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"HIV" – A Case of Mistaken Identity, Political Profiteering, and Pharmaceutical Murder

by Liam Scheff

What is AIDS

The AIDS mainstream butters its significant pile of bread with the notion that there is in existence a single particle, which they call "HIV," which is known to do one thing, and that is to "kill T-Cells." But behind the public pronouncements of HIV-AIDS-sex-panic-mania, the details in the published research paint a different, and frankly more interesting picture.

The mainstream will tell you that AIDS is caused by HIV, and HIV is the cause of AIDS; and you're left with a tautology – a circular definition that doesn't define either term.

In real-world use, **AIDS** is "immune deficiency," and it's real enough, but is has no one cause. AIDS is a **brand name** for fatal poverty in the 'third' world, and for major drug abuse, and the attending illnesses, in the West.

HIV, too, is a kind of brand name. This time, for a group of laboratory artifacts and events; **a collection of proteins** drawn from experiments with leukemia cells, that were mixed with tissue that came from gay men who were sick.

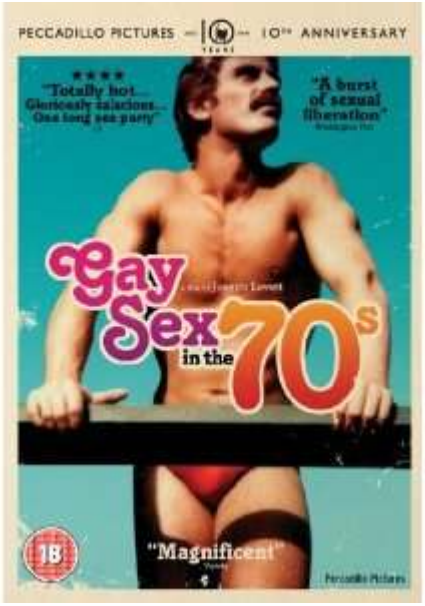
"Previously Healthy Young Gay Men"

The young gay men who were the first AIDS patients were drug users of a very high order. Life was a stream of party drugs, antibiotics, and a 'fast-lane' lifestyle, that encouraged life as a non-stop good time, in the urban 'gay ghettos' of the 1970s (and now). This bit of history is 'forgotten' by papers like the *New York Times,* which likes to **pretend** that it was "previously healthy gay men," who mysteriously fell ill.

But the medical and social literature of the era tells the non politically-corrected truth. The first illness called AIDS occurred in young gay men who using too many drugs, having too many partners – dozens to hundreds per week in the 'fast lane' life, having a pile-up of STDs, and taking so many antibiotics so regularly, that they gutted themselves, stripped their intestines of all protective layers, and became susceptible to every illness.

Drugs, STDs and Antibiotics, not Sexual Identity

But it wasn't all gay men who got sick. Missing from the first AIDS *(or GRID – Gay Related Immune Disease – as it was called)* cases was the vast majority of gay men – those who did not live in the 'fast lane.' It wasn't sexual identity that caused the various AIDS illnesses. These were caused by the lifestyle of a very specific group of people responding to the availability of drugs in the 'liberated' ideology of the 1970s. It was post 'sexual revolution,' post-Vietnam War, post-Nixon America. That is to say, were were a discouraged, cynical, and drug-addled nation.



Gay activists don't like to hear the toxicological arguments about AIDS; they consider it "homophobic" to talk about how drugs and antibiotics ruin a system, for some reason. But this isn't an indictment of gay men. It's a description of toxic compounds working in the body. (I personally support gay marriage, rights, and all that attends).

The urban gay population was a ghettoized, isolated, and rather 'incestuous' non-stop party in the 1970s, in the thrall of deep drug use and multiple partner swapping, specifically in the inner city 'islands' of New York, Los Angeles and San Francisco. These were **safety zones** where young men would flee to for protection, from the violent and mortally-dangerous homophobia that was rampant in the US.

But this reality was politically altered by activist groups in the early 1980s, who were pushing back against the ultra-conservatives, who claimed that homosexuality was a sin, and that AIDS was a "punishment from God." Given a spiteful Right wing, and a conservative, **pharmaceutically-minded Left wing**, the gay community, in tatters, chose the lesser of two real evils.

AIDS+Propaganda=Sex

Through carefully-crafted propaganda campaigns, like that created and expedited by amFAR, under Dr. Matilda Krim, we were made to believe that everyone was at risk for immune deficiency from having sex. The 'sexual AIDS' notion was **a political maneuver, and not a scientific principle**, and has been revealed as such time and again in the press, in

official documents, and in medical studies. But the culture at large took to it. It was as though we needed some moral bulwark to reframe sex, in the wake of the morbid and depressing "anything goes" era of the late 60s and 70s.

In no time, the medical authorities cast the "AIDS" diagnosis over a wider group of drug users – including heroin addicts, and then expanded it to Hemophiliacs, and then the poorest of the poor Africans. So, AIDS became any illness in "populations at risk." But, "at risk" for what? "At risk for AIDS!" is the answer. And we have our second circular definition in the AIDS campaign.

It's not politically-correct to talk about AIDS as a toxicological illness; but that's what it is. It's also highly treatable, when treated as an illness of toxic overload. When it's treated as a 'one cause viral infection,' however, it proves fatal. Because the drugs used on the body to "treat" a "virus" are deadly, and do not rebuild the immune system…

LAV, er, HTLV-iii, um, "HIV" Loves, em, Kills? T-Cells….

But, back to the 1980s. Cancer-virologists were mixing cells from "AIDS" patients to see what they could get. Different labs took these different leukemia T-Cell mixtures – called **"immortal" because they didn't die**, and then siphoned out some proteins, and decided, over time, that some proteins were more important than others.

Some labs found a 25, or a 24, or a 41, or 120-ish kilo-dalton (really small!) protein in their drug-addled patients cells, once they'd stimulated them and mixed them with a line of cancer cells. Did they ever find one, resilient, consistent particle?

Some labs decided that they had **a class C particle. Others a class D.** Some had protein 51, and some had protein 25, and some had protein 17. But it was "together," (though separately), that they "*inferred*" their "killer virus."

The presence of a "single virus" was never observed strictly by the old-fashioned "purification and isolation" process. It may surprise the newcomer to discover that in the entire annals of "HIV" research, you can't find a purified sheet of particles, anywhere, that can be passed on, and re-purified, and then is demonstrated, bio-mechanically, to kill or eat or bite or pinch T-Cells.

The mainstream knows this, but has ready excuses. From the start, they claimed that "HTLV-iii," or "LAV," as different labs called their cell mixtures, was considered to be "**too fragile**" to purify. That it tore itself apart after popping out of the cell wall; that it would be "difficult to transmit." Beyond that "it's" genetic material **always seemed to be different**.

So, a theory was born: The cellular garbage, debris, and the various proteins were given a new name: "HIV." And the press would report that it was "wily," and "always mutating," and "fragile!"

And yet somehow it still killed T-Cells.

But remember, to this day, these special proteins (which get called "HIV") are grown in T-Cells. "Immortally." For any serious-minded researcher, the end of AIDS theory was the beginning. T-Cells and whatever researchers are calling "HIV" have **nothing but warm regards** for one another.

Tiny Bubbles

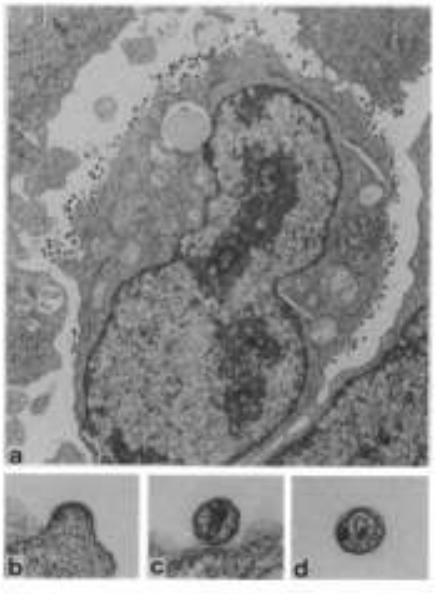


Fig. 1. Transmission electron micrograph of fixed cells obtained from the saliva of patient No. 8. Procedures for preparation of saliva are described in the legends to Table 1. (a) Leukocyte expressing HTLV-III (×10,000). (b) Budding virus (×150,000). (c) Mature virus particles (×150,000).

Even though researchers couldn't isolate and purify their golden goose, they could take pictures of stimulated cells in lymph tissue, popping out protein bubbles. These bubbles, some of the time, and in some cases, they called "LAV" or "HTLV-iii," and later "HIV."

Funny thing though, they could find these same protein bubbles **in human saliva!** Which means that all 8th graders are now at risk for the deadly AIDS plague!! (If the little bubble causes immune deficiency, by killing T-Cells).

Does it? Let's find out.

First, let's go to a response from the researchers at *Rethinking AIDS* to Robert Gallo, the multi-millionaire inventor of the AIDS paradigm. What do they have to say?

> "Until recently, prevailing dogma said HIV causes AIDS by killing T-cells. Most people, including Robert Gallo, still adhere to this belief even though there is no evidence to support it. On the contrary, the wealth of evidence available clearly shows that HIV does not, in fact, kill T-cells. This is not surprising since **the hallmark of retroviruses (HIV included) is that they do not kill cells** [1, 2].
>
> The discoverer of HIV, Luc Montagnier, heads [3] a list [4-6] of virologists who have confirmed that HIV does not kill T-cells in culture. Neither does it kill T-cells in human beings. Mario Roederer of Stanford University said in an editorial in 1998 [7] that the results of Pakker et al. [8] and Gorochov et al. [9] "provide the **final nails in the coffin** for models of T cell dynamics in which a major reason for changes in T cell numbers is the death of HIV-infected cells."
>
> [...]
>
> The fact that HIV does not kill T-cells has caused a remarkable about-face in mainstream thinking. Commenting on a recent paper by Hellerstein et al. [12], Guido Silvestri and Mark Feinberg summarized in 2003 the latest speculation that HIV causes AIDS not by killing T-cells but by over-stimulating the immune system [13]. Silvestri and Feinberg inform us that, **"Prevailing views...have shifted from models that**

> **focus primarily on direct HIV-mediated killing of CD4+ T cells to models that emphasize the pathogenic role of generalized immune system activation."** In other words, HIV no longer causes AIDS by killing our immune cells, as Gallo contends, but by boosting our immune system." Rethinking Aids.com/Gallo Rebuttal

Huh! So, "HIV," however they're quantifying or qualifying such an entity, used to kill T-Cells. But now it "**activates our immune system?**" That's what we call an "about face!" And it means that the mainstream should certainly retire the T-Cell theory.

But how do they know what "HIV" does, if they can't purify it? Answer – they "infer."

When researchers talk about "HIV," they're not talking about a particle. They're talking about finding evidence of **an agreed-upon consensus-described protein, or an enzyme**. And that's it. Not a biting, toothsome little particle. Not even a soft, happy little protein bubble. Just some "p24" or maybe "p41." It depends on who's doing the study, and what their limited criteria are.

No joke, that's how it works.

So, "p24," (or "HIV") doesn't kill T-Cells? But, maybe we're giving those RA guys too much credit. Let's see what mainstream AIDS researchers think about T-Cells and "HIV/LAV/HTLV-iii."

Dear Experts, How Does "HIV" Smash Up T-Cells?

From the inception of the paradigm in 1984, to the present, the answer is the same: "We don't know, but keep sending money":

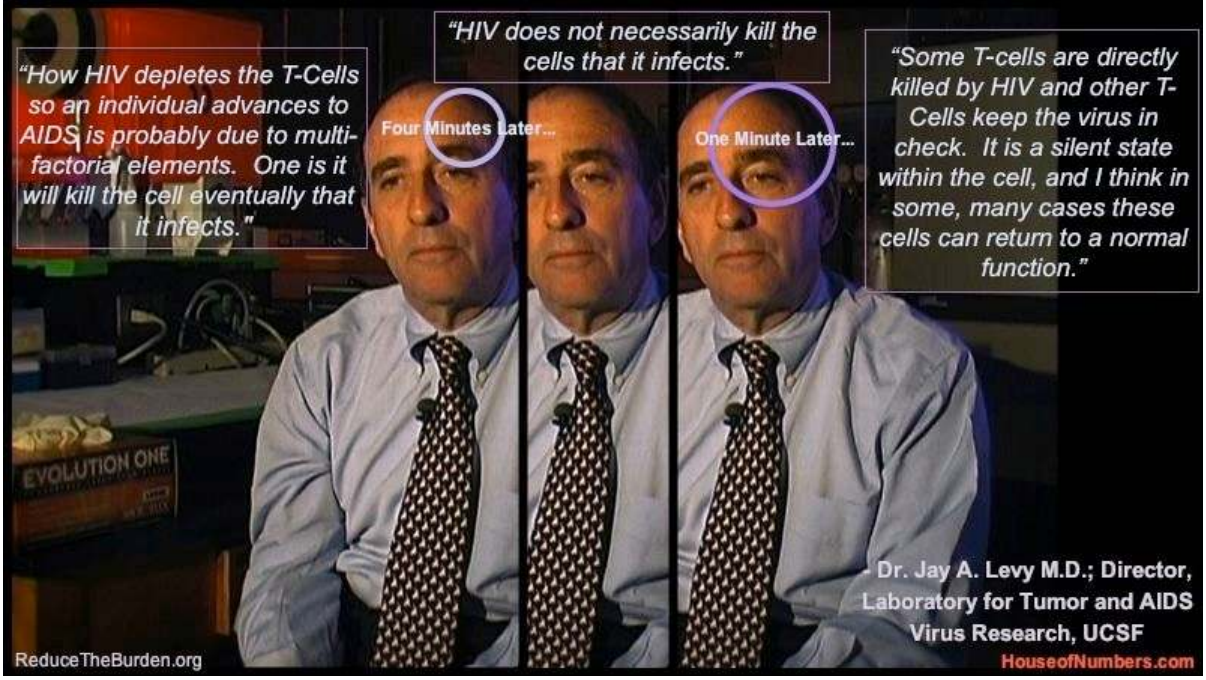
- "We are **still very confused** about the mechanisms that lead to CD4 T-cell depletion, but at least now we are confused at a higher level of understanding." — *Dr. Paul Johnson, Harvard Medical School (Balter 1997)*

- "We still **do not know how,** in vivo, the virus destroys CD4+ T cells.... Several hypotheses have been proposed to explain the loss of CD4+ T cells, some of which **seem to be diametrically opposed**." — *Joseph McCune, immunologist (McCune 2001)*

- "Despite considerable advances in HIV science in the past 20 years, the reason why HIV-1 infection is pathogenic is still debated... There is **a general misconception** that more is known about HIV-1 than about any other virus and that all of the important issues regarding HIV-1 biology and pathogenesis have been resolved. On the contrary, what we know represents only a **thin veneer on the surface** of what needs to be known." — *Mario Stevenson, virologist (Nature Medicine 2003)*

- "Twenty-five years into the HIV epidemic, a complete understanding of what drives the decay of CD4 cells – the essential event of HIV disease – **is still lacking**.... The puzzle of HIV pathogenesis keeps getting more pieces added to it." — W. Keith Henry, Pablo Tebas, and H. Clifford Lane *(Henry 2006)*

So, it's still a "puzzle" to the mainstream. Do T-Cells die when in the presence of "HIV" DNA? (Which is always different!) No. Or, *"We're confused at a much higher level of understanding"* is the official answer.



Here's a paper from 1994, ten years after terrifying the world with their 'discovery.' Gosh, they do seem to be having trouble...

> Indirect mechanisms of HIV pathogenesis: how does HIV kill T cells?
>
> Although twelve years have passed since the identification of HIV as the cause of AIDS, **we do not yet know how HIV kills its target, the CD4+ T cell**, nor how this killing cripples the immune system. **Prominent theories include direct killing** of infected CD4+ T cells by the action or accumulation of cytopathic viral DNA, transcripts or proteins, or by virus-specific cytotoxic T lymphocytes, **and indirect killing of uninfected CD4+ T** cells (and other immune cells) by autoimmune mechanisms, cytokines, superantigens, or apoptosis. In the past year, studies have provided *tantalizing clues as to why infected cells may not die* and how these infected cells kill innocent bystander cells. [LINK]

Huh. "Infected" cells (those that test positive some garbage protein or another), "may not die?" Do tell! "Theories include" either "direct or indirect" killing of T-Cells. Maybe. Maybe not! *"Tantalizing clues"* are offered as to why they don't! Well, golly. How very...expensive.

What you're reading in that single paragraph is billions of wasted tax-payer dollars. And hundreds of thousands of patients killed by extremely toxic drugs, used to "kill the virus" that "kills T-Cells."

"Life-Saving" Life-Ending Drugs

AIDS drugs are strong and toxic. They can wipe out a fungal infection faster than most chemicals, which can bring temporary relief to people with chronic Candida (a symptom of a ruined intestine and lowered immune and cellular strength). But they very quickly work on the patients body. These drugs dont' differentiate between your proteins and those of a bacteria or fungus.

The drugs given to AIDS patients don't kill any viruses. That's the advertising lie under which they're sold. **They kill cells and tissue tissue in which 'virus' is supposed to replicate.** That tissue is bone marrow and blood vessel, liver and spleen, stomach and intestinal lining, skin and heart, and, well, you get the picture. AIDS drugs kill AIDS patients. That's not really in doubt – not even in the mainstream AIDS 'theology.' They just accept the death of patients based on their "model." (You can find the thousand complaints by patients taking the drugs at sites like TheBody.com).

But if the model would change from "angry virus" to **"toxicological damage,"** then treatments

focusing on reversing intestinal and organ damage could be promoted- and lives could actually be improved. Wouldn't that be "tantalizing?"

I bet it would to people given the AIDS death sentence. It would also be a lot cheaper than drugging them to death. *(That also means, "less profitable for drug companies"- and maybe you can see some of the obstacles to taking this road!)*

But that was 1994. Let's look at a more recent paper, from 2003, having all the same troubles. Now we see the "models are shifting." In other words, well… the old theory is dead. Long live the new (old) theory! *(Which is a "model." ie. Not real).*

> "Prevailing **views** concerning the pathogenic mechanisms of AIDS **have shifted from models** that focus primarily on direct HIV-mediated killing of CD4+ T cells to models that emphasize the pathogenic role of generalized immune system activation. The observation that increases in T cell turnover seen in HIV-infected individuals primarily reflect increased proliferation of effector-memory T cells supports the concept that **chronic immune activation plays a prominent, if not predominant, role** in the pathogenesis of AIDS." [LINK]

At a certain point in any journey, you've got to make a decision. Do you stay on the road you're on, or do you find a new path? You might think we can more than fairly say that the "HTLV-iii/HIV/LAV/crazy protein garbage kills T-Cells" road is dead. And the mainstream is pretty close to agreeing with you. And now they want to kill something new.

Exosome, Not HIV

Let's visit a passage from Janine Robert's excellent 2008 book, Fear of the Invisible. Remember, "HIV" *(whichever consensus-agreed protein any one researcher is talking about)*, is supposed to be a "retrovirus." Here, Janine explains that the mainstream is coming to understand that retroviruses are not 'viruses.' They are They are messengers.

> "The National Institutes of Health (NIH) reported that retroviruses **'are so irregular and so labile that we have been unable to apply the tools of structural analysis to good effect.'** It also reported that retroviral DNA 'closely resembles a cellular mRNA' messenger vesicle. Retroviruses are also said to be '**unique among animal viruses in that some groups exhibit considerable polymorphism in receptor usage.**' They are thus particularly well suited for carrying messages – as they can deliver 'irregular', or varying, code 'similar to' mRNA to many kinds of cellular receptors." [LINK]

That is, these particles, these bubbling proteins, fragile and sensitive and ever-changing, are flexible precisely because they have to be. They are message carriers, not specific pre-determined entities. **They change from cell to cell, and function to function.**

Here's the quote from the NIH:

> "The continuing mystery of retroviral structure reflects less a lack of will—or skill—on the part of researchers than on a quirk of nature. Mature virions are so irregular and so labile that we have been unable to apply the tools of structural analysis to good effect." [LINK]

In other words, *"the damned things change too much to be classified."*

> "Retroviruses are unique among animal viruses in that some groups exhibit considerable polymorphism in receptor usage among otherwise closely related viruses." [LINK]

Again, they come and go. They're flexible. They carry genetic messages of variable size and code. They're never the same. **They don't eat T-Cells. They carry messages.** So, are they "viruses?"

or “retroviruses?”

Retroviruses Are Exosomes, and so is “HIV”

Take this quote from the latest “HIV” research. “HIV is an exosome?” *(What does it mean? What is an “exosome?”)*

> “Hildreth now proposes that “**the virus is fully an exosome in every sense of the word**.” Others have found that HIV particles contain MHC, but by the exosome hypothesis they may also contain proteins that exosomes use to fuse with target cells and to avoid attack by complement. As Gould points out, an exosome makes a perfect vector for HIV, because an exosome “is not just proteins in a vesicle, it’s something that is meant to traffic.” [LINK]

An “endo-some” is a protein bubble (vesicle) made inside (“endo-“) the cell, for carrying information, or goods and services, to another part of the cell. An “exo-some” is the same thing, but for use outside “exo-” the cell.

In other words, exosomes are a **non-toxic part of every living thing on earth**. They are little protein bubbles which are made by the cell, of the cell, for the cell; for healing, transport, messaging. For evolutionary adaptation!

And so is “HIV.” *“Fully an exosome, in every sense of the word.”*

The mainstream has now given up on the delusion that there is anything abnormal or unnatural occurring in “HIV” pictures. They now relate it to normal cellular processes. And so now, in their infinite wisdom, they want to kill all exosomes.

No, I’m not kidding. Now they want to create drugs that **destroy your exosomes**.

> **“To block all entry, suggests Hildreth, perhaps the MHC should be the target.** Alloimmunization—immunization with a wide range of MHC and other protein variants (e.g., by injecting killed leukocytes)—might allow a newly infected individual to mount a quick attack on the incoming HIV, which is packed with foreign MHC. Gould even suggests, “this is why we have tissue rejection responses— [they evolved] to protect us from retroviruses.” He points out that alloimmunity predates and thus could not have arisen from adaptive immunity.”

What is MHC? “Major Hisocompatability Complex.” It’s part of the genome of most and maybe all living things. AIDS researchers are now devising ways to destroy it in AIDS patients. What could possibly go wrong? *(You excited to find out? Me neither).*

> “The more extreme idea of xenoimmunization does work in monkeys, which can reject SIV grown in human cells. And for Thomas Lehner (Guy’s Hospital, London, UK), who has been pushing the idea for several years, alloimmunization “is far better than anything we have at the moment.” But it has languished since the monkey experiment, perhaps based on fears that it would **prevent later transplants, cause rejection during pregnancies,** and fail to catch a handful of HIV particles before they replicate and thus incorporate self-MHC.”

Huh. *“Rejection during pregnancies. Prevent transplants.”* And who knows what else.

So, yes, having discovered that “HIV” isn’t “HIV,” and doesn’t kill T-Cells, AIDS researchers now want to “block all” exosomes. But, didn’t we just learn that exosomes are required for life? Yes. Yes, we did.

Why the Hysteria?

Why are these researchers so hell-bent on the idea that one particle, which is clearly not one particle, has to be responsible for every of the dozens or hundreds of illnesses lumped together and called AIDS?

Why do they cling to the notion that something that is clearly a normal part of the body, an "exosome, in every sense of the word," is also eating T-Cells? Or, stimulating the pants of off them? Or….whatever it was that Jay Levy said. *"Returning to normal!*"

It seems to me that if the original researchers – Gallo, Montagnier, Levy, Weiss, Francis, etc – had been honest from the start, they would have said the following:

"Well, of course the genetic material we're finding is always different; we're just smashing up cancer cells and mixing them with tissue from sick people, and measuring DNA fragments. And we find something different, and call it the same thing! That's crazy!"

But they didn't say that, because they really, really wanted the idea of a single particle that caused cancer to be true.

When "AIDS" was "Cancer"

When you think of AIDS, you've been programmed to think of a sex-virus. But the first AIDS disease in the young gay men who were the first 'AIDS' cases was a cancer – **Kaposi's Sarcoma**. And the cancer researchers *really* wanted to prove that a virus was the cause, because they'd been trying to prove it for years and years. They'd spent billions of dollars. And it had all been a bust. And then, the young gay men came around with their broken immune systems, and they saw an opportunity – and took it.

That is, cancer-virologists had failed for 10 years or more to demonstrate that viruses caused cancers. In the waning days of their tax-payer funded excursion into oblivion, young gay men turned up with suppressed immune systems. These men had been taking handfuls of drugs, antibiotics and had a pile up of concurrent STDs. And some of them also had **a cancerous growth, a sarcoma** – a big ruddy purple spot on the skin.

The cancer researchers really, *really* wanted it to be caused by a virus, so they elected their own failed experiments to prove it. Robert Gallo recycled his HTLV-i and HTLV-ii, and decided that in these same cultures, was an new one, which he called…*HTLV-iii*.

Physicians at the time said that the sarcoma was caused by **a mutagenic drug, called "poppers,"** that these young, feverish gay men had been huffing all night long, for years on end. But, remember, the cancer virologists *really, really, really* wanted it to be caused by a virus. **So, that's the theory they supported.**

By 1994, everyone admitted that the sarcoma was *not caused* by any of the whatevers that they called HTLV-iii, or LAV, or *"HIV*.. It was probably – you guessed it – caused by the mutagenic, DNA-damaging drug they were huffing all those many nights.

Instead of working on **preventing toxic damage**, AIDS researchers have targeted human cells, organs and tissue with truly deadly cell-smashing chemotherapies, in the hopes of killing all retro-elements, or, as they're now called, "exosomes." The result has been drugs that kill bone marrow, peel skin, and cause morbid disfiguring illness, organ failure, and slow, painful death.

They've even taken pictures of their accomplishments, though they hide them when they advertise **"Product R(ed)."**

Save the Exosomes!

Will killing exosomes bring back the dead? That seems to be the purpose of all AIDS research – to stop what happened to rescue the poor souls who drugged themselves to death, and then were finished off in hospitals. The result has been to kill of an entire generation of young gay men with drugs like AZT, and to re-create a **Eugenics movement in Africa,** under the banner of the 'red ribbon.'

If we now kill all exosomes, will poor Africans suddenly have food and safe water to drink? Will young gay men who overdo drugs or pharmaceuticals rebuild their immune systems? Will heroin addicts suddenly be rendered healthy? Because these are the people targeted with the AIDS brand.

The medical and social literature admit that AIDS patients **can be successfully treated by intensive immune-rebuilding programs,** and that they slowly die without such intervention. It's also well-known that AIDS drugs kill, either quickly or slowly.

It is time for AIDS patients to turn away from the medical establishment in seeking more "cures" that kill, and start to look toward long-term, intensive and rigorous immune-restoration protocols. These won't be endorsed by the AIDS mainstream for some time, but you don't need their permission to get better. And figuring out how to make people better – not sicker – should be the central work of anyone working on this issue. And that means, letting go of bad theories, and starting to look at actual, living immunity, which starts in the intestine, and not under the microscope.

Further Reading:

http://reducetheburden.org/ – click on 'recovery'
http://www.fearoftheinvisible.com/why-our-cells-make-retroviruses
http://en.wikipedia.org/wiki/Major_histocompatibility_complex
http://en.wikipedia.org/wiki/Endosome
http://www.retrovirology.com/content/6/1/40

Notes for the RA citation:

1. Rubin H, Temin H. A radiological study of cell-virus interaction in the Rous sarcoma. Virology 1958,7:75-91.
2. Weiss R, Teich N, Varmus H, Coffin J. Molecular Biology of RNA Tumor Viruses. Plainview, NY: Cold Spring Harbor Lab. Press; 1985.
3. Lemaitre M, Guetard D, Henin Y, Montagnier L, Zerial A. Protective activity of tetracycline analogs against the cytopathic effect of the human immunodeficiency viruses in CEM cells. Res Virol 1990,141:5-16.
4. Langhoff E, McElrath J, Bos HJ, et al. Most CD4+ T cells from human immunodeficiency virus-1 infected patients can undergo prolonged clonal expansion. J Clin Invest 1989,84:1637-1643.
5. Anand R, Reed C, Forlenza S, Siegal F, Cheung T, Moore J. Non-cytocidal natural variants of human immunodeficiency virus isolated from AIDS patients with neurological disorders. Lancet 1987,2:234-238.
6. Hoxie JA, Haggarty BS, Rakowski JL, Pillsbury N, Levy JA. Persistent noncytopathic infection of normal human T lymphocytes with AIDS-associated retrovirus. Science 1985,229:1400-1402.
7. Roederer M. Getting to the HAART of T cell dynamics. Nature Medicine 1998,4:145-146.
8. Pakker NG, et al. Biphasic kinetics of peripheral blood T cells after triple combination therapy in HIV-1 infection: a composite of redistribution and proliferation. Nature Medicine 1998,4:208-214.
9. Gorochov G, et al. Perturbation of CD4+ and CD8+ T-cell repertories during progression to AIDS and regulation of the CD4+ repertoire during antiviral therapy. Nature Medicine 1998,4:215-221.
10. Grossman Z, Herberman RB. T-cell homeostasis in HIV infection is neither failing nor blind: modified cell counts reflect an adaptive response of the host [see comments]. Nat Med 1997,3:486-490.
11. Hellerstein M, Hanley MB, Cesar D, et al. Directly measured kinetics of circulating T lymphocytes in normal and HIV-1-infected humans [see comments]. Nat Med 1999,5:83-89.

12. Hellerstein MK, Hoh RA, Hanley MB, et al. Subpopulations of long-lived and short-lived T cells in advanced HIV-1 infection. J Clin Invest 2003,112:956-966.
13. Silvestri G, Feinberg MB. Turnover of lymphocytes and conceptual paradigms in HIV infection. J Clin Invest 2003,112:821-824.