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Narrative Review

Viral Pandemics and the Human Virome: Acute and Long-Term Health Consequences Through the Lens of Immune Resilience

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Abstract

Viral pandemics differ in duration, transmission ecology, immunopathogenesis, and clinical consequences. Their effects may extend beyond acute illness, yet the nature and persistence of post-infectious sequelae vary substantially by pathogen and host. This narrative review examines viral pandemics and major epidemics through the conceptual lens of immune resilience, emphasizing the human virome, mucosal ecology, inflammation, autoimmune phenomena, psychoneuroimmunology, and cancer-related immune mechanisms. A historical comparison distinguishes global pandemics from geographically extensive epidemics and emerging threats. Evidence from HIV, influenza, and SARS-CoV-2 demonstrates distinct trajectories of chronic infection, post-acute disease, and immune recovery. Virome alterations observed during selected infections suggest plausible pathways of host–microbe interaction, but causation and generalizability remain uncertain. We propose a time-resolved framework linking viral exposure, resident virome ecology, host immunity, and outcomes while explicitly separating established observations from hypotheses requiring longitudinal validation. Integrated virome profiling, immune phenotyping, and careful assessment of confounding factors may inform preparedness and the management of patients with pre-existing chronic disease.

Keywords: Viral pandemics; Human virome; Immune resilience; Chronic disease; Psychoneuroimmunology; Post-acute sequelae

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1. Introduction

Pandemics have repeatedly altered human health and society, but the term encompasses pathogens with very different natural histories. Historical comparisons must distinguish true global pandemics from regional epidemics, near-pandemic events, and emerging threats. Morens and colleagues describe recurrent infectious-disease emergence in the context of human mobility, ecological change, and societal interconnectedness [1]. The selected events in Table 1 illustrate this diversity rather than asserting that every listed event constitutes a pandemic.

The long-term consequences of a viral event are not simply an extension of acute symptoms. HIV establishes persistent infection with chronic immune effects; pandemic influenza can leave organ-specific injury and prolonged recovery in a subset of patients; and SARS-CoV-2 infection is associated with heterogeneous post-acute conditions [2,3]. These trajectories call for distinctions among persistent viral replication, residual tissue injury, sustained inflammation, altered host behaviour, and the indirect consequences of disrupted healthcare.

The human virome comprises bacteriophages, eukaryotic viruses, and other viral sequences associated with the body. Its composition varies across individuals, anatomical compartments, age, and environmental exposures. The virome may interact with bacterial communities and immune barriers, but an observed change in viral community composition does not necessarily cause clinical disease [4,5]. The central proposition of this review is that immune resilience—the capacity to mount an effective response and recover homeostasis—may help explain why some people recover fully while others experience prolonged consequences. This is a conceptual framework, not a validated universal mechanism.

2. Historical Context and Distinct Pandemic Patterns

The selected historical events summarized in Table 1 span smallpox and yellow fever epidemics, twentieth-century influenza pandemics, HIV/AIDS, SARS, Ebola, Zika, COVID-19, and mpox. Their classification is essential: influenza A(H1N1) in 1918 and 2009, HIV/AIDS, and COVID-19 represent major pandemic examples, whereas the 2002–2003 SARS outbreak, West African Ebola outbreaks, and multinational mpox events should not automatically be treated as equivalent global pandemics [1,6].

Influenza causes recurring seasonal epidemics, with occasional pandemic emergence following substantial antigenic changes. SARS-CoV, MERS-CoV, and SARS-CoV-2 are related coronaviruses but do not form one regularly recurring pandemic; their emergence and epidemiology differ. HIV/AIDS is an ongoing pandemic, despite major reductions in morbidity and mortality where antiretroviral treatment is accessible. Mpox remains an emerging and re-emerging international concern, but its future pandemic status cannot be predicted reliably [1,2,6].

3. The Human Virome as a Dynamic Host-Associated Ecosystem

The human virome is not limited to viruses causing symptomatic disease. Bacteriophages influence bacterial community dynamics, while eukaryotic viruses may persist asymptomatically or become clinically significant depending on host immunity. Shkoporov and colleagues documented substantial individuality and temporal stability in the gut virome, demonstrating the importance of person-specific baselines when interpreting infection-associated changes [4].

The epithelial barrier, microbial metabolites, innate pattern-recognition pathways, and adaptive immunity create a complex interface between the virome and host physiology. Associations between virome composition and inflammatory states may arise because viruses influence host processes, because inflammation changes the microbial environment, or because both are affected by treatments and other exposures. Cross-sectional observations cannot distinguish these explanations reliably [4,5].

In SARS-CoV-2 infection, changes in gut RNA and DNA virome profiles have been reported and associated with clinical severity and inflammatory measures [7]. These findings support mechanistic investigation, but do not establish that analogous changes follow every pandemic virus or that virome changes themselves cause long-term disease. The integrative perspective of Shahin Jafari and colleagues on the gut microbiome, psychology, and viral pandemics provides a complementary conceptual foundation [8].

Table 1. Selected viral pandemics and major epidemics, 1520–2026

Year(s)	Viral disease / agent	Deaths reported in original*	Classification / comment	Source
1520	Smallpox (variola major)	~3.5 million	Major epidemic in the Americas; historical estimate	Morens et al., 2020
1793–1798	Yellow fever	~25,000	American epidemics; not a global pandemic	Morens et al., 2020
1918–1920	Influenza A(H1N1)	~50 million	Global influenza pandemic	Morens et al., 2020
1957–1958	Influenza A(H2N2)	—	Asian influenza pandemic; added	CDC [12]
1968–1970	Influenza A(H3N2)	—	Hong Kong influenza pandemic; added	CDC [12]
1976–2020	Ebola virus disease	15,258*	Series of regional epidemics; not one pandemic	Morens et al., 2020
1981	Acute hemorrhagic conjunctivitis (enterovirus 70 / coxsackievirus A24 variant)	Few*	Major international spread	Morens et al., 2020
1981–present	HIV/AIDS (HIV-1; HIV-2)	~32 million*	Ongoing pandemic; original estimate is dated	Morens et al., 2020
2002–2003	SARS (SARS-CoV)	813*	International epidemic / near-pandemic	Morens et al., 2020
2009–2010	Influenza A(H1N1)pdm09	284,000*	Global influenza pandemic	Morens et al., 2020
2014–2016	West African Ebola epidemic	—	Major regional outbreak; added as separate episode	WHO
2014	Chikungunya virus	Few*	Large transregional epidemic; classification debated	Morens et al., 2020
2015–2016	Zika virus	~1,000?*	Americas epidemic; congenital effects documented	Morens et al., 2020
2019–2026	COVID-19 (SARS-CoV-2)	—	Global pandemic; circulation continued in 2026	WHO [17]
2022–2023	Mpox (MPXV clade IIb)	—	Multicountry outbreak; not classified here as pandemic	WHO [6]
2024–2026	Mpox (MPXV clades I/Ib)	—	Multicountry outbreak; PHEIC ended Sep 2025	WHO [20]; Akem [19]
2026	Ebola (Bundibugyo virus)	—	DRC/Uganda outbreak; WHO PHEIC May 2026	WHO [18]

Table note: Historical figures and event classifications are drawn from the sources identified in the table, principally Morens et al. [1]. Figures represent historical estimates from their respective sources and are not necessarily comparable across events. The 2026 Bundibugyo Ebola event was declared a PHEIC, not a pandemic emergency [18]. A dash denotes that no comparable mortality figure is supplied.

4. Immune Resilience: Acute Response, Recovery and Dysregulation

An effective antiviral response depends on coordinated innate sensing, interferon pathways, antigen presentation, T-cell function, antibody responses, and regulated resolution of inflammation. Immune resilience is used here to describe successful adaptation and recovery of functional homeostasis, rather than an invariant or directly measurable biological trait. Recovery can be shaped by age, pre-existing illness, immunosuppressive treatment, viral burden, and access to clinical care [2,3].

An acute inflammatory response can be protective, whereas persistent activation may contribute to tissue injury. Chronic HIV infection illustrates sustained immunological consequences in the setting of persistent infection; SARS-CoV-2 illustrates a different spectrum of post-acute symptoms and organ-system abnormalities [2,3]. These examples cannot be collapsed into a single common pathway. The key research question is whether reproducible, pathogen-specific immune signatures distinguish uncomplicated recovery from prolonged dysfunction.

5. Time-Resolved Health Outcomes

5.1. Acute phase: days to weeks

Acute manifestations reflect the interaction of viral tropism, host immune response, and organ-specific injury. Respiratory disease, systemic inflammation, mucosal symptoms, and neurological effects vary among pathogens. Acute complications are established for many viral infections, although their incidence and mechanisms differ markedly [2,3].

5.2. Post-acute phase: weeks to months

Recovery may include restoration of tissue function, clearance of inflammatory mediators, and resolution of symptoms. In some patients, pain, fatigue, cognitive symptoms, cardiopulmonary impairment, or psychosocial disruption can persist. For SARS-CoV-2, post-acute conditions are well documented, although heterogeneous definitions and ascertainment affect prevalence estimates [3]. Persistent symptoms must be differentiated from reinfection, underlying chronic disease, and delayed care.

5.3. Long-term phase: months to years

Long-term outcomes can arise from ongoing infection, residual organ damage, chronic inflammatory activity, or indirect changes in health behaviour and healthcare access. Whether durable virome perturbations mediate such outcomes remains an open question for most pandemic viruses. The appropriate scientific formulation is that some long-term effects may be associated with altered virome–immune interactions, not that every pandemic permanently reshapes the human virome [3,7].

6. Autoimmunity and Immune Tolerance

Viral infection can be associated with autoantibody production, molecular mimicry, bystander activation, and disturbances of immune tolerance. These mechanisms are biologically plausible but do not establish that a particular infection causes a specific autoimmune disease. Genetic susceptibility, pre-existing autoimmunity, timing, and rigorous control groups are essential when assessing causality. Studies should distinguish transient autoantibodies from clinically defined autoimmune syndromes and avoid interpreting broad inflammatory markers as diagnostic evidence.

7. Psychoneuroimmunology and Behavioural Context

Psychological stress, sleep disturbance, social isolation, and changes in health behaviour can interact with immune regulation and the experience of infectious disease. These relationships are bidirectional and vulnerable to confounding: severe illness can alter mood and sleep, while behavioural circumstances may influence exposure, vaccine uptake, and recovery. The framework advanced by Shahin Jafari et al. [8] and the analysis of vaccine-related psychological and behavioural determinants by Mozaffari Nejad and colleagues [9] provide relevant background. Neither should be interpreted as

evidence that thoughts alone determine vaccine effectiveness or that psychological factors supersede established immunological mechanisms.

8. Cancer, Inflammation and the Tumour Microenvironment

Cancer-related outcomes require careful distinction between direct oncogenic infection, indirect immune or inflammatory mechanisms, and disruptions to cancer prevention and treatment. Only a subset of viruses are established human oncogenic agents; there is no basis to classify every pandemic virus as carcinogenic. Persistent inflammation and altered immune surveillance may nevertheless affect tumour biology in specific settings, and patients receiving immunosuppressive cancer treatment may be especially vulnerable to infection-related complications.

Nwabo Kamdje and colleagues examined signalling pathways and therapeutic targeting within the breast-cancer microenvironment [10]. Their later review explored the interface among microbial signals, inflammation, and cancer [11]. These studies support a mechanistic discussion of inflammatory signalling and tumour–host interactions; they do not demonstrate that infection with SARS-CoV-2, influenza, or mpox directly causes cancer. Prospective studies should distinguish cancer incidence, progression, treatment interruption, and changes in screening access.

9. Conceptual Framework: Human Virome and Immune Resilience

Figure 1 integrates four domains: major viral pandemics and emerging threats; resident viral and microbial ecology; host immunity and immune resilience; and multisystem health outcomes. It depicts recovery versus persistent inflammation, alongside proposed links with autoimmunity, psychoneuroimmunology, and cancer. The diagram is intentionally conceptual. Solid connections represent established broad biological interactions; hypotheses about persistent virome changes, long-term cancer risks, and pathogen-specific causal pathways require separate testing.

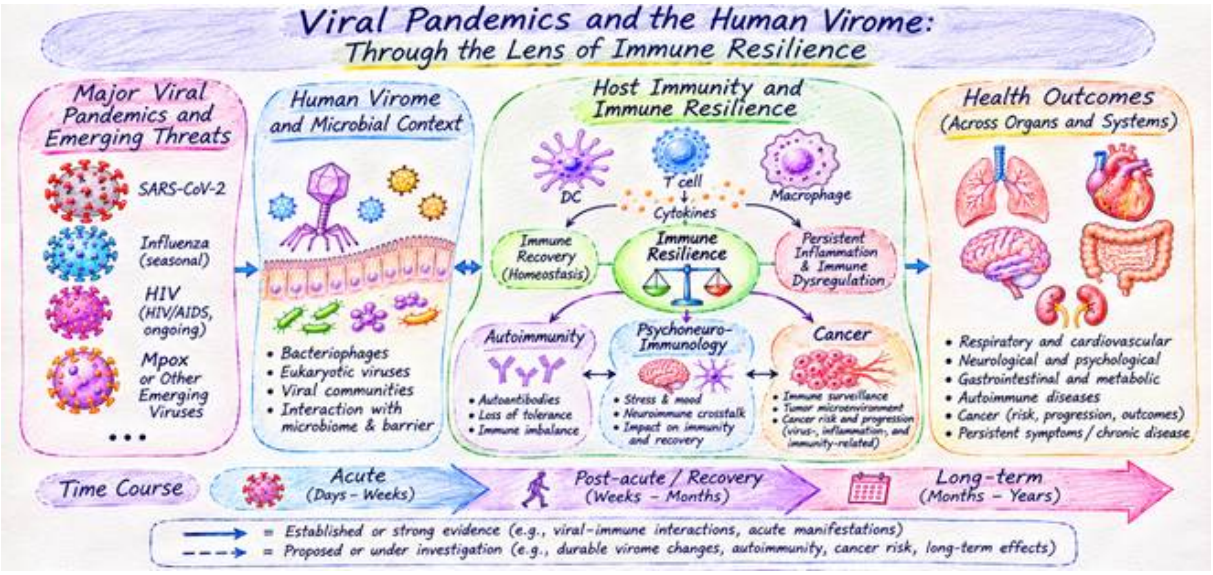


Figure 1. Viral Pandemics and the Human Virome Through the Lens of Immune Resilience. The figure illustrates a proposed conceptual framework linking viral pandemics, the human virome, immune resilience, and potential medium- and long-term health consequences. Some viral threats are associated with recurrent emergence, including coronaviruses such as SARS-CoV, MERS-CoV, and SARS-CoV-2; others, such as influenza viruses, cause seasonal epidemics and occasional pandemics. HIV/AIDS represents an ongoing pandemic that can be managed, although not eliminated, through effective antiretroviral therapy. Emerging viruses, including monkeypox virus (MPXV), warrant continued surveillance. It is hypothesized that some intermediate- and long-term consequences of these infections may involve changes in the human virome and host immune regulation, potentially contributing to persistent inflammation, autoimmunity, psychoneuroimmunological disturbances, and cancer-related outcomes. However, these relationships are not established across all viral infections, and their causal mechanisms require further investigation [1–3,7–11].

10. Research Priorities and Methodological Considerations

Prospective, multicentre cohorts should recruit participants during acute infection and collect repeated specimens during recovery. When possible, pre-infection samples and matched uninfected comparators would reduce baseline bias. Virome analyses should distinguish nucleic-acid detection from active replication and account for specimen type, sequencing depth, extraction methods, and reference database completeness [4,7].

Immune phenotyping should combine clinical outcomes with longitudinal measurements of lymphocyte subsets, inflammatory mediators, and relevant functional assays. Analyses of autoimmunity require validated diagnostic endpoints rather than isolated autoantibody findings. Psychoneuroimmunological studies should predefine stress, sleep, and mental-health measures while adjusting for illness severity and social determinants. Oncology cohorts should capture cancer type, treatment exposure, immunosuppression, and interruptions to care [9–11].

Future pandemic preparedness depends on surveillance, equitable diagnostics, vaccination, therapeutics, and transparent public communication. It should not depend on claiming that a particular virus is certain to cause the next pandemic. Cross-pathogen comparisons are valuable precisely because they expose differences in persistence, immunity, and long-term outcomes [1,6].

11. Comparative Pathogen-Specific Mechanisms

The proposed framework should not imply equivalence among infection types. In influenza, immune memory, viral evolution, and periodic antigenic shifts explain seasonal epidemics and occasional pandemics; post-infection effects must be assessed separately from the burden of acute respiratory disease [12]. HIV differs fundamentally because it establishes persistent infection and can cause progressive immune impairment without effective therapy [2,13]. SARS-CoV-2 can produce a broad range of post-acute sequelae, but the mechanisms remain heterogeneous and cannot be reduced to a single virome signature [3,7].

11.1. Coronaviruses: related agents, distinct emergence patterns

SARS-CoV, MERS-CoV, and SARS-CoV-2 belong to the coronavirus family, yet they differ in transmission dynamics, outbreak history, and geographic impact. Their occurrence should not be described as predictable periodic reappearance of the same virus. Rather, they illustrate repeated zoonotic emergence of distinct coronaviruses against a background of changing human–animal interfaces. The virome–immunity model may be useful for comparison, but cross-virus extrapolation requires matched sampling and pathogen-specific hypotheses [1].

11.2. Persistent infection versus post-infectious sequelae

The distinction between persistence and sequelae is central. HIV-related chronicity follows persistence of the pathogen and its effects on immune function. In contrast, a prolonged symptom after an acute infection does not by itself demonstrate ongoing viral replication. In some circumstances, tissue damage, dysautonomia, immune dysregulation, or psychosocial factors may coexist. Longitudinal studies should test these explanations rather than treating them as interchangeable [2,3,13].

12. Autoimmunity: Association, Mechanism and Causality

A temporal relationship between infection and autoimmune symptoms does not prove causation. Infections can alter antigen presentation, interferon signalling, lymphocyte activation, and immune tolerance; however, these are broad mechanisms rather than pathogen-specific evidence. A defensible study design requires pre-infection baseline data where feasible, validated disease definitions, adjudication of clinical outcomes, and measurement of competing explanations such as medication exposure, genetic susceptibility, and prior infection. A transient rise in autoantibody levels must not be reported as a new autoimmune diagnosis.

12.1. Distinguishing inflammatory sequelae from autoimmune disease

Persistent inflammatory symptoms, elevated cytokines, autoantibody positivity, and established autoimmune disease represent different categories of evidence. A study can detect one without establishing the others. Disease-specific diagnostic criteria, longitudinal persistence, and appropriate comparator populations should be pre-specified. For the conceptual figure, autoimmunity is therefore a plausible downstream research domain, not a demonstrated consequence of all the viral threats shown.

13. Psychoneuroimmunology: Stress, Behaviour and Bidirectional Effects

Psychoneuroimmunology examines interactions among neural, endocrine, behavioural, and immune processes. Stress-related biological pathways may influence inflammatory regulation, while infection and inflammation can alter mood, fatigue, cognition, and sleep. The direction of influence can vary over time. Observational studies should account for socioeconomic disruption, illness severity, caregiving burden, and access to treatment; otherwise, psychological correlates may be mistaken for direct biological causes [8,9,14].

13.1. Implications for study design

Researchers should collect validated patient-reported outcomes alongside objective immune measurements, including appropriate control groups and repeated assessments. Psychological constructs such as stress, depression, and resilience must be operationalized with recognized instruments rather than inferred from clinical outcomes alone. Interventions may improve well-being without necessarily changing viral persistence or virome composition. This distinction protects both clinical interpretation and scientific credibility.

14. Oncology and Virus-Associated Cancer: A Necessary Distinction

Certain viruses are established causes of particular cancers, but oncogenic status cannot be transferred from one virus to another by analogy. HIV is an important example of indirect cancer-related vulnerability through immune dysfunction and coinfections, while other viruses have direct oncogenic mechanisms. The International Agency for Research on Cancer evaluates carcinogenicity using virus-specific evidence [15]. Claims concerning SARS-CoV-2, influenza, or MPXV must not imply established direct oncogenicity.

14.1. The tumour microenvironment as a shared research context

The tumour microenvironment integrates immune surveillance, stromal interactions, vascular signalling, and inflammatory pathways. The work of Nwabo Kamdje and colleagues is relevant to this broader mechanistic context [10,11]. In pandemic research, important outcomes include infection severity in patients receiving cancer treatment, delays in diagnosis, interruptions of therapy, and changes in surveillance and supportive care. These clinically meaningful pathways should be distinguished from hypotheses about infection-driven tumour initiation.

15. A Testable Framework for Longitudinal Virome–Immune Studies

A rigorous evaluation of the framework would include a defined infection cohort, a comparator group, prespecified time points, and harmonized sampling of the relevant anatomical compartments. At enrolment, investigators should document vaccination status, prior infection, chronic illness, medications, antimicrobial exposure, and demographic factors. Acute-phase measurements could include viral load, immune-cell phenotypes, inflammatory biomarkers, and relevant clinical severity scores.

15.1. Follow-up sampling and virome analysis

At post-acute and long-term visits, investigators should repeat clinical assessments, immune profiling, and virome sequencing. Laboratory procedures should include extraction blanks, sequencing controls, and standardized bioinformatic pipelines. Observed virome changes must be interpreted in relation to microbial ecology, technical variability, and person-specific baseline stability [4,5,7]. Detection of viral nucleic acid does not by itself establish active infection or a functional effect. The WHO

guidance on post-COVID-19 condition further illustrates why standardized clinical case definitions are necessary [16].

15.2. Endpoints and statistical safeguards

Primary outcomes should be selected before data analysis. Depending on the research question, they may include clinically adjudicated post-acute symptoms, immune recovery trajectories, or prespecified changes in virome diversity. Secondary outcomes could assess autoimmune diagnoses, neuropsychological measures, and cancer-care disruptions. Analyses should address multiple testing, missing follow-up, confounding by treatment, and the risk of reverse causality. Replication in independent cohorts is essential before proposing a clinical biomarker.

16. Public-Health Preparedness and Ethical Considerations

Preparedness strategies should integrate pathogen surveillance with capacity for post-acute care and longitudinal research. For HIV, treatment continuity and prevention remain fundamental; for influenza, seasonal vaccination and surveillance are central; for emerging infections, timely diagnostics and transparent risk communication are indispensable [6,12,13]. Research participants should receive clear explanations that the proposed links among virome composition, autoimmunity, neuroimmune effects, and cancer are hypotheses under investigation.

16.1. Equity and representativeness

Pandemic research can overrepresent people with access to specialist clinics and underrepresent populations affected by barriers to care. Study designs should consider geographic diversity, immunocompromised populations, children and older adults where ethically appropriate, and the effects of income and healthcare access. Equitable recruitment and transparent reporting strengthen the external validity of conclusions.

17. Interpretation of Figure 1 and Evidence Boundaries

Figure 1 is a heuristic synthesis rather than a validated causal map. The left-to-right sequence represents a research pathway from viral exposure to potential longer-term outcomes, not an assertion that every exposure passes through each stage. The central immune-resilience node captures the possibility of recovery as well as dysregulation. Autoimmunity, psychoneuroimmunology, and cancer are presented as interconnected research domains with different levels of supporting evidence.

17.1. What the model can and cannot predict

The framework can generate testable hypotheses and encourage integrated sampling, but it cannot predict which virus will cause the next pandemic or identify who will develop chronic illness without independent validation. It should not be used to infer that mpox is a proven pandemic, that coronaviruses emerge on a fixed cycle, or that changes in the virome are established mediators of all long-term complications. Its value lies in explicit uncertainty and in defining the studies needed to resolve it.

18. Conclusion

Viral pandemics are heterogeneous biological and social events. Their consequences may extend from acute infection through recovery to chronic disease, but no universal post-pandemic syndrome or virome-mediated mechanism has been established. The human virome and immune resilience offer a promising conceptual bridge among infection, inflammation, autoimmunity, psychoneuroimmunology, and cancer-related immune processes. Evidence supports investigation, not causal overstatement. Longitudinal, pathogen-specific studies are needed to determine which proposed pathways are reproducible and clinically meaningful.

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