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A REVIEW OF EMERGING AND RE-EMERGING ZOONOTIC VIRAL DISEASES OVER FIFTY-EIGHT YEARS WITH A 'ONE HEALTH' PERSPECTIVE IN BANGLADESH

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ABSTRACT

Background: Zoonotic viral diseases (ZVDs), caused by RNA viruses, especially dengue, were first reported in 1966 in East Pakistan (now Bangladesh) and have since been followed by a vast number of studies on emerging and re-emerging ZVDs from Bangladesh. Over the past few decades, approximately 30 emerging viral diseases have been reported in outbreaks globally, including those in Bangladesh, of which 70% are zoonotic in origin. However, a review of these emerging and re-emerging ZVDs has been reported elsewhere, but not explicitly noted in Bangladesh.

Objective: This review provides an overview of significant emerging and re-emerging ZVDs, with special emphasis on Bangladesh.

Materials and Methods: A literature search was conducted using the Web of Science, PubMed, Google Scholar, and Scopus databases, and reports on emerging and re-emerging ZVDs were reviewed from 1966 to June 2025, approximately 58 years of reported findings.

Results: The emerging and re-emerging ZVDs are reported as sporadic and outbreak forms globally, with Bangladesh experiencing a significant burden from most of these diseases. This review has detected both the emerging (e.g., Avian influenza, Nipah virus infection (NVI), severe acute respiratory syndrome (SARS), Middle east respiratory syndrome (MERS), and Coronavirus-19 (COVID-19) and re-emerging (e.g., Japanese encephalitis, Dengue, Swine influenza, Rotavirus, Chikungunya fever and Zika virus infection) ZVDs reported in Bangladesh associated with morbidity and mortality in human populations. The pandemic ZVDs have been reported in Bangladesh from 1966 to June 2025 and were categorized into three groups. The first group comprises highly pathogenic avian influenza (HPAI-H5N1) reported in 11 human cases with one death. Moreover, HPAI-H5N1 infection caused 90 to 100% death in poultry and economic losses of > US$10 billion worldwide. Similarly, H1N1 caused a severe infection in swine, resulting in pandemic swine influenza, which led to 6,000 human deaths in Bangladesh in 2009. This outbreak also resulted in Dhaka incurring US$6.1 million in direct medical costs for patients. In addition, both outdoor (6.45%) and indoor (100%) patients reported positive for H1N1, with deaths due to respiratory failure. Swine influenza virus is circulating in pig populations, including those in the Hill Tract districts (12.22%) of Bangladesh. The second group consists of SARS-CoV-2, which caused a total of 2,049,377 clinical cases, of which deaths have been reported in 29,493 (22.34%) of the human population up to 13 April 2024 in Bangladesh. The MERS-CoV infection has only been recorded in a Bangladeshi man who lives in the USA, was diagnosed with this virus, probably infected while traveling by air from the USA to Bangladesh via Abu Dhabi airport. However, 31.0% imported camels tested with ELISA showed positive for MERS-CoV infection in Bangladesh. The third group consists of Zika virus infections, with the first confirmed case detected in 2016, with the sample collected in 2014, and 420 patients with Guillain-Barre syndrome (GBS) were diagnosed with ZIKV infection over five years from 2011 to 2015 in Bangladesh. Eight cases of ZIKA virus infection were reported in Dhaka in 2024. Additionally, five ZIKV-positive cases were identified, including those with dengue-ZIKV co-infections, during the 2023 study outbreak in Bangladesh. Bangladesh experienced a sharp increase in dengue cases in 2023, with 321,179 reported cases and 1,705 deaths. Chikungunya was first reported in 2008, followed by six recorded hospital patients in Dhaka city. Then Bangladesh experienced the largest CHIKV outbreak with 13,176 cases in 17 out of 64 districts, primarily in Dhaka. ZVDs can be transmitted to humans through various routes, including direct (e.g., Rabies) or indirect (e.g., Hantavirus) contact, nosocomial (e.g., Ebola virus), aerosols (SARS coronavirus), vertical (e.g., Zika virus- in utero), and vector or arthropod-borne (e.g., ZIKV and CHIKV). Over the past three decades, outbreaks of emerging and re-emerging ZVDs underscore the urgent need for integrated surveillance systems, early detection strategies, and susceptible intervention to mitigate future risks. Risk factors such as deforestation, climate change, unregulated wildlife trade, and intensive farming practices exacerbate the spread of zoonotic diseases. The etiology, clinical manifestations, transmission routes, and prevention of these ZVDs are briefly described and discussed.

Conclusions: The extreme diversity of emerging and re-emerging zoonotic RNA viral pathogens, along with changes in human lifestyle, the globalization of travel, business exchanges, and tourism, all potentiate the risk of the emergence of highly pathogenic infectious viral zoonotic diseases. Therefore, any public health prophylactic strategy requires a holistic approach to the health problem, considering the interactions of the 'One Health' approach. The program of this complex issue of emerging and re-emerging ZVDs should include modernizing the epidemiological surveillance system based on universal monitoring, an interconnecting 'One Health' approach, and an effective national health monitoring system compatible with its global counterpart.

Keywords: Fifty-eight years, Zoonotic viral diseases, RNA viruses, Emerging and re-emerging ZVDs, Rabies, Japanese encephalitis, Dengue fever, Avian influenza, Nipah virus infection, Swine influenza, Rotavirus, COVID-19, SARS-CoV-2, MERS-CoV, Chikungunya, Zika virus infection, 'One Health' approach, Bangladesh

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INTRODUCTION

The term zoonosis comes from the Greek words 'zoon', meaning animal, and 'osis', meaning condition (illness). Any disease or infection that can spread naturally from vertebrate animals to humans or from humans to vertebrate animals is referred to as a zoonotic disease.[1] However, most zoonotic pathogens are transmitted from animals to humans. Conversely, susceptible animals can also be infected with pathogens from humans, a phenomenon known as reverse zoonosis.[2] Zoonotic diseases are estimated to be responsible for 2.5 billion cases of human illness globally per year, resulting in 2.7 million deaths.[3,4] A comprehensive literature review identifies 1,415 species of pathogens known to infect humans, of which 868 (61.0%) are zoonotic, meaning they can be transmitted between humans and animals.[5] Additionally, 175 (75%) pathogenic species are associated with diseases considered to be emerging zoonotic.[5] Both the emerging and re-emerging zoonotic viral diseases (ZVDs) represent a significant public health problem globally, including Bangladesh. The 'emerging diseases' refer to the new occurrence of a disease, infection, or infestation that causes a significant impact on animals and/or public health, such as SARS and Avian flu.[6] Most of the emerging ZVDs are often previously unrecognized diseases or have increased virulence in populations lacking immunity, such as henipavirus,[7] severe acute respiratory syndrome (SARS), and influenza virus (swine-origin H1N1 or avian influenza H5N1).[8] A re-emerging disease that was once a major health problem, then declined, but has recently reoccurred, leading to significant health complications like plague and yellow fever.[9] Over 60% of existing and 75% of emerging and re-emerging human diseases are zoonotic,[5,6] and of these diseases, approximately 72% originate in wildlife. In contrast, only 25% of these pathogens originate from domestic animal species.[1,5] Mammals, birds, reptiles, and possibly amphibians are reservoirs or amplifying hosts for zoonotic viral infections (ZVI).[10] Over 36% of emerging and re-emerging zoonotic diseases are associated with food-producing animals.[11] Transmission of zoonotic viruses may occur by a variety of routes, including direct (e.g., rabies) or indirect (e.g., hantavirus) contact, nosocomial (e.g., Ebola virus), aerosol transmission (SARS-CoV), vertical in utero (Zika virus), and vector- or arthropod-borne (e.g., yellow fever virus and West Nile virus).[12] A range of US$22-31.2 billion per year would be needed globally for prevention measures that reduce the risks of zoonotic spillover.[13] Bangladesh is a high-risk location for zoonotic spillover, characterized by its tropical latitude, high wildlife diversity, dense human and domestic animal populations, and high connectivity between people, domestic animals, and wildlife.[14] Some review articles on the major zoonotic diseases have been reported from Bangladesh,[15-18] and bacterial zoonotic diseases in the light of the 'One Health' approach with multidrug resistance status have been documented in Bangladesh.[19] This paper presents a comprehensive review of emerging and re-emerging zoonotic viral diseases, primarily based on inland research reports published from Bangladesh.

MATERIALS AND METHODS

A literature search for zoonotic viral diseases (ZVDs) was carried out, and articles were retrieved by performing a search using online electronic databases (Google Scholar, PubMed, Medline Plus, Research Gate, Web of Science, and Scopus). The keywords used for the search include emerging ZVDs, re-emerging ZVDs, Ebola virus infection, COVID-19, MERS-CoV, SARS-CoV, Zika virus, H1N1, H5N1, zoonosis, and 'One Health.' The review encompassed major outbreaks caused by zoonotic viral infections that occurred over a period of more than half a century (1966 to June 2025). Based on the keywords used for the literature search, a total of 390 documents were reviewed from the databases of these websites, comprising 209 articles from inland sources and 181 from overseas sources. Relevant scientific information on the etiologic agents, public health impact (morbidity and mortality in humans), animal health impacts (morbidity and economic losses), and control measures was extracted from these publications. The cumulative morbidity and case fatalities of individual viral diseases were calculated using the number of deaths and cases.

RESULTS AND DISCUSSION

Zoonotic viruses are primarily associated with emerging and re-emerging zoonotic diseases, which constitute an ongoing threat to both human and animal health worldwide. There are three classes of zoonotic diseases, which include (a) endemic zoonoses, which are present in many countries and affect many people and animals, (b) epidemic zoonoses which are sporadic in temporal and spatial distribution, and (c) emerging and re-emerging zoonoses which are newly appearing in a population or have existed previously but are rapidly increasing in incidence or geographical range. The emergence of several high-impact zoonotic viruses during the past few decades represents a growing global concern for human health and severe economic consequences. Most important of these zoonotic viruses and their induced diseases are Avian influenza (H5N1 and H7N9), Ebola virus disease, Middle East respiratory syndrome (MERS), severe acute respiratory syndrome (SARS), SARS-CoV-2 (COVID-19), Yellow fever, Nipah virus, Monkeypox (Mpox), Zika virus, Rift Valley fever, Hantavirus, West Nile virus, Marburg virus disease, and Rabies. Over 30 new human pathogens have been detected in the last three decades, 75% of which have originated in animals[1] and these pathogens responsible for emerging and re-emerging animal diseases present a potential transgression of interface between interspecies establishing favorable conditions for genetic exchange leading to the emergence of new highly pathogenic variants and strains of which the animals are often the host reservoir.[20] General factors contributing to the emergence and re-emergence of zoonotic diseases are: (a) Habitat destruction and deforestation- these activities can bring into closer contact with wildlife and increase the risk of disease transmission, (b) Climate change- Changes in temperature, precipitation, and other environmental factors can impact the distribution of animal populations and vectors that carry infectious diseases, (c) Increased human population and urbanization- these factors can lead to greater exposure to animals and increased opportunities for disease transmission, (d) Globalization and international travel- these factors can facilitate the rapid spread of infectious diseases across geographical boundaries, and (e) Changes in agricultural practices- intensive farming and the proximity of livestock can increase the risk of zoonotic disease outbreaks.

Acute respiratory signs, including highly pathogenic avian influenza (H5N1, H7N7, and H9N2) and Middle East respiratory syndrome (MERS), have periodically led to international panic and necessitated the implementation of health measures to control outbreaks. Vector-borne viral diseases (West Nile, Chikungunya, Zika, Dengue fever, Rift Valley fever, etc.) generate approximately one million deaths annually. Bat-borne zoonotic diseases add a supplementary challenge to the global health community, and their role has been demonstrated in the emergence and re-emergence of many serious and highly publicized viral infectious diseases [Ebola and Marburg virus (re-emergence in 2014), Hendra virus (emergence in 1994), Nipah virus (emergence in 1997).[20] Most of the emerging zoonotic viruses are RNA types that pose a particularly high zoonotic risk because they can emerge and spread rapidly. A statistical analysis of 146 livestock viruses revealed that the ability of a virus to replicate in the cytoplasm is the strongest single predictor of cross-species transmission and its capacity to infect humans.[21] Table 1 provides some epidemiological features of major viral zoonotic infections.

Characteristics of the zoonotic viruses

There are two classes of viruses: DNA viruses and RNA viruses. Either DNA or RNA viruses may have single or double strands of genetic material. RNA viruses can be divided into two categories: single-stranded RNA (ssRNA) and double-stranded RNA (dsRNA). Among these, single-stranded RNA is further classified into positive-strand (+ssRNA) and negative-strand (-ssRNA) RNA. Zoonotic negative-sense RNA viruses pose a major threat to animal and human health and have caused numerous significant outbreaks, including the 1918 Spanish influenza pandemic, the 2009 swine influenza pandemic, Ebola virus outbreaks, Rift Valley fever virus outbreaks, and the H5 highly pathogenic avian influenza virus (HPAIV) panzootic [22]

Viral zoonotic diseases caused by RNA viruses have several characteristics in common, including: (a) genomes that may undergo genetic recombination or reassortment when infecting a single cell and thus have a high preponderance of forming new virus variants; (b) the natural reservoirs of all these viruses, or their precursors, are vertebrate animals, and (c) all of the RNA viruses associated with zoonotic infection can jump the species barrier by crossing from their natural hosts into new species, including humans, in which they may cause deadly disease.[23]

Epidemiology and public health impact of zoonotic virus diseases

Table 1 presents the epidemiology of the major emerging and re-emerging viral zoonotic infections. Table 2 provides an overview of zoonotic viral infections, while Table 3 presents an overview of genomic features, classification, therapy, and prevention of zoonotic viral diseases. Among the ZVDs caused by RNA viruses recorded in this review, the highest cumulative case fatalities of HPAI-H5N1 (53.5%), Ebola virus (39.5%), MERS-CoV (34.3%), whereas comparatively lower cumulative case fatalities in Zika virus (0.02%), H1N1 (0.5%), and SARS-CoV-2 (2.0%) infections in humans (Table 4).

Table 1. Epidemiology of the major emerging and re-emerging viral zoonotic infections[24]

SN Geographical distribution	Causal virus	Animal hosts/Reservoirs	Mode of transmission
① Global distribution	Rabies virus	All warm-blooded animals. especially dogs	Bite of an infected animal primarily dogs.
	Hantavirus	Rodents	Aerosolized rodent urine/feces, saliva, or bite of infected rodents
	Yellow fever virus	Non-human primates (NHP)	Bites of infected mosquitoes (primarily *Aedes* spp.)
	Chikungunya virus	NHP, rodents, birds, small mammals	Bites of infected mosquitoes (primarily Aedes spp.)
	Crimean-Congo Hemorrhagic fever virus	Domestic ruminants (cattle, sheep, goats & camels)	Bite of infected ticks (mainly *Hyalomma* spp.). Direct contact with blood or tissues of infected ticks, viremic livestock, or viremic humans.
	West Nile Virus	Birds	Bite of infected mosquitoes (primarily *Culex* spp.)
②. Africa / Middle East	Rift Valley fever virus	Domestic ruminants (cattle, sheep, goats & camels)	Bite of infected mosquitoes (*Aedes* spp.), contact with blood or organs of infected animals, or inhalation of aerosol during slaughter, unpasteurized milk
	Ebola virus	Fruit bats, NHP, antelopes, porcupines	Contact with blood, secretions, organs, or other body fluids of infected animals or humans.
	Marburg virus	Fruit bats (*Rousettus aegyptiacus*)	Contact with infected bat feces or aerosols or with blood, secretion, organs, or other body fluids of humans
	Lassa fever virus	Rodents (*Mastomys natalensis*)	Rodent urine or feces; nosocomial spread.
	Monkeypox	Rodents, NHP	Contact with blood, bodily fluids, or lesions of infected animals. or with respiratory secretions
	Middle East Respiratory Syndrome virus	Dromedary camels	Direct or indirect contact with infected camels, close contact with infected persons, contact with fomites
③ Americas	Equine encephalitis viruses (EEEV, WEEV, VEEV)	Wild birds; equids (for VEEV only)	Bite of infected mosquitoes (*Culex*, *Culiseta*, and *Aedes* spp.)
	Colorado tick fever virus	Wild rodents	Bites of infected Rocky Mountain wood ticks (*Dermacentor andersoni*)
	Zika virus	Non-human primates	Bite of infected mosquitoes (*Aedes* spp.), transplacental, blood transfusion, organ transplantation
④ Europe	Tahyna virus	Small (hares, rabbits, rodents, hedgehogs), large (red deer, roe deer, wild boar) mammals	Bite of infected *Aedes vexans* mosquitoes.
	Tick-borne encephalitis virus	Small rodents (voles, mice), wild & domestic mammals (foxes, bats, hares, deer, wild Boar, sheep, cattle, goats)	Bites of infected *Ixodes* and *Dermacentor* spp. ticks, consumption of unpasteurized milk.

Contd. Table 1. Epidemiology of the major emerging and re-emerging viral zoonotic infections[24]

SN	Geographical distribution	Causal virus	Animal hosts/Reservoirs	Mode of transmission
		Usutu virus	Wild and domestic birds (black birds, doves, jays, magpies, partridges, owls, sparrows)	Bite of infected mosquitoes (mainly *Culex* spp.)
⑤	Asia	Influenza virus	Wild waterfowl, domestic poultry, swine, horses, dogs, bats	Inhalation of droplets from infected persons; direct contact with infected animals or environments contaminated with their nasal secretions, saliva, or feces.
		Severe Acute Respiratory Syndrome	Variety of domestic and wild animals (palm civets, bats, pangolins, dogs, and cats	Contact with droplets from infected persons, contact with contaminated surfaces/fomites.
		Japanese encephalitis virus	Pigs, wading birds	Bite of infected Culex species mosquitoes
		Nipah virus	Fruit bats, pigs	Direct contact with infected bats or their body fluids. Consumption of palm juice contaminated with bat saliva
⑥	Australia	Hendra virus	Fruit bats, horses	Exposure to body fluids and tissues/excretion of infected horses.
		Kunjin virus	Wading birds	Bite of infected mosquitoes (e.g., *Culex annulirostris*)

Table 2. Overview of zoonotic viral infections[25]

S/N	Infection/Disease	Causative virus	Reservoir host (s)	Transmission host(s)	Mode of transmission	H-to-HT
01	Chikungunya fever	Chikungunya virus	Non-human primates (NHP)	*Aedes aegypti* and *Aedes albopictus*	Mosquito bite	No
02	Ebola/Marburg hemorrhagic fever	Ebola virus, Marburg virus	Fruit bats	Gorillas & Chimpanzees African green monkey	Contact with body fluids (CBF) of infected animals	Yes
03	Yellow fever	Yellow fever virus	NHP	Aedes mosquitoes	Mosquito bite	No
04	Monkeypox	Monkeypox virus	NHP, rodents	Rodents, monkeys	CBF or mucosal lesions	Rare
05	Nipah virus infection	Nipah virus	Fruit bats, flying foxes	Bats, pigs	CBF, drinking date palm sap	Yes
06	Lassa fever	Lassa virus	Rodents (mouse)	Rodents	CBF or excreta, surface, food	Yes
07	Rift Valley fever	RVF virus	Livestock	Mosquitoes	Mosquito bite, CBF	No
08	Tick-borne encephalitis	TBE virus	Rodents	Ticks	Tick bite	No
09	Japanese encephalitis	JE virus	Birds, pigs	Culex mosquitoes	Mosquito bite	No
10	West Nile fever	West Nile virus	Wild birds	Culex mosquitoes	Mosquito bite	No
11	Hantavirus infection	Hantavirus	Rodents, mice, rats,	Rodents	Rodent bite, inhalation excreta	Rare
12	Crimean-Congo Hemorrhagic fever	CCHF virus	Livestock, hare, boars	Ticks	Tick bite, CBF	Yes
13	Severe fever with Thrombocytopenia Syndrome (SFTS)	SFTS virus	Cats, dogs, cattle, sheep, chickens, minks, pigs, goats, rodents	Ticks	Direct CBF of infected animals	Yes
14	Influenza	Influenza A, B, C, D viruses	Wild birds	Horses, poultry, pigs, whales, seals, mink	Direct contact, aerosols, surface	Yes

H-to-HT = Human-to-human transmission

Table 3. Overview of genomic features, classification, therapy and prevention of zoonotic viral diseases

SN	Infection/Disease	Genome type	Genome structure	Viral family	Genus	Therapeutics	Emerging vaccines
01	Chikungunya virus	RNA	ss (+)	Togaviridae	Alphavirus	NSAIDs, DMARDs Supportive care	Ixchiq® vaccine Vimkunya® vaccine[26]
02	Ebola virus, Marburgvirus	RNA	ss (-)	Filoviridae	Ebolavirus Marburgvirus	mAb114, REGN-EB3 supportive care	Ervebo® vaccine[27,28] NLV, cAd3-MARV®
03	Yellow fever virus	RNA	ss (+)	Flavivirus	-	Supportive care, analgesics, antipyretic, Avoid aspirin & NSAIDs	YF-VAX®, Stamaril®, Sanofis vYF-247®[29]

Contd. Table 3. Overview of genomic features, classification, therapy and prevention of zoonotic viral diseases

SN	Infection/Disease	Genome type	Genome structure	Viral family	Genus	Therapeutics	Emerging vaccines
04	Monkeypox virus	DNA	ds	Poxviridae	Orthopoxvirus	Tecovirimat (TPOXX or, ST-246	ACAM2000®, MVA-BN (Jynneos® & LC16® [30]
05	Nipah virus	RNA	ss (-)	Paramyxoviridae	Henipavirus	Ribavirin , m102.4, Hu1F5	NLV, Viral vectored, mRNA-1215 & HeV-sG-V[31]
06	Lassa virus	RNA	ss (-)	Arenaviridae	Mammarenavirus	Supportive care	MV-LASV, EBS-LASV, INO-4500 vaccine[32]
07	Rift Valley fever virus	RNA	ss (-)	Phenuiviridae	Phlebovirus	Supportive care	NLV, TSI-GSD-200, hRVFV-4s, ChAdOx1 RVF[33]
08	Tick-borne encephalitis Virus	RNA	ss (+)	Flaviviridae	Flavivirus	Supportive care	TicoVac® vaccine[34]
09	Japanese encephalitis virus	RNA	ss (+)	Flaviviridae	Flavivirus	Supportive care	Inactivated: Ixiaro® [35] Attenuated: Imojev®
10	West Nile virus	RNA	ss (+)	Flaviviridae	Flavivirus	Supportive care	NLV, ChimeriVax-WN02, & HydroVax-001[36]
11	Hantaviruses	RNA	SS (-)	Hantaviridae	Orthohantavirus	Supportive care	Hantavax® (China & Korea)[37]
12	Crimean-Congo Hemo-rrhagic fever virus	RNA	ss (-)	Nairoviridae	Orthonairovirus	Supportive care	NLV, ChAdOx2CCHF (ISRCTN12351734)
13	SFTS virus	RNA	ss (-)	Phenuiviridae	Banyangvirus	S care, Favipiravir	NLV
14	Influenza	RNA	ss (-)	Orthomyxo-viridae	A (α-), B (β-), C (χ-), D (δ-) influenzavirus	Oseltamivir, Peramivir, Zanamivir, Baloxavir marboxil	Inactivated (Fluzone, Fluarix, Afluria, Vaxigrip Tetra®), Live attenuated (FluMist, Recombinant (Flublok)[38]

SFTS = Severe fever with thrombocytopenia syndrome NLV = No licensed vaccine

Table 4. Epidemiology of emerging and re-emerging zoonotic RNA virus infections worldwide[23]

SN	Name of disease	Duration of outbreak	No. of countries reported	Cumulative status No. of cases	No. of deaths	Case fatality, %	Vaccine availability	Genus and Family of the causative of viruses
①	HPAI-H5N1	1997-2015	016	000,907	483	53.5	Yes	Influenza A virus; Orthomyxoviridae
②	SARS-CoV	2002-2003	031	008,098	774	09.6	No	Beta coronavirus; Coronaviridae
③	H1N1	2009-2010	214	028,774	144	50.0	Yes	Influenza A virus; Orthomyxoviridae
④	MERS-CoV	2012-2015	027	002,499	858	34.3	No	Beta coronavirus; Coronaviridae
⑤	Ebola virus	2013-2016	015	028,646	11,323	39.5	Yes	Ebolavirus; Filoviridae
⑥	Zika virus	2014-2019	086	575,677	084	0.02	No	Flavivirus; Flaviviridae
⑦	SARS-CoV-2	2019-2021	222	271,963,258	5,331,019	2.0	Yes	Beta coronavirus; Coronaviridae

Rabies

Rabies is one of the ancient, globally prevalent zoonotic diseases caused by the Rabies virus, a member of the genus Lyssavirus, which is one of the seven genera that form the family Rhabdoviridae within the order Mononegavirales. It is a serious public health problem in over 150 countries and territories, predominantly diagnosed in Asia and Africa, accounting for 95% of positive cases in the world. Rabies is a fatal neurological zoonotic disease affecting warm-blooded animals, causing nearly 60,000 human deaths annually in developing Asia and Africa.[39] An estimated 29 million people receive post-exposure prophylaxis (PEP) for rabies each year, and more than 59000 people die of rabies globally, primarily due to poor rabies control measures.[40]

Livestock and humans become infected with rabies after a bite or scratch from a rabid animal, usually a dog in Bangladesh.[41] Analysis of the results from two studies on the prevalence of rabies, tested in 4,728,407

animals, showed that 14,145 (0.3%) animals were positive for rabies.[39] It is a vaccine-preventable disease in both humans and animals, but causes 100% case fatality in clinical cases. Globally, Rabies is maintained by dogs, foxes, jackals, raccoons, skunks, and bats. Among animals, foxes had the highest test prevalence of rabies at 78.3%, followed by dogs at 38.1%. In contrast, the incidence of rabies in tested animals was only 0.5%, and there were no cases in tested humans.[39]

More than 15 million people globally receive a post-exposure vaccination (PEV) annually, but it is still the cause of approximately 59,000 deaths annually, of which 60% occur in Asia.[41]

Rabies is not a notifiable disease in Bangladesh, so surveillance data on animals and humans are scarce. A comprehensive study reported 732 human rabies cases (0.5%) out of 150,068 animal bite cases treated from 2005 to 2008, with the majority of cases resulting from dog bites (90.7%).[42] In a retrospective study, 3425 (24.32%) animal fatalities were reported out of 14,085 dog bites in livestock during 2010-2012.[43]

Rabies is an endemic disease in Bangladesh, and domestic stray dogs are the primary reservoir of the Rabies virus; however, jackals and cats occasionally act as carriers of this virus. A review of the literature reveals that more than three lakh dog bites in humans have been reported annually, and an estimated 2000-2500 by Rabies associated with dog bites in Bangladesh.[45]

Research findings on the occurrence of rabies in street dogs,[46,47] pediatric population,[48] rural population,[49] clinico-epidemiological features of human rabies,[50] investigation into dog bites in animals,[51,52] and management of rabies in dogs[53-55] have been reported.

Rabies is a neglected disease responsible for several thousand deaths annually. It incurs a significant cost, particularly for the 15 million people who receive a post-exposure vaccination, especially in developing countries. The WHO, FAO, and OIE initiated the Global Alliance for Rabies Control (GARC) and launched the 'United Against Rabies' collaboration, which includes a global strategic plan aimed at eliminating human deaths from dog-mediated rabies by 2030, known as the 'zero by 30' initiative.[56] This progress is evident through increased access to dog rabies vaccines, improved human rabies vaccination rates, and enhanced rabies awareness in various countries. However, certain countries in Southeast Asia have begun rabies elimination campaigns. Bangladesh launched an elimination program in 2010 with the target of regional elimination by 2020. Bangladesh is situated in the Indian subcontinent, a region with a high prevalence of rabies, and has an estimated canine population of 1.6 million, of which 83% are considered free-roaming dogs. The annual incidence of humans being bitten by dogs is greater than 300,000. Before the start of the program, more than 2000 people died annually from rabies. In three years, human deaths from rabies decreased by 50%, with the number of reported rabies-related deaths being 52 in 2016.[41] One of the bases of the Global Alliance program is the implementation of rabies-awareness campaigns adapted to the local situation of the countries in question, to motivate dog owners to vaccinate against rabies, to prevent dog bites, and to administer first aid for bite victims, including wound washing and post-exposure rabies vaccinations.[57]

Ownerless street dog population control, awareness about the method of Rabies virus transmission, the fate of the clinical cases, dog bite wound management, and pre- and post-exposure vaccination should be considered to control Rabies in human and animal populations. The article on rabies concluded that Bangladeshi citizens' knowledge about rabies is low. It is essential to develop an intensive plan to enhance people's understanding of dog bites, the risk of contracting the rabies virus, and the measures to take to reduce this risk, including the necessity of completing post-exposure treatment.[41] Another survey study on rabies reported that the majority of people have heard about rabies (73%), and there was a high level of awareness that dog bites are the leading cause of rabies (86%) and that rabies can be prevented by vaccination (85%). However, 59% of dog bite victims first seek treatment from traditional healers instead of visiting hospitals, 29% receive the rabies vaccine, and 2.0% practice proper wound care for dog bites by washing with soap and water. In comparison, 4.8% have not taken any measures.[45] Many Asian countries have eradicated rabies by

implementing control measures, including animal registration, quarantine, isolation, and mandatory mass vaccination. However, rising fox populations now pose a potential risk for the spread of rabies in Asian regions.[39]

Japanese encephalitis

Japanese encephalitis (JE) is a zoonotic disease, primarily affecting pigs and humans, caused by the Japanese encephalitis virus (JEV), a single-stranded RNA virus belonging to the genus Flavivirus and the family Flaviviridae, which is transmitted via the bite of an infected mosquito.

Epidemiology

Mosquitoes of the *Culex Vishnu* subgroup, particularly *Culex tritaeniorhynchus*, are the main vectors of JEV.[58] This zoophilic mosquito species is maintained in an enzootic cycle, with pigs and wading birds serving as amplifying hosts. JE is endemic throughout most of Asia, parts of the western Pacific, and is the leading cause of viral encephalitis in Asia, causing an estimated 100,000 cases and 25,000 deaths per year.[59] Approximately three billion people in 25 countries are affected by JEV globally.[60] Globally, an estimated 68,000 clinical cases occur annually, and over 13,000 deaths.[61] It is an endemic disease in South Asian countries, including Bangladesh, India, Pakistan, and Myanmar.[62]

JEV is a zoonotic flavivirus transmitted primarily by Culex mosquitoes in an enzootic cycle involving water birds and pigs; humans are considered dead-end (incidental) hosts (Fig. 1).[63,64] JEV enzootic transmission cycle, and the JEV is maintained in a natural transmission cycle between mosquitoes (primarily *Culex* spp., primarily *C. tritaeniorhynchus*) and reservoir hosts (primarily Ardeid waterbirds) such as herons and egrets (Fig 1). Feral and domestic pigs act as amplifying hosts of JEV. Humans and other vertebrates, such as horses, are considered dead-end (incidental) hosts because they do not develop sufficient concentrations of JEV in their bloodstreams to infect feeding mosquitoes. JEV is transmitted to humans through the bites of infected mosquitoes. There have only been rare case reports of human-to-human transmission via blood transfusion and liver transplant.[64] JE causes reproductive disorders in pigs and has a 100% case fatality rate in piglets.

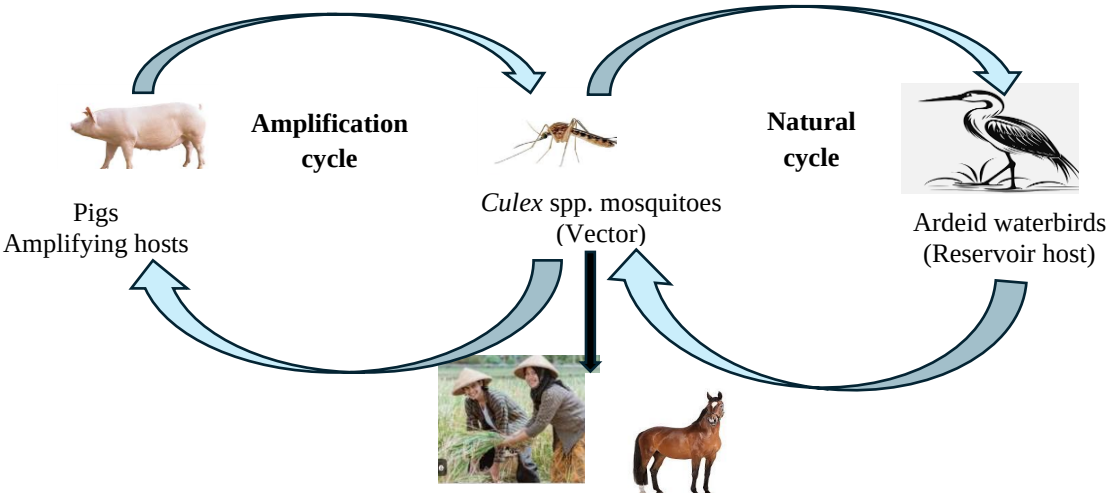


Fig. 1. Transmission cycles of Japanese encephalitis virus with amplification and natural cycles

Swine acts as an intermediate and amplifying host for JE. Wading ardeid water birds, including herons and egrets, carry the virus as reservoir hosts.[58]

JEV was first isolated in 1935 from an infected human in Tokyo, Japan,[64] whereas the first reported human case of JEV occurred in 1977 in Bangladesh.[65,66] Currently, JEV comprises five genotypes (G1-GV) that differ in their geographic distribution and disease manifestations. Genotypes I, II, and III are the most prevalent, accounting for approximately 98% of JEV strains, which are distributed throughout Asia.

The first outbreak of JE infection among humans in the Madhupur forest area of Bangladesh was reported in 1971, and a high titer of JE antibodies was detected in human patients, their contacts, and pigs.[66] In another study, of the 288 pig sera tested, 110 (38.19%) animals were found to carry HI antibodies for JE, with a comparatively higher prevalence in pigs from the Madhupur forest (64.06%) than in those from Dhaka (17.50%) areas.[67]

Hospital-based encephalitis surveillance detected that 4-6% of encephalitis patients had antibodies against JE.[68] A higher number of human cases of JE have been reported from the Rajshahi region,[69] which has been linked to previous exposure to JE virus in 30.0% of domestic pigs in the same area.[70] Pigs can act as a reservoir of JE virus and contribute to its transmission to humans through the bite of mosquitoes.

JE incidences have been reported to be higher in Rajshahi than in Khulna and Chittagong. This is possibly associated with a higher concentration of pigs, a primary vertebrate host in the JEV transmission cycle, in the northwestern areas of Bangladesh. [71]

Most JEV infections are asymptomatic, with less than 1% of infections overall progressing to encephalitis. Symptomatic infections can cause a spectrum of clinical manifestations ranging from undifferentiated febrile illness and aseptic meningitis to acute encephalitis (Table 5). One-third of the JE infections are fatal, and half of the survivors develop permanent neurological sequelae.[72] The case fatality rate of reported JE cases is 14-21%, with almost 50% of survivors having persistent neurological deficits at 1-year post-hospital discharge. Two reviews on Japanese encephalitis from a Bangladeshi perspective, as well as a clinical review, have been reported.[72,73]

Table 5. Epidemiological and clinical features of Japanese encephalitis in humans [72]

SN	Regions	Epidemiolo-logical pattern	Seasonality	Affected age group (year)	Unique characteristics	SN	Clinical features	% cases
①	Northern Asia & others	Epidemic	Seasonal	<15	Outbreaks with high morbidity & mortality	①	Altered sensorium	96
						②	Seizures	86
②	Southern Asia & other	Endemic	Year-round	All ages	Sporadic cases, ongoing environmental transmission	③	Hyperkinetic involuntary movements	46
③	Bangladesh (Specific data)	Mixed	July-Nov.	Median age 30	Adult >15 years more affected in some regions	④	Paralytic features	17
						⑤	Neuropsychiatric sequelae	30-50

Reports on the prevalence of JE in pigs and humans are primarily based on serological investigations in Bangladesh. Bangladesh is an endemic area for JE. Although the WHO recommends incorporating the JE vaccine in all endemic areas, Bangladesh does not have a JE immunization program. Therefore, detecting the JE virus in humans, pigs, and the mosquito population would be required for its control in risk areas in Bangladesh (Table 5).

Preliminary diagnosis relies on the individual's clinical presentation and thorough exposure history, including recent travel and outdoor activities. Detection of JEV RNA in the whole blood, CSF, urine, or brain tissue by nucleic acid amplification testing (NAAT) confirms the diagnosis (Table 6)

Treatment focuses on supportive care, as no specific antiviral treatment options are currently available. Supportive therapy should concentrate on controlling intracranial pressure, maintaining adequate cerebral perfusion pressure, controlling seizures, and preventing secondary complications.

Table 6. Diagnostic criteria and tests for Japanese encephalitis in humans [72]

SN	Diagnostic tests	Specimen	Sensitivity	Comments
①	JEV-specific IgM antibody	CSF	70-90% sensitivity	Highly specific diagnostic of JE
②	JEV-specific IgM antibody	Serum	60-70% sensitivity	Indicate recent infection or vaccination
③	Nucleic acid amplification	CSF/Blood	Low sensitivity due to transient viremia	Useful if performed early in infection
④	MRI (Thalami T2W1 hyperintensities	Brain imaging	High specificity for JE in endemic regions	Distinguished JE from other encephalitis

Prevention of JE by using vaccines that have been available for decades, but their introduction and uptake have been limited by high cost and (in some cases) multiple-dose regimens. Currently, three newer generation vaccine classes are in use: inactivated Vero cell-derived, live attenuated (i.e., CD-JEV), and live recombinant (chimeric) vaccines (i.e., JE-CV), all of which are safe and immunogenic. All licensed vaccine viruses are genotype III JEV but have been found to elicit protective levels of neutralizing antibodies against genotypes I-IV. Neutralizing antibody data and differences between genotype V and III viruses suggest that current vaccines may not be as effective against genotype V viruses. Another key preventive strategy for individuals living in or visiting endemic areas is avoiding mosquito bites. Strategies to prevent bites include using insect repellents, wearing clothing and gear treated with insecticides such as permethrin, and sleeping in screened or air-conditioned rooms or under an insecticide-impregnated bed net.[64]

In Bangladesh, 1 in 1000 JE infections in humans causes severe disease, 1 in 10,000 causes death, and hospital surveillance misses 76% of severe cases.[74] JE has a high fatality rate, and survivors develop long-term neurological sequelae; therefore, a childhood JE vaccination program should be considered at least in endemic areas.

Dengue fever

Dengue fever is a mosquito-borne viral illness caused by the Dengue virus (DENV) infection in humans, that is a significant public health concern globally, particularly in tropical and subtropical regions, and clinically characterized by fever, muscle and joint pain, myalgia, cutaneous rash, dengue hemorrhagic fever (DHF), and dengue shock syndrome (DSS), which can be fatal.

Etiology

Flavivirus is a genus of viruses within the family Flaviviridae, composed of more than 70 members, of which 40 are associated with zoonotic diseases, including Dengue virus (DENV), Zika virus (ZIKV), Yellow fever virus (YFV), West Nile virus (WNV), and Japanese encephalitis virus (JEV) are most important globally, causing extensive morbidity and mortality in humans. These viruses are primarily transmitted to humans and animals through arthropod vectors, particularly mosquitoes and ticks.

The dengue virus causes dengue fever, which is characterized by four distinct serotypes (DENV-1 to DENV-4) of single-stranded RNA viruses. All serotypes can cause human infection. The prevalent serotypes of dengue in Bangladesh until 2000 were DENV-1, DENV-2, and DENV-3, with the highest number of reported cases attributed to DENV-3.[75,76] Bangladesh has experienced the circulation of all four dengue virus serotypes, with DENV-1 and DENV-2 being historically dominant, and DENV-3 becoming more prevalent in recent years.[77,78]

Epidemiology

Dengue virus (DENV) primarily infects humans. Still, its infections have been detected in a variety of animals, including mammals like pigs, non-human primates (NHPs), bats, rodents, dogs, sheep, cattle, goats, and horses, as well as birds.[79-81] While these animals can carry the virus, their role as reservoirs for human transmission remains under investigation. Epidemiological survey indicates that DENV infection is spread

to approximately two-fifths of the world's population, infecting nearly 390 million people annually, resulting in 500,000 hospitalizations and 20,000 deaths. It is mainly distributed in the Eastern Mediterranean, Southeast Asia, Africa, the Western Pacific, and South America. Approximately 2.5 billion people are at risk of contracting dengue, and the reported cases of dengue fever each year are around 100 million; up to 500,000 individuals go on to develop the potentially fatal DHF or DSS. The majority of DHF and DSS cases are brought on by a subsequent viral infection with a different serotype or secondary infection.[82]

Dengue virus (DENV) infection in humans was first reported in East Pakistan (now Bangladesh) in 1966,[83] followed by sporadic cases in succeeding years with an acute outbreak of dengue as Dacca fever reported in 1967,[84] and then major epidemic of dengue fever and life-threatening dengue hemorrhagic fever occurred in 2000,[85,86] where four serotypes with DEV-3 predominance that remained in the circulation till 2002.[87,88,89] Bangladesh first experienced a large outbreak of Dengue in 2000, with 5,551 cases and a case fatality of 93.[90] The worst outbreak occurred in 2002 with 6,232 cases and 58 deaths.[75,91] The prevalence of dengue cases and deaths has fluctuated annually in Bangladesh. Some examples include 2019: 101,354 cases and 179 deaths, 2020: 1,405 cases and three deaths, 2021: 28,429 cases and 105 deaths, 2022: 62,382 cases and 281 deaths, 2023: 321,179 cases and 1,705 deaths, and 2024: 1,00,000 cases and 575 deaths.[92]

The year 2023 has witnessed a record high number of dengue-related deaths in Bangladesh, with cumulative deaths for the year surpassing all totals of the previous 23 years (2000-2022: 853 deaths vs. 2023: 1,705 deaths).[93] Over the past 23 years, a total of 244,246 dengue cases have been reported, including 849 deaths. The mean annual number of dengue cases increased eightfold during the second decade, rising from 2,216 cases between 2000 and 2010 to 18,321 cases between 2011 and 2022. The mean number of deaths doubled (21 vs 46).[94] A total of 565,438 dengue cases and 2,587 fatalities from January 2000 to March 2024 in Bangladesh.[95] There are a total of 2154 and 7,713 clinical cases of Dengue fever, of which 21 and 21 affected instances died in the City Corporation of Dhaka and outside the City Corporation from January to June 2025.[96] Thus, the mosquito-borne, life-threatening dengue virus disease has become a severe and alarming problem in recent years in Bangladesh.[93,97,98]

Aedes mosquitoes, primarily including the female vectors *Aedes aegypti* and *Aedes albopictus*, transmit the virus and are common in tropical and subtropical regions worldwide. *Aedes aegypti* was identified as the primary vector responsible for the dengue epidemic in Bangladesh, and *Aedes albopictus* was recognized as a potential vector in Chittagong.[75] These mosquitoes become infected by feeding on the blood of a person already infected with the dengue virus.

Dengue epidemics occur yearly in the Americas, Asia, Africa, and Australia. It is the fastest-spreading mosquito-borne viral disease globally, affecting over 100 million people annually. This disease also leads to 20 to 25,000 deaths, primarily among children, and is prevalent in more than 100 countries.[82] In another report, there are over 400 million cases of dengue fever worldwide, and 22,000 fatalities.[99]

The dengue virus is maintained by the following two transmission cycles: (a) mosquitoes carry the virus from a nonhuman primate to another nonhuman primate, and (b) mosquitoes transmit the virus from human to human. The correlation between dengue and meteorological factors[100] has been evaluated, along with a geospatial multi-criteria approach that integrates environmental and demographic characteristics, in Bangladesh. [101]

The primary DENV infection may be asymptomatic or result in mild fever, but of it becomes severe, it can cause coagulopathy, increased vascular fragility, and increased permeability; this condition is called dengue hemorrhagic fever (DHF), and after that, it may progress to hypovolemic shock, which is called dengue shock syndrome (DSS). These two diseases are life-threatening and can be potentially fatal.[82,102]

Diagnosis

A tentative diagnosis of DF can be based on the acute onset of fever, headache, body aches, and sometimes

a rash spreading from the trunk in patients from disease-endemic areas. Serological diagnosis and sero-surveillance of dengue fever in humans have been evaluated in Bangladesh.[103-107] Confirmatory diagnosis can be performed by detecting the virus, viral nucleic acids, antigens, anti-DENV antibodies, or combinations of these techniques. Serum samples from the acute phase (7 days after the onset of illness) can be used to detect viral RNA sequences by RT-PCR. IgM antibody capture ELISA can be used for the qualitative detection of DENV IgM antibodies on a micro-titer plate using anti-human IgM antibody.

Treatment

There is no specific treatment for DENV-infected patients due to the virus's etiology. Clinical signs of muscle pain, fatigue, and fever can be alleviated and reduced with treatment using the pain reliever acetaminophen. Non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen and aspirin, are not recommended because these anti-inflammatory drugs have a blood-thinning effect and blood anticoagulants that can worsen the prognosis of diseases with a risk of hemorrhage.

Prevention and Control

Infection by one serotype confers lifelong immunity to that serotype but not to others. Dengvaxia® is the only licensed dengue vaccine approved and in use in the United States for children aged 9 to 16 years; however, it is neither approved nor available in other countries. Mosquitoes can be controlled through the use of insect repellents, wearing long-sleeved shirts and long trousers, and using mosquito repellent both inside and outside the home. It can be concluded that Bangladesh has recorded higher dengue fatality rates in recent years, primarily due to the failure of the relevant national authority to manage dengue effectively.

Avian influenza

The A(H5N1) Avian influenza virus (AIV) first emerged in southern China in 1996. Those viruses caused large poultry outbreaks in Hong Kong in 1997, which resulted in 18 human infections. These viruses again spread widely in poultry birds in 2003 throughout Asia and later Africa, Europe, and the Middle East, causing poultry outbreaks and sporadic human infections. Since 2003, more than 23 countries have reported over 880 sporadic human infections with A(H5N1) AIVs to the WHO.[108] The zoonotic AIVs primarily affect poultry birds and humans. Since the first report of H5N1 Avian influenza in Bangladesh in early 2007, 585 outbreaks have been reported in 54 of the country's 64 districts.[109,110] During the two waves of H5N1 in 2007 and 2008, 547 commercial chicken farms were impacted and culled, involving 1.7 million poultry birds, resulting in an estimated economic loss of US$746 million.[111,112,113] However, the poultry industry faced annual waves of AIV outbreaks from 2007 until 2012. In addition to HPAI (H5N1), other subtypes of high (HPAI) and low (LPAI) pathogenicity have been reported in Bangladesh, including H9N2.[114,115] These outbreaks of AI in poultry birds resulted in significant production losses and substantial economic losses in both commercial and backyard farms in Bangladesh. Both high- and low-pathogenic AIVs are currently circulating and exist as endemic states in both domestic and wild birds in Bangladesh.

Highly pathogenic avian influenza (HPAI) has been reported to cause repeated outbreaks in poultry, primarily chickens, and sporadic infections in humans. H5 and H7 subtypes of the Type A influenza virus, belonging to the Orthomyxoviridae family, are known to be highly pathogenic in poultry. Commercial farm chickens are the primary hosts of HPAI, which causes outbreaks with a high mortality rate and is also recorded in backyard chicken flocks, as well as in turkeys, ducks, geese, and crows.[112,116] In 2015, human influenza surveillance identified a human infection with A/H9N2 in Dhaka, Bangladesh, with evidence of exposure to a sick quail.[117] Wild birds, including shorebirds, gulls, and domestic ducks, serve as reservoirs for the HPAI virus.

The first outbreak of HPAI (H5N1) in a chicken flock was reported in March 2007 in Bangladesh.[111,118,119]

More than 585 outbreaks of Avian influenza caused by HPAI H5N1 in poultry birds and 11 human cases since 2007 have been reported from Bangladesh.[120]

Approximately 585 HPAI H5N1 outbreaks in poultry and wild birds, from 2007 to 2022, have been reported in Bangladesh, with 90% of those cases originating from commercial poultry farms.[117] A recent report showed that H5 and H9 subtypes of AIV are circulating in backyard poultry, particularly in ducks (3.6%) and chickens (3.2%), with or without clinical symptoms, in Bangladesh.[121] LPAIVs and HPAIVs, including the highly

Table 7. Avian influenza situation in Bangladesh: HPAI outbreaks data (year-wise) from 2007 to 2018

Outbreak period/ Reported year	No. of districts/ sub-district	No. of commercial flocks	No. of backyard flocks	Wildlife (House crow)	AIV sub-types	No. of sub-types*	No. of clades	Total outbreaks / +ve	Ref. No.
January to July 2007	17/64	35	20	0	H5N1	-	-	55	123
March 2007-July 2009	154/486	-	-	-	H5N1	-	-	325	129
Up to April 2008	47/64	-	-	-	H5N1	-	-	-	130
2007-2016	-	-	-	-	H5N1	14	06	550	131
2010	-	-	-	-	-	2a	-	-	111
Aug 2010 to Dec 2013	LBMs	-	-	-	3	-	-	-	132
2013	49/64	-	-	-	-	-	-	536	133
Until 2013	52/64	-	-	-	H5N1	07	01	556	134,138
2007-2018	-	-	-	-	H5N1	-	-	561	135
January 2017	Dhamrai	732 chickens	-	-	H5N1	-	-	1	115
Jan.2017-July 2019	6/64	225	-	-	-	2b	-	39.6%	136
March 2007-Dec 2020	-	-	-	-	H5N1	-	-	556	137
2007- update	-	+	+	+	H5N1	-	-	580	109
2007-2022	-	+	+	+	H5N1	-	-	585	120

*Both HPAI and LPAI viruses: 14 Sub-types = H1N1, H1N3, H3N2, H3N8, H4N1, H4N6, H5N1, H5N2, H6N1, H7N9, H9N2, H11N2, H11N3 & HxN1
7 Sub-types = H1N2, H1N3, H3N6, H4N2, H5N6, H10N7 and H9N2 (predominant)
H5N1 Clades 6 = 2.2, 2.3.2.1, 2.3.4, 2.2.2, 2.3.2.2, 2.3.4.2 1 Clade predominant = 2.3.2.1a
AIV sub-types 2a = H5 (n = 323) and H9 (n = 3) 2b = H5 (13.0%) and H9 (14.0%) 2c = A/H5 (18.8%) and A/H9 (15.4%)
LBMs = Live-bird markets AIV sub-types 3 = H5N1, H7N3 and H9N2
6 districts = Dhaka, Narsingdi, Gazipur, Mymensingh, Pabna, and Rajshahi
Continual surveillance of Bangladeshi HP H5N1, H7N3, and H9N2 (n = 37) is warranted to identify further evolution and adaptation to humans[132]

pathogenic H5N1 viruses, have been reported in waterfowl, pet birds, wild birds, and chickens in Bangladesh[122-126] Over 580 outbreaks of HPAI H5N1 have been reported in poultry and wild birds since 2007 in Bangladesh (Table 7).[112,127,128] This indicates that the circulation of H5 and H9 in the backyard poultry could threaten nearby small-scale commercial poultry with less biosecurity.

The endemic circulation of AIVs in Bangladesh's poultry populations poses a threat to the poultry industry and human health (Fig. 2). Low-pathogenic AIV subtype H9N2 was first detected in Bangladesh in 2006 and is known to cause reduced egg-laying and hatching.[139] The HPAI A(H5) has been recognized as being associated with outbreaks in domestic (227) and wild (414) birds across 26 countries in Europe. Most HPAI outbreaks reported in poultry were primarily caused by the introduction of the virus by wild birds.[140] The average number of reported AIV outbreaks caused by AIV subtype H5 per year has declined, dropping from 83 outbreaks in commercial poultry and 10 outbreaks in backyard poultry between 2007 and 2012 to zero epidemics between 2013 and 2019.[120]

Subtypes of influenza A viruses

Influenza A viruses are classified into subtypes based on two surface proteins, hemagglutinin (HA protein) and neuraminidase (NA protein). There are 18 known HA subtypes and 11 known NA subtypes. In birds, 16 HA and 9 NA subtypes have been identified. Two additional subtypes, H17N10 and H18N11, have been identified in bats. There are many different combinations of HA and NA proteins, resulting in a total of 144 (16 × 9) possible combinations of subtypes.[141,142,143]

Influenza A viruses have been detected and are known to circulate in seven different animal species or groups, including humans, wild waterfowl, domestic poultry, swine, horses, dogs, and bats. Equine (horse) influenza A(H3N8) virus routinely circulates and can cause illness in horses, and canine (dog) influenza A(H3N2) virus routinely circulates and can cause disease in dogs.

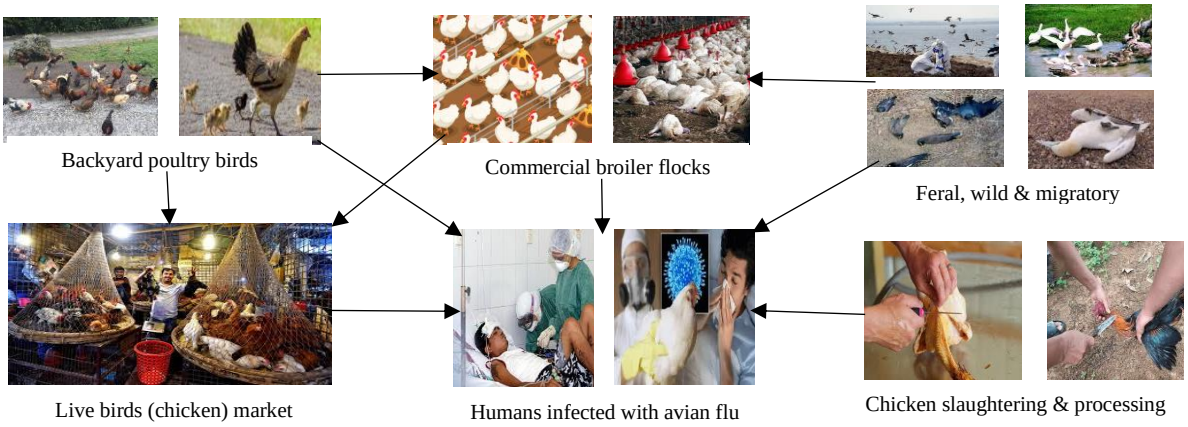


Fig. 2. Transmission and spread of zoonotic pathogenic avian influenza virus (H5N1) in birds and humans

High and low pathogenic avian influenza A viruses

Based on pathogenicity, Avian influenza A viruses are classified into two categories: low pathogenic avian influenza (LPAI) A viruses and highly pathogenic avian influenza (HPAI) A viruses. The LPAI viruses cause either no signs of disease or mild disease in poultry birds, but some LPAI viruses can mutate into HPAI viruses in poultry birds. The HPAI viruses cause severe disease and high mortality in infected poultry. Only some avian influenza A (H5) and A(H7) viruses are classified as HPAI A viruses, while most A (H5) and A (H7) viruses circulating among birds are LPAI A viruses.

A study showed that, until April 2015, clade 2.3.2.1a of the H5N1 virus remained unchanged. However, a new genotype of H5N1 viruses with clade 2.3.2.1a was identified in June 2015, which quickly became predominant in Bangladesh. In addition, migratory birds, domestic ducks, and waterfowl in free-range farms in Tanguar haor-like wetlands played a crucial role in the emergence of this novel genotype of highly pathogenic H5N1 viruses.[112,116,127,128,144]

Out of 506 avian specimens collected from live bird markets and backyard flocks, 50 samples reported positive for AI A viruses, with frequent subtypes among LPAI isolates being H9N2, H11N3, H4N6, and H1N1. Less frequently detected subtypes included H1N3, H2N4, H3N2, H3N6, H3N8, H4N2, H5N2, H6N1, H6N7, and H7N9.[125] The H9N2 subtype identified in Bangladesh was phylogenetically and antigenically related to previous human-derived H9N2 viruses detected in Bangladesh, representing a potential source for human infection. It appears that the domestic poultry birds sold in live markets carried a wide range of LPAI

virus subtypes and a high degree of genotypic diversity.

Since 2008, HPAI H5 viruses have been circulating in Bangladesh, with the predominant clade initially being 2.2.2, which was subsequently replaced by clade 2.3.2.1a viruses, which continue to circulate.[145] Other HPAI viruses have been transiently identified in Bangladesh, including clade 2.3.4.4b H5N6 viruses in domestic poultry, clade 2.3.4.4h H5N6 viruses in migratory birds, and clade 2.3.4.4 h H5N6 viruses in domestic ducks.[136,146,147]

Eight different clades (2.2, 2.2.2, 2.2.3, 2.3.2, 2.3.4, 2.3.2.1, 2.3.2.1a, and 2.3.4.2) of the H5N1 virus were detected from 2007 to 2019.[135] More recently, a new genotype of clade 2.3.4.4b H5N1 HPAI A viruses has emerged in Bangladesh.[147] It appears that the HPAI A (H5N1) viruses have circulated continuously in Bangladesh since 2007, and active surveillance has detected viral evolution driven by mutation and reassortment.[148]

Avian influenza has been recognized as an infectious zoonotic disease of birds; however, it has recently been reported in mammals, with symptoms ranging from asymptomatic to severe and mortal. Therefore, the maintenance, reservoir role, immunity, and the role of mammals in a potential pandemic need to be investigated.

The HPAIV subtype H5N1 poses a threat to poultry, wildlife, and human health. It is enzootic in Bangladesh, with live bird markets (LBMs) identified as potential hotspots for its maintenance, amplification, and spread. Surveillance of LBMs detected an alarmingly high rate of AIV infection, with 49% of the stalls selling AIV-infected live birds, of which 27.0% of stalls tested positive for HPAI H5, and 12.0% of stalls tested positive for H9.[149]

Live poultry markets should introduce centralized slaughter facilities to reduce environmental contamination. The live poultry market is considered a high-risk area for the transmission of HPAI virus in both poultry and humans.[123,137,138,148,150] The environment of poultry shops that slaughtered poultry within the shop was more contaminated with influenza A viruses compared with shops that did not allow slaughter.[151] Poultry workers at live poultry markets are frequently exposed to avian influenza viruses, and antibodies against the H5N1 virus were detected in 2.0% of poultry workers. Poultry workers who fed poultry birds, cleaned feces and utensils, and handled sick poultry birds were at high risk of getting H5N1 infection. Slaughtering, keeping poultry overnight, implementing weekly rest days, improving infrastructure, and implementing disinfection practices could be targeted for interventions to reduce environmental contamination.[151]

Human avian influenza

Avian influenza A viruses rarely infect humans; however, five subtypes of avian influenza A viruses (H5, H6, H7, H9, and H10) are known to have caused human infections. The most frequently identified subtypes of avian influenza A viruses that have caused human infections are H5, H7, and H9 viruses. Specifically, A(H5N1) and A(H7N9) viruses have caused the majority of human infections with avian influenza A virus. As of April 22, 2025, the WHO has recorded a total of 976 human cases of avian influenza A (H5N1) since the beginning of 2003, from 25 different countries globally. The cumulative number of deaths from these cases is 470, resulting in a case fatality rate of 48%.[152] The majority of human cases of avian influenza A(H5N1) and A(H7N9) virus infection have been associated with direct or indirect contact with infected live or dead poultry birds.

The first human case of H5N1 infection was identified in January 2008.[130] Since then, 11 human cases of AI have been reported in Bangladesh, with eight caused by HPAIV (A/H5N1) and three by LPAIV (A/H9N2). [113,153] It appears that Bangladesh has experienced a low number of human cases of H5N1 infection with lower-case fatality. Several factors, including the baseline immune status of the human population, have been proposed as contributing to humans' low prevalence. The identification of AIV A/H5 and A/H9 in

LBMs in Bangladesh may lead to genetic reassortment and the evolution of new AIV strains in poultry and human populations.[125]

A review of reports on human cases of avian influenza in Bangladesh during the period between 2007 and 2020 found eight reported human cases of H5N1 infection, causing one death. Seven of the eight human cases occurred within the poultry epidemic waves (Table 8). Of these, six were in the Dhaka division and five within the Dhaka district, with the following distribution of cases across the poultry epidemic waves: one in wave 2, two in wave 5, and three in wave 6 (Table 8). Another review report revealed a total of eight confirmed human cases of avian influenza in Bangladesh, comprising one case from 2003 to 2009, six cases with one death from 2010 to 2014, and one case from 2015 to 2019, with no further occurrences reported up to 2023.[154-160]

Table 8. Breakdown of H5N1 HPAI poultry epidemic waves associated with human infection in Bangladesh[161]

Wave No.	Outbreak start month	Outbreak end month	Reported bird cases	Reported human cases	Wave No.	Outbreak start month	Outbreak end month	Reported bird cases	Reported human cases
Wave 1	March 2007	July 2007	055	-	Wave 2	September 2007	May 2008	232	1
Wave 3	November 2008	June 2009	037	-	Wave 4	January 2010	June 2010	031	-
Wave 5	January 2011	May 2011	161	2	Wave 6	November 2011	April 2012	026	3

The most common clinical findings in affected humans include eye redness (conjunctivitis), respiratory difficulties, fever, cough, sore throat, and pneumonia. However, more than half of the world's human cases (463) resulted in death.[154] Multiple factors, including rapidly growing poultry production, densely populated countries, the culture of household farms and live bird markets, and the sale of unprocessed birds, make Bangladesh prone to zoonotic disease outbreaks, such as avian influenza.

The occurrence of multiple clades of H5N1 in different poultry species and the reassortment of AIVs with varying patterns of infection have complicated the epidemiological situation for prevention and control, creating conditions that increase the virulence of the virus, its host range, and potential zoonotic transmission. The risk of AIV transmission at the human-poultry interface is increasing over time due to inadequate surveillance and early detection strategies and practices, ineffective biosecurity practices among poultry raisers, and the complex supply chains of backyard and commercial poultry and live bird market systems in Bangladesh.[110,139] Avian influenza surveillance in retail pet birds, wholesale bird markets, mobile vendors, and households can identify cases promptly and reduce the risk of virus transmission.[117]

The circulation of both zoonotic HPAIV A/H5N1 and LPAIV A/H9N2 throughout the country has resulted in several poultry outbreaks, causing severe economic losses to Bangladesh's poultry industry. The emergence of new strains, subtypes, and clades in various domestic poultry species, including wild birds and migratory birds, continues to pose a risk to poultry and humans in Bangladesh. An effective and well-coordinated 'One Health' policy for AIV in Bangladesh will not only reduce public health risks but also economic losses of future outbreaks.

Avian influenza virus (AIV) in waterfowl

Out of 4,308 samples of waterfowl and live market environments tested, 191 (4.4%) were positive for AIV-A, including 74 (1.9%) AI A/ H5 subtypes. Approximately 99.0% (n = 73) of the AI A/H5 -positive samples were from apparently healthy waterfowl. Multiple subtypes, including H1N1, H1N3, H3N2, H3N6, H3N8, H4N1, H4N2, H4N6, H5N1 (clades 2.2.2., 2.3.2.1a, 2.3.4.2), H5N2, H6N1, H7N9, H9N2, H11N2, and H11N3, H11N6, were detected in waterfowl and environmental samples (Table 9). AIVs, including H5N1 and H9N2 subtypes, were also identified in backyard and small-scale raised poultry. Live bird markets could be high-rise sites for harboring the viruses and have the potential to infect naïve birds and humans exposed to them.[112,150]

Table 9. Avian influenza virus surveillance in domestic waterfowl of rural live markets in Bangladesh[150]

S/N	Categories of parameters	Population / sample No. (%)	Influenza A all sub-types No. (%)	Influenza A / H5 No. (%)	S/N	Categories of parameters	Population / sample No. (%)	Influenza A all sub-types No. (%)	Influenza A / H5 No. (%)
A.	**Species of birds**				**D.**	**Types of swabs**			
1.	Ducks	3800 (88.2)	186 (4.9)	71 (1.9)	1.	Oropharyngeal	0705 (16.4)	003 (0.4)	01 (0.1)
2.	Geese	0508 (11.8)	005 (1.0)	03 (0.6)	2.	Cloacal	3354 (77.8)	176 (5.3)	72 (2.2)
B.	Age groups of birds				3.	Fecal	0249 (05.8)	012 (4.8)	01 (0.4)
1.	Juvenile	0610 (14.2)	022 (3.6)	06 (1.0)	E.	Waterfowl flock size			
2.	Adult	3698 (85.8)	169 (4.6)	68 (1.8)	1.	Backyard	3991 (18.9)	172 (4.3)	74 (1.9)
C.	**Health status of birds**				2.	Small scale	0317 (??	019 (5.9)	0
1.	Clinically healthy	4124 (95.7)	188 (4.6)	73 (1.8)					
2.	Sick/dead	0184 (04.3)	003 (1.6)	01 (0.5)					

Through active live poultry markets (LPM) surveillance, HPAI A (H5N6) viruses, along with 14 other subtypes of influenza A viruses, were detected in Bangladesh. The HPAI A (H5N6) viruses belonged to clade 2.3.4.4 and were likely introduced in Bangladesh around March 2016.[146]

Endemic co-circulation of AIV subtypes

The endemic co-circulation of various avian influenza virus (AIV) subtypes, including both highly pathogenic (HP) and low pathogenic (LP) strains, with H5N1 and H9N2 being the most prevalent in Bangladesh. This co-circulation, particularly of H5N1 and H9N2, creates an environment conducive to viral evolution and reassortment, raising concerns about potential spillover to humans and other animals. Multiple virus introductions of different clades of HPAIV H5N1, reassorted genotypes, and ongoing diversification of LPAIV H9N2 create a highly volatile virological environment, which potentially implicates increased virulence, adaptation to new host species, and subsequent zoonotic transmission.[162] Since 2007, the potentially zoonotic HPAI H5N1 and H9N2 viruses have co-circulated endemically in commercial and backyard poultry, moving between different poultry species and causing sporadic spillovers into wild bird populations in Bangladesh.[163,164] Several reassortments between HPAI H5N1, H9N2, and H7N3 viruses have been recorded in Bangladesh. However, some reassortments took place before the incursion into Bangladesh.[132,163] In addition, Bangladesh suffered from successive incursions and replacements by three different GS/GD HPAI H5N1 clades: 2.2.2, 2.3.2.1, and 2.3.4.2.[164] Since 2012, clade 2.3.2.1a viruses have replaced previously co-circulating gs/GD viruses.[165] Exposure of humans to these viruses is emphasized by nine and three cases of human infection with HPAI H5N1 (including one death) and H9N2, respectively, to date.[119,166] Two clinical samples from poultry, obtained in 2016, yielded five different subtypes, including highly pathogenic H5N1, H5N2, H7N1, H7N2, and H9N2, as well as eight genotypes of AIV by plaque purification.[167]

Human avian influenza in Bangladesh

Since 2008, Bangladesh has recorded a total of 11 human H5N1 cases, including one death, and three human H9N2 cases. Three of these cases involved workers in live bird markets due to exposure to infected poultry. Bangladesh has reported two new human cases of H5N1 avian influenza, marking the first such cases since 2015. A bird flu case has been reported from a government poultry farm Jashore, the first in Bangladesh since 2018. An outbreak of avian influenza in Jashore district killed 1,900 birds out of a flock of 3,978. All remaining birds were culled.[168] Bangladesh has reported two new human H5N1 avian flu infections in two boys in Khulna division, which appear to be the country's first since 2015 (Table 10).[169] Emergence of new subtypes and genotypes of AIV, potential vaccination failures due to factors like improper administration or viral mutation, and risks associated with live bird markets.

Table 10. Human Avian influenza cases recorded in Bangladesh

S/N	Recorded date	Age of patient	Place with district	Source and Clinical findings	AIV ST	Ref. No.
01.	27 January 2008	16-month-old boy	Kamalapur, Dhaka	Fever and difficulty breathing.	H5N1	119,130,155
02.	5 March 2011	13-month-old girl	Kamalapur, Dhaka	Cough and fever, RT-PCR	H5N1	156,157
03.	1 March 2011	31-month-old boy	Dhaka	Cough and fever, RT-PCR	H5N1	156, 157
04.	26 February 2012	40-year-old man	Dhaka city	LBM, coughing and recovered	H5N1	158
05.	7 March 2012	26-year-old man	Dhaka city	LBM, coughing and recovered	H5N1	159
06.	7 March 2012	18-year-old man	Dhaka city	LBM, coughing and recovered	H5N1	159
07.	11 February 2013	2-year-old boy	Comilla	BPB, died 18 Feb. 2013	H5N1	160
08.	2007-2020	8 cases in humans	Bangladesh	-	H5N1	137
09.	2015	A human case	Dhaka	Exposure to a sick quail	A/H9N2	117
10.	February 2025	A boy	Khulna division	Sick, Recovered	H5N1*	169
11.	April 2025	A boy	Khulna division	Sick, Recovered	H5N1*	169

LBM = Live bird markets AIV ST = Avian influenza virus subtype BPB = Backyard poultry birds *Older 2.3.2.1a clade

Bangladesh has been vaccinating poultry against Avian influenza since 2012; however, HPAI H5N1 remains enzootic in the country. Vaccinating poultry flocks has high rates of H5N1 prevalence, and spillover to wild birds has increased. Vaccination thus bears the risk of supporting silent spread, where the vaccine only protects against the disease, not against infection.[170,171]

Nipah virus infection

Nipah virus (NiV) is an emerging bat-borne zoonotic paramyxovirus, causing outbreaks with severe respiratory disease and deadly encephalitis in humans with high mortality in Southeast Asia and South Asia, including Bangladesh and India, and Australia, countries in the region are at risk wherever there are susceptible animals, the presence of the virus, and a pathway for transmission.[172-174] In 1915, WHO recognized the NiV as one of the most dangerous emerging zoonotic disease threats because of its high case fatality and ability to transmit from person to person.[175]

Nipah virus (NiV) is a single-stranded RNA Paramyxovirus in the family Paramyxoviridae, genus Henipavirus (HNV), subfamily Paramyxovirinae, and order Mononegavirales.[176] Henipavirus includes Hendra virus (HeV), which has caused spillover infections in horses in Australia and has also resulted in human infections with severe clinical outcomes.[177,178] Hendra and Nipah are two hazardous zoonotic viruses that belong to the group of henipaviruses. In August 2022, a third zoonotic virus in the genus Henipavirus (HNV), Langya virus (LayV), was discovered in China.[179] The emergence of HeV, NiV, and LayV highlights the persistent threat of HNV to human and animal health. The natural reservoirs for HeV and NiV include pteropodid fruit bats, whose habitats span from South and Southeast Asia to East Africa and Australia. [180]

Nipah virus outbreaks in Bangladesh and India have been reported since 2001 and have regularly been associated with person-to-person transmission.[175,177] Phylogenetic analysis genetically characterized NiV into two major distinct genotypes: NiV-Malaysia (NiV-MY), recognized in Malaysia and Cambodia, and NiV-Bangladesh (NiV-BD), identified in Bangladesh and India.[181,182] The Bangladesh Nipah virus strain (NiV-B) is often associated with severe respiratory disease, whereas the Malaysian strain (NiV-M) is typically linked to severe encephalitis.[183] The Nipah virus found in Kerala has been identified as the Indian genotype, also known as I-Genotype, and is similar to the Nipah virus strain found in Bangladesh. [173]

The Indian NiV isolate exhibited nucleotide and amino acid identities of approximately 97% and 95%, respectively, with NiV-B (Bangladesh variant) sequences, and 91% and 83%, respectively, with NiV-M (Malaysian variant) sequences.[184] NiV is a biosafety level 4 pathogen[183] that is primarily spread by reservoir host fruit bats (Pteropus species), also known as flying foxes.

NiV can be transmitted to humans through direct contact with infected animals (bats or pigs), drinking contaminated palm sap, or eating contaminated fruits, and also spread through person-to-person contact (Fig. 3). The primary pathways of transmission from bats to people in Bangladesh are through contamination of raw date palm sap by bats with subsequent consumption by humans and infection of domestic animals (cattle, pigs, and goats), presumably from consumption of food contaminated with bat saliva or urine, with subsequent transmission to people. Approximately one-half of recognized Nipah case patients in Bangladesh developed their disease following person-to-person transmission of the virus.[185]

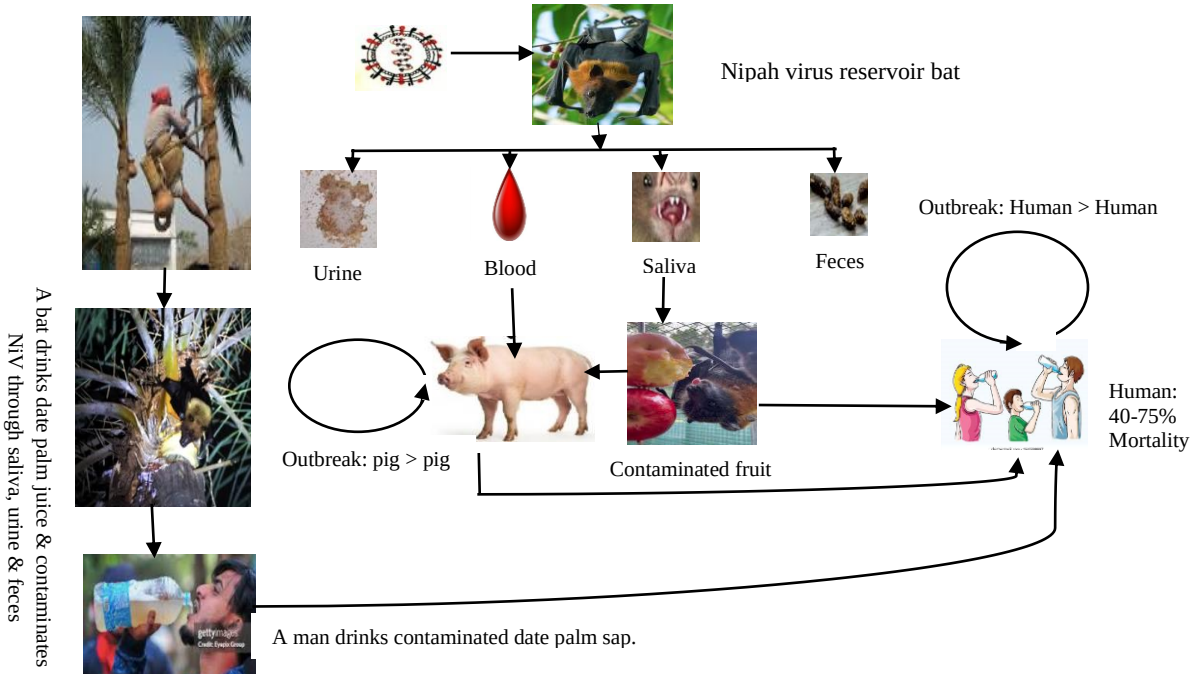


Fig. 3. Zoonotic Nipah virus transmission and mortality in humans

Epidemiological evidence of person-to-person transmission across countries has led to the suggestion that differences in transmissibility may be driven by genetic differences in Henipavirus strains,[181] and that the NiV Bangladesh/India strain is better adapted to human spread than other Henipaviruses.[180] However, an outbreak of Henipavirus in the Philippines in 2015 was associated with person-to-person transmission, and the virus implicated in the outbreak was more closely related to the Malaysian NiV strain than the South Asian strain.[186] A new Henipavirus infecting shrews and humans, with no evidence of person-to-person transmission, was reported from China in 2022.[179] The first outbreak of NiV infection in humans and pigs was reported near Sungai Nipah (Nipah River village) in the northern part of Peninsular Malaysia in 1998 from which the name of the Nipah virus is derived.[183,187,188]

The outbreak subsequently spread to various regions in Malaysia and Singapore (11 male abattoir workers affected, of which one died) in the south due to the movement and import of infected pigs. This outbreak resulted in nearly 300 human cases, with 246 laboratory-confirmed cases and 105 deaths reported in Malaysia and Singapore; pig farmers were mainly infected with NiV from NiV-infected pigs in Malaysia.[189] This disease caused a substantial economic impact as more than one million pigs were killed to help control the

outbreak.[190] While there have been no other known outbreaks of NiV in Malaysia and Singapore since 1999, outbreaks of NiV infection have been reported annually in countries of South and Southeast Asia, including in Bangladesh and India, with a mean case fatality rate of more than 70%.[191,192]

Bangladesh supports 31 species of bats, including three species of fruit bats (*Pteropus giganteus*, *Cynopterus sphinx*, and Rousettus leschenaulti) that are abundant and known to roost and forage near human settlements. *Pteropus giganteus* (the Indian flying fox), is the largest-bodied frugivorous species in Bangladesh and is of key interest as a zoonotic disease reservoir as it is both the natural reservoir for Nipah virus in South Asia and has also been associated with more than 55 others recently identified viruses, some of which may have the potential to cause disease in other animal or human hosts.[193] Most bat populations in Bangladesh are declining due to habitat destruction, and an estimated 80% of the known roosts of the flying fox have been lost over the past three decades. The species has been reported from old temples in Rajshahi, western Bangladesh, and a railway station in Dinajpur, northern Bangladesh.[194] However, their movements were recorded for a median of 120 (range 47-342) days with a median total distance traveled of 393 (range 76-3420 km per individual.[195]

P. giganteus is a social species, with large groups of several individuals living in the same tree. The roosting tree is the area in which bats spend the majority of the day. This fruit bat is reported to travel up to 15 km in search of food.[196]

The mixed evergreen forest in the neighboring Indian States of Assam and Mizoram to the east and the Chin and Rakhine states of Myanmar to the southeast. Deciduous forest is located at the center and north center of Bangladesh, bordering Tripura state, and faces intensive anthropogenic disturbance

Some authors predicted that 2-7% of Bangladesh's land area is suitable roosting habitat for bats. Nipah virus outbreak villages were 2.6 times more likely to be in areas predicted as highly suitable habitats for *P. giganteus* compared to non-outbreak villages.[197]

Fruit bats were identified as the natural reservoir of this virus.[198] A single genus (Pteropus) of frugivorous bats appears to be the main reservoir for henipaviruses throughout Asia and Australia.[199,200] This includes *Pteropus medius* (formerly *Pteropus giganteus*, the only pteropod bat present in Bangladesh and India.[191,200,201]

Outbreaks of NiV were also reported in Siliguri, India, in early 2001, resulting in the deaths of 45 people. The outbreak in the Nadia district was associated with five deaths, with 100% mortality, and the outbreak in Kerala in 2018 caused 21 deaths. Henipavirus infection caused severe disease among humans and horses in the southern Philippines in 2014, with close contact with horses or consumption of horse meat reported in 10 of the 17 confirmed cases. In the same period, 10 horses died, 9 of which were reported to have neurological disorders.[186] Horse-to-human and human-to-human transmission occurred in the Philippines.[186]

India borders Bangladesh, and the first outbreaks of Nipah virus infection occurred in 2001 in both Bangladesh and India. Since 2001, seasonal epidemics of NiV have occurred in Bangladesh during the winter months, primarily in 20 districts in central and northwestern Bangladesh, which are considered the 'Nipah belt' where the majority of spillover events occur.[177]

Bangladesh, especially the western region, is a known area of frequent spillover events with outbreaks of NiV, a zoonotic paramyxovirus reservoir of Pteropus fruit bats, identified almost yearly with a seasonal pattern, especially during winter from December to March since 2001.[198,202,203]

Consumption of raw date palm sap is the most common form of transmission of infection from bats to humans.[204] Outbreaks coincide with the sap harvesting season (December to May). Pteropus bats have been found to visit date palm trees and lick the sap streams being used for collection. Bats may also contaminate the sap collection pots with urine or feces.[205] Domestic animals may also serve as a route of transmission from bats to humans.

Domestic animals may also serve as a route of transmission from bats to humans. Pigs show high seroprevalence against NiV in Bangladesh,[206] and close contact with pigs was found to be a risk factor in one outbreak.[207] Cattle and goats have also been found to be susceptible, as indicated by seroprevalence studies.[187,206,208] Person-to-person spread is an important mode of transmission in Bangladesh, and has been identified in all outbreaks, with the largest person-to-person outbreak occurring in Faridpur in 2004.[209]

This review highlights that the Bangladesh strain of NiV (NiVB) poses a significant threat to humans compared to other Henipavirus strains (NiVM, HeV) reported elsewhere; however, it cannot dismiss the risk that other known and yet undiscovered viruses may pose serious health problems.

Since the outbreak of NiV was first reported in Bangladesh in 2001, approximately 335 people have been infected with the Nipah virus, of which 237 people died[210,] with a case fatality rate was 70% in affected humans and known human-to-human transmission.[175,209] During the period between 2001 to 2011 clinical Nipah cases in humans were initially detected in 22 districts in the northwestern part of Bangladesh and from 2012 to 2021 cases were recorded in another 10 districts (Table 11), that is up to 2021 it spread to a total of 32 districts and then in 2023 it spreads to another district to a total of 33 districts, and in 2025 in 35 districts (Table12), so the NiV infection is spreading in more districts in Bangladesh. Therefore, the term 'Nipah belt' is not appropriate for Nipah virus infection in Bangladesh, as there has been no further incidence of Nipah virus infection in the Meherpur district after the first outbreak; moreover, it is spreading to new districts.

Table 11. Year, district, and division-wise distribution of human clinical cases of Nipah virus infection in Bangladesh (first decade of outbreak)

S/N	District & Division	Year-wise occurrence of human clinical cases of Nipah virus infection																									
		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	Total
01.	Jhalokathi	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	01
02.	Bhola	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01	01
A.	**Barishal division**	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	01	02
02.	Comilla	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01
B.	**CTG division**	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01
03.	Dhaka	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01
04.	Faridpur	-	-	-	37	-	-	-	01	-	12	-	01	01	08	03	-	02	01	-	05	-	-	-	-	-	71
05.	Gopalgonj	-	-	-	01	-	-	-	-	-	02	-	01	01	01	01	-	-	-	-	01	-	-	-	-	-	08
06.	Madaripur	-	-	-	-	-	-	-	-	-	01	-	-	-	03	02	-	-	-	-	-	-	-	-	-	-	06
07.	Manikgonj	-	-	-	06	-	-	-	04	-	-	-	-	06	01	-	-	-	-	-	-	-	-	-	01	-	18
08.	Rajbari	-	-	-	14	-	-	-	06	01	02	02	01	02	-	01	-	-	-	-	-	01	01	04	-	-	35
09.	Tangail	-	-	-	-	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	12
10.	Shariatpur	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	01	01	-	03
11.	Narsingdi	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	01
C.	**Dhaka division**	-	-	-	59	12	-	-	11	01	17	04	03	10	14	07	-	02	01	-	08	01	01	06	02	01	161
12.	Chuadanga	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	01	02
13.	Jhinaidah	-	-	-	-	-	-	-	-	-	-	-	-	01	01	-	-	-	-	-	-	-	-	-	-	-	02
14.	Khulna	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	01	-	02
15.	Kushtia	-	-	-	-	-	-	08	-	-	01	-	-	02	01	-	-	-	-	-	-	-	-	-	-	-	12
16.	Meherpur	13	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	13
17.	Magura	-	-	-	-	-	-	-	-	-	-	-	-	01	04	01	-	-	-	-	-	-	-	-	-	-	06
18.	Norail	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	01
D.	**Khulna division**	13	-	-	-	-	-	08	-	-	01	-	-	04	09	01	-	-	-	-	-	-	-	-	01	01	38
19.	Mymensingh	-	-	-	-	-	-	-	-	-	-	-	-	02	-	-	-	-	-	-	-	-	-	-	-	-	02
E.	**Mymensingh division**	-	-	-	-	-	-	-	-	-	-	-	-	02	-	-	-	-	-	-	-	-	-	-	-	-	02
20.	Bogra	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	02	-	-	-	-	-	-	-	03
21.	Chapai-Nawbgonj	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	01
22.	Naogaon	-	-	12	02	-	-	01	-	-	-	-	-	02	01	04	-	-	-	-	01	01	02	02	01	-	29
23.	Natore	-	-	-	01	-	-	01	-	-	-	-	01	02	01	01	-	-	-	01	-	-	-	01	-	-	09
24.	Pabna	-	-	-	-	-	-	01	-	-	-	-	-	02	-	-	-	01	-	-	-	-	-	01	-	01	06
25.	Rajshahi	-	-	-	-	-	-	-	-	-	-	-	04	02	03	01	-	-	01	01	-	-	-	01	01	-	14
F.	**Rajshahi division**	-	-	12	03	-	-	03	-	-	-	-	06	08	06	06	-	01	03	02	01	01	02	05	02	01	62

Table 11. Year, district, and division-wise distribution of human clinical cases of Nipah virus infection in Bangladesh (first decade of outbreak)

S/N	District & Division	Year-wise occurrence of human clinical cases of Nipah virus infection																									Total
		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	
26.	Dinajpur	-	-	-	-	-	-	-	-	01	-	06	01	-	01	-	-	-	-	-	-	-	-	-	-	-	09
27.	Gaibandha	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	-	01
28.	Joypurhat	-	-	-	04	-	-	-	-	-	-	-	06	-	-	-	-	-	-	-	-	-	-	-	-	-	10
29.	Kurigram	-	-	-	-	-	-	-	-	-	-	01	-	01	-	01	-	-	-	-	-	-	-	-	-	-	03
30.	Lalmonirhat	-	-	-	-	-	-	-	-	-	-	23	-	01	-	-	-	-	-	-	-	-	-	-	-	-	24
31.	Nilphamari	-	-	-	-	-	-	-	01	-	01	-	03	01	-	-	-	-	-	-	-	-	-	-	-	-	06
32.	Panchagar	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	01
33.	Rangpur	-	-	-	01	-	-	-	-	01	-	09	01	-	04	-	-	-	-	-	-	-	-	-	-	-	16
34.	Thakurgaon	-	-	-	-	-	-	07	-	-	-	-	-	-	-	-	-	-	-	05	-	-	-	-	-	-	12
G.	**Rangpur division**	-	-	-	05	-	-	07	01	02	01	39	11	04	06	01	-	-	-	05	-	-	-	-	-	-	82

The highest number of Nipah virus cases (n = 71) was reported from the Faridpur district (n = 68), followed by Rajbari (n = 35), Naogaon (n = 28), and Lalmonirhat (n = 24) districts (Tables 11 & 12). Historically, the first Nipah outbreak was recorded in Meherpur District in May 2001, where nine (69%) of the 13 cases resulted in death.[191] Table 13 presents the case fatality rates caused by Nipah virus infection, which varied from 25 to 100% with an overall average of 66.99% in Bangladesh (Table 13).

Table 12. Year and district-wise occurrence of Clinical cases of Nipah virus infection in Bangladesh

SN	District	Year-wise Nipah cases recorded in outbreaks																									Total
		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	
01.	Bhola	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01	01
02.	Bogra	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	02	-	-	-	-	-	-	-	03
03.	CNB	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	01
04.	Chuadanga	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	01	02
05.	Comilla	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01
06.	Dhaka	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01
07.	Dinajpur	-	-	-	-	-	-	-	-	01	-	06	01	-	01	-	-	-	-	-	-	-	-	-	-	-	09
08.	Faridpur	-	-	-	37	-	-	-	01	-	12	-	01	01	08	03	-	02	01	-	05	-	-	-	-	-	71
09.	Gaibandha	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	-	01
10.	Gopalgonj	-	-	-	01	-	-	-	-	-	02	-	01	01	01	01	-	-	-	-	01	-	-	-	-	-	08
11.	Jhalokathi	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	01
12.	Jhinaidah	-	-	-	-	-	-	-	-	-	-	-	-	01	01	-	-	-	-	-	-	-	-	-	-	-	02
13.	Joypurhat	-	-	-	04	-	-	-	-	-	-	-	06	-	-	-	-	-	-	-	-	-	-	-	-	-	10
14.	Khulna	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	01	-	02
15.	Kurigram	-	-	-	-	-	-	-	-	-	-	01	-	01	-	01	-	-	-	-	-	-	-	-	-	-	03
16.	Kushtia	-	-	-	-	-	-	08	-	-	01	-	-	02	01	-	-	-	-	-	-	-	-	-	-	-	12
17.	Lalmonirhat	-	-	-	-	-	-	-	-	-	-	23	-	01	-	-	-	-	-	-	-	-	-	-	-	-	24
18.	Madaripur	-	-	-	-	-	-	-	-	-	01	-	-	-	03	02	-	-	-	-	-	-	-	-	-	-	06
19.	Magura	-	-	-	-	-	-	-	-	-	-	-	-	01	04	01	-	-	-	-	-	-	-	-	-	-	06
20.	Manikgonj	-	-	-	06	-	-	-	04	-	-	-	06	01	-	-	-	-	-	-	-	-	-	-	01	-	18
21.	Meherpur	13	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	13
22.	Mymensingh	-	-	-	-	-	-	-	-	-	-	-	-	02	-	-	-	-	-	-	-	-	-	-	-	-	02
23.	Naogaon	-	-	12	02	-	-	01	-	-	-	-	-	02	01	04	-	-	-	01	-	01	02	02	01	-	29
24.	Natore	-	-	-	01	-	-	01	-	-	-	-	01	02	01	01	-	-	-	01	-	-	-	01	-	-	09
25.	Nilphamari	-	-	-	-	-	-	-	-	01	-	01	-	03	01	-	-	-	-	-	-	-	-	-	-	-	06
26.	Norail	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	01
27.	Norsingdi	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	01
28.	Pabna	-	-	-	-	-	-	01	-	-	-	-	-	02	-	-	-	01	-	-	-	-	-	01	-	01	06
29.	Panchagar	-	-	-	-	-	-	-	-	-	-	-	-	-	01	-	-	-	-	-	-	-	-	-	-	-	01
30.	Rajbari	-	-	-	14	-	-	-	06	01	02	02	01	02	-	01	-	-	-	-	-	01	01	04	-	-	35
31.	Rajshahi	-	-	-	-	-	-	-	-	-	-	-	04	02	03	01	-	-	01	01	-	-	-	01	01	-	14
32.	Rangpur	-	-	-	01	-	-	-	-	01	-	09	01	-	04	-	-	-	-	-	-	-	-	-	-	-	16
33.	Shariyatpur	-	-	-	-	-	-	-	-	-	-	-	-	-	02	-	-	-	-	-	-	-	-	01	01	-	04

Contd. Table 12. Year and district-wise occurrence of Clinical cases of Nipah virus infection in Bangladesh

SN	District	Year-wise Nipah cases recorded in outbreaks																									
		2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	Total
34.	Tangail	-	-	-	-	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	12
35.	Thakurgaon	-	-	-	-	-	-	07	-	-	-	-	-	-	-	-	-	-	-	05	-	-	-	-	-	-	12
Total		13	0	12	67	12	0	18	11	04	18	43	23	25	36	15	0	03	04	08	07	02	03	11	05	03	343

CNB = Chapai-Nawbabgonj

Table 13 presents the reports on the prevalence and case fatality of Nipah virus in humans from a single district and the combination of multiple districts recorded during the period from 2001 to 2025.

Table 13. Overall human Nipah virus cases in Bangladesh

S/N	Year of outbreak	Districts	Total No. of cases	Case fatality No. (%)	Transmission methods	Reference No.
01.	2001	Meherpur	013	09 (69.23)	Bats, RDPS	191
02	2002	-	-	-	-	-
03.	2003	Naogaon	012	08 (66.67)	Bats, RDPS	191
04.	2004	09 district	067	50 (74.63)	Bats, RDPS	213
05.	2005	Tangail	012	11 (84.62)	Bats, RDPS	204
06	2006	-	-	-	-	-
07.	2007	Kushtia, Naogaon, Natore, Pabna, Thakurgaon	018	09 (50.00)	Bats, RDPS	213
	2001-2007	Sub-total (17 districts):	122	87 (71.00)	Bats, RDPS	202
08.	2008	Faridpur, Manikganj, Rajbari, Nilphamari	011	09 (81.82)	Bats, RDPS	213
09.	2009	Faridpur, Rajbari, Dinajpur, Rangpur	004	01 (25.00)	Bats, RDPS	213
10	2010	05 districts	018	16 (88.89)	Bats, RDPS	221
11.	2011	06 districts	043	38 (85.71)	Bats, RDPS	221
	2001-2011	20 districts	196	150 (77.0)	Bats, RDPS	207
12.	2012	09 districts	023	23 (100%)	-	-
	2001-2012	-	280	211 (75.0)	-	222
13.	2013	16 districts	026	22 (84.62)	Bats, RDPS	223
14.	2014	18 districts	038	15 (39.47)	Bats, RDPS	214
	2001-2014	14 years data	248	82 (33.06)	PPT	175
15.	2015	08 districts	018	11 (61.11)	Bats, RDPS	-
16.	2016	-	-	-	-	-
17.	2017	Faridpur and Pabna	003	02 (66.67)	Bats, RDPS	
18.	2018	Faridpur, Boghra and Rajshahi	004	03 (75.00)	Bats, RDPS	
19.	2019	Natore, Rajshahi and Thakurgaon	008	07 (87.50)	Bats, RDPS	
20.	2020	Jalokati, Faridpur, Gopalgonj, Naogaon	006	04 (66.67)	Bats, RDPS	
21.	2021	Comilla and Rajbari	002	00		
	2001-2021	-	322	229 (71.0)	50% RDPS, 29% PPT	213
22.	2022	Naogaon and Rajbari	003	02 (66.67)	-	-
23.	2023	Rajbari, Shariatpur, Narsingdi, Naogaon,	011	08 (72.73)	-	173,174
	2023	Natore, Pabna, and Rajshahi	014	10 (71.43)	-	224
24.	2024	Manikganj, Shariatpur, Rajshahi, Noagan	005	05 (100)	-	225,226
25.	2025	Pabna, Bhola and Chuadanga	003	03 (100)	-	227
	Overall	Bangladesh	1530*	1025 (66.99)*		

RDPS = Raw date palm sap
RDPJ = Raw date palm juice
PPT = Person-to-person transmission
Horse contact = Consumption of horse meat
*The same data is included in multiple articles
Bats = NiV-RNA detected in 19% (38/456) of bat roost urine samples act as a reservoir in Bangladesh[228]

The identified risk factors of NiV infection in humans are primarily associated with drinking NiV-contaminated raw date palm sap. This delicacy is likely to become contaminated by bat feces, urine, and saliva during sap collection, and transmission to another human usually occurs through caregiving.[204,205] Both animal-to-human and human-to-human transmission have been documented in Nipah virus (NiV) infections in humans in Bangladesh. [211]

From 2001 to 2023, 000 human cases of NiV infection in humans have been reported in Bangladesh, of which 000 (75%) died (Table 00). Outbreaks of NiV cases are distributed over 33 districts in Bangladesh and out of the eight administrative divisions, of which most cases were reported from four divisions, including Dhaka, Khulna, Rajshahi, and Rangpur (Table /)

The administrative structure of Bangladesh currently comprises eight divisions (Bengali: Bibhag) which include Barishal, Chattogram, Dhaka, Khulna, Rajshahi, Rangpur, Mymensingh, and Sylhet. So far, the outbreaks of Nipah virus infection in humans have been reported from seven divisions except the Sylhet division (Tables 11 & 12). Bangladesh is divided into 64 districts, of which NiV infections have been reported in 35 districts (Table 12).

In Bangladesh, wild date palm is found everywhere, especially in Jessore, Faridpur, Kustia, Khulna, and Rajshahi districts, and it is very popularly known as khejur. Date palm juice (sap) is consumed as raw drinks or further processed for concentrated juice and jaggery manufacturing.[212] The potential high risk of Nipah virus transmission in South and Southeast Asia, and its hidden and ongoing threat to global human health, has been assessed. [176,229]

Global perspective

The initial Nipah virus infection outbreaks reported in 1998-1999 in Malaysia resulted in 283 cases with 109 deaths, while Bangladesh has faced almost yearly epidemics since 2001, totaling 352 cases with a case fatality rate of approximately 74%. India has also experienced irregular outbreaks, reported in Kerala and West Bengal, with 1,001 infected and 72 deaths to date (Table 14).[214] Overall, 311 Nipah virus-infected human cases, with 119 deaths (CFR: 38%), were reported in Southeast Asia (Malaysia, Singapore, and the Philippines), while 443 cases, with 316 deaths (CFR: 71%), were reported in South Asia (Bangladesh and India). The epidemiological characteristics of NiV outbreaks

Table 14. Geographical distribution of Nipah virus cases outbreaks in humans [213]

S/N	Outbreaks year	Country, area, districts		Total No. of cases	Case fatality No. (%)	Transmission methods	Reference No.
1.	1998	Malaysia		265	105 (39.60)	Pig-humans	187
	-	Malaysia		283	109 (38.52)	-	214
2.	1999	Singapore		11	01 (09.00)	Pig-humans	215
3.	2001	Siliguri, West Bengal, India		66	45 (68.20)	Bats-humans	216,217
	2007	Nadia district, West Bengal		05	05 (100)	Bats-humans	218
	2018	Kozhikode district, Kerala, India		23	17 (73.91)	Fruits- products Human-to-human	219
	2021	Kerala State, India		01	01 (100)	Horse contact	
	2023	Kerala, India		06	02 (33.33)	-	220
	Sub-total	India		100	69 (69.00)		
4.	2014	Philippines	Encephalitis	11	09 (53.00)	Horse-to-human[1]	186
			Influenza-like	05	0	Person-to-person	
			Meningitis	01	0		
			Total	17	09 (82.0)		
	-	Philippines	-	17	14 (82.35)	-	214
5.	2001-2024	Bangladesh	-	343	245 (71.43)	-	214
6.	2025	Bangladesh	-	03	03 (100)	-	227

[1]Death of 10 horses, slaughter & meat consumption

Table 15. Nipah virus outbreaks in humans in South and Southeast Asia (1998-2024)[214]

SN	Country	Infected	Death (%)
①	Malaysia	283	109 (38.52)
②	Singapore	011	001 (09.09)
③	Bangladesh	343	245 (71.43)
④	India	100	072 (72.00)
⑤	The Philippines	017	014 (82.35)

in South Asia differs from Southeast Asia in terms of fatality rates and transmission patterns. In South Asia, NiV infections have high fatality rates and are not associated with known intermediate hosts.[230] The different fatality rates between Southeast Asian and South Asian countries can be attributed to genetic variations of NiV, which may affect its pathogenicity and virulence. Two clades of NiV, which exhibit greater than 90% genetic identity and amino acid homology (Clade 1: NiV-B, responsible for outbreaks in South Asia; Clade 2: NiV-M, accountable for epidemics in Southeast Asia), may be one of the reasons associated with the different fatality rates. The NiV primarily spreads from pigs or horses to humans in Southeast Asia. In contrast, in South Asia, it is often transmitted through the consumption of contaminated date palm sap or partially eaten fruits by infected bats. Additionally, human-to-human transmission and vertical transmission have been reported in Bangladesh. [230]

Table 16. Country-wise effects of Henipavirus outbreaks in humans occurred from 2002 to 2018[180]

SN	Parameters	HeV (No. +ve/No. tested (%)		NiV_M (No. +ve/No. tested (%)		NiV_B (No. +ve/No. tested (%)	
		Australia	Singapore	Malaysia	Philippines	Bangladesh	India
1.	Asymptomatic infection	-	10/1469 (0.7)[231]	4/1412 (3.0)[189]	-	0/1863 (0.0)[175]	3/279 (1.1)[219,232,233]
2.	Total No. of cases*	6	22	269	17	248	97
3.	Symptomatic cases	6/6 (100)	12/22 (55.00)	265/269 (98.5)	17/17 (100)	248/248 (100)	94/97 (97.0)
4.	Incubation period**	5-16 (7.5)		-	4-20 (8.0)	6-14 (9.0)[202]	6-18 (10.0)
5.	Patients with RS	3/6 (50.0)	6/12 (50.0)[231,234]	26/126 (21.0)[235,236]	16/17 (94.0)	152/243 (63.0)[175]	63/94 (67.0)
6.	Case fatality	3/6 (50.0)	1/12 (0.08)	105/265 (40.0)[236]	9/17 (53.0)	193/248 (78.0)	26/28 (93.0)

HeV = Hendra virus NiVM= Nipah virus, Malaysia NiVB = Nipah virus, Bangladesh
*Asymptomatic and symptomatic cases ** Range and median (%) - Not reported RS = Respiratory sings

Table 17 presents demographic and clinical features of Nipah virus infection in humans. However, Nipah virus infection has been reported to be associated with late-onset and relapsed encephalitis, in addition to acute encephalitis. [237]

Table 17. Demographic and clinical features of Nipah virus infection in humans in Faridpur 2010[238]

S/N	Parameters	1st outbreak (n = 4)	2nd outbreak (n = 4)	Sporadic (n = 8)	Total (n = 16)
1.	Mean age, year (range)	28 (10-45)	55 (32-60)	23 (4-45)	35 (4-60)
2.	Male sex	01 (25)	03 (75)	05 (63)	09 (56)
1.	Fever	4 (100)	04 (100)	08 (100)	16 (100)
2.	Altered mental status	04 (100)	03 (75)	08 (100)	15 (94)
3.	Unconscious	04 (100)	02 (50)	08 (100)	14 (88)
4.	Difficulty breathing	02 (50)	03 (75)	07 (88)	12 (75)
5.	Headache	04 (100)	02 (50)	04 (50)	10 (63)
6.	Vomiting	04 (100)	02 (50)	04 (50)	10 (63)
7.	Convulsion	03 (75)	01 (25)	03 (38)	07 (44)
	Case fatality rate	04 (100)	03 (75)	07 (88)	14 (88)
	Median days (range) from onset to death	7 (4-8)	6 (3-7)	4 (4-17)	5 (3-17)+

+N= 14, all died

Tables 11 to 13 show that Nipah virus is a public health importance zoonotic disease that has been associated with recurrent outbreaks in Bangladesh since 2001 and has been reported up to the current year,

2025. Outbreaks of Nipah virus (NiV) infection are seasonal in Bangladesh, with cases usually occurring annually between December and April, corresponding with the harvesting and consumption of date palm sap.

Nipah virus has several characteristics that make it a significant threat to human and animal health: (a) Its bat reservoir hosts are widely distributed throughout Asia and occur within dense human and livestock populations, leading to widespread frequent spillover events and outbreaks, (b) It can be s transmitted directly to humans by bats or via domestic animals, (c) It can be transmitted from person to person, (d) Spillover has repeatedly occurred in highly populous and internationally connected regions, (e) It is associated with high mortality rates in people, and (f) There are currently no commercial available vaccines or drug to mitigate disease.[200,239,240,241]

The NiV has been shown to spread from person to person in these outbreaks, raising concerns about its potential to cause a global pandemic.[190] Analysis of data on 57 spillovers during 2007-2013 revealed that temperature differences explained 36% of the year-to-year variation in the total number of spillovers each winter and that distance to surveillance hospitals explained 45% of spatial heterogeneity.[242] In addition to human population density, the composition and structure of the landscape shared by *P. giganteus* and humans may influence the geographical distribution of Nipah virus spillovers in Bangladesh.[197] Some authors predicted that 2 to 17% of Bangladesh's land area is suitable roosting habitat, and NiV outbreak villages were 2.6 times more likely to be located in areas indicated as highly suitable habitat for bats compared to non-outbreak villages.[197,243] Bamboo, Dhoincha, jute stick, and polythene skirts covering the sap-producing regions of a tree effectively prevented bat-sap contact. Community interventions should promote the application of these strategies to avoid occasional Nipah spillovers to humans.[244] Implementing strategies to increase awareness about the risks of Nipah virus and protect sap from bats might reduce the risk of NiV transmission.[245] The primary route of transmission of NiV in Bangladesh is through the consumption of raw date palm sap, which is often contaminated by bat saliva or urine. The persistence of Nipah virus RNA in the semen of survivors has been reported in India.[219] Public health campaigns are focusing on educating the public about the risks of consuming raw date palm sap and encouraging boiling it before consumption to reduce the risk of infection. The NiV outbreak highlights the importance of a 'One Health' approach, which involves collaboration between human, animal, and environmental health sectors to address the complex factors contributing to zoonotic Nipah virus transmission.

WHO South-East Asia Regional Strategy for the prevention and control of Nipah virus infection 2023-2030.[174]

- Enhance policy, strategy, and regulatory capacity.
- Increase multi-sectoral, 'One Health' system capacity and readiness for detection, early warning, and response to cases and outbreaks.
- Enhance risk communication and awareness to reduce spillover and spread.
- Promote research and development.
- Promote behavioral changes to reduce risk.
- Enhance control of diseases in domestic animals through improved biosecurity.
- Increase laboratory diagnostic capability in human, animal, and wildlife health sectors.
- Increase surveillance and information-sharing among human, animal, and wildlife health sectors.
- Improve clinical diagnosis and case management.
- Develop and improve access to medical countermeasures.
- Ensure resilience

Although they have been known for more than 20 years, no human drug or vaccine has been invented for them. Due to the high pandemic threat posed by henipaviruses, further research into medicines and vaccines is required. It is also essential to develop effective bio-assurance plans, introduce controls on their operation,

and educate the population on the issue. Reservoir animals, due to anthropogenic environmental changes, are altering their habitats and feeding sites, making more territories vulnerable to disease. New species of henipaviruses are emerging all the time, posing an epizootic challenge to public health. Therefore, a key action is to increase research into the epidemic development of the virus and conduct it as widely as possible.[246]

Swine influenza

Swine influenza, caused by influenza A viruses, is a respiratory disease affecting both pigs and humans, with pigs considered a significant reservoir for influenza viruses. H1N1 influenza, also known as 'swine flu,' because in the past, the people who caught it had direct contact with pigs. H1N1, a subtype of influenza A virus, is an infectious viral illness that infects the epithelium of the respiratory tract of pigs, typically causing an acute disease of variable severity, characterized by rhinorrhea, coughing, decreased appetite, fever, lethargy, rigors, myalgia, and anorexia.

Etiology

Influenza viruses in pigs are referred to as swine influenza viruses. Influenza A virus (IAV) is a common pathogen caused by the Alphainfluenza virus, which belongs to the family Orthomyxoviridae. IAV is a single-stranded negative-sense RNA virus with eight genome segments. As in all viruses with an RNA genome, mutation is an essential source of diversity in these viruses, due to errors in the replication of their enzyme (RNA polymerase), affecting multiple species. Viruses with segmented genomes, such as the influenza virus, have another mechanism for generating diversity: reassortment.

There are four types of influenza viruses: A, B, C, and D. The influenza A virus (IAV) is more predominant because of its capacity to cause higher mortality and morbidity than the influenza B virus (IBV). IAV affects both animals and humans, whereas IBV has no animal reservoir. Almost 75% of influenza cases are caused by IAV. Since humans are the natural host for influenza B, influenza B viruses do not create a pandemic. IBV causes infection in extremes of age groups and persons who are immunocompromised, and those suffering from metabolic and cardiovascular disease. Influenza C virus (ICV) is less well-known and comparatively rare in humans, typically causing mild respiratory infections. Influenza D virus (IDV) primarily affects cattle, with no documented cases in humans to date.[247] During co-infections with multiple strains of IAV, the genome's segmentation allows for gene reassortment, referred to as a genetic shift. Influenza A viruses are classified into different subtypes based on the antigenic differences of the surface HA and NA proteins. Currently, 18 different HA subtypes and 11 different NA subtypes have been identified.

Classification of influenza A viruses

The WHO has classified influenza A viruses into subtypes based on the surface proteins H (hemagglutinin, HA) and N (neuraminidase, NA), which respectively control the entry and exit of viruses into and out of affected cells (Table 18).

The IAVs are subclassified according to the species in which they are isolated, like swine influenza (H1N1), avian influenza (H5N1), equine influenza (H7N7), canine influenza (H3N2), and bat influenza (H17N10 and H18N11).

If a cell is infected with two different virus strains, the RNA of both viruses is copied into the nucleus. When new virus particles are assembled at the plasma membrane, each of the eight RNA segments can come from either of the infecting viruses. The offspring that inherit RNA from both parents are called 'reassortment.' This is the situation observed in the respiratory tract of pigs, whose cells have receptors for both avian and mammalian strains. An example of the evolutionary importance of reassortment is the exchange of RNA segments between mammalian and avian influenza viruses, which leads to the emergence

Table 18. Classification of influenza A viruses into subtypes based on surface proteins H and N[248]

SN	Host species	No. of subtypes	Influenza A subtypes
①	Waterfowls	115	H1-H16, N1-N9
②	Poultry	Numerous	H1-H7 & H9-H12, N1-9
③	Marine mammals	7 (+Virus B)	A (H7N7), A (H4N5), A (H3N3), A (H4N6), A (H13N2), A (H13N9), A (H18N11)
④	Bats	2	A (H17N10), A (H18N11)
⑤	Cats	1	A (H3N2)
⑥	Dogs	2 (+Virus C)	A (H3N8), A (H3N2)
⑦	Horses	2	A (H7N7), A (H3N8)
⑧	Swine	4 (+Viruses C, D)	A (H3N2), A (H1N1), A (H5N2), A (H3N1)
⑨	Humans	2 (+ Viruses C, B)	A (H3N2), A (H1N1)
⑩	Others*	2 known (+ virus D)	A (H10N4), A (H3N2)

*Squirrel, cattle, ferret

of pandemic influenza. In 1957, 1968, and 2009, reassortment events in the intermediate host (pigs) led to the emergence of new viruses, resulting in the Asian, Hong Kong, and Mexican influenza pandemics. The 1957 influenza virus (type A H2N2) acquired three genetic segments from an avian species, and the 1968 influenza virus (type A H3N2) acquired two genetic segments from an avian species.[247,248] The pandemic H1N1 2009 is a reassortment of avian, human, and swine influenza viruses (Figs. 4 & 5). It is necessary for such a reassortment virus to acquire, at a minimum, through mutations, the ability to recognize the human receptor to spread in the human population to develop a pandemic status. [249]

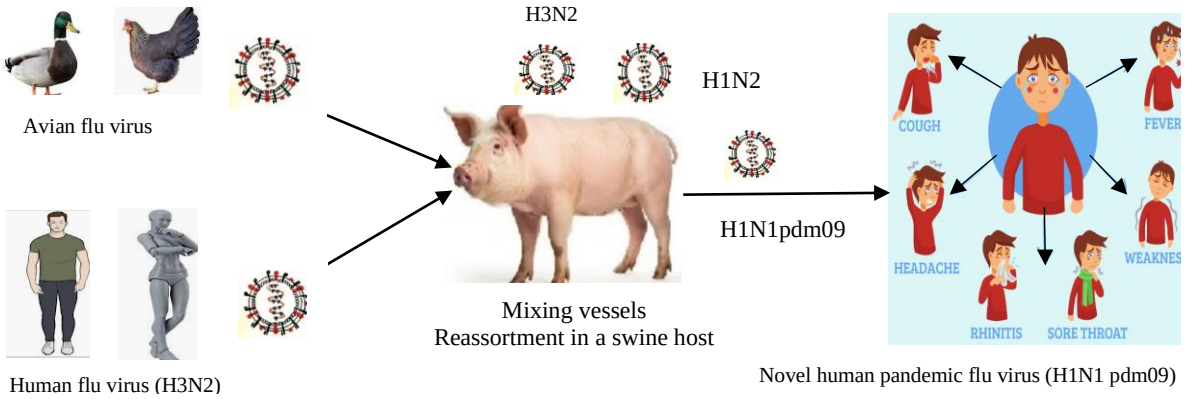
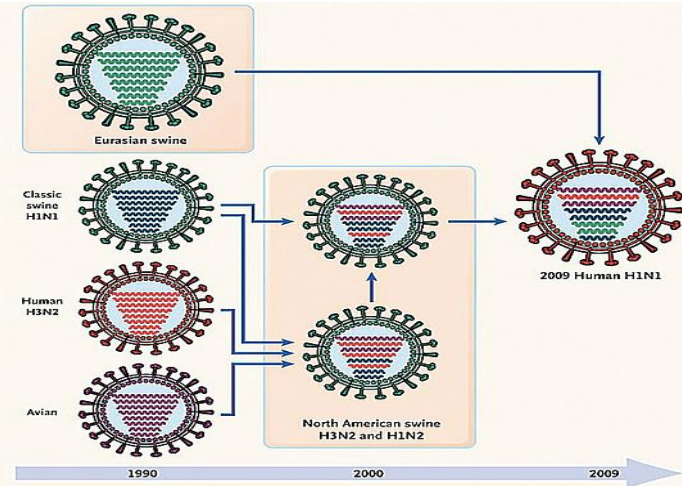


Fig.4. The triple reassortment that caused the 2009 swine influenza pandemic



The 2009 H1N1 influenza A virus, which caused a pandemic, emerged through a complex process of reassortment, where different gene segments from various influenza viruses combined in a swine host. This virus contained gene segments from North American swine, Eurasian swine, human, and avian influenza viruses. Specifically, the pandemic H1N1 virus was a quadruple reassortment, meaning it had gene segments from four different lineages (Fig. 5).

Fig.5. History of reassortment events in the evolution of the 2009 influenza A (H1N1) virus.[250]

Swine serve as 'mixing vessels' for reassortment of avian, swine, and human influenza viruses because they have specific avian and human influenza receptors in the tracheal epithelium (Fig. 4).[251] Until the 1990s, classical swine influenza A (H1N1) was the most commonly circulating swine influenza virus, and it remained relatively constant among pigs. However, by the late 1990s, different subtypes (H1N1, H3N2, and H1N2) had emerged and become predominant among North American pig herds. These swine influenza A viruses acquired gene segments from avian, human, and swine viruses through reassortment, and various genetic lineages can be distinguished within each subtype. A significant fraction of the currently circulating influenza A (H1N1) viruses in pigs derive from the 2009 pandemic influenza A (H1N1) virus, now officially designated A (H1N1) pdm09 influenza virus.[249]

Epidemiology

Influenza A virus (IAV) infects a variety of species, which range from ducks, geese, and waterfowl to humans, equines, dogs, and pigs. Pigs play a crucial role in IAV epidemiology, as they facilitate the replication of human, avian, and swine-origin viruses. Pigs are considered the mixing vessels for the influenza virus, which can reassort and transform into a whole new virus ready to infect animals and humans (Fig. 5).[252] There are multiple subtypes of influenza A viruses that infect pigs, with the most predominant being H1N1, followed by the less common yet significant H1N2 and H3N2 subtypes, which are endemic worldwide.[247] Pigs become infected with IAV through both direct and indirect contact, as well as airborne transmission. The three most important sources of IAVs are (a) infected swine, (b) infected humans, and (c) aerosol.[253] People slaughtering pigs for pork meat, and those who are in very close association with them, are increasingly susceptible to swine influenza. Swine influenza has the potential to cause outbreaks, making it a significant public health concern. The virus spreads through respiratory droplets, posing a high risk of transmission.[254]

Swine influenza viruses can potentially infect humans if their antigenic characteristics change through the reassortment of different influenza strains. Such reassortments have led to pandemics, as seen in 1918 and 2009, when the virus acquired the ability to transmit efficiently from person to person. In 1918, the H1N1 influenza virus, commonly known as the Spanish flu, sparked a devastating pandemic that infected roughly 500 million individuals and led to the deaths of an estimated 50 to 100 million people, accounting for 3-5% of the global population at the time.[255] This virus also caused another pandemic in 2009, which infected over a billion people,[256] and the death toll ranged between 150,000 and 575,000 worldwide.[257]

The first outbreak of the swine-origin novel subtype of influenza virus A (H1N1) in humans was reported in the border area between Mexico and the USA in April 2009. Within a short span of two months, it became the first pandemic, spreading to more than 170 countries worldwide.[258] In India, swine influenza has been reported in various regions, with significant outbreaks occurring due to close interactions between humans and pigs in farming and animal markets.[259,260]

Swine influenza is one of the critical diseases associated with morbidity and mortality in humans globally, but reports on its burden in low-income tropical countries, including Bangladesh, are limited. Swine influenza, particularly the H1N1 strain, has been detected in humans in Bangladesh, with the first confirmed case reported in June 2009. During June-July 2009, event-based surveillance identified 30 case-patients (57.0%) of whom were travelers; starting July 29, sentinel sites identified 252 case-patients (1.0% of whom were travelers).[261] While the initial outbreak was linked to travel to the United States, subsequent cases have occurred, highlighting the potential for local transmission. The 2009 pandemic influenza A (H1N1) virus caused approximately 6,000 deaths in Bangladesh, resulting in Dhaka incurring US$6.1 million in direct medical costs for patients. [262]

RT-PCR assay has been used to detect swine influenza virus (H1N1) in highly suspected 833 registered outdoor (flu corner) and 28 indoor patients from August to November 2009 in Dhaka Medical College

Hospital, of which 57 (6.45%) of outdoor patients were found positive for H1N1 influenza. In contrast, all the 28 (100%) indoor patients were found positive, treated with the capsule Oseltamivir, although two patients (7.14%) died from respiratory failure. [263] Three swine flu-infected humans with the H1N1 flu virus have been reported, which causes the potentially fatal swine flu in Bangladesh.[264] The seroprevalence of influenza A in swine populations of Rangamati and Khagrachari districts was 12.22%, with a higher prevalence recorded in Rangamati (15.0%) compared to Khagrachari (10.0%) districts.[265] This indicates that the swine influenza virus is circulating among pig populations in the Hill Tract districts of Bangladesh.

The H1N1 influenza strain in 2009 was classified as a pandemic by the World Health Organization (WHO).[266] The 2009 H1N1 virus spread through airborne droplets from human to human, possibly via fomites contaminated with the virus, and subsequently transferred to the mucosa or upper respiratory tract. Notably, similarities in symptoms of H1N1 in both humans and pigs arose, potentially due to the viral reassortment of preexisting strains.[267] Influenza A can cause influenza in humans, birds, pigs, and other mammals. In 2009 and 2010, a pandemic caused by the H1N1pdm09 strain, popularly known as 'swine flu' because it contained genetic sequences from avian, swine, and human influenza, resulted in thousands of human deaths worldwide. The situation in Bangladesh is part of a broader picture of swine influenza impacting human health, with regular surveillance and public health measures being implemented to mitigate the risk.

Economic important

Swine influenza can not only cause substantial economic losses for the pig industry but can also lead to epidemics or pandemics in the human population. Swine influenza commonly causes a high morbidity (up to 100%) and low mortality in infected pigs; however, it can lead to 10-15% mortality in naïve pigs. Swine influenza A virus (IAV-S) has a significant impact on the pork industry. It is deemed a significant threat to global public health due to its zoonotic potential.[268]

Current estimates indicate that the US loses $ 1-5 per pig each year due to swine influenza. Considering that global pork production yields ~700 million hogs per year, IAV-S infection has a significant impact on the global economy every year.

Clinical findings

Clinical symptoms of swine influenza A virus (H1N1) have been reported in both indoor and outdoor hospital patients in Bangladesh.[263] This disease in human patients is typically manifested as pyrexia, anorexia, lethargy, coughing, labored breathing, and respiratory distress. Though IAV-S can cause high levels of morbidity in infected herds, this usually does not translate to high rates of mortality. However, co-infections of IAV-S with other pathogens within the porcine respiratory disease complex (PRDC), such as *Mycoplasma hyopneumoniae*, porcine reproductive and respiratory syndrome virus (PRRSV), and porcine circovirus type 2 (PCV2), can result in high mortality rates.[268]

Diagnosis

Clinical diagnosis of swine influenza is challenging because it exhibits similar symptoms to those of rhinoviruses, parainfluenza, adenoviruses, and respiratory syncytial virus. Influenza virus spreads rapidly within 24 hours, and samples can be collected within 2-5 days from nasal and tracheal-throat swabs introduced into transport media to maintain viability until they reach specific laboratories. There, the media samples are used to detect influenza rapidly. Figure 6 shows the tests, that can be used to detect swine influenza.[269]

A confirmatory diagnosis of swine flu involves the detection of the virus, viral nucleic acid, or an antigen-antibody reaction in serologic assays (Fig. 6). A respiratory sample, such as a nasopharyngeal swab, aspirate, or wash, may be collected. Swine influenza viruses can be isolated in embryonated chicken eggs or cell cultures. These viruses can be isolated from lung tissues at necropsy, and from nasal or pharyngeal swabs

collected from acutely ill pigs. Serological tests include HI test, AGIDT, IFAT, VN, and ELISA. RT-PCR assays, which can also detect viral RNA in tissue samples or respiratory fluids, are often used in influenza diagnosis. However, currently, the WHO-recommended methods for detecting swine flu include RT-PCR in specific testing centers, which takes 3 to 4 hours (Table 19). More recently, several methods such as Antigen-Antibody or RT-LAMP and DNA biosensors have also been developed that are rapid and more sensitive (Table 19).

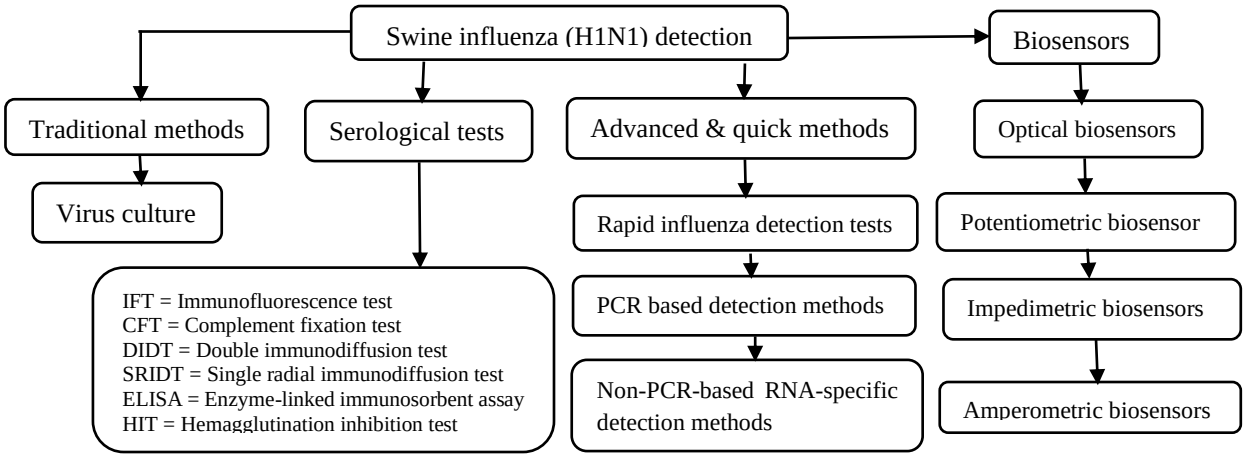


Fig. 6. Diagnostic methods used for the diagnosis of swine influenza

Table 19. Conventional methods for the diagnosis of A (H1N1) virus[269]

SN	Detection method	Sample type for detection	Detection time	Gene / Protein	Sensitivity, % /L.O.D	Reference No.
①	Cell culture	H1N1 virus	7 days	-	86-94	270
②	ELISA	H1N1 virus	4-5 hours	HA antibody	93.7	271
③	RIDT kits	H1N1 virus	<30 minutes	NA/antibody	1.13 HAU	272
④	RT-LAMP	Extracted RNA	3-4 hours	HA gene	93.8	273
⑤	RT-PCR	Extracted RNA	3-4 hours	HA gene	5 copies/reaction	274
⑥	HI assay	Sera	-	HA	92	275
⑦	CRT-PCR	Extracted RNA	3-4 hours	HA gene	1.0104 TCID50	276

RIDT = Rapid influenza detection test H1 = Hemagglutinin 1 NA = Neuraminidase
RT-LAMP = Reverse transcription-Loop mediated isothermal amplification HA = Hemagglutination
TCID = Tissue culture Infective Dose CRT-PCR = Conventional RT-PCR

Public health impact

IAV-S is considered a zoonotic pathogen because it can efficiently transmit between swine and humans. One Health initiative ranked zoonotic IAV as the top priority disease due to its pandemic potential. Swine play a unique role in the evolution of pandemic IAV due to their susceptibility to swine, avian, and human influenza A viruses (IAV). While avian species have a high prevalence of α-2,3-linked sialic acid receptors in their gastrointestinal tract and humans have α-2,6-linked sialic acid receptors in their respiratory tract, swine respiratory tracts contain both types of sialic acid-linked receptors. Co-infection, or 'superinfection' with multiple strains of IAV from different species hosts, can facilitate the swapping of gene segments and the development of new gene constellations that can then be transmitted to immunologically naïve humans. The 2009 H1N1 'swine flu' pandemic represents an example of gene reassortment in pigs and zoonotic

transmission to susceptible human populations. The novel IAV was initially recognized in Mesoamerica, then quickly spread and infected ~24% of the global population within the first year after its emergence. This zoonotic transmission event established a new and stable lineage of H1 in humans, known as H1N1pdm09, that completely replaced the previously circulating H1N1 lineage.

Prevention of swine influenza in pigs

Methods of preventing the spread of influenza among swine include facility management, herd management, and vaccination. Facility management involves implementing measures such as disinfection and maintaining optimal ambient temperatures to minimize the replication of viruses in the environment. Herd management strategies include preventing overcrowding, all-in/all-out production, and quarantining pigs with influenza-like illnesses away from unexposed pig groups. Isolating newly acquired pigs, biosecurity plans, and testing before release also reduces the risk of transmission to the rest of the herd. Sanitation and routine hygiene help prevent the transmission of pathogens on fomites and through mechanical vectors. Additionally, vaccination plays a crucial role.

The rapid evolution of these viruses through genomic mutations poses significant challenges in the development of effective vaccines. Furthermore, mutations create new viral strains, preventing the growth of herd immunity acquired through vaccination and previous infections.

The IAV vaccines that have been created include recombinant, live attenuated, inactivated split, and subunit types. Quadrivalent vaccine Fluarix® (0.5 ml, single dose, GlaxoSmithKline licensed), Flulaval®, Fluzone® (US-licensed trivalent vaccine), and Afluria® (quadrivalent vaccine, Seqirus Pty Ltd.).

SIV vaccines used may not induce strong immunity, and in pigs, they are not 100% effective; however, they can reduce the levels of virus shed by infected animals, thereby reducing the potential for human exposure and zoonotic infection. Licensed vaccines against IAV-s include whole inactivated virus vaccines, Alphavirus replicon particle vaccines, Live attenuated influenza virus vaccines, Double-attenuated LAIV, DNA vaccines, and Viral vectored vaccines. The efficacy of vaccines against emerging influenza viruses is limited due to the continuous alteration of their genomes. A previous vaccine may therefore not protect against future virus outbreaks in the population.

Prevention of swine-to-human viral transmission

Swine are susceptible to both avian and human strains of H1N1 influenza, earning them the nickname ‘mixing vessels.’ In these animals, viral reassortment can occur, leading to the emergence of new strains of swine influenza through antigenic shift.[276]

Transmission of the influenza virus from swine to humans is typically observed in individuals closely associated with pigs, such as farmers, pork handlers, and veterinarians. These individuals are strongly encouraged to wear face masks when dealing with infected animals to prevent transmission through respiratory droplets.

Prevention of human-to-human transmission

The primary routes of zoonotic swine influenza virus transmission between humans are most likely via inhalation of respiratory droplets or aerosols, mucosal contact, and fomite exposure, such as through the nose, mouth, or eyes. Documented strategies to prevent the spread of the virus include frequent handwashing with soap and water or alcohol-based sanitizers and disinfecting household, hospital, and public settings by cleaning with a diluted bleach solution. Anyone who resides in an area where the disease is prevalent, suspects an infection, or presents with flu-like symptoms should stay home, avoid public transportation, and consult a doctor immediately. There is a need to control swine influenza in pigs to prevent the potential of swine-source flu pandemics developing in humans.

Rotavirus

Rotaviruses are a group of highly contagious viruses that can infect both animals and humans, primarily causing gastroenteritis, often characterized by diarrhea, vomiting and fever, leading to high rates of morbidity and mortality in young individuals, predominantly in low- and middle-income countries in the world.

Etiology

Rotaviruses are a group of viruses with a segmented, double-stranded RNA genome (16-21 kbp), belonging to the Reoviridae family (formerly Sedoreviridae).[277] The wheel arrangement of the inner and outer capsid layer gives the virion a wheel-like appearance under electron microscopy, leading to the name 'Rotavirus,' derived from the Latin word 'rota' meaning wheel, which is now the accepted genus name. The rotavirus genome has eleven segments, encoding six structural proteins (VP1, VP2, VP3, VP4, VP6, and VP7) and six nonstructural proteins (NSP1 to NSP6). Rotaviruses are categorized into 10 main groups (RVA-RVJ) based on VP6 serotypes, as shown in Table 20. Among these groups, RVA is the most common and clinically significant species worldwide.

Table 20. Rotavirus (RV) groups and susceptible species [278]

SN	RV group	Species
①	A	Humans, pigs, cows, goats, ostriches, chickens, dogs & horses
②	B	Humans & some animal species (cattle, sheep, pigs, dogs, & rats)
③	C	Humans, pigs, cattle, ferrets, and dogs.
④	D, F, G	Birds
⑤	H	Humans and pigs
⑥	E	Pigs
⑦	I & J	Dogs and bats

Humans are primarily infected by species A, B, and C, most commonly by species A. In contrast, almost all the species cause disease in animals. Their genome, consisting of 11 segments of double-stranded RNA, is characterized by genetic variability including (a) point mutation, (b) genomic reassortment, and (c) genome rearrangements, thus leading to the diversity of rotaviruses. Animal rotaviruses are regarded as a potential reservoir for genetic exchange with human rotaviruses. There is now increasing evidence that animal rotaviruses can infect humans, either by direct transmission of the virus or by contributing one or several genes to reassortments with essentially a human strain genetic background. As mixed infections are a prerequisite for reassortment events, co-surveillance of animal and human rotavirus strains will be vital for gaining a better understanding of the relationships between cocirculating viruses, as well as for assessing any relevant vaccination programs.[279]

Certain animal rotavirus strains exhibit antigenic similarities to some human strains; this may indicate that animals play a role in the transmission of rotavirus infection to humans. Groups A to C have been shown to infect both humans and animals. Rotaviruses are further classified into different G and P genotypes based on the VP7 and VP4 genes, respectively.[277] The most prevalent G and P genotypes for human RVA strains worldwide are G1P[8], G2P[4], G3P[8], G4P[8], G9P[8], and G12P[8]. However, the specific G and P genotypes can vary among animal species. The most prevalent RVA genotypes in felines are G3P[3] and G3P[9], whereas in canines, G3P [3] Fig. 7. The most detected strains in both humans and animals are G2, G3, G4, and G9, P.[280]

Epidemiology

Zoonotic rotavirus, especially Rotavirus A (RVA), is a significant health concern due to its ability to spread between humans and animals. RVA is a significant cause of acute, dehydrating diarrhea in both humans and

animals, and its zoonotic potential is supported by studies demonstrating the presence of shared strains and cross-species transmission.

Regarding animals, calf diarrhea is primarily caused by rotavirus, with prevalence rates ranging from 7 to 94% worldwide.[281] The prevalence of porcine group A rotavirus (RVA) infections is highly variable, ranging from 3.3% to 67.3%, and is often elevated at the farm level, despite the availability of vaccines.[282] The feco-oral route is considered the main transmission route, and risk factors include unsanitary surfaces, contaminated food and water, and inadequate hygiene.

Rotavirus infection is mostly host-specific, but animal rotaviruses can infect humans.[283] Reassortment is one of the crucial mechanisms for generating genetic diversity in rotaviruses and, ultimately, for viral evolution. This means that rotaviruses can be transmitted from animals to humans, and they are therefore classified as zoonotic viruses.

Rotaviruses have a broad host range, infecting a wide range of animal species, as well as humans. As it was found that certain animal rotavirus strains had antigenic similarities to some human strains, this may be an indication that animals play a role as a source of rotavirus infection in humans. Groups A to C have been shown to infect both humans and animals. The most detected strains in both humans and animals are G2, G3, G4 and G9, P.[280] Rotaviruses A are a leading cause of diarrhea in children and the young of a large variety of mammalian and avian host species.

In Bangladesh, some research findings on the different aspects of rotavirus in humans, animals, and birds have been reported with special emphasis on prevalence, epidemiology, characterization of serotypes and strains of mostly diarrheic children,[284-300] calves,[289,290,300-306] buffalo calves,[307] goats,[305,308] poultry birds,[293,294,300] fruit bats,[309] and rodents and shrews.[310]

Host-specific rotavirus infections have been reported in humans and animals in Bangladesh.[298,305] Animal-like rotaviruses, such as bovine-like human VP4 mono-reassortment G6P (8) and novel G11P rotavirus, have been reported in humans.[297] About 23% of calf diarrhea cases were associated with BRV, and G6P was the predominant genotype (94.4%), followed by G10P (5.6%) cases.[306] Rotavirus A (RNA) is the primary cause of acute dehydrating diarrhea in humans and numerous animal species. Animal-to-human interspecies transmission is one of the evolutionary mechanisms driving the diversity of rotavirus strains in humans. Screened fresh feces from 416 bats for RVA using rRT-PCR detected a prevalence of 7.0% and 2.0% in *Pteropus medius* and *Rousettus leschenaultii*, respectively. The identified RVA strains reported are similar to human strains of G1 and G8, as determined by sequence-based genotyping, which underscores the importance of including wildlife species in surveillance for zoonotic pathogens to better understand pathogen transmission and evolution.[309] Rodents and shrews living near humans have been identified as important hosts of zoonotic pathogens. Rectal swab samples of 417 small mammals (rodents and shrews) were tested by using rRT-PCR, detecting Group A rotavirus (RVA) in 6.7% of both rodents and shrews in Bangladesh [310]

Approximately 64% prevalence of rotavirus among hospitalized children < 5 years of age admitted with acute gastroenteritis has been reported in Bangladesh.[298] This presents an opportunity for cross-species transmission of zoonotic pathogens, including rotaviruses, due to the higher percentage of human cases, the high density of the human population, frequent contact with animals, including small animals, and a lack of awareness regarding hygiene and sanitation.[310] Table 21 shows the prevalence of rotavirus infection in fecal samples in humans and animals in Bangladesh

Economic importance

Rotavirus causes significant economic losses in both humans and animals due to its impact on health and production. In humans, it's the leading cause of severe diarrhea in young children, leading to healthcare costs, lost productivity, and in some cases, mortality. In animals, particularly livestock like calves and piglets,

rotavirus infections result in diarrhea, retarded growth rates, and increased mortality, leading to substantial economic losses for livestock farmers.

Table 21. Prevalence of rotavirus infection in fecal samples in humans and animals in Bangladesh

SN	Host tested	No. of hosts	Tests used	Positive No. (%)	Rotavirus types	References No.
1.	Children (diarrheic)	1110	ELISA, RT-PCR	351 (32.0)	RVA	295
2.	Children (diarrheic)	574	RT-Multiplex PCR	140 (24.4)	RVA	296
3.	Cattle (diarrheic)	241	qRT-PCR	015 (06.2)	RVA	305
4.	Calves (diarrheic)	200	RTSBK, RT-PCR	000 (23.0)	BRV	306
5.	Goats (diarrheic)	279	qRT-PCR	046 (16.5)	RVA	305
6.	Bats (Apparently healthy = AH)					
	Pteropus medius	201	rRT-PCR	014 (07.0)	RVA	
	Rousettus leschenaultia	165	rRT-PCR	003 (02.0)	RVA	
	Taphozous melanopogon	050	rRT-PCR	0	RVA	
	Total (bats)	416	rRT-PCR	017 (04.1)	RVA	309
7.	Rodents (AH)	267	rRT-PCR	018 (06.7)	RVA	
	Shrews (AH)	050	rRT-PCR	010 (06.7)	RVA	
	Total	417	rRT-PCR	028 (06.7)	RVA	310

RVA = Group A rotavirus RT=PCR = qRT-PCR = Quantitative Real-time reverse transcription PCR
rRT-PCR = Real-time reverse-transcriptase polymerase-chain-reaction RTSBK = Rapid test-strips BIOK 152

Globally, an estimated 30-50% of pediatric hospitalizations for diarrhea are caused by rotavirus infections.[311] Rotavirus infections are also linked to > 2 million child fatalities every year in children under five, with the highest death rates occurring in Southern Asia and Sub-Saharan African countries. [278]

Rotavirus infections are a leading cause of diarrhea-related deaths, especially in children, with estimates of ~611,000 child deaths annually, mainly in developing countries.[312] Likewise, rotavirus-associated enteritis is a significant problem in young calves, weaning and post-weaning piglets, and foals. [313] Group A rotaviruses (GARVs) account for up to one million child deaths each year, chiefly in developing countries.[314]

Clinical findings

Rotavirus infections in humans and animals share common clinical signs of gastrointestinal distress, particularly diarrhea, but the severity and specific symptoms can vary. Humans, especially infants and young children, often experience severe watery diarrhea, vomiting, fever, and abdominal pain. In animals, while diarrhea is also a primary symptom, other signs, such as lethargy, loss of appetite, and dehydration, can be more pronounced, and secondary infections can lead to more severe outcomes. Porcine rotavirus A is a significant cause of enteritis in piglets during the weaning and post-weaning periods. Dehydration, lethargy, and loss of appetite, with mortality possible in severe cases, particularly in young animals.[313]

Diagnosis

Clinical diagnosis of rotavirus infections can be based on the age of affected hosts, clinical signs of diarrhea, and dehydration. Rotavirus diagnosis in humans and animals utilizes a variety of methods, including stool sample analysis for different methods including (a) etiological methods- electron microscopy, and virus isolation and cultivation, (b) serological methods- antigen detection assay (like ELISA, immunoflurences, immunochromatography, and latex agglutination test), (c) molecular techniques- RT-PCR, qPCR, and LAMO, and (d) other methods- NGS and biosensors.[315]

Prevention

Rotavirus prevention in humans and animals focuses on vaccination, hygiene, and sanitation practices. For

humans, especially for infants, it is the most effective measure. In addition to vaccination, good hand hygiene, proper sanitation, and avoiding contaminated food and water are crucial for preventing rotavirus transmission. In animals, vaccination of pregnant mothers and proper hygiene around animals can help reduce infection. In 2006, two new oral live-attenuated rotavirus vaccines, including Rotarix® (GlaxoSmithKline, Rixensart, Belgium) and Rota Teq® (Merck and Co., Sanofi Pasteur MSD, Lyon, France) became available for use.[316] The rotavirus vaccine is estimated to have averted approximately 28,000 deaths in 2016, and approximately 83,200 additional children could have been saved had full vaccine coverage been achieved that year.[317]

Zoonotic transmission of rotavirus

Zoonotic transmission of rotavirus, though not as frequent as person-to-person transmission, can occur when animal rotaviruses infect humans. This can happen through direct contact with animals, consumption of contaminated water or food, or environmental contamination from animal waste. Reassortment, where different rotavirus strains combine within a host, can also lead to new strains with zoonotic potential.

Group A rotavirus (Rotavirus A; RVA) is primarily known for causing gastroenteritis (acute dehydrating diarrhea) in young children and numerous animal species, which means it has zoonotic potential, can be transmitted between animals and humans. While generally species-specific, rotaviruses can sometimes jump from animals to humans, and vice versa, due to factors like close animal-human contact, environmental contamination, and genetic reassortment. While humans are primarily infected by group A, B, and C rotaviruses, other animal species can also be affected by these and other rotavirus groups. Specifically, group A rotaviruses are classified into different strains based on their outer capsid proteins (G (VP7) and P (VP4) types, and these strains can be found in both humans and various animal species, including cattle, pigs, and sheep (Fig. 7). So far, 36 G (VP7) types and at least 51 P (VP4) types of group A rotaviruses circulating in humans and animals have been reported.

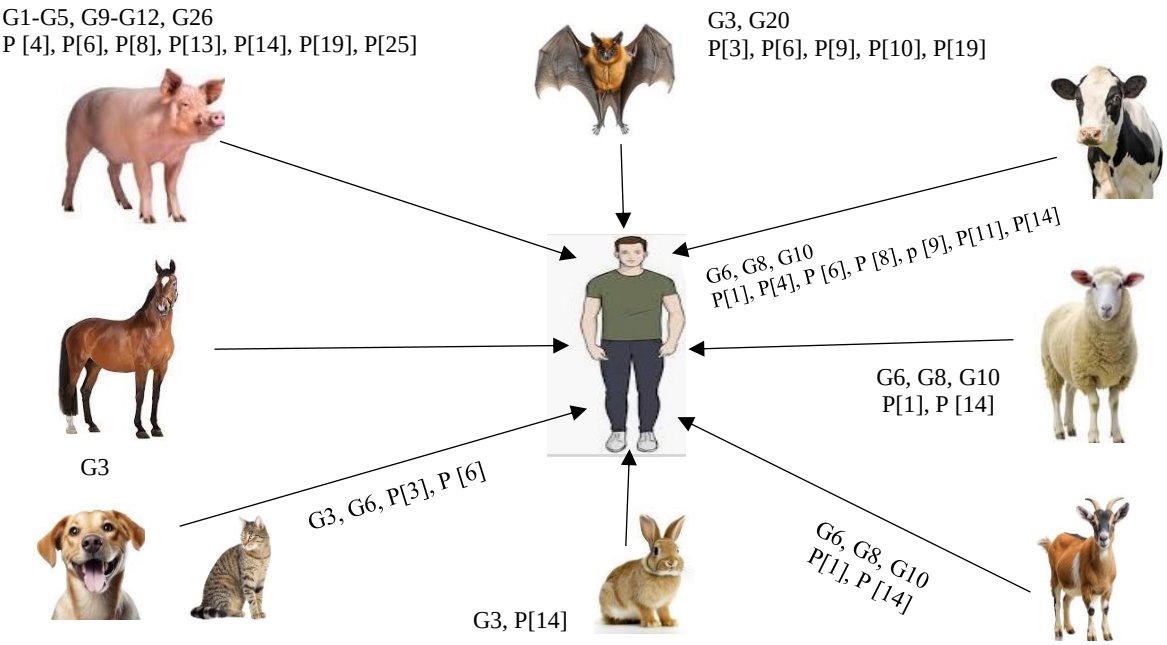


Fig. 7. Zoonotic transmission of group A rotaviruses from animals to humans.

Animal rotaviruses significantly contribute to the gene segment pool of human RVA strains, enhancing their genetic diversity. Specific G- and P-types transmitted from animals to humans are depicted for each animal species.

Interspecies transmission

Rotaviruses are generally species-specific, but cross-species transmission is possible. The diversity of rotavirus is influenced by interspecies transmission. Genetic reassortment is an essential mechanism in the evolutionary process.[283] Interspecies transmission is an important mechanism of rotavirus evolution and contributes to the diversity of human RBA strains.[318] Humans can contract rotavirus strains from animals through direct transmission, or the reassortment of genome segments between human and animal rotaviruses, can lead to antigenic shift &/or increased virulence (Fig. 8).

As mixed infections are a prerequisite for reassortment events, joint surveillance of animal and human rotavirus strains will be pivotal in gaining a better understanding of the relationships between co-circulating viruses, as well as assessing any relevant vaccination.

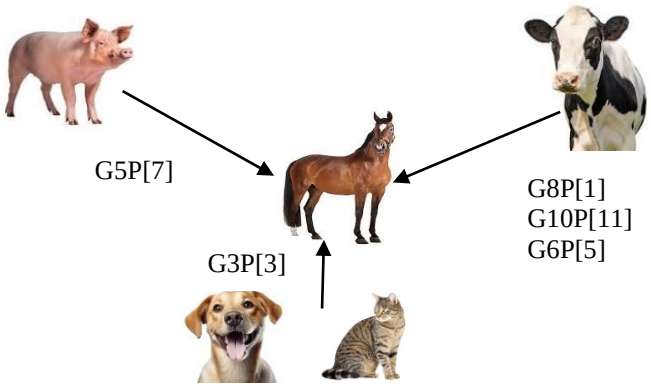


Fig. 8. Cross-species transmission of infrequent RVAG- and P-types from other animals to horses

Surveillance of rotavirus in animal populations is crucial for identifying the source and spread of zoonotic strains. Control measures may include vaccination programs for animals, enhanced sanitation practices, and education on preventing the transmission of diseases between humans and animals. Rotavirus vaccinations, such as RotaTeq and Rotarix, have avoided severe gastroenteritis.[319] Addressing zoonotic rotavirus requires a collaborative 'One Health' approach, considering the interconnectedness of human, animal, and environmental health. This involves understanding the epidemiology of rotavirus in both humans and animals, as well as efforts to prevent its transmission across species. Research is ongoing to understand better the genetic diversity of rotaviruses, their evolutionary relationships, and the factors that contribute to cross-species transmission. This knowledge is crucial for developing more effective vaccines and other preventive measures.

Coronavirus disease (COVID-19)

The Novel Coronavirus has officially been named COVID-19 by the WHO, 'CO' stands for 'Corona,' 'VI' for 'virus,' and 'D' for 'disease,' while '19' stands for the year when the outbreak was first identified (on December 31, 2019). Viruses, and the diseases they cause often have different names. Examples: HIV is the virus that causes AIDS. Similarly, the '2019 novel coronavirus was named 'Coronavirus disease' (COVID-19) and the causal agent as severe respiratory syndrome coronavirus 2 (SARS-CoV-2). WHO announced 'COVID-19' as the name of this new disease on 11 February 2020, and the International Committee on Taxonomy of Viruses (ICTV) announced 'severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) as the name of the virus on 11 February 2020.

Coronaviruses get their name from the crown-like spikes that cover the virion particle's outermost surface. These viruses comprise one of the most prominent families of viruses in the *Nidovirales* order. *Coronavirinae* and *Toronavirinae* are two subfamilies of the *Coronaviridae* family. *Coronavirinae* is divided into four genera: Alpha, Beta, Gamma, and Delta. Human coronavirus 229E (HCoV-229E), Human coronavirus OC43 (HCVoV-OC43), Human coronavirus NL63 (HCoV-NL63, New Haven coronavirus), SARS-CoV, Human coronavirus HKU, Middle East respiratory syndrome coronavirus (MERD-CoV), Novel coronavirus (2019-nCoV)/COVID-19 are the seven SARS CoV and MERS CoV.[320]

Several coronavirus species have been linked to various human and livestock diseases. Due to their recombination capability, random mutation, and ability to infect multiple species, new variants continue to emerge, affecting human health. The novel SARS-CoV-2 coronavirus has posed a significant threat to human life and global health, leading to the COVID-19 pandemic since its emergence in December 2019.[321]

The SARS-CoV-2 virus has instigated a global pandemic, leading to approximately seven million human fatalities worldwide as of 13 April 2024.[322] Bangladesh's first case of COVID-19 was identified on 8 March 2020. Subsequently, as new variants of the SARS-CoV-2 virus emerged, the country encountered multiple waves of COVID-19 infections, resulting in a cumulative total of over 20 million cases and 29,498 deaths to date.[323] Several variants, characterized by their increased transmissibility, have emerged, including alpha (B.1.1.7), beta (B.1.351), gamma (P.1), delta (B.1.617.2), and omicron (B.1.1.529), which have spread globally, including within Bangladesh.[322] A total of 229 countries and Territories around the world have reported 704,753,890 confirmed cases of COVID-19, the coronavirus that originated in Wuhan, China, and a death toll of 7,010,681 as of April 13, 2024.[324]

Within two decades, there have been three documented highly pathogenic and lethal human coronaviruses, namely SARS-CoV, MERS-CoV, and SARS-CoV-2, because of their dreadful impacts on humans (Table 22). The economic burden and health threats caused by these coronaviruses are exceedingly terrible and getting more serious as the increasing number of global infections and attributed deaths of SARS-CoV and MERS-CoV.

Since the first reports of a novel SARS-like coronavirus in December 2019 in Wuhan, China, there has been intense interest in understanding how SARS-CoV-2 emerged in the human population. Both these SARS-CoV emergence events were associated with markets selling live animals and involved species, particularly civets and raccoon dogs, that were also sold live in Wuhan markets in 2019 and are known to be susceptible to SARS-CoV-2 infection. [325]

Table 22. Comparative characteristic features among SARS-CoV, MERS-CoV and SARS-CoV2 up to 13,2024[320,324,326,327]

SN	Characteristic features	SARS-CoV	MERS-CoV	SARS-CoV-2
01.	Clade and lineage	Clade I, lineage B	Clade II, lineage C	Clade I, lineage B
02.	No. countries infected	29	27	229
03.	Mean incubation period (days)	4.7	5.8	5.6-6.7
04.	First emergence	Wuhan, China 2019	Saudi Arabia 2012	Foshan, Guangdong, China
05.	Outbreak beginning date	7 November 2019	4 April 2012	Nov. 2002/ 16 December 2002
06.	No. of cases	8,422 (since 2019)	2,553 (since 2012)	704,753,890
07.	No. of deaths	916	876	7,010,681
08.	No. cases in Bangladesh	-	-	2,049,377
09.	No. of deaths in Bangladesh	-	-	29,493
10.	Case fatality rate, %	10	36	6.8
11.	Time to infect first 1000 people (days)	130	903	48
12.	Incidental hosts	Masked palm civet	Dromedary camels	Malayan pangolin
13.	Recent status	Completely controlled.	Sporadic, continuous	Pandemic is ongoing

SARS-CoV, MERS-CoV, and SARS-CoV-2 are known to produce asymptomatic, mild, or severe respiratory syndromes, with potential fatality rates of 10%, 35%, and 6%, respectively.[328] SARS-CoV and MERS-CoV are zoonotic and known to infect civet cats and camels, respectively. (Fig. 9).[328] However, the first reported spillover of SARS-CoV-2 from animals to humans was recorded in Wuhan, China, in 2019. Since then, the disease has spread worldwide at an unprecedented rate, with the affliction of over 215 countries, with a case fatality rate of 6%.[328]

SARS-CoV-2 natural infection in animals

SARS-CoV-2 virus that is spreading globally through human-to-human transmission, but has also demonstrated the ability to infect multiple animal species with spillover potential from one animal species to another. As of 17 November 2024, there have been 776,897,200 confirmed cases of COVID-19, including 7076329 deaths reported to the WHO. In the last seven days, 41711 new human cases and 558 deaths were reported worldwide.[329] Since the beginning of the pandemic in March 2020, 232 countries, states, and territories have reported COVID-19 cases among humans across five geographic regions: Africa, the Americas, Asia, Europe, and Oceania.[329]

Naturally acquired infection of a SARS-CoV-2 susceptible animal requires close contact with COVID-19-infected humans. Among domesticated animal settings, cats, dogs, and ferrets were reported to be highly susceptible to SARS-CoV-2, while dogs experienced only mild infection. Among captive zoo animals, lions, tigers, minks, snow leopards, pumas, gorillas, otters, and others were also found to be naturally infected (Table 23).[328,329]

Table 23. Animal species naturally infected (RNA detection) by SARS-CoV-2[329]

SN	Animal species	Scientific name	No. of country	Sites (Sources of animals)	Year reported (animal cases/production/ farms/markets)
01.	Domestic cats	*Felis catus*	32	Household	2020 (76), 2021 (94), 2022 (31), 2023 (02)
02.	Domestic dogs	*Canis lupus familiaris*	30	Household	2020 (76), 2021 (76), 2022 (93), 2023 (07)
03.	Domestic American mink	*Neovison vison*	12	Farm	2020 (349), 2021 (32), 2022 (2), 2023 (1)
04.	Domestic ferret	*Mustela furo*	02	Household	2020 (1), 2021 (1), 2024 (1)
05.	Wild Am mink	*Neovison vison*	02	Free range	2020 (no data), 2021 (2)
06.	Western lowland gorilla	*Gorilla gorilla gorilla*	04	Zoo	2021 (12), 2022 (5)
07.	White-tailed deer	*Odocoileus virginianus*	02	Park, Wild	2021 (350), 2022 (625), 2023 (1), 2024 (7)
08.	Binturong	*Arctictis binturong*	USA	Zoo	2021 (1)
09.	Coatimundi	*Nasua nasua*	02	Zoo, Urban park	2021 (3)
10.	Fishing cat	*Prionailurus viverrinus*	USA	Zoo	2021 (1)
11.	Tiger	*Panthera tigris*	07	Zoo, Wild	2020 (1), 2021 (24), 2022 (4), 2023 (1)
12.	Lion	*Panthera leo*	11	Zoo	2020 (2), 2021 (26), 2022 (3), 2023 (1)
13.	Puma	*Puma concolor*	03	Wild, Rescue	2020 (2), 2021 (1)
14.	Snow leopard	*Panthera uncia*	USA	Zoo	2020 (3), 2021 (2)
15.	Indian leopard	*Panthera pardus fusca*	India	Free range	2021 (1)
16.	Canada lynx	*Lynx canadensis*	USA	Zoo	2021 (1)
17.	Spotted hyenas	*Crocuta crocuta*	USA	Zoo	2021 (2)
18.	Asian small-clawed otters	*Aonyx cinereus*	USA	Aquarium zoo	2021 (9)
19.	Hamsters	Unspecified	02	Pet shop	2022 (2)
20.	Wild Eurasian River otter	*Lutra lutra*	Spain	Free range	2021 (1)
21.	Hippopotamus	*Hippopotamus amphibius*	02	Zoo	2021 (1), 2022 (2)
22.	Black-tailed marmoset	*Mico melanurus*	Brazil	Free range	2022 (1)
23.	Mule deer	*Odocoileus hemionus*	USA	Natural park	2022 (1), 2023 (2)
24.	Antillean manatees	*Trichechus m. manatus*	Brazil	Captive	2020 (2)
25.	Giant anteater	*Myrmecophaga tridactyla*	Brazil	Free range	2022 (1)

Contd. Table 23. Animal species naturally infected (RNA detection) by SARS-CoV-2[329]

SN	Animal species	Scientific name	No. of country	Sites (Sources of animals)	Year reported (animal cases/production/ farms/markets)
26.	Mandrill	*Mandrillus sphinx*	USA	Zoo	2022 (1)
27.	Monkey squirrel	*Saimiri sciureus*	USA	Zoo	2022 (1)
28.	Red fox	*Vulpes vulpes*	01	Zoo	2022(1)
29.	Cattle	Unspecified	03	Domestic	2021/2022 (32), 2023 (1)
30.	Buffalo	Unspecified	India	Domestic	2021/2022 (13)
31.	Goat	*Capra hircus coreanae*	02	Unspecified	2021/2022 (46), 2023 (1)
32.	B&B headed spider monkey	*Ateles fusciceps*	Ecuador	Captive	2022 (20)
33.	Common wooly monkey	*Lagothrix lagothricha*	Ecuador	Captive	2022 (1)
34.	White rhinoceros	*Ceratotherium simum*	Senegal	Natural reserve	2023 (1)
35.	Duck	Unspecified	Nigeria	Household farms	2021/2022 (2)
36.	Chicken	Unspecified	Nigeria	Household farms	2021/2022 (10)
37.	Turkey	Unspecified	Nigeria	Household farms	2021/2022 (1)
38.	Sheep	Unspecified	Nigeria	Household farms	2021/2022 (50)
39.	Pig	Unspecified	Nigeria	Household farms	2021/2022 (4)
40.	Lizard	*Agama agama*	Nigeria	Household farms	2021/2022 (19)
41.	Eurasian beaver	*Castor fiber*	Mongolia	Farms	2021 (1)
42.	White-fronted capuchin	*Cebus unicolor*	Peru	Captive	2022/2023 (9)
43.	House mouse	*Mus musculus*	Mexico	Urban	2020 (4)
44.	Brown rat	*Rattus norvegicus*	Mexico	Urban	2020 (3)
45.	Big hairy armadillo	*Chaetophractus villosus*	Argentina	Captive	2022 (3)

Current data shows that over 45 different animal species tested positive for SARS-CoV-2 (Table 23). These 45 animal species include pets, captive animals, farmed animals, and wild animals. Human-to-human, animal-to-animal, and animal-to-human transmission events have been reported in various outbreaks involving animal infections with SARS-CoV-2.[330]

Coronaviruses are zoonotic viruses that include human epidemic pathogens such as the Middle East Respiratory Syndrome virus (MERS-CoV), and the severe acute respiratory syndrome virus (SARS-CoV). Bats were the source of both MERS-CoV and SARS-CoV that infect humans through civet cats and camels, respectively (Fig. 9). Additionally, bats have been identified as the natural hosts of SARS-CoV-2 through phylogenetic studies comparing SARS-CoV-2 with other CoVs, which revealed that the novel virus is 96% identical to two SARS-like CoVs isolated from bats, namely bat-SL-CoVZX45 and bat-SL-CoVZX21. An intermediate host, which enables the novel virus to cross the species barrier and infect humans, remains unknown. Snakes act as an intermediate host to the virus, where homologous recombination within the S protein had occurred, transferring the virus from bats to humans. The ant-eating, long-snouted pangolins are the prospective intermediate host of SARS-CoV-2, depending on a 99% match in genetic identity between the CoV discovered in pangolins and SARS-CoV-2. A schematic-labeled diagram of coronavirus (SARS-CoV) and MERS-CoV and their transmission directly to humans from civet cats and dromedary camels, respectively, is illustrated in Fig. 9.[326]

The coronavirus disease 2019 (COVID-19) is a highly transmissible disease that has infected more than four million people worldwide as of May 12, 2020. COVID-19 is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[327]

The virus was first detected in the city of Wuhan, Hubei Province, China, and the initial outbreak was epidemiologically linked to a wet animal and seafood market.[331] SARS-CoV-2 is thought to have originated from wild animals, possibly a bat coronavirus.[332] The virus exhibits 96.2% nucleotide homology with the bat-origin CoV RaTG13 virus.[333] As of April 13, 2024, a total of 2,049,377 cases of SARS-CoV-2 have been reported, of which deaths have been recorded in 29,493 (22.34%) populations in Bangladesh.[334]

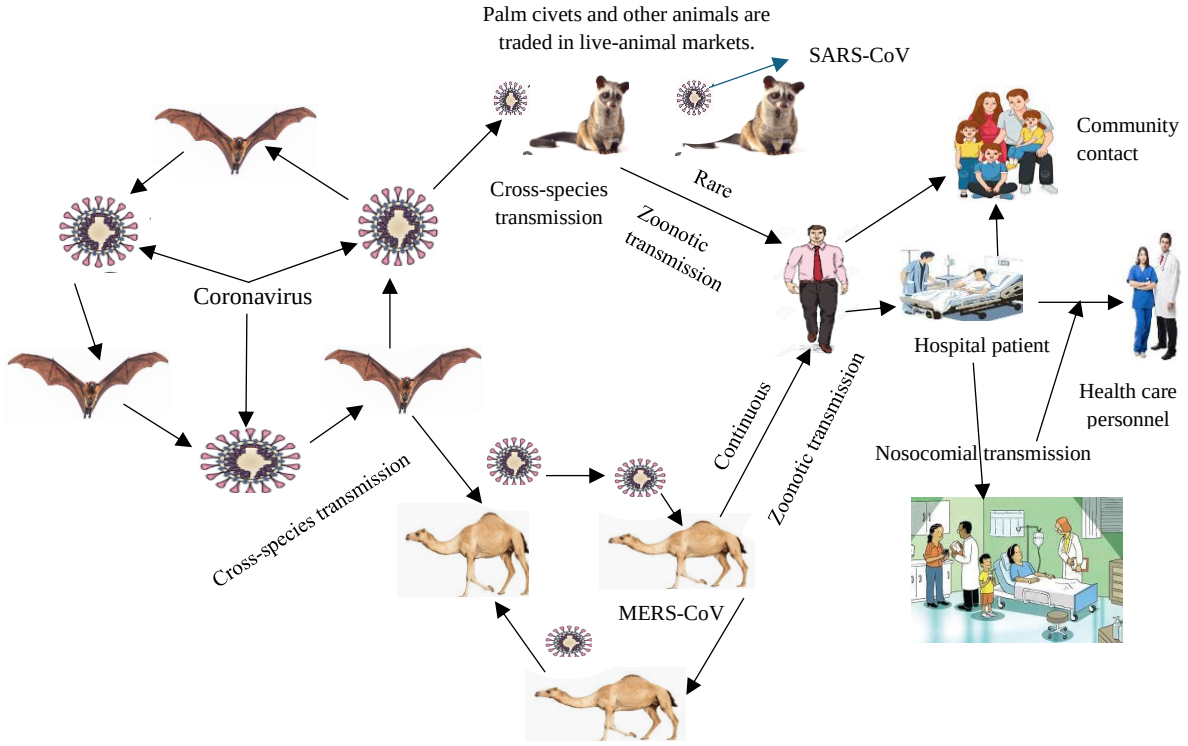


Fig. 9. Emergence and cycle of SARS-CoV and MERS-CoV

In Bangladesh, social perspectives of the COVID-19 pandemic,[335] lesions learned on the SARS-CoV-2 variants and COVID-19,[336] investigation of SARS-CoV-2 infection in domestic animals from COVID-19 affected households,[337] a review on COVID-19 infection caused by SARS CoV-2, [338] and reported the first MERS case.[339] Table 24 presents the COVID-19 confirmed cases in Bangladesh up to July 2020. [340]

Table 24. COVID-19 confirmed cases in Bangladesh (up to July 2020) [340]

SN Division	Total No. of confirmed cases	Positive %	Deaths No. (%)
1. Dhaka	77,354	53.61	1,305 (48.91)
2. Chattogram	29,661	20.56	0673 (25.22)
3. Rajshahi	10,477	07.27	0144 (05.40)
4. Khulna	09483	06.58	0173 (06.49)
5. Sylhet	06672	04.62	0125 (04.69)
6. Barishal	04021	02.79	0100 (03.75)
7. Mymensingh	03687	02.55	0058 (02.17)
8. Rangpur	02969	02.02	0090 (03.37)

Co-infection of dengue and COVID-19 has been reported among 31.0% of the human participants from Dhaka City from December 2021 to November 2023. Coinfected participants had a four times higher risk of developing severe health conditions in Bangladesh. [341]

MERS-CoV is a zoonotic virus transmitted sporadically from camels to humans. Most reported human MERS-CoV cases have occurred in or near the Arabian Peninsula. Limited human-to-human transmission can occur after close contact and has resulted in health care-associated outbreaks. Bangladesh has reported its first MERS-CoV case, involving a Bangladeshi man who lives in the USA and got sick after flying to his native land via Abu Dhabi. The patient, age 53, flew to Bangladesh on June 4 and fell ill on June 6. Most probably, he contracted the virus during the three hours at Abu Dhabi airport or on the plane.

Dromedary camels are bred domestically and imported into Bangladesh. In 2015, of 55 camels tested for MERS coronavirus in Dhaka, 17 (31.0%) were seropositive with ELISA, but none were PCR positive. The potential for infected camels to be sold in urban markets could have significant public health implications.[342,343] Continued and targeted MERS-CoV surveillance is crucial for maintaining preparedness and responding promptly to potential MERS cases.

Chikungunya

Chikungunya is a mosquito-borne viral zoonotic re-emerging disease caused by the chikungunya virus (CHIKV), an RNA virus in the alphavirus genus of the family *Togaviridae*, which is maintained by nonhuman primates. An abrupt febrile illness, polyarthralgia, and a maculopapular rash characterize chikungunya fever. The word Chikungunya means 'to walk bent over' and its name originates from the Makonde, a Bantu language spoken by the Makonde ethnic group in Tanzania and Mozambique. It refers to the curved position of the patient due to debilitating joint pain, meaning that which 'bends up' and describes the contorted posture of people with severe joint pain.

Epidemiology

CHIKV was first identified in Tanzania in 1952 and subsequently in other countries in Africa and Asia. Urban outbreaks were first reported in Thailand in 1967 and in India in the 1870s. CHIKV has now (2024) been identified in more than 119 countries and territories, primarily in Asia, Africa, Europe, and the Americas.[344] Since the beginning of 2025, and as of the beginning of June, approximately 220,000 CHIKV cases and 80 CHIKV-related deaths have been reported in 14 countries/territories, which include the Americas, Africa, and Asia. As of the beginning of June 2025, over 33,000 CHIKV disease cases were reported in Asia from India, Sri Lanka, and Pakistan.[345] All distributed regions with established populations of *Aedes aegypti* or *Aedes albopictus* mosquitoes have now experienced local transmission of mosquito-borne diseases (Fig. 10). The CHIKV infects approximately three million people annually, with an estimated 1.3 to 2.7 billion people currently residing in areas at risk of CHIKV transmission.[346]

When an uninfected mosquito feeds on a person who has CHIKV circulating in their blood, the mosquito can ingest the virus. The virus then replicates in the mosquito over several days, enters its salivary glands,

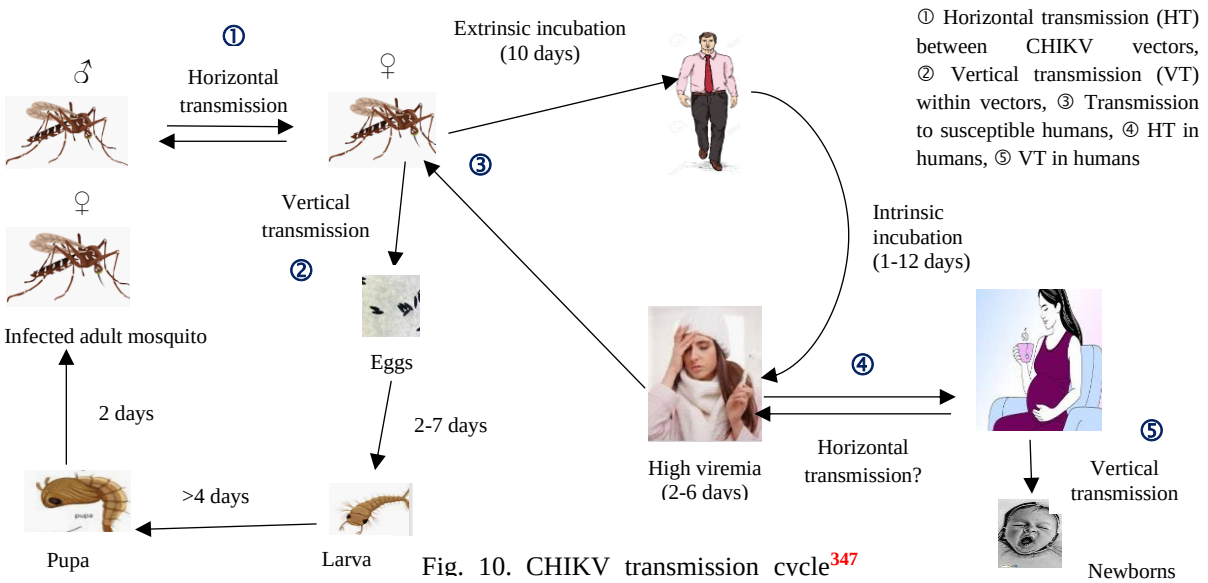


Fig. 10. CHIKV transmission cycle[347]

and can be transmitted to a new human host when the mosquito bites them. The virus again begins to replicate in this newly infected person and reaches high concentrations in their blood. At this point, they can further infect other mosquitoes and perpetuate the transmission cycle (Fig. 11)

Chikungunya in Bangladesh

CHIKV was first reported in Bangladesh in December 2008 in two adjacent north-western districts, Rajshahi and Chapai Nawabgonj. Subsequently, outbreaks were reported in 2009, 2011, and 2012.[448,449] The IgM antibodies against chikungunya virus were detected in 166 (69%) of 242 symptomatic individuals and 48 (28%) of 171 individuals without symptoms consistent with chikungunya infection in the Tangail district.[450] Chikungunya has been reported as an emerging infection in Bangladesh, with six recorded clinical cases in patients at a tertiary teaching hospital in Dhaka city.[451] In 2017, Bangladesh experienced the largest CHIKV outbreak with 13,176 clinically confirmed cases in 17 out of 64 districts of the country, primarily in Dhaka.[352] A cross-sectional study of the Chikungunya outbreak between July 24 and August 5, 2017, recorded 1,326 chikungunya cases in Dhaka city, of which 18% (239/1326) constituted confirmed cases and 82% (1087/126) were probable cases.[353] A modeling study predicted a peak prevalence of 47 cases per 1,000 people in Dhaka City during the 2017 outbreak.[354]

Nationwide surveillance conducted between 2015 and 2016 reported a seroprevalence of 2.4% and predicted that 4.99 million people would be infected with CHIKV before the 2017 major outbreak in Bangladesh.[355] The re-emergence of CHIKV in Dhaka followed a near disappearance of the virus after the 2017 outbreak, highlighting a serious concern, especially in the context of the ongoing and large-scale dengue epidemic in Bangladesh. [345,355]

A chikungunya outbreak in Dhaka city was reported in the third week of October 2024, affecting 394 suspected patients, of whom 138 (35%) were confirmed positive for CHIKV. The cases were predominantly male (64.5%) and over 30 years old (83.3%), with 98.6% residing in Dhaka.[346] Sequence data analysis of 12 samples confirmed the circulation of the ECSA genotype, with all samples containing the E1-K211E substitution.[354] Total cases of chikungunya have been reported to be 1326, of which 239 (18.02%) were confirmed cases and 1087 (81.08%) were probable cases during the outbreak of this disease in humans.[353]

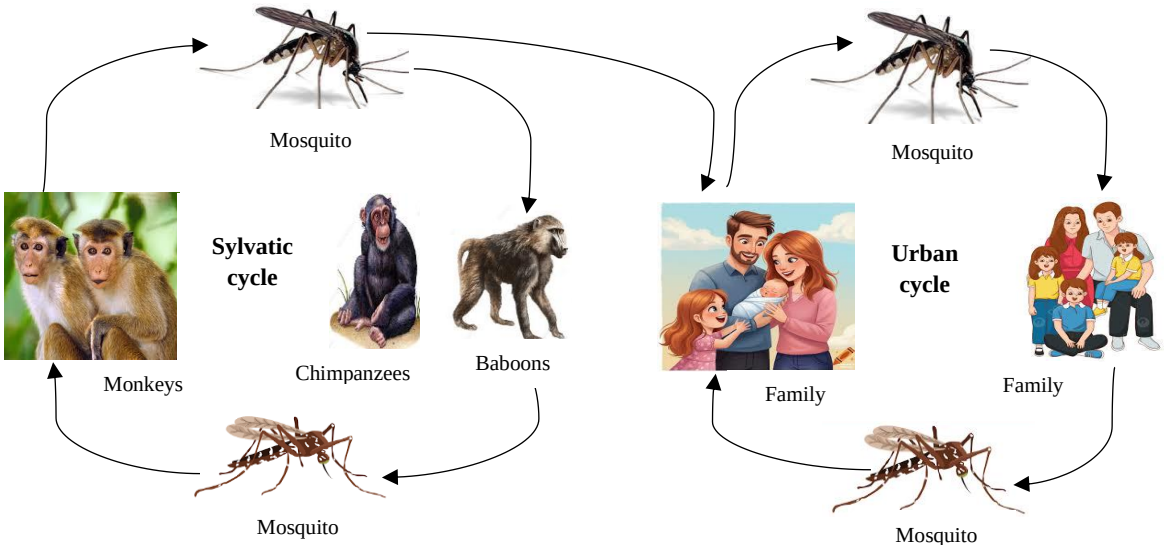


Fig.11. Sylvatic and urban cycles of chikungunya virus.

Chikungunya virus (CHIKV) has two distinct transmission cycles. During epidemics, humans are the primary reservoir. In Africa, a sylvatic cycle involves non-human primates (like monkeys), rodents, and birds, with wild Aedes mosquitoes acting as vectors (Fig. 11). While a sylvatic cycle hasn't been definitively identified outside Africa, studies suggest other vertebrate species, like certain reptiles and amphibians, may play a role in maintaining the virus.[356]

Pathogenesis

CHIKV directly enters the subcutaneous capillaries following intradermal inoculation by infected mosquitoes and infects susceptible cells in the skin: macrophages, fibroblasts, and endothelial cells, where limited replication occurs. Locally produced viruses are transported to secondary lymphoid organs, where they infect migratory cells and release viruses into the lymphatic circulation, which then proceeds to the blood. Once in the blood, the virus has access to various parts of the body, including the liver, muscle, joints, and brain. In these tissues, the infection is associated with a marked infiltration of mononuclear cells. Mononuclear cell infiltration and viral replication in muscles and joints are associated with pain.[357]

Clinical findings

After an incubation period of 4-8 days following the bite of an infected mosquito, infection with CHIKV typically causes symptomatic illness in most infected individuals, characterized in the acute phase by fever, joint pain, and a rash on the skin.[358-362] Other signs include joint swelling, muscle pain, headache, nausea, fatigue, and edema (Tables 25 & 26). Joint pain and dysfunction may persist for months to years following infection.

Table 25. Comparison between chikungunya and dengue fever[363]

SN	Signs & symptoms	Chikungunya	Dengue fever
01.	Presentation	Fever with joint pain	Fever, headache, myalgias, bleeding
02.	Characteristic of fever	Abrupt onset, lasting 3-5 days	Acute onset, lasting 5-7 days
03.	Skin rash	Appears on day 2 or 3	Appears between days 5-7
04.	Polyarthralgia/Polyarthritis	Frequent	Less common
05.	Musculoskeletal system	Arthralgia	Myalgia
06.	Bleeding manifestations	Uncommon	Common
07.	Organ involvement	Rare	Common
08.	Hypovolemic shock	Rare	Frequent in severe form
09.	Leukopenia	Infrequent	Common
10.	Thrombocytopenia	Infrequent	Common
11.	Hematocrit	Normal	High

Table 26. Clinical findings of Chikungunya in humans in Bangladesh [353]

SN	Symptom	Types of cases (n = 1326)		
		Total No. (%)	Confirmed No. (%)	Probable No. (%)
①	Arthralgia	1326 (100)	239 (100)	1087 (100)
②	Pain	0990 (74.7)	177 (70.1)	813 (74.8)
③	Skin rash	0923 (69.6)	187 (78.2)	736 (67.7)
④	Itching	0807 (60.9)	148 (61.9)	659 (60.6)
⑤	Headache	1025 (77.3)	165 (69.0)	860 (79.1)
⑥	Myalgia	0919 (69.3)	155 (64.9)	764 (70.3)
⑦	Edema	0239 (18.0)	053 (22.2)	186 (17.1)

Diagnosis

The symptoms of CHICKV infection overlap with dengue and Zika virus infection, and cases can be misdiagnosed. In addition, coinfections of chikungunya, dengue, and malaria have been reported globally, including in Bangladesh.[364-367] Therefore, laboratory diagnosis requires the use of direct methods, including virus isolation and RNA detection through nucleic acid amplification testing such as real-time PCR (RT-PCR).[368] Indirect methods, such as ELISA, measure IgM antibody responses to recent infections as early as the first week after illness onset and can remain detectable for approximately two months.[369] Interpretation of IgM and IgG serological tests in chikungunya includes IgM +ve & IgG -ve: Recent infection; IgM -ve & IgG +ve: Past infection; both IgM & IgG +ve: Recent or Recent past infection; and both IgM & IgG -ve: Negative infection.

Treatment

There is no specific antiviral drug treatment for chikungunya virus disease (CVD). Treatment for signs and

symptoms include rest, fluids, and the use of analgesics and antipyretics. Dengue is endemic in Bangladesh, while chikungunya has emerged as a significant mosquito-borne viral infection. Due to their shared vector, Aedes mosquitoes, co-infections of mosquito-borne zoonotic viral infections are possible and have been reviewed.[367] However, in dengue-endemic areas, acetaminophen or paracetamol is preferred as first-line treatment for fever and joint pain to reduce the risk of hemorrhage until dengue can be ruled out. For chikungunya-affected patients without any co-infection with persistent joint pain, nonsteroidal anti-inflammatory drugs, corticosteroids, including topical preparations, and physical therapy might help lessen the symptoms.

Prevention and control

Avoidance of mosquito bites offers the best protection against CHCKV infection. The primary method to reduce the transmission of CHCKV infection is through the control of mosquito vectors and the reduction of mosquito breeding sites.[370,371] Insecticides may be sprayed to kill flying adult mosquitoes, applied to surfaces in and around containers where the mosquitoes land, and used to treat water in containers to kill the immature mosquito larvae. Window and door screens should be used to prevent mosquitoes from entering homes. Repellents can be applied to exposed skin or clothing in strict accordance with product label instructions. Repellents should contain DEET, IR3535, or picaridin. Insecticide-treated mosquito nets should be used against day-biting mosquitoes by individuals who sleep during the daytime, such as young children, sick people, or older adults.[347]

Once an individual is recovered, available evidence suggests that they are likely to be immune to future chikungunya infections. There are currently two chikungunya vaccines that have received regulatory approval and/or have been recommended for use in populations at risk in several countries; however, the vaccines are not yet widely available or in widespread use.

Two chikungunya vaccines are currently licensed and approved for use in the United States. These are: (a) the live-attenuated vaccine (IXCHIQ® Valneval) @ 0.5 ml/ single dose IM for 18-year-old humans, and (b) the virus-like particle vaccine (VIMKUNYA® Bavarian Nordic) @ 0.8 ml single dose IM for ≥12 years old humans.[372]

Zika virus infection

Zika virus (ZIKV) infection is one of the critical zoonotic viral diseases affecting humans, caused by ZIKV, a member of the genus Flavivirus within the family Flaviviridae, which comprises 53 other Flavivirus species, including dengue and chikungunya viruses.[373] While most infections are mild or asymptomatic. Zika virus can cause serious complications, especially during pregnancy, leading to congenital birth defects like microcephaly. It is also linked to neurological complications in adults, such as Guillain-Barre syndrome.

Epidemiology

Although the virus was first isolated from a rhesus monkey (*Macaca mulatta*) in 1947 from the Zika forest of Uganda, it was confirmed in 1952 by a serological survey in the same country that Zika can infect humans.[374,375] ZIKV is a mosquito-borne flavivirus that is the focus of an ongoing pandemic and public health emergency (Fig. 12). Previously limited to sporadic cases in Africa and Asia, the emergence of the Zika virus in Brazil in 2015 heralded rapid spread throughout the Americas.[376] Currently, ZIKV infections have been reported around the world, including Asian countries.[377]

Zika virus has been detected in Bangladesh, including the identification of a cluster of cases in Dhaka. The first confirmed case was detected in 2016, with the sample collected in 2014. A total of 420 patients with Guillain-Barré syndrome (GBS) were diagnosed with ZIKV infection using internal standard criteria, including RT-PCR, ELISA, and sero-neutralization assays, over five years from 2011 to 2015 in

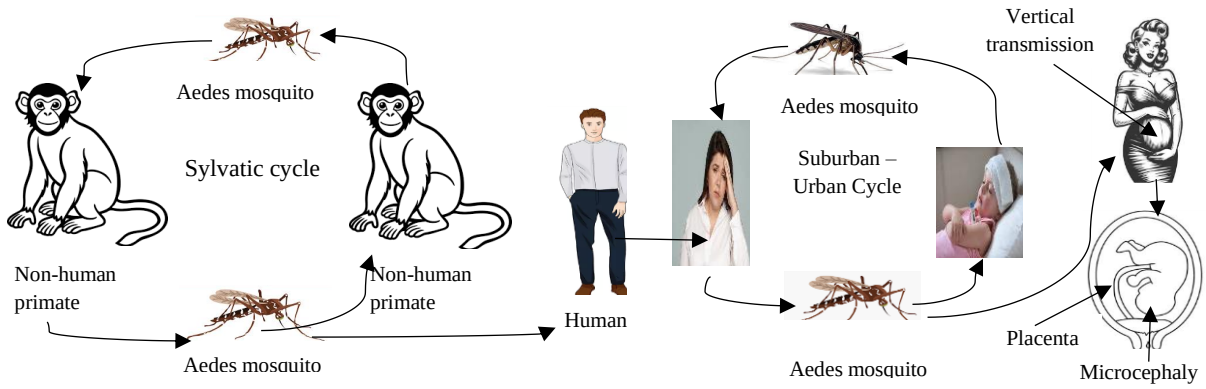


Fig. 12. Transmission cycle of Zika virus with vertical transmission

Bangladesh.[378] However, ZIKV infection in an endemic area may trigger a distinct clinical and electrophysiological subtype of GBS, although the lack of association between ZIKV and GBS indicates that ZIKV may precede a specific GBS subtype. Still, the risk is low.[379] Eight cases of Zika virus infection were reported in Dhaka in 2024.[380]

ZIKV infection remains underdiagnosed and underreported in Bangladesh due to its symptomatic similarities to the other two endemic arboviruses- dengue (DENV) and chikungunya (CHIKV). Bangladesh experienced a sharp increase in dengue cases in 2023, with 321,179 reported infections and 1,705 deaths, resulting in a case fatality rate of 0.5%. Five ZIKV-positive cases were identified, including DENV-ZIKV co-infections, during the 2023 study outbreak in Bangladesh.[381] More recently, a cluster of five Zika cases was identified in the Mohakhali area of Dhaka in 2023. Zika virus is a zoonotic virus primarily transmitted to humans through the bite of infected Aedes mosquitoes (*Aedes aegypti* and *Aedes albopictus*). It can also be transmitted through sexual contact, from mother to fetus during pregnancy, and through blood transfusion.[373]

Mosquitoes of the Aedes genus primarily transmit ZIKV. The Aedes mosquitoes lay eggs in areas of high moisture, which undergo metamorphosis from larvae to pupae and eventually to the adult form. This cycle takes approximately one and a half to three weeks to complete. Once the female mosquito takes a blood meal, it can produce on average 100 to 200 eggs per batch, yielding on average five batches of eggs during her lifetime.[382]

The adult female form acquires blood to produce eggs by biting humans and animals. The virus originated in non-human primates in tropical rainforests and infected them as described in the sylvatic cycle. The subspecies of the Aedes mosquitoes, Aedes africanus, Aedes furcifer taylori, and *Aedes dolphins take blood meals from chimpanzees*, monkeys, and baboons. Humans are affected when the sylvatic cycle transitions into the urban cycle. In this case, the subspecies of the Aedes mosquitoes, *Aedes aegypti* and *Aedes albopictus* takes a blood meal from humans. Lastly, it can be transmitted from human to human through sexual intercourse, blood transfusions, and in utero (Fig. 12).

Clinical findings

Most infections are mild and self-limiting. The incubation period for ZIKV disease is around 2 to 7 days. When symptoms are present, they are typically characterized by an influenza-like syndrome accompanied by conjunctivitis, mild fever, malaise, headache, dizziness, joint pain, and a maculopapular rash on the skin. While most infections are asymptomatic or mild, Zika virus can cause serious complications, including microcephaly and other congenital brain abnormalities in babies born to infected mothers (Fig. 12), as well

as arthralgia, Guillain-Barre syndrome, and cardiovascular anomalies in adults.[382,383]

Diagnosis

Virus isolation and serological methods are used for laboratory diagnosis and examination of ZIKV.[273] Virus isolation needs several days (1-2 weeks), while convalescent and acute sampling and cross-reactions among flaviviruses are the limitations for serological methods. RT-PCR is used for confirmation of ZIKV infections, whereas ELISA can detect IgM against ZIKV. RT-PCR is a timesaving, specific, and sensitive method for detecting ZIKV in serum, cell culture, blood, or saliva samples.

Public health impact

The Zika virus is believed to have originated in non-human primates (via a zoonotic cycle) and then spread to humans through the bite of infected mosquitoes. While Aedes mosquitoes are the primary vectors, other mosquito species have also been implicated in transmission.

The potential for severe congenital complications and the spread of the virus through various transmission routes make Zika a significant public health concern.

Treatment and Prevention

There is no specific treatment due to the viral causative agent. Symptomatic treatment of ZIKV-associated conditions, paracetamol to relieve pain and fever, drugs to treat heart rhythm problems, pain medication for arthralgia, glaucoma management, and other palliative treatment options. Nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided for hemorrhagic side effects.

Prevention involves the installation of various mosquito control measures to curb the spread of diseases transmitted by mosquitoes. Pregnant women and individuals at high risk should receive proper medical care to control this infection.

Zoonotic viruses pose a significant threat to global human and animal health, frequently causing emerging and re-emerging pandemics. Pathogenic RNA viruses are the most important group involved in zoonotic disease transmission, posing a challenge to global disease control due to their rapid mutation rates, high transmissibility, and ability to adapt to host immune responses and antiviral agents. There are currently 180 recognized species of RNA viruses that can infect humans, with an average of two new species being added each year.[384] RNA viruses tend to mutate much more rapidly than DNA viruses. This is because the enzymes responsible for replicating their genetic material (RNA-dependent RNA polymerases) are less accurate than the DNA polymerases used by the DNA viruses. This high mutation rate enables RNA viruses to rapidly evolve resistance to antiviral drugs and evade host immune responses, thereby increasing their likelihood of establishing zoonotic infections. The faster mutation rate contributes to their rapid evolutionary rate, enabling them to infect a broader range of host species, including humans, more efficiently. A significant proportion of emerging and re-emerging infectious diseases are caused by RNA viruses, underscoring their importance in zoonotic transmission. This also translates to the fact that pathogenic RNA viruses, which cause numerous diseases and deaths in humans, represent the major viral group involved in zoonotic disease transmission and are responsible for worldwide pandemics.[385]

RNA viruses pose particularly high zoonotic risks, and a statistical analysis of 146 livestock viruses indicated that the ability of a virus to replicate in the cytoplasm (without nuclear entry) is the strongest single predictor of cross-species transmission and the ability to infect humans.[21] Previous studies have reported that RNA viruses are responsible for approximately 94% of zoonotic diseases documented between 1990 and 2010. Approximately 160 species of human-infectious RNA viruses (89% of the total) are considered zoonotic, meaning they can also infect other types of vertebrate hosts.[384]

Seven major pandemic zoonotic RNA virus infections occurred from 1997 to 2021 and were categorized

into three groups. The first group comprises highly pathogenic avian influenza (HPAI-H5N1) and swine-origin influenza (H1N1) viruses, with cumulative fatality rates of 53.5% and 0.5% in humans, respectively. Moreover, HPAI-H5N1 infection caused 90-100% death in poultry and economic losses of >$ 10 billion worldwide. Similarly, H1N1 caused a severe infection in swine and financial losses of 0.5-1.5% of the GDP of the affected countries. The second group consists of severe acute respiratory syndrome-associated coronavirus infection (SARS-CoV), Middle East Respiratory Syndrome (MERS-CoV), and coronavirus disease 2019 (COVID-19), with case fatalities of 9.6%, 34.3%, and 2.0%, respectively, in humans. Still, this group only caused mild infections in animals. The third group consists of Ebola and Zika virus infections, with case fatalities of 39.5% and 0.02%, respectively, in humans but causing only mild infections in animals.[23]

Both the emerging and re-emerging viral infections represent a continuous problem to human and animal health. RNA viruses are often highlighted as the most common class of pathogens responsible for new human diseases, with a rate of 2 to 3 novel viruses being discovered each year.[386]

The biological diversity and rapid adaptive rates have proven to be challenging to overcome, and this has stimulated the continuous development of pharmaceutical and medical technologies. The constant monitoring of viral genetics and phenotypes in wildlife reservoirs is also crucial, as reservoirs may be a continuous source of novel pathogenic material for humans. Therefore, RNA viral investigations in animals have been emphasized in preparation for future viral zoonoses, particularly in the search for viruses capable of infecting humans.

During the last 25 years, it has been painfully evident that modern medicine, despite all its remarkable advances, is woefully unprepared for the epidemic threats, especially viral zoonotic threats. One need only examine the recent human morbidity from epidemics of West Nile virus, SARS-CoV-1, Marburg virus, H1N1 influenza A pandemic virus, MERS-CoV, H7N9 avian influenza virus, Ebola virus, Zika virus, Yellow fever virus, Lassa virus, and SARS-CoV-2 to affirm this position.[387] Part of the difficulty in preventing such zoonotic viral epidemics lies in a lack of understanding of the nature of these threats. Zoonotic viral spillover and spillback, which are not well understood, are essential for preventing these outbreaks. Spillover refers to the transmission of a pathogen from one species (typically the reservoir, but potentially an amplifying or bridge host) to a novel, susceptible species, thereby establishing an infection in this new host. Spillback is a specific case of spillover, where a pathogen spills from a new host back to its original host. Example: a novel virus (blue virus) emerges in pigs and is transmitted to humans in a spillover event. This virus is amplified in humans and spills back to another group of pigs. In this second group of pigs, the virus combines with other swine viruses and undergoes further changes. This new virus has the potential to cause new morbidity in both pigs and humans.

Spillover and spillback events are addressed in five ways that clinicians can improve detection and containment responses for emerging microbial pathogens, especially viral ones.[387] (a) Viruses are everywhere, (b) Extant medical diagnostics can miss clinically meaningful viral infections, (c) All viral spillovers are not the same, (d) Viral spillback is a global and domestic public health threat, and (e) Clinicians have obligations to be knowledgeable, vigilant agents of detection.[387]

The warm, humid tropical climate of Bangladesh, combined with its long monsoon season, provides optimal breeding conditions for Aedes mosquitoes, making the country particularly vulnerable to various mosquito-borne diseases. Seasonal epidemics of dengue have been a growing public health concern in recent years, culminating in the most significant outbreak to date in 2023, with 321,179 reported infections and 1,705 deaths.[388] Other emerging viral pathogens, such as chikungunya and Zika, have led to global epidemics in the past two decades, including a major chikungunya outbreak in Bangladesh in 2017.[352] Zika virus is present in Bangladesh, with the first confirmed case reported in 2016.[389] A retrospective surveillance study in 2016

revealed one confirmed Zika-positive case from 2014, suggesting that the Zika virus had been circulating in Bangladesh even before the 2015 outbreak in Brazil. More recently, a cluster of five Zika cases was identified in Dhaka in 2023, highlighting the need for wider surveillance.[390] One of the five Zika virus cases was also infected with dengue virus- the first time this coinfection has been detected in Bangladesh. [381]

Modern viral diagnostics are finely tuned to be both sensitive and specific concerning previously recognized viral pathogens. However, the modern diagnostics are still not widely available in low- and middle-income countries in Africa and South Asia, including Bangladesh.

CONCLUSIONS

Emerging and re-emerging viral zoonotic diseases pose a significant and ongoing threat to global human and animal health, including Bangladesh, leading to illness, disability, and death, with the potential to cause pandemics and major economic disruptions. These diseases, which originate in animals and can be transmitted to humans, are influenced by factors such as encroachment of human activities into wilderness areas, movement of wild animals into areas of human activity, climate change, and increased global travel and trade. Outbreaks can also result in substantial economic losses, including poultry culling and healthcare costs. A 'One Health' approach, integrating human, animal, and environmental health perspectives, is crucial for effective prevention and control. Effective surveillance, public health awareness campaigns, and improved sanitation and hygiene practices are essential to mitigating the risks associated with zoonotic diseases. The 'One Health' approach involves virological scientists, veterinarians, physicians, public health professionals, and other stakeholders working together to monitor disease outbreaks, prevent transmission, and develop effective treatments and interventions.

ETHICAL APPROVAL

Reviews do not need any ethical approvals or informed consent.

CONFLICTS OF INTEREST

The author declares no conflict of interest.

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