On Human Immunodeficiency Virus’s Potency to Freeze the Immune Response

By: GX8

This Paper explains why the immune system (either of human or animals) cannot eradicate the virus that has been labeled Human Immunodeficiency Virus (HIV) once a critical mass of it infects its host. In proving HIV’s machinery, This Paper also reveals how the T killer cell (also known as CD8, T8, and Cytotoxic Killer Cell, know as “Killer Cells” for This Paper) communicates with the “Helper Cells” (also known as CD4 and T4).

To date, there is no clear or comprehensive explanation as to how the virus works, or the communication system between the Killer and Helper Cells. A number of theories have been explained, but all fall short in explaining why the infected has a distorted growth population of the virus; why, after infection, the white blood cell concentrations decay and die, specifically helper white blood cells; and the odd relationship the Helper Cell population has in contrast with the increasing viral population; (See HIV Charts); and why the virus appears dormant for a period of time.

This Paper will prove that the virus kills its host by freezing the Helper Cells’ immune response, and hence, cripples the infected’s immune system to the point where a secondary infection, by virus, bacteria, or cancer, kills the person. As such, This Paper lays the groundwork in proposing a solution against the disease and vaccination of it.

Part I introduces This Paper. Part II presents a brief history and the theories that have developed to explain the uncanny nature of the virus. Part III outlines the undisputed facts of the virus. Part IV explains the HIV lifecycle. Part V explains the communication system between Helper and Killer cells within the immune system. Part VI discusses how HIV freezes the immune response. Part VII conclude This Paper.

II. A BRIEF HISTORY OF HIV’S EMERGENCE IN THE UNITED STATES

There are disputed accounts of when the HIV virus first appeared in human history. Let’s take the prevailing view.

On June 5, 1981, the Center for Disease Control and Prevention (“The CDC”) first identified five infected homosexual men, ranging from the age of 29 to 36, whom displayed symptoms of a type of pneumonia, which is usually seen in only those with compromised immune systems. Two of the patients died shortly after being admitted to the hospital. Id. A month later, similar patterns of infections were being seen in San Francisco and New York.
Over the next 18 months, patients, mainly from the states of California, Florida, New Jersey, New York, and Texas, were diagnosed with the same pneumonia that appeared in Los Angeles. Also seen in these patients was Kaposi's sarcoma, a type of tumor that usually infects those with weak immune systems.

The new disease was named “Gay-Related Immune Deficiency” or “GRID” by Michael Gottlieb, a UCLA researcher, and his four associates, at the May 1982 conference of the American Federal for Clinical Research in Washington, DC. The CDC proposed calling it 4-H, named after the type of people infected: homosexuals, hemophiliacs, heroin-users, and Haitians. In 1983, the disease was relabeled as Human Immunodeficiency Virus (HIV), and its mature stages of progression was called Acute Immunodeficiency Syndrome (AIDS).

According to the World Health Organization, since the virus’s genesis, to the date of drafting This Paper, HIV has been estimated to infect 71 million people, of which 34 million have died from the disease. As of the date of submitting This Paper, there is no known cure, which could eliminate it. There is also no clear theory as to why the immune system can’t eradicate it.

There are, however, three common theories proposed. The prevailing theory is the mutation theory. The mutation theory states that the virus is constantly mutating faster than the body’s immune system could react. A modern theory – the hiding theory - has also emerged. In this theory, HIV “hides” or escapes detection from the immune system. Another common theory is the denial theory – which states that HIV is not real. All are incorrect, but after understanding the sinister nature of the virus, it’s clear why they have varying degrees of appeal.

THE UNDISPUTED FACTS OF HIV

Before discussing the theories that explain HIV’s lethal nature, one first has to understand the undisputed facts of the virus. They are as follows:

1. HIV is a virus, meaning it has the traits that classify all viruses, which are: a) it has a protein shell, b) genetic material (either coded as DNA or RNA) and c) needs a host to replicate. In other words, HIV cannot reproduce, without a host.

2. Unlike other viruses, as a result of being infected with HIV, the host’s immune response, particularly T4 or (CD4) becomes impaired, decimating the body’s T Killer blood cell population.

3. HIV doesn’t kill the host directly, but a secondary illness, usually by an influenza-type virus or tumor, (which results, because of the impaired immune system), kills the host when the patient’s immune system is too crippled to fight off these secondary illness. This stage is called AIDS (which This Paper will no longer use in its terminology after this point.)

4. Once the virus infects the host, the immune system cannot eradicate the virus population.

5. HIV targets Helper Cells, a type of white blood cell, to parasitize them, in order to survive. (See FN10).

6. HIV can be transmitted by three fluids: blood, semen, and milk.

THE LIFECYCLE OF HIV (IN RELATION TO ITS HOST)

Unlike other viruses, HIV has four stages in the lifecycle of its host. The first stage is initial infection. The second stage, which is unique to HIV, is commonly called “latency” or dormancy, or “asymptomatic phase” but This Paper will call this stage, from this point forward, “The Slow Doom-Phase.” (The latter term becomes self-evident later in This Paper.) The third stage, commonly known as AIDS, sometimes referred to as “eventual end-stage”, is the “The Imminent Doom-Phase”. The fourth stage, and final stage, is that the host perishes. (See HIV Charts attached for a visual model.)

A viral infection usually ends in the host being able to eradicate the virus or the virus eradicating the host. Hence, the virus progress into a two or three-stage lifecycle.

Take the first scenario. The virus, one; infects its host, and two; is, then, eradicated.

Take the second scenario. The way it works is the virus: one; infects its host; two; populates beyond the host’s control (analogous to the HIV’s Imminent Doom-stage); and three; kills the host.
Thus, it’s the slow doom-phase that has perplexed mankind, about the nature of the HIV virus. How does HIV just linger in the host for so long? The question has stood as a Sphinx’s enigma, blocking mankind’s journey in the realm of medicine, which has resulted in her monstrous devouring of tens of millions of human lives. Now that the riddle can be answered, the beast can be vanquished.

THE COMMUNICATION SYSTEM BETWEEN HELPER AND KILLER CELLS IN RESPONSE TO VIRAL INFECTIONS

One cannot understand HIV, without first understanding the basics of how the body’s immune response reacts to a viral infection. Part of the problem with understanding this disease, is that the communication system between the Killer Cells and Helper Cells haven’t been well established. vi Because of the complexity of the discovery, This Paper uses analogies to simplify the point.

Not to over-employ analogies, but seeing that a viral infection is generally operating in an invisible world (as viruses cannot be seen without the use of an electronic microscope), an understanding of the relationship between the host and HIV cannot be understood without analogies. Also, as Einstein stated on his theory of relativity, one cannot understand phenomena without understanding the context and relationship in which these phenomena play out.

The first principle to know about a viral infection is that, at the point the infection occurs, the body is at war. The host intends to eradicate the invader-virus, and the invader-virus attempts to conquer the host. And as Sun Tze, the philosopher of war stated, “If you know the enemy and know yourself, you need not fear the result of a hundred battles. If you know yourself but not the enemy, for every victory gained you will also suffer a defeat. If you know neither the enemy nor yourself, you will succumb in every battle.” Hence, the immune feedback loop, which currently isn’t known, and the virus both need to be understood better to solve this problem.

Of the white blood cells, there are two types vii that are relevant to a viral infection, the Killer Cells and the Helper Cells. viii (Over the years, there appears to be much confusion as the label of these cells as well as their functions. They’ve been called helper cells, killer cells, CD4+, T4, CD8, and T8.)

Killer cells, (what This Paper, for purposes of understanding, will now call (“Patrol Cells”)), have a constant number in the blood stream, meaning, in general, at any given time, the body does not make more or less of them. (See HIV Charts – Figure 7.15, which shows before Eminent Doom, the body makes a consistent amount of Patrol Cells – which is CD8). Helper cells, (what This Paper, for purposes of understanding, will call (“Reinforcement Cells”)), in contrast, varies its numbers, depending on whether the body is invaded or not. This is why.

After the invader-virus enters the host, the Patrol Cell detects it ix, engages it, and kills it. (See How HIV Freezes the Immune Response –attached.) A Patrol Cell appears to be designed to confront any type of invader, but as such, logically, it must be energetically more expensive for the body to produce because Patrol Cells needs to have various weapons at its disposal. This is because different viruses require a different defense; so to be equipped to engage any kind, requires it to be multifunctional – like a Swiss Army Knife. (After the virus and the patrol cell die, two events must occur. See How HIV Freezes . . .).

First, the patrol cell, which has killed the invader-virus must send a signal x for Reinforcement Cells to arrive. Second, the patrol cell needs to send a signal for the host to produce a specific type of Reinforcement Cell, a message as to what weapon Reinforcement Cells need to be equipped with to combat the viral-invader. (The latter is required because once the body has the lock-and-key-weapon, specific to that virus, it can kill it faster – instead of spending time to figure out what is required upon each engagement.)

As a result, the host responds by releasing clones, replicas of the same type of Reinforcement Cells in mass to overwhelm the invaders. After the swarm of Reinforcement Cells enters the blood stream, it can quickly engage and eliminate the invading viruses. Nonetheless, for every virus it kills, the Reinforcement Cell also needs to send a message to the host’s communication center that a virus has been killed by it (which would be a distinctly different signal than the one the Patrol Cell sends); this is so, the body can shut off the production of these Reinforcement Cells. At this point, there’s a gradual decrease in the production of Reinforcement Cells, until, by receiving these various signals, the host registers that victory has been achieved. The body then stops manufacturing the Reinforcement Cells and returns to status quo.

VI. DISCUSSION: HOW HIV FREEZES THE IMMUNE RESPONSE
The problem with HIV, whether the body enters into the Slow-Doom-Phase or the Imminent Doom-Phase, is that Reinforcement Cells are being phased out. To start, like other viruses, HIV enters into an initial infection phase. Then, the body responds accordingly by producing the right type of Recruitment Cells. But before the eradication of the virus, the body lags in producing Recruitment Cells, when it shouldn’t, and eventually, it ceases production of them.

What’s special about HIV, however, is that at a certain point – (“The Doom’s Point” – see it marked on the drawing attachment), the body shuts off production of Recruitment Cells permanently, even when the virus is still present, although at a small concentration.

This Paper refers to it as the Doom’s Point because from that point forward, Recruitment Cells are ceasing to be produced to combat HIV or later viral infections. It’s at this point, that immune system is permanently crippled, and the host is doomed to die, whether it be a slow death or slower death.

HIV then begins to repopulate slowly and gradually, but consistently. This marks the Slow Doom-Phase. One can see now why it’s a slow doom, because once the host’s immune system passes The Doom’s Point – there can be no return to producing Recruitment Cells at the concentrations needed, and hence, fighting viral infections becomes permanently impaired.

And it’s a slow doom because, although the virus is replicating slowly, one’s fate, after passing that threshold, is certain: death. It’s only a matter of time, where HIV is present in such a high concentration, and Recruitment Cells in such a low concentration, that the odds of a secondary infection are certain and fatal.

But why does the host shut off production of the Recruitment Cells prematurely? Given the logic of the immune response, there could only be one possible answer.

While it parasitizes a cell (or when the HIV-parasitized cell dies), HIV is in the active business of sending the exact same message that the Recruitment Cell sends to tell the command center that the virus is being destroyed, when it actually is not. In other words, because of the signals HIV is releasing, the host is tricked into thinking that the host has eradicated the disease, when it has not. Hence, The Doom’s Point is the point where the body is flooded with such a signal, that it overwhelms the immune system permanently into believing that victory has been achieved when it has not. In other words, after the Doom’s Point, the body is frozen in a mode that believes that it has achieved full and complete victory of all current and future viral infections for the rest of the host’s remaining life.

As a result, even though there’s a constant production of Patrol Cells, there can never be enough of them to fight off the constantly growing HIV population or later infections. The ultimate result is an early death for the host.

Now, one can see why the three theories listed in the introduction hold some appeal but are wrong. First, the mutation theory is flawed because even though a RNA-based virus like HIV does mutate, the rate of mutation given the rate of mutations and the odds of achieving the correct combination to create a new type of protein coat would be slower than the rate of the immune system’s rate of eradicating the virus. In other words, the immune system would be able to eliminate the virus faster than it could mutate to escape detection from it. The logic itself also makes no sense, as a great number of viruses are also RNA based, and we don’t see the Slow Doom-Phase in these type of infections.

Second, the hiding theory is also wrong. Although the conclusion of it hiding from the immune system explains the ceasing of Recruitment Cells and the increasing growth of HIV’s viral load, it doesn’t explain how.

Finally, the HIV-denialism is based mainly off two points. The thesis of the theory goes like this: One, HIV infections fails Kock’s Theorem, which is a four point test designed to determine if one is infected with an organism. Two, there’s no explanation for the Imminent Doom-Phase. Therefore, HIV doesn’t exist.

Repeated, the argument goes: Because it’s not explainable, it doesn’t exist. This is illogical. One’s absence of explaining an event, doesn’t mean the event doesn’t exist. And regarding applying the HIV and its lifecycle to Kock’s theorem, it can be done, but it is not within the scope of this paper. Therefore, HIV-denialism is also incorrect.

VII. CONCLUSION

As discussed thoroughly in This Paper, HIV works by releasing a signal that freezes the host’s immune system from producing Helper Cells (or “Recruitment Cells”). As a result, the
host’s immune system is permanently crippled and vulnerable to secondary infections. This Paper clearly explains why the immune system cannot eradicate HIV, and in doing so, has dispelled the prevailing theories on HIV’s lifecycle. Based on this understanding, it is possible to treat the host in a manner that can eliminate the virus. It is also now possible to vaccinate against the disease.

END NOTES

i Upon an offer publication, the author will reveal her true identity, which she currently entrusts to her lawyer. Upon offer of publication, she offers to write another paper on the significance of critical mass and why a critical mass is required to trigger AIDS.

ii Upon an offer of publication, the author will release the experiment that proves that Patient L.C.’s model of the communication system is valid; though, she fully accepts that it may be proven to be much more complex than the simplified model she’s arrived at.


iv Karposi’s sarcoma and Pneumocystis pneumonia among homosexual men--New York City and California. MMWR 1981 Jul 3; 30(25):305-8


vii Even the term, Human Immunodeficiency Virus is misleading, HIV can infect more than just humans. But for the sake of simplicity, since that’s what the world knows it as, This Paper will adopt the term.


ix One reason that the virus’s potency hasn’t been understood is because the communication mechanism between the Killer cells and Helper cells hasn’t been discovered, until now.


xi AIDS is a confusing term, and has done more harm in our understanding of the lifecycle of HIV. The label AIDS makes it seem like the virus has mutated into a different virus, when really AIDS is describing the state of a person’s immune system in relation to the virus’ population growth.

In other words, AIDS describes the critical point of a host’s immune system, but the way it’s used, makes it seem like the virus has changed its character into another virus. HIV is HIV, even when it becomes “AIDS”; it’s just at a greater population, at the stage of AIDS, but nothing about it has changed physically.

xii Principles of Virology, 36-54.

xiii See Principles of Virology p. 219, which has the three stages, but doesn’t mention death, the fourth phase.

xiv Once again, all these terms are misleading. It’s not that the virus is dormant, sleeping, or latent. It’s that it’s replicating at a slower rather than viruses usually do. This Paper will stop using such terms.

xv Take a look at Principles of Virology, pp 98-132 principle of virology. This textbook has no mention of the communication system between the Killer Cells and Helper Cells.

xvi There may be more, but for the purposes of the model presented, only these two need to be discussed.

xvii Harvard Researchers say that the NK Killer cell can also play a role, but for the purposes of This Paper, the finding is out of scope. GX8 believes that research into this area can be potentially helpful in understanding the immune system.

xviii Or more specifically, detects the infected host cell, which it must now kill.
“This is a very overly simplified model, constructed for the purposes of explaining HIV. In reality, there could be thousands, if not tens of thousands, of biochemical reactions that facilitate this process, which I’m convinced that we, as a race, have barely scratched the surface of in the realm of science. I confess now, that this model is most likely not fully correct, and current and later discoveries in science will adjust and correct it with the passage of time. This assumes the human race doesn’t regress into a dark age.” – GX8.

Upon an offer of publication, the author will reveal himself and also reveal the peptide signal that the virus is most likely releasing in mass.

Must mutations of a virus, would result in a non-functioning virus – not a functioning, different one.

Upon publication, the author will release the experiment to prove this theory wrong.