Research
Coronavirus Disease 2019—Review

Ecological Barrier Deterioration Driven by Human Activities Poses Fatal Threats to Public Health due to Emerging Infectious Diseases

Dayi Zhang [a], Yunfeng Yang [a], Miao Li [a], Yun Lu [a], Yi Liu [a,*], Jingkun Jiang [a], Ruiping Liu [a], Jianguo Liu [a], Xia Huang [a], Guanghe Li [a], Jiuhui Qu [a,b,*]

[a] *School of Environment, Tsinghua University, Beijing 100084, China*
[b] *Key Laboratory of Drinking Water Science and Technology, Research Center for Eco-Environmental Sciences, Chinese Academy of Sciences, Beijing 100085, China*

A B S T R A C T

The coronavirus disease 2019 (COVID-19) and concerns about several other pandemics in the 21st century have attracted extensive global attention. These emerging infectious diseases threaten global public health and raise urgent studies on unraveling the underlying mechanisms of their transmission from animals to humans. Although numerous works have intensively discussed the cross-species and endemic barriers to the occurrence and spread of emerging infectious diseases, both types of barriers play synergistic roles in wildlife habitats. Thus far, there is still a lack of a complete understanding of viral diffusion, migration, and transmission in ecosystems from a macro perspective. In this review, we conceptualize the ecological barrier that represents the combined effects of cross-species and endemic barriers for either the natural or intermediate hosts of viruses. We comprehensively discuss the key influential factors affecting the ecological barrier against viral transmission from virus hosts in their natural habitats into human society, including transmission routes, contact probability, contact frequency, and viral characteristics. Considering the significant impacts of human activities and global industrialization on the strength of the ecological barrier, ecological barrier deterioration driven by human activities is critically analyzed for potential mechanisms. Global climate change can trigger and expand the range of emerging infectious diseases, and human disturbances promote higher contact frequency and greater transmission possibility. In addition, globalization drives more transmission routes and produces new high-risk regions in city areas. This review aims to provide a new concept for and comprehensive evidence of the ecological barrier blocking the transmission and spread of emerging infectious diseases. It also offers new insights into potential strategies to protect the ecological barrier and reduce the wide-ranging risks of emerging infectious diseases to public health.

© 2020 THE AUTHORS. Published by Elsevier LTD on behalf of Chinese Academy of Engineering and Higher Education Press Limited Company. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Background

Since 1970, over 1500 pathogens have been identified and isolated, 70% of which come from animals. The World Health Organization (WHO) has listed 15 pathogens as global threats causing infectious diseases [1–4]. In recent decades, numerous viruses such as Ebola, hydrophobia, avian influenza, dengue, Zika, and human immunodeficency virus (HIV) have infected over 1 billion people and killed 80 million, and their area of influence and the populations affected by them are increasing (Table 1)

[5–54]. Important cases include: West Nile River disease, which has infected 4161 people and caused at least 277 deaths [23,55]; severe acute respiratory syndrome coronavirus (SARS-CoV), which infected 8422 people and caused 919 deaths in 2003 [43,56]; and Middle East respiratory syndrome coronavirus (MERS-CoV), which has infected 701 people and caused 249 deaths since 2012 [51]. In particular, the coronavirus disease 2019 (COVID-19), caused by the novel severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has infected over 43 million people and caused over 1.1 million deaths as of 25 October 2020 [57], raising extensive attention from both scientific and social communities.

The increasing frequency of emerging infectious diseases has raised the question of how viruses can spread from natural hosts in their wildlife habitats to human societies. As events that

Table 1
Data on the first record of viral epidemics or pandemics.

Virus	Type	First record	Epidemic area	Infected cases	Death	Mortality	References
Dengue virus	Flavivirus, enveloped, single-stranded ribonucleic acid (ssRNA)	16th century	Global	~390 million·a^{-1}	Unknown	1%–5%	[5–8]
Hanta virus	Bunyaviridae, enveloped, ssRNA	1913, Soviet Union	Global	> 1 000 000	Unknown	1%–60%	[9–11]
Spanish flu, H1N1 pandemic (pdm)	Orthomyxovirus, enveloped, ssRNA	1918, United States	Global	~500 to 1000 million	~25 to 50 million	0.1%–5%	[12–14]
West Nile virus	Flavivirus, enveloped, ssRNA	1937, Uganda	Global	> 57 354	>2447	3%–15%	[15–20]
Zika virus	Flavivirus, enveloped, ssRNA	1947, Uganda	Global	> 200 000	Unknown	Unknown	[21,22]
Chikungunya virus	Alphaviruses, ssRNA	1952, Tanzania	Global	> 1.5 million	Unknown	< 1%	[23–26]
Kyasanur forest disease virus	Flavivirus, enveloped, ssRNA	1957, India	India	3 263	Unknown	2%–10%	[27–29]
Marburg virus	Filovirus, enveloped, ssRNA	1967, Germany	Africa	587	475	24%–88%	[30,31]
Ebola virus	Filovirus, enveloped, ssRNA	1976, South Sudan	Africa	31 161	12 999	20%–90%	[32–34]
Hendra virus	Paramyxoviruses, enveloped, ssRNA	1994, Australia	Australia	7	3	30%–60%	[35–37]
H5N1	Orthomyxovirus, enveloped, ssRNA	1997, Hong Kong (China)	Hong Kong (China), Thailand	650	386	53%	[38–40]
Nipah virus	Paramyxoviruses, enveloped, ssRNA	1998, Malaysia	Southeast Asia	477	248	52%	[35,41,42]
SARS-CoV	Coronavirus, enveloped, ssRNA	2002, China	32 countries	8 439	812	9.6%	[43–45]
H1N1 pdm	Orthomyxovirus, enveloped, ssRNA	2009, Mexico	Global	0.7 billion–1.4 billion	18 449 (confirmed); 201 200 (estimated)	0.01%	[46–50]
MERS-CoV	Coronavirus, enveloped, ssRNA	2012, Saudi Arabia	27 countries	815	313	38.4%	[51]
H7N9	Orthomyxovirus, enveloped, ssRNA	2013, China	China	1 568	616	39%	[40,52]
SARS-CoV-2	Coronavirus, enveloped, ssRNA	2019, China	Global	> 40 million	> 1 million	~3%	[53,54]

SARS-CoV: severe acute respiratory syndrome coronavirus.

hypothetically correlate with viral transmission across both species [58] and physical space, pandemic outbreaks are predominantly linked to the relationship between the natural environment and humanity. Human activities have caused increasing environmental problems around the world, including intensive contamination [59], global warming [60], frequent natural disasters [61], destruction of wildlife habitats [62], loss of biodiversity [63], and so forth. These challenges have significantly altered the global ecosystem and thereby shaped the behavior and habits of wildlife, including natural pathogens and viruses to some extent [64,65], consequently influencing the emergence and distribution of infectious diseases. As a result, emerging infectious diseases are appearing with high frequency, and the epidemic areas of some controlled infectious diseases are expanding or even changing, causing severe outbreaks and threatening public health [66]. New trends challenging the prevention and control of emerging infectious viruses include the increasing number of viruses, diverse infection and transmission routes, and the scope and frequency of pandemics [67,68].

A vaccine is currently the most effective and cost-efficient strategy to prevent susceptible populations from infection. However, most RNA viruses and emerging zoonoses have no vaccines with satisfactory protection efficiency [69]. For emerging infectious diseases, prevention in advance is far more effective and economical than treatment after an outbreak has occurred [70]. It is therefore important to unravel the viral transmission routes from viruses' natural hosts to human societies and to understand the underlying mechanisms in order to design timely and long-lasting prevention strategies [71].

Many studies have addressed the cross-species (molecular) barrier or the endemic barrier against emerging infectious diseases [58,72]; in fact, they are the two sides of the ecological barrier that determines the possibility of emerging viruses transmitting to and spreading among human societies from both the micro and macro perspectives. The cross-species barrier represents the rarity of viruses spreading efficiently within new hosts that have not been previously exposed or susceptible [58]. Effective breakthrough of

the cross-species barrier—namely, spillover infection into alternative hosts—is mainly attributed to viral mutation or evolution, which allows viruses to gradually adapt to new host cells and eventually spread into new populations [58,73]. On the other hand, crossing the endemic barrier depends on the probability and frequency of viral spread, which is closely correlated with contacts between viruses' natural hosts and potential hosts or humans [72]. Although many studies have explored the initial outbreaks and epidemics of infectious diseases and examined how they are linked to the cross-species or endemic barriers, these studies mainly focus on epidemiology and immunology; there is a lack of a comprehensive and systematic analysis of viral migration and transmission in ecosystems from a macro perspective.

Viruses have low genetic stability, and their evolution or variation is closely related to changes in the ecological environment [74]. In addition, viral transmission is determined by interactions among viruses, environmental media, and hosts, and the influential factors vary across geographic regions [75]. It is worth noting that human activities have a significant influence on ecosystems, such as encroachment on the wild habitats of viruses' natural hosts [72] and a shifted geographical distribution of viruses' natural or intermediate hosts driven by climate change [76]. Accordingly, the increasing intensity of human activities might deteriorate ecological barriers by shaping the contacts between humans and the natural environment, and thus accelerate viral transmission into human societies. The sudden appearance and global spread of COVID-19 as a representative of emerging infectious diseases hint at the relationship between human activities and the destruction of the ecological barrier, which is a key issue for both public health and sustainable development in the future. As there is limited knowledge on the relationship between ecological barrier deterioration and human activities, it is necessary to systematically summarize the viral transmission routes in ecosystems and reveal the mechanisms of viral transmission across the ecological barrier, thereby uncovering how human activities can deteriorate the ecological barrier and accelerate viral transmission. This will assist in the prevention and control of emerging infectious diseases.

2. Key factors in the ecological barrier to viral transmission from natural hosts to humans

Viral transmission and infection normally occur within limited species; thus, viral infection of a human must break the ecological barrier. More precisely, four key factors in the ecological barrier play critical roles in viral transmission across either the molecular or the endemic barrier: transmission routes, contact probability, contact frequency, and viral characteristics (Fig. 1). The ecological barrier integrates all the potential challenges to viral transmission from viruses' natural or intermediate hosts to human society, and acts as a key node for emerging infectious diseases.

2.1. Transmission route

Viral transmission across the ecological barrier primarily depends on the mechanisms of virus spillover and transmission between species, via either natural hosts, domestic hosts, or wild vectors. Natural hosts can release viruses to the surrounding environment through secretions, feces, urine, corpses, and so forth [77]. Viruses can survive in soil or water and on various surfaces in wild habitats for a prolonged period of time [78], causing potential infection in other species including humans via direct contact or intake. Domestic animals can also be infected by viruses through contact with environmental media polluted by wild animals carrying viruses [35] which makes it easier for viruses to break the ecological barrier and enter human societies through the excreta, fluids, or wastes of domestic animals during the life cycle of breeding, transportation, slaughter, and sale. Alternatively, wild virus vectors such as mosquitoes can directly deliver viruses by biting domestic animals and humans, thereby behaving as a key group of intermediate hosts transmitting viruses across species with a larger range and higher risk [79].

Increasing human activities in recent decades have resulted in regional and even global climate change, which significantly alters the habitats and movement trajectories of wild animals [80,81]. Climate change can enlarge the living area of natural and intermediate hosts carrying viruses, allowing viruses to spread over greater distances; furthermore, global warming can release ancient viruses from the permafrost [82,83]. In addition, rapid urbanization processes increase the demands placed on land resources, leading to frequent land use change and the massive destruction of wildlife habitats [84]. Ecosystems such as forests and grasslands have been gradually eroded, and the living space of wild animals has been significantly compressed to smaller scales. The improvement in human living standards and the development of agriculture and animal husbandry have increased the numbers and distribution of domestic animal populations [85], inadvertently providing a new breeding habitat and route for zoonoses to cross the ecological barrier. These human-driven factors work together to increase the transmission routes of emerging viruses from natural environments into human society.

2.2. Transmission possibility

Besides diversified transmission routes that offer opportunities for viruses to break through the ecological barrier, the possibility of viral transmission from wildlife to humans depends on the probability of contact within a given area between virus hosts and humans. The range and intensity of human activities are key factors in the possibility of transmission, especially in overlapping areas used by both humans and wildlife. Changes in wildlife habitats driven by global and regional climate change may lead to wildlife invading human residential areas, increasing the probability of direct contact between humans and natural hosts carrying viruses [76]. In fragmented areas where both wildlife and humans are active, the dynamic numbers and geographical distribution of viruses' natural hosts result in higher contact probability in comparison with closed wildlife habitats [72]. Furthermore, rapid urbanization processes produce massive human gathering areas and promote the high-density reproduction and activity of domestic animals in urban areas, which unintentionally raise the transmission possibility through breeding, transportation, slaughter, and sale [86]. The emergence of poverty, slums, and shantytowns in urban areas can result in potential centralized transmission places for emerging infectious diseases [87].

2.3. Contact frequency

Contact frequency, which refers to the level of human exposure to hosts carrying viruses over a time scale, is another key factor in viral transmission across the ecological barrier. Given certain transmission routes and possibilities, contact frequency is strongly associated with the density and active intensity of human populations in fragmented areas that are the habitats of wildlife [72]. As for most zoonoses, contact frequencies between domestic animals and humans are closely linked to living habits and urbanization level [35].

2.4. Viral characteristics

Viral survival time, load, and infectivity are key features affecting the possibility of viruses breaking through the ecological barrier. Viruses of different types have distinct survival times and
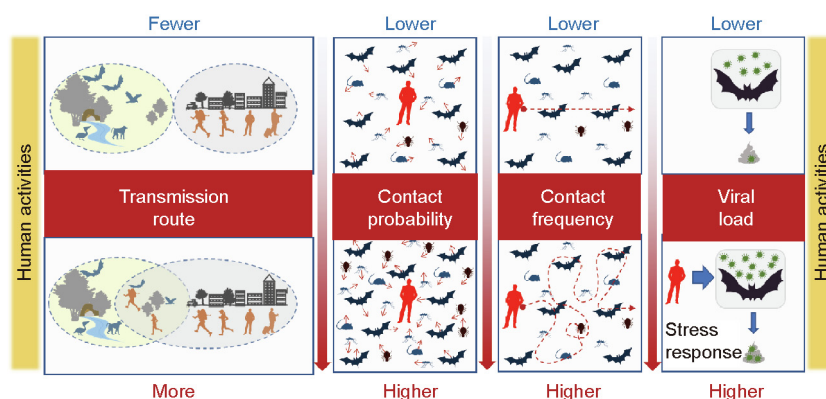

Fig. 1. Key factors of the ecological barrier to viral transmission from natural or intermediate hosts to human, including transmission routes, contact probability, contact frequency, and viral characteristics. Human activities can deteriorate the ecological barrier and intensify the emergence and spread of infectious diseases.

decay patterns across environmental media and conditions, and numerous studies have reported on the survival time and influential factors of classic viruses on solid surfaces or in domestic water, sewage, air, and soil. Temperature is a critical factor influencing viral activity and is generally inversely proportional to viral survival time [88–93]. Coronaviruses lose 99.9% of their activity after ten days in filtered water, and even survive after 100 days at 4 °C [94]. Airborne viruses mainly exist on aerosol surfaces and transmit via airflow over hundreds of meters, far beyond the range of droplets, forming an important transmission route for influenza and other respiratory diseases [95]. Soil is also an important environmental source of or carrier for viruses. Many spherical, tailless viruses and phages have been detected in the soils of deserts, farmlands, forests, wetlands, and pastures around the world at a high level (2.2×10^3–5.8×10^9 g^{-1}) [96–103]. Viruses causing respiratory diseases such as influenza (H1N1, H9N9, and H5N1) and coronavirus (MERS-CoV, SARS-CoV, and other human coronavirus) have a relatively shorter survival time in soils. Influenza viruses can survive for several hours to three days on solid surfaces and to six days on masks, latex, and feathers [104–113] and coronaviruses can survive on solid surfaces or in soils for 2–6 days [114–120]. It is worth noting that soil moisture content is normally proportional to viral activity [121,122]; however, the mechanisms of virus inactivation differ under dry and wet conditions. Viral capsid proteins are easy to dehydrate and inactivate in dry soils, causing the virus to lose its capability to protect RNA, infect, and reproduce, whereas viral RNA may not be destroyed. In contrast, RNA lyase activity is stronger under wet conditions due to the higher microbial activity in soils, resulting in a higher rate of virus capsid dissolution and RNA degradation [123].

Viral loads in viruses' natural hosts have been reported to be affected by human invasions [124]. Wildlife habitat destruction can create extra environmental stresses on wild animals and trigger a stress response to increase the viral load in urine and saliva secretion [125]. Furthermore, viral infectivity and pathogenicity in other hosts are key factors in the frequent occurrence of emerging infectious diseases. Viruses with a longer survival time or more transmission routes have a greater possibility of transmitting from their natural hosts to others, and RNA viruses are prone to greatly mutate in response to environmental changes and to rapidly replicate, contributing to their higher chance to break through the ecological barrier and adapt to new hosts [126].

3. Ecological barrier deterioration driven by human activities

The prevalence of emerging infectious diseases crossing the ecological barrier is related to many ecological processes that are intensively affected by the consequences of human activities, such as global climate change, invasions in fragmented wildlife habitats, diverse human habitats and agricultural development, and rapid urbanization [124,127,128]. In this context, the ability of the ecological barrier to block viral transmission from natural hosts to human society is related to the burden of viral transmission in wild intermediate hosts, breeding animal hosts, and environmental media, respectively (Fig. 2).

3.1. Expanding and emerging infectious diseases driven by global climate change

Global climate change has caused a series of problems including sea level rise, extreme weather, flood, drought, and air/water quality degradation [129]. It can also affect vector ecology to promote the spread of emerging infectious diseases in many ways [130]. In general, global climate change shifts the range and distribution of the habitats of viruses' natural or intermediate hosts and

releases ancient viruses from the permafrost, thereby increasing the wide-ranging risks of emerging infectious diseases.

3.1.1. Wildlife habitats

Global climate change driven by industrialization significantly alters the habitat range and movement trajectory of wild animals [80,81]. The population and distribution of viral natural hosts or vectors that benefit from global climate change will expand, thereby increasing the possibility and frequency of contact with humans in order to transmit emerging infectious diseases across the ecological barrier [76]. The West Nile, chikungunya, Zika, and dengue viruses are all arboviruses. Although their natural hosts differ, their vectors are all arthropod mosquitoes that transmit these arboviruses in a mosquito–natural host–mosquito cycle. Humans are easily infected by mosquitoes carrying viruses [79] among which *Aedes aegypti* (*A. aegypti*) and *Aedes albopictus* (*A. albopictus*) are two typical climate-sensitive vectors. Temperature, rainfall, and humidity are key factors in the reproduction, expansion, and activity of *A. aegypti* and *A. albopictus* [131,132]. Outbreaks of West Nile virus disease are mostly related to high temperature, and a drop in temperature from 26 to 18 °C can decrease the infection rate of *Culex pipiens* from 97% to 18% [133]. As global warming can drive mosquitoes to spread in higher altitudes [134], the *Lancet Countdown to 2030: Public Health and Climate Change* points out that climate change is increasing dengue fever transmissibility by *A. aegypti*; dengue fever has already increased by 9.4% from 1950 to 2015 [135]. Such shifting habitats and the expanding transmission of arboviruses driven by global climate change pose a serious threat to public health.

3.1.2. Ancient virus release

Global climate change can also bring about another acknowledged risk of emerging infectious diseases, widely known as the release of ancient viruses from the melted permafrost. Giant icosahedral DNA viruses [136] and *Mollivirus sibericum* [82] identified in 30 000-year-old permafrost still retain their ability to infect after resuscitation. A recent study found 33 viral populations representing four known genera and likely 28 novel viral genera from about 15 000-year-old ice in the glaciers of the Tibetan Plateau [83]. In a worst-case scenario, all the ancient viruses might be released from melted permafrost or glaciers alongside global warming.

3.2. Higher contact frequency and transmission possibility by increasingly intensive human activities

The intensity of human activities determines the level of human invasion into wildlife habitats, the viral load in viruses' natural hosts, and the number and density of domestic animals, including livestock, poultry, and pets. Human activities in wildlife habitats increase the contact frequency between humans and wild animals carrying viruses and shorten the effective contact time, thereby remarkably increasing the risks of viral transmission across the ecological barrier [137]. Although habitat fragmentation isolates populations with low mobility, it provides access to mobile animals and thus changes the diversity in undisturbed areas [125]. Such impacts change both the habitats of vectors and the patterns of emerging diseases [138].

3.2.1. Immune response in fragmented areas

Environmental stress caused by human activities may lead to an immune response in wild animals, which changes the viral load in the viruses' natural or intermediate hosts. Stress response is reported to alter immune function and change the transmission and infection patterns among wild animals, domestic animals, and humans [139]. Although no convincing experimental data has illustrated the relationship between stress response and host
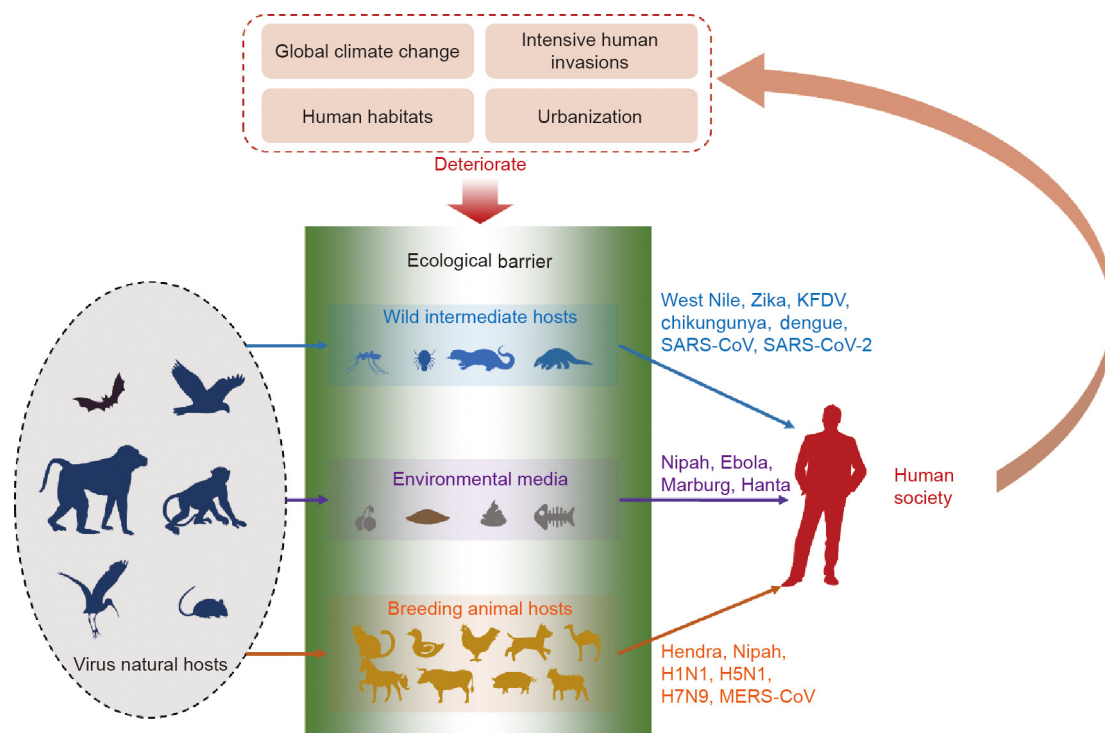
Fig. 2. Ecological barrier deterioration driven by human activities. The ecological barrier blocks the three main viral transmission routes (wild intermediate hosts, breeding animal hosts, and environmental media) from natural hosts to human society. Human activities deteriorate the ecological barrier through global climate change, intensive human invasions, growing human habitats, and urbanization. KFDV: Kyasanur forest disease virus.

viral load, two hypotheses have been raised to explain this phenomenon. The "accidental spillover" hypothesis suggests that the immune response of virus hosts suppresses the persistent infection, and replication and periodic shedding of viruses only occur when the immune response is weakened by internal or external pressure, breaking the balance between viruses and their hosts [140,141]. This hypothesis explains one of the driving mechanisms of Hendra virus as the immune response of fruit bats induced by human-caused stress [125,142]. In contrast, the "transient epidemic" hypothesis describes a dynamic balance between local virus extinction and re-colonization between hosts. Accordingly, an infection pulse is generated as a wave of infection across hosts. The key factor triggering a transient epidemic of non-lethal viruses is recovery after infection and subsequent immunity [125]. As time passes, the immunity of the whole population decreases, and the viral load then increases. A study on the significant correlation between land use change and the outbreak of Ebola disease reported that, among the 11 first-reported infectious cases of Ebola disease, eight cases occurred in areas with a high degree of forest destruction [72]. Such areas are all habitats of bats carrying the Ebola virus, and the viral load in bats is only detectable in the case of Ebola disease. The migration and distributions of ticks in damaged forests are strongly correlated with the case numbers and geographical features of Kyasanur forest disease [143], Lyme disease [144], and Crimean-Congo hemorrhagic fever [145].

3.2.2. Contact with environmental media

Besides directly coming into contact with wildlife, human infection can occur by touching environmental media containing viruses in wildlife habitats. Natural viral hosts can release viruses into the surrounding environmental media in many ways, such as through saliva on fruit, animal carcasses during feeding, feces or urine entering the water or soil, and even dead corpses. Typical examples include the Nipah [146,147], Ebola [30,33,148,149], Marburg, and Hanta [9] viruses. Nipah virus was first identified in

1998 in Malaysia; its natural hosts are *Pteropus giganteus* (*P. giganteus*). The habitats of *P. giganteus* are close to jujube trees, and the hosts' excreta of urine and feces containing Nipah virus can contaminate the jujube juice and juice collection jars, resulting in human infection [146,147]. Fruit bats are natural Ebola hosts, and the most recognized transmission routes from fruit bats to humans include eating bat meat with live viruses, consuming foods contaminated by bat excreta, and coming into direct contact with fruit bats in caves [30,33,148,149]. Hanta virus disease can be traced back to 1913; its natural hosts are rodents, including *Apodemus agrarius*, *Rattus norvegicus*, *Apodemus dahlia*, and *Apodemus agrarius*. The main transmission route of Hanta virus is animal-derived contact through saliva, urine, and feces, which release viruses into the surrounding environment. Infection can occur through the respiratory tract due to dusts carrying viruses, the digestive tract from contaminated food and water, direct contact with rodents or their excreta, and invasion through damaged skin [9].

More importantly, viruses can survive in environmental media for a prolonged length of time, waiting for opportunities to infect animals and humans and cause an outbreak of an emerging infectious disease. Under suitable conditions, viruses can survive for hundreds or even thousands of days in environmental matrices. Porcine parvovirus can survive for more than 43 weeks in soil [150] and human norovirus retains at least 10% activity after 1266 days in groundwater [151]. SARS-CoV-2 viral RNA has been detected on the floor in COVID-19 patient rooms [152] and even in the soil surrounding the outpatient department [153]. Accordingly, environmental media in wildlife habitats are hypothesized to receive and store viruses from natural virus hosts—particularly in soil, silt, or fallen leaves in caves or forest interiors, which are cold, dark, and humid, and thus allow viruses a longer survival time. These residual viruses may contaminate the surface water through rainfall and the groundwater through infiltration. Human activities that involve directly touching these media, eating contaminated fruits, or drinking contaminated water in these wildlife

habitats offer opportunities for viruses to break through the ecological barrier during wilderness backpacking, mining, logging, or poaching. As SARS-CoV-2 is reported to survive on plastic surfaces for at least three days [154], it is strongly suspected that virus-contaminated clothing might spread a wide range of viruses in crowd-gathering areas, eventually causing an outbreak of an emerging infectious disease in human society.

Outbreaks of Kyasanur forest disease are a good example of a transmission route through direct contact with environmental media containing viruses. Kyasanur forest disease is caused by the Kyasanur forest disease virus (a member of the virus family Flaviviridae). Monkeys and rodents such as *Macaca mulatta* and *Rattus rattus* are its natural hosts, and ticks (mainly *Haemaphysalis*, especially *Haemaphysalis spinigera*) are its wild vectors [155]. Epidemics of Kyasanur forest disease exhibit an obvious seasonal behavior and are consistent with the life habits of local ticks carrying viruses. Patients or susceptible people predominantly include young farmers, herdsmen, and forestry workers, who frequently enter wildlife habitats in their daily work, resulting in increased exposure to intermediate virus hosts and a much higher chance of getting infected in comparison with other people [27–29,156].

3.2.3. International trading

International trading provides new opportunities for the long-distance transmission of viruses or pathogens. Frequent international or transnational trading increases the exchange of wild or domestic animals carrying viruses, thus increasing the likelihood of a global outbreak [157]. For example, the outbreak of monkeypox in the United States in 2003 originated from the transnational pet trade [158] and avian influenza in Asia presents a high risk to countries in other continents through the international poultry trade [159]. In addition, some crop pathogens can be transmitted by international trading and can infect humans and animals in other countries [160]—a transmission route that explains many outbreaks of foodborne diseases. A significant case is the emergence of *Salmonella* in the United States in 1998–2003, which was linked with imported mangoes from Brazil [161].

3.3. Increasing transmission routes driven by human habits

Natural virus hosts can transmit viruses to other wild animals (i.e., intermediate hosts), including predators (through being bitten or eaten by intermediate hosts), and parasites (ticks or fleas). After adaptation and evolution, viruses can infect and spread in the new intermediate hosts, expanding natural virus reservoirs in wider habitats. This process effectively breaks through the ecological barrier and poses a threat to human societies, as an increasing number of wild animals and parasites can cause outbreaks of emerging infectious diseases. Residents' living habits can also affect the transmission routes and infectivity of viruses, including eating wild animals, domestic breeding, farming, and personal sanitation.

3.3.1. Eating wild animals

Some residents of East Asia and Africa consider wild animals to be nourishing foods to maintain one's health; therefore, eating wild animals is common behavior in some countries [162]. This habit sets up an industrial chain of wildlife poaching, feeding, and slaughtering, which increases the risk of viral transmission from their natural or intermediate hosts to human societies. Among all natural virus hosts, bats are an important reservoir of coronaviruses, including those related to SARS-CoV and SARS-CoV-2 [163]. A possible transmission route is through wild intermediate hosts (e.g., civets and weasels) to humans. Cooks and employees in wildlife food markets have a greater chance of being infected with such viruses due to their frequent contact with SARS-CoV or SARS-CoV-2 intermediate hosts [44,56,164]. Eating wild

animals in restaurants further encourages the whole supply chain, exacerbating the possibility of direct or indirect contact between wild animals carrying viruses and hunters, breeders, butchers, or consumers, and offering additional opportunities for viral evolution and human infection.

3.3.2. Domestic breeding

Besides wild intermediate hosts, domestic breeding animals such as horses, camels, chickens, and ducks can become intermediate hosts after infection. Domesticated animals can come into close contact with natural hosts carrying viruses by sharing foods in cribs or through biting and predation, which permits frequent viral transmission to occur between wild and domestic animals [85]. Livestock polyculture also accelerates viral mutation and interspecific transmission, making domesticated breeding animals into key intermediate hosts for many zoonoses [86]. These viruses can infect and spread within domestic breeding animal populations and directly enter human societies through the processes of raising, sales, and eating. Thus, breeders, transporters, farmers, and seafood market sellers are highly susceptible to infection by emerging infectious diseases including Hendra virus, MERS-CoV, and influenza (H1N1, H5N1, etc.).

Hendra virus was first identified in Hendra (Brisbane, Queensland, Australia) in 1994, where it caused the deaths of 22 horses and three people; its natural hosts are fruit bats [165]. Breeding increases the horse populations close to the living habitats of fruit bats carrying Hendra virus [35]. These breeding farms provide additional habitats for fruit bats, which gives the horses more chances to touch the urine or secretions of the fruit bats, allowing the spread of Hendra virus among horses and then among farm staffs in close contact with the horses [166–169].

MERS-CoV was first isolated from the lung tissues of deceased cases of severe pneumonia in Saudi Arabia in 2012, and its natural hosts may be bats. The MERS outbreak is attributed to the domestic breeding of single humped camels in the Middle East, as the single humped camel is an important intermediate host of MERS-CoV. As thousands of single humped camels are imported into Saudi Arabia from African countries every year, the outbreak of MERS is strongly correlated with the traffic of camels [170]. Sufficient evidence shows that MERS-CoV is transmitted from camels to humans, as the infection rate of breeding staffs who come into close contact with camels is much higher than that of other people, and as serological studies have documented that the positive rates of MERS-CoV antibody in breeding and slaughterhouse staff members are 15 and 23 times higher, respectively, than in the general population [43,51,171,172].

Influenza A viruses are mutagenic in their natural hosts, showing huge potential to infect poultry and invade humans. H5N1 is a zoonosis with first recorded infection case in Hong Kong, China in 1997 and widely detected in other Asian countries [173]. Wild birds and poultry are considered to be the natural and intermediate hosts of H5N1, respectively [174]. Wild birds are responsible for virial long-distance transmission from Qinghai Lake in China to India, Siberia, and Southeast Asia, and interspecies transmission between poultry populations has promoted the regional transmission of H5N1 into human societies [39,40]. H1N1 was first identified in Mexico in 2009 and affected 214 countries and regions, infecting millions of people and causing at least 18 449 deaths. Domestic pigs are considered to be the intermediate hosts in which H1N1 virus obtains the capability to infect humans [46,175]. H7N9 virus comes from wild birds and can eventually infect humans by recombining genes with other influenza viruses in breeding chickens and ducks [127]. During the adaptation process in domestic poultry, H7N9 evolved from a low pathogenic avian influenza into a highly pathogenic one, causing a serious epidemic in China in 2017 with 1564 infected cases and more than 600 deaths [38,176,177].

In conclusion, domestic breeding animals including poultry and livestock are key intermediate hosts of influenza viruses and are key factors in influenza outbreaks in human society.

3.3.3. Farming

Some traditional farming practices can increase the risk of viral transmission to human society. Untreated sewage and sludge are commonly used for irrigation or fertilization in many rural areas around the world; however, a variety of pathogens can be found in fresh sewage or feces, such as viruses (norovirus, enterovirus, hepatitis E virus, etc.) [178,179], bacteria (*Salmonella*, *Escherichia coli* (*E. coli*), *Vibrio cholera*, etc.) [180], and parasitic eggs (*Ascaris* eggs, *Trichuris* eggs, etc.) [181,182]. Hepatitis E viruses are found in pig manure or wastewater from pig-breeding facilities [183]. Infectious hepatitis E viruses were detected in pig manure from 15 out of 22 pig farms in Iowa, USA [184]. As the fecal–oral pathway is a main transmission route of hepatitis E viruses, pig manure applications and surface wastewater runoff may contaminate agricultural products and the surrounding water sources [185,186]. The positivity of hepatitis E viruses in untreated and concentrated sewage samples was found to be as high as 10.97% in a wastewater treatment plant in Puna, India [187], indicating that sewage treatment workers face a higher risk of hepatitis E infection. Therefore, sewage irrigation can directly increase viral transmission from sewage to humans, or can indirectly contaminate soil, water, and food products to cause foodborne infectious diseases. Farming and storage products can also attract the active approach of wild animals, thereby increasing the transmission routes and contact frequency of some viruses, allowing them to cross the ecological barrier [72]. For example, the Ebola virus can be transmitted from fruit bats to humans through contaminated fruit; this process is aggravated by the fruit storage in villages, which draws fruit bats from caves into an area of human activity [162].

3.3.4. Personal sanitation

Personal sanitation is a critical way to block viral infection [188]. Inappropriate cultural norms and personal habits related to personal cleanliness can increase the exposure and infectious probability of viruses and pathogens [189]. Hand, foot, and mouth disease (HFMD) is caused by enterovirus (mainly coxsackievirus A16 and enterovirus 71); the susceptible population is mainly children with weak immunity [190]. Studies show that children who have a habit of sucking their fingers face a significant higher risk of such infection than others, whereas children who wash their hands before meals face only half the risk in comparison with others [191]. In addition, burying a corpse for several days and then touching the corpse is part of traditional funeral customs in some rural areas of West Africa, and can increase the risk of spreading emerging infectious diseases [192].

3.4. New hotspots generated by urbanization and sanitary conditions

The increasing level of urbanization has changed the global pattern of infectious diseases [193]. Although urbanization can improve the basic infrastructure and sanitation conditions to protect the public health to some extent [84], it can also alter the numbers, diversities, and community structure of wildlife in city areas [194]. As a result, urbanization generates new hotspots that cause the outbreak of infectious diseases. Some municipal infrastructures provide new points or networks for viral transmission in human societies. For example, Zika virus is mainly transmitted by *A. aegypti* and other *Aedes*, which have already adapted to a densely populated urban environment [195]. Driven by both global climate change and urbanization processes, these vectors have a wider distribution and significantly contribute to the global outbreak of Zika virus disease [196]. SARS-CoV-2 has been detected in the wastewater in many countries, and wastewater-based epidemiology (WBE) not only offers a new diagnostic tool for disease prevention and control, but also highlights the signs of disease spread through the urban pipeline network [197].

3.4.1. Landfills

Landfills are typical sources for viral transmission in urban areas. Due to the complexity of the wastes dumped in landfills, it may include animals that die of infection or contaminated medical wastes carrying many infectious viruses, making landfills a virus sink. In the United States, poultry and livestock with infectious diseases are generally disposed in landfills; during an epidemic, some medical wastes carrying viruses are also buried. These activities increase the chance of viral secondary transmission and generate new hotspots of emerging infectious viruses in urban areas. Studies have demonstrated that avian influenza H6N2 virus in poultry carcasses remains infectable for nearly two years—or even for more than 30 years in municipal landfills under appropriate temperature and pH conditions—and that viruses can survive in landfill leachates for at least 30 days [198]. The prolonged survival time of infectious pathogens can result in wide distributions of bacterial pathogens (e.g., *E. coli* and *Salmonella*) and viruses causing avian influenza, HFMD, Newcastle disease, and porcine epidemic diarrhea [199,200]. Studies have also reported that the waste treatment facilities in landfills can continuously release a variety of viruses in the form of bioaerosols, which spread emerging infectious viruses to wild animals and even to the landfill staff [201].

In addition, a large number of foraging rodents and birds in or around landfills can transport and excrete feces containing viruses as a transmission route, as they forage in groups during the day and return to their residence communities at night. Rodents living in landfills can carry and transmit viruses by ingesting organic wastes. For example, rodents living at landfill sites in the Istra peninsula are more likely than wild rodents to be infected by zoonotic viruses, such as lymphocytic choriomeningitis virus and tick-borne encephalitis virus [202]. Birds and their stools have been reported to carry over 60 types of pathogens, including bird influenza [203] and other human epidemic viruses such as H1N1, H2N1, and H3N2 [204,205]. Mutated influenza viruses can accumulate in migratory birds until they break through the ecological barrier to infect humans [73,206]. Studies in the United Kingdom have shown that landfills provide a wide range of foraging opportunities for rotifers such as seagulls and crows, which are known carriers of human infectious pathogens such as *Salmonella*, *E. coli*, *Campylobacter*, and influenza A viruses [207]. Their daily shuttle between landfills and reservoirs introduces the risk of water contamination by pathogens and viruses and challenges drinking water safety. White storks nesting in landfills carry more pathogens than those nesting naturally [208]. According to the wild migratory bird surveillance program for highly pathogenic avian influenza by the United States, wild ducks living in landfills are the birds most likely to be infected by influenza viruses [209]. By taking organic waste as a food source, American black vultures living in a Patagonia landfill (Argentina) were reported to be infected with and to spread zoonotic pathogens such as *Salmonella* [210]. It can be concluded that birds around landfills are potential hosts carrying and transmitting viruses to humans via stools, contaminated water, and dead bodies. As pathogen reservoirs, landfills are high-risk sites for birds and rodents to transmit emerging infectious diseases across the ecological barrier.

3.4.2. Water-supply and sewage systems

Human agricultural activities can also provide breeding grounds and living habitats for the vectors of insect-borne infectious diseases by constructing dams, ponds, and other water-storage facilities for irrigation. The construction of the Aswan Dam in Egypt, the Jama Dam on the Senegal River, and the Manantari Dam in Mali and

Guinea has intensified the outbreak frequency of schistosomiasis mansoni [211]. In addition, about 60 species of *Anopheles* are vectors of mosquito-borne malaria, and they can breed in the open ponds constructed close to farmlands, becoming an important reasons for the malaria epidemics in many countries [212].

Backward municipal infrastructure—such as water-supply and drainage networks, sewage treatment systems, and improper water storage—provides extra habitats in cities for some vectors carrying and spreading emerging infectious diseases; for example, *Aedes* and *Culex* flourish in urban sewage, causing outbreaks of Rift Valley fever and other diseases [138,212].

The occurrence of SARS-CoV-2 in wastewater and rivers has been reported in China (Wuhan) [213], France (Paris) [197], and Australia [214]. High levels of SARS-CoV-2 viral RNA in wastewater, ranging from several to thousands of copies per milliliter, suggest the potential transmission and spread of SARS-CoV-2 viruses in the urban and rural water cycle, presenting a potential threat to public health [215].

3.4.3. Sanitation conditions

Sanitation conditions are closely related to viral transmission in urban areas, and improved sanitation conditions can protect the public health from emerging infectious diseases. The transfer of huge populations from rural areas to cities in the early stage of rapid urbanization is often accompanied by poverty caused by urban expansion and underdeveloped infrastructure [87]. From 1963 to 2010, over 110 000 cases of hemorrhagic fever renal syndrome (HFRS) caused by hantavirus were reported in Hunan province (China), and a positive correlation was detected between the number of city migrants and the incidence rate of HFRS in the initial stage of urbanization [84]. In addition, urban public health problems are a key factor in the emergence of infectious diseases [216]. Many cities have slums or shantytowns with poor sanitation conditions, and some infectious diseases are rampant in these densely populated and relatively closed areas. Some developing countries in Asia and Africa with poor sanitation conditions, limited basic medical capabilities, and insufficient vaccine coverage cannot sufficiently deal with emerging infectious diseases, which have a higher possibility of breaking out there [185]. The largescale outbreak of acute viral hepatitis (AVH) from 1955 to 1956 in New Delhi was mainly located in slums with poor sanitation conditions and a low socio-economic level [217].

4. Conclusions and future perspectives

In summary, the ecological barrier is the key to viral transmission from viruses' natural or intermediate hosts to human societies. The strength of the ecological barrier determines the possibility and scale of epidemics caused by emerging infectious viruses. Future studies should focus on the dynamic process of viruses crossing the ecological barrier, which is a critical step for the prevention and control of emerging infectious diseases. The main influential factors affecting the ecological barrier include transmission routes, contact probability, contact frequency, and viral characteristics; environmental media are also an important component of the ecological barrier. Emerging infectious diseases are currently exhibiting global spreading patterns owing to the deterioration of the ecological barrier by intensive human activities. Global climate change driven by industrialization and globalization processes has triggered and expanded the emergence of infectious diseases, and the increasing levels of human disturbance in fragmented wildlife habitats are significantly promoting greater contact probability and a higher frequency of emerging infectious viruses breaking through the ecological barrier. With the rapid development of the social economy, international transportation

through the air and over sea and land is becoming more intensive. As a result, such transportation is advancing a cross-border exchange of emerging infectious viruses that is driving the globalization of epidemics and introducing great challenges for public health and biosafety management. The presence of diverse human habitats across countries also increases the transmission routes of viruses, and global urbanization is shaping new hotspots of poverty and poor sanitary conditions in urban areas that promote the spread of emerging infectious diseases in human societies.

It is extremely urgent to further explore the quantitative effects of human activities on the strength of the ecological barrier and to understand the mechanisms by which emerging infectious diseases are transmitted and spread across the ecological barrier. As viruses are "dark matter" in many environmental media that behave as viral reservoirs, more studies should focus on building the environmental virus database for wildlife habitats. A comprehensive investigation into the interactions between viruses, their hosts, and environmental media can grant us better insight into the effects of ecological barrier deterioration on the spread of emerging infectious diseases, as well as the underlying influential factors.

For the effective prevention and control of emerging infectious diseases, potential strategies should be considered to protect the ecological barrier and block the transmission of viruses from their natural reservoirs into human societies. Firstly, largescale environmental surveys on viruses in wildlife habitats are suggested in order to map the origin and distribution of emerging infectious viruses and visualize "hot" or weakened spots in the ecological barrier. Secondly, dynamic monitoring of natural (e.g., bats, pangolins, birds) or intermediate (e.g., camels, mosquitoes, and ticks) hosts of zoonoses should focus on fragmented wildlife habitats being invaded by human activities. In addition, we propose a biosafety "skynet" as a novel strategic concept for preventing and controlling the rapid outbreak of emerging infectious diseases in urban areas. It consists of online diagnostic devices for monitoring viral load in environmental media like aerosol and water, and a real-time big data management system for early warning and emergency management. Regular biosafety management and emergency measures are necessary to enable the ecological barrier in either the natural environment or human societies to effectively control emerging infectious viruses of great concern. Lastly, but most importantly, in following a path of sustainable development, human society must reconsider the correlation between human and global ecology and pay more attention to the protection of the ecological barrier.

Acknowledgments

Compliance with ethics guidelines

Dayi Zhang, Yunfeng Yang, Miao Li, Yun Lu, Yi Liu, Jingkun Jiang, Ruiping Liu, Jianguo Liu, Xia Huang, Guanghe Li, and Jiuhui Qu declare that they have no conflict of interest or financial conflicts to disclose.

References

[1] Managing epidemics: key facts about major deadly diseases [Internet]. Geneva: World Health Organization; c2018 [cited 2020 Apr 10]. Available from: https://apps.who.int/iris/handle/10665/272442.

[2] Sampathkumar P, Sanchez JL. Zika virus in the Americas: a review for clinicians. Mayo Clin Proc 2016;91(4):514–21.

[3] Guzman MG, Gubler DJ, Izquierdo A, Martinez E, Halstead SB. Dengue infection. Nat Rev Dis Primers 2016;2(1):16055.

[4] Dudas G, Carvalho LM, Bedford T, Tatem AJ, Baele G, Faria NR, et al. Virus genomes reveal factors that spread and sustained the Ebola epidemic. Nature 2017;544(7650):309–15.

[5] Zhu M, Lin ZG, Zhang L. Spatial–temporal risk index and transmission of a nonlocal dengue model. Nonlinear Anal Real World Appl 2020;53:103076.

[6] Maljkovic Berry I, Rutvisuttinunt W, Sippy R, Beltran-Ayala E, Figueroa K, Ryan S, et al. The origins of dengue and chikungunya viruses in Ecuador following increased migration from Venezuela and Colombia. BMC Evol Biol 2020;20(1):31.

[7] Bhatt S, Gething PW, Brady OJ, Messina JP, Farlow AW, Moyes CL, et al. The global distribution and burden of dengue. Nature 2013;496(7446):504–7.

[8] Brady OJ, Gething PW, Bhatt S, Messina JP, Brownstein JS, Hoen AG, et al. Refining the global spatial limits of dengue virus transmission by evidence-based consensus. PLoS Negl Trop Dis 2012;6(8):e1760.

[9] Lee HW, Lee PW, Baek LJ, Song CK, Seong IW. Intraspecific transmission of Hantaan virus, etiologic agent of Korean hemorrhagic fever, in the rodent *Apodemus agrarius*. Am J Trop Med Hyg 1981;30(5):1106–12.

[10] Jonsson CB, Figueiredo LTM, Vapalahti O. A global perspective on hantavirus ecology, epidemiology, and disease. Clin Microbiol Rev 2010;23(2):412–41.

[11] Lee HW. Epidemiology and pathogenesis of hemorrhagic fever with renal syndrome. In: Elliott RM, editor. The bunyaviridae. Boston: Springer; 1996. p. 253–67.

[12] Johnson NPAS, Mueller J. Updating the accounts: global mortality of the 1918–1920 "Spanish" influenza pandemic. Bull Hist Med 2002;76(1):105–15.

[13] Olson DR, Simonsen L, Edelson PJ, Morse SS. Epidemiological evidence of an early wave of the 1918 influenza pandemic in New York City. Proc Natl Acad Sci USA 2005;102(31):11059–63.

[14] Taubenberger JK, Morens DM. 1918 Influenza: the mother of all pandemics. Emerg Infect Dis 2006;12(1):15–22.

[15] Smithburn KC, Hughes TP, Burke AW, Paul JH. A neurotropic virus isolated from the blood of a native of Uganda. Am J Trop Med Hyg 1940;20:471–92.

[16] Shi PY, Kramer LD. Molecular detection of West Nile virus RNA. Expert Rev Mol Diagn 2003;3(3):357–66.

[17] Petersen LR, Brault AC, Nasci RS. West Nile virus: review of the literature. JAMA 2013;310(3):308–15.

[18] Sambri V, Capobianchi M, Charrel R, Fyodorova M, Gaibani P, Gould E, et al. West Nile virus in Europe: emergence, epidemiology, diagnosis, treatment, and prevention. Clin Microbiol Infect 2013;19(8):699–704.

[19] Chancey C, Grinev A, Volkova E, Rios M. The global ecology and epidemiology of West Nile virus. Biomed Res Int 2015;2015:376230.

[20] West Nile virus statistics & maps [Internet]. Geneva: World Health Organization; c2020 [cited 2020 Oct 6]. Available from: https://www.cdc.gov/westnile/statsmaps/index.html.

[21] Kazmi SS, Ali W, Bibi N, Nouroz F. A review on Zika virus outbreak, epidemiology, transmission and infection dynamics. J Biol Res 2020;27(1):5.

[22] Baud D, Gubler DJ, Schaub B, Lanteri MC, Musso D. An update on Zika virus infection. Lancet 2017;390(10107):2099–109.

[23] Powers AM. Chikungunya virus outbreak expansion and microevolutionary events affecting epidemiology and epidemic potential. Res Rep Trop Med 2015;6:11–9.

[24] Pialoux G, Gaüzère BA, Jauréguiberry S, Strobel M. Chikungunya, an epidemic arbovirosis. Lancet Infect Dis 2007;7(5):319–27.

[25] Rezza G, Weaver SC. Chikungunya as a paradigm for emerging viral diseases: evaluating disease impact and hurdles to vaccine development. PLoS Negl Trop Dis 2019;13(1):e0006919.

[26] Weaver SC, Chen R, Diallo M. Chikungunya virus: role of vectors in emergence from enzootic cycles. Annu Rev Entomol 2020;65(1):313–32.

[27] Yadav PD, Patil S, Jadhav SM, Nyayanit DA, Kumar V, Jain S, et al. Phylogeography of Kyasanur forest disease virus in India (1957–2017) reveals evolution and spread in the Western Ghats region. Sci Rep 2020;10:1966.

[28] Tandale BV, Balakrishnan A, Yadav PD, Marja N, Mourya DT. New focus of Kyasanur forest disease virus activity in a tribal area in Kerala, India, 2014. Infect Dis Poverty 2015;4(1):12.

[29] Ajesh K, Nagaraja BK, Sreejith K. Kyasanur forest disease virus breaking the endemic barrier: an investigation into ecological effects on disease emergence and future outlook. Zoonoses Public Health 2017;64(7):e73–80.

[30] Smith DH, Isaacson M, Johnson KM, Bagshawe A, Johnson BK, Swanapoel R, et al. Marburg-virus disease in Kenya. Lancet 1982;319(8276):816–20.

[31] Chronology of major Marburg virus disease outbreaks [Internet]. Geneva: World Health Organization; 2018 Feb 15 [cited 2020 Oct 6]. Available from: https://www.who.int/en/news-room/fact-sheets/detail/marburg-virus-disease.

[32] Liu WB, Li ZX, Du Y, Cao GW. Ebola virus disease: from epidemiology to prophylaxis. Mil Med Res 2015;2:7.

[33] Murray MJ. Ebola virus disease: a review of its past and present. Anesth Analg 2015;121(3):798–809.

[34] Chronology of previous Ebola virus disease outbreaks [Internet]. Geneva: World Health Organization; 2020 Feb 10[cited 2020 Oct 6]. Available from: https://www.who.int/news-room/fact-sheets/detail/ebola-virus-disease.

[35] Eaton BT, Broder CC, Wang LF. Hendra and Nipah viruses: pathogenesis and therapeutics. Curr Mol Med 2005;5(8):805–16.

[36] Barclay AJ, Paton DJ. Hendra (equine morbillivirus). Vet J 2000;160(3):169–76.

[37] Hendra virus infection [Internet]. Geneva: World Health Organization; c2020 [cited 2020 Oct 6]. Available from: https://www.who.int/health-topics/hendra-virus-disease#tab=tab_1.

[38] Herfst S, Imai M, Kawaoka Y, Fouchier RAM. Avian influenza virus transmission to mammals. In: Compans RW, Oldstone MBA, editors. Influenza pathogenesis and control—volume I. Cham: Springer; 2014. p. 137–55.

[39] Bui C, Bethmont A, Chughtai AA, Gardner L, Sarkar S, Hassan S, et al. A systematic review of the comparative epidemiology of avian and human influenza A H5N1 and H7N9—lessons and unanswered questions. Transbound Emerg Dis 2016;63(6):602–20.

[40] Li YT, Linster M, Mendenhall IH, Su YCF, Smith GJD. Avian influenza viruses in humans: lessons from past outbreaks. Br Med Bull 2019;132(1):81–95.

[41] Aditi SM, Shariff M. Nipah virus infection: a review. Epidemiol Infect 2019;147:e95.

[42] Nipah virus infection. 2009 Jun 15 [cited 2020 Oct 6]. Available from: https://www.who.int/publications/i/item/10665-205574.

[43] Lu G, Wang Q, Gao GF. Bat-to-human: spike features determining 'host jump' of coronaviruses SARS-CoV, MERS-CoV, and beyond. Trends Microbiol 2015;23(8):468–78.

[44] Guan Y, Zheng BJ, He YQ, Liu XL, Zhuang ZX, Cheung CL, et al. Isolation and characterization of viruses related to the SARS coronavirus from animals in southern China. Science 2003;302(5643):276–8.

[45] SARS outbreak contained worldwide [Internet]. Geneva: World Health Organization; 2003 Jul 5 [cited 2020 Oct 6]. Available from: https://www.who.int/mediacentre/news/releases/2003/pr56/en/.

[46] York I, Donis RO. The 2009 pandemic influenza virus: where did it come from, where is it now, and where is it going? In: Richt JA, Webby RJ, editors. Swine influenza. Berlin: Springer; 2013. p. 241–57.

[47] Kelly H, Peck HA, Laurie KL, Wu P, Nishiura H, Cowling BJ. The age-specific cumulative incidence of infection with pandemic influenza H1N1 2009 was similar in various countries prior to vaccination. PLoS ONE 2011;6(8):e21828.

[48] Dawood FS, Iuliano AD, Reed C, Meltzer MI, Shay DK, Cheng PY, et al. Estimated global mortality associated with the first 12 months of 2009 pandemic influenza A H1N1 virus circulation: a modelling study. Lancet Infect Dis 2012;12(9):687–95.

[49] Pandemic (H1N1) 2009—update 112 [Internet]. Geneva: World Health Organization; 2010 Aug 6 [cited 2020 Oct 6]. Available from: https://www.who.int/csr/don/2010_08_06/en/.

[50] Wong JY, Kelly H, Ip DKM, Wu JT, Leung GM, Cowling BJ. Case fatality risk of influenza A (H1N1pdm09): a systematic review. Epidemiology 2013;24(6):830–41.

[51] Al-Tawfiq JA, Memish ZA. Middle East respiratory syndrome coronavirus: transmission and phylogenetic evolution. Trends Microbiol 2014;22(10):573–9.

[52] H7N9 situation update [Internet]. Rome: Food and Agriculture Organization of the United Nations; c2020 [cited 2020 Oct 6]. Available from: http://www.fao.org/ag/againfo/programmes/en/empres/h7n9/situation_update.html.

[53] Seah I, Agrawal R. Can the coronavirus disease 2019 (COVID-19) affect the eyes? A review of coronaviruses and ocular implications in humans and animals. Ocul Immunol Inflamm 2020;28(3):391–5.

[54] Coronavirus disease (COVID-19) situation dashboard [Internet]. Geneva: World Health Organization; c2020 [cited 2020 Oct 05]. Available from: https://covid19.who.int.

[55] Weaver SC, Lecuit M. Chikungunya virus and the global spread of a mosquito-borne disease. N Engl J Med 2015;372(13):1231–9.

[56] Bolles M, Donaldson E, Baric R. SARS-CoV and emergent coronaviruses: viral determinants of interspecies transmission. Curr Opin Virol 2011;1(6):624–34.

[57] Coronavirus disease (COVID-19) situation report [Internet]. Geneva: World Health Organization; 2020 Oct 6 [cited 2020 Oct 6]. Available from: https://www.who.int/publications/m/item/weekly-epidemiological-update—5-october-2020.

[58] Parrish CR, Holmes EC, Morens DM, Park EC, Burke DS, Calisher CH, et al. Cross-species virus transmission and the emergence of new epidemic diseases. Microbiol Mol Biol Rev 2008;72(3):457–70.

[59] Schwarzenbach RP, Egli T, Hofstetter TB, von Gunten U, Wehrli B. Global water pollution and human health. Annu Rev Environ Resour 2010;35(1):109–36.

[60] Root TL, Price JT, Hall KR, Schneider SH, Rosenzweig C, Pounds JA. Fingerprints of global warming on wild animals and plants. Nature 2003;421(6918):57–60.

[61] Dilley M, Chen RS, Deichmann U, Lerner-Lam AL, Arnold M. Natural disaster hotspots: a global risk analysis. Technical report. Washington, DC: The World Bank; 2005.

[62] Markovchick-Nicholls L, Regan HM, Deutschman DH, Widyanata A, Martin B, Noreke L, et al. Relationships between human disturbance and wildlife land use in urban habitat fragments. Conserv Biol 2008;22(1):99–109.

[63] Smith RJ, Muir RD, Walpole MJ, Balmford A, Leader-Williams N. Governance and the loss of biodiversity. Nature 2003;426(6962):67–70.

[64] Kansky R, Kidd M, Knight AT. A wildlife tolerance model and case study for understanding human wildlife conflicts. Biol Conserv 2016;201:137–45.

[65] Whittaker D, Knight RL. Understanding wildlife responses to humans. Wildl Soc Bull 1998;26(2):312–7.

[66] Shuman EK. Global climate change and infectious diseases. N Engl J Med 2010;362(12):1061–3.

[67] Spencer JH, Finucane ML, Fox JM, Saksena S, Sultana N. Emerging infectious disease, the household built environment characteristics, and urban

planning: evidence on avian influenza in Vietnam. Landsc Urban Plan 2020;193:103681.

[68] Zumla A, Hui DSC. Emerging and reemerging infectious diseases: global overview. Infect Dis Clin North Am 2019;33(4). xiii–xix.

[69] Wu T, Perrings C, Kinzig A, Collins JP, Minteer BA, Daszak P. Economic growth, urbanization, globalization, and the risks of emerging infectious diseases in China: a review. Ambio 2017;46(1):18–29.

[70] Langwig KE, Voyles J, Wilber MQ, Frick WF, Murray KA, Bolker BM, et al. Context-dependent conservation responses to emerging wildlife diseases. Front Ecol Environ 2015;13(4):195–202.

[71] Pike J, Bogich T, Elwood S, Finnoff DC, Daszak P. Economic optimization of a global strategy to address the pandemic threat. Proc Natl Acad Sci USA 2014;111(52):18519–23.

[72] Rulli MC, Santini M, Hayman DT, D'Odorico P. The nexus between forest fragmentation in Africa and Ebola virus disease outbreaks. Sci Rep 2017;7 (1):41613.

[73] Lloren KKS, Lee T, Kwon JJ, Song MS. Molecular markers for interspecies transmission of avian influenza viruses in mammalian hosts. Int J Mol Sci 2017;18(12):2706.

[74] Duffy S. Why are RNA virus mutation rates so damn high? PLoS Biol 2018;16 (8):e3000003.

[75] Jaramillo D, Fielder S, Whittington RJ, Hick P. Host, agent and environment interactions affecting Nervous necrosis virus infection in Australian bass *Macquaria novemaculeata*. J Fish Dis 2019;42(2):167–80.

[76] Gould EA, Higgs S. Impact of climate change and other factors on emerging arbovirus diseases. Trans R Soc Trop Med Hyg 2009;103(2):109–21.

[77] Leroy E, Gonzalez JP, Pourrut X. Ebolavirus and other filoviruses. In: Childs JE, Mackenzie JS, Richt JA, editors. Wildlife and emerging zoonotic diseases: the biology, circumstances and consequences of cross-species transmission. Berlin: Springer-Verlag; 2007. p. 363–87.

[78] Li M, Yang Y, Lu Y, Zhang D, Liu Y, Cui X, et al. Natural host–environmental media–human: a new potential pathway of COVID-19 outbreak. Engineering 2020;6(10):1085–98.

[79] Londono-Renteria B, Troupin A, Colpitts TM. Arbovirosis and potential transmission blocking vaccines. Parasit Vectors 2016;9(1):516.

[80] Ecology WB. How climate change alters rhythms of the wild. Science 2000;287(5454):793–5.

[81] Colwell RK, Brehm G, Cardelús CL, Gilman AC, Longino JT. Global warming, elevational range shifts, and lowland biotic attrition in the wet tropics. Science 2008;322(5899):258–61.

[82] Legendre M, Lartigue A, Bertaux L, Jeudy S, Bartoli J, Lescot M, et al. In-depth study of *Mollivirus sibericum*, a new 30,000-y-old giant virus infecting *Acanthamoeba*. Proc Natl Acad Sci USA 2015;112(38):E5327–35.

[83] Zhong ZP, Solonenko NE, Li YF, Gazitúa MC, Roux S, Davis ME, et al. Glacier ice archives fifteen-thousand-year-old viruses. 2020. bioRxiv: 2020.01.03. 894675.

[84] Tian H, Hu S, Cazelles B, Chowell G, Gao L, Laine M, et al. Urbanization prolongs hantavirus epidemics in cities. Proc Natl Acad Sci USA 2018;115 (18):4707–12.

[85] Kuiken T, Leighton FA, Fouchier RAM, LeDuc JW, Peiris JSM, Schudel A, et al. Pathogen surveillance in animals. Science 2005;309(5741):1680–1.

[86] Pearce-Duvet JMC. The origin of human pathogens: evaluating the role of agriculture and domestic animals in the evolution of human disease. Biol Rev Camb Philos Soc 2006;81(3):369–82.

[87] Normile D. China's living laboratory in urbanization. Science 2008;319 (5864):740–3.

[88] Sobsey MD, Shields PA, Hauchman FH, Hazard RL, Caton III LW. Survival and transport of hepatitis A virus in soils, groundwater and wastewater. Water Sci Technol 1986;18(10):97–106.

[89] Kimura M, Jia ZJ, Nakayama N, Asakawa S. Ecology of viruses in soils: past, present and future perspectives. Soil Sci Plant Nutr 2008;54(1):1–32.

[90] Blanc R, Nasser A. Effect of effluent quality and temperature on the persistence of viruses in soil. Water Sci Technol 1996;33:237–42.

[91] Allison L, Salter M, Mann G, Howard CR. Thermal inactivation of Pichinde virus. J Virol Methods 1985;11(3):259–64.

[92] Kuzyakov Y, Mason-Jones K. Viruses in soil: nano-scale undead drivers of microbial life, biogeochemical turnover and ecosystem functions. Soil Biol Biochem 2018;127:305–17.

[93] Cook N, Bertrand I, Gantzer C, Pinto RM, Bosch A. Persistence of hepatitis A virus in fresh produce and production environments, and the effect of disinfection procedures: a review. Food Environ Virol 2018;10(3):253–62.

[94] Gundy PM, Gerba CP, Pepper IL. Survival of coronaviruses in water and wastewater. Food Environ Virol 2009;1:10–4.

[95] Lakdawala SS, Lamirande EW, Suguitan Jr AL, Wang W, Santos CP, Vogel L, et al. Eurasian-origin gene segments contribute to the transmissibility, aerosol release, and morphology of the 2009 pandemic H1N1 influenza virus. PLoS Pathog 2011;7(12):e1002443.

[96] Gonzalez-Martin C, Teigell-Perez N, Lyles M, Valladares B, Griffin DW. Epifluorescent direct counts of bacteria and viruses from topsoil of various desert dust storm regions. Res Microbiol 2013;164(1):17–21.

[97] Ashelford KE, Day MJ, Fry JC. Elevated abundance of bacteriophage infecting bacteria in soil. Appl Environ Microbiol 2003;69(1):285–9.

[98] Williamson KE, Radosevich M, Wommack KE. Abundance and diversity of viruses in six Delaware soils. Appl Environ Microbiol 2005;71(6):3119–25.

[99] Swanson MM, Fraser G, Daniell TJ, Torrance L, Gregory PJ, Taliansky M. Viruses in soils: morphological diversity and abundance in the rhizosphere. Ann Appl Biol 2009;155(1):51–60.

[100] Williamson KE, Corzo KA, Drissi CL, Buckingham JM, Thompson CP, Helton RR. Estimates of viral abundance in soils are strongly influenced by extraction and enumeration methods. Biol Fertil Soils 2013;49(7):857–69.

[101] Chen L, Xun W, Sun L, Zhang N, Shen Q, Zhang R. Effect of different long-term fertilization regimes on the viral community in an agricultural soil of Southern China. Eur J Soil Biol 2014;62:121–6.

[102] Amossé J, Bettarel Y, Bouvier C, Bouvier T, Tran DT, Doan TT, et al. The flows of nitrogen, bacteria and viruses from the soil to water compartments are influenced by earthworm activity and organic fertilization (compost vs. vermicompost). Soil Biol Biochem 2013;66:197–203.

[103] Williamson KE, Fuhrmann JJ, Wommack KE, Radosevich M. Viruses in soil ecosystems: an unknown quantity within an unexplored territory. Annu Rev Virol 2017;4(1):201–19.

[104] Van Doremalen N, Bushmaker T, Munster VJ. Stability of Middle East respiratory syndrome coronavirus (MERS-CoV) under different environmental conditions. Euro Surveill 2013;18(38):20590.

[105] Coulliette AD, Perry KA, Edwards JR, Noble-Wang JA. Persistence of the 2009 pandemic influenza A (H1N1) virus on N95 respirators. Appl Environ Microbiol 2013;79(7):2148–55.

[106] Zuo Z, de Abin M, Chander Y, Kuehn TH, Goyal SM, Pui DYH. Comparison of spike and aerosol challenge tests for the recovery of viable influenza virus from non-woven fabrics. Influenza Other Respir Viruses 2013;7(5):637–44.

[107] Mukherjee DV, Cohen B, Bovino ME, Desai S, Whittier S, Larson EL. Survival of influenza virus on hands and fomites in community and laboratory settings. Am J Infect Control 2012;40(7):590–4.

[108] Greatorex JS, Digard P, Curran MD, Moynihan R, Wensley H, Wreghitt T, et al. Survival of influenza A (H1N1) on materials found in households: implications for infection control. PLoS ONE 2011;6(11):e27932.

[109] Dublineau A, Batéjat C, Pinon A, Burguière AM, Leclercq I, Manuguerra JC. Persistence of the 2009 pandemic influenza A (H1N1) virus in water and on non-porous surface. PLoS ONE 2011;6(11):e28043.

[110] Wood JP, Choi YW, Chappie DJ, Rogers JV, Kaye JZ. Environmental persistence of a highly pathogenic avian influenza (H5N1) virus. Environ Sci Technol 2010;44(19):7515–20.

[111] Sakaguchi H, Wada K, Kajioka J, Watanabe M, Nakano R, Hirose T, et al. Maintenance of influenza virus infectivity on the surfaces of personal protective equipment and clothing used in healthcare settings. Environ Health Prev Med 2010;15(6):344–9.

[112] Tiwari A, Patnayak DP, Chander Y, Parsad M, Goyal SM. Survival of two avian respiratory viruses on porous and nonporous surfaces. Avian Dis 2006;50 (2):284–7.

[113] Bean B, Moore BM, Sterner B, Peterson LR, Gerding DN, Balfour Jr HH. Survival of influenza viruses on environmental surfaces. J Infect Dis 1982;146 (1):47–51.

[114] Chan KH, Peiris JSM, Lam SY, Poon LLM, Yuen KY, Seto WH. The effects of temperature and relative humidity on the viability of the SARS coronavirus. Adv Virol 2011;2011:734690.

[115] Ijaz MK, Brunner AH, Sattar SA, Nair RC, Johnson-Lussenburg CM. Survival characteristics of airborne human coronavirus 229E. J Gen Virol 1985;66(Pt 12):2743–8.

[116] Sizun J, Yu MWN, Talbot PJ. Survival of human coronaviruses 229E and OC43 in suspension and after drying on surfaces: a possible source of hospital-acquired infections. J Hosp Infect 2000;46(1):55–60.

[117] Kim SH, Chang SY, Sung M, Park JH, Kim HB, Lee H, et al. Extensive viable Middle East respiratory syndrome (MERS) coronavirus contamination in air and surrounding environment in MERS isolation wards. Clin Infect Dis 2016;63(3):363–9.

[118] Rabenau HF, Cinatl J, Morgenstern B, Bauer G, Preiser W, Doerr HW. Stability and inactivation of SARS coronavirus. Med Microbiol Immunol 2005;194(1–2):1–6.

[119] Warnes SL, Little ZR, Keevil CW. Human coronavirus 229E remains infectious on common touch surface materials. mBio 2015;6(6):e01697–e1715.

[120] Weber DJ, Sickbert-Bennett EE, Kanamori H, Rutala WA. New and emerging infectious diseases (Ebola, Middle Eastern respiratory syndrome coronavirus, carbapenem-resistant Enterobacteriaceae, *Candida auris*): focus on environmental survival and germicide susceptibility. Am J Infect Control 2019;47(Suppl):A29–38.

[121] Zhao B, Zhang H, Zhang J, Jin Y. Virus adsorption and inactivation in soil as influenced by autochthonous microorganisms and water content. Soil Biol Biochem 2008;40(3):649–59.

[122] Yeager JG, O'Brien RT. Enterovirus inactivation in soil. Appl Environ Microbiol 1979;38(4):694–701.

[123] Yeager JG, O'Brien RT. Structural changes associated with poliovirus inactivation in soil. Appl Environ Microbiol 1979;38(4):702–9.

[124] Taylor LH, Latham SM, Woolhouse MEJ. Risk factors for human disease emergence. Philos Trans R Soc Lond B Biol Sci 2001;356(1411):983–9.

[125] Plowright RK, Eby P, Hudson PJ, Smith IL, Westcott D, Bryden WL, et al. Ecological dynamics of emerging bat virus spillover. Proc Biol Sci 2015;282 (1798):20142124.

[126] Murthy S, Couacy-Hymann E, Metzger S, Nowak K, De Nys H, Boesch C, et al. Absence of frequent herpesvirus transmission in a nonhuman primate predator-prey system in the wild. J Virol 2013;87(19):10651–9.

[127] Gao R, Cao B, Hu Y, Feng Z, Wang D, Hu W, et al. Human infection with a novel avian-origin influenza A (H7N9) virus. N Engl J Med 2013;368(20):1888–97.
[128] Daszak P, Zambrana-Torrelio C, Bogich TL, Fernandez M, Epstein JH, Murray KA, et al. Interdisciplinary approaches to understanding disease emergence: the past, present, and future drivers of Nipah virus emergence. Proc Natl Acad Sci USA 2013;110(Suppl 1):3681–8.
[129] Parmesan C, Yohe G. A globally coherent fingerprint of climate change impacts across natural systems. Nature 2003;421(6918):37–42.
[130] McMichael AJ, Woodruff RE, Hales S. Climate change and human health: present and future risks. Lancet 2006;367(9513):859–69.
[131] Paz S. The West Nile virus outbreak in Israel (2000) from a new perspective: the regional impact of climate change. Int J Environ Health Res 2006;16(1):1–13.
[132] Gale P, Brouwer A, Ramnial V, Kelly L, Kosmider R, Fooks AR, et al. Assessing the impact of climate change on vector-borne viruses in the EU through the elicitation of expert opinion. Epidemiol Infect 2010;138(2):214–25.
[133] Jupp PG. Laboratory studies on the transmission of West Nile virus by Culex (Culex) univittatus Theobald; factors influencing the transmission rate. J Med Entomol 1974;11(4):455–8.
[134] Epstein PR, Diaz HF, Elias SA, Grabherr G, Graham NE, Martens WJM, et al. Biological and physical signs of climate change: focus on mosquito-borne diseases. Bull Amer Meteor Soc 1998;79(3):409–18.
[135] Watts N, Amann M, Ayeb-Karlsson S, Belesova K, Bouley T, Boykoff M, et al. The Lancet countdown on health and climate change: from 25 years of inaction to a global transformation for public health. Lancet 2018;391 (10120):581–630.
[136] Legendre M, Bartoli J, Shmakova L, Jeudy S, Labadie K, Adrait A, et al. Thirty-thousand-year-old distant relative of giant icosahedral DNA viruses with a pandoravirus morphology. Proc Natl Acad Sci USA 2014;111(11):4274–9.
[137] Hu H, Nigmatulina K, Eckhoff P. The scaling of contact rates with population density for the infectious disease models. Math Biosci 2013; 244(2):125–34.
[138] Sutherst RW. Global change and human vulnerability to vector-borne diseases. Clin Microbiol Rev 2004;17(1):136–73.
[139] Hing S, Narayan EJ, Thompson RCA, Godfrey SS. The relationship between physiological stress and wildlife disease: consequences for health and conservation. Wildl Res 2016;43(1):51–60.
[140] Apanius V. Stress and immune defense. Adv Stud Behav 1998;27:133–53.
[141] Dietrich MO, Zimmer MR, Bober J, Horvath TL. Hypothalamic Agrp neurons drive stereotypic behaviors beyond feeding. Cell 2015;160(6):1222–32.
[142] Bradley CA, Altizer S. Urbanization and the ecology of wildlife diseases. Trends Ecol Evol 2007;22(2):95–102.
[143] Walsh MG, Mor SM, Maity H, Hossain S. Forest loss shapes the landscape suitability of Kyasanur forest disease in the biodiversity hotspots of the Western Ghats, India. Int J Epidemiol 2019;48(6):1804–14.
[144] MacDonald AJ, Larsen AE, Plantinga AJ. Missing the people for the trees: identifying coupled natural–human system feedbacks driving the ecology of Lyme disease. J Appl Ecol 2019;56(2):354–64.
[145] Estrada-Peña A, Zatansever Z, Gargili A, Aktas M, Uzun R, Ergonul O, et al. Modeling the spatial distribution of Crimean–Congo hemorrhagic fever outbreaks in Turkey. Vector Borne Zoonotic Dis 2007;7(4):667–78.
[146] Luby SP, Rahman M, Hossain MJ, Blum LS, Husain MM, Gurley E, et al. Foodborne transmission of Nipah virus, Bangladesh. Emerg Infect Dis 2006;12(12):1888–94.
[147] Khan MSU, Hossain J, Gurley ES, Nahar N, Sultana R, Luby SP. Use of infrared camera to understand bats' access to date palm sap: implications for preventing Nipah virus transmission. EcoHealth 2010;7(4):517–25.
[148] Leroy EM, Epelboin A, Mondonge V, Pourrut X, Gonzalez JP, Muyembe-Tamfum JJ, et al. Human Ebola outbreak resulting from direct exposure to fruit bats in Luebo, Democratic Republic of Congo, 2007. Vector Borne Zoonotic Dis 2009;9(6):723–8.
[149] Swanepoel R, Leman PA, Burt FJ, Zachariades NA, Braack LEO, Ksiazek TG, et al. Experimental inoculation of plants and animals with Ebola virus. Emerg Infect Dis 1996;2(4):321–5.
[150] Bøtner A, Belsham GJ. Virus survival in slurry: analysis of the stability of foot-and-mouth disease, classical swine fever, bovine viral diarrhoea and swine influenza viruses. Vet Microbiol 2012;157(1–2):41–9.
[151] Seitz SR, Leon JS, Schwab KJ, Lyon GM, Dowd M, McDaniels M, et al. Norovirus infectivity in humans and persistence in water. Appl Environ Microbiol 2011;77(19):6884–8.
[152] Ong SWX, Tan YK, Chia PY, Lee TH, Ng OT, Wong MSY, et al. Air, surface environmental, and personal protective equipment contamination by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) from a symptomatic patient. JAMA 2020;323(16):1610–2.
[153] Zhang D, Yang Y, Huang X, Jiang J, Li M, Zhang X, et al. SARS-CoV-2 spillover into hospital outdoor environments. 2020. medRxiv: 2020.05.12.20097105.
[154] Van Doremalen N, Bushmaker T, Morris DH, Holbrook MG, Gamble A, Williamson BN, et al. Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1. N Engl J Med 2020;382(16):1564–7.
[155] Mackenzie JS, Williams DT. The zoonotic flaviviruses of southern, south-eastern and eastern Asia, and Australasia: the potential for emergent viruses. Zoonoses Public Health 2009;56(6–7):338–56.
[156] Mansfield KL, Lv JZ, Phipps LP, Johnson N. Emerging tick-borne viruses in the twenty-first century. Front Cell Infect Microbiol 2017;7:298.
[157] Bondad-Reantaso MG, Subasinghe RP, Arthur JR, Ogawa K, Chinabut S, Adlard R, et al. Disease and health management in Asian aquaculture. Vet Parasitol 2005;132(3–4):249–72.
[158] Hutson CL, Lee KN, Abel J, Carroll DS, Montgomery JM, Olson VA, et al. Monkeypox zoonotic associations: insights from laboratory evaluation of animals associated with the multi-state US outbreak. Am J Trop Med Hyg 2007;76(4):757–68.
[159] Kilpatrick AM, Chmura AA, Gibbons DW, Fleischer RC, Marra PP, Daszak P. Predicting the global spread of H5N1 avian influenza. Proc Natl Acad Sci USA 2006;103(51):19368–73.
[160] Schikora A, Garcia AV, Hirt H. Plants as alternative hosts for Salmonella. Trends Plant Sci 2012;17(5):245–9.
[161] Strawn LK, Schneider KR, Danyluk MD. Microbial safety of tropical fruits. Crit Rev Food Sci Nutr 2011;51(2):132–45.
[162] Leroy EM, Kumulungui B, Pourrut X, Rouquet P, Hassanin A, Yaba P, et al. Fruit bats as reservoirs of Ebola virus. Nature 2005;438(7068):575–6.
[163] Tang X, Wu C, Li X, Song Y, Yao X, Wu X, et al. On the origin and continuing evolution of SARS-CoV-2. Natl Sci Rev 2020;7(6):1012–23.
[164] Chen W, Yan M, Yang L, Ding B, He B, Wang Y, et al. SARS-associated coronavirus transmitted from human to pig. Emerg Infect Dis 2005;11 (3):446–8.
[165] Murray K, Selleck P, Hooper P, Hyatt A, Gould A, Gleeson L, et al. A morbillivirus that caused fatal disease in horses and humans. Science 1995;268(5207):94–7.
[166] Selvey LA, Wells RM, McCormack JG, Ansford AJ, Murray K, Rogers RJ, et al. Infection of humans and horses by a newly described morbillivirus. Med J Aust 1995;162(12):642–5.
[167] Playford EG, McCall B, Smith G, Slinko V, Allen G, Smith I, et al. Human Hendra virus encephalitis associated with equine outbreak, Australia, 2008. Emerg Infect Dis 2010;16(2):219–23.
[168] Hooper PT, Gould AR, Russell GM, Kattenbelt JA, Mitchell G. The retrospective diagnosis of a second outbreak of equine morbillivirus infection. Aust Vet J 1996;74(3):244–5.
[169] O'Sullivan JD, Allworth AM, Paterson DL, Snow TM, Boots R, Gleeson LJ, et al. Fatal encephalitis due to novel paramyxovirus transmitted from horses. Lancet 1997;349(9045):93–5.
[170] Muhairi SA, Hosani FA, Eltahir YM, Mulla MA, Yusof MF, Serhan WS, et al. Epidemiological investigation of Middle East respiratory syndrome coronavirus in dromedary camel farms linked with human infection in Abu Dhabi Emirate, United Arab Emirates. Virus Genes 2016;52(6):848–54.
[171] Bermingham A, Chand MA, Brown CS, Aarons E, Tong C, Langrish C, et al. Severe respiratory illness caused by a novel coronavirus, in a patient transferred to the United Kingdom from the Middle East, September 2012. Euro Surveill 2012;17(40):20290.
[172] Al-Tawfiq JA, Zumla A, Memish ZA. Travel implications of emerging coronaviruses: SARS and MERS-CoV. Travel Med Infect Dis 2014;12(5):422–8.
[173] Van Reeth K. Avian and swine influenza viruses: our current understanding of the zoonotic risk. Vet Res 2007;38(2):243–60.
[174] Alexander DJ. An overview of the epidemiology of avian influenza. Vaccine 2007;25(30):5637–44.
[175] Michaelis M, Doerr HW, Cinatl Jr J. An influenza A H1N1 virus revival-pandemic H1N1/09 virus. Infection 2009;37(5):381–9.
[176] Tanner WD, Toth DJA, Gundlapalli AV. The pandemic potential of avian influenza A (H7N9) virus: a review. Epidemiol Infect 2015;143(16):3359–74.
[177] Tang RB, Chen HL. An overview of the recent outbreaks of the avian-origin influenza A (H7N9) virus in the human. J Chin Med Assoc 2013;76(5):245–8.
[178] Katayama H, Haramoto E, Oguma K, Yamashita H, Tajima A, Nakajima H, et al. One-year monthly quantitative survey of noroviruses, enteroviruses, and adenoviruses in wastewater collected from six plants in Japan. Water Res 2008;42(6–7):1441–8.
[179] Lodder WJ, de Roda Husman AM. Presence of noroviruses and other enteric viruses in sewage and surface waters in the Netherlands. Appl Environ Microbiol 2005;71(3):1453–61.
[180] Sahlström L. A review of survival of pathogenic bacteria in organic waste used in biogas plants. Bioresour Technol 2003;87(2):161–6.
[181] Wu C, Maurer C, Wang Y, Xue S, Davis DL. Water pollution and human health in China. Environ Health Perspect 1999;107(4):251–6.
[182] Nithiuthai S, Anantaphruti MT, Waikagul J, Gajadhar A. Waterborne zoonotic helminthiases. Vet Parasitol 2004;126(1–2):167–93.
[183] Karetnyi YV, Gilchrist MJR, Naides SJ. Hepatitis E virus infection prevalence among selected populations in Iowa. J Clin Virol 1999;14(1):51–5.
[184] Kasorndorkbua C, Guenette DK, Huang FF, Thomas PJ, Meng XJ, Halbur PG. Routes of transmission of swine hepatitis E virus in pigs. J Clin Microbiol 2004;42(11):5047–52.
[185] Meng XJ. From barnyard to food table: the omnipresence of hepatitis E virus and risk for zoonotic infection and food safety. Virus Res 2011;161(1):23–30.
[186] Yugo DM, Meng XJ. Hepatitis E virus: foodborne, waterborne and zoonotic transmission. Int J Environ Res Public Health 2013;10(10):4507–33.
[187] Vaidya SR, Tilekar BN, Walimbe AM, Arankalle VA. Increased risk of hepatitis E in sewage workers from India. J Occup Environ Med 2003;45(11):1167–70.
[188] Strunz EC, Addiss DG, Stocks ME, Ogden S, Utzinger J, Freeman MC. Water, sanitation, hygiene, and soil-transmitted helminth infection: a systematic review and meta-analysis. PLoS Med 2014;11(3):e1001620.
[189] Bancalari A, Martinez S. Exposure to sewage from on-site sanitation and child health: a spatial analysis of linkages and externalities in peri-urban Bolivia. J Water Sanit Hyg Dev 2018;8(1):90–9.
[190] Chen KT, Chang HL, Wang ST, Cheng YT, Yang JY. Epidemiologic features of hand–foot–mouth disease and herpangina caused by enterovirus 71 in Taiwan, 1998–2005. Pediatrics 2007;120(2):e244–52.

[191] Xing W, Liao Q, Viboud C, Zhang J, Sun J, Wu JT, et al. Hand, foot, and mouth disease in China, 2008–12: an epidemiological study. Lancet Infect Dis 2014;14(4):308–18.

[192] Ryder RW, Nsa W, Hassig SE, Behets F, Rayfield M, Ekungola B, et al. Perinatal transmission of the human immunodeficiency virus type 1 to infants of seropositive women in Zaire. N Engl J Med 1989;320(25):1637–42.

[193] Hay SI, Guerra CA, Tatem AJ, Atkinson PM, Snow RW. Urbanization, malaria transmission and disease burden in Africa. Nat Rev Microbiol 2005;3 (1):81–90.

[194] Pickett STA, Cadenasso ML, Grove JM, Boone CG, Groffman PM, Irwin E, et al. Urban ecological systems: scientific foundations and a decade of progress. J Environ Manage 2011;92(3):331–62.

[195] Weaver SC, Costa F, Garcia-Blanco MA, Ko AI, Ribeiro GS, Saade G, et al. Zika virus: history, emergence, biology, and prospects for control. Antiviral Res 2016;130:69–80.

[196] Counotte MJ, Kim CR, Wang J, Bernstein K, Deal CD, Broutet NJN, et al. Sexual transmission of Zika virus and other flaviviruses: a living systematic review. PLoS Med 2018;15(7):e1002611.

[197] Rimoldi SG, Stefani F, Gigantiello A, Polesello S, Comandatore F, Mileto D, et al. Presence and infectivity of SARS-CoV-2 virus in wastewaters and rivers. Sci Total Environ 2020;744:140911.

[198] Graiver DA, Topliff CL, Kelling CL, Bartelt-Hunt SL. Survival of the avian influenza virus (H6N2) after land disposal. Environ Sci Technol 2009;43 (11):4063–7.

[199] Hatch JJ. Threats to public health from gulls (Laridae). Int J Environ Health Res 1996;6(1):5–16.

[200] Mudge GP, Ferns PN. The feeding ecology of five species of gulls (Ayes: Larini) in the inner Bristol Channel. J Zool 1982;197(4):497–510.

[201] Carducci A, Federigi I, Verani M. Virus occupational exposure in solid waste processing facilities. Ann Occup Hyg 2013;57(9):1115–27.

[202] Duh D, Hasic S, Buzan E. The impact of illegal waste sites on a transmission of zoonotic viruses. Virol J 2017;14(1):134.

[203] Costa T, Akdeniz N. A review of the animal disease outbreaks and biosecure animal mortality composting systems. Waste Manag 2019;90:121–31.

[204] Simonsen L, Chowell G, Andreasen V, Gaffey R, Barry J, Olson D, et al. A review of the 1918 herald pandemic wave: importance for contemporary pandemic response strategies. Ann Epidemiol 2018;28(5):281–8.

[205] Vincent A, Awada L, Brown I, Chen H, Claes F, Dauphin G, et al. Review of influenza A virus in swine worldwide: a call for increased surveillance and research. Zoonoses Public Health 2014;61(1):4–17.

[206] Short KR, Richard M, Verhagen JH, van Riel D, Schrauwen EJA, van den Brand JMA, et al. One health, multiple challenges: the inter-species transmission of influenza A virus. One Health 2015;1:1–13.

[207] Andrew TB, Allan JR. Use of raptors to reduce scavenging bird numbers at landfill sites. Wildl Soc Bull 2006;34(4):1162–8.

[208] Camacho M, Hernández JM, Lima-Barbero JF, Höfle U. Use of wildlife rehabilitation centres in pathogen surveillance: a case study in white storks (Ciconia ciconia). Prev Vet Med 2016;130:106–11.

[209] Munster VJ, Baas C, Lexmond P, Waldenström J, Wallensten A, Fransson T, et al. Spatial, temporal, and species variation in prevalence of influenza A viruses in wild migratory birds. PLoS Pathog 2007;3(5):e61.

[210] Plaza PI, Blanco G, Madariaga MJ, Boeri E, Teijeiro ML, Bianco G, et al. Scavenger birds exploiting rubbish dumps: pathogens at the gates. Transbound Emerg Dis 2019;66(2):873–81.

[211] Southgate V, Tchuem Tchuenté LA, Sène M, De Clercq D, Théron A, Jourdane J, et al. Studies on the biology of schistosomiasis with emphasis on the Senegal river basin. Mem Inst Oswaldo Cruz 2001;96(Suppl):75–8.

[212] Reiter P. Climate change and mosquito-borne disease: knowing the horse before hitching the cart. Rev Sci Tech 2008;27(2):383–98.

[213] Zhang D, Ling H, Huang X, Li J, Li W, Yi C, et al. Potential spreading risks and disinfection challenges of medical wastewater by the presence of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) viral RNA in septic tanks of Fangcang Hospital. Sci Total Environ 2020;741:140445.

[214] Ahmed W, Angel N, Edson J, Bibby K, Bivins A, O'Brien JW, et al. First confirmed detection of SARS-CoV-2 in untreated wastewater in Australia: a proof of concept for the wastewater surveillance of COVID-19 in the community. Sci Total Environ 2020;728:138764.

[215] Bhowmick GD, Dhar D, Nath D, Ghangrekar MM, Banerjee R, Das S, et al. Coronavirus disease 2019 (COVID-19) outbreak: some serious consequences with urban and rural water cycle. npj Clean Water 2020;3(1):1–8.

[216] Zhang YZ, Zou Y, Fu ZF, Plyusnin A. Hantavirus infections in humans and animals. China Emerg Infect Dis 2010;16(8):1195–203.

[217] Chandra V, Taneja S, Kalia M, Jameel S. Molecular biology and pathogenesis of hepatitis E virus. J Biosci 2008;33(4):451–64.