

Chapter 15

Viral Zoonoses: Wildlife Perspectives



Arockiasamy Arun Prince Milton, Govindarajan Bhuvana Priya, Sandeep Ghatak, and Samir Das

Abstract Wildlife plays an important and complex role in the emergence of new diseases and the maintenance of endemic infectious diseases. The majority of the recent emerging diseases were caused by zoonotic viruses of wildlife origin and had significant impacts on public health and economies. Wildlife can act as a reservoir or maintenance or spill-over or amplifier hosts or simply a liaison host of diseases transmissible to human beings and farmed livestock. Anthropogenic factors like agricultural expansion, habitat destruction, urbanisation, trade of exotic or domestic animals and global travel comprise major drivers of the emergence of zoonotic disease. The viral families *Arenaviridae*, *Coronaviridae*, *Flaviviridae*, *Filoviridae*, *Hepeviridae*, *Hantaviridae*, *Herpesviridae*, *Nairoviridae*, *Orthomyxoviridae*, *Peribunyaviridae*, *Paramyxoviridae*, *Phenuiviridae*, *Poxviridae*, *Reoviridae*, *Rhabdoviridae*, and *Togaviridae* enclose viruses which represent most of the viral zoonoses of wildlife origin. The basic factors influencing the disease emergence from wildlife species are also the major drivers of biodiversity loss. Therefore, emerging zoonotic viruses are not only potential threats to human beings but can also be harmful to wildlife species. Thus, there is a convincing and effective chance for mutual gains for the conservation of wildlife and public health by collective and collaborative attempts.

Keywords Wildlife · Viral zoonoses · Emerging viruses · Spill-over · Spill-back · Reservoirs · Migratory birds

A. A. P. Milton (✉) · G. B. Priya · S. Ghatak · S. Das
Division of Animal Health, ICAR Research Complex for NEH Region, Umiam, Meghalaya,
India

15.1 Prologue

Today's world does not have any barrier between the animal and human medicine. The majority of infectious diseases affecting human are of zoonotic origin, and for several emerging diseases, wildlife serves as a reservoir (Jones et al. 2008). In the emergence of new diseases as well as maintenance of endemic infectious diseases, wildlife plays an important and complex role. The word "emerging disease" has received greater importance in the last 20 years in the popular press, owing to well-publicised disease outbreaks such as severe acute respiratory syndrome (SARS), Ebola haemorrhagic fever, Monkeypox, West Nile fever, Nipah, and Hendra viral encephalitis. These events have augmented global attention to the association of wildlife in emerging diseases (Travis et al. 2011). Over the past few decades, roughly 75% of emerging diseases including zoonoses had wildlife origin (Jones et al. 2008) and more than 70% of emerging or reemerging infectious agents are thought to have wildlife as their natural reservoirs (Taylor et al. 2001). Although wildlife has a crucial role in preserving the integrity of planet's ecosystem, it frequently embodies a significant risk of emerging zoonotic diseases (Daszak et al. 2000; Thompson et al. 2009). In significant ways, wildlife differs from other domestic animal species. They are elusive, usually have no owners or custodians, not always well recognised by zoologists and are often taken emotionally by the general public (Artois et al. 2011). Wildlife can act as a reservoir or maintenance or spill-over or amplifier hosts or simply a liaison host of diseases transmissible to human beings and farmed livestock. For example, in continental Europe, it was well-known that rabies had gone astray as a disease maintained by dogs but instead turned into disease spread by a red fox (*Vulpes vulpes*). Such situations arise where wildlife hosts are responsible for maintaining and spreading zoonotic diseases; thus, there is a rising concern in developing means to control the transmission of disease from wild animal population to humans or farm animals (Artois et al. 2011).

The majority of the recent emerging diseases were caused by zoonotic viruses of wildlife origin and had a significant influence on public health and economies (Murray et al. 2016). The part of wildlife species in diseases like severe acute respiratory syndrome (SARS), Influenza, Ebola haemorrhagic fever, Nipah viral encephalitis, and monkeypox is well-acknowledged. The emergence and rapid spread of such fatal diseases have been most important arousing public health episodes that accentuated the want for group effort between the veterinarian, wildlife professionals and public health specialists (Chomel et al. 2007). It has also increased the interests of the general public on diseases of wildlife origin, and as a result wild game managers, conservationists and government agencies have shown greater interest in surveillance and control of wildlife diseases (Gortazar et al. 2007). Pathogens of wildlife origin spill over into domestic animals, into humans and other wild animals. Zoonoses of wildlife origin have a negative bang on public health, wildlife conservation, and agricultural production (Chomel et al. 2007). It is now far and widely accepted that the complete purge of such shared pathogens is impractical if wildlife reservoirs are ignored (Gortázar et al. 2015). The viral families

Arenaviridae, *Coronaviridae*, *Flaviviridae*, *Filoviridae*, *Hepeviridae*, *Hantaviridae*, *Herpesviridae*, *Nairoviridae*, *Orthomyxoviridae*, *Peribunyaviridae*, *Paramyxoviridae*, *Phenuiviridae*, *Poxviridae*, *Reoviridae*, *Rhabdoviridae*, and *Togaviridae* contain viruses which represent most of the viral zoonoses of wildlife origin. The present chapter will focus on viral zoonoses involving wildlife hosts, their ecology and transmission modes, drivers of emergence, geographical distribution, and control strategies.

15.2 Spill-Over and “Spill-Back”

The spread of pathogens from domestic reservoir animals to the sympatric wild animal population, termed “spill-over”, underlines the emergence of a variety of emerging infectious diseases from wildlife. Spill-over is a scrupulous threat to endangered fauna, as the existence of infected reservoir animals can reduce the infectious agent’s threshold density and lead to the extinction of local populace (Daszak et al. 2000). African wild dog population (*Lycaon pictus*) has been waning since the 1960s and is now endangered and, with a patchy population of <5000, is vulnerable to stochastic events like outbreaks of disease. In 1991, synchronised with canine distemper epizootic in domestic sympatric dogs, wild dogs have become extinct in Africa.

Similarly, rabies was responsible for mortality in wild dogs, and a common viral variant has been identified in wild and sympatric dogs. In Serengeti, the emergence of rabies in wild dogs was due to the spatial expansion of human dwellings and resulting infringement of rabid domestic dogs. Spill-over outbreaks embody a stern threat to wild fauna and through “spill-back” (reverse spill-over) to the sympatric domestic animal population. Brucellosis was possibly ingrained into America through cattle. The occurrence of brucellosis in elk and bison in Yellowstone National Park (USA) is considered a probable threat to cattle grazing at the boundaries of the park. Other instances of spill-over events include bovine tuberculosis (global), sarcoptic mange in wombats (Australia) and foxes (Europe). Bovine tuberculosis also frightens to spill back to domestic livestock and eventually, to humans (Daszak et al. 2000).

15.3 Wild/Migratory Birds, Exotic Pets, Bats, and Rodents: As Reservoirs of Zoonotic Viruses

Emerging zoonotic agents have originated from numerous wildlife species like ungulates, carnivores, birds, non-human primates, bats, and rodents (Fig. 15.1) (Singh and Gajadhar 2014). Many zoonoses were originated from wildlife, and the list is expanding over time, but the relative significance and mechanism driving the

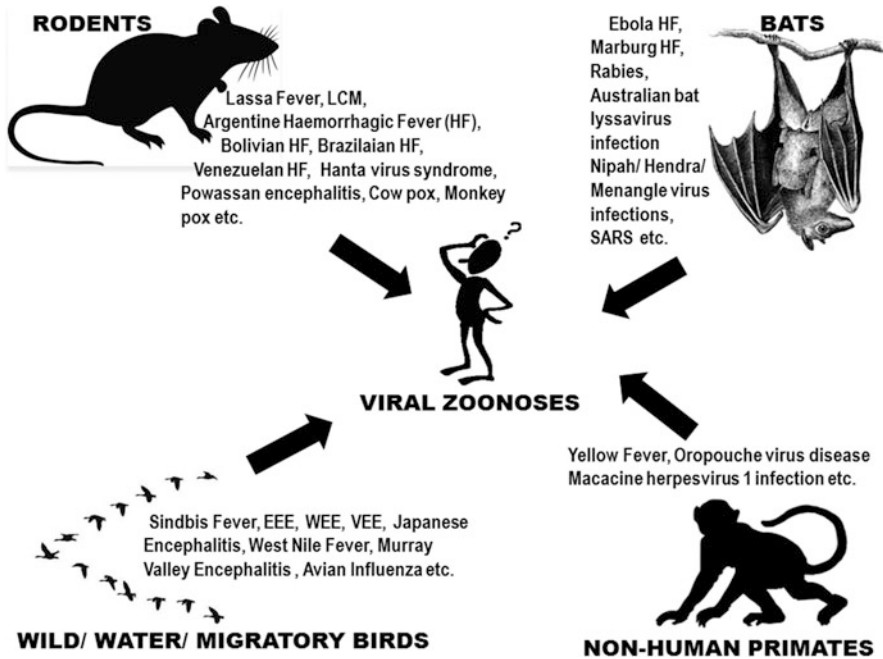


Fig. 15.1 Major wildlife reservoir group of viral zoonotic diseases

differences of various set of wild hosts in disease emergence remain unclear. With more than 1 billion cases of human zoonoses occurring each year, identifying wildlife reservoirs of the disease remains a perennial priority of public health (Han et al. 2015). Therefore identifying which species are most expected to play reservoir role of upcoming zoonotic infections and in which provinces/regions new outbreaks are expected to occur are an essential move towards a pre-emptive method to minimising zoonotic illness risk in humans.

Birds have a vital role in the transmission and spread of numerous emerging zoonotic pathogens. The West Nile virus emergence in the USA is a prominent example of how rapidly a novel zoonotic disease can become extensively dispersed. Wild birds are well-acknowledged to be reservoirs for many emerging zoonotic diseases, such as WNV, influenza A virus, Western equine encephalitis, St. Louis encephalitis, etc. Besides, wild/migratory birds are also infested with arthropod species (vector), which can disseminate zoonotic pathogens along their routes of migration, even if that particular bird species is not a capable reservoir of disease. Furthermore, avian hosts migrating over intercontinental and national borders can act as long-range competent vectors for any zoonotic pathogen. This establishes new endemic disease foci along the routes of migration (Reed et al. 2003).

Several latest epidemics have been connected with exotic pets or wildlife hosts including Ebola, SARS, and Monkeypox. For instance, the monkeypox outbreak in the USA in 2003 started after importing African rodents, which infected prairie dogs

in pet shops. Many imported species of African rodents were revealed positive for the monkeypox virus related to the outbreak (Souza 2011). From 1991 to 1998, eight cases of rabies due to the new variant of rabies virus were reported in Brazil. Marmosets (*Callithrix jacchus jacchus*) reared as pets were found to be a source of transmission. In a pet care shop in France, encephalitis was detected in an Egyptian rousette bat which was recently imported from Belgium. The pet bat was found to be carrying Lagos bat lyssavirus which leads to treatment of around 120 exposed individuals (Chomel et al. 2007).

Bats (Order *Chiroptera*) offer substantial ecosystem services, like arthropod control, pollination, and seed dispersal, over a broad range of habitats. On the other hand, bats are gaining more attention as prospective reservoirs for many emerging zoonotic diseases after the recent recognition of their association with Ebola and Marburg viruses, SARS coronavirus, and Nipah and Hendra viruses. Subsequently, there has been frequent speculation that they may be exclusive in their potential to serve as a host for viruses of zoonotic nature (Calisher et al. 2006). Generally, traits of bats that may make them suitable to harbour more viruses include moderately long life spans, which helps viral persistence; flight, letting movement and spreading over long distances and extended torpor, which can diminish both immune function and viral replication (Munshi-South and Wilkinson 2010). Also, the gregarious nature of some bat species like Mexican free-tailed bat allows them to live in dense aggregations (3000 per m²). Roosting spots can even house a diverse group of several species of bats (Luis et al. 2013). These high inter- and intraspecific contacts can favour speedy pathogen transmission, and hefty population volumes could maintain acute-immunising infections.

Furthermore, in evolutionary terms, they are ancient mammals, so it has been assumed that zoonotic viruses which evolved in them may use extremely conserved cellular receptors, hence enhancing the bat's capability to pass on viruses to other mammal species (Calisher et al. 2006). Numerous species of bats have peri-domestic behaviours, roosting in human dwellings, houses, and trees in cities, leading to repeated human contact with their excreta. In recent decades, bat-human contact is escalating due to habitat encroachment and the exploitation of bats as bushmeat (Mickleburgh et al. 2009; Luis et al. 2013).

Rodents are the diversified and superabundant living mammals on the earth. From the middle ages, it is well-known that rodents can transmit human diseases, as black rats were involved in the distribution of plague. Even today, rodents possess a significant threat to human health. Diseases distributed by rodents take two different ways. The first is a direct way, wherein rodents spread disease-causing agents to humans by biting or contaminating the food and water with their faeces or urine or through inhalation route (Hantaviruses). The second is an indirect way, wherein rodents serve as an amplifier host and transmit the pathogen to human through arthropod vectors like ticks, fleas, and mites (Crimean-Congo Haemorrhagic fever). Rodents can help to sustain the infectious agent's transmission cycles in diverse environments, varying from rural to densely populated urban areas and in the wilderness (Meerburg et al. 2009). Some rodent-borne zoonotic viral diseases are Hantavirus infections, Crimean-Congo haemorrhagic fever, Kyasanur Forest

Disease, Omsk haemorrhagic fever, Tick-borne encephalitis, Lymphocytic choriomeningitis, Lassa fever, Cowpox, etc.

15.4 Drivers of Zoonotic Disease Emergence from Wildlife

Zoonotic disease emergence is a multi-factorial event. The factors may be changes in, among others, genetics of microbes, vector distribution, human behaviour, trading, and farming practices. It is also imperative to make out that various drivers play distinctive functions in the emergence of a range of viruses, even it can be for viruses of the same family (Wang and Crameri 2014). In a joint consultation meeting of WHO/FAO/OIE held in 2004, it was ended with a conclusion that anthropogenic factors like agricultural expansion, habitat destruction, urbanisation, trade of exotic or domestic animals, and global travel comprise major drivers of zoonotic disease emergence (WHO/FAO/OIE 2004). Most of these anthropogenic factors bear negative implications for wildlife, and subsequently for human health. Nipah virus emergence demonstrated the interplay between various ecological risk factors like intensive animal agriculture, habitat destruction, and animal transport to longer distances (Greger 2007).

Agricultural drivers include intensification of farming, habitat clearing for grazing and cropping, modernisation, and newer agricultural practices. These major changes have multiple effects including pushing different wildlife species together and commingling domestic livestock and wildlife, thus facilitating spill-over and spill-back events including the transfer of novel pathogens into naive and susceptible hosts (Wang and Crameri 2014). In 1957, in India, a new flaviviral disease named after Kyasanur forest occurred owing to clearance of woods which was in turn used for grazing of cattle. Cattle are the most important host for the tick (*Haemaphysalis spinigera*) that passed the virus out from its small mammal reservoir and simian hosts. This disease now causes thousands of human cases in India each year (Greger 2007; Singh and Gajadhar 2014).

Hardwood trees cut in south-western Wisconsin, USA, form basal tree holes which collect water and increase the numbers of breeding sites for *Aedes triseriatus*, the natural mosquito vector of La Crosse virus. The reservoir of the virus is small forest mammals. The virus is also transmitted to humans, causing encephalitis, principally in pre-school age children (Williams et al. 2002).

Natural climate change is another important driver of zoonotic disease emergence from wildlife. Climate changes enhance host abundance and transmission of pathogens as in the case of the emergence of Sin Nombre Hantavirus in the USA. Higher rainfall resulted in increased grass setting and spreading out of rodent population (*Peromyscus* spp.) that are key reservoirs of the Hantavirus. Consequently, human contact with the excretions of these mice increased, which resulted in the manifestation of Hantavirus pulmonary syndrome (HPS) in humans. Even though the virus was certainly endemic in rodents for centuries, causing intermittent cases of HPS in human, until 1993, the aetiology was not discovered (Schmaljohn and Hjelle 1997).

Another important risk factor associated with zoonotic diseases emergence from wildlife is the significant increase in bushmeat consumption in several parts of the globe (Chomel et al. 2007). Although hunting of wild species for food has been in practice for millennia, a marked increase has been observed over the past few decades, and this tendency is likely to endure as one of the paramount threats to biodiversity. Bushmeat is consumed to the tune of 1–3.4 million tonnes annually in Central Africa alone (Brown 2004). The commercial trade of bushmeat in Asia, mainly in Guangdong province of China has led to the SARS epidemic (Donnelly et al. 2003) and the emergence of the H5N1 subtype of influenza virus (Chen et al. 2004). Such bushmeat consumption and illegal hunting may expose people to new or previously unknown pathogens. Increased reliance on wildlife to meet dietary protein might have increased due to land-use change, deforestation activities, and food insecurity in various parts of the world, predominantly in tropical developing countries. Change in climate is also likely to affect food security in various parts, further encouraging greater reliance on bushmeat. This is set in contradiction of increasing air travel around the globe, which already poses an important risk to public health globally employing the transportation of infectious agents (Murray et al. 2016).

Another important driver of disease emergence from wildlife is the trade of wildlife and wildlife products. Recently, globalisation has caused an unprecedented amount of such trades across the globe, both legally and illegally in the form of exotic pets, medicines, crafts, trophies, bushmeat, etc. Such trade represents a considerable risk for the public, domestic animals, and wildlife health globally (Travis et al. 2011). International legal wildlife trade is roughly US\$159 billion annually (Brown 2004). Given the covert nature and large size of the business, no estimate of the volume of wildlife trafficked throughout the globe is present. The USA is involved in the maximum consumption of wildlife and wildlife products with the legal importation of live animals to the tune of 1.5 billion between 2000 and 2006 and closely 90% of which were meant for the pet industry. And as far as non-live wildlife is concerned, an average of 25 million kilograms enters the USA annually. The Monkeypox outbreak displayed that a single consignment of infected pet animals can end up in a serious impact on human health, underlining the challenges encountered by agencies trying to regulate or control the legal and illegal business of wildlife (Smith et al. 2012).

Zoological collections are also places where pathogens could spread from one species to others, to initiate a new disease. An African rodent species born and raised in an Asian zoo could be found in a South American Asian zoological collection housed adjacent to Arctic mammals from North America. Therefore the number of permutations of novel organismal biomes for pathogens or commensals to explore has increased exponentially (Brown 2004).

15.5 *Arenaviridae* Zoonotic Infections

Arenaviruses have been categorised based on their antigenic traits into two sero-complex groups, the Tacaribe group and Lassa-Lymphocytic choriomeningitis group. This has been further classified into four evolutionary lineages. All arenaviruses are more or less strongly associated with a specific mammalian host. The host distribution decides the distribution of each arenavirus (Salvato et al. 2005). The diversity of the viruses is mostly due to the long-time shared evolutionary association (co-speciation or -evolution) between the *Muridae* family of rodents and viruses of the *Arenaviridae* family (Bowen et al. 1997). In nature, the long-time persistence of arenaviruses depends on the chronic infection of the rodent host along with chronic viraemia. Out of 23 species in *Arenaviridae* family, five arenaviruses are established to cause a terrible haemorrhagic fever with a 20% case fatality rate. They are Lassa, Machupo, Junin, Guanarito, Sabia distributed in western Africa, Bolivia, Argentina, Venezuela, and Brazil, respectively (Table 15.1) (Delgado et al. 2008; Briese et al. 2009; Charrel and de Lamballerie 2010). Any manipulation of these viruses has to be done in BSL 4 facilities as they are incorporated in Category A list of pathogens designated by CDC. Lymphocytic choriomeningitis virus (LCMV) (Table 15.1) can cause congenital malformations and central nervous system infection; it has also recently been identified as a significant cause of grave infection in immunocompromised patients and organ transplantation recipients (Emonet et al. 2007; Charrel and de Lamballerie 2010). The natural hosts of the arenaviruses are rodents. Old World arenaviruses like Lassa fever virus, LCMV are allied with rodents of subfamily *Murinae* in family *Muridae*. While New World arenaviruses are related to new world rodents in the subfamily *Sigmodontinae* of family *Muridae* (Wilson and Reeder 2005). Human beings may contract arenaviruses through bites or any means of direct contact with virus-infected rodents or via inhalation of infected rodent secreta or excreta. Hence, one of the chief determinants of human infection is probably the dynamics of rodent populations. The major contributing factor aiding virus transmission to human from rodent is the peri-domestic and domestic behaviour of these rodent reservoir hosts.

Nevertheless, in majority cases, arenavirus transmission occurs after agricultural or recreational incursions into environments giving critical habitat for reservoir rodent hosts. Besides, professionals working with infected rodents in laboratory and field are at greater risk (Sewell 1995). Generally, natural ecological changes and anthropogenic modifications of the environment have been incriminated in the arenaviruses infection emergence in humans due to changes in the behaviour of the rodent population (Charrel and de Lamballerie 2010).

Table 15.1 List of *Arenaviridae*, *Peribunyaviridae*, *Phenuiviridae*, *Nairoviridae*, and *Hantaviridae* zoonotic diseases involving wildlife species

S. no	Viral zoonotic diseases	Virus aetiology genus, family (virus)	Wildlife reservoirs/ amplifiers/natural hosts/spill-over hosts	Major transmission route to humans	Geographical distribution	Human disease	BSL level	References
1	Argentine hemorrhagic fever	<i>Mammarenavirus</i> , <i>Arenaviridae</i> (Junin virus)	Reservoir—dry-land vesper mouse (<i>Calomys musculinus</i>)	Direct contact with infected rodents/inhalation of rodent excreta	Argentina	Severe hemorrhagic fever	4	Charrel and Lamballerie (2010)
2	Bolivian hemorrhagic fever	<i>Mammarenavirus</i> , <i>Arenaviridae</i> (Machupo virus)	Reservoir—large vesper mouse (<i>Calomys callosus</i>)	Direct contact with infected rodents/inhalation of rodent excreta; person-to-person	Bolivia	Severe hemorrhagic fever	4	Charrel and Lamballerie (2010)
3	Brazilian hemorrhagic fever	<i>Mammarenavirus</i> , <i>Arenaviridae</i> (Sabia virus)	Rodents	Direct contact with infected rodents/inhalation of rodent excreta	Brazil	Severe hemorrhagic fever	4	Charrel and Lamballerie (2010)
4	Lassa fever	<i>Mammarenavirus</i> , <i>Arenaviridae</i> (Lassa virus)	Reservoir—natal multimammate mouse (<i>Mastomys natalensis</i>)	Direct contact with infected rodents/inhalation of rodent excreta	West Africa	Severe hemorrhagic fever	4	Charrel and Lamballerie 2010
5	Lymphocytic Choriomeningitis	<i>Mammarenavirus</i> , <i>Arenaviridae</i> (LCM virus)	Reservoir—house mice (<i>Mus musculus</i> , <i>M. domesticus</i>)	Direct contact with infected rodents/inhalation of rodent excreta	Worldwide	Acute central nervous system disease and congenital malformations	2/3	Charrel and Lamballerie (2010)

(continued)

Table 15.1 (continued)

S. no	Viral zoonotic diseases	Virus aetiology genus, family (virus)	Wildlife reservoirs/amplifiers/natural hosts/spill-over hosts	Major transmission route to humans	Geographical distribution	Human disease	BSL level	References
6	Venezuelan hemorrhagic fever	<i>Mammarenavirus</i> , <i>Arenaviridae</i> (Cuanarito virus)	Reservoir—common cane mouse <i>Zygodontomys brevicauda</i> , Alston's cotton rat (<i>Sigmodon alstoni</i>)	Direct contact with infected rodents/inhalation of rodent excreta	Venezuela	Severe hemorrhagic fever	4	Charrel and de Lamballerie (2010)
7	La Crosse encephalitis	<i>Orthobunyavirus</i> , <i>Peribunyaviridae</i>	Amplifying hosts—Chipmunks, squirrels	Mosquito bite (<i>Aedes</i>)	USA	Febrile encephalitis with seizures	2	Harding et al. (2018) and Maes et al. (2019)
8	Oropouche virus disease	<i>Orthobunyavirus</i> , <i>Peribunyaviridae</i>	Reservoir—pale-throated sloths (<i>Bradypus tridactylus</i>), non-human primates, rodents and some wild birds	Mosquito bite (<i>Culicoides paraensis</i> , <i>Culex</i>)	Brazil, Panama, Peru, and Trinidad and Tobago	Self-limiting dengue-like, acute febrile illness with arthralgia	3	Sakkas et al. (2018) and Maes et al. (2019)
9	Rift valley fever	<i>Phlebovirus</i> , <i>Phenuiviridae</i>	Wild spill-over hosts—African buffaloes, Warthog, black rhino, zebra, Thompson's gazelle, lesser kudu, impala waterbuck, lions, cheetahs, African wild dogs and jackals	Mosquito bite (<i>Aedes</i> , <i>Culex</i>)	Eastern and Southern Africa, Saudi Arabia	Self-limiting dengue-like, acute febrile illness with arthralgia	3	Bird et al. (2009) and Maes et al. (2019)

10	Crimean-Congo haemorrhagic fever	<i>Orthonairovirus</i> , <i>Nairoviridae</i>	Natural hosts hedgehogs, hares and ground-feeding birds	Tick bite and person-to- person	Western areas of the former Soviet Union; Southeast- ern and Southwest- ern Europe; central eastern; central Asia; the Middle East and Turkey; and Africa	Mild and nonspecific febrile illness to severe hemorrhagic disease	4	Bente et al. (2013) and Maes et al. (2019)
11	Diseases caused by hantaviruses	<i>Orthohantavirus</i> , <i>Hantaviridae</i>	Reservoir hosts— rodents, shrews, moles, and bats	Inhalation, ingestion and transcutaneous	Worldwide	Hemorrhagic fever with renal syn- drome (HFRS), Nephropathia epidemica (NE) and Hantavi- rus cardiopulmo- nary syndrome (HCPS)	3	Jiang et al. (2017)

15.6 *Peribunyaviridae* Zoonotic Infections

La Crosse Encephalitis and Oropouche Virus Disease are the two important zoonotic viral infections associated with wildlife belonging to *Peribunyaviridae* (Maes et al. 2019) (Table 15.1). La Crosse Encephalitis caused by La Crosse (LAC) virus was initially isolated in La Crosse, the USA in 1964 from the brain of a young girl diagnosed with encephalitis. In North America, LAC encephalitis is the second most frequently described mosquito-borne disease next to West Nile viral encephalitis. According to a CDC report, an inconsistent number of 30–130 human severe clinical cases has been reported in the USA annually with majority victims being children under the age of 16 (Harding et al. 2018). Unlike Yellow fever and dengue fever, LAC encephalitis infections are generally contracted in or near the wilderness. Suggested reservoir or amplifying hosts are the eastern grey squirrel and the eastern chipmunk. These animals drink from the tree holes, wherein they transmit the virus to the tree hole mosquito, *Aedes triseriatus*, which is a vector mosquito for this disease (Sutherland 2008; Harding et al. 2018). The important risk factor for LAC infection is the proximity to artificial or natural breeding sites. Humans are generally dead-end or incidental hosts and occasionally acquire an adequate dose of LAC virus from mosquito bites to build up an infection (Bewick et al. 2016; Harding et al. 2018).

Oropouche fever, similar to dengue fever is an acute febrile disease caused by Oropouche virus (OROV). OROV was initially isolated from the forest conservation worker in Trinidad. This disease is currently endemic, causing sporadic outbreaks and cases in some parts of Central and South America. OROV is an arbovirus transmitted to humans mainly by the *Culicoides paraensis* (biting midge). This virus is maintained in nature by an urban and sylvatic cycle which may comprise quite a few different vector species. In the urban cycle, the primary vector is *C. paraensis*, which has been associated with larger epidemics (Mourao et al. 2015; Sakkas et al. 2018). Wild mammals and birds are the natural reservoir hosts in the sylvatic cycle. OROV antibodies have been found in non-human primates such as black and gold howler monkeys, capuchin monkeys, black-tufted marmosets, pale-throated three-toed sloths, rodents (*Proechimys* spp.), and birds (*Thraupidae*, *Fringillidae*, *Columbidae*). These wild species may have some role in the transmission of OROV. Humans are most likely the link host between the two cycles of transmission because OROV is typically invading urban localities through a viraemic person who visits the forest and gets back to the urban residential area during viraemia (Cardoso et al. 2015; da Rosa et al. 2017). It is now well acknowledged that OROV is circulating in wildlife and humans at very low levels, and whenever a deviation in the natural environment (deforestation/loss of vegetation and habitat) and/or in the general population (immigration of animal and/or human) occurs, or pouches fever outbreaks are emerging (Sakkas et al. 2018).

15.7 *Phenuiviridae* Zoonotic Infections

Rift Valley Fever (RVF) is an important zoonotic viral infection caused by *Phlebovirus* of *Phenuiviridae* family (Maes et al. 2019) (Table 15.1). RVF is a mosquito-transmitted emerging zoonotic disease of animals and human beings in Africa and the Middle East region that is directly related to high rainfall conditions. This virus was first discovered from aborted sheep in 1930 in Kenya (Linthicum et al. 2016). A change from enzootic to epizootic RVF virus activity characteristically occurs following extended episodes of exceptionally plentiful rainfall and consequent inundation of dambos, which facilitates the emergence of abundant *Aedes* mosquitoes. These infected mosquitoes feed on livestock (e.g., cattle and sheep) that rapidly build up clinical disease and high-titre viraemias and in sequence; the infected animals infect bridge mosquitoes such as *Anopheline* or *Culex* spp. Humans develop disease following an infected mosquito bite or exposure to aerosols or from handling aborted materials or transcutaneous injury during necropsy or slaughtering of viraemic animals (Bird et al. 2009; Linthicum et al. 2016). The ungulate livestock, especially sheep, goats, and cattle, assume a central role in RVF epidemics and epizootics. The role of wildlife species in the maintenance of RVF virus during inter-epizootic times or as amplifier hosts has been well-studied since the discovery of the virus. Serological evidence from South Africa suggests wild rodents may play some role in the virus maintenance. A high prevalence of antibody was found in many species of wild animals, including giraffe, African buffalo, black rhino, common warthog, Thompson's gazelle, zebra, impala waterbuck, lions, African wild dogs, jackals, cheetahs, and lesser kudu during and immediately after the 2006–2007 East African epizootic (Bird et al. 2009; Linthicum et al. 2016).

15.8 *Nairoviridae* Zoonotic Infections

Crimean-Congo Haemorrhagic Fever (CCHF) is the important zoonotic viral infection caused by *Orthonairovirus* of *Nairoviridae* family (Maes et al. 2019) (Table 15.1). CCHF is the tick-borne viral zoonotic disease, causing outbreaks or sporadic human cases across a vast geographical area, from China to the Middle East and Europe (south-eastern) and many parts of Africa. It was first described in the Crimea region of the post-Soviet states in 1944 and the Congo (present DR Congo) in 1956 (Spengler et al. 2019). The CCHF virus in nature is maintained in a tick–vertebrate–tick endemic cycle, wherein the ixodid ticks serve as both vector and true reservoir of the virus as they remain infected throughout their lifetime unlike transient viraemia in mammals. *Hyalomma* ticks are the primary source of human illness, most likely because both adult and immature forms vigorously look for hosts for blood meal during every stage of maturation specifically during spring and summer. The broad distribution of *Hyalomma* ticks reveals their tolerance of varied environments, including steppe, savannah, and small forest areas, and the capability

of their vigorously questing larvae and nymphs to feed on a range of hosts, including hedgehogs hares and ground-feeding birds, whereas the adults aggressively seek out sheep, cattle, and other large ruminants (Bente et al. 2013; Spengler et al. 2019). Human beings most frequently agricultural workers, slaughterhouse workers, and medical personnel contract the infection through an infected tick bite, contact with infected blood/tissues of animals, and contact with secretions of infected patients, respectively. Climate change is frequently considered as a major factor for the virus spreading out, but evidence proposes that other factors such as agricultural abandonment, landscape fragmentation, and proliferation of wildlife hosts are also instrumental in disease emergence and outbreaks (Spengler et al. 2019).

15.9 *Hantaviridae* Zoonotic Infections

Hantaviruses (Table 15.1) of the *Hantaviridae* family are considered as emerging viruses with a rising number of clinical cases of humans worldwide. The earliest pathogenic Hantavirus was isolated in 1976, by the side of the Hantan River, in South Korea and was named as Hantaan virus. These viruses have a worldwide distribution and are major zoonotic pathogens causing severe infection in humans. More than 50 strains of Hantaviruses have been identified so far, and 24 of them have pathogenic bearing to humans (Jiang et al. 2017). The latest data states that, globally, more than 20,000 clinical cases of Hantaviruses have been estimated to occur every year, with most of the cases reported in Asia (Jiang et al. 2017). Naturally, Hantaviruses are maintained in asymptomatic specific reservoir hosts. Rodents, moles, shrews, and bats are the regular reservoir hosts of Hantaviruses. *Rattus norvegicus* and *Apodemus agrarius*, which are host species for the *Hantaan virus* and *Seoul virus*, are the principal reservoirs in the residential area and wild, respectively (Zhang et al. 2014). Though chronic and persistent infections are well established along with high-titre neutralising antibodies, these reservoirs stay as asymptomatic infected hosts (Yu and Tesh 2014). Like arenaviruses, each Hantavirus is connected with a specific rodent host, and spill-over to other species of rodents seems to provoke specific antibody production and virus clearance (Spengler et al. 2013). In general, Hantaviruses coevolve with their specific hosts (Vaheiri et al. 2013). Recently a Hantavirus (Xuan Son virus) has also been found in bats in Vietnam (Arai et al. 2013). Two acute diseases are caused by hantaviruses in humans, haemorrhagic fever with renal syndrome (HFRS), and Hantavirus cardiopulmonary syndrome (HCPS). HFRS primarily came to the notice of Western physicians between 1951 and 1954, when 3200 United Nations soldiers fell ill in Korea. In Europe, more than 3000 HFRS cases occur annually (Zhang et al. 2014). HFRS outbreaks are caused by Hantaan, Dobrava, Seoul, and Puumala viruses which are prevalent mainly in Asia and Europe and are called as Old World Hantaviruses (Jiang et al. 2017). Nephropathia epidemica (NE), which is a mild type of HFRS characterised by acute kidney damage, and thrombocytopenia were first identified in Sweden (Krautkramer et al. 2013). Depending on the season, HFRS

outbreaks can vary, with most cases recorded in the winter to the early spring season in epidemic areas. Farmers are most commonly affected, especially in China (Zhang et al. 2014; Jiang et al. 2017). Lately, endemic zones have expanded beyond rural areas forming new foci of infection. Factors thought to be associated with such expansion of endemic trend are due to climate change, urbanisation, human migration, and rapid economic development (Zuo et al. 2011). HCPS, a previously unrecognised syndrome, was described first in 1993 in the USA. HCPS outbreaks are chiefly caused by Andes and Sin Nombre viruses, which are widespread in North and South America and are called as New World Hantaviruses. In contrast to HFRS, most cases of HCPS occur during early summer and late spring months (Jiang et al. 2017). The expanding geographical distribution of Hantaviruses and the variation between the “New World” and “Old World” viruses are slowly becoming less apparent.

15.10 *Togaviridae* Zoonotic Infections

Alphaviruses of *Togaviridae* family enclose zoonotic viruses which are generally transmitted by mosquitoes (Table 15.2). Alphaviruses are usually referred to as New World’ and “Old World” viruses with “New World” viruses (which include Venezuelan, Eastern and Western Equine Encephalitis viruses) principally related with the serious encephalitic disease in the Americas. Old World viruses are associated with rheumatic or arthritogenic diseases in humans.

The arthritogenic alphaviruses encompass Chikungunya virus (CHIKV), the Sindbis group of viruses, Ross River virus (RRV), Barmah Forest virus (BFV), and Mayaro virus. These viruses are responsible for endemic diseases and rarely, large epidemics; for example, chikungunya epidemic in 2004–2011 resulted in 1.4–6.5 million morbidities in almost 40 countries (Suhrbier et al. 2012). Symptoms in adults due to alphaviruses infection are always associated with rheumatic ailments, principally polyarthritis and/or polyarthralgia, which can be debilitating and chronic. CHIKV was primarily isolated in Tanzania in 1952. Following the isolation, regular epidemics have been witnessed in Africa and Asia, with former outbreaks baffled with dengue fever. The largest Chikungunya epidemic was linked with the emergence of viruses that were transmitted by *Aedes albopictus* (Ng and Hapuarachchi 2010; Burt et al. 2012; Suhrbier et al. 2012). RRV and BFV were isolated in 1959 and 1974 from mosquitoes trapped in the Ross River in Queensland and Barmah Forest, Victoria, in Australia, respectively. These viruses are enzootic and endemic in Australia with RRV also identified in Papua New Guinea. Most cases occur in Northern Australia from December to February, when vector mosquitoes are at their peak. BFV and RRV infections are notifiable to Australian public health authorities (Harley et al. 2001; Jacups et al. 2008; Suhrbier et al. 2012). Sindbis virus was isolated for the first time in 1952 from mosquitoes in Egypt. Sindbis viral diseases are endemic with and restricted to Northern Europe with cases in early autumn or late summer. Sporadic cases are also reported in South Africa,

Table 15.2 List of *Togaviridae* and *Filoviridae* zoonotic diseases involving wildlife species

S. no	Viral zoonotic diseases	Virus aetiology genus, family (virus)	Wildlife reservoirs/amplifiers/natural hosts/spill-over hosts	Major transmission route to humans	Geographical distribution	Human disease	BSL level	References
1	Chikungunya virus disease	<i>Alphavirus, Togaviridae</i>	Sylvatic natural hosts—non-human primates, bats and monkeys	Mosquito bite (<i>Aedes</i>)	South and South-east Asia, Africa, Indian Ocean Islands	Dengue-like, acute febrile illness with acute and persistent polyarthralgia	3	Burt et al. (2012)
2	Ross river fever	<i>Alphavirus, Togaviridae</i>	Reservoir—agile wallaby, and dusky rat	Mosquito bite (<i>Culex annulirostris, Aedes vigilax</i>)	Australian and South and Western Pacific regions	Febrile arthritogenic illness	2	Jacups et al. (2008)
3	Barmah forest virus	<i>Alphavirus, Togaviridae</i>	Reservoir—brush-tail possums	Mosquito bite (<i>Culex, Aedes</i>)	Australia	Febrile arthritogenic illness	2	Jacups et al. (2008)
4	Sindbis fever	<i>Alphavirus, Togaviridae</i>	Amplifying hosts—migratory and water birds	Mosquito bite (<i>Culex, Culiseta, Aedes</i>)	northern Europe, South Africa, Australia	Febrile arthritogenic illness	2	Adouchief et al. (2016)
5	Mayaro fever	<i>Alphavirus, Togaviridae</i>	Wild vertebrate hosts—non-human primates, birds, marsupials and rodents	Vector transmitted— <i>Haemagogus, Culex</i> spp., <i>Aedes</i> spp., <i>Psorophora</i> spp., <i>Coquillettidia</i> spp.	South and Central America	Febrile arthritogenic illness	3	de Oliveira Mota et al. (2015)
6	Eastern equine encephalitis	<i>Alphavirus, Togaviridae</i>	Enzootic cycle—wild birds and mosquitoes	Mosquito bite (<i>Culiseta melanura</i>)	North and South America	Febrile disease with encephalomyelitis	3	Armstrong and Andreadis (2013)
7	Western equine encephalitis	<i>Alphavirus, Togaviridae</i>	Enzootic cycle—passerine birds (reservoir) and mosquitoes	Mosquito bite (<i>Ochlerotatus melanion, Aedes dorsalis, and Ae. campestris</i>)	North America	Mild febrile disease with encephalomyelitis in few cases	3	Arechiga Ceballos and Aguilar Setien (2015)

8	Venezuelan equine encephalitis	<i>Alphavirus, Togaviridae</i>	Principal reservoir hosts epizootic cycles—bats, rodents, and some birds Enzootic cycles—sympatric rodents in the genera <i>Sigmodon</i> , <i>Oryzomys</i> , <i>Zygodontomys</i> , <i>Heteromys</i> , <i>Peromyscus</i> , and <i>Proechimys</i>	Mosquito bite	Central and South America	Febrile illness; Children-fatal encephalitis and permanent neurological sequelae; pregnant women-birth defects, spontaneous abortions and stillbirths	3	Weaver et al. (2004)
9	Ebola disease	<i>Ebola virus, Filoviridae</i>	Reservoir—fruit bats	Person-to-person transmission/direct contact with non-human primates	Sub-Saharan Africa and Reston (REBOV), identified in the Philippines	Hemorrhagic fever and multiple organ failure	4	MacNeil and Rollin (2012)
10	Marburg virus disease	<i>Marburg virus, Filoviridae</i>	Reservoir—fruit bats	Person-to-person transmission/direct contact with non-human primates	sub-Saharan Africa	Hemorrhagic fever and multiple organ failure	4	MacNeil and Rollin (2012)

Australia, and China (Laine et al. 2004; Adouchief et al. 2016). Mayaro virus, initially isolated in 1954 is enzootic in the northern part of South America. This virus causes recurrent smaller outbreaks and sporadic cases in humans. Usually, human cases are associated with forest visits or human dwelling near the forest (de Oliveira Mota et al. 2015). All these rheumatic alphaviruses are maintained in the wilderness in the transmission cycle between mosquitoes and vertebrate hosts: non-human primates for CHIKV (Burt et al. 2012), macropods (wallabies and kangaroo) for BFV and RRV (Jacups et al. 2008), migratory and wild birds for Sindbis virus (Adouchief et al. 2016), birds, marsupials, rodents and primates for Mayaro virus (de Oliveira Mota et al. 2015). On various occasions, these reservoir hosts infect human via mosquito bite, however larger epidemics generally associated with consequent urban transmission cycles.

Alphaviral encephalomyelitis is caused by Western equine encephalomyelitis virus (WEEV), Eastern equine encephalomyelitis virus (EEEV), and Venezuelan equine encephalomyelitis virus (VEEV) (Table 15.1). All these equine alphaviruses cause a mosquito-transmitted infection that causes serious neurological disease and mortality in humans and horses in the Americas. Though related, these viruses are antigenically and genetically distinct. The first isolation of WEEV was from a horse brain in 1930 in California. WEEV is maintained in an enzootic cycle between vertebrate hosts–mosquito (*Culex tarsalis*)–passerine birds cycle. *Culex tarsalis* is associated with stream drainage and irrigated agriculture in the western United States. Bridging mosquito vectors, *Aedes dorsalis*, *Ochlerotatus melanimon*, and *Aedes campestris*, are implicated in the transmission of the virus to horses and humans in Utah, California, and New Mexico, respectively (Arechiga Ceballos and Aguilar Setien 2015). The first isolation of EEEV was from an infected horse brain in 1933 in New Jersey and Virginia. The EEEV is maintained in a primary transmission cycle between birds and mosquito vector, *Culex melanura*. Transmission to humans and horses is mediated by *Aedes* spp., *Culex* spp., and *Coquillettidia* spp. Transmission of virus usually occurs around the Gulf Coast of the USA and hardwood swamps in the Atlantic and the Great Lakes region (Armstrong and Andreadis 2013). During the 1930s, VEE was first identified as a disease of mules, donkeys, and horses in northern South America. In spite of wide vertebrate host range of VEE which includes humans, dogs, sheep, birds, bats, and rodents, major epidemics have not occurred in the nonexistence of equine cases. The principal reservoir hosts of VEEV are believed to be sylvatic rodent genera, *Heteromys*, *Oryzomys*, *Peromyscus*, *Sigmodon*, *Proechimys*, and *Zygodontomys* as they are regularly infected in nature, develop viraemia from moderate to high titre, and have high degrees of immunity (Weaver et al. 2004).

15.11 *Filoviridae* Zoonotic Infections

Under family *Filoviridae*, Ebola haemorrhagic fever and Marburg haemorrhagic fever are the two analogous diseases caused by two virus genera, Ebola virus (EBOV) and Marburg virus (MARV), respectively (Table 15.2). Despite the general rarity of their incidence, these diseases are well recognised due to the sensationalist accounts of its outbreaks. Nevertheless, EBOV and MARV are potentially pathogenic and have typically been connected with shattering outbreaks, with 25–90% case fatality rate range (Leroy et al. 2011; MacNeil and Rollin 2012). Besides, these viruses are recognised as potential bioweapons and as such are categorised as class A select agents. Present facts suggest fruit bats (*Pteropus* spp.) as the reservoir of both the viruses, and the dispersal appears to be restricted to sub-Saharan Africa (except Reston Ebola virus, spotted in the Philippines, and not documented to be related with human infection) (Taniguchi et al. 2011; MacNeil and Rollin 2012). Generally, zoonotic source of the exposure is not recognised always in outbreaks, but the introduction of these fatal viruses to have always been associated with hunting or processing bushmeat (EBOV) or persons entering mines and caves (MARV) (MacNeil and Rollin 2012). Outbreaks and clusters are principally the outcomes of person-to-person transmission. Three distinctive contact modes attribute for transmission of virus during outbreaks: (1) transmission between, close contacts, members of the family and caretakers of infected individuals; (2) direct contact with cadaver in preparation and funeral events; and (3) nosocomial transmission from infected persons to other patients or medical staff by reusing medical equipment or infringing barrier nursing (MacNeil and Rollin 2012; Cross et al. 2018).

15.12 *Flaviviridae* Zoonotic Infections

Zoonotic flaviviruses under family *Flaviviridae* are generally transmitted to humans by tick and mosquitoes. Despite being present in blood and body secretions during acute illness, flaviviruses do not get transmitted from person-to-person (contagious). Consequently, reservoirs of virus and abundance of vectors are prerequisites for epidemics. Zoonotic flaviviral diseases involving wildlife hosts can be grouped into mosquito- and tick-borne. Mosquito-borne flaviviral zoonotic diseases are Japanese encephalitis, St. Louis encephalitis Murray Valley encephalitis, Wesselsbron disease, West Nile fever, and Yellow fever. Tick-borne flaviviral zoonotic diseases are Kyasanur forest disease, Powassan encephalitis, Omsk haemorrhagic fever Tick-borne encephalitis, and Tyulenyi virus infection (Table 15.3).

Japanese encephalitis virus (JEV) causes neurological infection in humans all over Asia, affecting 70,000 people each year with nearly 10,000 fatalities. The JEV was first isolated in Japan at the beginning of the 1930s. JE is now endemic in eastern and southern Asia with more number of cases from China, Japan, India, Pakistan, and the Philippines (Erlanger et al. 2009). No cases have been reported from Europe,

Table 15.3 List of *Flaviviridae* zoonotic diseases involving wildlife species

S. no	Viral zoonotic diseases	Virus aetiology genus, family (virus)	Wildlife reservoirs/amplifiers/natural hosts/spill-over hosts	Major transmission route to humans	Geographical distribution	Human disease	BSL level	References
1	Japanese encephalitis	<i>Flavivirus, Flaviviridae</i>	Reservoirs—cattle egret, pond herons; other wild host species—flying foxes, snakes and frogs	Mosquito bite (<i>Cx. tritaeniorhynchus</i>)	Asia, Western Pacific countries, and Northern Australia	Febrile encephalitis and permanent neurologic or psychiatric sequelae	3	Mansfield et al. (2017)
2	Kyasanur forest disease	<i>Flavivirus, Flaviviridae</i>	Natural hosts—Blanford's rat, striped forest squirrel house shrew, ground birds; Amplifiers—black-faced langur and red-faced bonnet monkey	Nymph bite (<i>Haemaphysalis</i> spp.)	India	Haemorrhagic febrile illness and encephalitis	3	Mourya and Yadav (2016)
3	Murray valley encephalitis	<i>Flavivirus, Flaviviridae</i>	Reservoirs—water birds, such as herons and egrets	Mosquito bite (<i>Culex</i> species)	Endemic to Australia and New Guinea	Febrile encephalitis and neurological deficits	3	Floridis et al. (2018)
4	Omsk hemorrhagic fever	<i>Flavivirus, Flaviviridae</i>	Natural host—narrow-headed vole (<i>Microtus gregalis</i>); water vole (<i>Arvicola terrestris</i>) and musk rats	Tick bite (<i>Dermacentor</i>)	Western Siberia	Hemorrhagic fever	3	Dobler (2010)
5	Powassan encephalitis	<i>Flavivirus, Flaviviridae</i>	Reservoirs—woodchucks (<i>Marmota monax</i>) and white-footed mice (<i>Peromyscus leucopus</i>)	Tick bite (<i>Ixodes, Dermacentor</i>)	North America, Russian Far East	Febrile illness with neurological symptoms	3	Birge and Sonnesyn (2012)

6	Tick-borne encephalitis	<i>Flavivirus, Flaviviridae</i>	Primary reservoirs and hosts—small rodents	Hard tick bites (<i>Ixodes</i>); consuming infected milk or milk products	Europe and Asia	Febrile illness with CNS involvement (meningitis, meningoencephalitis)	3	Bogovic and Strle (2015)
7	St. Louis encephalitis	<i>Flavivirus, Flaviviridae</i>	Amplifying hosts—passerine and columbiform avian species	Mosquito bite (<i>Culex</i>)	USA, Panama, and South America	Mild febrile to severe neuroinvasive disease	3	Diaz et al. (2018)
8	Wesselsbron disease	<i>Flavivirus, Flaviviridae</i>	Cape short-eared gerbil and black rats (maintenance hosts?)	Mosquito bite (<i>Aedes</i>)	Africa	Acute, influenza-like illness with arthralgia and myalgia	3	Diagne et al. (2017)
9	West Nile fever	<i>Flavivirus, Flaviviridae</i>	Reservoir hosts—House sparrow (<i>Passer domesticus</i>), common grackles (<i>Quiscalus quiscula</i>)	Mosquito bite (<i>Culex</i> species)	Endemic in Africa, Europe, the Middle East, West and Central Asia, and most recently, North America and is spreading into Central and South America	Mild febrile to severe illness affecting the CNS (encephalitis or meningitis)	3	Chancey et al. (2015)
10	Yellow fever	<i>Flavivirus, Flaviviridae</i>	Reservoir hosts—Non-human primates	Mosquito bite (<i>Aedes</i> species)	Africa and South America	Acute haemorrhagic fever and some patients develop jaundice	3	Gardner and Ryman (2010)
11	Tyuleny virus infection	<i>Flavivirus, Flaviviridae</i>	Natural cycle between ticks and sea birds	Tick (<i>Ixodes</i>) bite or direct contact with birds	Far Eastern Russia, France, USA	Febrile illness	2	Dobler (2010)

Africa, or the Americas. The virus is maintained in an enzootic transmission cycle between mosquitoes and wild birds, particularly large ardeid water birds such as cattle egret and pond herons. Domestic and wild pigs act as amplifier hosts. Irrigated rice fields offer a breeding ground for vector mosquitoes and also invite migratory wading birds facilitating virus maintenance in the sylvan cycle (van den Hurk et al. 2003; Miller et al. 2012; Jeffries and Walker 2015). *Culex* species of mosquitoes especially *Culex tritaeniorhynchus*, which is both ornithophilic and mammalophilic mosquito, helps in virus circulation between avian species and also acts as a bridge vector to infect livestock and humans (Guo et al. 2014; Mansfield et al. 2017). Other potential hosts in wildlife species are flying foxes, ducks, frogs, and snakes. However, these are taken as dead-end hosts as they seldom develop adequate viraemia to infect vector mosquitoes (Miller et al. 2012). JEV has not spread to Africa and Europe in spite of the presence of *Cx. tritaeniorhynchus* in these regions. This may be due to the absence of competent vectors in Europe or the non-migration of birds from tropical Asia to Africa or restricted movement of livestock from Asia to Europe (Mansfield et al. 2017).

Murray Valley encephalitis (MVE) is a mosquito-borne viral zoonotic disease endemic to Australia and New Guinea. It affects mostly children living in remote and rural areas and is potentially fatal. An enzootic cycle between *Culex annulirostris* mosquitoes and water birds maintains the virus. Apart from the primary vector, *C. annulirostris*, *Aedes normanensis* also supports the MVEV transmission to humans (Floridis et al. 2018).

St. Louis encephalitis (SLE) is again a mosquito-borne zoonotic viral disease endemic to the USA and some cases are occurring in a wide area ranging from Argentina to Canada. The virus is transmitted by several mosquito vectors in the genus *Culex*. Columbiform and passerine birds are the amplifying hosts. Most SLE cases are present with a flu-like illness and very few signs of progress to invasive encephalitis is unusual and is more common in older people (Ortiz-Martínez et al. 2017; Diaz et al. 2018).

Wesselsbron (WSL) disease is a zoonotic mosquito-borne flavivirus infection that causes teratogenic defects and abortions in sheep and cattle in Africa. These domestic animals ought to play a role in the viral life cycle, but some shreds of evidence suggest that wild animals may also be involved in virus maintenance in nature. This assumption is only supported by the isolation of the WSL virus from a black rat and Cape short-eared gerbil in Africa. The virus can also infect humans and produce dengue-like syndrome (Diagne et al. 2017).

West Nile virus (WNV) is a mosquito-transmitted virus which causes flu-like illness to fatal neuroinvasive diseases in humans. It was first described in Uganda in 1937 from a febrile case. WNV has caused sporadic outbreaks in Israel, India, Egypt, France, and South Africa. An enzootic cycle maintains the virus between birds and mosquitoes. Birds are reservoir hosts for the WNV as they can mount high viraemia to infect mosquitoes. American crows and blue jays become commonly ill or die; however, birds like common grackles and house sparrows build up high viraemia with lesser death rates. House finches and American robins are two important

amplifiers of WNV in the USA. Additionally, 30 other vertebrate hosts such as mammals, reptiles, and amphibians are susceptible to WNV infection.

Nevertheless, only a few vertebrates including brown lemurs, eastern grey squirrels, lake frogs, hamsters, eastern chipmunks, fox squirrels, and eastern cottontail rabbits have been described to mount viraemia expected to help vector transmission. Generally, humans and horses may endure serious infection or death, but they are considered only as incidental hosts as they do not mount sufficient level of viraemia to infect vector mosquitoes. Although mosquito bite transmission is common in humans, transmission by organ transplantation, blood transfusion, transplacental route, and via breast milk is also possible. *Culex* spp. of mosquitoes that feed on both birds and mammals are considered as bridge vectors as they pass on the virus from infected birds (reservoirs) to mammalian (incidental) hosts (Van der Meulen et al. 2005; Kilpatrick et al. 2006; Chancey et al. 2015).

Yellow fever (YF) is a mosquito-transmitted flaviviral disease endemic to tropical areas of Africa and the Americas. From Africa, it was introduced into the Americas and Europe as a consequence of the slave trade. YF virus primarily affects humans and non-human primates via mosquito bite and causes devastating epidemics of grave haemorrhagic disease. The transmission to humans occurs in sylvatic (humans who enter forests), intermediate (epidemics in rural villages), and urban cycles (urban mosquito species, *Aedes aegypti*). In recent decades, the intermediate transmission cycle causing small scale epidemics in rural villages is most common in Africa, wherein the infected semi-domestic mosquitoes species feed on both humans and monkeys (Gardner and Ryman 2010). As per the World Health Organization (WHO), an outbreak involving *Ae. aegypti* is referred to as urban YF, while outbreaks associated with other species of mosquitoes are categorised as jungle YF (Bres 1986). YF is differentiated from other viral haemorrhagic fevers by the distinctive severity of liver injury and jaundice (Monath 2008; Gardner and Ryman 2010).

Kyasanur Forest disease (KFD) is a zoonotic tick-transmitted viral disease endemic in southern India. KFD virus circulates between small mammals like shrews and rodents; ground birds and a range of tick species. The natural cycle of the virus involves two monkey species, red-faced bonnet monkey, and black-faced langur and a variety of tick species, predominantly ticks of *Haemaphysalis* spp. After getting infected, monkeys amplify and broadcast the virus to a large number of ticks feed on them. In humans and monkeys, the virus causes serious haemorrhagic disease and death (Mourya and Yadav 2016).

Omsk haemorrhagic fever (OHF) is a tick-transmitted zoonotic flaviviral disease endemic in Western Siberia. The virus is naturally maintained by two independent transmission cycles (grassland cycle and wetland cycle) which are connected by migration. In the grassland cycle, the vole *Microtus gregalis* is the main maintenance host developing high viraemia levels, which infects *Dermacentor reticulatus* ticks, which is the important vector of OHF virus. In the wetland cycle, *Ixodes apronophorus* appears to play a vital role as a vector, and the other water vole, *Arvicola terrestris* which migrates from grassland to wetland develops high viraemia for a longer period and seems to be an important maintenance host. In this cycle,

muskrats seem to be another key vertebrate host. Apart from humans getting OHF infection from mosquito bites, direct contact with the infected muskrat's blood and bites from infected animals is other possible routes of transmission (Dobler 2010).

Tick-borne encephalitis (TBE) is another important flaviviral infection prevalent in Europe and some regions of Asia (northern China, Japan, Siberia, and Far Eastern Russia). Three subtypes of TBE virus, namely Far-Eastern, Siberian, and European, cause the disease. Adults are often infected than children. Small rodents are the primary reservoirs which maintain the virus in nature and humans are only accidental hosts. *Apodemus flavicollis* of *Muridae* family may play a key role in the European subtype viruses transmission. TBE virus is transmitted to human mostly by tick bites. The principal mosquito vector species in Europe and Japan are *Ixodes ricinus* and *Ixodes ovatus*, respectively. While in far-east Asia, Russia, and parts of Eastern Europe, it is *Ixodes persulcatus* (Dobler 2010; Bogovic and Strle 2015). In humans, around 1% of all TBE infections are most likely acquired by consuming contaminated unpasteurised milk and milk products from farm animals, particularly goats (Mansfield et al. 2009).

Powassan virus infection is a rare tick-transmitted flaviviral infection in North America and Russia. The common reservoirs are small and medium-sized mammals like white-footed mice, woodchucks and tick species like *Ixodes* and *Dermacentor* act as vectors (Birge and Sonnesyn 2012). Tyuleniy virus infection is other rare flaviviral zoonoses at the island of Tyuleniy in Far Eastern Russia. After its first isolation from sea birds in 1969, it was also isolated from the Atlantic coast of France, Norway, and the USA. Tyuleniy virus appears to be transmitted in an enzootic cycle involving seabirds and ticks. So far three human cases have been reported with fever, pharyngitis, nausea, joint pain, and petechial exanthema. All the three cases reported were ornithologists who had direct contact with sea birds and their ticks (Hubalek and Halouzka 1996; Dobler 2010).

15.13 *Reoviridae* Zoonotic Infections

Under family *Reoviridae*, Orungo fever and Colorado tick fever are the two important viral zoonotic diseases involving wildlife species caused by orbivirus and colitivirus, respectively (Table 15.4). Orungo virus was primarily isolated in 1959 in Uganda from the blood of a human infected with the virus. Orungo virus with its four distinct serotypes is transmitted by *Aedes*, *Anopheles*, and *Culex* mosquitoes. This virus is extensively dispersed in tropical Africa. It has been isolated from humans, cattle, camels, sheep, goats, and monkeys. Antibodies against the Orungo virus have been detected in primates, cattle, and sheep. Despite high prevalence in human, only a few clinical cases and three deaths reported in Uganda. High co-infection with yellow fever has been described, revealing their analogous geographical distribution and vector mosquito (*Aedes*) species (Attoui and Jaafar 2015).

Colorado tick fever or mountain fever is a tick-borne disease prevalent in North America, particularly in the Rocky Mountain region. This virus maintains in an

Table 15.4 List of *Reoviridae*, *Rhabdoviridae*, *Paramyxoviridae*, *Orthomyxoviridae*, *Coronaviridae*, *Hepeviridae*, and *Poxviridae* zoonotic diseases involving wildlife species

S. no	Viral zoonotic diseases	Virus aetiology genus, family (virus)	Wildlife reservoirs/amplifiers/natural hosts/spill-over hosts	Major transmission route to humans	Geographical distribution	Human disease	BSL level	References
1	Orungo fever	<i>Orbivirus</i> , <i>Reoviridae</i>	Wild host—monkeys	Culicine mosquitoes	Uganda	Febrile illness	3	Attoui and Jaafar (2015)
2	Colorado tick fever	<i>Coltivirus</i> , <i>Reoviridae</i>	The major naturally infected host species include the golden-mantled ground squirrel, least chipmunk, Columbian ground squirrel, yellow pine chipmunk, porcupine, deer mouse, and busy tailed wood rat	Tick-to-human transmission (<i>Dermacentor andersoni</i>)	North America	Febrile illness with neurologic symptoms in children	2	Romero and Simonsen (2008)
3	Rabies	<i>Lyssavirus</i> , <i>Rhabdoviridae</i>	Wild reservoirs/vectors—vampire bats, raccoons, skunks, wild cats, foxes jackal, wolf, badger and mongoose	Bite/wounds or cuts/inhalation/organ transplant	Except Antarctica and Australia, distribution of the disease cover all continents	Two forms of human disease—furious, or encephalitic and paralytic or dumb	3	Singh et al. (2017)
4	Australian bat lyssavirus infection	<i>Lyssavirus</i> , <i>Rhabdoviridae</i>	Bats are the natural reservoir	Direct contact/bite of infected bats	Australia	Serious illness which results in paralysis and convulsions	3	Singh et al. (2017)

(continued)

Table 15.4 (continued)

S. no	Viral zoonotic diseases	Virus aetiology genus, family (virus)	Wildlife reservoirs/ amplifiers/natural hosts/spill-over hosts	Major transmission route to humans	Geographical distribution	Human disease	BSL level	References
5	Nipah virus infection	<i>Henipavirus</i> , <i>Paramyxoviridae</i>	Fruit bats (flying foxes) are the natural reservoir	Direct contact (bats/pigs), food-borne, person-to-person	Southeast Asia-India, Bangladesh, Malaysia, Singapore, Philippines	Asymptomatic to acute respiratory infection, and fatal encephalitis	4	Kulkarni et al. (2013)
6	Hendra virus infection	<i>Henipavirus</i> , <i>Paramyxoviridae</i>	Fruit bats (flying foxes) are the natural reservoir	Direct contact with infected horses	Australia	Mild influenza-like illness to fatal respiratory or neurological disease	4	Field (2016)
7	Menangle virus infection	<i>Rubulavirus</i> , <i>Paramyxoviridae</i>	Fruit bats (flying foxes) are the natural reservoir	Direct contact with infected pigs	Australia	Severe influenza-like illnesses	3	Barr et al. (2012)
8	Influenza A virus infections	<i>Influenza virus A</i> , <i>Orthomyxoviridae</i> (H5N1, H7N9, H9N2, H1N1, H3N2, etc.)	AI-Natural reservoirs—wild and aquatic birds SI (H1N1)—wild boars, badgers, ferrets, skunks, cheetahs, giant anteater are some spill-over hosts	Direct contact with infected birds/animals; person-to-person	worldwide	Respiratory tract infections and multiple organ failure	3	Horman et al. (2018), Schrenzel et al. (2011), and Delogu et al. (2019)
9	SARS	<i>Betacoronavirus</i> , <i>Coronaviridae</i> (SARS coronavirus-SARS-CoV)	Reservoir—Bats Other incidental wild hosts—palm civets, raccoon dog	Human-to-human transmission	2003 outbreak-China, Hong Kong, Vietnam, Canada and several other countries	Severe acute respiratory syndrome	3	de Wit et al. (2016)

10	Hepatitis E virus infection	<i>Orthohepevirus, Hepeviridae</i> (Hepatitis E virus HEV3, HEV4)	Wild hosts—wild boar and deer	Consumption of contaminated meat from deer and wild boar	worldwide	3	Doceul et al. (2016)
11	Macacine herpesvirus 1 (B virus or Herpesvirus simiae, or Herpes virus B) Infection	<i>Simplexvirus, Herpesviridae</i>	Enzootic natural hosts— <i>Macaca</i> spp.— <i>M. mulatta</i> , <i>M. fascicularis</i>	Transcutaneously (via bites) or per mucosally	Asia, USA	4	Lee et al. (2015)
12	Cowpox	<i>Orthopoxvirus, Poxviridae</i>	Wild rodents are reservoir	Direct contact	UK, Europe, adjacent USSR	2	Essbauer et al. (2010)
13	Monkeypox	<i>Orthopoxvirus, Poxviridae</i>	Reservoirs—rodents, including squirrels and giant pouched rats (monkeys are considered disease hosts)	Indirect (touch, bite, or scratch) or direct contact with live or dead animals and person-to-person	Endemic in Central and West Africa, cases reported in USA, UK, and Israel	3	Peterson et al. (2019)
14	Tanapox	<i>Yatapox, Poxviridae</i>	Monkeys (natural host)	Direct contact, arthropod vectors	Endemic in East Africa, few case reports in USA, Europe	2	Dhar et al. (2004)
15	Contagious ecthyma/orf	<i>Parapoxvirus, Poxviridae</i>	Wild small ruminants (spill-over host)	Direct contact, fomites, meat	Worldwide	2	Kuhl et al. (2003)

Self-limiting acute hepatitis and fulminant hepatic failure in patients with chronic liver disease

Influenza-like illness and encephalitis

Papular-vesicular-pustular-ulcerative skin lesions and scar formation

Papular-vesicular-pustular (similar to that of smallpox) skin lesions

Febrile illness with nodules on extremities

Solitary or multiple papules that advance through a sequence of stages, terminates in complete resolution

enzootic tick–mammalian host–tick cycle involving *Dermacentor andersoni* ticks (larval and nymphal stages). The major reservoir and vector for the disease is the wood tick, *D. andersoni*. The main naturally infected vertebrate hosts include the Columbian ground squirrel, golden-mantled ground squirrel, yellow pine chipmunk, least chipmunk, porcupine, deer mouse, and bushy-tailed woodrat. Virus transmission to humans through tick bite coincides with the activity of *D. andersoni* ticks, which is generally from late March to late October (Romero and Simonsen 2008).

15.14 *Rhabdoviridae* Zoonotic Infections

Rabies and Australian lyssavirus (ALV) infection are the two important viral zoonotic diseases involving wildlife species under genus *Lyssavirus* of *Rhabdoviridae* family (Table 15.4). Rabies is a potentially zoonotic and fatal disease caused by the rabies virus. All the warm-blooded animals, including human, are affected by the virus. Rabies is distributed throughout the world and endemic in several countries except Australia and Antarctica. Every year, over 60,000 people expire due to rabies, and roughly 15 million people get the vaccine as post-exposure prophylaxis every year. Bite of infected animals and saliva of rabid hosts are mostly responsible for disease transmission. Apart from domestic dogs, wildlife like foxes, raccoons, skunks, and bats are chief reservoirs for rabies, from the enormous amount of rabies cases reported every year (Davis et al. 2013; Ellison et al. 2013; Streicker et al. 2013; Kuzmina et al. 2013). Rabies virus circulates in an urban and sylvatic cycle involving dogs, cats, and wild animals like a racoon, skunk, jackal, fox, badger, mongoose, bats, etc., as reservoirs/vectors, respectively (Condori-Condori et al. 2013; Blackwood et al. 2013; Escobar et al. 2013).

Nevertheless, both cycles may overlie in several geographical situations. Rabies was eradicated officially in the UK in 1920. But in 2002, a bat conservationist died after contracting a rabies virus (European bat lyssavirus type 2) from a bat (Fooks 2007). Presently rabies virus is absent in terrestrial animals in Australia. However Australian bat lyssavirus is present in bats. This virus is transmitted to humans and animals from bats. It was first identified in Queensland, in 1996 and so far, only three human cases have been accounted for due to bite or scratch by bats (Francis et al. 2014; Singh et al. 2017). Additionally, some rabies-related lyssaviruses have been described in Eurasia from insectivorous bats: Irkut, Aravan, Khujand, Bokeloh bat lyssavirus, West Caucasian bat viruses, and Ikoma lyssavirus (Singh et al. 2017). Till now, five human deaths have been connected to rabies-related viruses (Singh et al. 2017).

15.15 *Paramyxoviridae* Zoonotic Infections

Nipah, Hendra (genus *Henipavirus*), and Menangle virus (genus *Rubulavirus*) infections are the three important viral zoonotic diseases involving wildlife species under the *Paramyxoviridae* family (Table 15.4). All the three viruses are transmitted

through bats. The emergence of these zoonotic viruses is alleged to be due to ecological modifications like deforestation, urbanisation, and drought that have compelled the bat populations to shift their usual habitats to agricultural areas subsequently resulting in animal and human diseases (Allocati et al. 2016; Kulkarni et al. 2013).

Nipah virus first emerged in Malaysia in 1998. It has caused an outbreak of encephalitis and respiratory illness in pigs. Nipah virus transmission from pigs to human has resulted because of direct contact with infected animals. The human-to-human transmission is also reported. In outbreaks of Bangladesh and India, an intermediary animal was not recognised, suggesting direct bat-to-human and human-to-human spread. *Pteropus* bats (*P. hypomelanus* and *P. vampyrus*) are believed to be the natural hosts. Pigs play the role of amplifying host. In human outbreaks of Malaysia and Singapore, it has been proved that infected swine was the source (Parashar et al. 2000; Kulkarni et al. 2013).

Hendra virus first emerged in 1994 in Australia has caused fatal respiratory infection in two humans and 20 horses and further several outbreaks. Pteropid bats are the reservoir of the Hendra virus. Horses infected by the secretions and excretions of infected bats are the intermediate hosts that transmit the infection to humans, who come in close contact with them. The human-to-human transmission has not been documented until now (Allocati et al. 2016). The majority of human cases have been veterinary assistants or veterinarians, underlining the prominent risk profile of this cohort (Field 2016).

Menangle virus is another zoonotic paramyxovirus able to cause disease in pigs and humans. This virus was first isolated in Australia in 1997 from stillborn piglets at a commercial pig farm. This virus was shown to infect people; 2 workers in piggery developed a serious influenza-like illness and found to have neutralising antibodies to Menangle virus. For the outbreak, bats were identified as a source, as *Pteropus poliocephalus* and *Pteropus scapulatus* bats were noticed to be roosting close to the piggery implicated in an outbreak (Barr et al. 2012).

15.16 *Orthomyxoviridae* Zoonotic Infections

Influenza A viruses under the *Orthomyxoviridae* family (Table 15.4) time and again have posed a significant threat to public health, both through pandemic outbreaks and seasonal infections. Avian influenza (AI) viruses, especially highly pathogenic (H5N1, H7N9) variants have emerged as a major zoonosis, and they circulate naturally in wild bird populations as well as in waterfowl and ducks and be able to spill over to domestic poultry birds like chickens. Aquatic birds play the role of natural reservoirs for all influenza A subtypes except some novel strains being isolated in bats (Horman et al. 2018). Many AI virus antigenic subtypes have been recovered from swine, demonstrating an ideal “mixing pot” of influenza A viruses possibly pandemic for humans. There is serological evidence of AI in one duck

hunter and two wildlife professionals with considerable exposure to wild water bird and game bird (Gill et al. 2006; Horman et al. 2018).

Swine influenza viruses (H1N1, H3N2), especially H1N1, which created a global pandemic, seem to have high infectivity for a wide array of domestic and wild animal species. The domestic animals in which the virus was detected are swine, dogs, turkeys, cats, and domestic ferrets, whereas wildlife species include skunks, cheetahs, American badger, black-footed ferret binturong, giant anteaters, and wild boar (Schrenzel et al. 2011; Delogu et al. 2019). In a few cases, animal to animal spread may have occurred, lifting apprehension about the possible development of new wild reservoirs (Schrenzel et al. 2011). The omnipresence of H1N1 pandemic strain and its capability to infect a broad range of hosts is a concern for the health of wildlife and for the likelihood of creating extra reservoirs that could change the evolution of subtype H1N1 viruses by causing diverse selection pressures and creating new ways of producing novel reassortant strains (Schrenzel et al. 2011).

15.17 *Coronaviridae* Zoonotic Infections

Coronaviruses (CoV) preceding SARS outbreak was only acknowledged to be the second reason for common cold infection next to rhinoviruses. Of late, 2 very important zoonotic-CoV were recognised: Severe Acute Respiratory Syndrome Coronavirus (SARS-CoV) and the Middle East Respiratory Syndrome Coronavirus (MERS-CoV) (Table 15.4). SARS-CoV was first spotted in February 2003 in China, and soon after 4 months, 48,000 cases had been reported with about 800 fatalities in 27 countries around the world. This virus has a broad host range, and it is linked to the bushmeat industry. Bats are the principal hosts that transmit the virus to intermediate amplifier hosts such as raccoon dogs and mask palm civets that then could transmit to humans (Allocati et al. 2016). These amplifier animals are only incidental hosts as in wild or breeding facilities, no circulation of SARS-CoV-like viruses has been seen in them. Rather, bats are the natural reservoir of a broad range of coronaviruses, like SARS-CoV-like and MERS-CoV-like viruses (de Wit et al. 2016). In large scale epidemics, person-to-person transmission is considered as the major route of transmission. MERS-CoV was first recognised in 2012 in Saudi Arabia and then distributed to a few other countries causing many deaths. MERS-CoV is phylogenetically associated with SARS-CoV, and it also shares the origination that is from bats. Clinical characteristics of MERS-CoV are also similar to SARS-CoV, while this virus has also been related to some extrapulmonary manifestations, like renal complications. Experimental studies in bats prove that bats are the reservoir for MERS-CoV, as the virus replicated without manifesting any overt clinical signs (Munster et al. 2016). Animal-to human transmission is acknowledged to be important in MERS outbreak as recent studies have pointed out that dromedary camels may work as a potential source of MERS-CoV to humans (Allocati et al. 2016; de Wit et al. 2016).

15.18 *Hepeviridae* Zoonotic Infections

Under *Hepeviridae*, Hepatitis E virus (HEV) infection caused by the Hepatitis E virus is an important zoonotic viral disease from the wildlife perspective (Table 15.4). Globally, HEV is the foremost cause of hepatitis that is enterically transmitted. HEV-1 and HEV-2 genotypes infect only humans and responsible for large water transmitted epidemics. HEV-3 and HEV-4 infect both human beings and animals and are the major cause of hepatitis E cases in industrialised countries. The natural host of genotypes, HEV-3, and -4 is swine. Recently, HEV transmission from wild boar to other wild boar and domestic pigs by direct contact between animals was established (Doceul et al. 2016).

Additionally, the zoonotic spread of HEV-3 and -4 to human from wild boar, deer, and domestic pigs and human by eating contaminated meat has been confirmed (Tei et al. 2003; Doceul et al. 2016). Several reports in France, Japan, Australia, Germany, and Spain have also correlated sporadic hepatitis E cases or outbreaks with the consumption of pork or wild boar meat and offals. In France, a nationwide survey showed that consumption of pork, pork liver sausages, offal, and game meat was an important contributor for the prevalence of anti-HEV antibodies (Doceul et al. 2016).

15.19 *Herpesviridae* Zoonotic Infections

Macacine herpesvirus 1 (MaHV1 aka B virus) under the genus *Simplexvirus*, of *Herpesviridae* (Table 15.4) is a zoonotic agent enzootic among macaque (*Macaca* spp.) all over Asia. This virus is related to herpes simplex virus (HSV 1 and 2) of humans and other herpes viruses infecting non-human primates like baboons. Macaques can shed the virus without manifesting any overt clinical symptoms and also manifest vesicular lesions on the buccal cavity and genital areas. Human transmission can occur permucosally (exposure to infected macaque secretions and excretions) and transcutaneously (via bites). Among human cases, ≈40 laboratory workers have reported MaHV1 encephalitis following direct contact with the long-tailed macaques and the rhesus macaques or their infected tissues during the research (Cohen et al. 2002; Lee et al. 2015).

15.20 *Poxviridae* Zoonotic Infections

The genus *Orthopox* contains two zoonotic virus species with the involvement of wildlife species: cowpox and Monkeypox. The other two important genera that are important viral zoonotic pathogens from wildlife perspective are *Parapox* and *Yatapox*, causing contagious ecthyma/orf and Tanapox, respectively (Table 15.4).

Earlier cowpox virus was generally spotted in milking cows with the rare zoonotic transmission to milkers. Today, cowpox is renowned for infecting a wide array of hosts, including cats, zoo animals, and humans. More than 400 cases of cowpox infections have been reported in domestic cats. Human cowpox cases are mainly due to direct contact with infected cats, cows, on rare occasions with rats or zoo and circus animals (Essbauer et al. 2010). Incidental evidence of rodents being a source of infection to humans has been described in two suspected cases and one proven wild rat to women transmission (Wolfs et al. 2002).

Monkeypox was identified first in 1958 among captive monkeys imported to Denmark from Africa for research purposes. As the animal reservoir of the monkeypox is rodents, including giant pouched rats and squirrels; the given name seems to be inappropriate. From its discovery, this disease has been endemic to Central and West Africa with, sporadic and intermittent cases reported among humans transmitted from local wildlife. This virus has been identified in a range of animal species such as rats, striped mice, squirrels (rope and tree), dormice, and monkeys. Direct and indirect contact with infected live and dead animals is believed to be the driver of human cases. Monkeypox had gained international attention in 2003 when the first human cases outside Africa were reported in the USA. Several people developed a rash, fever, and respiratory symptoms, and source of exposure was investigated as pet prairie dogs (*Cynomys* sp.) (Peterson et al. 2019).

Orf, or contagious ecthyma, is a rare zoonotic disease commonly transmitted from infected sheep and goats. Humans get infection either by direct contact with infected animals or indirect contact with fomites contaminated with the virus. A case report of human Orf contracted by handling deer carcasses with bare hands is available (Kuhl et al. 2003).

Tanapox infection is extremely rare outside Africa and endemic to equatorial Africa. Non-human primate-to-human and human-to-human transmission have been described. Arthropod mediated transmission is also suggested. So far, only four human cases have been reported in the USA: where three cases were of research personnel who handled laboratory animals, and one case was a traveller recently returned from Sierra Leone. In Europe, a tanapox case was reported in a person who had recently arrived from Africa to Germany. A typical tanapox case was reported in Africa in a student working with orphaned chimpanzees (Dhar et al. 2004).

15.21 Control Strategies

Diseases shared with wildlife species are multi-host infections, which have a potential impact on public health, economy; wildlife management and conservation where wildlife itself plays a major role in the maintenance of the infection. The complete eradication of shared zoonotic pathogen is impossible ignoring its wildlife reservoir hosts. The control of such diseases needs the development of policies and strategies that will decrease the transmission of the pathogen between wildlife species and both human beings and domestic livestock. Also, a collaborative trans-disciplinary effort

in a One Health context is vital to protect the health of human, livestock, wildlife, and the environment. The following are some options that are suggested to control viral zoonotic diseases at the wildlife–livestock–human interface.

1. Establishing suitable disease surveillance and monitoring in wildlife species. Monitoring targets on the known infected population of wildlife to map temporal and spatial trends, whereas surveillance focuses on healthy wildlife to demonstrate the disease absence. After the identification of disease, descriptive studies are to be undertaken to assess whether the disease and the role of wildlife is relevant for public or animal health or for wildlife conservation and management (Artois et al. 2009; Gortázar et al. 2015).
2. Alternate options like no-action or zoning or compartmentalisation should also be given a thought, especially considering cost/benefit estimation, but monitoring of disease and population is constantly required. Compartmentalisation and zoning can be and have been employed by states or countries to define sub-populations of different health statuses for controlling the disease. Zoning is defining a particular geographical area in which a disease exists (Artois et al. 2011).
3. Translocation control (“movement control”) is a well-known preventive option in controlling the disease for both livestock and wildlife. It prevents the introduction or re-introduction of infectious agents through the release of infected captive or free-living wildlife (Gilbert et al. 2005; Gortázar et al. 2015).
4. Barrier concept, which includes the use of small or large scale fencing or any other barrier, to prevent the spread of diseases by decreasing contact between animal populations. Farm biosecurity is one of the most prominent methods used to reduce wildlife–livestock–human interactions (Engeman et al. 2011; Judge et al. 2011).
5. Wildlife population control solves the problem of an increased reservoir population. Population control methods like feeding bans, increased harvesting, habitat management, random or selective culling, and reproductive control may be deployed (Gortázar et al. 2015).
6. Vaccination of wildlife emerges as a precious alternative or complementary method in disease control. As opposed to culling methods, general public easily accepts vaccination methods as it is sustainable and non-destructive (Beltrán-Beck et al. 2012).
7. Control of arthropod vectors employing insecticides, acaricides, and vaccines (tick) in the urban areas and use of protective clothing or repellents when visiting the forest areas are truly helpful methods as most of the viral zoonotic diseases are vector transmitted (Gortázar et al. 2015).
8. Proper removal of harvested wild animals (carcass, offal, and other remains) limits the potential spread of the infection mainly by mammals (Vicente et al. 2011).
9. Farming of wildlife species could diminish the risk of zoonotic infection spill over if comparable biosecurity and health measures are implemented to farmed wildlife as to domestic livestock (Murray et al. 2016).

10. Strong regulations can be instituted to prohibit and grant disincentives for illegal and legal trade of bushmeat to beat growing demand as an elegant commodity. High taxation charges may elevate the price to decrease demand and afford revenue for surveillance and enforcement efforts. Enacting high penalties may prevent participation in the illegal trade of wildlife (Murray et al. 2016).
11. Education of the general public about the risks connected with wildlife, bushmeat, and exotic pet trades (Chomel et al. 2007).
12. Future research on zoonoses involving wildlife hosts needs to embrace a collaborative trans-disciplinary approach to identify primary causes and to control their transmission (Daszak et al. 2000). Extensive studies to improve understanding of rodent–human/bat–human interactions to disrupt transmission cycles are needed to design innovative control strategies in the future.

15.22 Conclusions and Prospects

It is now well recognised in the global community that zoonotic diseases have emerged from wildlife hosts and are still emerging as a result of human and domestic livestock exposure to wildlife. The present chapter has comprehensively reviewed most of the viral zoonotic diseases from wildlife perspectives. The major pathways of disease transmission to humans from wildlife are direct exposure due to encroachment into formerly wild areas (fragmentation and degradation); growing co-mingling of domestic livestock and wildlife owing to land-use changes (habitat loss); increasing amount of international wildlife movement, overexploitation of wildlife, unsustainable practices in agriculture and other enterprises, and effect of invasive species. These basic factors influencing the disease emergence from wildlife species are also the major drivers of loss of biodiversity. Therefore, emerging zoonotic viruses are not only potential threats to humans but can also be pathogenic to wild host species. Thus, there is a convincing and effective chance for mutual gains for the conservation of wildlife and public health by collective and collaborative attempts.

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