World Applied Sciences Journal 35 (2): 188-198, 2017
ISSN 1818-4952

DOI: 10.5829/idosi.wasj.2017.188.198

Emerging Viral Zoonoses and Their Implications on Public Health

[1]*Fitsum Dulo and* [2]*Mahendra Pal*

[1]Department of Microbiology, Immunology and Veterinary Public Health,
School of Veterinary Medicine, Wolaita Sodo University, P.O. Box No.138, Wolaita Sodo, Ethiopia
[2]Department of Microbiology, Immunology and Veterinary Public Health, College of Veterinary Medicine
and Agriculture, Addis Ababa University, P.O. Box No.34, Debre Zeit, Ethiopia

Abstract: Microbiologic infections acquired from animals, known as zoonoses, pose a great risk to public health. It is estimated that 60% of emerging human pathogens are zoonotic. Of these pathogens, >71% have originated from wild life. These microbial agents can switch hosts by acquiring new genetic combinations that have altered pathogenic potential or by changes in behavior or socioeconomic, environmental or ecologic characteristics of the hosts. The emergence of these zoonotic diseases such as Hanta viral infections, Nipah virus disease, avian influenza, Ebola hemorrhagic fever, severe acute respiratory disease, Rift Valley fever, swine flu, West Nile fever are very distinct and hence their prevention and control will require unique strategies, apart from traditional approaches. Such strategies require rebuilding a cadre of trained professionals of several medical and biologic sciences. Since there is no way to predict when or where the next important new zoonotic pathogen will emerge or what its ultimate impact on human and animal health might be, investigation at the first sign of emergence is particularly highly important. The success of detection and control of these emerging zoonoses is largely based on international solidarity and cooperation. The emphasis is given on the permanent placement of the "Public Health Veterinarian "In public health as the animals serve as reservoirs of most of the zoonotic pathogens. In addition, veterinarians are encouraged to report the incident of zoonotic diseases, since animal infections are often a sentinel for human infection.

Key words: Control · Emerging Viral Zoonotic Infections · Public Health · Public Health Veterinarian

INTRODUCTION

Infectious zoonotic diseases are a continuing threat to our animal and human populations worldwide. They produce suffering and death and impose enormous financial burdens on society. The threat will continue to be extensive morbidity and mortality in both animal and human populations, disruption of global trade and financial burdens to healthcare systems and agricultural production systems [1].

Review in various part of world identifies 1415 species of infectious organism known to be pathogenic to humans, including 217 viruses and prions, 538 bacteria and rickettsia, 307 fungi, 66 protozoa and 287 helminths. Out of these, 868 (61%) are zoonotic, that is, they can be transmitted between humans and animals and 175 pathogenic species are associated with diseases considered to be 'emerging'. Out of the emerging pathogens, 132 (74%) are zoonotic and overall, zoonotic pathogens are twice as likely to be associated with emerging diseases as non-zoonotic pathogens. However, the result varies among taxa, with protozoa and viruses particularly likely to emerge and helminths particularly unlikely to do so, irrespective of their zoonotic status [2].

A number of factors involved for disease emergence which often follows ecological change caused by human activities such as agricultural changes, urbanization, migration, deforestation and dam building [3-5]. In addition, international travel and commerce, industrialization of food products, break down of public health measures and microbial adaptation also led to the emergence of the zoonotic diseases [5,6]. There is no way to predict when or where the next new zoonotic pathogen will emerge or what its ultimate importance might be. The effective surveillance and control of zoonotic diseases pose a significant challenge [5].

Corresponding Aauthor: Fitsum Dulo, Department of Microbiology, Immunology and Veterinary Public Health,
School of Veterinary Medicine, Wolaita Sodo University, P.O. Box No.138, Wolaita Sodo, Ethiopia.

A wide variety of animal species, both domestic and wild, act as reservoir for the new emerging and re-emerging diseases of humans [6]. Many of these diseases were either unknown because we were not able to isolate the infectious agent or to distinguish them from other clinical syndromes of discovered accidentally. Much of the recent identification of new pathogens has been based on new molecular biology tools or epidemiological studies. The role of veterinary profession is very important in public health and on the rise again in the USA, as it should be in many countries of the world. Veterinarians, therefore, must use all the available resources to meet the challenge of new emerging zoonoses [5].

Emerging zoonoses are those zoonoses that have been newly recognized or newly evolved, or are rapidly increasing in their incidence or expansion in geographical, host or vector range [3].The objective of this paper was to delineate the public health threat due to emerging viral zoonotic diseases.

Recent Examples of Emerging Viral Zoonotic Diseases

Ebola Viruses: Ebola viruses are members of the *Filoviridae* family. They are among the most virulent pathogens for humans and great apes, causing acute haemorrhagic fever and death within a matter of days and are potential bioweapons [7].

The reservoir hosts have not been definitively identified, but bats are the most likely candidates [8]. Ebola virus is transmitted through contact with infected patients or primates. During outbreaks, the virus appears to be transmitted by close physical contact with infected persons, their clothing and their body fluids including blood, vomitus, stools, saliva and sweat. The Ebola virus has also been detected in breast milk, urine and semen [9].

The risk of transmission from patients to other persons or to the hospital staff varies significantly. The chance the virus may be transmitted from a patient to nearby persons in the early stages of the disease is lower. The chances of transmission increase when there has been contact with a patient in the later stages of illness [10].

Incubation ranges from 2 to 21 days; in most cases, symptoms appear in 4 to 10 days. The symptoms usually appear abruptly; the initial signs may include fever, chills, headache, severe malaise, muscle aches, abdominal pain, nausea, vomiting and diarrhea [4]. Some patients develop conjunctivitis or pharyngitis with a non productive cough. A purplish-red, maculopapular rash may also be seen; this rash is especially common on the trunk and shoulders of patients infected with *Zaire ebolavirus*. After a few days, patients may develop mild to severe bleeding tendencies. In mild cases, this may be limited to bruising, bleeding of the gums, epistaxis, petechiae and/or mild oozing from venepuncture sites. In severe cases, patients have hemorrhage from the gastrointestinal tract or other sites, go into shock and develop multi-organ failure. Although many patients die, some begin to recover after a week or two. Recovery is more likely if the bleeding tendencies are mild. During convalescence, which can be slow, some patients develop joint pain, deafness, orchitis or pericarditis [8].

Filoviruses cause hemorrhagic fever in nonhuman primates. Wild chimpanzees and gorillas are often found dead. Clinical signs observed in dying wild animals (Of various species) during Ebola outbreaks include vomiting, diarrhea, hair loss and emaciation, as well as bleeding from the nostrils. Whether all of these signs are associated with filovirus infections or some were caused by other diseases is unknown [8].

ELISA techniques have been developed for detection of the viral antigen on inactivated specimens, such as blood, serum, or tissue suspensions [11]. Nucleic acid detection (RT-PCR) is more sensitive, but not often available in developing countries [7].

Presently, no specific therapy is available. As most Marburg hemorrhagic fever cases are highly fatal in both humans and nonhuman primates, it is highly important to euthanize the infected animals [8].

During outbreaks, suspects and exposed animals should be isolated and euthanized after confirmation of the disease. Strict infection control procedures are necessary to prevent virus transmission on fomites. Prevention of human exposure is vital. No vaccine is commercially available, but some vaccines being tested in nonhuman primates have been promising [8].

Severe Acute Respiratory Syndrome: Severe acute respiratory syndrome (SARS) is an emerging infectious viral respiratory illness caused by a coronavirus, called SARS-associated coronavirus (SARS-CoV) [4].

The SARS virus is capable of infecting a number of animal species and humans, with virus being detected in live animal markets and in the wider environment in rats, palm civets, raccoon dogs and ferret badgers [12].

The main way that SARS seems to spread is by direct contact with respiratory secretions and/or body fluids of diseased patient; transmitted most readily by respiratory droplets [4,12]. Cockroaches, faeces and sewage have been suspected to be the transmitter [4]. The use of aerosol-generating procedures (such as endotracheal intubation, bronchoscopy and treatment

with aerosolized medication) in hospitals may amplify the transmission of SARS-CoV and outbreaks have involved more than 100 patients on occasion [13]. There have been no reports of foodborne or waterborne transmission [4,12].

SARS-CoV disease has an incubation period of approximately 2–7 days; most patients become ill within maximum of 10 days after exposure. SARS is clinically characterized by high fever, chills, difficult breathing, cough, shortness of breath, myalgia, most of the patients develops pneumonia and case fatality may reach 10-50 percent. In animals, respiratory, gastrointestinal, liver and neurological symptoms are evident [4].

The presence of SARS-CoV has been demonstrated by molecular tool such as reverse-transcriptase polymerase chain reaction (RT-PCR) for early diagnosis of disease and the isolation of the virus from clinical samples such as respiratory secretions, blood, faeces on cell line. Immunological tests (ELISA, IFA) are also used to detect SARS-CoV IgG antibodies in the patient's sera [4].

Although a number of strategies have been used for specific treatment and prevention, controlled studies documenting the efficacy of therapies are lacking [13]. Supportive therapy with cefuroxime, clarithromycin and erythromycin may be tried [4]. In the absence of a vaccine, effective drugs, or natural immunity to SARS-CoV, the key to controlling SARS is the classic public health control strategy of case identification and containment [14].

Droplet precautions should be added to standard precautions for any patient known or suspected to have an acute respiratory infection, including patients with suspected or confirmed infection with novel coronavirus. Encourage the use of respiratory hygiene [4].

The most consistent association of increased risk of transmission to healthcare workers (based on studies done during the SARS outbreaks of 2002–2003) was found for tracheal intubation. Health care workers should strictly adhere to use of appropriate personal protective equipment and the potential for transmission to other non- SARS patients and hospital visitors should be minimized through administrative controls. Contact tracing, evaluation for illness and monitoring should be an immediate high priority to maximize the chance to rapidly control an outbreak [14].

Rift Valley Fever: Rift Valley fever (RVF) is an emerging zoonotic disease distributed in sub-Saharan African countries and the Arabian Peninsula. The disease is caused by the Rift Valley fever virus (RVFV) of the family *Bunyaviridae* and the genus *Phlebovirus* [15,16].

The bite of infected mosquitoes is the main transmission mechanism of RVF in ruminants during inter-epizootic periods. Ruminant-to-human transmission is the main infection route for humans, although they can also be infected by mosquito bites. Body fluids such as the blood (During slaughtering and butchering), foetal membranes and amniotic fluid of viraemic ruminants are highly infective for humans. Fresh and raw meat may be a source of infection for humans, but the virus is destroyed rapidly during meat maturation. Empirical field observations indicate that ruminants can also become infected by contact with material containing virus (e.g. fetus and foetal membranes after abortion), however, this route of transmission has not yet been confirmed [17,18]. Persons in contact with animals, such as veterinarians and workers in the livestock industry are at risk [16,19].

RVF usually presents in epizootic form over large areas of a country following heavy rains and flooding and is characterised by high rates of abortion and neonatal mortality, primarily in sheep, goats and cattle. The susceptibility of different breeds to RVF varies considerably. Some indigenous African animals may have only inapparent infections, while exotic or other breeds suffer severe clinical disease with mortality and abortion. Susceptible, older nonpregnant animals and some other species usually do not show signs of disease. Camels have been regularly involved in the RVF epidemics in East Africa and Egypt. Clinical disease is not seen in adult camels, but abortion occurs and some early post-natal deaths have been observed [20].

Clinical manifestations vary depending on age and animal species. During epidemics the occurrence of numerous abortions and mortalities among young animals, together with disease in humans, is characteristic [16]. RVF has a short incubation period: 12–36 hours in lambs. A biphasic fever of up to 41°C may develop and the fever remains high until shortly before death. Affected animals are listless, disinclined to move or feed and may show enlarged superficial lymph nodes and evidence of abdominal pain. Lambs rarely survive longer than 36 hours after the onset of signs of illness. Animals older than 2 weeks may die peracutely, acutely or may develop an inapparent infection. Some animals may regurgitate ingesta and may show melaena or bloody, foul-smelling diarrhoea and bloodstained mucopurulent nasal discharge. Icterus may sometimes be observed, particularly in cattle. In addition to these signs, adult cattle may show lachrymation, salivation and dysgalactia. In pregnant sheep, the mortality and abortion rates vary from 5% to almost 100% in different outbreaks and between different flocks. The death rate in cattle is usually less than 10% [20].

In most cases, human infections remain inapparent, or with mild, influenza-like symptoms. However, infected people may experience an undifferentiated, severe, influenza-like syndrome and hepatitis with vomiting and diarrhoea. Complications may also occur. Severe forms are manifested in three different clinical syndromes. The most frequent one is a maculo-retinitis, with blurred vision and a loss of visual acuity due to retinal haemorrhage and macular oedema. Encephalitis may also occur, accompanied by confusion and coma. This form is rarely fatal but permanent sequelae are encountered. The third and most severe form is a haemorrhagic fever, with hepatitis, thrombocytopenia, icterus and multiple haemorrhages [18].

Viral antigens and RNA can be detected in blood and tissue samples by various antigen detection tests and reverse transcription polymerase chain reaction (RT-PCR) assays. Enzyme-linked immunoassay (ELISA) and other serologic tests can detect specific IgM or rising titers [18,20,&21].

There is no specific treatment for either humans or animals [18]. Mosquito repellents, long shirts and trousers, bed nets and other arthropod control measures should be used to prevent transmission by mosquitoes and other potential insect vectors. Outdoor activities should be avoided, if possible, during periods of peak mosquito activity. Insecticides may be helpful. During epidemics, vaccination of susceptible animals can prevent amplification of the virus and protect people as well as animals [21].

Swine Influenza: H1N1 influenza (Swine influenza or swine flu) is an emerging respiratory disease of pigs caused by type A influenza virus that regularly causes outbreaks of influenza in pigs and present a terror effects all-round the globe [22].

The main route of transmission is through direct contact between infected and uninfected animals. These close contacts are particularly common during animal transport. Intensive farming may also increase the risk of transmission, as the pigs are raised in very close proximity to each other. The direct transfer of the virus probably occurs either by pigs touching noses, or through dried mucus. Airborne transmission through the aerosols produced by pigs coughing or sneezing is also an important means of infection [23].

Flu viruses are spread mainly from person to person through coughing or sneezing by people with influenza. Sometimes people may become infected by touching something, such as a surface or object with flu viruses on it and then touching their mouth or nose. The symptoms of 2009 H1N1 flu virus in people include fever, cough, sore throat, runny or stuffy nose, body aches, headache, chills and fatigue. Some people may have vomiting and diarrhea [23].

In pigs, influenza infection produces fever, depression, coughing (barking), discharge from the nose or eyes, sneezing, breathing difficulties, eye redness or inflammation and going off feed [24].

For diagnosis of swine influenza A infection, respiratory specimen (Nasopharyngeal swab, throat swab nasal aspirate, nasal washing) would generally need to be collected within the first 4 to 5 days of illness. Swine influenza can be diagnosed by virus isolation, the detection of viral antigens or nucleic acids and serology. Mammalian influenza viruses can be isolated in embryonated chicken eggs or cell cultures [24].

Methods of preventing the spread of influenza among swine include facility management, herd management and vaccination. Recommendations to prevent spread of the virus among humans include using standard infection control against influenza. This includes frequent washing of hands with soap and water or with alcohol-based hand sanitizers, especially after being out in public. Chance of transmission is also reduced by disinfecting household surfaces, which can be done effectively with a diluted chlorine bleach solution [24]. Antiviral drugs can be given to treat those who become severely ill, two of which are recommended for swine flu symptoms include: oseltamivir (Tamiflu) and Zanamivir (Relenza) [23].

Hanta Viruses: Hantaviruses are the most widely distributed zoonotic rodent-borne viruses and can cause two important clinical syndromes: haemorrhagic fever with renal syndrome (HFRS) and hanta virus pulmonary syndrome (HPS) in Asia and the Americas, respectively [25]. HPS is also referred to as hanta virus cardiopulmonary syndrome (HCPS) as deaths have been attributed to cardiac failure rather than pulmonary edema [26].

Currently up to 21 species and more than 30 genotypes of hanta viruses have been described. The important species include Hantaan virus (HTNV), Seoul virus (SEOV), Puumala virus (PUUV), Sin Nombre virus (SNV) and Dobrava-Belgrade virus (DOBV) [26,27].

Transmission among rodents and to humans is generally through respiratory secretions, urine and saliva and the aerosol route is felt to be most important. Direct excrement on aerosolization may contaminate open wounds, conjunctiva, food etc. Cat can get infected from rodents and act as potential reservoir. Human-to-human transmission has not been reported [4].

In man, symptoms include fatigue, fever and muscle aches, especially in the large muscle groups-thighs, hips, back and sometimes shoulders, dry cough, pneumonia, headache, nephritis, anemia, hematuria, oliguria, gastrointestinal bleeding, petechiae, renal dysfunction, shock, rapid respiratory failure and death. In animals, clinical signs are not described [4].

Hantavirus infections are often diagnosed by serology. Serological tests include the immunofluorescent antibody test (IFA), enzyme-linked immunosorbent assays (ELISA), immunoblotting and virus neutralization [4,28].

Hantavirus infections can also be diagnosed by finding antigens in tissues with immunohistochemistry. Viral RNA can be detected in blood or tissues with reverse transcriptase- polymerase chain reaction assays (RT-PCR). PCR assays can differentiate some hanta viruses [28].

There are no effective anti-viral drugs for the treatment of all hantavirus infections [4,26]. Ribavirin (1-*â*-D-ribofuranosyl-1, 2, 4-triazole-3-carboxamide) has been used in clinical trials for treatment of HFRS patients in the People's Republic of China and has shown reduction in fatality. However, it remains ineffective for treatment of HPS. Supportive therapy is the best to control progression towards life threatening symptoms [4,25,&26].

Prevention of exposure to rodent excreta is the best way to avoid infection. Simple preventive steps are: use of protective clothing while handling of rodents, decontamination of human dwelling having signs of rodent activity, maintaining rodents as pets should be discouraged and proper storage of food should be practiced. There are a few inactivated vaccines (Hantavax) licensed for use in Korea but the protective response is short lived [26].

Nipah Virus Disease: Nipah virus (NiV), a neurotropic, zoonotic paramyxovirus was first identified during a swine and human outbreak in peninsular Malaysia in 1998 and 1999 [29]. In pigs, the disease presented as primarily a respiratory syndrome which is locally known as "One –mile cough "Or "Barking pig syndrome" [30-32]. In humans, the virus was strongly neurotropic, causing encephalitis and ultimately killing 105/265 infected people who were mostly farmers and abattoir workers who came into close contact with infected pigs [4,29].

The natural reservoirs of NiV are fruit bats (Flying foxes) which belong to the family *Pteropididae* and genus *Pteropus*. They are asymptomatic carriers that shed the virus in saliva, urine, birthing fluid and products [31,32]. Disease is frequently encountered in humans and pigs [30,32]. Moreover, natural infection is also recorded in the cat, dog, goat, horse and sheep [4,30,31,&33]. Experimentally, the infection has been established in the golden hamster, cat and pig [30,31].

In humans, transmission of Nipah virus infection occurs via respiratory droplets, contact with nasal or throat secretions from the pigs or handling the tissues of sick animal [30-33]. Consumption of raw fruits or raw date palm juice contaminated with saliva or urine from fruit bats was considered the most likely source of infection in Bangladesh and India outbreaks [30,31,&34]. Person to person transmission through close contact with patient's secretions and excretions during outbreak in Bangladesh and India has also been recorded [34]. In domestic animals, the routes of transmission of Nipah virus from fruit bats to domestic animals is uncertain but pigs may be infected by eating fruits that may be contaminated with bat urine or saliva or by drinking contaminated water. The domestic animals may get infected mainly by contact with pigs [33].The cat can become infected through contact or by eating the tissues of bat [4,30,&31].

Human infections range from asymptomatic infection to fatal encephalitis. Infected people initially develop influenza-like symptoms of fever, headaches, myalgia, vomiting and sore throat. This can be followed by dizziness, drowsiness, altered consciousness and neurological signs that indicate acute encephalitis. About a quarter of the patients have seizures and about 60% become comatose and require mechanical ventilator. In the long term, persistent neurological dysfunctions are observed in more than 15 % of the patients [30,32].The case fatality may range from 40 to 75% [30-32].

In animals, typical clinical symptoms are observed in pigs where respiratory symptoms dominate. Nipah virus disease in pigs is also known as porcine respiratory and neurologic syndrome as well as barking pig syndrome based on clinical observation [30-32]. The mortality rate may reach to 40% in suckling pigs [4].

Nipah virus disease should be suspected if pigs have an unusual barking cough or if human cases of encephalitis are present [30,32]. Nipah virus infection can

be diagnosed by a number of different tests: serum neutralization, enzyme-linked immunosorbent assay (ELISA), polymerase chain reaction (PCR) assay, immunofluorescence assay, virus isolation by cell culture [30-32].

There is no effective treatment for Nipah virus disease, but ribavirin may alleviate the symptoms of nausea, vomiting and convulsions. Treatment is mostly focused on managing fever and the neurological symptoms. Severely ill individuals need to be hospitalized and may require the use of a ventilator [30,31].

Prevention activities include reducing the risk of bat-to-domestic animal transmission or other exposure to bat-contaminated fruits or fruit products and routine cleaning and disinfections of pig farms (With sodium hypochorite or other detergents). In case of an outbreak, potential control measures include quarantine of animal premises, culling or depopulating of infected animals or herds, restriction of animal movements and establishment of active animal and human health surveillance systems for early warning for veterinary and human public health authorities [30,31].

Avian Influenza Viruses (Bird flu): A disease capable of causing extremely high mortality in infected fowls was first defined in 1878 and was known as `fowl plague'. Only type A influenza viruses are known to cause natural infections of birds, but viruses of all 15 (H1-H15) haemagglutinin (HA) and all nine (N1-N9) neuraminidase (NA) influenza A subtypes in the majority of possible combinations have been isolated from avian species [35].

Influenza A viruses infecting poultry can be divided into two distinct groups on the basis of their ability to cause disease in chickens. The very virulent viruses cause `fowl plague', now termed highly pathogenic avian influenza [HPAI], in which mortality may be as high as 100%. These viruses have been restricted to subtypes H5 and H7, although not all viruses of these subtypes cause HPAI. All other viruses cause a much milder primarily respiratory disease designated low pathogenicity avian influenza [LPAI], which, nevertheless, may be exacerbated by other infections or environmental conditions resulting in a much more serious disease [36].

Avian influenza viruses are rare zoonotic pathogens. Even H5N1 has an extremely low incidence, globally, with a total of 602 cases and 352 fatalities over 10 years in 16 countries [37] with over 86% of cases occurring in only 4 countries – China, Vietnam, Egypt and Indonesia, all with a common agro ecological systems, conducive to the evolution and spread of these viruses [37].

Infected birds shed influenza virus in their saliva, nasal secretions and feces; the virus can survive for considerable lengths of time outside of the host. Susceptible birds become infected when they have contact with contaminated secretions or excretions or with surfaces that are contaminated with secretions or excretions from infected birds [37].

Domesticated birds, including chickens, ducks and turkeys, may become infected with Avian Influenza virus through direct contact with infected waterfowl or other infected poultry, or through contact with surfaces (Such as dirt or cages) or materials (Such as water or feed) that have been contaminated with the virus [4,37].

Infection with avian influenza viruses in domestic poultry causes two main forms of disease that are distinguished by low and high extremes of virulence. The ‘Low pathogenic’ form may go undetected and usually causes only mild symptoms (Such as ruffled feathers and a drop in egg production). However, the ‘highly pathogenic’ form spreads more rapidly through flocks of poultry. This form may cause disease that affects multiple internal organs and has a mortality rate that can reach 90-100%, often within 48 hours [37].

Avian influenza can be diagnosed by a variety of techniques including virus isolation. These viruses can be recovered from oropharyngeal, tracheal and/ or cloacal swabs in live birds and organ samples (Trachea, lungs, air sacs, intestine, spleen, kidney, brain, liver and heart) are tested in dead birds [38,39]. Real-time (RT)-PCR is the method of choice for diagnosis in many laboratories and serological tests including agar gel immunodiffusion, hemagglutination, hemagglutination inhibition and ELISAs are useful as supplemental tests [4,38,&39].

The quarantining of infected farms and destruction of infected or potentially exposed flocks are standard control measures aimed at preventing spread to other farms and eventual establishment of the virus in a country’s poultry population. Apart from being highly contagious, Avian Influenza viruses are readily transmitted from farm to farm by mechanical means, such as by contaminated equipment, vehicles, feed, cages, or clothing. Highly pathogenic viruses can survive for long periods in the environment, especially when temperatures are low. Stringent sanitary measures on farms can, however, confer some degree of protection [37]. People working with infected birds should follow good hygiene practices and wear appropriate protective clothing such as boots (Or shoe covers), coveralls, gloves and respirators.

Health education should be imparted to poultry farmers, workers, attendants and veterinarians about the mode of transmission, source of infection and preventive measures [4].

West Nile Virus Infection: West Nile virus (WNV) is an emerging mosquito-borne neurotropic flavivirus infection that was first identified in 1937 in the West Nile district of Uganda in East Africa [38,39].

The virus is maintained in a bird-mosquito-bird cycle and human infection results from mosquito bites. Other atypical modes of transmission that have been reported include transplacental transmission, breast-feeding, blood transfusion, organ transplantation and percutaneous inoculation [40].

West Nile Fever (WNF) is a potentially serious illness for humans and only 1 in 150 people infected with WNV will develop a neuroinvasive illness resulting in fatality (Mostly in the elderly). Neurological effects permanently presented may include meningitis, encephalitis and acute flaccid paralysis. Up to 20% of the infected people have milder symptoms and approximately 80% of people who are infected with WNV will show no symptoms at all [38-40]. As with the number of animal species affected, a variety of systemic effects have been observed. Neurological symptoms of an infected bird consist of ataxia, tremors, abdominal head posture, circling and/or convulsions. Pathology of the virus determined that it is non-discriminatory as to which organs are infected [41].

The most efficient diagnostic method is detection of IgM antibody to West Nile virus in serum or cerebrospinal fluid. Virus isolation in Vero cell culture remains the standard for confirmation of WNV positive pools. However, Vero cell culture is expensive and requires specialized laboratory facilities; thus, nucleic acid assays have largely replaced virus isolation as detection and confirmatory assay methods of choice [42].

Although no human vaccine is available to date, there are currently three WNV vaccines licensed for horses [43].West Nile infection prevention depends on two broad general strategies [44]; 1) reducing the number of vector mosquitoes through actions taken by the public or by municipal authorities and 2) preventing vector mosquitoes from biting humans by using mosquito repellents; avoiding locations where vector mosquitoes are biting; and using barrier methods, such as window screens or long-sleeved clothing. There is no specific treatment for WNV infection [4].

Zika Virus: Zika virus (family *Flaviviridae*) is an emerging a mosquitoborne arbovirus. It was first discovered in Uganda in 1947 and later in humans elsewhere in sub-Saharan Africa, arriving in south-east Asia at latest by the mid-twentieth century. In the twenty-first century, it spread across the Pacific islands reaching South America around 2014. Since then it has spread rapidly northwards reaching Mexico in November 2015[45] and more recently in Brazil (2015) and 31 countries in the Americas (March 2016) have reported outbreaks [46].

The Zika virus natural transmission cycle involves mosquitoes, especially *Aedes spp.* [47], but perinatal transmission [48] and potential risk for transfusion-transmitted Zika virus infections has also been demonstrated [49]. Moreover, Zika virus transmission by sexual intercourse has been suggested [49,50].

The most common symptoms of Zika infection are rash, fever, arthralgia, myalgia and conjunctivitis. Most of the patients manifested mild disease, but severe neurologic complications have been described in other patients in French Polynesia [46,49,51,&52]. Its clinical profile associations with Guillain–Barre´ syndrome and microcephaly have appeared recently [45]. However clinical identification of ZIKV is difficult due to similarities with other co-circulating viruses (e.g. dengue and chikungunya) and testing is currently the responsibility of specialized laboratories [46].

Serum samples were most commonly used to diagnose ZIKV infection, but other samples including saliva, nasopharyngeal swabs, urine, semen, amniotic fluid and breast milk have also been used to detect ZIKV infection with RT-PCR [46].

Prevention of Zika is achieved by vector control and insect bite precautions [46,52,&53]. *Aedes* spp. is adapted for indoor and daytime biting in urban areas. They are known to breed in aquatic environments such as small puddles, open water storage containers and plants that hold water between the leaves and stems. Insect bite precautions (during early morning and late afternoon peak biting times) and vector control should be tailored to known epidemiology [52,53]. To date, there is no vaccine for the prevention of Zika and a few studies have been carried out on this emerging arbovirus [53].

Factors Affecting the Emergence of Viral Zoonotic Diseases: The factors affecting the emergence are complex [54] and need to be better understood if events are to be anticipated and their impacts mitigated. In addition to mutation and recombinant events in the

organisms themselves, casual factors range from climate change that affects vector abundance and distribution to cultural and socio-economic factors such as movement of infected animals and people, tourism, changes in land use, changes in livestock management practices and a developing taste for exotic pets and meats. As is clear from the examples set out by the WHO, such factors usually interact in complex path way and can influence disease emergence. So, it is worth considering several prominent and overlapping issues that will impact emergence. These issues include, among others, ecological disruption, globalization and livestock movement [55].

Globalization and Livestock Movement: The volume of trade in animals around the world is large and continues to grow in line with increased demand of protein [55]. Global travel around the world is faster than the incubation period of zoonotic diseases. The distance and speed of travel increased 1000 fold since 1800 [5]. As the human population expands and moves from continent to continent and as various species are mixed together for trade, personal satisfaction, or technological advances, microorganisms are transferred to novel niches, with pathogenic results [57]. Moreover, agribusiness has become global and whereas the intensive rearing of animals for consumption dictates considerable savings in terms of economies of scale and efficiency, in intensive agriculture, larger quantities of goods and materials flow within a country and between countries, so potential for disease spread is high. Also, it is intuitive that larger collections of animals provide more optimal incubating conditions for the expansion of an emerging zoonotic disease, e.g. Nipah virus and highly pathogenic avian influenza [57].In recent years, live animal trading markets and the consumption of bush meat have received increased attention, largely due to their connections with outbreaks of SARS and Ebola [5,57].

Ecological Disruption: Ecological disruption is another major issue to address and may be one of the most dangerous factors in the emergence of new zoonotic diseases. As humans encroach on new habitat, it is a certainty that they will be exposed to novel pathogens that could move from their four-footed or avian niches into humans to engender disease [57]. It comes as no surprise, that wildlife may modify feeding practices as a consequence of changing land use, bringing them closer to humans and livestock. This modification was suggested to have been instrumental in the Nipah virus outbreak that affected pigs and humans in Malaysia in 1999. Nipah virus persists as a serious problem in many rural areas of Bangladesh and India, where infected bats living near human dwellings, urinate in date palm sap, which is later consumed raw by humans [58].

Surveillance and Control Methods of Emerging and Re-emerging Viral Zoonoses: Unfortunately, it is difficult to predict which zoonotic disease emerge obviously in the future due to the multifactorial and constantly evolving nature of the risk factors involved [5,56]. In these conditions the early and accurate detection of new outbreaks of epidemic diseases, including emerging zoonoses and an improved capacity to understand the underlying causes of the emergence of disease and the ecology of the agents and their hosts will assist in the effective prevention or rapid containment of future emergence events [56]. A number of international networks have been established in recent years to better detect and respond to such events. Some of these networks, such as the global early warning system for transboundary animal diseases (GLEWS) and international food safety authorities network (INFOSAN) emergency, global outbreak alert and response network (GORAN) focus on early detection and response in outbreak situations [56].

Advanced surveillance systems exist in a few countries [3] but most countries, especially developing countries, are ill equipped to develop, implement and maintain such systems. Emerging zoonotic diseases are likely to occur in countries that have weakest infrastructures for detection and response. In the light of recent global events (e.g. the emergence of SARS and outbreaks of avian influenza), there is an urgent need to enhance the capacity of these countries and subsequently, to connect the various surveillance and early warning, alert and response system at regional and international levels [56].

To recognize and combat zoonotic diseases, the epidemiology of these infections must be understood. We need to identify pathogens, their vertebrate hosts and their methods of transmission. Prevention and control strategies chosen must be in keeping with the characteristics of the virus, its transmission patterns and environmental stability, its pathogenesis and threat to animal health, productivity and profitability, zoonotic risk etc. [59].

There are a number of control methods and tools currently available at the animal reservoir, vector and human levels that are appropriate for the prevention and control of emerging zoonotic diseases. For domestic animals, the common methods and tools used in disease control are such as vaccination of pets or livestock, proper biosecurity and quarantine (e.g. excluding wildlife from domestic stock, hygienic practices in husbandry and among farm workers), eradication programmes (Depopulation), appropriate veterinary care, proper herd health programmes [3].

There is currently no international organization dedicated to monitoring, reporting and studying wild animal health on a global scale. The following methods and tools may be undertaken after careful evaluation of the species involved and its ecology: isolating and creating of physical barriers to exclude wild animals from farms or human residences, population control by culling, treating and vaccinating defined populations (e.g. oral rabies vaccination of foxes), limiting wildlife movement, conducting preliminary testing of all live import and exports, exercising care in adopting and translocating wild animals [3].

Vector control is an effective tool in the prevention and control of vector-borne zoonotic diseases, for example by, spraying against fleas and mosquitoes during plague and Rift Valley fever outbreaks, respectively. Other effective methods involve environmental management through elimination of vector breeding habitats as well as limiting anthropogenic activities that promote vector breeding, such as land-clearing, unplanned development and the destruction of habitats that support vector predators [3].

When available and legally permitted, the most valuable preventive measure is vaccination, not merely for the protection of the individual animal, but also to build up a level of population immunity sufficient to break chains of disease transmission. The importation of exotic disease into countries should be prevented by surveillance and quarantine programs [59].

CONCLUSIONS

As the result of virus genetic variation (Mutation, recombination and reassortment) and environmental factors, many zoonotic diseases get emerged along with re-emergence of some new viruses that pose major global threat to human health and sustainable development.

An estimated approximately 60% of emerging human pathogens posses zoonotic potential. Control of these emerging zoonotic infection including viral infection and protection of the public health will become even more challenging as world population increases. So, timely prevention and control of these diseases are very distinct and there is no way to predict when or where the next new zoonotic pathogen will emerge or what its ultimate importance might be, investigation at the first sign of emergence is particularly paramount. The success of detection and control of these emerging zoonoses is largely based on international solidarity and cooperation. However, the weak infrastructure in most countries specially in developing world needs capacity building of these countries and subsequently, to connect the various surveillance and early warning, alert and response system at regional and international levels.

Based on above facts and nature of global events, the following recommendations are forwarded:

- Coordinated action across the traditional professional lines of veterinary and human medicine.
- Enhancing record keeping, reporting and active disease surveillance programmes.
- Strengthening the capacity of diagnostic and research laboratories.
- Increasing the awareness of the society on the disease epidemiology.
- Keeping the natural environment from disasters (Avoiding the global warm).

REFERENCES

1. King, L.J., 2004. Emerging zoonoses and pathogens of public health significance. Revue Scientific Technique Office International des Epizooties, 23: 429-433.
2. Taylor, L.H., S.M. Latham and M.E.J. Woolhouse, 2001. Risk factors for human disease emergence. Transactions of Royal Society of London B: Biological sciences, 356: 983-89.
3. WHO, 2004. Report of the WHO/FAO/OIE Joint Consultation on Emerging Zoonotic Diseases, 3-5 May, Geneva. WHO, Geneva, pp: 46-49.
4. Pal, M., 2007. Zoonoses, 2nd ed. Satyam Publisher, Jaipur, India, pp: 21-90.
5. Pal, M., 2013. Public health concern due to emerging and re-emerging zoonoses. International Journal of Livestock Research, 3: 56-62.

6. Pal, M., 2005. Importance of zoonoses in public health. Indian Journal of Animal Sciences,75: 586-591.
7. Leroy, E. M., J.P. Gonzalez and S. Baize, 2011. Ebola and Marburg haemorrhagic fever viruses: major scientific advances, but a relatively minor public health threat for Africa. Clinical Microbiology and Infection, 17: 964-76.
8. CFSPH, 2009. Viral Hemorrhagic Fevers–Ebola & Marburg. Iowa State University, Ames, Iowa, USA, pp: 1-9.
9. WHO, 2014. What we know about transmission of the Ebola virus among humans.Media center News. 6 October 2014.
10. Ustun C. and O. Ozgurler, 2005. Ebola: A significant threat as an infectious disease and as a potential bioterrorism agent. Turkish Journal of Medical Sciences, 35: 1-4.
11. Saijo, M., M. Niikura, T. Ikegami, I. Kurane and T. Kurata, 2006. Laboratory Diagnostic Systems for Ebola and Marburg Hemorrhagic Fevers Developed with Recombinant Proteins. Clinical and Vaccine Immunology, 13: 444-451.
12. Shakespeare, M., 2009. Zoonoses. Pharmaceutical Press, Great Britain.
13. Peiris, J.S.M., K.Y. Yuen, A.D.M. Osterhaus and K. Stöhr, 2003. The severe acute respiratory syndrome. New England Journal of Medicine, 349: 2431-41.
14. Parashar, U.D. and L.J. Anderson, 2004. Severe acute respiratory syndrome: review and lessons of the 2003 outbreak. International Journal of Epidemiology, 33: 628-634.
15. Ikegami, T. and S. Makino, 2011. The pathogenesis of Rift Valley Fever. Viruses, 3: 493-519.
16. Pal, M., N. Akililu and B. Giro, 2012. Rift Valley fever: Rift Valley fever: A fatal viral disease of neonatal animals. International Journal of Livestock Research, 2: 14-20.
17. Gerdes, G.H., 2004. Rift Valley fever. Revue Scientific Technique, 23: 613-23.
18. Chevalier, V., M. Pepin, L. Plee and R. Lancelot, 2010. Rift Valley fever- a threat for Europe? EuroSurveillance, 15(10): 1905.
19. Flick, R. and M. Bouloy, 2005. Rift Valley fever virus. Current Molecular Medicine, 5: 827-834.
20. OIE, 2008. Terrestrial Manual. Rift Valley fever. OIE, Paris, France. pp: 323-333.
21. CFSPH, 2007. Rift Valley Fever. Iowa State University, Ames, Iowa, USA, pp: 8-11.
22. Parvathareddy, S.K., 2010. Knowledge and Awareness about H1N1 Influenza (Swine flu). MSc Thesis. Minnesota State University, Mankato, Minnesota.
23. Lade, K.S., S.D. Sawant and M.C. Singh, 2011. Review on influenza with special emphasis on Swine flu. International Journal of Pharmaceutical and Chemical Science. 3: 97-107.
24. Parmar, S., N. Shah, M. Kasarwala, M. Virpura and D.D. Prajapati, 2011. A Review on Swine flu. Journal of Pharmaceutical Science and Bioscientific Research, 1: 11-17.
25. Kruger, D.H., R. Ulrich and A.A. Lundkvist, 2001. Hantavirus infections and their prevention. Microbes Infection, 3: 1129–44.
26. Chandy, S., P. Abraham and G. Sridharan, 2008. Hantaviruses: an emerging public health threat in India? A review. Journal of Bioscience, 33: 495-504.
27. Lednicky, J.A., 2003. Hanta viruses: A short review. Archives of Pathology Laboratory Medicine, 127: 30-35.
28. Khairnar, H.K., R.J. Oswal and R.V. Antre, 2012. A review on Hantavirus. International Journal of Pharmaceutical and Chemical Science, 1: 347-352.
29. Chua, K.B., W.J. Bellini, P.A. Rota, B.H. Harcourt, A. Tamin, S.K. Lam and B.J. Mahy, 2000. Nipah virus: A recently emergent deadly Paramyxovirus. Science, 288: 1432-1435.
30. Pal, M. and J. Abdo, 2012. Nipah virus disease: A newly recognized viral zoonosis. International Journal of Livestock Research, 2: 65-68.
31. Wahed, F., S. Abdul Kader, Akhtarunnessa and M.M. Mahamud, 2011. Nipah Virus: An emergent deadly paramyxovirus infection in Bangladesh. Journal of Bangladesh Society of Physiology, 6: 134-139.
32. WHO, 2009. Nipah virus, Fact sheet No.262. WHO, Geneva, Switzerland.
33. CFSPH, 2007. Nipah virus infection. Iowa State University, Ames, Iowa, USA.
34. ICDDR, B., 2007. Person-to- person transmission of Nipah virus in Bangladesh. Health and Science Bulletin, 5: 1-6.
35. Alexander, D.J., 2000. A review of avian influenza in different bird species. Veterinary Microbiology, 74: 3-13.
36. WHO, 2012. H5N1 avian influenza: Timeline of major events, World Health Organization, Geneva, Switzerland, pp: 1-59.

37. Ahmed, A.N., 2008. Bird flu and Bangladesh: Review and update. Journal of Bangladesh College of Physician and Surgeon, 26: 32-38.
38. Paz, S. and J.C. Semenza, 2013. Environmental drivers of West Nile fever epidemiology in Europe and Western Asia. International Journal of Environmental Research and Public Health Environment., 10: 3543-3562.
39. Swayne, D.E., 2008. Avian influenza. In: Foreign Animal Diseases. United States Animal Health Association. Boca Raton, Florida, pp: 137-46.
40. Ravindra, K.V., A.G. Freifeld, C. Andre, A.C. Kalil, D.F. Mercer, J. Wendy, W.J. Grant, F. Jean, J.F. Botha and R.B. Stevens, 2004. West Nile virus–associated encephalitis in recipients of renal and pancreas transplants: case series and literature review. Clinical Infectious Diseases, 38: 1257-60.
41. Steele, K.E., M.J. Linn, R.J. Schoepp, N. Komar, T.W. Geisbert and R.M. Manduca, 2000. Pathology of fatal West Nile virus infections in native and exotic birds during the 1999 outbreak in New York City, New York. Veterinary Pathology, 37: 208-24.
42. Lanciotti, R.S., A.J. Kerst, R.S. Nasci, M.S. Godsey, C.J. Mitchell and H.M. Savage, 2000. Rapid detection of West Nile virus from human clinical specimens, field- collected mosquitoes and avian samples by a TaqMan reverse transcriptase-PCR assay. Journal of clinical Microbiology, 38: 4066-71.
43. De Filette, M., S. Ulbert, S.M. Diamond and N.N. Sanders, 2012. Recent progress in West Nile virus diagnosis and vaccination. Veterinary Research, 43: 16.
44. Petersen, L.R. and A.A. Marfin, 2002. West Nile Virus: A primer for the Clinician. Annales of Internal Medicine, 137: 173-179.
45. Gatherer, D. and A. Kohl, 2016. Zika virus: a previously slow pandemic spreads rapidly through the Americas. Journal of General Virology, 97: 269-273.
46. Waddell, L.A. and J.D. Greig, 2016. Scoping Review of the Zika Virus Literature. PloS ONE, 11(5): e0156376.
47. Hayes, E.B., 2009. Zika virus outside Africa. Emerging Infectious Diseases. 15: 1347-50.
48. Besnard, M., S. Lastère, A. Teissier, V.M. Cao-Lormeau and D. Musso, 2014. Evidence of perinatal transmission of Zika virus, French Polynesia. Euro Surveillance, 19: 20751
49. Musso, D., C. Roche, E. Robin, T. Nhan, A. Teissier and V.M. Cao Lormeau, 2015. Potential Sexual Transmission of Zika Virus. Emerging Infectious Diseases, 21: 359-361.
50. Foy, B.D., K.C. Kobylinski, J.L. Chilson Foy, B.J. Blitvich, A. Travassos da Rosa, A.D. Haddow, *et al*., 2011. Probable non-vector-borne transmission of Zika virus, Colorado, USA. Emerging Infectious Disease, 17: 880-2.
51. Oehler, E., L. Watrin, P. Larre, I. Leparc-Goffart, S. Lastere, F. Valour, *et al.,* 2014. Zika virus infection complicated by Guillain-Barré syndrome—case report, French Polynesia, December 2013. Euro Surveillance, 19: 7-9.
52. Summers, D.J., R.W. Acosta and A.M. Acosta, 2015. Zika Virus in an American Recreational Traveler. Journal of Travel Medicine, 22: 338-40.
53. Rodríguez-Morales, A.J., 2015. Zika: the new arbovirus threat for Latin America. The Journal of Infection in Developing Countries, 9: 684-685.
54. Wolfe, N.D., C.P. Dunvan and J. Diamond, 2007. Origins of major human infectious diseases. Nature, 447: 279-83.
55. Cutler, S.J., A.R. Fooks and W.H.M. Van Der Poel, 2010. Public health threat of new, reemerging and neglected zoonoses in the industrialized world. Emerging Infectious Disease, 16: 1-7.
56. Meslin, F.X., 2008. Public Health Impact of Zoonoses and International approaches for their detection and containment. Veterinaria Italiana, 44: 583-590.
57. Brown, C., 2004. Emerging zoonoses and pathogens of public health significance. Revue Scientific Technique Office International des Epizooties, 23: 435-442.
58. Luby, S.P., M. Rahman, M.J. Hossain, L.S. Blum, M.M. Husain and E. Gurley, 2006. Foodborne transmission of Nipah virus, Bangladesh. Emerging Infectious Disease, 12: 1888–94.
59. Manojkumar, R. and V. Mrudula, 2006.Emerging viral disease of zoonotic importance. International Journal of Tropical Medicine, 1: 162-166.