
5 she “had been lecturing and learning about (HIV) for a couple of years before then,”  
6 which represents the year after she received her medical license in 1980 and at least  
7 three years *before* doctors Robert Gallo and Luc Montagnier claim to have discovered  
8 the virus in 1984. Based upon her testimony and evidence produced from this case, I  
9 am not surprised that “half of (her) practice died on a regular basis” or that “hundreds  
10 and hundreds (died)... over the years” since then (p161 14-17). This is consistent with  
11 reports that a million Americans are injured or killed annually by iatrogenic (doctor-  
12 caused) preventable errors and complications<sup>8</sup> and adverse drug reactions (ADRs)<sup>9</sup>.  
13 Shortly after admitting this, prosecutors “tendered” that Dr. Sweet was “an HIV  
14 expert” with no opposition from DC or the Court (!).

15 18. More importantly, Dr. Sweet never acknowledged that the definition of what  
16 constituted an AIDS diagnosis was expanded in 1993, just at a time when the original  
17 cohort of AIDS patients who often presented with opportunistic infections such as  
18 pneumocystis carinii of the lungs and a skin cancer, Kaposi’s sarcoma, were on the  
19 decline and the predicted epidemic failed to manifest. When the antiviral drug AZT  
20 was introduced, this hastened the demise of many of the early patients. AZT is an  
21 “anti-cancer” drug that had known severe toxicities: it is mutagenic, carcinogenic,  
22 suppresses bone marrow production.<sup>10</sup> However, it was introduced to the market for  
23 the treatment of very sick AIDS patients after the shoddiest of clinical trials. It  
24 accounted for many of the deaths in the original AIDS cohort. The cohort of patients  
25 who were tested and found positive after 1993 were generally found to have entirely  
26 different sets of diseases – or no disease whatsoever. In 1992, the CDC claimed that a  
27 low CD count along with a positive HIV test was sufficient to diagnose AIDS and that

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28 <sup>8</sup> [JAMA, December 21 1994 – Vol 272, No 23](#)

<sup>9</sup> [JAMA. 1998;279\(15\):1200-1205 \(doi:10.1001/jama.279.15.1200\)](#)

<sup>10</sup> see [Zidovudine label information](#)

1 “anti-retroviral” (ARV) drugs could be given. The number of diseases now considered  
2 AIDS-defining diseases is greater than 29. Dr. Sweet did not seem to be aware of the  
3 changing population characteristics of what constitutes an AIDS diagnosis and how  
4 this change in diagnostic criteria effected both demographics and patient longevity.

5 19. Dr. Sweet claims that the HIV test, “has become very, very sensitive and specific  
6 with the new – especially rapid test” (p164 3-4). **Sensitivity** measures how often a test  
7 is positive when you already know what you are testing for is present. **Specificity**  
8 expresses how often a test is positive when a patient DOES NOT have the condition.  
9 In all HIV test literature, sensitivity and specificity are not based on measurements of  
10 the test against an isolated virus, but is a concordant measure between two tests that do  
11 not detect HIV or HIV antibodies. Careful examination of the manufacturer’s label  
12 information makes this clear. So, for example, while breath, blood and urine tests  
13 accurately measure known blood-alcohol content as it is consumed and metabolized,  
14 HIV tests more closely resemble lie detector devices, which are limited (like HIV  
15 tests) on the operator’s subjective evaluation. So while lie detector tests may be 99%  
16 sensitive and specific to each other, neither test can establish whether someone has  
17 actually lied. For this reason, clinicians are admonished by manufacturers and  
18 laboratories not to use the test for diagnostic purposes. Any clinician that relies on the  
19 results of such tests for diagnostic purposes is demonstrating questionable competency.

20 20. Given these limitations, let’s assume that HIV tests said to have a 99% sensitivity  
21 and specificity were applied in a military setting. If one uses the law of positive  
22 predictive value, any test, regardless of its accuracy, performs poorly in a population  
23 where few people are expected to be infected. According to demographic studies done  
24 on military recruits, the expected frequency of HIV positivity per 100,000 is ~.12%.<sup>11</sup>  
25 If the ~.12% is rounded off to .1%, under the best of circumstances, the positive  
26 predictive value of this test in a military population would be less than 10%. For this  
27 reason alone, Dr. Sweet statement that the tests are “very, very sensitive and quite

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28 <sup>11</sup> Dondero, T.J., & Gill, O. N. (1991). Large scale HIV surveys. What has been learned? *AIDS*, 5,  
Supplement 2, S63-S69.]

1 specific” is at best grossly inaccurate. Given that Gutierrez is a heterosexual military  
2 veteran who would fall into this low risk category, Dr. Sweet knew, or should have  
3 known, this information and applied it to her ASYMPTOMATIC patient. In light of  
4 these facts, the medical record and Dr. Sweet’s testimony, it’s hard to imagine how a  
5 competent physician would not have considered the likelihood that Gutierrez’ alleged  
6 test results were false-positive.

7 21. Although Dr. Sweet claims that HIV tests are **sensitive** and **specific**, she never  
8 mentioned – nor was she ever asked about – HIV test **reliability**. An “HIV expert”  
9 would have known that there have been repeated recalls of “HIV” antibody tests  
10 during the past decade. Between 2004 and 2011, the FDA Biologic Website listed no  
11 fewer than ten.<sup>12</sup> There is no evidence that Dr. Sweet or any of Gutierrez’ other  
12 clinicians made any attempt to identify which HIV tests were used to diagnose their  
13 patient to ensure that his test was not subject to an FDA recall. Dr. Sweet appears to  
14 have made no effort to examine what test was used to diagnose her patient, how it was  
15 administered or whether his test fell into one of the FDA or manufacturers’ recall  
16 categories.

17 22. Dr. Sweet testified that once an individual is diagnosed with HIV, “The next step  
18 is to find out – to stage them essentially just like you would cancer or any other  
19 disease” (page 165 line 1). Given that a) manufacturers and labs warn clinicians that  
20 HIV tests cannot be used for diagnostic purposes and b) researchers have established  
21 that HIV test have a low positive predictive value in the defendant’s population  
22 category and c) that Gutierrez’ test is 10 times as likely to be false positive as it is to be  
23 a true positive,<sup>13</sup> an examination of his recent medical history would have been the  
24 first appropriate response before a complete physical examination for physical  
25 symptoms of HIV infection is conducted. Once a diagnosis is made based on a  
26 complete and thorough assessment of the patients history and physical examination, a

26 <sup>12</sup> <http://www.fda.gov/BiologicsBloodVaccines/SafetyAvailability/Recalls/default.htm>

27 <sup>13</sup> Burke, D.S., Brundage, J.F., Redfield, R.R. et al. 1988. Measurement of the False Positive Rate in a  
28 Screening Program for Human Immunodeficiency Virus Infections. *NEJM* 319:961-964..

1 competent physician would consider all possible diagnostic alternatives before staging  
2 the progression of the alleged disease.

3 23. False-positive reactions are known to be caused by influenza vaccinations,<sup>14 15 16</sup>  
4 <sup>17 18 19</sup> exposure to viral vaccines,<sup>20</sup> natural occurring antibodies,<sup>21 22</sup> tetanus vaccines  
5 <sup>23</sup> and cross-react with at least 70 other factors.<sup>24</sup> According to medical records,  
6 Gutierrez received 43 vaccinations, 17 of which were administered concurrently with  
7 his initial HIV test and subsequent tests. These vaccines contain pathogens and  
8 adjuvants that are known to compromise immune function, cause injury and death.  
9 There is no evidence in the medical record or testimony that Dr. Sweet made any  
10 attempt to rule out known cross-reactors.

11 24. Since the initial test was never presented during the testimony, it is unknown  
12 which if any antibodies on the HIV antibody test were noted to be positive. There is a  
13 body of literature on the specific cross-reactivity of each of the identified "HIV"  
14 glycoproteins from the three identified sites: env (envelope), gag (group specific  
15 antigen) and pol (polymerase-and enzyme). These are the identified genetic areas of  
16 the virus that are said to code for the glycoproteins. However, there are multiple  
17 indications that the HIV antibody tests may be the result of antigenic stimulation other  
18 than HIV. For example, Amazonian Indians who had no contact with individuals

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18 <sup>14</sup> Mackenzie W, Davis J, Peterson D. et al. 1992. Multiple false-positive serologic tests for HIV, HTLV-1  
19 and hepatitis C following influenza vaccination, 1991. JAMA. 268:1015-1017.

20 <sup>15</sup> Challakere K, Rapaport M. 1993. False-positive human immunodeficiency virus type 1 ELISA results in  
21 low-risk subjects. West. J. Med. 159(2):214-215.

22 <sup>16</sup> Arnold NL, Slade RA, Jones MM, et al. 1994. Donor follow up of influenza vaccine-related multiple viral  
23 enzyme immunoassay reactivity. Vox Sanguinis. 67:191.

24 <sup>17</sup> Hisa J. 1993. False-positive ELISA for human immunodeficiency virus after influenza vaccination. JID.  
25 167:989.

26 <sup>18</sup> Cordes R, Ryan M. 1995. Pitfalls in HIV testing. Postgraduate Medicine. 98:177.

27 <sup>19</sup> Profitt MR, Yen-Lieberman B. 1993. Laboratory diagnosis of human immunodeficiency virus infection.  
28 Inf. Dis. Clin. North Am. 7:203

<sup>20</sup> Cordes R, Ryan M. 1995. Pitfalls in HIV testing. Postgraduate Medicine. 98:177.

<sup>21</sup> Barbacid M, Bolgnesi D, Aaronson S. 1980. Humans have antibodies capable of recognizing oncoviral  
glycoproteins: Demonstration that these antibodies are formed in response to cellular modification of  
glycoproteins rather than as consequence of exposure to virus. Proc. Natl. Acad. Sci. 77:1617-1621.

<sup>22</sup> Healey D, Bolton W. 1993. Apparent HIV-1 glycoprotein reactivity on Western blot in uninfected blood  
donors. AIDS. 7:655-658

<sup>23</sup> Pearlman ES, Ballas SK. 1994. False-positive human immunodeficiency virus screening test related to  
rabies vaccination. Arch. Pathol. Lab. Med. 118-805

<sup>24</sup> <http://www.cwbpi.com/AIDS/reports/HIV-false%20list.pdf>

1 outside their communities and have no AIDS have a 3.3 to 13.3% HIV Western Blot  
2 seropositivity rate – a higher rate than military recruits.<sup>25</sup> Germaine to the Gutierrez  
3 case, in a study of 1.2 million applicants to the U.S. military considered low risk, 1%  
4 or 12,000 had a first time positive HIV ELISA test. Only 2,000 were shown to be WB  
5 positive, which left 10,000 positive ELISAs that probably reacted for reasons other  
6 than “HIV antibodies” – a prime example of cross-reactivity<sup>26</sup>. The medical records  
7 and Dr. Sweet’s testimony suggest that neither Dr. Sweet nor any of Gutierrez’ other  
8 clinicians ever considered this possibility.

9 25. Mice of certain autoimmune strains make antibodies against HIV proteins. A  
10 study from the University of California Davis Veterinary teaching hospital reported  
11 that 50% of dogs’ blood samples “reacted with one or more HIV recombinant  
12 proteins.”<sup>27</sup> Assuming that California dogs are not infected with HIV (as did the  
13 authors), one must conclude that the data are further proof of antibody cross reactivity  
14 to many proteins. This is the caveat that an “HIV expert” should be familiar with.  
15 This means that so-called HIV+ individuals are not necessarily infected with what  
16 their antibodies imply in HIV tests.

17 26. Even if the HIV antibody test are positive, the medical “standard of care” requires  
18 “HIV experts” to conduct a thorough history and physical examination of their patient  
19 to evaluate their identifiable risks (if any) for the possibility of developing a future  
20 cellular immune deficiency status. Gutierrez’ overall health was satisfactory at the  
21 time that Dr. Sweet first evaluated him and there is no indication that Gutierrez was  
22 ever evaluated for known metabolic defects in the cellular immune system that would  
23 indicate that he might be a candidate for progression to AIDS. Such metabolic  
24 deficiencies are well-described in the medical literature and include nutrient  
25 deficiencies such as selenium<sup>28</sup> and glutathione, among others.

26 <sup>25</sup> Rodriquez, L., Dewhurst, S., Sinangil, F. et al. 1985. Antibodies to HTLV-III/LAV among Aboriginal  
27 Amazonian Indians in Venezuela. *Lancet* II:1098-1100.

28 <sup>26</sup> Burke et al, *NEJM* 319:961-964.

<sup>27</sup> Strandstrom, *Cancer Research* 1990;50:5628s-5630s

<sup>28</sup> J.P. Breux, J.P. Venot, et al., “Selenium deficiency in HIV infected patients”, *Int Conf AIDS*, 1996 Jul 7-  
12;11:124 (abstract no. Mo.B.1395)

1 27. When asked about CD4 counts, Dr. Sweet stated that the “CD4 count really kind  
2 of comes down, the virus HIV literally destroy. It’s a lymph destructive virus that  
3 destroys those cells such that you end up with no immune system, and that’s why  
4 people with AIDS die.” (page 166 11-15) Again, Dr. Sweet is misinformed. After  
5 successfully mapping the retrovirus genome and being admitted to the National  
6 Academy of Sciences, UC Professor Peter Duesburg PhD assured the world 25 years  
7 ago that retroviruses are not and have never been cytopathic – they don’t kill the host  
8 cell.<sup>29</sup> More recent research confirms this, concluding that “the HIV replication elicits  
9 little cytopathic effect in productively infected cells and that CD4+T lymphocytes are  
10 eroded by other mechanisms.” The researchers noted that...”no evidence was found  
11 for marked direct or indirect cytopathic effects in HIV-infected cells. It suggests that  
12 HIV erodes CD4+T lymphocytes by other mechanisms.”<sup>30</sup> By analogy, although fire  
13 trucks generally appear at structure fires and rarely visit buildings where no emergency  
14 exists, there is no evidence that fire trucks cause structure fires – or that the  
15 suppression of fire trucks would prevent the progression of structure fires.

15 28. It is still an unproven supposition that HIV causes immune deficiency by  
16 destroying CD4+T lymphocytes. What is even more important is that it has been  
17 discovered that there are subsets of CD4+T cells (TH1 and TH2) that perform different  
18 functions and respond to different cell signals. Therefore, if HIV indeed were killing  
19 CD4+T cells, it would be doing it selectively. In 1989, experts discovered that  
20 there are at least 2 subsets of CD4+T cells (more have since been discovered) that have  
21 different functional capabilities. This knowledge is fundamental to understanding the  
22 underlying pathology of those people who are at risk for developing an “immune  
23 deficiency”. They do not develop a global immune deficiency like the boy in the  
24 bubble who developed SCID--severe combined immune deficiency in which neither  
25 the TH1 or the TH2 system is working. The problem in AIDS remains largely

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26 <sup>29</sup> [Duesberg, Proc. Nat'l Acad. Sci. USA, Vol. 86, pp. 755-764, February 1989](#)

27 <sup>30</sup> George A., Funk, Annette Oxenius, et al, “HIV Replication Elicits Little Cytopathic Effects in Vivo:  
28 Analysis of Surrogate Markers for Virus Production, Cytotoxic T Cell Response and Infected Cell Death”,  
*J of Med Virology*, 78:1141-1146 (2006)

1 confined to the TH1 system. The TH1 CD4+T cells produce a poison gas, nitric  
2 oxide, and are responsible for what is called “cell mediated immunity.”<sup>31</sup> These TH1  
3 cells, using this poison nitric oxide gas, function to kill intracellular parasites. The  
4 decrease in the ability of these cells to make nitric oxide is the cellular defect said to  
5 be the problem in people who develop AIDS. This is why they develop what are called  
6 “opportunistic infections”--the nitric oxide gas is no longer being produced and  
7 therefore the intracellular parasites can survive and cause disease. A Nobel Prize was  
8 awarded in 1998 to the researchers who discovered the important cell signaling and  
9 other functional roles for nitric oxide.<sup>32</sup>

10 29. The other functional type of CD4+T cell, TH2 migrates out of the blood stream  
11 and into the bone marrow and lymph where it signals to other immune cells known as  
12 B cells to make more antibody. This is called the humoral response. In AIDS, unlike  
13 the “bubble boy” syndrome, the entire immune system is not dysfunctional but is  
14 primarily the TH1 system. The TH2 system is working and is why patients can  
15 continue to mount an antibody response and fight off most bacterial infections. These  
16 two systems respond to different messengers called cytokines and there is a fine  
17 balance between the two cell populations in a healthy individual. It is the imbalance in  
18 this system and a TH1 to Th2 switch that appears to be the critical step in the  
19 progression to AIDS.<sup>33</sup> Dr. Sweet appeared to have no awareness or knowledge of the  
20 dual nature of the CD4+T cell system and how a working knowledge of this system  
21 could have been used to initiate appropriate compensatory therapy..

22 30. Glutathione is a tri-peptide (3 amino acid molecule) and it is levels of glutathione  
23 in antigen-presenting cells that determine whether Th1 or Th2 response patterns  
24 predominate.<sup>34</sup> “HIV experts” have known since 1989 that total and reduced  
25 concentrations of glutathione in the plasma of HIV infected subjects who go on to

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26 <sup>31</sup> Mossman and Coffman (TH1 and TH2 Cells: Different Patterns of Lymphokine Secretion Lead to  
27 Different Functional Properties”, *Ann Rev Immunol*, 1989.7:145-73).

28 <sup>32</sup> <http://www.experiment-resources.com/nitric-oxide-in-cardiovascular-system.html>

29 <sup>33</sup> Mario Clerici and Gene M. Sheares, “A TH1 →TH2 switch is a critical step in the etiology of HIV  
30 infection, *Immunology Today*, Vol. 14:3 p 108-111.

31 <sup>34</sup> Jeffrey D. Peterson, Leonore A. Herzenberg, et al, “Glutathione levels in antigen-presenting cells  
32 modulate Th1 versus Th2 response patterns, *Proc Nat Acad Sci USA*, Vol. 95, pp 3071-3076, March 1998.

1 develop AIDS are about 30% of those in normal individuals and 60% lower in the  
2 epithelial lining of the lung.<sup>35</sup>

3 31. Dr. Sweet testified that “the CD4 cell... is part of the cell-mediated immune  
4 system that keeps us protected from intracellular pathogens” (p 165 19-21). It is well  
5 known that glutathione depletion inhibits lymphocyte activation and that glutathione is  
6 critical for the function of natural killer cells and for lymphocyte mediated  
7 cytotoxicity. In other words, glutathione is necessary for the functioning of the cell  
8 mediated system that Dr. Sweet testified was the problem in people who develop  
9 AIDS. Yet the medical record and testimony provide no evidence that Dr. Sweet or  
10 any other clinician evaluated Gutierrez for nutritional deficiencies such as glutathione.

11 32. It is also well known that many other factors can suppress the CD4+T cells. For  
12 example women have a higher count than men. Other viral infections can suppress the  
13 count as well as time of day. The count is lower in the morning than in the afternoon  
14 due to the diurnal variation in cortisol output from the adrenal glands. The World  
15 Health Organization (WHO) conducted a study in Africa which showed that HIV  
16 negative populations can have T-cell counts below 350, a number that would,  
17 according to WHO guidelines, qualify for an AIDS diagnosis in HIV+ populations.  
18 The same study found that HIV-positive patients who started AIDS drug treatment  
19 with low T cell counts had the same survival outcomes as HIV positives that began  
20 treatment with high T cell counts.<sup>36</sup> Researchers at the University of California at Los  
21 Angeles School of Medicine found that 5% of healthy persons seeking life insurance  
22 had abnormal T cell counts and that the numbers appeared to be a stable finding.<sup>37</sup>  
23 There is no evidence in the medical record or testimony that Dr. Sweet considered  
24 reasons unrelated to HIV for Gutierrez’s T cell count to be falling. Low CD4+  
25 lymphocyte counts are associated with a variety of conditions including many viral  
26 infections, bacterial infections, parasitic infections, sepsis, tuberculosis,

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25 <sup>35</sup>(R. H. Buhl, et al, “Systemic Glutathione Deficiency in HIV positive symptom free individuals”, *Lancet*  
26 1989: 1294-98

26 <sup>36</sup>[Journal of Infectious Diseases, Vol. 194 p 1450](#)

27 <sup>37</sup>[Rett, K., Wicklmayr, M., Dietze, G. J. & Schwabing, K., 1988. Abnormal T-cell subsets in normal  
28 persons. NEJM 319:1608-1609.](#)

1 coccidiomycosis, burns, trauma, intravenous injections of foreign proteins ( e.g.  
2 vaccines), malnutrition, over-exercising, pregnancy, corticosteroid use, diurnal  
3 variation, psychological stress and social isolation. By analogy, using an HIV, viral  
4 load, CD4 and other blood tests to identify HIV infection is like using a lie detector  
5 test to detect lies. Without a careful examination of the corroborating evidence, the  
6 tests are meaningless.

7 33. Chronic hypercortisolism (elevated cortisol) as found in people under chronic  
8 stress has been shown to selectively reduce the number of CD4+T cells. Telling  
9 someone that they have an incurable life-ending sexually transmitted disease is reason  
10 enough to create a long term stress pattern in an individual, which in Gutierrez's case  
11 was compounded by his status as a career USAF sergeant in isolation, facing a court  
12 martial, prison and a dishonorable discharge by accusations by his own wife and the  
13 willing testimony of his personal physician.<sup>38</sup> There is no evidence in the record that  
14 Dr. Sweet ever considered any of these factors.

15 34. Regarding viral loads (page 167 21-23), Dr. Sweet testified that a person is "More  
16 easily infective to someone else; the higher your viral load, the more likely you are to  
17 infect someone either with blood or sexual secretions." Dr. Sweet failed to explain  
18 that – if billions of HIV are present – why an arcane DNA fragment amplification  
19 measuring technique was used to locate them. Other common viruses infect other  
20 immune cells, including T cells, Cytomegalovirus (which infects 40% of all  
21 Americans), Epstein Barr (50%), Hepatitis B (5%), Herpes simplex 1 (65%) and 2  
22 (40%). These viruses are cytotoxic and actively infect one third or more of the target  
23 cells. Since HIV can be barely found in the target cell and can only be identified by  
24 this gene amplification technique, it defies logic and science that HIV is cytotoxic  
25 when it infects fewer cells than other viral diseases that are self limiting.

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27 <sup>38</sup> A. S. Fauci, D. C. Dale, "The effect of hydrocortisone on the kinetics of normal human lymphocytes,  
28 *Blood*, 46:235, 1975.

1 35. In a 2006 study of 2800 HIV+ people, doctors Benigno Rodriguez and Michael  
2 Lederman<sup>39</sup> found that viral load failed to predict or explain immune status in more  
3 than 90% of cases: "Viral load is only able to predict progression to disease in 4% to  
4 6% of HIV-positives studied, challenging much of the basis for current AIDS science  
5 and treatment policy." Dr. Rodriguez said that "If you look at the number of cells that  
6 are actually infected (with HIV), we are talking less than 1 percent... But in reality, the  
7 individual may have lost 20, 30, 50 percent of his immune cells."<sup>40</sup> This again would  
8 underscore the lack of evidence for HIV being cytopathic.

9 36. Dr. Sweet testified that "absent medical intervention" (drugs), a person will die  
10 from HIV (p172 1-3). However a study from Denmark's Dr. Nicoliah Lohse found  
11 that 25% of those "HIV positive" in the year 2000-2005 cohort who refused anti-viral  
12 drugs had a lower mortality rate compared to those who were treated by drugs. The  
13 study also found that those HIV positives typically had high health risks such as  
14 smoking, alcohol and other drug addiction. These habits, absent HIV infection, are  
15 known to compromise immune function and shorten the life span.<sup>41</sup> According to  
16 Nobel Laureate (2009) and HIV discoverer Luc Montagnier MD, individuals with a  
17 healthy immune system can "get rid of HIV within a few weeks without drugs," but  
18 that clinicians push drugs and vaccines because they cannot profit by selling clean  
19 water and good nutrition.<sup>42</sup> These and other reports clearly demonstrate that having  
20 "HIV" antibodies does not always demand viral treatments.

21 37. The use a antiretroviral drugs described by Dr. Sweet are known to cause severe  
22 injury and death attributable to non-AIDS diseases. Increased mortality from liver,  
23 cardiovascular and pulmonary diseases as well as non-AIDS malignancies were  
24 associated with higher CD4 cell counts at HAART (highly active anti-retroviral  
25 therapy) initiation.<sup>43</sup> What these studies and others demonstrate is that it is possible to

26 <sup>39</sup> Rodriguez B, Sethi AK, Cheruvu VK, et al. Predictive value of plasma HIV RNA level on rate of  
27 CD4 T-cell decline in untreated HIV infection. JAMA 296 (12):1498-506, 2006

28 <sup>40</sup> <http://www.medicalnewstoday.com/articles/79047.php>

<sup>41</sup> *Annals of Internal Medicine*, Lohse et al, January 16, 2007 vol. 146 no. 2 87-95

<sup>42</sup> Video, [House of Numbers \(2009\)](#)

<sup>43</sup> J Acquir Immune Defic Syndr. 2007 Mar 1;44(3):364

1 live close to a normal life span not taking ARVs and that taking ARVs has the  
2 potential risk of death, not from any AIDS related disease, but from the complications  
3 of the drugs themselves.

4 38. As to the risk of infecting a partner through sexual contact. Dr. Sweet testified  
5 that “It’s difficult to have a lot of evidence. But the quote is that it is somewhere  
6 between 10 and 20 positives per 10,000 encounters. That’s sort of high-end. There  
7 are other people that would say 1 out of 10,000 to 1 out of 100,000 given encounters”  
8 (p178 21-23). This very low recorded transmission rate is not characteristic of a  
9 sexually transmitted disease. Before the AIDS era, more than 20 STDs were  
10 identified. These diseases have a high infectivity rate that varies from 30 to 60%  
11 infectivity after one contact. With male to female transmission rates recorded to be 1  
12 per 1000 encounters, assuming on average 3 encounter per week, it would take from  
13 six to twenty four years for the virus to be transmitted. Hemophiliacs like Ryan White  
14 are seldom discussed, because studies on discordant HIV positive hemophiliacs with  
15 their partners has shown very low to zero transmission even without condom use.<sup>44</sup>

16 39. Based upon the medical records and Dr. Sweet’s testimony, it is clear that Dr.  
17 Sweet failed to meet the minimum medical standard of care and that she did not  
18 perform as a competent medical expert on the topic of HIV and AIDS. By her own  
19 account, “hundreds of patients have died” while under her care, a feat that is rarely  
20 matched by other medical professionals. Dr. Sweet’s testimony falsely suggests and  
21 supports the prosecution’s unsupportable contention that Defendant Gutierrez was  
22 infected with an incurable and deadly infectious disease that could have only been  
23 transferred, once every six to twenty-four years. Assuming that evidence of  
24 cytotoxicity exists, the risks involved and cited by Dr. Sweet are even less than the  
25 known mortality of women who are injured or killed by complications related to  
26 pregnancy and childbirth. The idea of charging fathers with aggravated assault or  
27 manslaughter is an absurdity. This may be why prosecutors were unable to produce a

28 <sup>44</sup> [JAMA, September 25, 1991 – Vol. 266. 12](#)

1 credible witness who was willing to render a formal sworn medical opinion that David  
2 Gutierrez was, in fact, infected with HIV.

3 40. I reviewed the office medical records that Dr. Sweet submitted before trial. The  
4 records are astounding for an absence of a confirmation that Gutierrez actually did  
5 have a positive HIV test. Every year thousands of patients are unnecessarily injured  
6 because physicians fail to follow a bare minimum standard of care. There are  
7 numerous stories of patients having the wrong appendage operated on, having the  
8 wrong organ removed and sometimes even operating on or doing a procedure on the  
9 wrong patient. These errors usually result because the treating physician failed to  
10 double check the results of imaging studies and blood work to assure that a.) he/she  
11 has the correct patient and b.) the patient has the correct diagnosis. Relying on  
12 hearsay has never met the minimum standard of care when someone's life is on the  
13 line. The office records indicate that Dr. Sweet relied on a report that Gutierrez was  
14 HIV positive, but the records indicated that she failed to make any attempt to verify  
15 this finding.

16 41. An ESR (erythrocyte sedimentation rate) is a nonspecific screening test for  
17 various inflammatory diseases. The test measures the distance that red blood cells  
18 settle in unclotted blood toward the bottom of a specially marked tube. For men under  
19 50, the results should be under 15 mm/hr. On January 20, 2009 it was noted that  
20 Gutierrez had an very elevated ESR of 120. It was noted that the laboratory that ran  
21 the test was called to assess if that were a correct number. The lab assured that it was  
22 and a note was made to recheck the ESR at a future date, but no further ESR studies  
23 are found in the chart. An ESR can be elevated in a variety of conditions, including  
24 the after effects of various vaccines and can result from both the vaccine and the  
25 adjuvants that are placed in the vaccines to improve their efficiency.

26 42. Gutierrez received a total of 43 different vaccines between 1991 and 2010. On  
27 6 November 2008, Gutierrez received an "influenza split virus." Two months later, he  
28 was scheduled for his first appointment with Dr. Sweet. The record shows that his

1 initial and subsequent evaluations (13 Jan 2009 to 6 May 2010) were conducted not by  
2 Dr. Sweet, but by nurse practitioner Kathryn D. Thiessen ARNP. Nurse Thiessen  
3 noted that Gutierrez was not acutely ill: there was no fever, lymphadenopathy or  
4 elevated temperature, yet he had a very abnormal ESR, which was never explored as a  
5 part of his “differential diagnosis.”

6 43. During the 12 months before Gutierrez’ first visit at Dr. Sweet’s office in January  
7 2009, clinicians at the 31<sup>st</sup> Medical Group treated Sgt. Gutierrez for a knee injury and  
8 prescribed the medication, PIROXICAM ([Feldene](#)) 20 mg.<sup>45</sup> Feldene is one of the  
9 non-steroidal anti-inflammatory drugs (NSAIDs) which Gutierrez was taking for his  
10 torn meniscus (7 Feb 2008). Feldene can cause skin rashes and is also noted to cause  
11 leukopenia – a decrease in the white blood cell count (wbc). This is important because  
12 the patient had a history of skin rashes and three months later was noted to have a dip  
13 in his wbc. There is no evidence that Dr. Sweet ever considered the possibility that the  
14 patient’s chronic use of this particular NSAID could have been associated with the dip  
15 in his CD4+T cell lymphocyte count.

16 44. As noted above, there are many reasons for a decrease in the CD4+T cell count  
17 including diurnal variations. Notes also indicate that Gutierrez had elevated  
18 triglycerides. It is recommended that the lipid profile be done on a “fasting stomach,”  
19 which generally means a morning blood test. If the CD4 was drawn at the same time  
20 as a fasting test, it could have been lower simply because it was morning. It is also  
21 known that the CD4 has an inverse relationship with cortisol levels. Cortisol rises in  
22 the morning, pushing the absolute number of lymphocytes in the blood stream into the  
23 periphery.

24 45. Although the medical record shows that Gutierrez remained ASYMPTOMATIC  
25 through 17 Nov 2010, someone from Dr. Sweet’s office issued a prescription to him  
26 for ATRIPLA<sup>46</sup> on 16 Feb 2010.<sup>47</sup> Atripla is one of the many highly toxic “black box”

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26 <sup>45</sup> see [label information, Pfizer Labs, Rev. Aug. 2010](#)

27 <sup>46</sup> [Atripla label information](#), 21-937-GS-006 January 2010

28 <sup>47</sup> [History & Physical Report #7](#), 16 Feb 2010

1 drugs, which is a combination of the “black box” drugs TENOFOVIR<sup>48</sup>,  
2 EMTRICITABINE<sup>49</sup>, and EFAVIREZ<sup>50</sup>. According to the product label information,  
3 the combination of these three toxins are known to **cause** bone reduction, fat  
4 redistribution, lactic acidosis, hepatomegaly, steatosis, neuropathy, hypersensitivity  
5 reactions and liver toxicity and immune reconstitution syndrome (IRS)<sup>51</sup>, which  
6 presents symptoms virtually identical to those used to identify the onset of “full blown  
7 AIDS.” Like AIDS, those symptoms can include fever, night sweats, weight loss,  
8 fatigue, generalized aching, enlarged lymph nodes, draining sinus, a cough or wheeze,  
9 dyspnea, chest or abdominal pain, nausea, vomiting, diarrhea, bloating, headache, joint  
10 pain/swelling, back pain/paraparesis, chest wall swelling, skin lesions, and urinary  
11 retention.<sup>52</sup>

12 46. On 20 Jan 2009, a Truegene HIV-1 test is noted in the chart. The HIV-1 typing  
13 tests simply look at two gene codes for enzyme (a type of protein) regions for protease  
14 and reverse transcriptase. Both of these enzymes are found in all of life processes and  
15 are not unique to the HIV retrovirus. This is why the package insert contains the  
16 disclaimer that the test is not definitive for assessing the presence of a unique HIV  
17 virus. The “indirect markers” indicate that you might have a virus but that those  
18 markers can be present even in the absence of a particular viral infection. The test  
19 does not establish whether the enzymes detected belong to any particular virus, or even  
20 to the patient’s own protease and reverse transcriptase genomes. Despite these facts,  
21 there is no indication that Dr. Sweet, her nurse practitioner, or any other HIV-treating  
22 clinician made any attempt to competently diagnose Gutierrez before treating him with  
23 Atripla.

24 47. Based upon the aforementioned facts as well as my training and expertise, it is my  
25 opinion that prosecutors have produced no evidence that can be used to reasonably

26 <sup>48</sup> [Tenofovir label information](#), 21-356-GS-025 March 2010

27 <sup>49</sup> [Emtriva label information](#), DGS-21-500-896 22 06 2007

28 <sup>50</sup> [Sustiva label information](#), rev. September 2009

<sup>51</sup> [Atripla label information](#), 21-937-GS-006 January 2010

<sup>52</sup> [HIV/AIDS, CID 2005;41 \(15 November 2006\), 1483](#)

1 conclude with any degree of medical or scientific certainty, that Technical Sergeant  
2 David Gutierrez was ever, or currently is, infected with what is called HIV.

3  
4 I, NANCY BANKS, do hereby swear under penalty of perjury under the laws of  
5 the State of Kansas that the foregoing, consisting of TWENTY of TWENTY pages is  
6 true and correct.

7 Executed this 19th day of APRIL, 2011 in GUADALAJARA, Jalisco Mexico.

8  
9   
10 \_\_\_\_\_  
11 NANCY BANKS, M.D.