



Mini Review

Emerging Viral Pathogens: The Perpetual Challenge of Viral Emergence and Re-emergence

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Abstract

As the COVID-19 pandemic demonstrated, the continued emergence and spread of viral pathogens is a continuing global public health concern. This mini-review examines the factors contributing to the emergence and resurgence of viral pathogens and how viral agents, animal reservoirs, and human activities interact to facilitate the spread of new and emerging viruses. Recent research on the genetic plasticity of viruses - especially RNA viruses with high rates of mutation and recombination and recombination mechanisms that can lead to different variants infecting new hosts - is integrated into this strategy. It also recognises that the vast, largely unexplored reservoir of zoonotic viruses in wildlife, especially bats, rodents and non-human primates, is a source of spill-over effects and that anthropogenic drivers such as land use change, climate change, globalisation and urbanisation are accelerating the spread of pathogenic agents. Findings underscore that viral emergence is not a chance event. It is a predictable process caused by documented human, animal, and environmental interactions. This certainty demands an immediate and fundamental shift in global health strategy toward comprehensive solutions. These solutions must actively integrate proactive public health measures with greatly enhanced global surveillance networks. Also, it is necessary to have One-Health collaboration, the targeted ecosystem protection initiatives, and the investment in fast vaccine technology through platforms to be able to implement the successful mitigation. All these actions must be taken together to control future viral hazards and to challenge them successfully.

Introduction

The 21st century has been persistently defined by the spectre of emerging and re-emerging viral pathogens. From the sudden global devastation of the SARS-CoV-2 pandemic to the recurrent, focal outbreaks of Ebola virus in Central and West Africa and the relentless expansion of arboviruses like Zika and Chikungunya, the narrative of a fragile truce between humanity and the viral world has been decisively shattered. The term emerging viral pathogen describes a virus that has either newly appeared in a population or has existed but is rapidly increasing in incidence or geographic range [1]. This phenomenon is not novel; history is replete with examples, from the influenza pandemic of 1918 to the introduction of HIV in the 20th century. However, the current pace of emergence appears to be accelerating, prompting a critical and urgent question: why do these pathogens keep coming back?

The appearance and return of viral threats aren't random; they follow a cycle. This cycle—where viruses appear, are suppressed, and then come back—is driven by a complex, linked mix of biological, ecological, and human factors (Figure 1). The return of old diseases like Yellow fever and the arrival of new ones like SARS-CoV-2 are fundamentally the same problem. Both stem from a failure to control the core reasons that push viruses to move between species and environments. This mini-review will argue that the perpetual challenge of emerging viral pathogens is a direct consequence convergent causality, where viral evolution, environmental perturbation, and human behaviour create a perfect storm for spillover and spread. By examining the principal drivers—including viral genetic plasticity, zoonotic reservoirs, anthropogenic environmental changes, globalization, and the inadequacies of public health infrastructure—this review will synthesise current understanding to illustrate that viral emergence is not an unforeseeable catastrophe but a predictable outcome of modern human existence.

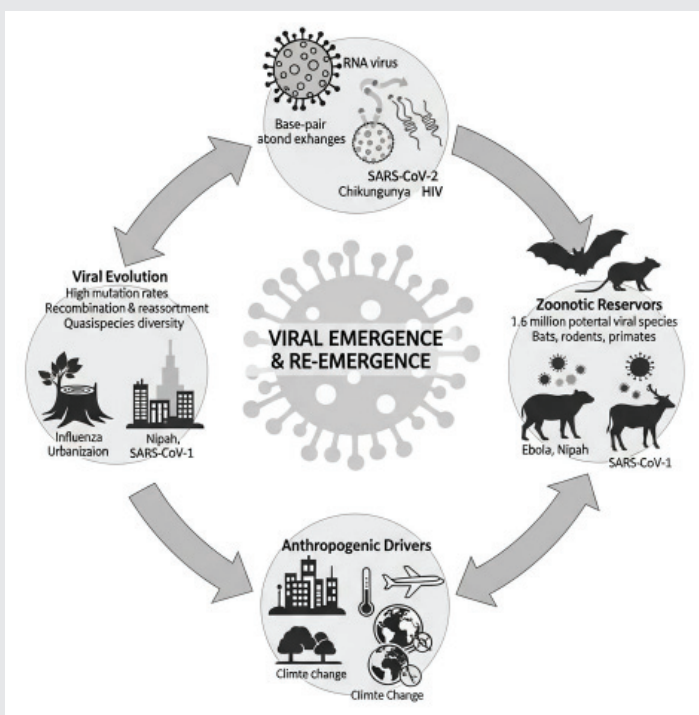


Figure 1: Convergent Causality of Viral Emergence and Re-emergence. This diagram depicts a synergistic feedback loop between four domains: 1) Viral Evolution (e.g., mutation in SARS-CoV-2); 2) Zoonotic Reservoirs (e.g., bats, rodents); 3) Anthropogenic Drivers (e.g., deforestation, urbanization); and 4) Public Health Factors (e.g., global travel). Their convergence at the central Spillover Event initiates outbreaks. Bidirectional arrows show feedback; for example, spillover impacts public health, which can alter viral evolution and environmental pressures, perpetuating the cycle of emergence and re-emergence.

The inherent mutability: viral evolution and adaptation

At the most basic level, the ability of viruses to emerge and re-emerge is encoded in their very nature. Viruses, especially RNA viruses, have an evolutionary system that naturally creates a lot of diversity. Many major threats that have recently emerged, such as influenza, HIV, SARS-CoV-2, and Ebola, are all RNA viruses. Their RNA-dependent RNA polymerases or reverse transcriptases are notoriously error-prone, lacking the proofreading mechanisms of cellular DNA polymerases. This results in high mutation rates, creating vast and dynamic populations often described as quasispecies—clouds of genetically related variants [2].

This genetic plasticity is the raw material for adaptation. A single mutation in a surface protein, such as the haemagglutinin of influenza or the spike protein of SARS-CoV-2, can alter host range, tissue tropism, or transmissibility. For example, the Chikungunya virus outbreak on La Réunion Island in 2005–2006 was caused by a very small change. A single part of the virus's outer layer, called the E1 envelope glycoprotein, gained a new amino acid (A226V). This single change made the virus much better at infecting the mosquito species *Aedes albopictus* [3]. Since *A. albopictus* is more widespread and lives better in cities than the usual carrier (*Aedes aegypti*), this tiny change led to a huge outbreak and helped the virus spread worldwide.

Furthermore, recombination and reassortment provide mechanisms for more dramatic evolutionary leaps. The 2009

H1N1 influenza pandemic virus was a quintessential re-assortant, containing gene segments from avian, swine, and human influenza viruses that had co-mingled in pig reservoirs [4]. Coronaviruses also frequently use recombination. This process, which lets them swap genetic material, was likely key to creating both SARS-CoV-1 and SARS-CoV-2. Recombination allowed these viruses to gain the traits needed to spread efficiently from one person to another [5]. Therefore, the constant generation of genetic diversity ensures a perpetual pool of potential variants that are pre-adapted or readily adaptable to new hosts, raising the question not if a new variant will emerge, but when.

The zoonotic reservoir: An immense and uncharted library of viruses

Most of the emerging infectious diseases come from animals, meaning they are zoonotic. The number of viral species that are still undiscovered and can be found in mammals and birds is more than 1.6 million, and out of these, about 700,000 are thought to have the capability to infect humans [6]. This is a huge, and mostly unstudied, collection of possible pathogens. The interface between human and wildlife populations is the critical zone where spillover events occur.

Different animal orders act as disproportionate contributors to viral emergence. Bats, in particular, have been identified as a major reservoir for virulent zoonoses, including Ebola, Nipah, SARS-like coronaviruses, and the original SARS-CoV-1. The physiological peculiarities of bats—such as their ability to fly, their unique immune systems that allow for viral persistence without clinical disease, and their social roosting behaviour—make them ideal viral reservoirs and dissemination vehicles [7]. Similarly, rodents and non-human primates are significant sources of zoonotic viruses.

The process of spillover is complex and multifactorial, requiring a chain of events a human must come into contact with an infected animal or its excretions; the virus must be capable of infecting human cells (a property influenced by the compatibility of viral surface proteins with human receptors, like ACE2 for SARS-CoV-2); and the virus must achieve sufficient replication within the human host to be transmitted onward. Most of the time, spillover events do not lead to anything, as the new virus cannot spread in human populations. Still, the great variety of interactions at the human–animal interface daily makes it possible for a virus to acquire a perfect combination of characteristics for the emergence, though the probability is low for any single event, it becomes a statistical certainty over time.

Anthropogenic drivers: Forcing the hand of nature

Though viral evolution and zoonotic reservoirs generate the biological potential for emergence, it is primarily humans who open it up and make its occurrence a possibility. Drivers of viral emergence are strongly anthropogenic and generate and exacerbate interfaces through which spillover can occur.

- a. **Land-use change and agricultural expansion:** Deforestation, urbanisation, and the expansion of

agriculture are among the most significant drivers. As natural habitats are fragmented and destroyed, wildlife is forced into closer proximity with human settlements and livestock. This compression of ecological niches dramatically increases contact rates. The emergence of the Nipah virus in Malaysia in 1999 is a classic case study. The establishment of intensive pig farms at the edge of forests, where fruit bats (the natural reservoir) fed, allowed the virus to spill over from bats to pigs, and then from pigs to humans, resulting in a devastating outbreak [8]. Similarly, the hunting and trade of bushmeat, a practice often intensified by habitat loss and economic need, is a direct route of exposure, as suspected in the early cases of Ebola virus disease and HIV.

b. Climate change: The impact of climate change on disease emergence is complex but increasingly undeniable. Changes in temperature, precipitation, and humidity can directly affect the distribution and abundance of arthropod vectors. An increasing range of *Aedes aegypti* and *Aedes albopictus* mosquito vectors for Dengue, Chikungunya, and Zika viruses is closely associated with climate change, leading these diseases to become established within naïve populations in temperate zones [9]. Additionally, climate-change-related effects can also disrupt wildlife migration and ecosystems, which may bring about new interspecific contacts or transmission routes of viruses.

c. Globalisation and urbanization: The modern world is hyper-connected. With international air travel, a germ incubated in a small village might be flown to the capital city within the time of viral incubation. The vulnerability of allowing entrance to unimpeded international travel, as witnessed during the rapid spread around the world of SARS-CoV-1 in 2003 and SARS-CoV-2 in 2020, has been realised such that it cannot be ignored. Overcrowded, spontaneous urban communities with insufficient sanitation and health care provide the best environment for the perpetuation and spread of respiratory and now also enteric viruses. Those cities act as amplifiers, with pathogens that break out from the slaughterhouses spreading and radiating along transportation networks.

The cycle of panic and neglect: Societal and public health amplifiers

Human response to viral threats tends to cycle between damaging levels of panic and neglect. In a time of crisis (eg, the 2014–2016 outbreak of Ebola in West Africa or the COVID-19 pandemic), vast financial and political resources are marshalled. Surveillance improves, research funding booms, and the public is riveted. But after the immediate crisis passes, attention flags, resources wane, and surveillance systems are dismantled or neglected until the next emergency [10]. It is precisely this cyclical amnesia that enables resurgence.

This neglect is exacerbated by the structural deficiencies in global public health infrastructure. Molecularbrand Many

of the areas most at risk for viral emergence are also plagued by the poorest health systems, with weak surveillance, poor diagnostic capacity, and a lack of a robust primary care system. The One Health initiative, acknowledging the interdependence of human, animal, and ecosystem health, has received notable rhetorical attention but is underresourced and lowly integrated into policy. Without sustained, deliberate investment in underpinning public health capacities — from local clinics to global surveillance networks — the world remains in an inherently reactive stance, constantly at risk of yet another spillover. Moreover, vaccine hesitancy, misuse of antimicrobials, and the flow of misinformation are secondary social problems but can also hinder attempts to control emerging and re-emerging diseases such as measles and polio.

Conclusion

The ongoing appearance of emerging and re-emerging viruses is a predictable result, not a random event, driven by a convergence of factors—a “syndemic” of causes. This threat stems from the intrinsic mutability of viruses, whose biological drive to adapt interacts with the immense, stressful environment created by human activity. The continuous threat is fueled by the vast, simmering zoonotic reservoir (viruses in animals) and strong human-caused (anthropogenic) drivers like land-use change, climate change, and globalisation. Asking, “Why do they keep coming back?” is misleading because it implies they left. In fact, many viruses never truly left their animal hosts. The conditions necessary for them to spillover and spread are now a permanent and escalating feature of our human-dominated world, making events like the COVID-19 pandemic an anticipated “grey rhino” threat that was broadly foreseen yet consistently ignored.

Addressing this perpetual challenge requires a fundamental shift from a reactive to a proactive stance. This means sustained investment in global surveillance and One Health initiatives to identify threats at their source, along with deciphering the virome of wildlife to understand the potential threat landscape. Additionally, protecting and restoring natural ecosystems is crucial to limit human-wildlife contact. We need to strengthen public health systems worldwide and advance platform technologies to quickly develop vaccines and treatments. Unless we tackle the combined causes of new viruses with the same focus and resources used during a crisis, humanity will be stuck in a Sisyphean struggle, always caught off guard by the next emerging pathogen.

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