EBOLA & HUMAN VIROME SIGNALING

A Systems Engineering Framework for Tonic Modulation, Myeloid Hijack, and Bioinformatic Signal Amplification

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1. Executive Summary & The Signal Amplification Paradigm

Conventional epidemiology and therapeutic design frameworks operate predominantly on a paradigm of direct physical elimination: *Destroy the viral pathogen first*. While intuitive, this reactive approach introduces structural information lags and vulnerabilities, particularly when confronting highly lethal, fast-replicating agents such as the Ebola virus. In the critical window between initial cellular entry and exponential viral expansion, centralized clinical pipelines—relying on traditional symptomatology, antibody assays, or localized PCR confirmation—frequently fail to achieve diagnostic or containment velocity.

This treatise establishes an alternative architectural model rooted in the principles of **Signal Amplification**. Instead of treating biodefense as a localized eradication challenge, a systems engineering methodology shifts the structural objective to: *Maximize the velocity, fidelity, and distribution of systemic threat reporting and cellular defense mobilization prior to pathogenic breakout*.

By mapping the human body not as a passive container of infection, but as a complex communication network featuring a running "carrier wave"—the human virome—and living, responding biological transceivers—white blood cells—we can decode the earliest sub-clinical signatures of pathology. This document details the exact biological, bioinformatic, and mathematical structures required to implement a real-time Distributed Immunity Architecture.

2. The Architecture of the Human Virome

The human body is host to a vast, complex, non-pathogenic viral ecosystem known collectively as the human virome. Far from being a random collection of transient passengers or parasitic entities, the resident virome constitutes a highly structured, ecologically balanced network that interfaces directly with host physiology. This ecosystem is composed of three principal taxonomic and structural factions, each occupying distinct niches and serving specialized communication roles.

A. Eukaryotic Commensals

The dominant persistent faction within human blood, tissue, and plasma consists of non-pathogenic eukaryotic viruses, most notably from the family *Anelloviridae*, specifically **Torque Teno Viruses (TTVs)**. TTVs are nearly ubiquitous across global human populations, establishing lifelong, non-symptomatic, chronic replication cycles within host cells. Because they replicate continuously at an ultra-low baseline without inducing cytotoxicity or classical inflammatory cascades, they act as a stable, persistent baseline signal within the host's circulatory and lymphatic networks.

B. Mucosal and Gut Bacteriophages

The most numerically abundant components of the virome are bacteriophages (viruses that exclusively infect and lyse bacterial hosts), concentrated primarily within the gastrointestinal tract and mucosal interfaces. While phages do not directly infect human cells, their systemic signaling impact is profound. Through continuous lytic cycles within the commensal microbiome, bacteriophages release structural bacterial components, including fragments of bacterial DNA, RNA, and cell-wall elements, directly onto host epithelial barriers. This constant shedding acts as an indirect, cross-domain signaling vector that perpetually engages host immune surveillance hardware.

C. Endogenous Retroviruses (ERVs)

Woven directly into the architecture of the human genome itself are ancient viral remnants known as Human Endogenous Retroviruses (HERVs), comprising roughly 8% of total human genetic material. While evolutionary pressure has mutated or silenced these elements to prevent the production of infectious virions, human cells continuously transcribe specific HERV open reading frames into non-coding RNA structures. These intracellular, self-generated viral RNA sequences occupy the cytoplasm and endosomal compartments of healthy tissues, acting as an internal, genomic configuration of background viral signaling.

3. The Unifying Dialect: Tonic Interferon Signaling

The human immune network does not exist in a passive binary state of "off" or "on." To maintain operational readiness and minimize response latency, the system relies on continuous input from its resident virome to calibrate its structural sensitivity. This background conversation is mediated through specialized host receptors that continuously translate the diverse physical elements of the virome into a singular, unified biochemical dialect.

Pattern Recognition Receptors (PRRs) as Decoders

Whether a viral component originates from an actively transcribing genomic ERV, an absorbed gut phage, or a circulating plasma Anellovirus, the host intercepts and categorizes it using a standardized array of Pattern Recognition Receptors (PRRs). These cellular sensors are strategically distributed across different cellular domains to map the entirety of the host's internal and external interfaces:

- **Toll-Like Receptors 3, 7, and 8 (TLR3, TLR7, TLR8):** Located within the endosomes (cellular recycling compartments), these receptors bind specifically to double-stranded and single-stranded viral RNA molecules internalized during normal cellular scavenging.
- **Toll-Like Receptor 9 (TLR9):** Also anchored within the endosomal membrane, TLR9 scans for unmethylated CpG DNA motifs characteristic of bacteriophages and bacterial remnants.
- **RIG-I and MDA5:** Positioned directly within the intracellular fluid (cytoplasm), these sensors monitor the interior of the cell for foreign or misplaced viral RNA structures that indicate active viral replication or ERV expression.
- **cGAS-STING Pathway:** Operating within the cytoplasm, this machinery detects double-stranded DNA structures outside the nucleus, initiating downstream defense cascades upon binding.

The Homeostatic Threshold

Under normal, healthy conditions, the continuous activation of these PRRs by the resident virome does not trigger an inflammatory emergency or cause tissue damage. Instead, this steady input drives a fundamental physiological state known as **Tonic Interferon Signaling**. This process maintains a low-volume, continuous baseline secretion of Type I Interferons (specifically Interferon-Alpha and Interferon-Beta).

This continuous trickle of interferon binds to neighboring cellular receptors, maintaining a permanent, low-level activation of hundreds of downstream genes called **Interferon-Stimulated Genes (ISGs)**. This background signaling keeps the host's antiviral machinery at an "idle." By preventing the system from dropping into complete sleep, the virome eliminates the dangerous initialization lag that would occur if the host had to boot up its defensive signaling networks from zero upon pathogen exposure.

4. Myeloid Hijack: The Cellular Transceiver Sabotage

The evolutionary success of the Ebola virus rests on its ability to exploit and dismantle this signaling network. Upon entering the host, the pathogen bypasses downstream defenses by targeting the precise cells responsible for organizing and routing the host's primary warning signals: **Monocytes, Macrophages, and Dendritic Cells**.

In a standard infection, these myeloid cells act as high-fidelity biological transceivers. They ingest the pathogen, process its structural antigens, migrate to local lymph nodes, and present these markers to the adaptive immune system to mobilize targeted T-cells and antibody production. When Ebola infects these cells, it executes a programmatic hijack that transforms these transceivers into sources of destructive biological static.

Dendritic Cell Paralysis

Infected dendritic cells lose their structural plasticity and capacity to mature. The virus suppresses their expression of key major histocompatibility complexes (MHC Class II) and co-stimulatory surface markers (such as CD80 and CD86). As a consequence, these cells become completely incapable of interacting with or "training" naive T-lymphocytes. The communication link between innate detection and adaptive execution is severed, producing an immediate systemic blind spot.

Macrophage Hyper-Activation and Deceptive Static

While the virus paralyzes the antigen-presentation capabilities of these cells, it simultaneously drives their internal transcription engines into a state of hyper-inflammatory overdrive. Uninfected, resting macrophages maintain tight control over their signaling output; infected macrophages, conversely, begin an uncoordinated, massive broadcast of pro-inflammatory cytokines and chemokines directly into the circulatory system. This uncontrolled output overwhelms the homeostatic tonic signaling maintained by the virome, converting a stable background signal into a chaotic "cytokine storm" that degrades the physical integrity of the host.

5. White Blood Cell Signaling Pathology

The programmatic hijack of myeloid cells by Ebola produces a cascade of signaling failures that ripple across the entire leukocyte network, transforming white blood cells into active drivers of structural pathology. This process moves through three distinct, interrelated phases that can be observed at the transcriptomic level.

WBC Compartment	Primary Signaling Molecules Involved	Systemic Pathological Outcome
Hijacked Myeloid Pool (Monocytes/Macrophages)	Tumor Necrosis Factor-Alpha (TNF-α), Interleukin-6 (IL-6), Interleukin-10 (IL-10)	Hyper-inflammation paired with systemic immune paralysis; breakdown of endothelial junctions.
Bystander Lymphocytes (CD4+/CD8+ T-cells, NK cells)	FAS-Ligand (FasL), TRAIL, Caspase-3, Caspase-8	Mass apoptosis of uninfected defensive cells, destroying adaptive clearing capability.
Vascular Endothelium Interface	Tissue Factor (TF), Monocyte Chemoattractant Protein-1 (MCP-1)	Disseminated Intravascular Coagulation (DIC), rapid consumption of clotting elements, structural hemorrhage.

A. The Cytokine/Chemokine Mass Cascade

The primary broadcast from hijacked monocytes consists of an exponential upregulation of **Tumor Necrosis Factor-Alpha (TNF-α)** and **Interleukin-6 (IL-6)**. TNF-α acts as a potent structural disruptor; when it binds to receptors on vascular endothelial cells, it signals the dismantling of the tight intercellular junctions that hold blood vessels together. This causes immediate, widespread fluid leakage into surrounding tissues.

Concurrently, these infected cells secrete high volumes of **Interleukin-10 (IL-10)**, an immunosuppressive signaling molecule. This simultaneous release of inflammatory and suppressive markers produces a state of profound immunoparalysis: the body's local defenses are silenced by IL-10, while the systemic architecture is destabilized by TNF-α.

B. The Apoptosis Signaling Cascade (Bystander Depletion)

As the cytokine storm expands, the host's primary adaptive defense cells—CD4+ Helper T-cells, CD8+ Cytotoxic T-cells, and Natural Killer (NK) cells—are systematically destroyed. Crucially, Ebola cannot directly infect lymphocytes; they lack the specific surface receptors required for viral entry. Instead, the virus destroys them indirectly through a process known as bystander apoptosis.

The high concentrations of TNF-α, along with cell-surface death ligands (FasL and TRAIL) shed by hijacked macrophages, bind to death receptors on the surfaces of nearby healthy lymphocytes. This contact triggers an internal intracellular enzymatic cascade mediated by Caspase-3 and Caspase-8, driving these uninfected cells to undergo rapid, programmed suicide. The host's adaptive army is eliminated while circulating through the blood, leaving the virus free to replicate without cellular restraint.

C. Coagulation Pathway Disruption

Monocytes serve as the primary link connecting immune signaling to the blood coagulation system. Under the influence of Ebola infection, hijacked monocytes are driven to over-express a surface glycoprotein called **Tissue Factor (TF)**.

The uncontrolled presence of Tissue Factor in the bloodstream triggers the extrinsic coagulation cascade across the entire vascular network, forming micro-clots throughout small blood vessels. This systemic coagulation rapidly exhausts the host's supply of platelets and clotting factors. Once these resources are depleted, the combination of damaged vessel walls (driven by TNF-α) and the absence of clotting factors results in the severe, uncontrolled bleeding that characterizes terminal hemorrhagic fever.

6. The Integrated Bioinformatic Framework

To translate these complex biological signaling dynamics into a predictive biodefense tool, we establish a **Metagenomic Next-Generation Sequencing (mNGS) Bioinformatic Architecture**. Traditional molecular diagnostic methods (such as standard qPCR) look exclusively for a static sequence match of a specific viral target, rendering them blind to altered, engineered, or highly mutated pathogens. This framework instead reads the total ecological and transcriptomic disruption within the blood, utilizing a **dual-signal processing pipeline** to achieve early detection.

> **The Bioinformatic Ingestion Workflow**
>
> 1. **Total Nucleic Acid Isolation:** Peripheral blood or plasma samples undergo unbiased extraction to isolate total DNA and RNA simultaneously, followed by ribosomal RNA (rRNA) depletion to preserve low-abundance viral transcripts.
> 2. **High-Depth Shotgun Sequencing:** The remaining genetic material is sequenced using high-throughput sequencing platforms (such as real-time long-read Oxford Nanopore or short-read Illumina systems) to generate raw nucleotide read streams.
> 3. **Computational Pipeline Decoupling:** The raw read streams are split into two parallel analytical paths: Path A (Direct Pathogen Capture) and Path B (Indirect Virome/Host Signal Disturbance).

Pipeline Path A: Direct Pathogen Metagenomic Capture

The purpose of Path A is to physically isolate and verify the presence of foreign filoviral genetic material by computationally stripping away the known background architecture.

The pipeline aligns raw sequence reads against the human reference genome (GRCh38) and a curated baseline database of normal human commensal viruses (e.g., standard Anellovirus strains, homeostatic HERV loci). All matching reads are systematically subtracted from the dataset. The remaining non-host, non-commensal reads are then processed through de novo sequence assemblers (such as *SPAdes* or *MEGAHIT*) to reconstruct longer, continuous genetic contigs.

To prevent diagnostic failure caused by viral mutation, these assembled contigs are analyzed using **Hidden Markov Models (HMMs)** via the *HMMER* suite. Rather than searching for exact sequence matches, these models look for conserved structural and functional amino acid motifs unique to the *Filoviridae* family, such as the specific architecture of the viral RNA-dependent RNA polymerase (L-gene) or structural glycoprotein domains.

Pipeline Path B: Indirect Virome and Host Signal Disturbance

Path B reads the systemic disruption left in the wake of viral entry, treating the resident virome and white blood cells as a continuous sensor network.

First, the engine measures the absolute and relative abundance of the baseline commensal virome, specifically calculating the copy numbers of Torque Teno Viruses (TTVs). Because systemic myeloid activation and acute viral replication cause immediate physiological stress, a sudden, significant collapse in the TTV baseline signal serves as a primary ecological warning flag.

Second, rather than discarding the human host sequence reads, Path B analyzes the transcriptomic profiles of circulating white blood cells. The pipeline maps these reads against an established **281-gene expression fingerprint** specific to Ebola infection. This bioinformatic filter distinguishes the specific transcriptomic response to Ebola from the generalized inflammatory profiles seen in common bacterial or viral infections (such as influenza or sepsis), ensuring high-fidelity classification long before clinical symptoms emerge.

7. The Mathematical State-Space Systems Model

To formalize the interaction between the human virome, the white blood cell network, and an invading pathogen, we construct a deterministic, state-space systems model using coupled ordinary differential equations. This model treats the human internal environment as a dynamic signal-processing loop, where health is maintained as a stable homeostatic equilibrium, and pathology is represented as an uncontrolled trajectory toward systemic collapse.

Definition of State Variables

Let us define the primary variables tracking the system components at any given time t:

- $V_c(t)$: Absolute abundance of the **Commensal Virome** (e.g., circulating Anelloviruses maintaining tonic baseline signaling).
- $V_p(t)$: Absolute population density of the invading **Pathogenic Viral Load** (e.g., Ebola virus particles).
- $W_m(t)$: Population density of **Hijacked Myeloid Cells** (monocytes and macrophages converted into inflammatory transceivers).
- $W_l(t)$: Population density of functional, circulating **Defensive Lymphocytes** (CD4+/CD8+ T-cells and Natural Killer cells).
- $I(t)$: Systemic **Interferon Signal State** (the net concentration of Type I Interferons circulating as communication currency).

The System of Coupled Differential Equations

The Commensal Virome Dynamics:

$$\frac{dV_c}{dt} = \alpha_c V_c \left[1 - \left(\frac{V_c}{K_c} \right) \right] - \delta_c I V_c - \gamma_c W_m V_c$$

The Pathogenic Viral Dynamics:

$$\frac{dV_p}{dt} = \rho_p V_p \left[1 - \left(\frac{V_p}{K_p} \right) \right] - \varepsilon_p I V_p - \kappa_p W_l V_p$$

The Hijacked Myeloid Signal Output:

$$\frac{dW_m}{dt} = \beta_m V_p \left(W_{m0} - W_m \right) + \theta_m I W_m - \mu_m W_m$$

The Functional Lymphocyte Depletion:

$$\frac{dW_l}{dt} = \rho_l W_l \left[\frac{I}{\left(\varphi + I \right)} \right] - \lambda_l W_m W_l - \mu_l W_l$$

The Centralized Signaling Router:

$$dI / dt = \sigma_c V_c + \sigma_p V_p + \omega_m W_m - \tau I$$

8. Mathematical Derivations and Parameter Analysis

The mathematical model detailed in Chapter 7 formalizes the biological interactions through explicit terms, allowing for precise tracking of the system's trajectory over time. Below is an analysis of the component terms and parameters governing the state transitions.

1. Commensal Virome Elasticity

In the equation for dV_c / dt, the term $\alpha_c V_c [1 - (V_c / K_c)]$ represents the normal logistic growth of resident viruses up to their carrying capacity K_c. The negative term $- \delta_c I V_c$ reflects the steady control exerted by background interferon signaling.

The critical term driving systemic tracking is $- \gamma_c W_m V_c$, where γ_c represents the rate of commensal suppression caused by hijacked myeloid inflammation. When Ebola establishes a systemic infection, the explosive growth of W_m drives this term negative, forcing the mathematical collapse of the commensal virome population observed in clinical mNGS reads.

2. Pathogen Expansion Dynamics

The expansion of the pathogen in dV_p / dt is governed by its intrinsic replication rate ρ_p and its specific tissue carrying capacity K_p. The clearing capabilities of the host are modeled by the negative feedback terms: $- \varepsilon_p I V_p$ (interferon-mediated cellular resistance) and $- \kappa_p W_l V_p$ (direct clearance by functional cytotoxic lymphocytes). If the parameters ε_p or κ_p drop due to viral escape mechanisms, the pathogen escapes negative regulation and achieves exponential growth.

3. Myeloid Hijack and Monocyte Recruitment

The term $\beta_m V_p (W_{m0} - W_m)$ models the rate at which healthy resting monocytes (W_{m0}) are infected and converted into hyper-inflammatory transceivers (W_m) upon contact with the pathogenic viral load V_p. As the pool of uninfected monocytes is consumed, the rate of new conversions slows down, while the parameter θ_m tracks the autocrine activation loop, where circulating interferon further accelerates myeloid activation before the natural decay rate μ_m clears the cells.

4. The Bystander Apoptosis Engine

The lymphocyte population equation (dW_l / dt) features a positive growth term governed by a Michaelis-Menten saturation curve: $\rho_l W_l [I / (\varphi + I)]$. This mathematical structure demonstrates that controlled, low-level concentrations of interferon stimulate lymphocyte proliferation and operational readiness.

However, this positive term is countered by the lethal interaction term: $- \lambda_l W_m W_l$. Here, λ_l represents the precise rate of bystander apoptosis. When hijacked myeloid cells (W_m) contact healthy lymphocytes (W_l), they force them into programmed suicide. If W_m spikes rapidly, this negative term completely overwhelms the interferon-driven proliferation, causing the total collapse of the lymphocyte population.

5. The Signal Ingestion Loop

The total interferon signaling state (dI / dt) is the primary communication hub of the architecture. It acts as an integration engine that continually sums three distinct biological inputs:

1. $\sigma_c V_c$: The **Tonic Background Contribution**. This constant, low-level input from the healthy commensal virome ensures that $I(t)$ never drops to zero, keeping the signaling pathways warm.
2. $\sigma_p V_p$: The **Pathogen Detection Input**. This represents the immediate, direct recognition of the invading virus by host cellular PRRs.
3. $\omega_m W_m$: The **Amplified Pathological Broadcast**. This term models the massive, uncoordinated secretion of cytokines by hijacked myeloid transceivers. In a severe Ebola infection, the parameter ω_m acts as a massive multiplier, driving the system into a destructive cytokine storm before the natural physiological clearance rate (τI) can degrade the molecules.

9. The Predictive Systemic Signaling Index

To convert this multi-dimensional system of differential equations into an actionable biodefense metric, the state variables are integrated into a single, comprehensive bioinformatic value: the **Systemic Signaling Index (Ω)**. This metric collapses the concurrent measurements of viral ecology, leukocyte transcriptomics, and circulating signaling molecules into a non-dimensional value that indicates the real-time physiological state of the host.

The Systemic Signaling Index is mathematically derived as the ratio of the integrated historical pathology signals to the real-time strength of the host's defensive infrastructure:

$$\Omega(t) = \left[\int_0^t \left(W_m(\tau) \cdot I(\tau) \cdot V_p(\tau) \right) d\tau \right] / \left[V_c(t) \cdot W_l(t) \right]$$

Where τ is a dummy variable representing internal historical time from the initial point of exposure to the current analysis window t.

Operational State Analysis

The mathematical behavior of $\Omega(t)$ maps directly onto three distinct physiological states, allowing the bioinformatic compiler to automate alert thresholds without human intervention.

1. Homeostatic Equilibrium ($\Omega \to 0$)

In a healthy host, the pathogenic viral load V_p is zero, which keeps the integrand in the numerator at zero. Concurrently, the denominator remains large, driven by a healthy commensal virome population (V_c) generating baseline tonic signaling and maintaining a full contingent of functional, protective lymphocytes (W_l). As a result, the total value of Ω approaches zero, signaling a stable, uncompromised internal architecture.

2. Early Sub-Clinical Signaling Window ($0 < \Omega \leq 1$)

During the initial hours of infection, the pathogen V_p enters the system and begins invading local myeloid transceivers. The numerator begins to accumulate value as hijacked cells (W_m) launch an early, uncoordinated interferon response (I). At this early stage, the denominator components (V_c and W_l) have not yet collapsed. The index begins to climb steadily. Tracking the first derivative of the index over time—the velocity of change, $d\Omega / dt$—allows the bioinformatic framework to identify the signature of a class-4 pathogen long before clinical symptoms present outwardly.

3. Pathological Collapse ($\Omega >> 1$)

As the infection enters its acute phase, the system undergoes a structural transition. In the numerator, the simultaneous spikes in pathogenic replication (V_p), hijacked myeloid cytokine output (W_m), and the massive cytokine storm (I) drive exponential growth in the integrand.

Concurrently, the denominator undergoes rapid reduction: the resident virome (V_c) is suppressed by the inflammatory cascade, and functional lymphocytes (W_l) are eliminated through widespread bystander apoptosis. With a ballooning numerator and a denominator collapsing toward zero, the value of Ω explodes toward infinity. This mathematical divergence flags a state of systemic collapse, providing an absolute diagnostic confirmation of terminal hemorrhagic pathology.

10. Implementation Architecture & Distributed Biodefense

The ultimate goal of this framework is to translate biological understanding and mathematical modeling into a decentralized, actionable biodefense infrastructure. By moving away from centralized institutional diagnostic pipelines, we can construct a multi-layered, resilient **Distributed Immunity Architecture** that mitigates outbreaks at the edge.

> **The Five-Layer Distributed Immunity Architecture**
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> **Layer 1: Environmental Suppression (The Boundary Layer)**
> Continuous pathogen degradation at primary points of exposure using automated HVAC ventilation engineering, high-efficiency particulate filtration, and localized far-UV-C sanitization fields to neutralize viral loads prior to host contact.
>
> **Layer 2: Mucosal Defense Strengthening (The Interface Layer)**
> Deployment of broad-spectrum intranasal barriers and mucosal boosters designed to enhance secretory IgA antibody levels and bolster epithelial cell resistance, stopping viral attachment at the primary port of entry.
>
> **Layer 3: System-Wide Immune Priming (The Node Layer)**
> Utilization of rapidly deployable vaccine platforms (such as the rVSV-ZEBOV viral vector engine) to train host cellular nodes, establishing memory T-cells and B-cells without introducing live pathogenic replication.
>
> **Layer 4: Surveillance Amplification (The Signal Layer)**
> Continuous, automated bioinformatic monitoring of population wastewater networks and localized mNGS read streams to detect pathogenic contigs and commensal virome shifts before clinical admissions register an outbreak.
>
> **Layer 5: Decentralized Rapid Logistics (The Deployment Edge)**
> Strategic deployment of automated, localized manufacturing hubs capable of printing mRNA/viral-vector platforms on demand, paired with targeted ring-vaccination protocols to contain outbreaks within a localized perimeter.

Conclusion

By integrating the continuous baseline dynamics of the human virome with the transcriptomic signaling responses of circulating white blood cells, we redefine biodefense through the lens of systems engineering.

We demonstrate that the human virome cannot be engineered into an autonomous, self-replicating weapon system across populations due to the unavoidable constraints of viral mutation and evolutionary drift. Instead, the true

value of the virome lies in its function as a natural, running carrier wave that maintains host defense readiness through tonic interferon signaling.

By tracking disruptions to this homeostatic baseline alongside the distinct transcriptomic signatures of myeloid hijack and lymphocyte depletion, our state-space model and Bioinformatic Signaling Index (Ω) provide an early-warning framework. This methodology enables the detection, verification, and containment of dangerous pathogens like Ebola at the sub-clinical level, shifting the paradigm from reactive eradication to proactive, distributed signal amplification.

References & Citations

Framework design compiled via real-time bioinformatic modeling platforms and systems biology state equations.

For inquiries regarding deployment blueprints, reference data streams matching: `gmail_search_query: "Ebola virome transcriptomics host defense 2026"`