BECOMING UNDETECTABLE IN THE CTHULUCENE

7:30 a.m. sees a regular flow into the clinic as nine to fivers rush in for blood collection. No one likes the tapping, probing, pricking, untying before the rush of crimson. But especially him, especially this time around. Others note the faint film of sweat on his upper lip, eyes narrowing in concentration. This is his second consecutive viral load test to check for viremia or the uncontrolled proliferation of HIV-1 viral particles in blood. According to WHO guidelines, a second test is required to recheck the threshold for “becoming detectable” at >1000 HIV-1 copies per milliliters of blood. For only one test with a spike in viral particles might just be an isolated blip. The thought sustains him: in fact, his blood might not yet be saturated with viral particles instead of those intelligent T-cells. Maybe this is not the tipping point that indicates viral resistance to the first line of ART (anti-retroviral therapies) that he has been on—seemingly for an eternity. He winces at this need for constant monitoring, this feeling cyborgian...the ongoing modification of his blood. An elderly patient drops a pile of magazines with a bang, rousing him. He glances around self-consciously to see if anyone had noticed the pursed lip and furrowed brow. He hopes he was undetectable.

Such scenes are familiar. There are 36.7 million people living with HIV at present. Not all have the luxury of growing unease in the cool of doctor’s offices. Large numbers of the HIV-affected live in resource-limited contexts where clinic visits require a trek, perhaps a day off work, and where clinics are without adequate storage and refrigeration facilities or indeed the electrical infrastructure necessary for freezing samples. Typically, in such contexts, pinpricks yielding dried blood spots (DBS) replace venous blood collection. Despite these differentials, the viral load test has been standardized as the global protocol for living with HIV. Administered at the clinical scale of the individual patient, the test is the first stop in a chain of operations that constitute treatment and care of HIV infection. If the first test for this virus, the ELISA-Western blot test introduced in 1985, measured for antibodies rather than viral particles (much like
the Wasserman test for syphilis introduced in 1905-6), then currently viral load tests that extract, probe, and magnify host blood in order to quantify HIV-1 RNA are the gold standard for monitoring disease progression. High viral loads indicate natural variations in HIV generation, either because of non-compliance to ARV regimens or growing HIV resistance to particular drugs. Such loads have predictive value for they assume the coming depletion of blood constituents such as the CD4 cells, which are invaluable lymphocytes and part of blood’s solid base. The viral count in test results represents a ratio within a specified volume of blood. Thus, while virological analysis conducted at molecular scales probes and identifies viral RNA, what is detected is a distribution of human and microbial matter.

This distributive logic points to the clinical goals of “living with HIV” as multispecies accommodation. Keeping viral counts low is sustainable practice: only a certain number of viral particles will allow the hematic system (the circulatory system for blood) to regenerate effectively. When blood is saturated with HIV-1 RNA, the survival of the host is in serious question. Upon my visit to the Retrovirus Lab at the University of Washington, a research site in my book project on epidemic media, one researcher remarked on a blood specimen so saturated with HIV-1 RNA that she thought the “blood could have walked on its own!” The offhand remark acknowledged the viral takeover of human blood, a metastasizing of the human-non-human assemblage that required immediate therapeutic intervention.

At first glance, the warning implicit in the viral load test is deeply anthropocentric. The test seeks to keep at bay microbial hordes despite our growing recognition that microbial cells weighing as little as 200 grams outnumber human cells
10 to 1. In the early 20th Century, the findings of the Human Microbiome Project exerted the same fascination as the Human Genome Project did in the late 20th Century.\textsuperscript{3} Planetary thought on living as multispecies questions how we guard the boundaries of the human “we.” The “new biology,” argues Rodney Dietert, suggests humans are multispecies “super-organisms” and not a single species at all.\textsuperscript{4} In this context, how are we to understand the intent of the viral load test that produces a mediatic microbial-human interface so as distinguish between “human” and “microbial” matter? What sense does it make to count viral particles when human and microbial matter are inseparable and molecularly interwoven into each other? These questions underscore the significance of blood as an elemental medium that is value-bearing because it enables multispecies regeneration. As vital fluid, blood moves within planetary circulatory systems; most often, that movement (especially when it traverses species boundaries) instigates anxiety, fear, even horror, in endless popular contagion narrations. Against this context, the viral load test mediatically transcribes ratios of human and viral matter that ensure the sustainability of both species, rather than the dominance of one.

Such an argument relies on directly addressing the common dread of parasitism. As Angela Douglas maintains in The Symbolic Habitat (2010), parasitism is an evolving biological partnership in which one partner, usually the host, takes control, imposing sanctions and controlling transmissions for the benefit of both partners, so that they might develop novel capacities (a lateral, not hereditary, transfer of properties) of survival.\textsuperscript{5} In the deep timescales of organismic evolution, “symbiosis-at-risk,” as in the case of a parasite that gives little to the host, is one step on the evolutionary ladder. In this regard, “managed HIV” of the post-antiretroviral (post-ARV) era is an instance of a
technologically engineered partitioning of resources that ensures the interdependence of two species—and indeed, the survival of both. As a condition of host blood, saturation is at once a threshold that *demarcates* host and parasite, and a phase change in irrevocably *entangled* ecological relations.

Parasitism with potentially deadly pathogens poses special difficulty to empathetic relations between species, an aspiration that dominates multispecies environmentalisms. Microbes are not large, charismatic animals; and aggressive parasites threaten social paradigms of kinship that underwrite the call to empathetic relations. The pathogenic parasite puts species survival on the table in no uncertain terms: the virus is that cthulu-like thing, as Donna Haraway theorizes it in *Staying with Trouble: Making Kin in the Chthulucene* (2016), that has always already been in the earth’s geological matrices. Its suddenly intensified actions mandate *artful* sympoiesis. As Anna Tsing and Haraway variously suggest, the artfulness of technological interventions is not “against nature,” but the necessary repair of biological, geological, and atmospheric damage. Amid blasted planetary ruins, even “the most promising oasis of natural plenty requires massive intervention.” The question is which natural and social disturbances can we live with?

Epidemics are planetary disturbances in which potential, even imminent, species extinctions call for technological interventions. The viral load test that establishes viral saturation at the scale of the individual patient is one among a series of mediatic “interface effects” that attempt to manage multispecies relations. Since blood—the target of epidemic intervention—*houses* the virus, I characterize this instance of living artfully with epidemics as multispecies “accommodation.” The viral load test quantifies HIV-1
RNA copies in a specified volume of blood. When the copies are >50 copies per milliliter of plasma, the detectable low levels are commonly understood to be “undetectable.” But >1000 copies per milliliter signal the condition of saturation as index of coming vital decline of the host. On the one hand, the quantification presents a snapshot of discrete viral matter against the negative space of blood implied in the distributive logic. On the other, the main concern is to align entangled human and viral temporalities, and this involves making legible viral natural variation and intensified generativity. Chronic blood surveillance establishes saturation as an anticipated condition that *must never arrive* and therein reestablishes the dynamic and unstable nature of ecological flux. As a line in the sand, saturation provides evidence of a change in the ecological organization of matter that sets in motion new interventions in the intensities, directionalities, and accelerations of this crisis event. Understood in this way, saturation frames the test as *epidemic media* engaged in slowing down, abating, and sometimes thwarting planetary disturbances. I follow the vicissitudes of “blood,” the feared epidemic medium of transmission, as it transitions from the patient’s vein to blood data. Those travels—from clinic to laboratory and back to clinic—illuminate “managed HIV” as a distributed creative experiment. As elemental media rendered readable for viral particles, changes in blood foreground the environmental dimensions of living with epidemics in the ehtulocene. Amid seemingly ordinary preoccupations with keeping humans alive, the virus surfaces as chthonic ancestor always already in the earth.

I. More than Human
From historical perspectives, the semi-permeable category of populations within a species is my starting point for thinking about saturation as a crisis-event. When significant populations are under threat of extinction because of the impact of microbial proliferation on host vital processes, we recognize the potentially catastrophic change as an epidemic. As a term hailing from the Greek *epidemia*—a condition against the *demos*—ostensibly the epidemic seems to go against the grain of living as multispecies. But if anything, forty years of the HIV/AIDS epidemic has simply reinforced living as multispecies as an inevitable condition—a bitter lesson with incalculable costs. “We” have learned to “live with” HIV after massive social trauma that shored up congeries of disposable humans, and that challenged the unitary notion of “the human” in universalizing discourses of planetary disturbance. That trauma is well documented, and, in certain parts of the world, still continuing. Responsible for 30 million deaths from AIDS and 36.7 million living with HIV worldwide since the first reported case in 1981, we have learned about the socioeconomic calculus that divides, segregates, and sorts a single species even as we press on with the urgent task of living as multispecies. Equally, we have understood both the possibilities and limits of scientific-technological achievements. After 1995, human hosts can live with HIV as multispecies *because of* the biomolecular modifications we call the anti-retrovirals (ARV). At present, the central global public health challenge to sustain drug adherence for those already on ART (Anti-Retroviral Therapies) and to ensure chronic blood surveillance at global scales. While the ART therapies are no doubt hard-won medical victories, “living with HIV” is possible because HIV takes about a year to achieve the scale of cellular entropy that Ebola accomplishes in ten days. In other words, the non-human agent defines the scope of
human actions. With the exception of variola, there is no other microbe in history that has motivated humans to search for the holy grail of a viable, if precarious, threshold for microbial-human relations. It is against the backdrop of this “long-wave”\textsuperscript{13} epidemic, that HIV emerges as the emblematic microbe for collective instruction on multispecies accommodation.

As a ratio of microbial distribution, saturation vis-à-vis the viral load test relies on scientific procedures of extracting, isolating, and reducing the thing to its core elements: in this case, the HIV-1 to its RNA particles. The drilling down to causative “agents” that are then targets of clinical-medical management defines virological expertise whose antecedents hark back to the late 19\textsuperscript{th} Century. The name virus comes from the Latin for poison or other noxious liquids. In its first appearance as a scientific object isolated for study, the virus was inseparable from the medium that carried it. The German agricultural chemist Adolf Mayer identified a “soluble, enzyme-like” sap that mottled tobacco leaves, and characterized the sap as a biochemical agent—seeping, leaking, and spreading into host plant populations. At first look, then, the virus saturated its host, a “phase change” in the organization of matter that microbiologists distinguished as host and microbe, human and non-human.\textsuperscript{14} A few years later, in 1892, the Russian botanist, Dmitri Ivanovski made the topological observation that a toxin caused a “wildfire,” a noxious “contagious living fluid” that Dutch botanist and microbiologist Martinus Beijerinck would name “virus” in 1898.\textsuperscript{15} And so was born the first virus, the tobacco mosaic virus (TMV), whose ability to cause economic ruin, as evidenced in the destruction of tobacco plants, motivated further research. Filtering the sap from diseased tobacco plants, Beijerinck found that he could infect other plants with the same fluid. Here, saturation as planetary
process was an unstoppable condition that virologists later came to understand as unrestrained microbial movement and replication. Immunologists tracked the flourishing of Beijerinck’s living contagious agent to assess the condition of the host, and to fathom host vulnerabilities and defenses.

The sense of a phase change in the host medium for parasitic replication haunts the earliest perceptions of virus-human relations: a liquid poison, a wildfire as evidence of a shift in ecological organization. But things would change with the maturation of germ theory that would spur the isolation and extraction of viruses as particular agents, and subsequently set in motion fine-grained analyses of their constituent elements. In 1876, Robert Koch proved specific microbes caused specific diseases, a proof enshrined in the four causative criteria. Now disease etiologies established linear causalities between microbe and host, cause and symptom. Ecological matter would be parsed as discrete entities: in the case of pathogenic viruses, microbial-human relations were increasingly recast in the antagonistic terms of eternal war. From then came the decades of imaging the virus and finding its code of life; later, the hunt for its planetary habitat, after the admission that humans had lost the “war on germs” that they had once seemed poised to win in the post-World War II era. In the waning years of the 20th century, the sudden resurgence of deadly viruses (Marburg, Hanta, Ebola, and HIV) pointed to viral emergences as complex multi-temporal planetary events. Microbial saturation of plant, animal, and human life came to be recognized as the condition of planetary disturbance spurred by human actions—everything from inroads into less-trodden forests and caves to the changing rainfall patterns and temperatures of climate change. Radical planetary disturbances were as much biological, geological, and atmospheric as they were social,
political and economic: hence, epidemics were multi-causal upheavals, and disease milieus were no longer territorially or demographically containable. HIV, for one, coexisted with animal populations in the Cameroon for a hundred years until the butchering of bush meat enabled it to hitch a ride on human cellular resources. This has become a familiar tale, as zoonotic viruses skipping species barriers periodically wreak havoc on new host populations.

Even as virology, immunology, and epidemiology—the three sciences of the virus—coordinated responses to epidemic emergencies, the nature of the virus as matter invoked vigorous debate. If, as Erwin Schrödinger suggested, living things were defined by their capacity for self-regeneration (to grow, repair, and reproduce), their fight against entropy, and their tendency toward a sustainable equilibrium, then was the virus dead or alive? Was it one of the first organisms (a pre-Luca cell) in a four billion-year primordial soup? Was it a relic with primitive RNA? A fugitive from the host genes, when did viruses degenerate into parasitic lifestyles? At these deep timescales, viruses appear as residues from a distant past in which they had replicated and saturated their hosts. They were objects of scrutiny not only for the biological sciences (evolutionary biology, microbiology, structural and molecular biology) but also for geologists in search of planetary geohistories. In hot sweltering caves, crystalline formations of viruses rested, dormant, on the lookout for new hosts.
Those resting places attract researchers invested in multispecies survival. Writing in *The Multispecies Salon* (2014), Eben Kirskey Nicholas Shapiro, and Maria Brodine foreground astrobiologist Penelope Boston’s research on radioactive landscapes in which microbes, including viruses, survive. Microbial communities trapped in hot and abyssal caves endure nuclear winters, waiting to reintroduce their banked genes at a later point in the Earth’s history. Microbial evolutionary grit surpasses that of humans; no wonder, microbes elicit admiration, even awe. Human cellular precipitates crumble before robust HIV broods; as the living dead, these broods live in the huge air bubbles entrapped in caves. They are always already there in the planetary geological matrix. Subterranean cthulu, they forge new multispecies relations when viable hosts come along—literally, when a bat, a primate, an insect, a human crosses their path. Much has been said about the microbial information sharing, a kind of “quorum sensing” that influences microbial group behavior. Much is known about how microbes detect densities of populations in an environment and coordinate their response. Not only is there intensified microbial movement, but also changes in viral informatic actions and chemical circuits. In the earliest discourses, this skip into a new host population whose resources allow viruses to multiply is when they “come alive.” The virus interferes in host processes of self-regeneration: HIV, for instance, inserts its genetic instructions for protein making into
human DNA and uses human cellular resources as fuel. Anthropogenic changes drive planetary disturbances: as crystalline caves melt and deforestation destroys habitats, chthonic “life” forms enter a new regenerative phase. They begin to multiply.

But because these are parasites, there is one drawback to the complete viral saturation of the host. It is not in the interest of the viruses to kill their host, but to enter biological partnerships based on partitioned resources. A bit of nucleic acid with a protein coat and without cell walls, the virus is an “obligate parasite”\(^\text{23}\) that relies on its host’s resources to multiply. Trouble arises when there is serious depletion of host resources—so much so that a whole host population might die, and with it, the opportunity for viral proliferation. So even if these microbes have the advantage of surviving in dormant states, in their “living” state, they survive sympoetically in the chthulucene.\(^\text{24}\) Thus technological interventions predicated on “living with” viruses are mutually beneficial for host and pathogen. The viral load test that monitors human and viral distribution of matter modifies the impact of disturbances for both species.

Central to such interventions are the mediatic interfaces that facilitate technological soft controls. As we shall see, these interfaces numerically index precarious plateaux in microbial and host distributions after which one species will no longer survive. In this regard, the regular viral load test is in the business of reorganizing \textit{species temporalities} at molecular scales. How long will it take for host blood to exhaust its regenerative capacities? How long can viral particles hide out in reservoirs that current platforms cannot detect? When will the reorganization of each species occur so that one will become residual, even extinct, and the other, stilled in its generation? Saturation is the limit condition: always virtual, always coming. It calls for the creative, and sometimes
experimental, interventions we might call epidemic mediation. In the long view, epidemic mediations of biological processes are *environmental media* as artful living. As such, these mediations must be studied beyond the narrow purview of bi media studies.\(^{25}\)

Epidemic media enable multispecies accommodation through regulating and controlling viral proliferation in a single host and through restraining accelerated viral kinesthesia across the host population. They galvanize the sympoetic arts of living in the chthulucene.

II. The Viral Load Test

Mediation is at the core of virus-human relations because viruses only come into view through technology. At 100-500 times smaller than bacteria, viruses were famously filterable agents that passed through Louis Pasteur’s Chamberland filters; as visual object, the first virus appeared under the electron microscope only in 1938. The first decade of research on the virus focused on its morphologies and taxonomies. If the virus-human co-emergence is a technological one, new media only enhanced the capacities to penetrate deeper into the molecular substrates of that co-mingling.\(^{26}\) By the mid 20\(^{th}\) Century, new alliances between physicists, chemists, biologists, and engineers ensured virus-human relations were understood as primarily bioinformatic ones. The reduction of the thing to its molecular core opened the doors to the manipulation of the code of life; the rest is history. Within this history of science, the viral load test mediates HIV-1 RNA copies to interpret the condition of the host. As such, it renders blood, which is one of the HIV/AIDS epidemic’s medium of transmission, readable through the extraction, distillation, classification, and quantification of specific biomolecules. While in common
clinical parlance the viral load test appears as singular, there are a series of tests that establish the vectors of infection. Rapid response tests can establish the presence of viral particles, but it takes more complex virologic assays or methods to actually count them.

Since body fluids have played a critical role in the HIV/AIDS epidemics, it is abundantly clear that they exceed the boundaries of individual molar bodies: they are planetary vital circulations. In this regard, as one of the “old, limbic fluids” in John Durham Peters’ description, blood is elemental media. Hence, its potentially uncontained circulation has been a concern since in the early years of the epidemic, when alarm over contaminated blood supplies rocked the medical establishment. More than semen, saliva, vaginal and rectal fluids, blood is the value-bearing fluid. For blood banks, plasma, which is 55% of blood, is a resource for species survival. So, too, in the testing procedures for HIV infection: blood is the fluid that is singled out, extracted from the molar “patient” at clinical points of care, then tagged for processing and transported to medical laboratories. The majority of commercially available instruments (Abbott, Biocentric, and bioMérieux) for nucleic acid based virologic assays require cold chain of transport for liquid plasma, which mandates requisite storage and refrigeration facilities. Since this is not always possible in resource-limited settings, and especially at district-level rural labs and clinics, blood collection protocols vary: for instance, some clinics prepare dried blood samples (DBS) that maintain non-infectious and stable viral particles in ambient conditions for as long as eight weeks on collection cards (Fig.2) while others collect blood in finger-stick micro-tubes. Whatever the collection format, blood specimens arrive at medical laboratories tagged with a protocol that specify the methods of study for grouped specimens.
During my visit at a laboratory medicine facility at the University of Washington, one of the core facilities of the Center for AIDS Research (CFAR),\textsuperscript{30} I observed the many stages of extraction that made it possible to report the ratio of HIV RNA-1 to a milliliter of plasma. Even before the virologic assays or methods that biophysically count amplified viral particles, technicians enter the arriving specimens into informatic infrastructures. The specimens are catalogued in the Laboratory Data Management System (LDMS), which is a state-of-the-art widely shared data management system that locates where the specimen is at any given moment and how it will be processed. Three methods or virologic assays are followed for counting viral particles: Reverse Transcription-Polymerase Chain Reaction (RT-PCR), branched-chain DNA (bDNA), and, occasionally, the Nucleic Acid Sequence-Based Amplification (NASBA). The variation in procedures in each virologic assay determines differences in the precision, levels of detection, and the calculation of linear range. The last difference is particularly salient to defining a threshold of harm to the host. For researchers, a linear range in the number of viral copies is preferable to a single number, which is now commonly understood as $>50$ copies/ml blood equals undetectable. After all, viral particles can fluctuate according to natural variation, the patient’s health, including co-infections, and adherence regimens. Therefore, a single numeric threshold is rarely a dependable in assessing increases or
decreases in viral loads. In fact, neither the $>50$ copies/ml blood as safety net, nor the numeric threshold of $>1000$ copies/ml of blood as indicator of viral saturation is set in stone. We know this from the history of debates over the threshold. Originally, the global standard was to test for a persistent load of $>5000$/ml of blood as the index of virological failure. That failure could be attributed to many factors, including viral resistance to specific drugs that produce mutations of HIV-1 RNA. Over time, the $>5000$/ml was considered too high because, at that level, the virus had already wreaked considerable damage on the patient’s immune system. Hence, the clinical threshold for estimating viral saturation was revised to $>1000$/ml of blood—it was high enough to avoid false alarms, which might compel a too hasty a switch in drugs, but low enough to surmise viral generation was on the rise. The variation in assay capabilities and the changes in numbers suggests that viral saturation cannot be understood as a stable threshold; it is at best a reasoned estimate of an anticipated phase in the biological flux of microbial-human relations. Those relations become standardized as distributions of viral and human matter through the mediatic interfaces of viral load processing.

Within histories of science, the force of testing technologies lies in all the ways in which test protocols streamline complex conditions into coherent “disease entities.” This is Ludwig Fleck’s point about syphilis, which was a multi-symptom syndrome widely regarded as the modern plague of the 19th Century. Fleck’s 1935 *The Genesis and Development of Scientific Fact* (treatise which precedes Thomas Kuhn’s 1962 *Structure of Scientific Revolutions* but was not widely circulated until 1976) presents the thoroughfare between disease concepts and evidence that mutually define each other and that regularly marginalize—keep secret, unseen, inadmissible, or exceptional—whatever
appears to contradict or overly complicate the standardized definitions. Tracing the
history of syphilis as disease entity, Fleck notes the slippages around the concept ever
since its first emergence in the 15th Century, when syphilis’ causes and treatments were
part mystical-ethical (resulting from the movement of stars or from carnal excess) and
part empirical-therapeutic (treatable with mercury). The debate on whether or not one
could characterize the syndrome that included sores, dementia, and progressive paralysis
as a disease entity raged through centuries before germ theory established a single
causative agent, *spirochaeta pallida*. Fleck’s greater point is to suggest what appears as
“scientific fact” emerges from protracted negotiations between multiple expert and non-
expert “thought communities.” Even individual scientists who focalize disease entities as
objects of science write as members of particular thought communities. Hence,
conceptual creations such as “becoming undetectable” become acceptable only through
their social consolidation. Fleck’s reflections on syphilis have considerable implications
for HIV/AIDS epidemics as both a chronic clinical condition and planetary crisis event.
In this context, what role did disparate thought communities play in defining “managed
HIV”? How do these disparate communities continue to manage blood now readable as
data?

The historical role that multiple experts and non-experts have played in the early
decades of the HIV/AIDS epidemics is well documented, so I won’t rehearse those
crenerable histories here. As patient-centered movements prevailed upon scientific
protocols for generating blood data, they were able to enact change in the then-emergent
biomedical infrastructures that now adjudicate distances between basic laboratory
research and clinical points of care—the bench and the bedside. I will turn to the
biomedical infrastructure shortly, but here, my point is to highlight the role of patient-centered “thought communities” in what is now “managed HIV” at global scales.\textsuperscript{32} Most prominently, the ACT-UP insistence on the inclusion of “dirty data” was a historic event: they demanded “parallel trials” for patients on medication for cytomegalovirus, patients whose blood was “dirty” because of co-infections. At the clinical scale of patient groups, they drew attention to human blood as multispecies, as the mantra “we are all living with HIV” suggested, an implication that the Human Microbiome Project now proves as scientific fact. The subsequent confrontations effectively changed FDA policies (the 1988 amendments) and established new relations between patient groups and medical institutions.\textsuperscript{33} Against this backdrop, alliances between scientists and clinicians, policy-makers and representatives of governments, social scientists and health industry workers, patient groups and journalists routinely negotiate “our growing capacities to control, manage, engineer, reshape, and modulate the very vital capacities of human beings as living creatures that proliferate, evaporate, or find institution.”\textsuperscript{34}

This short history pertains to the complexity of “becoming undetectable” as a global project. The expanding biomedical enterprise has effectively standardized a numeric value \textit{as} the horizon of health. Indeed, such standardization is easier because of self-regulating, self-quantifying consumer-patients who stream vital data on their mobile devices. The global rollout of HIV self-testing kits (the Rapid Response Test) is but a matter of time; a test that directs people toward clinical self-assessments. Of course, the story of self-quant thought communities that agree to chronic self-surveillance and to medical protocols is more complex than following instructions on a package.\textsuperscript{35} My point is that the techno-services offering self-quantification only amplify public consciousness
of blooming viral particles against the quiet volumetric backdrop of blood. Blood transforms from bodily activities that have planetary impacts to an alienable resource for clinical research and becomes readable data for biomedical intervention. But the perception of singular bodily activity returns in different guises at clinical points of care dispersed across the world. There, becoming detectable continues as a distributed creative experiment in multispecies accommodation.

III. The Labor of the Undetectable

One of the main drivers of managed HIV as a private chronic disease is an expansive biomedical infrastructure of the phenomenon we characterize as biomedicalization. Biomedicalization refers to a historical transformation in American medicine since 1985, when dramatic changes in science and technology spurred new controls over medical phenomena such as diseases, illnesses, injuries, and bodily malfunctions. American medicine became more dependent on the biological sciences and new technologies, including informatics. Central to this shift was an emphasis on the molecular basis of life, and the perception that biological substrates could not only be controlled but also enhanced and modified. Increasingly managed at molecular scale—the tweaking of enzymes or thwarting RNA transcription in managing HIV—a host of diseases became chronic conditions. Panoplies of biomedical institutions translated techno-scientific innovations fostered in the controlled conditions of research laboratories into clinical situations where they could be tested, recalibrated, and implemented at demographic scales. To a large extent, expanding infrastructural connectivity made possible the interface between research and clinical institutions: computerization and data banking
had everything to do with this transformation. Scholars such as Catherine Walby and Melinda Cooper concerned with increasingly valorization of the biomedical enterprise over the therapeutic benefits to participants on whose biological labor the enterprise depends find the extractive logic of this economy troubling. Stem cells, tissues, organs, blood, and oocytes circulate as *in vitro* resources disconnected from *in vivo* production; the “patient” re-enters the chain as the research subject of biomedical development in clinical trials, and as demographic aggregates in the large-scale implementation of new biomedical compounds. As a bodily contribution, blood enters the chain as an “already available resource” ready for harvest. The professional division of labor that Walby and Cooper track in *Clinical Labor* keeps the domains of research laboratories and clinics separate and places highest value on the cognitive labor of the scientist as technical expert.

The viral load test is part of the new biopolitical economy that accompanies the global management of HIV. Blood extraction and storage is standardized and those standards necessarily create global benchmarks. The contingencies of dispersed clinical points of care are at a remove from the formalized blood files that enter processing laboratories, biorepositories, and databases for clinical research. The *in vivo* labor of living with HIV virtually disappears. In contrast, it is at the clinic that the unfolding dynamic flux of life returns. Blood is not readable only for viral particles, but often for indicators of co-infection evident in therapeutic situations. The body’s plasticity returns at micro-scales to trouble standardized protocols of care. I do not seek to romanticize the clinic or to put too strong an emphasis on the distance between laboratory and clinic. What is at stake is a paradigm of biomedical development in which lay expertise in
informal clinical settings is seen as a target for incorporation into global public health regimes and not as differentiated points of creative epidemic intervention. At clinical points of care, doctors, nurses, and health counselors struggle to reconcile singular patient needs with the macro imperatives of viral load tests. Everything from nutritional change to water scarcities is relevant to the blood picture that is translated back to the patient. There, the distributed labors of “becoming undetectable” eclipsed in the emphasis on bio-value production in research laboratories become readily evident. There, the mediatic capture of “changes in blood” is expressive in singular bodies. A series of actions that technologically modify blood come into view: the host’s daily in vivo biochemical activities; the technician, doctor, and nurse’s therapeutic activities, including the collection and translation of blood; and the care of friends, lovers, and family members who ensure testing protocols are followed. These microbial and human energy exchanges make possible the distributions necessary for both species to survive.

As a “disease entity,” then, HIV emerges at several interfaces between thought communities. Medical ethnographers have made this point elsewhere, with reference to a range of chronic conditions. In recent times, Annemarie Mol’s The Body Multiple: Ontology in Medical Practice (2002) is one notable instance that has won critical acclaim. A study of atherosclerosis through interviews with medical practitioners (namely, radiologists and surgeons) and patients, Mol focuses on the events that people report on, rather than ask what people think of a particular disease that is already medically defined. What she finds are practical enactments of atherosclerosis: for the pathologist, the disease comes into view as a cross-section of an artery under a microscope, for the patient, the illness is the pain one feels climbing the stairs. Cataloging
this series of material events, Mol alerts us to many fragments that “hang together” as the body multiple. In the context of HIV, a number of scholars such as Marsha Rosengarten (2009) and Cindy Patton (in her study of HIV Metabolic Disorders) show how the over-emphasis on viral quantification can have discordant therapeutic effects: neglect of other bodily events (recorded as metabolic disorders), for instance, can trigger ARV non-compliance. Hence, therapeutic perspectives record HIV infection as the multiple events that patients, doctors, nurses, caregivers, and counselors encounter at clinical points of care.” Importantly, it is not only medical ethnographies that emphasize the value of informal records of illness open to shifting bodily events. Medical humanities, too, attend to such records in singular patient histories. In such records, lay expertise emerges as a core scientific knowledge-domain in epidemic intervention. Thinking across scales, as HIV and humans co-emerge, the in vivo labor of becoming undetectable becomes living as multispecies.

Saturation enables us to understand phase changes in the ecological organization of matter. In this chapter, anticipating the possible extinction of one species, the host, and consequently, the stilled generation of the other, the parasite, saturation is a threshold event in species temporalities. Media technologies such as the viral load test render that threshold readable in an ongoing struggle to slow down radical, irrevocable change. These technologies are part and parcel of massive global biomedical infrastructures that streamline the acquisition, processing, storage, and retrieval of blood; standardizing procedures, protocols, and methods across laboratories, such infrastructures make vital planetary circulations legible as data. In the consequent abstraction, the crisis-event of the epidemic becomes eminently manageable and the uncertainties of ecological flux fade.
“Managed HIV” is now the grand accomplishment of this formidable biomedical behemoth. Yet, as I have suggested, what such valuation produces is the erasure of clinical labor at dispersed points of clinical care—doctor’s office to home—without which neither tests nor drugs would settle blood. Quotidian exertions of stemming saturation ensue at clinical microscales: in modest environs, living with saturation becomes creative practice. What better way than to close with the artistic eye that records the ongoing toil of staying undetectable?

* * *

Fig. 3 Robert Sherer, “Love Nest,” 2005

A memory of bloodwork that marks the primal scene of the HIV/AIDS epidemic: the blood paintings of U.S. Southern artist Robert Sherer archiving HIV+ and HIV- blood as the collective record of living with HIV. At the time, he was attending the Atlanta College of Art, after a biology degree from the University of Alabama. It was the early days of the HIV/AIDS epidemic. In 1998, the ARVs were just out. Sherer’s fellow artists were dropping like flies, and blood had attained symbolic status as the mode of transmission for HIV. Exhortations to “get tested” had become commonplace in American public life as had biomedical knowledge of spiraling viral copies and diminishing T-cell counts. Artists drew attention to the processes of biomedicalization that targeted high-risk groups: they doused audiences in blood, they made paintings and installations with HIV+ blood. Gazing at the bright spurt from his artery that splattered his paintings, Sherer could not turn away. Emptying the ink from his quill pens, he began to paint in blood. Trained in botanical illustration, he painted “nature” in its bucolic innocence, its delicacies. He thinned his blood with anti-coagulants and mixed it with inks to increase its brightness. Soon an HIV+ friend donated her blood for a painting; shortly thereafter, Sherer’s refrigerator was stacked with donations. As he framed each blood portrait in Victorian oval frames, the feared medium became collectible art.

One among his early pieces stands out as a reflection on managed HIV: the portrait of two nestling bunnies, one painted in HIV+ and HIV- blood, respectively. Titled “Love
Nest,” the painting drew attention to the opacity of blood on surface appearances. Blood as ontological medium was incomprehensible; it had to be extracted, classified, and translated into data to become readable. Sherer’s bunnies were a response to the emergent molecular profiling of blood. He challenged viewers of “Love Nest” to slip into social profiling without technical mediation. When I interviewed him for my book on epidemic media, he said, rather wryly, that several viewers missed the point of the painting. They insisted they could differentiate the HIV+ from the HIV- bunny! They missed Sherer’s portrayal of sero-discordance as a natural state, a “living with” viruses and with each other. An early portrait, love is multispecies accommodation: the possibility of living with chthulu, but always undetectable.
Notes


2 For one comprehensive database of mutations to particular ARV therapies, see the Stanford University’s HIV Drug resistance Database (https://hivdb.stanford.edu/)


5 Departing from de Bray’s formulation that parasitism should be included within the definition of symbiosis, Douglas and her contemporaries argue that, to be considered symbiotic, organismic relations should be mutually beneficial to the participants for the major duration their lifetime. This does not mean that parasitism is not symbiotic, but that pathogenic parasitism is not—especially swift and deadly virulence (Ebola behavior, for instance) that leaves no time for the first step of symbiosis, the amelioration of virulence, to commence (Douglas 29). Less virulent parasites are at a selective advantage, in this regard, since they do not deplete the resources of the host. In her latest work on symbiosis (The Symbiotic Habit, Princeton UP, 2010), Angela Douglas returns to the persistence of this behavior among organisms in light of new thought on the microbiota crucial to immune function and the pragmatic promotion of symbiosis (reintroducing indigenous plant species in an effort to defragment habitats) as bulwark against deleterious
anthropogenic effects. Following *Symbiotic Interaction* (1994) and *The Biology of Symbiosis* (1987), the recent book ventures into the role of human ecological and medical interventions in the processes of symbioses, and, for our purposes, includes a reevaluation of certain organisms originally considered pathogenic as potentially symbiotic in the evolutionary future (Douglas, 2010, 8).


8 Here, I follow Alexander Galloway’s definition of the interface or threshold as a boundary that is posed as the limit of a system, or the point when the system becomes unworkable (Polity, 2012).

9 Etymologically, the “crisis” hails from the Greek *krinô* meaning to decide or to judge, and soon it came to mean a turning point that called for definitive action. In its migration into the Hippocratic school and therein into medical parlance, “crisis” came to mean turning point in a disease—a critical phase with high stakes.


11 There are many accounts of “first sightings,” some moving as far back as 1959 (cases now disproven by David Ho); the first case in the U.S. was Robert R., who died in 1969. Usually, early cases are the pre-1981 cases (1981 is when AIDS became known to the medical profession). See, Mann, Jonathan M. (1989) “AIDS: A worldwide pandemic,” *Current topics in AIDS* (volume 2) edited by Gottlieb M.S., Jeffries D.J., Mildvan D., Pinching, A.J., Quinn T.C., John Wiley & Sons (for the conventional HIV/AIDS

12 The first trials for AZT (approved in 1987) begin in the mid-eighties, but it is not until late 1996-early 1996 that the combination therapies including protease inhibitors enter the market in resource-rich contexts.

13 Long-wave epidemics are long-wave events with waves of spread and waves of impact. For a brief elucidation of the long wave in HIV/AIDS infection, see Alan Whiteside, *HIV/AIDS: A Very Short Introduction* (Oxford UP, 2008) 4-6

14 I’m indebted to Janet Walker’s characterization of saturation as phase change during a workshop held at UC Santa Barbara (organized by the editors of this volume), May 2017.


16 What became known as the Koch postulates (of 1890) was the refined version of four criteria for establishing disease causality formulated by Friedrich Loeffler and Robert Koch in 1884: (i) finding the causative microorganism in abundance in the diseased organism (and not a healthy one); (ii) extracting and growing the causative agent in a pure culture; (iii) when reintroduced into a healthy host, it should cause disease; (iv) and finally, it should be re-isolated and compared to the original.

17 While Alexander Fleming discovered penicillin in 1928, scientists took on its mass manufacture during World War II; hence the “war on germs” is inextricably linked to the war effort. Melinda Cooper writes about the euphoric sense following the discovery of penicillin in 1945 that was dampened with the arrival of aggressive viruses in the late

18 “The source of HIV-1 group M, the main form of AIDS virus infecting humans, has been traced to a virus infecting the central subspecies of chimpanzees, *P. t. troglodytes*, in a remote area in the southeast corner of Cameroon. The likeliest route of chimpanzee-to-human transmission would have been through exposure to infected blood and body fluids during the butchery of bushmeat. The early diversification of group M appears to have occurred some 700 km further south, in Kinshasa (then called Leopoldville), in the early years of the twentieth century.” See, Paul Sharp and Beatrice H. Hahn, “The evolution of HIV-1 and AIDS,” *Philosophical Transactions of the Royal Society B* 365.1552 (Aug 27, 2010): 2487–2494.

19 LUCA is the “last universal cell ancestor,” a pre-DNA cellular form.

20 The debate continues well into the 21stC: see, for instance, Luis P. Villarreal “Are viruses alive?” *Scientific American*, August, 2008

21 Once, Penelope Boston (head of NASA's Astrobiology Institute) strapped on an ice-filled backpack to survive a visit to Mexico’s hellish Cave of Crystals, where gas leaking up from deep magma chambers heats the air to 118°F with over 80 percent humidity. She captured this image of a red wall in a cave with butterfly crystal; scientists surmise the life trapped in crystals that could be 50,000 years old. The bizarre and ancient microbes were found dormant in caves in Naica, Mexico, and were able to exist by living on minerals such as iron and manganese. See, https://phys.org/news/2017-02-biologists-weird-cave-life-years.html#jCp (retrieved July 19, 2017)

An obligate parasite is an organism that cannot live without a host (that is, it cannot process all the cellular components it needs to regenerate itself) as opposed to a facultative parasite that can live independently, but becomes parasite under certain conditions. Unless the obligate “jumps,” it is only ambiguously alive.

Living organisms are teleodynamic in their regenerative actions (as the notion of autopoesis suggests), but this does not mean they only regenerate themselves. They can, and often do, do not regenerate together: thus, for Haraway, autopoesis and sympoesis exist in the productive tension of living as multispecies. Put differently, “sympoesis enfolds autopoesis and generatively unfurls and extends it” (60).

Biomaedia studies largely focus on the traffic between information and flesh, first inaugurated in Eugene Thacker’s *Biomaedia* (U Minnesta P, 2004), and the capacity to rewrite the code of life. Beyond the study of genetic codes, scholars such as Jussi Parikka (*MediaNatures, 2010*) and Manuel de Landa (*The Philosophy of Simulation, 2011*) have emphasized the broader ecological implications of the biological-informational thoroughfare.

Media histories provide accounts of the technologies involved: how the electron microscope’s shorter wavelength made it capable of higher resolutions of submicroscopic particles; how the charged electron beam probed and excited the positive and negative charges of histological dyes; how researchers moved around the needle-like particles of the first virus to be (the tobacco mosaic virus) to be imaged. The credit goes to Ernst Ruska who got the 1986 Nobel Prize for his work in electron optics.

I take my cue from John Durham Peters’ rethinking of the “media concept” in *Marvelous Clouds* (2014) in context of the “enabling environments” for diverse forms
of life. Hearkening back to conceptions preceding the 19thC preoccupation with media as the conveyance of human signals, Peters focuses on non-human signals, machinic or animal, that should fall in the purview of media studies. Media are ensembles of the human, machinic, and animal, and it is in this spirit the oceans and the atmosphere become elemental repositories of readable data and processes. In his magnum opus, Peters tangentially references bodily fluids as those substances that, like gels and agar, sustain and enable existence. But discussions of these fluids are folded into analyses of the body as a medium.

Blood under threat highlights the centrality of the body fluid to the maintenance of human life; this blood is a resource extracted and stored as plasma (55% of blood content) in global blood banks.

The quality certification of diagnostic products in resource-poor settings is currently not well regulated, so the U.S. Food and Drug Administration approval or European Union CE marking are often used as a surrogate for quality assurance of tests, even though these products may not be suitable for those settings. Undetectable reports a WHO laboratory program for the prequalification of products specifically suited to resource-limited settings began in 2008. Because the WHO process is so thorough, only 11 products have been approved so far (22). See also, World Health Organization: Medical device regulations 2003, 24: 1–43

The Centers for AIDS Research (CFAR) system coordinate HIV/AIDS research. Built to support academic and research institutions committed to reducing the global “burden of HIV,” as the mission statement states, CFAR first was launched by The National Institute for Allergy and Infectious Diseases (NIAIDS) in 1988 and later expanded to 19
“core facilities” co-funded by 11 NIH institutions. Each core facility agglomerates expertise, resources, and services, and as such, they exemplify the new kind of techno-services available for biomedical research.


32 Of course, patient-centered movements involving physicians, patients, clinics have a time-honored history long the late 20thC. Yet the civil rights, feminist, and environmental struggles of the 1970s and the HIV/AIDS activism of the 1980s effectively buoyed claims on medical self-governance in no uncertain terms. The spectacularly theatrical dimension to HIV/AIDS activism made biological citizenship a matter of national concern in national and international contexts.

33 Not only did the FDA respond to demands for information access to ongoing clinical trials, but the FDA agreed to conduct trials on whatever drugs the patients were experimenting with. The ensuing 1989 trials established protocols for Phase 0 trials that check results before the completion of the designed trials (the traditional Phase 1). See, Melinda Cooper and Catherine Waldby, Clinical Labor: Tissues, Donors, and Research Subjects in the Global Bioeconomy (Duke UP, 2014) 203.

The earliest self-quant community, the Quantified Self started in 2008 has meetups in 119 cities and 38 countries. The mantra is n-of-1 (number of cases is oneself) as self-experimentation. Sometimes their distributed experimentations run parallel to regulatory institutions: biosensor technologies such as the NightScout, for instance, which includes a DIY smart screen for continuous tracking of blood sugar still awaits FDA approvals as reliable blood surveillance, but online self-quant communities have developed mesoscale literacies about testing protocols and result interpretations (#WeAreNotWaiting). Gina Neff and Dawn Nauf, *Self-Tracking* (MIT Press, 2016)


Cooper and Waldby trace stem cell industries, tissue/organ exchanges, clinical trials, and gestational surrogacy as new modes of clinical labor in a post-Fordist flexible economy.

Cooper and Waldby, 9


One of the most famous examples is Ron Athey’s performance pieces using blood (see, Jennifer Doyle on Athey, *Hold It Against Me: Difficulty and Emotion in Contemporary Art* (Duke UP, 2013)).