



AIDS: “It’s the bacteria, stupid!”

Lawrence Broxmeyer ^{a,*}, Alan Cantwell ^b

^a C/o Med-America Research, 208-11 Estates Drive, Bayside, NY 11360, USA

^b Kaiser Permanente Medical Center, 11668 Sherman Way, North Hollywood, CA 91605, USA

Received 1 May 2008; accepted 4 June 2008

Summary Acid-fast tuberculous mycobacterial infections are common in AIDS and are regarded as secondary “opportunistic infections.” According to the National Institute of Allergy and Infectious Diseases, TB is the major attributable cause of death in AIDS patients. Could such bacteria play a primary or causative role in AIDS?

Certainly, In screening tests for HIV, there is frequent, up to 70%, cross-reactivity, between the gag and pol proteins of HIV and patients with mycobacterial infections such as tuberculosis. By 1972, five years before gays started dying in the U.S., Rolland wrote *Genital Tuberculosis, a Forgotten Disease?* And ironically, in 1979, on the eve of AIDS recognition, Gondzik and Jasiewicz showed that even in the laboratory, genitally infected tubercular male guinea pigs could infect healthy females through their semen by an HIV-compatible ratio of 1 in 6 or 17%, prompting him to warn his patients that not only was tuberculosis a sexually transmitted disease, but also the necessity of the application of suitable contraceptives, such as condoms, to avoid it. Gondzik’s solution and date of publication are chilling; his findings significant.

Since 1982 Cantwell et al found acid-fast bacteria closely related to tuberculosis (TB) and atypical tuberculosis in AIDS tissue. On the other hand molecular biologist and virologist Duesberg, who originally defined retroviral ultrastructure, has made it clear that HIV is not the cause of AIDS and that the so-called AIDS retrovirus has never been isolated in its pure state. Dr. Etienne de Harven, first to examine retroviruses under the electron, agrees. In 1993 HIV co-discoverer Luc Montagnier reported on cell-wall-deficient (CWD) bacteria which he called “mycoplasma” in AIDS tissue. He suspected these as a necessary “co-factor” for AIDS. Remarkably, Montagnier remained silent on Cantwell’s reports of acid-fast bacteria which could simulate “mycoplasma” in AIDS tissue. Mattman makes clear that the differentiation between mycoplasma and CWD bacteria is difficult at best and cites Pachas’s 1985 study wherein one mycoplasma was actually mistaken for a CWD form of a bacterium closely related to the mycobacteria.

It is important to realize that the statement “HIV is the sole cause of AIDS” is just a hypothesis. There are unanswered questions and controversy concerning the role of HIV “as the sole cause of AIDS.” And until they are resolved, a cure is not possible. This paper explores the possible role of acid-fast tuberculous mycobacteria as “primary agents” in AIDS.

© 2008 Elsevier Ltd. All rights reserved.

Introduction

In a startling new report, a team of Slovakian researchers headed by Vladimir Zajac has found

* Corresponding author. Tel./fax: +718 746 5793.
E-mail address: medamerica1@verizon.net (L. Broxmeyer).

genetic sequences of HIV (the human immunodeficiency virus) in various bowel bacteria cultured from AIDS patients [1]. It is well-known that HIV attacks blood cells of the immune system, but this is the first study indicating HIV can also infect bacteria naturally contained within the body.

Gastrointestinal disturbances are universal in AIDS. Early in the epidemic, microbiologists Beca Damsker and Edward Bottone found frequent infection with *Mycobacterium avium* in the colon and rectal tissues in gay men with [2] AIDS. It is not known if these mycobacterium contained gag-pol-env genes.

HIV as the “sole” cause of AIDS

Ever since HIV was isolated in 1984 by Robert Gallo, the scientific mantra has been that “HIV is the sole cause of AIDS.” However, since 1982 there have been reports by Cantwell et al. that “acid-fast bacteria”, closely related to tuberculosis (TB) and to non-tuberculosis (atypical tuberculosis) acid-fast mycobacteria, might play a primary role in the pathogenesis of AIDS [3–8]. Cantwell’s reports of acid-fast bacteria in AIDS have been largely ignored by the scientific community. However, a small but highly vocal group of scientists, called “the Perth Group”, denies that HIV is the sole cause of AIDS. Other “dissident” scientists, headed by molecular biologist and virologist Peter Duesberg[9,10], claim that HIV is not the cause of AIDS and that the so-called AIDS retrovirus

has never been isolated in a “pure state.” As one of the first to actually define retroviral structure, Duesberg’s opinion is not to be taken lightly.

Tuberculosis and AIDS

When viewed microscopically, mycobacteria are stained red by carbol-fuchsin, the color of the initial stain used in the acid-fast staining procedure. All other bacteria are decolorized by an acid-alcohol rinse, the next step in the staining procedure. Typical TB-causing bacteria are therefore referred to as “acid-fast”.

Tuberculosis (TB), caused by *Mycobacterium tuberculosis*, remains the most frequent cause of death worldwide from any single infectious disease, and the numbers are increasing yearly. According to the National Institute of Allergy and Infectious Diseases. TB is the major attributable cause of death in HIV/AIDS patients. One out of every three people with HIV/AIDS worldwide dies of TB [11]. People who are HIV-positive and infected with TB are 30 times more likely to develop active TB than people who are HIV negative. TB acid-fast bacteria enhance HIV replication and accelerate the natural progression of HIV infection. In developed countries such as the U.S., the most common acid-fast species found in AIDS is an atypical tuberculous bacterium called *Mycobacterium avium*, or fowl tuberculosis. These infections are often resistant to treatment with anti-TB medications.

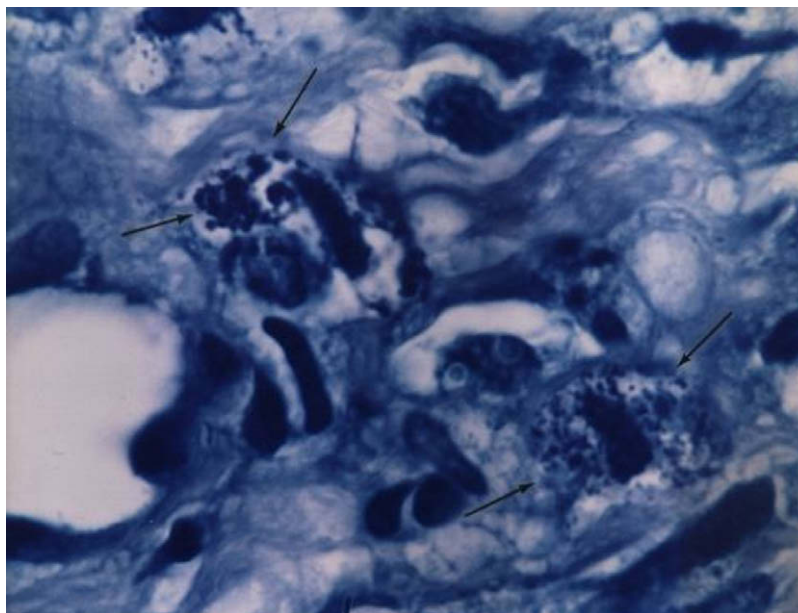


Figure 1 Microscopic tissue section of AIDS-related Kaposi’s sarcoma of the skin. Arrows point to variably-sized acid-fast coccoid (round) and granular forms in the tumor. Acid-fast (Fite) stain, magnification $\times 1000$, in oil.

A half century ago only a few species of mycobacteria were known to cause infection in humans. Leprosy is a well-known acid-fast mycobacterial disease caused by *M. leprae*.

Using modern molecular biology techniques such as PCR, there are now more than 130 recognized species of tuberculosis-like mycobacteria. Myco-

bacteria are found everywhere in nature, in soil, food and water.

Cell-wall deficient acid-fast mycobacteria

In laboratory culture and in tissue stained with an acid-fast stain, the red-stained and rod-shaped

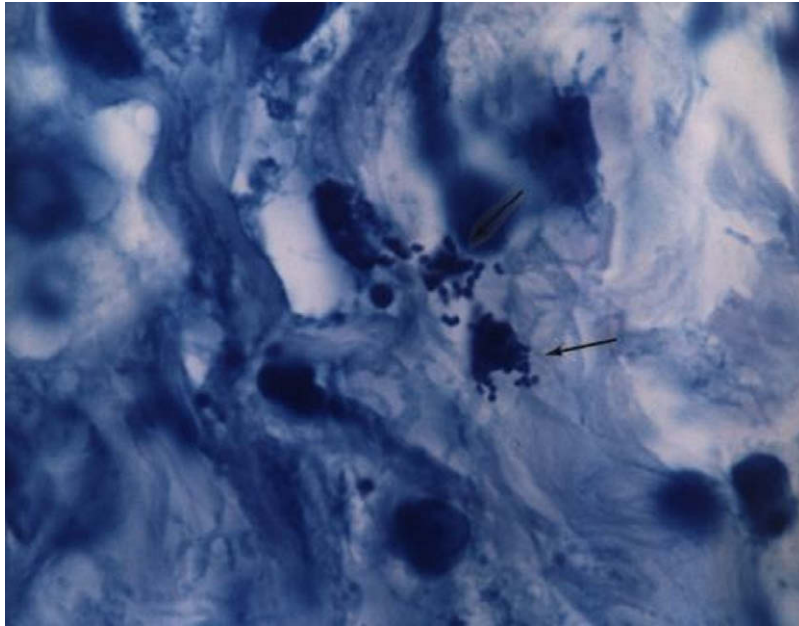


Figure 2 Microscopic tissue section of lung showing "interstitial pneumonitis" occurring in a fatal case of AIDS. Arrows point to round coccoid forms. Compare the size and form of these tiny round bodies to those seen in *Mycobacterium avium* (Figure 3), which was cultured from bronchoscopic washings in this case. Acid-fast stain, $\times 1000$, in oil.

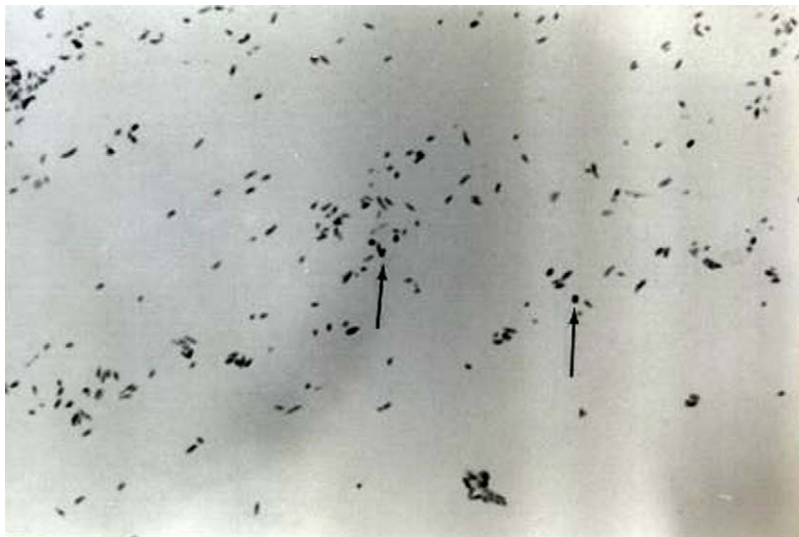


Figure 3 Laboratory culture of *Mycobacterium avium* cultured from a case of AIDS-related "interstitial pneumonitis". The culture is pleomorphic, in that it contains acid-fast rod forms and non-acid-fast round coccoid forms, as indicated by arrows. Compare the size of these cocci with the tiny round forms observed in the lung in Figure 2. Acid-fast stain, $\times 1000$, in oil.

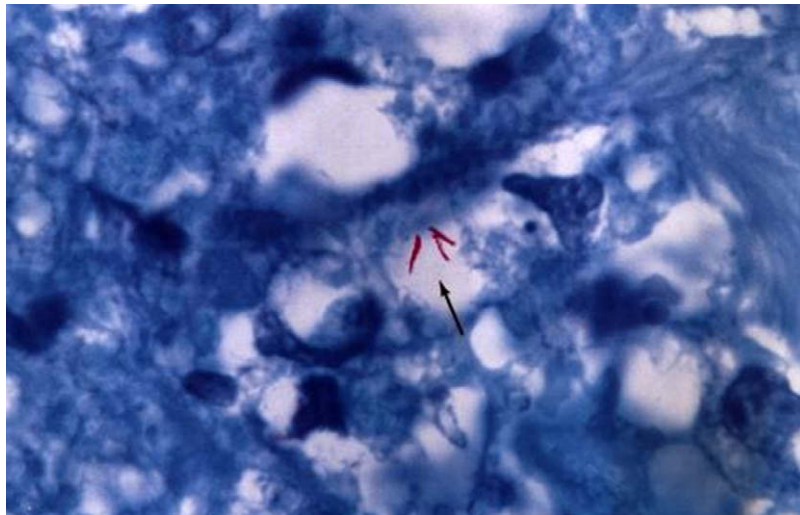


Figure 4 Microscopic tissue section of AIDS-related “immunoblastic sarcoma” tumor of the face showing 3 typical red-staining acid-fast rods. These rod forms were exceedingly rare. However, weakly acid-fast coccoid forms were more numerous in the tumor. *Mycobacterium avium* was cultured from this tumor. Acid-fast stain, $\times 1000$, in oil.

bacillus is the classic form of mycobacteria, easily identified by pathologists and microbiologists. (See Fig. 4).

However, in AIDS, the acid-fast bacteria reported by Cantwell et al in AIDS-damaged tissue sections are primarily in the cell-wall-deficient (CWD) form, and since the integrity of their cell wall is disrupted, they are easily mistaken for mycoplasma or viruses. Such forms are “pleomorphic” (i.e., they have many forms) and frequently appear as round coccoid forms which are not stained a bright red, but rather a magenta color. Because these cell-wall-deficient forms of tuberculosis are generally not recognized, and not looked for by pathologists, they generally go undetected in diseased tissue.

Beginning in 1982, these pleomorphic, acid-fast bacteria were reported by Cantwell in the enlarged lymph nodes of early AIDS, in the lesions of AIDS-related Kaposi’s sarcoma and throughout the various organs and connective tissue of AIDS victims at autopsy. (Figs. 1–4).

Bacteria as a necessary co-factor in AIDS

According to one website, 2610 scientists and educators have expressed doubts that “HIV” causes AIDS (<http://www.rethinkingaids.com/quotes/rethinkers.htm>). Nevertheless, the mantra that “HIV is the sole cause of AIDS” is so well-known and accepted universally that any suggestion to the contrary is usually met with disdain by the AIDS establishment. One notable example of this disdain was provided by TIME magazine’s Man of the Year

in 1996, AIDS researcher David Ho MD [12], who famously declared: “It’s the virus, stupid!”

Despite this, in 1993 Luc Montagnier, the original discoverer of HIV at the Pasteur Institute in Paris, reported on bacteria (via culture and biochemical techniques) in the form of mycoplasma (CWD bacteria) as a suspected necessary co-factor in AIDS [13]. And at the same time, remarkably, he remained silent on Cantwell’s reports of acid-fast bacteria which could simulate “mycoplasma” in AIDS tissue. At present, the precise connection between Montagnier’s mycoplasma and Cantwell’s cell-wall-deficient (CWD) acid-fast bacteria has not been established. In her classic text *Cell Wall Deficient Forms-Stealth Pathogens*, microbiologist Lida Mattman [14] makes clear that the differentiation between mycoplasma and CWD bacteria is difficult at best and cites a study by Pachas [15] et al which confirmed that one mycoplasma was actually mistaken for a CWD form of a bacterium closely related to the mycobacteria.

Virus-like forms of mycobacteria in AIDS

Lost in the history of microbiology is the concept of a “tuberculosis virus.” A century ago, in 1908, Hans Much [16] first described the tiniest virus-like granules of TB bacteria, which eventually became known as “Much’s granules,” the precise nature of which remains controversial to this day. Two years later, A. Fontes proved the granules were filterable, meaning they were able to pass through laboratory filters designed to hold back bacteria. [17] As they were too small to be seen microscopically, they became known as the TB ‘virus’.

When injected into guinea pigs the granules and other forms of the filterable TB bacteria reverted and transformed back into their classic acid-fast, rod-shaped bacillus form and produced immune system disease, and even tuberculosis in the animals.

Mellon and Fisher [18], appearing in *The Journal of Infectious Diseases* had actually warned that such filterable forms of *M. Avium* and *M. Tuberculosis* could easily be mistaken for both viruses and mycoplasmic-like forms, citing 'the common finding by French workers of acid-fast bacilli in the glands of guinea pigs into which viral-like (cell free) filtrates of tuberculosis material had been injected'. It is these tiny granular and coccal forms (not the typical bright-red-stained rod-shaped bacillus characteristic of the TB germ) that can be observed in acid-fast-stained AIDS tissue.

Physician Virginia Wuerthele-Caspe Livingston [19] was the first to discover that the virus-like tuberculous CWD forms of pleomorphic bacteria that she found in cancer and other immunologic diseases were "acid-fast" at some stage of their growth, and that this staining reaction was the key to identifying these "cancer microbes" in cancer and in tissue. To the extent that she also understood that these organisms could be sexually transmitted, she suspected the same type of organisms were implicated in AIDS.

The specificity of the HIV test and the "purity" of HIV

AIDS dissidents [20] question whether HIV is truly a virus, as well as the significance, accuracy and specificity of the HIV blood test. It should be noted that the manufacturers of the various HIV blood tests never claimed 100% specificity. A positive HIV test should be viewed as a starting point for physicians to follow-up with further blood tests to determine the immune system's status and the number of T cells in a patient suspected of having HIV. However, tuberculosis can also drastically decrease the number of the body's T cells [21]. A "positive" HIV test can be encountered in someone who is not infected with HIV. This can be due to "cross-reactivity". According to Kashala [22], false-positive HIV tests can occur in up to 70% of patients infected with acid-fast mycobacteria such as TB or leprosy.

AIDS dissidents [23] believe HIV has never been isolated in "pure" culture. HIV was originally isolated in Gallo's virology laboratory by pooling the blood of ten AIDS patients and seeding the mix into a cell-line comprised of leukemic (cancerous)

blood cells. Although this might make some bacteriologists cringe, this pooling of patients' blood enabled Gallo to grow enough HIV to devise a blood test for the virus. The HIV test has been highly successful in screening out HIV-infected blood donors.

But could "pure" cultures of HIV contain cellular elements and/or CWD/bacteria such as those found in tuberculosis? Etienne de Harven, M.D., a renowned electron microscopist and authority on the microscopical appearance of retroviruses [24], thinks so. He insists that so-called HIV purified cultures are contaminated with cellular debris, and that he has never been able to visualize any retroviral particle in the blood of an AIDS patient, even those with a very high "viral load."

But what about pictures of HIV widely promoted in the media? According to de Harven, "These pictures are extremely attractive, and are frequently rich in artificial colors. They clearly exemplify the danger of misinforming the public with computer graphics. To publish such images brings to the attention of the general public, and of the medical profession as well, an apparently crystal-clear message: 'Yes, HIV has been isolated since one can portray it under the electron microscope.' All these images represent computerized rationalizations and embellishments of actual electron microscopic pictures. ...but not one of these pictures originated directly from one single AIDS patient! They all originated from complex cell cultures prepared in various laboratories, cultures that have been described as 'real retroviral soups'." De Harven, the first researcher to ever visualize a retrovirus under an electron microscope, concludes: HIV has never been properly isolated, nor purified, and consequently, the HIV/AIDS hypothesis has to be fundamentally reappraised.

Microbiologist and electron microscopist Phyllis Evelyn Pease [25,26], author of *AIDS, Cancer and Arthritis: A New Perspective* (2005), agrees with de Harven. She takes issue with the new techniques of molecular biologists and virologists, by stating, "Reliability has been placed on physical/chemical techniques, which are greatly favored by biochemists but which are not suitable for microbiological materials unaccompanied by microscopic methods." Biochemists, Pease says, usually have a poor understanding of microorganisms as living creatures. They tend to regard bacteria as laboratory tools — as bags of enzymes, or as culture media, for example — and to believe that biochemical techniques are all that are necessary for identifying and isolating viruses. The result of this simplistic approach is that it has been accompanied by the virtual abandonment of that *sine qua non* for a properly trained microbiologist, the microscope,

and in the case of filterable forms of bacteria and viruses, this means the electron microscope. Without these aids and the controls that they offer, it has become apparent that what have passed as preparations of pure virions have in fact been contaminated not only with filterable forms of bacteria, but also with cellular materials derived from the tissue cultures in which the viruses have been cultured.

In a review essay of Pease's book, former London Times science editor Neville Hodgkinson concludes "Pease's book is the most authoritative and microscopically precise account to date of the failings of the HIV theory of AIDS." (http://www.immunity.org.uk/images/Neville_H_Review.pdf).

Could unrecognized elements of tuberculous acid-fast bacteria be present in the leukemic cell lines used to grow HIV? Largely unknown is the fact that acid-fast bacteria were identified and reported in leukemic, as well as in other forms of cancer, many decades ago by Seibert et al.

As a biochemist, Florence Siebert Ph.D. [27,28], (1897–1991) contributed greatly to the development of the modern TB skin test, still in wide use. An expert on contaminants, she discovered 'pyrogens' in water for injection, the removal of which set the standards for injectable water. Having done such, her opponents were put on notice that Florence Siebert knew exactly what a contaminant was. Seibert devoted the last years of her life to cancer microbe research and believed fully in bacteria as causative agents in cancer and leukemia. Along with dozens of other cancer microbe workers (including Pease), Seibert et al. reported on bacteria in leukemia at a New York Academy of Sciences symposium in 1969. Publishing her cancer work in 1970, she was inducted into the National Women's Hall of Fame in 1990, at the age of 92. Her discovery of virus-sized bacteria in leukemia suggests that it is unwise to grow a virus, such as HIV, in leukemic cells.

In her privately-published autobiography *Pebbles on the Hill of a Scientist* [29], she wrote: "One of the most interesting properties of these bacteria is their great pleomorphism...and even more interesting than this is the fact that these bacteria have a filterable form in their life cycle: that is, they can become so small that they pass through bacterial filters which hold back bacteria. This (ability to pass through filters) is what viruses do, and is one of the criteria of a virus, separating them from bacteria. But the viruses also will not grow live on artificial media like these bacteria do. They need body tissue to grow on. Our filterable form, however, can be recovered again on

ordinary artificial media and will grow on these. *This should interest the virus workers very much and should cause them to ask themselves how many of their viruses may not be filterable forms of our bacteria.*" (Italics ours).

AIDS bacteria and viral bacteriophages

Further complicating the precise role of bacteria in AIDS is the fact that bacteria can be infected with viruses called "bacteriophages" or phage for short. There is no evidence to suggest that HIV is a phage. However, the reality that viruses commonly infect bacteria gives further credence to Zajac's discovery of HIV genes in bacteria. Are tuberculous mycobacteria in AIDS patients infected with HIV, or do they generate the virus? No one seems to know.

Could "phage therapy" be useful in treating AIDS? Phages are ubiquitous in the environment and have antibacterial properties. They were discovered to be antibacterial agents and put to use as such soon afterwards, with varying success. However, with the discovery of broad spectrum, easily manufactured antibiotics, phage therapy was largely abandoned in the West, but continued, unabated, in the former Soviet Union throughout the 1940's. Phage therapy is now seen as a hope against multi-drug-resistant (MDR) strains of many bacteria. Broxmeyer wrote about a proposed phage therapy [30] and was the lead investigator in a *Journal of Infectious Diseases* study to prove its efficacy.[31]

Is HIV a virus-like form of tb-type mycobacteria?

A quarter-century ago, Cantwell et al first reported on acid-fast bacteria in AIDS. More recently, in a 2007 paper, Cantwell explored research leading him to ask, whether TB-type bacteria caused AIDS?

Previous to this, in 2003, Broxmeyer made a similar claim by suggesting that AIDS was not caused by "a virus", but was caused by infection with CWD forms of tubercular mycobacteria, in both a book [32] as well as his peer-reviewed *Is AIDS Really Caused by a Virus?*, [33] Broxmeyer reviews the literature and presents his argument for AIDS as being caused by a mix of atypical and typical tuberculous mycobacteria. His conclusion was supported by among other things, Cantwell's repeated microscopic findings of acid-fast bacteria in AIDS-damaged tissue.

How are cell-wall-deficient forms of mycobacteria created? Antibiotics can be used in bacterial

cultures in laboratories to transform ordinary bacteria into CWD forms. But supported by the previous work of Nelson and Pickett [34], Broxmeyer thinks it is mycobacteriophages inside the body that attack the cell wall of bacteria, thereby transforming them into CWD forms. Nelson's study showed that it was only after phage activation that antibiotics and other agents caused CWD forms. In the process, genetic information can be transferred from attacking phage to bacteria.

How does Zajac's research fit into this? Broxmeyer suspects the gag-pol-env genes of HIV are mycobacterial in origin. He bases this, in part, on genomic phage studies reported by Lawrence [35] and others in *The Journal of Bacteriology* entitled *Imbroglios of Viral Taxonomy: Genetic Exchange and Failings of Phenetic Approaches*. Based upon this, Broxmeyer feels that molecular sequencing of CWD mycobacteria and their phages should be ascertained to determine if there are comparable genetic elements that could "cross-react" with those genes (gag-pol-env), presently attributed solely to HIV. However, to this date, no such study has been undertaken.

In an attempt to make an AIDS vaccine, genetic engineers have inserted HIV genes into *Mycobacterium bovis*. In recombining HIV with a mycobacterium, Broxmeyer says HIV is acting more or less like a bacterial or mycobacterial phage. So far no recombinant AIDS vaccine has proved effective. Efforts to vaccinate infants against TB at birth by use of the time-honored BCG vaccine (which contains an attenuated strain of *M. bovis*) have recently caused illness and even death in some HIV-positive infants. The fact that a "live" anti-TB vaccine made with a "harmless" strain of mycobacteria (*M. bovis*) can cause death is proof of how susceptible AIDS patients are to mycobacterial infections, even those thought to be totally benign.

A proposed virus/bacteria hypothesis for AIDS

It is important to realize that the statement "HIV is the sole cause of AIDS" is a hypothesis, and that this hypothesis was hastily thrown together in the face of an epidemic emergency. However, most scientists still accept the HIV-AIDS hypothesis as fact.

The ability of bacteria to pass through filters – and the ability of viruses to infect bacteria – suggest a close (if not inseparable) relationship between viruses and bacteria. Tuberculosis mycobacterial infections are the ultimate cause of death in many

AIDS patients. In African AIDS cases there is an ever-increasing associated epidemic of XDR (extensively drug resistant) TB. There is suspicion that HIV and *M. tuberculosis* may have exchanged genetic material to account for these resistant strains. However, drug resistant strains of TB are routinely found in patients who do not have AIDS or HIV.

Bacterial cells and human cells routinely exchange genetic material via "horizontal" transmission using their bacteriophage viruses. Nevertheless, scientists still seem content to regard viruses and bacteria as separate and distinct from one other. Yet there is still much to be learned regarding the pathogenesis of AIDS and health professionals should keep an open mind on the matter of retrovirus versus cell-wall-deficient mycobacteria.

And we forecast the day when an important AIDS researcher will take the stage, with similar assurance and fervor to declare: "It's the bacteria, stupid!"

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at [doi:10.1016/j.mehy.2008.06.012](https://doi.org/10.1016/j.mehy.2008.06.012).

References

- [1] Zajac V, Stevurkova V, Matelova L, Ujhazy E. Detection of HIV-1 sequences in intestinal bacteria of HIV/AIDS patients. *Neuro Endocrinol Lett.* 2007;28(5):591–5.
- [2] Damsker B, Bottone EJ. *Mycobacterium avium*-Mycobacterium intracellulare from the intestinal tracts of patients with the acquired immunodeficiency syndrome: concepts regarding acquisition and pathogenesis. *J Infect Dis* 1985;151(1):179–81.
- [3] Cantwell Jr AR. Variably acid-fast bacteria in vivo in a case of reactive lymph node hyperplasia occurring in a young male homosexual. *Growth* 1982;46(4):331–6.
- [4] Cantwell AR. Variably acid-fast cell wall-deficient bacteria as a possible cause of dermatologic disease. In: Domingue GJ, editor. *Cell-Wall-Deficient Bacteria*. Reading: Addison-Wesley Publishing Co; 1982. p. 321–60.
- [5] Cantwell Jr AR. Necroscopic findings of variably acid-fast bacteria in a fatal case of acquired immunodeficiency syndrome and Kaposi's sarcoma. *Growth* 1993;47(2):129–34.
- [6] Cantwell Jr AR. Kaposi's sarcoma and variably acid-fast bacteria in vivo in two homosexual men. *Cutis* 1983;32(1):58–61. 63-4,6.
- [7] Cantwell Jr AR. *Mycobacterium avium-intracellulare* infection and immunoblastic sarcoma in a fatal case of AIDS. *Growth* 1986 Spring; 50(1): 32–40.
- [8] Cantwell AR, Rowe L. African "eosinophilic bodies" in vivo in two American men with Kaposi's sarcoma and AIDS. *J Dermatol Surg Oncol.* 1985;11(4):408–12.

- [9] Duesberg PH. HIV is not the cause of AIDS. *Science* 1988;241:514–51.
- [10] Duesberg PH. AIDS epidemiology: inconsistencies with human immunodeficiency virus and with infectious disease. *Proc. Natl Acad Sci* 1991;88:1575–9.
- [11] Fauci AS. NIAID: A commitment to global health U.S. *Medicine* 1999;3:34–61.
- [12] Ho DD, Neumann AU, Perelson AS, Chen W, Leonard JM, Markowitz M. Rapid turnover of plasma virions and CD4 lymphocytes in HIV-1 infection. *Nature* 1995;373:23–126.
- [13] Montagnier L, Blanchard A. Mycoplasmas as cofactors in infection due to the human immunodeficiency virus. *Clin Infect Dis*. 1993;17(Suppl 1):S309–15.
- [14] Mattman L. Cell wall-deficient forms – stealth pathogens. Boca Raton: CRC Press; 1993. p. 416.
- [15] Pachas WN, Schor M, Aulakh GS. Evidence for the bacterial origin of *Acholeplasma laidlawii* A. *Diagn Microbiol Infect Dis*. 1985;3(4):295–309.
- [16] Much H, granuläre Über die, Tuberkulosevirus nach Ziehl nicht färbbare Form des. *Beiträge zum Klinik der Tuberkulose* 1907;8:86–99. 17: 1908, 8: 85..
- [17] Fontes A. ;Bemerkungen ueber die Tuberculoese Infection and ihr virus. ; *Mem Instit Oswaldo Crus* 1910;2:141–6.
- [18] Mellon. Fisher New studies on the filterability of pure cultures of the tubercle group of microorganisms. *J Infect Dis* 1932;51:117–28.
- [19] Livingston V. *Cancer: A New Breakthrough*. Los Angeles: Nash Publishing; 1972.
- [20] The Perth Group. Papadopulos-Eleopulos E Turner VE A critical analysis of the HIV-T4-Cell AIDS hypothesis. *Genetics* 1995;95:5–24.
- [21] Hirsch CS, Toossi Z. Apoptosis and T Cell hyperresponsiveness in pulmonary tuberculosis. *J Infect Dis* 1999;179: 945–53.
- [22] Kashala O, Marlink R, Ilunga M, Diese M, Gormus B, Xu K, Mukeba P, Kasongo K, Essex M. Infection with human immunodeficiency virus type 1 (HIV-1) and human T cell lymphotropic viruses among leprosy patients and contacts: correlation between HIV-1 cross-reactivity and antibodies to lipoarabinomannan. *J Infect Dis*. 1994;169(2):296–304.
- [23] Duesberg, P., Review of Gallo R Virus Hunting: AIDS, Cancer, and the Human Retrovirus: A Story of Scientific Discovery, 1991. Reviewed in *The New York Native* 29 April 1991.
- [24] De Harven E. Remarks on methods for retroviral isolation. *Continuum Magazine* 1998;5(3). 1998b.
- [25] Pease PE. AIDS, Cancer and arthritis: a new perspective. Birmingham UK: Pease Associates; 2005. p. 134.
- [26] Pease P. Bacterial origin of certain viruses: identity of the eaton agent with streptococcus MG. *Nature* 1993;197:1132 (16th March, 1963).
- [27] Seibert FB, Feldmann FM, Davis RL, Richmond IS. Morphological, biological and immunological studies on isolates from tumors and leukemic bloods. *Ann N Y Acad Sci*. 1970;174(2):690–728.
- [28] Seibert FB, Farrelly FK, Shepherd CC. DMSO and other combatants against bacteria isolated from leukemia and cancer patients. *Ann N Y Acad Sci*. 1967;141(1):175–201.
- [29] Seibert FB. Pebbles on the hill of a scientist, In: Florence B. Seibert, author/publisher, St. Petersburg, FL 1968.
- [30] Broxmeyer L. Bacteriophages: antibacterials with a future? *Med Hypotheses* 2004;62(6):889–93.
- [31] Broxmeyer L, Sosnowska D, Miltner E, Chacón O, Wagner D, McGarvey J, Barletta RG, Bermudez LE. Killing of *Mycobacterium avium* and *Mycobacterium tuberculosis* by a mycobacteriophage delivered by a nonvirulent mycobacterium: a model for phage therapy of intracellular bacterial pathogens. *J Infect Dis*. 2002;186(8):1155–60.
- [32] Broxmeyer L. AIDS: What the discoverers of HIV never admitted. Chula Vista: New Century Press; 2003.
- [33] Broxmeyer L. Is AIDS really caused by a virus? *Medical Hypothesis* 2003;60(5):671–88.
- [34] Nelson E, Pickett MJ. The recovery of l-Forms of brucella and their relation to brucella phage. *J Infec Dis*. 1951;89:226–32.
- [35] Lawrence JG, Hatfull GF, Hendrix RW. Imbroglis of viral taxonomy: genetic exchange and failings of phenetic approaches. *Journal of Bacteriology* 2002;184(17): 4891–905.

Available online at www.sciencedirect.com

