

Is AIDS really caused by a virus?

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PROLOGUE

University of Liverpool, Liverpool, England, 1988

The results were disquieting. Researchers at the University of Liverpool, England, led by A.M. al-Sumidaie in 1988, had found a retrovirus in 97% of the women they tested for breast cancer (1).

Did this finding mean that breast cancer was also caused by a retrovirus? Certainly not. Al-Sumidaie knew very well as a viral researcher that he could take cells from a patient's body and coax out a lot of harmless retroviruses to which a patient had been exposed. When in the retroviral business, you detected retroviruses and over the years it had been done in multiple sclerosis, sarcomas and leukemias. Yet little more evidence than Al-Sumidaie now had in front of him was available when the HIV retrovirus was called the probable cause of AIDS.

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EPIDEMIC

Manhattan, 1979

By 1979, doctors in Manhattan began to notice a strange new disease killing what had been up to then healthy gay men. As reports mounted, the Centers for Disease Control (CDC) was forced to circulate similar notices of homosexual men in New York and Los Angeles with a weakened immune system dying from

heretofore rare causes (1). From its conception AIDS was a nightmare of anguished victims, washed with wave after wave of terrible disease, whose physicians, like so many medical priests, helplessly watched them die. U.S. Coastal hospitals in San Francisco, New York and Los Angeles soon turned into war zones.

Rare diseases like *Pneumocystis carinii*, a tiny one-celled protozoa, filled gay lungs to the point of suffocation and requests for pentamidine aerosols trickled and then poured into the CDC.

Another uncommon killer, Kaposi's Sarcoma (KS), became the most common form of AIDS cancer.

Nor was it only gays at risk. Drug addicts sharing needles and hemophiliacs, given pooled clotting factor VIII from blood so they would not bleed to death, soon became prey, again developing Kaposi's sarcoma and *pneumocystis* pneumonia. America's entire blood supply was in jeopardy, for by the early 1980s, gay and bisexual men accounted for 1 of 4 American blood donors.

AIDS throttled the immune system, in some cases shutting it down, and the primary site of attack always seemed traceable to the body's T-cells: white blood cell lymphocytes which held the body's invaders at bay.

By 1977 much evidence indicated that the basis of cellular immunity was tied in with T-cells lymphocytes-colorless, motile, cellular elements of lymph. Chief in importance among these was the T-helper or CD4 lymphocyte, which fought infection. It soon became apparent that in AIDS CD4 cells were either severely depleted or they fell off the blood map altogether.

Without delay, the same virologists who for decades had failed miserably to find a viral cause for cancer, pounced on AIDS, dismissing any possibility other than a virus: ignoring that it could just as easily be either one or a combination of older microbes presenting in an all-new way.

Virologists initially told physicians to pass on the word that Cytomegalovirus (1) caused AIDS. Doctors

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dutifully obeyed, not fully realizing that all people, with time, are infected with Cytomegalovirus.

Epidemiologist-retrovirologist Donald Francis, who would direct laboratory efforts for AIDS at the CDC, and was also assistant director of the CDC's Division of Viral Diseases, had his own peculiar theory which he shared with epidemiologist James Curran, eventual director of AIDS research at CDC: combine hepatitis with feline leukemia in cats—a retrovirus on which Francis wrote his doctorate and you had Kaposi's and the opportunistic infections seen in AIDS. Or maybe, just maybe, it was a retrovirus similar to the cat retrovirus alone that was solely responsible. Curran and Francis had worked together years ago developing the Hepatitis B vaccine.

Francis, one of the few at CDC who had actively wiped out smallpox worldwide, was considered an expert on both epidemics and the feline leukemic virus. He would now combine his fields of expertise and quickly conclude that AIDS was cat leukemia in people. It was an impulsive long shot and by most treated as such, at least initially.

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CANCER STRUGGLES

America, Turn of the century

The history of retroviruses mirrored cancer research. By the turn of the 20th century American medicine had come to the conclusion that it was not a matter of whether infectious disease caused cancer, but which one. Incredibly, just 20 years before, the germ theory of disease, ignored by organized medicine for 150 years, had finally been accepted. Robert Koch, discoverer of the cause of tuberculosis, had seen to that. But as the 'Father of Bacteriology' and perhaps the greatest physician that ever lived, when Koch presented his Berlin paper his immediate problem was in convincing the German medical establishment that TB was not a 'virus'.

Under this backdrop and in 1904, Ellermann and Bang, searching for an infectious bacterial cause for chicken leukemia (4), succeeded in its transfer from one fowl to another by injecting cell-free tissue infiltrates. They sought a bacteria, but simply because it passed through a filter, the responsible agent was assumed to be a virus.

That same year, the first lentivirus, related to HIV, was isolated as a filterable equine infectious agent by Valle and Carre at the Pasteur Institute (16). Yet Roux, an authority on 'invisible microbes' at the time, shrugged off Valle and Carre's finding as no more than 'small bacteria' (14).

Most authorities now realize that there are some viruses almost as large as bacteria and some bacteria as small as viruses, forms of which can easily pass through filters. This realization has been disputed by HIV enthusiasts Francis et al. (5) when they mentioned 'since the infectious agent had obviously passed through a filter, it had to be a virus'.

It did not.

Peyton Rous was credited with the discovery and isolation of the first retrovirus. By 1911, Rous wanted to know why if one chicken got cancer, others followed (13). Rous, who reproduced the tumor at will in Plymouth Rock fowls, favored a bacterial cause over a filterable virus. However, it was a question that he never definitely answered.

By 1933 Shope reported a viral tumor in cottontail rabbits. Bittner reported on a milk-born mouse breast cancer attributed to still another virus (1).

In the 1950s, and with the advent of the electron microscope, particles later questionably ascribed to retroviruses were readily being detected. As a result, and at a time when established medicine had about-faced and was now firmly set against an infectious cause for cancer, two controversial minority camps splintered from mainstream, each diametrically opposed.

There were the virologists, who claimed that cancer was viral, and another group which did careful, peer-reviewed research at the height of U.S. Post-World War II technology, demonstrating that the retroviruses in Rous, Bittner and Shope tumors were actually filterable forms of mycobacterial-like bacteria of the *Actinomycetales* (10).

Tuberculosis-like, these 'viruses' stained with acid-fast dyes; readily passed through a filter, but actually were a class of bacteria having many of the characteristics of the mycobacteria such as tuberculosis.

This work, spearheaded by physician-researcher Virginia Livingston of Rutgers (9), validated earlier work on Rous as a bacteria (3,6). Soon others would join (2,7,15).

Livingston's network questioned the very existence of retroviruses and the retrovirologists did not like it. A scientific life-and-death cancer struggle ensued.

By 1960, biologist turned retrovirologist Howard Temin sought an explanation for his observation as to why retroviruses, composed of RNA, Rous among them, were inhibited by Actinomycin D – an antibiotic and known bacterial DNA inhibitor. Based on this finding, Temin elaborately hypothesized the concept of reverse transcription. But since antibiotics did not affect viruses, Temin's observation regarding Actinomycin D's inhibition of Rous made more sense if Rous was bacterial.

Nevertheless, in reaction to Temin, cancer viral investigators of the 1960s and 1970s reacted by

misinterpreting his non-specific enzyme discovery (reverse transcriptase), which arose primarily as a function of normal cellular healing, for a primary indicator for the newly scrutinized retroviruses – which it was not. And in the 1970s, it was as a direct result of Temin's enzyme that 'oncoviruses', purported to cause cancer, suddenly became known as 'retroviruses'.

It was to Livingston's solid disadvantage that when Richard Nixon signed his National Cancer Act on December 23, 1971, he unwittingly placed virologist Frank Rauscher as director of the just established National Cancer Program (NCP). With Rauscher at the controls it was only a matter of time before cancer virologists, retrovirologists and immunologists were pushed to the vanguard of 'America's War on Cancer'. Once entrenched, they would remain at the helm even as, incredibly, their failed cancer attempts now turned towards finding a viral cause for AIDS.

Bacterial L-forms, the connecting link between viruses and bacteria, were first described by Klieneberger at England's Lister Institute for which they were named. L-forms were 'cell-wall-deficient' because they either had a disruption or lack of a rigid bacterial cell wall. This lack of rigidity allowed them the plasticity to assume many forms (pleomorphic), some of them viral-like but all different from their classical parent and poorly demonstrated by ordinary staining (8). Of all the bacteria, L-forms predominate and are crucial to the survival of the mycobacteria whose cell-wall-deficient (CWD) forms escape destruction by the body's immune system. And at the same time CWD forms of the mycobacteria react in Elisa blood tests (11), similar to the 'HIV virus', and can simulate it in every way.

Some years later, when HIV discoverer Luc Montagnier was interviewed for a French AIDS documentary, film-maker Djamel Tabi asked how he had isolated HIV. Incredibly, Montagnier's reply was that he did not isolate HIV, he just found something that looked like a retrovirus (12).

Klieneberger, as well as Livingston, also saw parallels between the filterable forms of tuberculosis and 'mycoplasmic-like forms' because without intact cell walls they were often mistaken for the virus-like bacteria mycoplasma, which has no cell wall (8). The differentiation between mycoplasma and cell-wall-deficient bacteria is difficult at best (11).

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SHYH-CHING LO

Armed Forces Institute of Pathology, Washington, 1989

Dr. Shyh-Ching Lo was a senior scientist at the prestigious, world-renowned Armed Forces Institute of Pathology in Washington. As he watched events unfold and it became obvious that it was going to be dictum that HIV caused AIDS, he just had one problem: whenever he examined someone who had died of AIDS, he could never find HIV, not even a trace of HIV-infected tissue damage. So Lo began his own search for an AIDS cause which led him to a 'virus-like infectious agent' (8). Knowing he was onto something, Shyh-Ching Lo followed his conscience, against the grain of most other scientists, and finally isolated a mycoplasma. And in one study of 24 people with AIDS, he found antibody titers to it in practically everyone (9). Shyh-Ching Lo co-published again with Saillard (13).

That same year Livingston died and a year later Luc Montagnier, the discoverer of HIV, almost got booed off a 1991 San Francisco podium at the Sixth International AIDS conference for endorsing Lo's mycoplasma as a necessary co-factor for the AIDS virus to become fatal

(10). This 'co-factor' theory was, in effect, Montagnier's way of admitting that HIV, the virus he had discovered, wasn't virulent enough in itself to even approach what happened in AIDS.

Montagnier and Lemaitre had done a hornet's nest of an experiment which put HIV advocates on the edge of their chairs. In 1990 they published that cells cultured with 'HIV', which normally died, grew well in the presence of two antibiotics, minocycline and doxycycline (5). Antibiotics do not affect viruses, so it was not working against HIV – it was a bacteria. Montagnier decided that that bacteria was probably Lo's mycoplasma. He had done so, unaware of the fact that the two particular antibiotics he was using also had activity against Livingston's mycobacteria (3,12,14,15).

Rutgers-Presbyterian Hospital Laboratory for the Study of Proliferative Diseases, Bureau of Biological Research, Rutgers University, New Jersey, 1950

Livingston associate and prominent Cornell microbiologist Eleanor Alexander-Jackson (1), a lifelong colleague, had a problem.

As long as she held her reputation as one of the leading tuberculosis experts in the world, American medicine would embrace her, but when she tried to attribute cancer to Livingston's tuberculosis-like germ, it would move to crush her.

Alexander-Jackson, whose advanced mycobacterial staining and culture techniques appeared in a 1944 issue of *Science*, carefully set a trap insured to ensnare virologists. Rous, as a retrovirus, was supposed to be an RNA virus; Alexander-Jackson knew that finding DNA in it would automatically mean that it was bacterial. It was understood that Retroviral DNA should be present only in human or animal cells and nowhere else. Her paper on the 'Ultraviolet Spectrographic Microscope Studies of Rous Sarcoma Virus Cultured in Cell Free Medium' demonstrated that there was DNA present in the Rous tumor agent, characteristic of bacteria (2). Why was it still being called a virus?

When Livingston confronted Rous that his 'retrovirus' could be dried, shelved, stored and mixed months later in saline only to grow out on bacterial culture plates, he reminded her that he had never said it was a virus, carefully using 'tumor agent'.

To be certain, the Livingston network concluded that oncogenic, supposedly cancer-causing viruses, were in fact L-forms of the mycobacteria and related organisms. And they came much too close to proving their point to suit American retrovirologists.

HIV co-discoverer Robert Gallo, threatened, huffed: 'What is going on in this country? This is insanity! She can have her theories and what can I say? I don't know of anything to support it. I can't see any basis and I don't

know what to say or what analogy to give you' (11). But Livingston's findings, with worldwide stature, were not about the theory of retroviruses yet to be isolated. She had, in fact, come much closer than any of the retrovirologists in proving a direct causation between her organism and cancer by showing that it manufactured human growth hormone, long associated with malignancy (7).

Before her death, Livingston would give one more clue towards unraveling what had become AIDS. There were 'less known' and 'little publicized' microorganisms that were transmitted sexually. Through bacteriological studies she had confirmed that the very same L-forms of mycobacteria she found in Rous, by some called mycoplasma, could be found in the semen of man (6).

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IN THE SEMEN OF MAN

Although tuberculosis was rarely thought of as a sexually transmitted disease, the potential for this had always existed. In the presence of prostatitis, it may be transmitted through the semen (20).

Anyone who watched AIDS evolve in not only gay America but heterosexually in Africa and Asia could not help but be struck by its travel and spread along the epidemiologic highways of sex, drugs, migrants, prostitutes, bath houses and venereal disease clinics.

Yet the realization that sexual transmission of AIDS could occur between a man with risk factors and a woman came late (3). Soon thereafter, female to male transmission, originally thought unlikely, was also found to occur (29). By 1984, the pivotal importance female prostitutes played in the propagation of AIDS in equatorial Africa had become evident (33).

But despite the magnifying forces of high tech tests such as Polymerase Chain Reactors (PCRs), protein broths which make multiple copies of hard to find pathogens, and which critics contest vastly exaggerates HIV by making numerous copies of fragments of nucleic acid which might or might not even be HIV, HIV can be detected only in a distinct minority of semen samples: one in 25 (34).

On the other hand, ignored and unnoticed, the very real possibility of the genital transmission of *M. tuberculosis*, a disease affecting almost 2 billion people, intimately linked with and considered a reliable sign of AIDS (4,5), and frequently found in the genitourinary tract (38). Tuberculosis and *Mycobacterium avium* (also called *Mycobacterium avium-intracellulare* or MAI) are not only the recognized leading causes of infectious disease in AIDS today, they are by far the most important infections in AIDS.

The Research Center for Genitourinary Tuberculosis, Kingsbridge Veterans Hospital, Bronx New York, 1954

By 1954, a pattern emerged at Dr. John Lattimer's Center for Genitourinary Tuberculosis. Men who developed tuberculosis epididymitis (inflammation of the testicles) were usually found to have an active focus of

tubercular infection in their prostate and cultures of their semen were frequently positive for tubercle bacilli (20).

Documenting sexual transmission, what puzzled John Lattimer most was why more husbands with prostatic TB were not infecting their wives. Two possibilities came to mind. First, the resistance of the thick stratified vaginal epithelium to tubercular infection; second, the scorched earth policy of prostatic tuberculosis, whereby it sought to destroy glandular elements of the prostate (Ibid), severely decreasing semen volume. Many of his male patients, in fact, complained that orgasm produced only slight moisture at the tip of their penis with over half of his experimental groups having a semen volume of less than 0.5cc: too scanty to infect the vaginal or vulvar epithelium, if it reached them at all. As almost a testament to this finding, of 40 men with tuberculous genital infections, only one produced a child (Ibid).

Nevertheless, seeing sexual spread in a disease with staggering numbers right in front of him, he gave notice to the scientific world (20). But few listened and adequate descriptions of tuberculosis as a sexually transmitted disease never really reached medical texts.

Niagara Peninsula Sanatorium, St. Catharines, Ont., Canada, December 1954

Dr. Edgar T. Peer was more than a bit skeptical as he reviewed Lattimer's study regarding the seminal transmission of tuberculosis.

He himself had recovered tubercle bacilli from a patient's semen in 1952, but dismissed the finding as lab error. He would later write that like most dismissals of the tubercle bacilli on similar grounds, it would come back to haunt him.

By 1954, Peer's cases of sexually transmitted tuberculosis were mounting and struck by similarities beyond coincidence, he saw an 'extremely probable source' of tuberculosis coming from the male genital tract.

Peer published, warning that if physicians did not wake up to the possibility of sexually transmitted genital tuberculosis, its diagnosis would continue to be unsuspected and underestimated (28), which one day could lead to potentially catastrophic consequences.

Nor were Peer and Lattimer alone. Netter mentioned that the spread of the tubercle bacilli through the female genital tract of the tubercle bacilli by coitus with a tuberculous male could not be denied (26). In fact, wherever culture of the seminal fluid showed *Mycobacterium tuberculosis*, there was a possibility of transmission of genital tuberculosis from male to female via the semen through sexual intercourse (6). While Lattimer (21) and Peer (28) showed that the development of tuberculous

ulcers in the vagina or vulva resulting in swollen lymph nodes in the groin was due to semen positive males harboring *M. tuberculosis*, Hellerstrom clocked the actual incubation period from the date of coitus during which the wife was exposed – to the development of the vaginal or vulval ulcer and enlargement of inguinal lymph nodes at approximately 3–4 weeks (14).

Heins offered a better idea of the potential potency of sexually transmitted mycobacteria such as tuberculosis, demonstrating that even the tame *Mycobacteria smegmatis* found in the smegma genital secretions of both men and women, when introduced into the vaginas of female mice, resulted in the immediate death of over half of an experimental group of 14 (13).

Lattimer's cases were compiled from European and American literature. The ulcer and enlarged nodes in the female, often misdiagnosed, closely resembled lymphogranuloma inguinale, syphilis or chancroids (Ibid) diseases that could coexist with tubercular sexually transmitted disease (20).

And, just as men could transmit mycobacterial disease to women, so too could women infect men (21). By 1870 Soloweitschnick had documented the first observation of a tuberculous ulceration of the penis (2).

Lewis cited 110 cases of tuberculosis of the penis written up before 1946. Twenty-nine additional cases were subsequently reported by Lal (19). In his series of primary cases, Lewis pointed to 14 of venereal origin – 12 penile ulcerations being definitely the result of coitus and 2 as a result of oral sex (21).

Penile ulcers attributed to primary HIV (15) were already well documented in TB literature (16,17,21,37). And 'giant cells' claimed to originate from HIV (22,30) were decades ago seen at the base of these ulcers (21). Long a hallmark of tuberculosis, multi-nucleated giant cells form and engulf the tubercle bacilli in an attempt to kill them (23).

Lewis mentions that of all the ways in which the penis could be infected with tubercle bacilli, direct contact was by far the most common. Although he documented transmission mostly through vaginal sex and occasionally oral sex, rectal transmission was not explored (21).

To explain cases claimed not to arise from direct vaginal inoculation, woman to man, Lewis borrowed from Verneuil's hypothesis, somehow overlooked by later writers. In 1883, Verneuil, in *Hypothesis On the Origin of Genital Tuberculosis In The Two Sexes*, proposed a mechanism whereby men with infected urine or semen first inoculated the vaginal vault of their partners, and then, through subsequent sex became themselves re-inoculated at the corona or frenulum of their own penises (35).

Years passed. Voices of warning persisted.

By 1972, five years before gays started dying in the U.S., Rolland wrote *Genital Tuberculosis, a Forgotten Disease?* (31) 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 probably a sexually transmitted disease, but also the necessity of the application of suitable contraceptives such as condoms to avoid it (12).

Gondzik's solution and date of publication are chilling; his findings significant. Even in syphilis at its most infectious stage, successful transmission in humans was possible only in 30% of contacts (32).

Two years later, investigators in South Africa, itself perched on the precipice of a devastating sexually transmitted AIDS epidemic, issued a report of 91 cases of tuberculosis of the penis (25,36). This was followed by documentation in which 'HIV' in young African females came only after first contracting genital TB (11).

Moreover, the fact that *Mycobacterium avium-intracellulare*-fowl or swine tuberculosis, considered an 'atypical' tuberculosis could also act like a sexually transmitted disease, set up an explosive scenario (8–10).

Avium had, in the short space of 30 years, gone from relative obscurity to the leading infectious disease in U.S. AIDS. And despite the fact that DePaeppe's group used only conventional Ziehl-Neelsen stain without culture of either testicular tissue or semen, they still found *M. avium* in these specimens in 32% of AIDS patients with systemic *M. avium*, the same *M. avium* that would eventually kill most U.S. AIDS patients that did not die from another AIDS-related disease (27).

Queens Hospital Center, Long Island Jewish-Hillside Medical Center, Jamaica, New York, 1984

Dr. Pascal De Caprariis saw before him a dying 30-year-old Haitian man with AIDS. A biopsy of the lymph node in the patient's groin showed *Mycobacterium avium-intracellulare* seemingly gone systemic, spreading to the liver. Despite using different combinations totaling seven different anti-mycobacterials, the patient died.

Then, just before death an ulcerative lesion of the corona of the penis formed. Tests for herpes were negative. Upon culture and only upon culture, *Mycobacterium avium* was isolated from the penile crater, and De Caprariis started speculating that with this and the right groin lymphatic swelling, sexual transmission of *Avium* seemed neither far-fetched nor improbable (9).

Department of Microbiology, Mount Sinai Hospital, New York, 1985

Avium, although ubiquitous, was also in animal reservoirs, swine among them (7). For American microbiologist Beca Damsker, who found overwhelming mycobacterial infections of the colon and rectal tissues in U.S. gay AIDS time and again, an anorectal portal of transmission had to be considered important. She had, with regularity, found *Avium* or swine tuberculosis in gay stool and biopsy specimens (8). There was also the specter of the predilection of some homosexuals for bestiality, sexual activities with animals, a potential vector of transmission to man, which when combined with the fragility of the rectal mucosa to local trauma or intercourse and antigenic challenge (24), could lead to calamitous unchecked multiplication of *M. avium-intracellulare*, possibly of animal origin, in the intestinal mucosa leading towards AIDS spread into the blood with resultant targeting of other systems (8).

Tuberculosis in swine is almost always caused by *M. avium* (18) and such avian tuberculosis leads to some of the highest financial losses in the swine and poultry farm industry (1).

Although *M. avium-intracellulare* was thought of as an 'opportunistic' infection which occurred only late in the immunosuppression of AIDS, Damsker encouraged a better analysis of the temporal relationship between *M. avium-intracellulare* infection and American AIDS, foreseeing that what doctors were documenting in AIDS could be nothing but a stepwise increment of *M. avium-intracellulare*, present from the beginning but ever increasing, both in the scope of its infection, immune suppression, and lowered CD4 count (8). In theorizing such, Beca Damsker presented a very possible cause for AIDS.

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TRAGIC COST OF PREMATURE CONSENSUS

Pasteur Institute, Paris, January, 1983

The Pasteur Institute squeezed head of cancer virology, Luc Montagnier, into the pressure cooker of finding an AIDS retrovirus when its production of Hepatitis B vaccine, accounting for a significant part of its income, and in part processed from pooled American homosexual blood, came under fire.

Montagnier, unsuspectingly looking for a virus closely related to Gallo's HTLV-1, instead came upon LAV (Lymphadenopathy Associated Virus), first identified by Francoise Barre-Sinoussi, a member of his team at Pasteur's Virology.

LAV was so named because the French homosexual fashion designer it was first isolated from had enlarged, inflamed neck nodes (lymphadenopathy), a common early AIDS feature. Thirty-three and promiscuous, he had also visited New York City in 1979 and had a 50-gay-partner-a-year history.

Betting the obvious that the agent responsible for AIDS could be more readily detected in these swollen lymph nodes, Barre, in January, 1983, packed a small piece of the homosexual's just biopsied lymph node *en Toto* in ice at Paris's Pitie-Salpetriere Hospital and delivered it to Montagnier at Pasteur. This patient did not yet have full-blown AIDS, but his history and symptoms were strongly suggestive (1). He would die 5 years later of the disease.

At Pasteur, Montagnier put the tissue into cell cultures of T-lymphocytes. Immediately questions arose as to the procedure. Looking only for a retrovirus, the team cultured whole tissue lymph node lysates. The 'virus' was never isolated in its pure form. It would be the beginning of a long, long line of research work based on indirect evidence.

Later, looking at Montagnier's tissue cultures microscopically there were many granules, some of which were felt to look like retroviruses; but they were inside cells and tissues – not whole viral particles and had different shapes and sizes. No two were alike. They seemed to show all forms of 'viral maturation', but were they viral?

As early as 1928, Eleanor Alexander-Jackson began discovering unusual, and to that point, unrecognized forms of the TB Bacillus. Jackson marveled at the many forms of tuberculosis, including the tiny granules which the German Much saw in 1908 and soon became known as Much's granules (4). In 1910 Fontes proved Much's granules, as a sub-classification of Kleinberger's L-forms, were filterable and therefore also often mistaken for viruses. In fact, in certain circles the variable acid-fast granules were called 'the TB virus' (2).

Even prior to Livingston (1970), Mellon and Fisher had warned that filterable forms of *M. Avium* and *M. Tuberculosis* could easily be mistaken for the virus Montagnier and Barre thought they had (3) and might explain '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 (3)'.

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THE RACE

Pasteur Institute, Paris, 1983

Gallo's leukemic retrovirus (HTLV), which Barre and Montagnier thought they had isolated, should have led to the wild proliferation of lymphocytes. But all that Barre found in subsequent trips to the lab was how well it was slaughtering them. What also puzzled her was that retroviruses typically didn't kill cells. How could she explain this?

By January 25th, 1983, Barre's reverse transcriptase radioactivity counter was showing increased numbers, which to her meant the 'retrovirus' LAV was multiplying.

But reverse transcriptase was nonspecific and was also found elevated in events leading to the death of CD4 lymphocytes by tuberculosis (16), as well as in *M. avium* infection of neck lymph nodes (2), probably the very event Barre was watching. Why had she not considered these as possible AIDS causes?

In actuality, previously having been viral cancer researchers, Barre and Montagnier were solely attuned, intellectually and technologically to detecting retroviruses. Barre had been trained in mouse retroviral techniques requiring the measurement of reverse transcriptase in Robert Bassin's National Cancer Institute (NCI) lab. Her procedures were not designed to explore for some unknown pathogen. In effect, Barre and Montagnier found a retrovirus because that was all that they were looking for.

Within weeks Montagnier called a staff meeting. The new 'retrovirus' wasn't Gallo's HTLV. He didn't assert that his group had proved that HAV caused AIDS, but it was possible. In the future he would send samples of the tissue culture to Gallo who headed AIDS research at NCI to stimulate further research.

Approximately 1 year later, after receiving French samples from Montagnier, retrovirologist Gallo isolated and announced HTLV-3 as the AIDS retrovirus, which proved to be the same as LAV: a productive Lentivirus infection with all forms of viral maturation.

A colleague had suggested that Montagnier characterize his virus as a Lentivirus (Lenti means slow) on a hunch. Lentiviruses were large viruses which, after entering cells, did not leap into activity at once, but later shot into action.

But so-called slow viruses had been implicated, but never proven, in diseases such as Creutzfeldt-Jacob and Alzheimer's. Prominent American retrovirologist Peter Duesberg, who did much of the pioneer work on retroviral ultrastructure, knew that as direct pathogens the retroviruses were not 'slow' viruses, even lentiviruses like visna with which HIV was often compared. Rather, if Visna reached high enough blood concentration, it was rapidly pathogenic (10). HIV, however, was not to be found in such high blood titer. Perplexed, Duesberg summed that there was no such thing as a slow virus, 'only slow virologists (7)'.

The discovery of LAV (HIV) allowed virologists to take the high ground in American medicine. No longer would practicing physicians like Livingston, who had seen disease face to face, assume leadership on policy issues. The new medical shamans would be laboratory gene splicers, molecular biologists, virologists and immunologists who told doctors what to think. A dangerous precedent was being set.

Cambridge University Clinical School, Cambridge, England, September, 1983

Former viral cancer researcher Abraham Karpas, worked out of the Department of Hematological Medicine at Cambridge. By September, 1983 he had identified a 'transmissible agent' through electron micrographs of the blood of a gay AIDS patient (8).

Karpas was having a problem with Gallo's HTLV1 and was unable to confirm previous reports of this purported AIDS retrovirus in Africans. Many blood tests finding HTLV1 positive by previous investigators were found negative when retested in Karpas's lab (9).

Karpas, probably the second man in the world to see the AIDS agent, fired off a quick report on his transmissible agent complete with a microphotograph, but was having difficulty getting the paper published. He had the honesty to admit that he wasn't certain that the 55 nm particles with their 10 nm electron-dense cores were viruses at all and began his paper with the phrase 'assuming it is a virus', though he later found them identical to Montagnier's HIV.

Experts sided with Karpas's restraint. Not only were retroviral particles 'no proof that a virus was involved', but such particles were ubiquitous – a statement supported by O'Hara's Harvard study which found 'viral particles, morphologically indistinguishable' in 90% of the enlarged lymph nodes in both AIDS and non-AIDS patients (13). O'Hara's study stood out as the one study to date which used suitable controls, finding 'viral particles' indistinguishable from HIV in a variety of swollen lymph nodes without HIV.

Similarly, African studies of the lymph nodes of patients with HIV also showed them indistinguishable from those with just tuberculosis and without AIDS (12,15). O'Hara concluded 'The presence of such particles do not, by themselves indicate infection with HIV'. Yet it was photomicrographs of the same particles which first informed the world that there was an HIV.

In *Reproduction of RNA Tumor Viruses*, Badar warned that in vitro cultures, even virus free, 'can be induced to produce particles which resemble RNA tumor viruses in every physical and chemical respect (3)', an event many saw applicable to the rigors Montagnier and Gallo put their AIDS tissues thru.

Oddly, it would not be until 1997 that two independent groups would examine these HIV particles in accordance with accepted international procedure (6,4). Both teams saw an excess of fluid filled, many formed (pleomorphic) 'contaminating' vesicles, ranging in size from 50 to 500 nm as opposed to a minor population of particles of about 100 nm.

The latter were assumed to be viral, but proved, according to critics, to be too large, of a wrong shape and

containing too much material to be retroviruses. In fact, both the particles and vesicles of Bess and Gluschankof shared common antigenic determinants could easily have been, and do seem reminiscent of the variably acid-fast mycobacterial L-forms pointed out and electronically microphotographed by Seibert (14), Alexander-Jackson (1), Livingston (11) and Cantwell (5). Livingston showed a protoplast with L-form inclusions budding out vesicles from its surface not unlike the vesicles in the 1997 AIDS verification studies, while Seibert and Cantwell showed particles similar to those attributed to HIV. And the 'substantial amount' of both RNA and DNA found by Bess points to bacterial or mycobacterial origin.

Suddenly, it seemed as if the world had been sold a bill of sale on a non-existent virus.

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SMOKE AND MIRRORS

Like chameleons, HIV scientists continued to change their colors to blend into whatever new facts came along, much of it from the mycobacterial literature found in AIDS. And since the doctors and scientists who bought into the HIV theory were in the clear majority, and that majority ruled, their funding and literature mushroomed into a self-fulfilling archive while those that did not agree found themselves labeled heretics, lost tenure, research funding and even their jobs.

By 1983, the certain knowledge that AIDS had begun its wholesale slaughter of Africans, mainly through heterosexual sex, sent shudders down the back of a world in which, not since the last great sexual pandemic of syphilis five centuries ago, had there been anything comparable. Men and woman were transmitting AIDS back and forth sexually in heretofore unheralded numbers.

Wave after wave of epidemic tuberculosis had hit the world. It was a disease of big numbers. In the 100 years from 1850 to 1950, it was estimated that 1 billion persons died from tuberculosis (3).

From England it spread to the shores of Western and then Eastern Europe and by 1900, North and South American waves began to peak. But in the developing countries of Asia and Africa, where the AIDS epidemic was new in 1983, epidemic waves of tuberculosis had not yet reached their zenith (10). As this epidemic continued to seethe, it was these very countries that would show the highest TB mortality and morbidity, even before AIDS came into the picture (7), and would prove to be the future epicenters for AIDS.

Foreigners called the all-too-common African AIDS wasting syndrome 'slim disease'; Africans, just 'slim'. Serwadda wrote about it in *Slim Disease: a New Disease in Uganda* (9) but of 82 patients diagnosed with this wasting syndrome, he found 44% to have disseminated tuberculosis. Referring to what he felt to be the tip of an iceberg, Serwadda suggested that a substantial proportion, if not all, Slim Disease was actually due to disseminated tuberculosis (2). In addition, disseminated *M. Avium* too was believed to be a major cause of wasting syndrome in patients with AIDS with approximately 40% having nausea or diarrhea (6,8).

True, tuberculosis and diarrhea, prominent in African AIDS, had been killing Africans for some time, but suddenly they had become untreatable. A new name was needed for an old affliction. And that name, AIDS, was supplied, hurriedly, perhaps too hurriedly.

It was in Africa that those who hailed HIV as the cause of AIDS faced their first and most serious challenge over extremely suspicious coincidences. Not only were over 65% of African AIDS patients not HIV-positive (*Lancet*, Oct. 17, 1992), but, of those that tested positive, data suggested that the antigens in HIV-1 Elisa and Western Blots, originally claimed to belong solely to HIV, were cross-reacting with the mycobacteria (5).

Mycobacterial cell wall components, phenolic glycolipid (PGL) and lipoarabinomannan (LAM) were noted not only to strongly cross-react with p24, the sacred cow of 'HIV isolation', but p31, also favored in the detection of HIV in the blood (5).

Even the most prominent and persistently detected antigen in AIDS tests (11), p41, could be found in bacteria such as the tuberculosis.

Defensively, HIV enthusiasts shot back that tuberculosis in AIDS was merely an 'opportunistic infection', a label that most North American AIDS experts were originally extremely reluctant to assign. To John and Kaur, in a *Lancet* article, the term 'opportunistic' seemed inappropriate. Infections due to normally non-disease-causing microbes were 'opportunistic' (4). *Mycobacterium tuberculosis* was the only infectious pathogen ever to force the UN to issue a (1993) global emergency.

Many physicians, wary of the invented terms used to describe HIV, still remained silent. Makeshift expressions, like ARC (Aids Related Complex), PGL (Persistent Generalized Lymphadenopathy) and 'pre-AIDS' were scrutinized in disbelief. There were similar forms of latent TB, yet none were ready to call them pre-tuberculosis or tuberculosis-related complex (TRC).

By 1986 Montagnier's group, puzzled, found a patient in West Africa with AIDS but no HIV antibodies in the blood. Rather than rethink the whole HIV hypothesis, the discoverer of HIV proceeded to simply say that it was another retrovirus at work: HIV2, said to be responsible for a large West African epidemic, mainly transmitted through heterosexual intercourse (1).

Lacor Hospital in Gulu, Uganda, was in effect a TB sanitarium, but roughly half of the patients who remained there for two months or more came down with AIDS. And so Africans died, with the bleeding gums and anemia claimed at different times to come from both HIV and TB, but their blood was HIV-negative. In a word, they died of wasting, etc. or consumption.

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AMBIGUOUS SLAUGHTER

The scientific vagueness and deception to bolster the HIV theory continued.

Important to the basic mechanism of AIDS is the destruction of CD4 (T-cells) lymphocytes, key to the body's resistance against infection. As this CD4 cell count falls in the blood of an AIDS patient, many treacherous infections are able to jump on board.

HIV was early on claimed to destroy these CD4 white blood cells, yet the exact mechanism for this was never made clear (17).

What Papadopulos-Eleopulos makes clear is that retroviruses were never known to kill cells. It was the one thing retrovirologists always knew and agreed upon. Therefore, she asked, how could they kill CD4? Instead, it seemed to her that CD4 T Lymphocyte death might be due to the many non-HIV factors present in HIV inoculate, including other infectious agents (28).

And although low CD4 has been made synonymous with HIV by many, the fact is that known AIDS-risk groups may have low CD4, even in the face of persistently negative HIV antibody tests (7,9,26).

That HIV is not the cause of apoptosis (or immune cell destruction) of CD4+ is indicated by the fact that in chronically infected retroviral cell lines, where HIV is incessantly produced, apoptosis is not detected (28). Discoverer Luc Montagnier and others have confirmed that HIV does not kill T-cells like CD4+ directly (11,20).

On the other hand, virulent TB can depress the CD4 count (34), and does kill T-cells like CD4+ directly, as well as macrophages through nitric oxide secretion (32).

In 1978, the first European measurement of a low CD4 in AIDS was on a patient with disseminated atypical *Mycobacteria fortuitum* (5), closely related to *Mycobacterium tuberculosis*.

By 1987, Canadian researchers realized that mycobacteria such as tuberculosis alone could be responsible for direct CD4 killing and much of the immunosuppression found in AIDS. Furthermore, such a tubercular immune system throttle could persist for life, even when the disease wasn't progressive (19).

In the same vein, Mudaki, in Zaire, showed how fast a CD4 count could shrink below 200/ul just by tuberculosis, without HIV (25). Moreover, TB often presented before the development of immune dysfunction, either with or without HIV (30).

In fact of all the infections involved in AIDS, none were associated with as low CD4 cell counts as were mycobacterial infections (27). And those patients with either *M. Avium* or *M. tuberculosis* in their blood had significantly lower CD4 counts (14).

Yet there had to be more – a missing link. It has long been known that a low CD4 count in and of itself did not automatically lead to the severe immunodepression found in AIDS (18).

Case Western Reserve University, Ohio, July, 1998

Although previously demonstrated (19,8), the ferocity of a tubercular attack shown in papers such as Hirsh's 1999 Ohio study amplified that not only were 30% of CD4 and non-CD4 cells slaughtered within 98 hours of co-culture with TB, a 20-fold increase (15), but B cells (6,22) and macrophages (12,24) were also decimated.

The fact that both TB specific and non-specific T cells were equally affected would account for tuberculosis's silent role in the depressed responsiveness towards non-TB antigens such as Candidal thrush, *Pneumocystis*, and other opportunistic organisms; non-CD4 lymphocytes hold a possible role in keeping these at bay.

But it was in the annihilation of infection swallowing macrophages, critical to reticuloendothelial ultrastructure, that *M. Tuberculosis*, *M. avium* (4), or both working together, furnished the key to AIDS devastation.

California Pacific Medical Center Research Institute, San Francisco, California, 1999

A stagnant HIV hypothesis, much in need of rejuvenation, was expanded to include infection of macrophages, long the home base of tuberculosis and now claimed to be the most important reservoir of the AIDS 'virus' from which a sustained, long term attack on the body's lymphocytes could proceed (16). Although Duesberg and Levy saw HIV infection of macrophages as possible, their subsequent killing by HIV was not (10,21).

Indeed, the missing link in AIDS was the macrophage. It had long been known that certain white blood cells called macrophages ate (phagocytosed) bacteria. But how tuberculosis and the mycobacteria, naturally endowed with their own thick, lipid-rich cell envelopes, became the greatest assassins ever, presently responsible for a human death every 15 s (<http://www.stoptb.org/tuberculosis/#facts.html>) had to do with how they resisted lysosomal degradation inside the macrophage, multiplied there (1), and ate it up, from the inside out (13). Inside every macrophage swim two thin-membraned vacuoles: one, the phagosome, containing ingested bacteria; the other the lysosome containing lysozyme, a destructive enzyme tailored to kill bacteria. Usually, with infection, the two fuse or join, the acidic and enzymatic content of the lysosome killing bacterial elements harbored in the phagosome. It is how the macrophage defends the body. But after eons of evolution virulent mycobacteria and tuberculosis have developed a survival strategy which includes coating the phagosomes they find themselves in with proteins to prevent their enzymatic destruction (29), punching holes into the phagosomal membrane for nutrition and release of toxic products (33), evading enzymatic destruction even in those cases where vacuolar fusion has taken place (2), and learning to escape from such fused vacuoles (23), only to eventually kill the macrophage as the hunter becomes the hunted. Thus TB and the mycobacteria enjoy and thrive in a macrophagal lifestyle deadly to most other pathogens (31).

At California Pacific Research Institute, for example, Bermudez, Parker and Petrofsky watched ferocious AIDS *Mycobacterium Avium* destroy 28–46% more macrophages than uninfected cultures (4). And although it was known that both *Avium* and tuberculosis could escape dying macrophages only to kill and infect others, in the case of AIDS *Avium*, Bermudez saw a particularly menacing event in front of him: macrophage kill only made *Avium* more virulent and hungrier than ever (3), as it sought out its next macrophage victim.

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VIRAL MIRAGE

By 1995 David Ho, head of New York's Aaron Diamond AIDS Research Center, assumed the mantle of titular head of the U.S. AIDS establishment; he and his colleagues proclaiming a new proactive stance, asserting that HIV was never inactive and multiplied astronomically in the body each day, killing CD4 cells. But there was still no hard physical evidence, only theory, as to how HIV killed. Ho speculated that the carnage took place in the lymph nodes, so that there were few signs of infected CD4 in the blood. Then HIV not involved in this hypothesized

slaughter shot out into the blood stream, creating a 'viral load'. To eradicate viral load, Ho was suggesting early and aggressive anti-viral drugs taken in potent 'cocktails', with serious side-effects, and probably for the life of the patient. The problem was, as Robert Gallo later noted, that just about everyone he knew realized from the start that Ho's theory was absolutely wrong.

Nevertheless, soon HIV scientists were proclaiming that the amount of virus in the blood was the most important determinant in AIDS prognosis (9). But the fact is that HIV is so sparse in the blood as to require Polymerase Chain Reactors (PCR), a nucleic acid broth which makes copious copies of hard-to-find pathogens, to even detect it.

PCR inventor Kary Mullis would not support the use of his test to amplify and exaggerate what is being perceived as HIV in measuring 'the viral load', as is currently being done. To many, the massive amounts of RNA supposedly representing HIV in the circulation were suspect. Furthermore, dissidents wanted to know if you made a thousand copies of a dollar bill, how many real dollar bills did you still really have?

The 'viral load test', presently in use makes only copies of fragments of nucleic acids attributed to HIV (6) – does not count HIV itself. Since it does not count HIV itself and other infections, in particular the mycobacteria, can also yield similar nucleic acid fragments, a positive viral load test cannot be regarded as signaling HIV itself. Meanwhile, nobody ever questioned the validity of using a non-quantitative PCR in the detection of another hard to find pathogen. tuberculosis.

By 1994, British researcher John Kay walked up to a New Hampshire podium before the Proteolytic Enzyme Conference and announced that Hoffman-LaRoche's protease inhibitor RO31-8959, now called saquinavir or Inverase, hadn't worked out clinically in an 18-month trial with 400 AIDS patients (3). The reason given was that after an initial improvement in symptoms, HIV developed resistance to the agent and that for the time being Roche was imposing a blackout on the disappointing trial. Biochemist Dr. David Rasnick, an expert on the proteases, saw things differently. The inhibitors were performing their job as designed and that was to block HIV production. It wasn't mutation or resistance that were the problems, it was that HIV did not cause AIDS (Ibid).

By 1984, Rasnick, with extensive protease experience, was in a pivotal position to capitalize on their momentum but quickly decided that to kill a harmless retrovirus was an exercise in futility; often at the risk of severe and as-yet-unknown side-effects, some fatal in animals.

Although it was generally acknowledged by the HIV establishment that by the 1990s Highly Active Antiretroviral Therapy (HAART) made headway in braking the

steep rise in both AIDS and AIDS-related deaths in the U.S., no randomized study has ever been done comparing those on these drugs to those that are not (10). Furthermore, the precipitous drop in AIDS deaths in 1995 predates the introduction of the protease inhibitors, which first came onto the market in late 1996, by one year. This seems much akin to tuberculosis, which began to decrease long before any specific measures or drugs were used against it (4).

HAART, to be sure, from its onset was palliative. Nevertheless, with HAART, in many cases, the CD4+ count is partially restored and supposedly therefore the necessity for continuing drugs specifically against *M. Avium* in certain cases stopped. But *M. avium* infection rebounds when these anti-HIV drugs are stopped or fail (8). Furthermore, the antiretrovirals in HAART were not the only agents which could restore a CD4 count. This restoration also occurred in patients with HIV and TB when anti-TB treatment was used, as in John's study where a CD4 count of 89/ μ l climbed to 760/ μ l (5).

In truth, the entire story has not been nearly unraveled regarding America's potent anti-retroviral drugs. Regush mentions that the types of antiviral drugs used in 'cocktails' 'have antimicrobial properties that could, to varying degrees, target other infections that are common to AIDS' (10).

FDA approval for any of these agents did not require information as to whether they were bactericidal. Therefore, studies which show that widespread HAART reduces the risk for TB or may bring about the further decline of TB among persons infected with HIV (7) can never answer with certainty that the reason for this is not some heretofore unknown direct antimycobacterial activity on the part of HAART.

For example, in 1999 Bermudez et al. (1) documented that the intense macrophage and lymphocyte killing by American AIDS *M. avium* was significantly reduced by protease inhibitors called caspases.

These investigators, in effect, established that certain protease inhibitors, a first line of defense against 'HIV' found in HAART, were able to curtail *Mycobacteria avium-intracellulare's* virulence, thus pinpointing a much more specific and satisfying reason as to why, all of a sudden, MAI prophylaxis was not necessary and symptomatic improvement noted then that HAART was 'bolstering the immune system'. But the question remained: At what price?

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LUCKY

Once upon a time, a small group of politically powerful scientists rammed a flawed theory on the origin and cause of AIDS down America's and then the world's throat.

Yet we are still led to believe that we are fortunate that retroviruses, only discovered in the 1970s, were uncovered just in time to label them the culprit in a killer AIDS epidemic. Lucky that two 'HIVs' were discovered in rapid succession and the technology and theory to link AIDS to the HIV retrovirus were fully in place, for the first time in history, only a few years prior to the recognition of the AIDS epidemic.

Lucky? As the 20th anniversary of the first reported AIDS cases passes, AIDS has infected nearly 60 million people of which almost 22 million, including nearly half a million Americans have died, and 8500 AIDS deaths occur daily. Yet the prospects for a cure or vaccine are as remote as they were two decades ago. And although it is currently difficult to find anyone who openly questions HIV as the cause of AIDS, a fast growing-number express their doubts privately.

Historically, not that long ago, and at the end of the 19th century, after 150 years of denial, the medical establishment recognized that there were bacteria, and suddenly every disease seemed to be caused by a bacteria. But with the electron microscope, unknown disease was more and more attributed to a virus, often to no avail. Thus it was that scientists were certain that a virus was behind Lyme's disease, Mycoplasma pneumonia, and Legionnaires' disease before their respective bacteria were found.

By 1931, Rudenberg, hoping to visualize the polio virus, filed a patent for his electron microscope and during WWII investigators never gave up on an electron search for a cancer virus, despite one dismal failure after another.

Then an AIDS 'virus' was found, primarily because it was looked for; and not because it caused AIDS-bolstered by the half-truths, flawed theory and downright hocus-pocus.

HIV scientists cited that 'unassailable epidemiological evidence' (2) has established HIV or a virus as the cause of AIDS, including those epidemiological studies carried out by the CDC on filtered factor VIII blood transfusions for hemophiliacs. Blattner cites Peterman in his article when he said 'it is also noteworthy that HIV infection, and not infection with any other infectious agent, is linked to blood transfusion-associated AIDS (35)'. But blood transfusions do not distinguish between HIV and other filterable infectious agents especially, as in the case of mycobacteria, if these other infectious agents are not screened for. Yet, one of the original factor VIII transfusion cases happened in Canton, Ohio, yielding a diagnosis of oral thrush and disseminated *Mycobacterium avium* (8).

John Lattimer could not foresee the unusual situation which, decades later, might be involved with the direct insemination of particularly virulent mycobacteria rectally onto the vulnerable one layered epithelium of the prostate during gay sex, nor the hypervirulent strains of AIDS *Mycobacterium tuberculosis* and *Mycobacterium avium* that would one day, decades later, be shared in much greater numbers, heterosexually, and in a worldwide epidemic called AIDS. But who could have?

Nor could Gondzik realize the profound significance of warning his patients, on the eve of AIDS epidemic in 1979 to wear condoms lest they acquire sexually transmitted tuberculosis (cf. 14).

Mycobacterial infections are the main cause of bacterial infection during AIDS (34); and often precede other infections by 1–10 months (1). The fact that two decades after AIDS started killing people, mycobacteria, despite their prevalence in AIDS, are not considered its cause, is in no small part due to the lack of scientists and the lay public to understand the ability of mycobacterial and tubercular infection, once contracted, to lie seemingly dormant for extended lengths of time in humans (49), and at the same time begin to melt the immune system (27).

Nor was it emphasized that both tuberculosis, whose chronic lymphadenitis is its most common extrapulmonary manifestation (22), and *M. avium*, the most common cause of lymphadenitis in children (18), can attack lymph nodes (41) and the entire body additively and simultaneously (42).

AIDS is a mycobacterial disease and patients with advanced TB or *Avium* are indistinguishable from those with 'HIV'. Even in the earliest AIDS cases on record, dating back to 1959, tuberculosis (47) and the atypical mycobacteria (40) (17) were clinically and bacteriologically diagnosed.

Perhaps one of the most convincing arguments for the intimate causal link between mycobacterial infection and AIDS comes from the widespread geographical overlap of the two. Worldwide, by conservative estimates, around one in three people is infected with *Mycobacteria tuberculosis* alone, or 1.8 billion people (48) and the very cities, in which up to 80% have tuberculosis, are the epicenters of AIDS. The last world AIDS conference in Barcelona admitted that 1/3 of all AIDS deaths were from diagnosed tuberculosis (30).

Almost a million new cases of AIDS were estimated to be attributable to tuberculosis in 1995, and by the year 2000 there were probably 8 million co-infected people worldwide (36). Figures such as these make it unconvincing that AIDS ever surpassed tuberculosis as the leading cause of death in the world, especially in lieu of cross-reacting sera that opens up the question as to just how many cases attributed to HIV are in fact from tuberculosis or allied mycobacteria.

When the AIDS epidemic officially began in June, 1981, allergist/immunologist Michael Gottlieb of UCLA, after first implicating cytomegalovirus (CMV), wrote, *Ongoing AIDS Epidemic Could Be Product of Dual Pathogen Infection*, concluding that AIDS resulted from not one but two microbial infections (15). Unexpected in early AIDS autopsies was the surprisingly high proportion of difficult to diagnose *Mycobacterium avium-intracellulare* (46), in up to 55% (24) of American cases. But in Haitian and African AIDS patients, undoubtedly just as exposed to *Avium*, death by *Mycobacterium tuberculosis* predominated.

Most convincing evidence points to Gottlieb's hypothetical dual pathogens as being atypical mycobacteria such as *Avium* in somehow getting into a human blood pool already harboring latent or active *Mycobacterium tuberculosis*. The 'acquired' in Acquired Immune Deficiency Syndrome is *Mycobacteria avium* or a similar nontuberculous mycobacteria; 'immune deficiency' but the result of a savage double attack on the immune system by an atypical mycobacterium (such as *M. avium* and *M. tuberculosis*). *Avium* has, in the short space of 30 years, gone from relative obscurity to the leading infectious disease in U.S. and European AIDS, and there is no dearth in the literature, ranging from sexual transmission (5,6), bestiality (5,23), certain Voodoo practices, African ritualistic drinking of animal blood (37), and the

medical or addict's use of shared needles (38) to explain just how it could have been introduced and spread throughout the human blood pool, whether in Africa, the U.S., Europe or the rest of the world.

By the late 1960s, lymph nodes from 368 swine from Transvaal and Natal South Africa were examined and found to mostly contain *Mycobacteria avium complex* (MAC) (25). Similar strains were then identified when attention shifted to humans in Africa in the early 1970s (26).

Only a few strains of MAC are found in human AIDS (24). Notably these are also found in simian AIDS (19). Weiszfeiler and Karczag (45) succeeded in isolating 50 strains of Mycobacteria, including MAC from (33) monkeys, a significant finding as millions of pre-AIDS African were vaccinated with an early polio vaccine attenuated in living monkey kidney tissue. Some see a correlation between where the bulk of polio vaccine was administered and the epicenters of AIDS (20). The Simiae-Avium (SAV) group of mycobacteria, which shares characteristics of both *M. avium* and *M. simiae*, by itself is entirely capable of causing a lethal AIDS infection (29).

Soil borne, MAC is found in cats, swine, and primates, all significant in early retroviral theories regarding AIDS.

The fertile soil upon which *Mycobacterium avium* and similar 'atypical' mycobacteria plants AIDS is pre-existing TB, often latent, always immunosuppressive. Despite WHO estimates, Fox maintained that nearly half the world has TB (10) but others feel that number to be much greater.

Cantwell, who repeatedly found acid-fast forms in AIDS, felt it reasonable to assume that the initial immunosuppression disease in that disease must also be present in many 'healthy' people as studies indicate that some promiscuous but otherwise 'healthy' gays were actually immunosuppressed to begin with (4). *Mycobacteria tuberculosis*, both in its vast reservoir of seemingly well and its human immune-killing potential, certainly fulfills this criterion. Papadopulos-Eleopulos 33 reminds us that in African AIDS, 'HIV infection' usually follows TB.

The first case of human disease due to *Mycobacterium avium-intracellulare* (MAC) was reported in a middle-aged Mesabi Range iron miner in 1943. His symptoms were pulmonary (9) and until the emergence of AIDS, lung infection alone typified *Avium*, though differentiation between fowl or *Avium* tuberculosis and tuberculosis itself was at times nearly impossible (32). The first reports of MAC in AIDS appeared in 1982 and Zakowski found it in eight or nine patients who died of AIDS at the UCLA Medical Center (50). Soon the devastatingly immunosuppressive potential of co-infection with *M. tuberculosis* and *M. avium* was shown (42). As already mentioned, this ability of mycobacteria to attack simul-

taneously is a recurrent theme in the literature, occurring over and over again. But in AIDS, it would bring on a combined immunosuppression the likes of which man had never had to deal with. The known ability for AIDS and TB to potentiate one another (13) is a result of such double-pronged mycobacterial attack between 'AIDS', the atypical mycobacterial infection, and tuberculosis.

In such a scenario HIV is simply one of the L-forms of an atypical mycobacteria, in particular *M. avium* and until it is recognized as such no 'retroviral' vaccine or cure will be possible.

In the U.S., on the surface, AIDS is characterized by the severe immunosuppression of *Mycobacteria avium* (MAC) and opportunistic infections like Kaposi's Sarcoma and Pneumocystis Carinii. In Africa it is a wasting disease characteristically ending with death by *Mycobacteria tuberculosis*. HIV should cause the same disease where it from the same cause.

In *Disseminated Mycobacterium avium Infection Among HIV Infected Patients in Kenya*, Gilks approaches this most perplexing AIDS enigma in terms of the mycobacteria, addressing the apparent relative rarity of disseminated MAC in AIDS in Africa and the developing countries (12). That MAC exists in the African as well as the American environment (43) cannot be denied. Nor can the fact that African skin tests prove antibodies to MAC already in African blood (44). Gilks acknowledges that AIDS patients in developing countries are probably dying of more virulent tubercular infections before they become immunosuppressed enough to show *Avium*. Inderlied (21) and O'Keefe (31) agree.

Gilks mentions that AIDS patients in Africa, already infected with latent tuberculosis, are more likely to reactivate this mycobacteria with catastrophic results before reaching the low CD4 level associated with clinical MAC. This, however, does not preclude the fact that *Avium* or a similar non-tubercular mycobacteria, as causative, plants AIDS in the soil of previous tubercular infection, whether in Africa or elsewhere.

The full extent of drug-resistant TB in African countries is unknown but at least as prevalent as it is in New York, Haiti or the Ivory Coast (28). Frieden found 30% of resistant strains of tuberculosis in New York (11) AIDS patients, but Shafer assured that with or without HIV, drug resistant tuberculosis was comparable (39). How much of multi-drug resistant (MDR) TB is in fact a fusion with the atypical mycobacteria like MAC or SAV is an open question. Meanwhile, there hasn't been a new TB drug in 30 years (30). For its part *Avium* (swine, or fowl tuberculosis) and the 'atypical' mycobacteria in man has never had a truly satisfactory treatment (16). Whether this situation will change with novel strategies now in the pipeline (3) remains to be seen.

Multi-drug-resistant strains always had the potential to render tuberculosis and *Avium* once again incurable infections (7). That this happened, and how new and virulent strains of mycobacteria found entry first into the human blood pool and then an enhanced portal of sexual transmission in the disease eventually called AIDS should come as a surprise to no one.

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