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The Rise Of New Emerging Viruses: Is Global Warming To Be Blamed?

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Abstract

The alarming rise of new emerging viruses and the re-emergence of previously known viruses is a major global health concern. Climate change, with its devastating consequences of biodiversity loss and habitat change, is disrupting ecosystems. This disarray creates prime conditions for the spillover of zoonotic viruses, exemplified by the recent COVID-19 pandemic. Melting permafrost further adds to the equation, potentially releasing ancient, unknown viruses trapped for millennia. Globalization facilitates the spread of these emergent viruses to new geographical areas where outbreaks have resulted in socio-economic crises. This review explores the rise of new emerging viruses with pandemic potential, focusing on the role of environmental change and human behavior in their emergence. We examine how these factors, including urbanization and increased human-wildlife interaction, are creating a breeding ground for novel viral threats. Recognizing the critical link between a changing environment and the rise of zoonotic infectious diseases is paramount for developing effective strategies to mitigate future pandemics.

1. Introduction

The Earth's climate is undergoing unprecedented changes driven by human activities, primarily the emission of greenhouse gases and the resulting global warming. This phenomenon manifests through rising temperatures, melting ice caps, sea-level rise, and altered weather patterns, impacting ecosystems, biodiversity, and ecological processes. Global warming isn't just upsetting the fragile harmony within environments; it may also be creating conditions that promote the rise and spread of infectious diseases, particularly viruses. The undeniable reality of anthropogenic climate change and its consequences for human health necessitates a deeper understanding of its influence on infectious diseases. Beyond the direct effects of global warming, such as heat stress and increased mortality in vulnerable populations, a concerning indirect consequence is the potential rise of novel and emerging infectious diseases (EIDs) [1].

Emerging infectious diseases (EIDs) are illnesses caused by pathogens that have recently increased in incidence, geographic range, or both, or which threaten to increase in the near future. These diseases present significant public health challenges globally due to their potential to cause widespread morbidity and mortality. Factors contributing to the emergence of these diseases include ecological changes like deforestation and urbanization, which modify the habitats of vectors and hosts; climate change, which can expand the geographic range of vectors; and human behaviors like increased international travel and trade, which facilitate the rapid spread of pathogens.

The evolution of pathogens through mechanisms like mutation and genetic recombination can lead to the development of new, more virulent strains. Biodiversity, under severe threat from various anthropogenic pressures including habitat loss, pollution, and climate change, plays a crucial role in maintaining ecological balance and preventing viral spillovers. However, disturbances in natural habitats can lead to the spread of viruses to new hosts, including humans, resulting in severe consequences ranging from localized outbreaks to global pandemics.

Viruses are ubiquitous in nature, existing in complex relationships with their hosts and the surrounding environment. Many viruses have co-evolved with specific host species, forming delicate balances within their natural reservoirs. These delicate interactions, however, can be upset by changes in environmental conditions brought about by habitat disturbances and global warming, which can open doors for viruses to spread to new hosts, including people. Viral spillover events can have severe consequences, ranging from localized outbreaks to global pandemics, impacting human health, agriculture, and economies.

Newly emerging viruses (NEVs) are viruses that have recently been identified and have the potential to cause significant public health concerns. The term "newly emerging virus" was first coined in the 1990s by the Institute of Medicine (IOM) in a report highlighting the growing threat of unknown viral pathogens [2]. These viruses often originate in animal reservoirs, and their emergence can be attributed to a complex interplay of factors, including human encroachment on wildlife habitats, changes in agricultural practices, and the global wildlife trade [3]. However, a concerning trend has emerged in recent decades, with growing evidence suggesting a potential link between global warming and an increased frequency of NEV outbreaks.

Climate change can alter ecological dynamics in several ways that may facilitate NEV emergence. Rising temperatures can expand the geographic range of insect vectors that transmit viruses, such as mosquitoes and ticks. Additionally, melting permafrost, a layer of permanently frozen ground in polar regions, can release previously frozen viruses that were trapped for millennia [4]. Furthermore, climate change can disrupt established ecological relationships, leading to increased interactions between humans and wildlife reservoirs of viruses, thereby increasing the spillover potential.

The recent past has witnessed a surge in NEV outbreaks, highlighting the urgency of this issue. The 2013-2016 Ebola virus epidemic in West Africa, the 2014 emergence of the Middle East Respiratory Syndrome Coronavirus (MERS-CoV), and the global pandemic caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) are just a few stark examples. These events underscore the potential for rapid and devastating spread of NEVs in a globalized world.

In this article, we will explore the newly emerging viruses documented in recent years, examining their outbreaks and the intricate interplay with global warming as a significant contributing factor. The main goal of this discussion is to highlight why it's so important to understand link between climate change and evolution of viruses so as to effectively respond present and future threats against public health.

2. New Emerging Viruses

The concept of emerging infectious diseases has evolved significantly over time. French doctor Charles Anglada (1809–1878) discussed the variability of pathologies in his 1869 book on extinct and new diseases, noting that diseases follow each other, with new ones asserting their presence. Nobel laureate Charles Nicolle further elaborated on this in his 1930 book, discussing the historical existence and future potential of infectious diseases.

The term "emerging disease" began appearing in scientific literature in the 1960s. David Sencer's 1971 article defined them as infectious diseases currently emerging as public health problems. The AIDS

epidemic in the 1980s spurred increased interest and action, with Mirko Grmek refining the concept of emerging diseases in his work on AIDS . In 1989, the NIAID/NIH conference on Emerging Viruses highlighted human behavior and zoonotic sources as key factors in viral emergence. Stephen S. Morse noted the opposite expectation of infectious diseases retreating, a view disrupted by the AIDS crisis. Following this, the Institute of Medicine's 1991 committee recommended surveillance programs and intervention strategies, leading to the formation of ProMED in 1994 and the launch of the CDC's Emerging Infectious Diseases journal in 1995.

Emerging viruses shouldn't be confused with re-emerging viruses. Emerging viruses are entirely new viruses that have not previously been identified in humans. They often jump the species barrier from animals, like the recent case of Nipah virus transmission from fruit bats to humans in Malaysia [5]. These viruses can be highly unpredictable as scientists have no prior knowledge of their behavior or potential for causing illness.

Re-emerging viruses were once known to cause illness in humans but have either declined significantly or disappeared entirely. However, they re-emerge due to various factors, such as changes in human behavior, mutations in the virus itself, or weakened immune systems in the population [6]. A recent example is measles, which has seen a resurgence in some parts of the world due to declining vaccination rates.

New vs. Re-emerging Viruses		
Feature	New/Emerging Viruses	Re-emerging Viruses
Definition	Viruses that are newly discovered and have not caused human illness before.	Viruses that were previously known but are experiencing a significant increase in incidence or geographic range.
Timeframe	Recently discovered, often within the past few decades.	May have been known for decades or even centuries.
Cause of increase	Often due to pen_spark zoonosis (jumping from animals to humans) or environmental changes.	Can be due to factors like: - Changes in human behavior (travel, hygiene) - Antibiotic resistance - Mutations in the virus itself
Examples	- SARS-CoV-2 (COVID-19) - Nipah virus - Ebola virus	- Measles - Dengue virus - HIV/AIDS (initially)
Public Health Impact	Often pose a significant challenge due to lack of existing immunity and treatments.	Can still be a major public health threat, especially if immunity wanes in the population.

Table 1. Distinction between newly emerging Vs. re-emerging viruses.

2.1 SARS-CoV-2

SARS-CoV-2, the causative agent of COVID-19, is an emergent virus that belongs to the coronavirus family, which includes other notable viruses such as SARS-CoV and MERS-CoV. Coronaviruses are characterized by their crown-like spike (S) proteins on the virus surface, a positive-sense single-stranded RNA genome, and an envelope structure. The virus's genome encodes several structural and non-structural proteins, facilitating its replication and pathogenicity [7].

Transmission of SARS-CoV-2 occurs primarily through respiratory droplets, with potential for airborne spread in certain conditions. The virus can also be transmitted via contact with contaminated surfaces, followed by touching the face [8]. Pathogenesis involves the virus binding to the angiotensin-converting enzyme 2 (ACE2) receptor, leading to viral entry into host cells, particularly in the respiratory tract, causing symptoms ranging from mild respiratory illness to severe pneumonia and acute respiratory distress syndrome (ARDS) [9].

The first major outbreak of SARS-CoV-2 was identified in Wuhan, China, in December 2019, rapidly escalating to a global pandemic by March 2020. The pandemic was characterized by widespread transmission, significant morbidity, and mortality, driven by factors such as high transmissibility, global travel, and delayed implementation of containment measures. Major outbreaks occurred worldwide, with significant impact in countries like the United States, Italy, Brazil, and India [10].

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) emerged through a probable zoonotic spillover event, with horseshoe bats identified as the primary reservoir based on genetic analysis. The role of intermediate hosts in facilitating transmission to humans remains under investigation, with pangolins being a candidate based on viral sequence homology [11]. Anthropogenic activities such as wildlife trade and habitat destruction are well-documented drivers of increased human-animal interactions, potentially influencing the emergence of SARS-CoV-2.

2.2 Ebola Virus

The emergence and re-emergence of Ebola virus disease (EVD) highlight the ongoing challenge of zoonotic viruses with high pathogenic potential. First identified in 1976 during outbreaks in what is now the Democratic Republic of the Congo (DRC) and Sudan, Ebola virus is a member of the Filoviridae family, characterized by its filamentous structure and negative-sense single-stranded RNA genome [12]. The virus primarily transmits through direct contact with bodily fluids of infected individuals or contaminated surfaces, leading to severe hemorrhagic fever with high mortality rates. Major outbreaks include the first recorded outbreak in 1976 in Sudan and the Democratic Republic of Congo, the 1995 outbreak in Kikwit, and the unprecedented 2013-2016 West African outbreak, which resulted in over 11,000 deaths [13].

The high mortality rate of Ebola virus disease (EVD), often exceeding 50%, is attributed to the virus's rapid replication and the body's intense inflammatory response, leading to multi-organ failure and severe haemorrhaging. The primary reservoir for the Ebola virus is believed to be fruit bats, with transmission to humans occurring through contact with infected wildlife, including primates and other mammals consumed as bushmeat [14].

Numerous variables, including poor healthcare infrastructure, significant population movement, crowded urban surroundings, and delayed discovery and response, contributed to the epidemic's global character, as demonstrated by the events of 2013–2016. To highlight the threat that EVD poses on a worldwide scale, the World Health Organization (WHO) designated it an international public health emergency.

There is still much to learn about the relationship between Ebola virus emergence and global warming. Increased interactions between humans and animal reservoirs may result from ecosystems being altered by climate change. More chances of zoonotic spillover events could arise from changes in wildlife distribution and behavior brought on by climate changes and habitat loss.

2.3 Crimean-Congo Hemorrhagic Fever (CCHF) Virus

Crimean-Congo Hemorrhagic Fever (CCHF) virus is a zoonotic pathogen causing severe viral hemorrhagic fever in humans. The World Health Organization (WHO) lists CCHF as a high-priority disease with the potential for epidemics. Crimean-Congo Hemorrhagic Fever (CCHF) virus, first recognized in 1944 in the West Crimean region, belongs to the genus *Orthonairovirus* within the family *Nairoviridae*. The virus has been detected in nearly 30 countries across Africa, Southeastern Europe, the Middle East, and Western Asia.

CCHF is endemic in Africa, the Balkans, the Middle East, and Asia, with the geographical range limited by the distribution of its primary tick vectors of the genus *Hyalomma*. Wild and domestic animals, particularly ruminants like sheep, goats, and cattle, act as asymptomatic reservoirs for the virus, shedding it in their

blood and secretions. Humans can become infected through tick bites, direct contact with infected animal blood or tissues during slaughter or nosocomial transmission in healthcare settings. The case fatality rate for CCHF varies from 10% to 40%, with a higher mortality rate observed in patients who lack access to prompt medical care [15].

CCHF outbreaks have been reported sporadically since its discovery, with notable clusters occurring in regions with high tick populations and extensive livestock farming. The first detection in India was in 2011, with subsequent outbreaks in Gujarat and Rajasthan [16]. The clinical course of CCHF includes an incubation period of 1-13 days, followed by phases characterized by high fever, severe headache, nausea, vomiting, diarrhea, and hemorrhagic symptoms. Diagnosis relies on molecular assays such as RT-PCR and serological tests like ELISA to detect specific antibodies.

CCHF poses a significant threat due to its hemorrhagic nature and the possibility of nosocomial transmission. Hemorrhagic fever symptoms like bleeding from various orifices can lead to rapid blood loss and organ failure. The lack of a specific antiviral treatment and the absence of a commercially available human vaccine further complicate the management of CCHF outbreaks [17].

Global warming, climate change, biodiversity loss, and habitat disturbance are emerging as potential factors influencing CCHF transmission dynamics. Rising temperatures and changes in precipitation patterns could expand the geographical range of *Hyalomma* ticks, potentially bringing them into closer contact with human settlements and livestock [3]. Warmer weather might extend the tick season, leading to a longer period of potential human exposure to infected ticks. Habitat loss and fragmentation can force wildlife species closer to livestock grazing areas, creating more opportunities for tick transmission between wild and domestic animals, potentially acting as a bridge to human infection [18].

2.4 Kyasanur Forest Disease Virus (KFDV)

Kyasanur Forest Disease Virus (KFDV), identified in 1957 in India, belongs to the genus *Flavivirus* within the family *Flaviviridae*. KFDV primarily spreads through the bite of infected hard ticks, particularly *Haemaphysalis spinigera*. Sylvatic monkeys like langurs serve as the natural reservoir for the virus, with ticks feeding on infected monkeys becoming the primary vectors for transmission to humans. The virus cycles between ticks and small mammals, with monkeys acting as amplifying hosts. Human infections occur through tick bites or contact with infected animals. KFDV has been endemic in the Karnataka state but has spread to other regions like Kerala, Tamil Nadu, Goa, and Maharashtra since 2012 [19].

KFDV causes a severe febrile illness with an incubation period of 3-8 days. Symptoms include high fever, headache, body ache, and occasionally, central nervous system involvement such as tremors and psychological disturbances. The case-fatality rate (CFR) ranges from 3-5%, with prolonged convalescence in some cases. Diagnosis is based on RT-PCR and ELISA tests for detecting viral RNA and specific antibodies. A formalin-inactivated vaccine is available, but no specific antiviral treatment exists [20].

Climate change may exacerbate KFDV spread by affecting tick distribution and density, thereby increasing human exposure. Habitat changes and increased interactions between humans and wildlife due to deforestation also contribute to the risk of spillover events.

2.5 Rift Valley Fever (RVF) Virus

Rift Valley Fever (RVF) virus, first identified in 1931 in Kenya, is a mosquito-borne, zoonotic pathogen belonging to the genus *Phlebovirus* within the family *Phenuiviridae*. It primarily infects domestic animals like cattle, sheep, and goats, causing significant economic losses due to abortions and deaths. However, RVF can also spill over to humans through mosquito bites or direct contact with infected animal blood or tissues

Major outbreaks have occurred in sub-Saharan Africa, with notable events in Kenya, Sudan, and South Africa.

The main mode of RVF transmission occurs through the bite of infected *Aedes* mosquitoes, particularly the species *Aedes vexans* and *Aedes cummins*. These mosquitoes acquire the virus by feeding on infected animals and subsequently transmit it to other animals or humans during subsequent blood meals. RVF also has a sylvatic cycle, where wild ruminants like buffalo and gazelle act as the primary reservoir hosts [21]. During heavy rainfall and flooding events, the virus can amplify in these wild ruminant populations and spill over to domestic animals through contact with contaminated water sources or pastures.

RVF presents in humans in mild and severe forms. Mild cases exhibit flu-like symptoms, while severe cases can progress to ocular disease, meningoencephalitis, or hemorrhagic fever, with a CFR of about 50% in hemorrhagic cases. Diagnosis involves RT-PCR and ELISA assays to detect viral RNA and antibodies. There is no specific vaccine or treatment for RVF, and management is largely supportive [22].

RVF outbreaks in India have been historically rare. However, in 2020, the first major outbreak was reported in Rajasthan and Gujarat, affecting both livestock and humans. This emergence highlights the potential for future outbreaks, particularly with changing environmental conditions [23].

Global warming may influence RVF emergence by expanding the range and breeding cycles of mosquito vectors, thereby increasing transmission rates. Flooding and heavy rainfall, associated with climate change, can create optimal breeding conditions for mosquitoes, facilitating outbreaks.

2.6 Yellow Fever (YF) Virus

Yellow fever is a mosquito-borne viral disease historically known for its devastating impact on human populations. Yellow fever has plagued humanity for centuries, with documented outbreaks impacting major historical events like the construction of the Panama Canal. Warfare was even influenced by yellow fever, as armies were often decimated by the disease [24].

Endemic in parts of Africa and South America, yellow fever is transmitted by *Aedes* and *Haemagogus* mosquitoes, with monkeys acting as the primary reservoir. The primary mosquito vectors, *Aedes aegypti* and *Haemagogus* spp., are known for their ability to thrive in urban and peri-urban environments, breeding in stagnant water sources like discarded containers. This close proximity to human settlements facilitates efficient transmission.

The virus is transmitted through the bite of infected mosquitoes. While most infected individuals experience mild illness, a significant portion (15-50%) develop severe illness with high fever, bleeding complications, and organ failure. The case fatality rate for severe cases can be as high as 50%. In 2017, India witnessed an outbreak of yellow fever, the first in decades, highlighting the potential for the virus to spread beyond its traditional geographical range.

Laboratory diagnosis of YF is conducted via RT-PCR and ELISA to detect viral RNA and antibodies, with PRNT used to address cross-reactivity with other flaviviruses. For those who develop severe illness, the case fatality rate is alarmingly high, highlighting the potential for significant mortality during outbreaks. There is no specific treatment for yellow fever, making supportive care and prevention paramount [25]. The spread of *Aedes* mosquitoes, the primary vectors, is facilitated by urbanization and deforestation, creating ideal breeding grounds near human populations.

Virus name	Outbreak frequency	Disease type	Case fatality rate	Reference
Ebola virus	Recurring	Haemorrhagic fever	25–90%	[26]
Crimean-Congo Haemorrhagic fever (CCHF)	Rare	Haemorrhagic fever	10-40%	[27]
Kyasanur Forest Disease virus (KFDV)	Very Rare	Haemorrhagic fever	3.4-5%	[28]
Rift Valley Fever Virus (RVF)	Very Rare	Haemorrhagic fever, meningoencephalitis	1%	[21]
Yellow fever Virus (YFV)	Very Rare	Haemorrhagic fever, Encephalopathy, coma	20% to 50%	[25]
Nipah Virus	Very Rare	Acute Respiratory illness	100%	[29]
Monkeypox Virus	Rare	Fever, muscle pains, swollen lymph nodes	3-8%	[30]
Dabie banda virus	Rare	Thrombocytopenia, multiple organ failure	12-30%	[31]
Marburg virus disease	Rare	Haemorrhagic fever, petechiae or purpura, hematoma, MODS	23–90%	[32]
Influenza Virus A subtype H7N9	Rare-Recurring	Severe respiratory illness	30%	[33].
Influenza Virus A subtype H5N1	Rare-Recurring	Severe respiratory illness	50%	[34]
Adenovirus serotype 14	Rare	Pharyngitis, Diarrhoea	>50%	[35], [36]

Table 2. A Comprehensive List Of New Emerging Viruses

2.7 Nipah Virus

Nipah virus (NiV), belonging to the genus Henipavirus within the Paramyxoviridae family, poses a significant threat as an emerging zoonotic disease with pandemic potential. First identified during outbreaks in Malaysia and Singapore (1998-1999), NiV is a highly pathogenic RNA virus with a mortality rate ranging from 40% to 75% depending on the outbreak strain [37].

The initial outbreak in Malaysia was attributed to close contact with infected pigs, highlighting the virus's ability to spill over from its natural reservoir, fruit bats, to domestic animals [38]. However, subsequent outbreaks in Bangladesh and India (2001 onwards) revealed a concerning shift in transmission dynamics. These outbreaks were characterized by human-to-human transmission and nosocomial infections, likely due to respiratory droplets and contaminated fomites [39]. This human-to-human transmission, coupled with a higher secondary attack rate compared to the Malaysian outbreak, suggests potential for broader community spread.

Furthermore, the association of NiV disease with consumption of contaminated date palm sap by fruit bats raises concerns about environmental factors influencing transmission. The virus's stability in date palm sap, along with its tolerance for a wide range of pH levels, suggests potential for indirect transmission through contaminated food and beverages [40]. This environmental dimension adds another layer of complexity to control efforts.

Genetic analysis suggests strain variation might play a role in the observed differences in transmission patterns. Studies comparing the Malaysian (MY) and Bangladesh (BD) strains indicate higher viral shedding and prolonged viremia in the BD strain, potentially contributing to the increased human-to-human transmission observed in the Indo-Bangladesh outbreaks.

The most recent outbreak in Kerala, India, occurred in September 2023. While limited in scale with six confirmed cases and two fatalities, it highlights the ongoing threat posed by NiV and the need for continued vigilance [41]. The Nipah virus poses a complex problem. Its capacity to move from bats to household animals, along with the possibility of transmission from person to person and environmental contamination, emphasizes the possibility of a pandemic.

2.8 Monkeypox Virus

Monkeypox virus (MPXV), an Orthopoxvirus within the Poxviridae family, has re-emerged as a global health concern. While historically endemic to Central and West Africa, recent outbreaks, particularly in 2022 involving the MSM population, highlight its potential for broader human-to-human transmission and pandemic spread. This essay explores the scientific basis for this concern [42].

MPXV is a zoonotic virus with a natural reservoir suspected to be African rodents. Transmission to humans can occur through contact with infected animals or their bodily fluids, or indirectly via contaminated fomites. Notably, the ongoing outbreak suggests a shift in transmission dynamics, with the predominant mode being sexual contact, particularly among men who have sex with men (MSM) [43]. This highlights the virus's ability to adapt to new transmission routes, potentially increasing its transmissibility.

The clinical presentation of MPXV infection is characterized by an initial prodromal phase with fever, headache, and lymphadenopathy, followed by a characteristic rash progressing through maculopapular, vesicular, pustular, and crusting stages. This presentation can be confused with other vesicular diseases like varicella-zoster or chickenpox, particularly in non-endemic regions. However, the presence of lymphadenopathy is a key differentiating factor for MPXV [44]. While there is no specific treatment for MPXV, supportive care and management of symptoms are crucial. However, the potential for severe complications, including encephalitis, pneumonia, and bacterial sepsis, underscores the need for effective control measures.

The ongoing outbreak has reignited interest in the role of vaccination for MPXV prevention. Historically, widespread vaccination against smallpox provided some degree of cross-protection against MPXV. However, the eradication of smallpox and subsequent cessation of routine vaccination have left populations vulnerable. Fortunately, the replication-deficient Modified Vaccinia Ankara (MVA) vaccine, originally developed for smallpox, has been shown to be safe and effective against MPXV in animal models and clinical trials [45].

2.9 Dabie bandavirus/ SFTS virus

During 2006-2007, several patients in China exhibited symptoms such as fever, stomach ache, gastrointestinal disorders, nausea, bloating, vomiting, and thrombocytopenia, leading to the identification of Severe Fever with Thrombocytopenia Syndrome (SFTS) in Shaanxi province. The causative agent, the SFTS virus (SFTSV), is a phlebovirus from the family Bunyaviridae. Initially mistaken for *Anaplasma phagocytophilum* bacterium, SFTSV was isolated from a patient in 2009. The virus's genome comprises

large (L), medium (M), and small (S) segments encoding the RNA-dependent RNA polymerase, glycoproteins Gn and Gc, and nucleocapsid and non-structural proteins, respectively [31].

SFTSV is zoonotically transmitted via ticks, though human-to-human transmission has also been reported. From 2013 to 2016, China confirmed 7,419 cases with 355 deaths, with cases also reported in Japan, South Korea, the USA, and Taiwan [46]. The disease's pathogenesis remains not fully understood, and no specific treatment or vaccine exists. Preventive measures focus on avoiding tick bites and using protective devices. Ribavirin's effectiveness is noted for similar viral infections but remains unproven for SFTS [47].

SFTS has become a significant health concern due to its high mortality rate and potential for human-to-human transmission. Current research emphasizes the need for improved understanding and development of therapeutics and vaccines. The fast mutation rate of SFTSV in tick vectors and animal hosts poses an ongoing threat to global health, underscoring the importance of continued epidemiological and vaccine research [48].

2.10 Marburg Virus

First identified in 1967 during outbreaks in Marburg, Germany, and Frankfurt, Germany, and Belgrade, Serbia, all linked to handling African green monkeys imported from Uganda, Marburg virus (MARV) poses a significant threat as a potential pandemic agent. Classified as a filovirus within the Filoviridae family, its single-stranded, negative-sense RNA genome shares significant similarities with the notorious Ebola virus [49].

A particularly concerning development was the 2007 co-circulation outbreak of MARV and Ravn virus (RAVV), another filovirus, in the Democratic Republic of Congo. This co-infection event raises concerns about potential immune evasion strategies employed by these viruses [50]. Filoviruses are known for their ability to suppress the host's innate immune response, hindering the body's initial defenses. Additionally, their ability to exploit antigen presentation pathways allows them to evade adaptive immunity, making them difficult for the immune system to target effectively [51].

Fruit bats of the Pteropodidae family are considered the natural reservoir for MARV. Transmission to humans primarily occurs through direct contact with bodily fluids of infected individuals or animals. Due to its high fatality rate, ranging from 23% to 90% depending on the outbreak strain, MARV is classified as a Biosafety Level 4 (BSL-4) agent, requiring the highest level of biocontainment for research.

Currently, there are no licensed vaccines or specific treatments for MARV infection. However, research efforts are underway to develop vaccines and antiviral therapeutics. One promising avenue involves a multivalent vaccine targeting both MARV and Ebola virus, leveraging immunological similarities between the two filoviruses [52].

The recent 2023 outbreaks in Equatorial Guinea and Tanzania highlight the ongoing threat posed by MARV. The swift response by the World Health Organization (WHO) and international health agencies demonstrates the global commitment to preparedness and containment. However, the lack of readily available vaccines and therapeutics underscores the urgency for continued research and development efforts.

MARV's high pathogenicity, potential for immune evasion, and lack of specific countermeasures warrant its classification as a potential pandemic threat. Continued research on diagnostics, vaccines, and antiviral therapies is crucial to mitigate the risk of a future MARV pandemic.

2.12 Influenza A virus subtype H7N9

The emergence of Influenza A virus subtype H7N9 (H7N9) in 2013 sent shivers down the spines of public health experts. While not currently efficient in human-to-human transmission, H7N9 possesses several worrying characteristics that, coupled with global warming, could create a "perfect storm" for a future pandemic [53].

One key concern lies in the viral receptor binding properties of H7N9's hemagglutinin (HA) protein. Unlike H5N1, which binds preferentially to avian receptors, H7N9 exhibits a concerning ability to bind to human-like receptors in the lower respiratory tract. Studies suggest these binding properties may have arisen from mutations in the HA gene, potentially acquired from ducks or wild birds through reassortment events. This ability to bind human receptors signifies a crucial step towards efficient human-to-human transmission, a hallmark of pandemics [54].

Global warming adds another layer of complexity. Rising temperatures are predicted to alter migratory patterns of birds, potentially increasing the geographic overlap between H7N9-infected birds and human populations. This increased interaction could provide more opportunities for the virus to jump the species barrier and establish itself in humans. Warmer temperatures might extend the influenza season by altering environmental factors that influence viral survival. A longer seasonal window translates to more opportunities for human exposure and potential viral evolution.

Furthermore, some research suggests that warmer temperatures might influence the virulence of influenza viruses. A warming climate could potentially favor the emergence of more virulent H7N9 strains, posing a greater threat to public health. While the current H7N9 strain exhibits lower mortality compared to H5N1, its ability to infect humans and its potential for adaptation under a changing climate necessitate continued research. Developing effective vaccines and antiviral treatments alongside robust influenza surveillance programs are crucial steps in mitigating the risk of a future H7N9 pandemic [55].

2.13 Influenza A virus subtype H5N1

Influenza A virus subtype H5N1, abbreviated as A/H5N1, is a influenza A virus that causes influenza (commonly known as flu), mostly in birds. This specific type of virus can be seen in birds often and is enzootic (it remains within the population), but it also spreads across various species - this makes it panzootic [56]. The A/H5N1 virus can infect mammals too, including humans who have come into contact with infected birds; symptoms are usually serious or fatal in these instances. Highly Pathogenic Avian Influenza A (HPAI) subtype H5N1 is a cause for serious public health concern. Its high virulence in birds, documented spillover to humans, and ongoing mutations raise the specter of a future pandemic [57].

HPAI H5N1 exhibits high pathogenicity in birds, causing devastating outbreaks in poultry farms and wild bird populations worldwide [56]. This extensive presence in the avian reservoir creates a constant platform for mutations and potential spillover events to humans. Studies have shown that specific mutations in the hemagglutinin (HA) gene of H5N1 can significantly alter its ability to bind to human respiratory tract receptors, potentially increasing its transmissibility in humans [58].

Influenza A viruses, including H5N1, have a high mutation rate and a segmented genome. This allows for genetic reassortment with other influenza strains if co-infection occurs in a host. Such reassortment events could create novel variants with increased ability to transmit between humans, a key factor in pandemics [59], [60]. The 2009 H1N1 pandemic virus, for instance, emerged from a complex reassortment event involving avian, swine, and human influenza strains.

Climate change is predicted to alter bird migration patterns, potentially increasing the geographic overlap between H5N1-infected bird populations and human settlements. This increased contact could create more opportunities for spillover events from birds to humans. Warmer temperatures might extend the influenza season by altering environmental factors that influence viral survival. This could translate to a longer window for human exposure to H5N1 and potentially more opportunities for viral evolution within the human population.

2.15 Adenovirus serotype 14

Adenovirus serotype 14 (Ad14) poses a unique challenge due to its potential for severe illness and its emergence as a new variant. Unlike most adenoviruses, which cause mild respiratory infections, Ad14 has been linked to outbreaks of severe pneumonia, with a high hospitalization rate and mortality in otherwise healthy individuals [61].

The concern surrounding Ad14 stems from its recent emergence and unique characteristics. Sequencing of isolates from geographically distinct outbreaks in the United States revealed a genetically identical strain, distinct from previously known Ad14 [61]. This suggests the emergence of a novel Ad14 variant with potentially altered virulence or transmission properties.

The ability to cause severe respiratory illness, including pneumonia, in healthy individuals raises concern for potential widespread morbidity and mortality during outbreaks [62]. The emergence of a new variant with unknown transmissibility characteristics necessitates close monitoring. Adenoviruses are typically transmitted through respiratory droplets and contact with contaminated surfaces. Globalization and increased population density could facilitate the spread of this emerging Ad14 variant.

Climate change could alter weather patterns and potentially influence the seasonality of respiratory viruses, although the specific impact on Ad14 is unknown. Climate change-driven disruptions to ecosystems could lead to unforeseen consequences for viral reservoirs or transmission patterns. The role of globalization in Ad14 spread is also uncertain, but increased travel and population mixing could theoretically facilitate the introduction and dissemination of novel viral strains. Hence, Adenovirus serotype 14 represents an emerging public health concern due to its potential for severe illness and the emergence of a novel variant.

3. Reasons For Emergence/Re-Emergence

Understanding the factors influencing viral emergence and re-emergence is crucial for mitigating the threat of infectious diseases.

3.1 The Intricacies of Infection Cycles

A pathogen's ability to establish itself within a vector and multiply there is a critical step in its transmission cycle. For diseases transmitted through respiratory droplets or direct contact (like influenza or tuberculosis), the cycle involves distinct stages: release of infectious particles from an infected person, their dispersal through air currents, and inhalation by a susceptible individual, leading to a new infection. This is less complex compared to pathogens that rely on various non-human species. A pathogen's biological characteristics can influence the difficulty of specific stages. For instance, a bacterium that thrives only inside host cells or survives briefly outside them would struggle to complete the cycle successfully.

Host-pathogen interactions further complicate certain stages. Some pathogens require specific receptors on host cells for successful entry. The absence or dysfunction of these receptors (due to genetic variations) can hinder the pathogen's entry phase. Evading the host's immune response is another hurdle. Many pathogens have developed immune evasion tactics over time. Without such strategies, diseases like smallpox might still be prevalent today. Eradication was largely possible because the virus lacked effective immune evasion, leading to repeated outbreaks until no susceptible individuals remained.

In essence, a complete infection cycle requires more than just a pathogen and susceptible hosts. It necessitates adaptation between species, considering ecological aspects alongside biological traits inherent to both pathogens and vectors involved in transmission.

3.2 Factors Contributing to Disease Outbreaks

Pathogens causing the most severe diseases are not particularly selective about their host's habitat, feeding behavior, or immune response. This aligns with the emergence of viruses like Ebola and Marburg, which can infect a variety of primates, including humans, increasing the potential for spillover events. Similarly, Nipah virus can jump from fruit bats to pigs and then to humans, demonstrating a lack of strict host preference. While Mpox primarily infects rodents, it can also infect humans, highlighting its adaptability across species. SARS-CoV-2, initially thought to originate in bats, likely infected humans through an intermediary animal host, showcasing its ability to establish a transmission cycle in a new species.

For instance, a study revealed that seven out of the ten diseases with the highest human impact had increased in occurrence during the analyzed period. A pathogen's capacity for productive infection within a host is a key factor influencing transmission. Mosquitoes, known vectors for diseases like malaria and dengue fever, have been extensively studied. Translocation (accidental transport of mosquitoes by humans or objects) and dispersal (natural mosquito movement) are two main modes of mosquito spread [63]. Before 1950, only two mosquito species caused significant arbovirus outbreaks: *Aedes aegypti* transmitted yellow fever virus, while *Culex fatigans* spread West Nile virus (WNV). Since then, both these species have become globally invasive, along with others like *Ae. albopictus*. This expansion is attributed to human activities like trade, which unintentionally introduces mosquito eggs (laid on tires and plants) into new locations where they hatch and establish populations [64].

Most disease-causing pathogens exhibit limited host preference, infecting domesticated livestock and wild game with similar ease. This confirms that while specific characteristics might enhance transmission opportunities within certain animal groups, the overall capacity for infection remains widespread across various potential hosts globally. Notably, *Ae. albopictus*, responsible for transmitting numerous viruses like yellow fever and four serotypes of dengue, was historically transported by early seafarers from Africa to tropical Asia and America. The intricate dynamics between viruses and mosquitoes showcase the importance of considering regional specificities. During the 1960s, campaigns to eliminate yellow fever and malaria nearly eradicated *Aedes* mosquitoes from the Americas. However, *Ae. aegypti* has since rebounded [65].

Understanding the historical prevalence of specific viruses in certain regions and the presence of potential animal reservoirs can inform public health preparedness and response strategies. For instance, the past outbreaks of Yellow fever and Rift Valley fever in Africa inform surveillance efforts for these diseases in the region.

3.3 The Influence of Coupled Ocean-Atmosphere Phenomena

Growing evidence suggests that, along with ENSO events, the Quasi-Biennial Oscillation (QBO) and the Indian Ocean Dipole (IOD) also impact weather patterns in tropical and subtropical zones, potentially influencing the emergence and progression of epidemics and enzootics (animal diseases). The Pacific Decadal Oscillation (PDO) seems to affect not only Pacific Ocean sea surface temperatures (SSTs) but also temperatures in America, Africa, and Asia. During its warm phase, El Niño events become more frequent, while La Niña events dominate its cold phase. Diseases potentially linked to ENSO include cholera, plague, malaria, hantavirus pulmonary syndrome, and possibly dengue fever in some Polynesian islands [66].

The Indian Ocean Dipole can sometimes influence how ENSO events affect the Indian Ocean. This was evident during a period with strong positive SST anomalies in the western Indian Ocean and negative SSTs in the eastern Indian Ocean, causing heavy rainfall in East Africa while Indonesia experienced a more severe drought than usual. The following year, the pattern reversed, leading to heavy precipitation in Southeast Asia. This interplay resulted in a devastating Rift Valley fever epidemic alongside outbreaks of cholera and plague in East Africa during 1997. The subsequent year saw an outbreak of dengue fever, Japanese

encephalitis, Nipah encephalitis, and enterovirus 71 encephalitis in Southeast Asia. It's possible that a similar severe dipole event, independent of ENSO, contributed to the 1961-1963 Rift Valley fever epidemics in East Africa [67].

3.4 Virus Spillover

As global warming continues to exacerbate these pressures on biodiversity, the delicate balance of ecosystems is being disrupted, potentially creating opportunities for viruses to spillover from their natural reservoirs into new hosts. This underscores the urgent need for concerted efforts to mitigate climate change, conserve biodiversity, and maintain the integrity of ecological systems to prevent potential viral spillover events and their associated consequences.

Viruses inhabit diverse ecosystems, from rainforests to oceans. Wildlife, particularly bats and rodents, serve as crucial reservoirs for various viruses. These animals harbor viruses without showing symptoms, allowing them to persist and evolve. Viral diversity within these reservoirs is immense, with frequent recombination and adaptation. Factors like habitat changes, host behavior, and viral traits influence spillover risk.

Bats and rodents, in particular, have drawn attention due to their unique immune systems and natural history features. Recent research in East Africa has shed light on this diversity. Using meta transcriptomic data from 844 bats and 250 rodents, scientists identified 251 vertebrate-associated viral genomes. These viruses showed host-related variability, high geographic specificity, and frequent recombination. Notably, the Coronaviridae and Circoviridae families displayed low host specificity and high divergence among viral entry proteins. Despite dispersal limitations, recurrent mutations and occasional gene flow contribute to the local diversity of viral genome [68].

While most viruses are host-specific, the rapid evolution of viruses and increased contact between wildlife and humans or domestic animals have led to the emergence of zoonotic pathogens. Understanding how viruses evolve and transmit within and among wildlife species is crucial for proactive pathogen surveillance. Recombination, mutation, and gene flow contribute to the genetic plasticity of viral communities, enabling host adaptation and maintenance.

3.5 Mechanisms of Viral Spillover

Viral spillover refers to the transmission of pathogens from animals to humans. While the exact process remains intricate, several factors contribute to this phenomenon. First, viruses must adapt to the host's cells and tissues, allowing successful replication. Second, the virulence and pathogenic characteristics of the virus influence its ability to cross species barriers. Third, adaptation and evolution within a novel host environment play a crucial role. Fourth, steric hindrance where viral proteins interfere with host cell factors affects transmission. Understanding these mechanisms is essential for preventing zoonotic outbreaks and developing effective interventions.

3.5.1 Climate-Induced Changes in Host Behavior and Distribution

Climate change significantly impacts wildlife behavior and distribution. As temperatures shift, species alter their ranges, migration patterns, and activity times. For instance, warmer temperatures may lead to earlier breeding seasons or changes in feeding habits. These shifts can affect interactions between wildlife and humans. For example, altered migration routes could bring species into closer proximity to human settlements, increasing the risk of zoonotic spillover.

3.5.2 Increased Human-Wildlife Interface and Habitat Encroachment

As human populations expand, we encroach upon natural habitats, leading to greater overlap with wildlife. Urbanization, deforestation, and agricultural expansion reduce available habitat for animals, forcing them into closer contact with humans. This proximity increases the chances of disease transmission. For instance, bats roosting in urban areas may shed viruses near human dwellings. Managing this interface—through wildlife corridors, protected areas, and sustainable land use—is essential to minimize spillover risks.

3.5.3 Emergence of Novel Vectors or Intermediate Hosts

Climate change can alter the distribution and behavior of vectors (such as mosquitoes and ticks) and intermediate hosts (organisms that transmit pathogens between reservoir hosts and humans). Warmer temperatures may expand the range of disease-carrying vectors, exposing new populations to infections. Additionally, changes in host availability can impact transmission cycles. Identifying and monitoring these novel vectors and hosts is critical for early detection and prevention of zoonotic outbreaks.

3.6. Consequences of Viral Spillover

Viral spillover events pose a significant risk of zoonotic disease emergence. When viruses jump from animals to humans, they can cause outbreaks with devastating consequences. Examples include Ebola, HIV, and SARS-CoV-2. These zoonotic diseases often originate from wildlife reservoirs. Understanding the factors that drive spillover—such as ecological changes, host behavior, and viral adaptation—is crucial for early detection and prevention. Surveillance, research, and public health preparedness are essential to mitigate this risk.

The direct impact of zoonotic diseases on human health is evident. Outbreaks can lead to illness, hospitalizations, and fatalities. Beyond health, these events disrupt economies. Travel restrictions, trade disruptions, and reduced workforce productivity affect global markets. For instance, the COVID-19 pandemic caused widespread economic losses. Investing in surveillance, rapid response systems, and healthcare infrastructure can mitigate these impacts.

Viral spillover affects ecosystems and their services. When wildlife populations decline due to disease outbreaks, ecosystem dynamics change. For example:

- o **Pollination:** Loss of pollinators impacts crop yields.
- o **Predator-Prey Relationships:** Disruptions affect species interactions.
- o **Nutrient Cycling:** Decomposers play a vital role in nutrient cycling.
- o **Biodiversity:** Reduced biodiversity affects ecosystem resilience.
- o **Tourism and Recreation:** Disease-related fears impact ecotourism. Protecting ecosystems and maintaining their services is essential for human well-being and planetary health.

4. Future Perspectives and Research Directions

As we continue to face the challenge of viral spillover and disease outbreaks, it's crucial to look forward and consider future research directions. This could involve studying the underlying mechanisms of viral spillover, improving our understanding of virus-host interactions, and developing new strategies for disease prediction and control. Additionally, research into the societal and economic impacts of outbreaks can help inform policy and public health responses.

To effectively manage the risk of viral spillover, it's essential to take a holistic approach that considers the complex interplay of factors. This includes integrating data from ecology, climate science, and public health. By understanding how changes in the environment and human behavior can influence the emergence and spread of diseases, we can develop more effective strategies for prevention and control. This could involve using advanced data analysis techniques, such as machine learning, to identify patterns and trends.

Early detection of potential outbreaks and rapid response can significantly reduce the impact of diseases. This could involve developing advanced surveillance systems that use a variety of data sources, such as satellite imagery, social media, and medical records, to identify signs of unusual disease activity. Once a potential outbreak is detected, rapid response strategies could be deployed, such as targeted vaccination campaigns, quarantines, and public health messaging.

The threat of viral spillover and disease outbreaks is a global issue, and requires a global response. This means strengthening international collaboration and information sharing. This could involve creating global networks of researchers, public health officials, and other stakeholders, who can work together to share data, expertise, and resources. It could also involve developing international guidelines and standards for disease surveillance, response, and control.

Conclusion

The emergence of novel viruses and the re-emergence of previously known viruses present a complex and multifaceted challenge to global public health. While the singular attribution of blame solely to global warming would be an oversimplification, it undeniably plays a significant role. Climate change acts as a potent driver of ecosystem disruption, fostering conditions conducive to viral spillover events from animal reservoirs to human populations. Melting permafrost harbors the potential to unleash ancient, previously unknown pathogens, further complicating the equation.

While attributing blame solely to global warming for the rise of new emerging viruses would be an oversimplification, it undeniably plays a significant role. Globalization, with its increased human mobility and interconnectedness, acts as a potent force in amplifying the geographic spread of emergent viruses, potentially sparking pandemics. Additionally, factors like urbanization and associated human encroachment on wildlife habitats further exacerbate the risk of zoonotic transmission.

To go on, we need to use many tactics. We must keep studying new viruses and how they might spread. Reducing climate change and related changes in environment is also crucial for preventing future outbreaks. Sustainable methods, safeguarding biodiversity, and lessening human-wildlife contact are all very important. Recognizing the interconnectedness of environmental health and human health, we can develop effective strategies to combat the rise of new emerging viruses and safeguard our future from devastating pandemics.

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