**Viral Zoonosis: A Comprehensive Review**

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**Abstract:** Zoonoses are human diseases caused by animal pathogens or animal diseases that are transmissible to humans. Zoonotic pathogens identified are mostly viral origin and are emerging and reemerging. Zoonotic viral infections are grouped based on the type of infection they produce in natural host. Some are associated with encephalitis/hemorrhages and others may cause only local lesions like rashes and arthralgia. Transmission of these viruses usually involves arthropod vectors, which sometime act either as mechanical and/or biological vectors. Some zoonotic agents may be transmitted directly through animal bite or close contact with infected animals or fomites. The zoonotic microbes continue to evolve and adapt with tremendous acceleration and expansion of global trade, human movement and population explosion for efficient adaptation in new host and ecosystem results in catastrophic effects. They continue to cause health hazards in most parts of world and are economically important and public health concern. Control of zoonotic diseases and protection of public health are challenging tasks as the world population is increasing proportionately. The prevention of these infections depends on improved diagnosis and highly effective therapeutics/prophylactics.

The collective effort of professionals from medical and veterinary and others is necessary to combat these zoonotic infections. In this review most important zoonotic infections along with their specific etiology, transmission (role of wild-life) manifestations and epidemiology and control/preventive measures are described, so as to create awareness to the scientific/public health community.

**Keywords:** Zoonosis, emerging diseases, vectors, epidemiology, control and prevention

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**INTRODUCTION**

Zoonosis (zoo-e-no-sis) is an infectious disease that may be transmitted from animals (wild and domestic) to humans or from humans to animals. The word zoonosis is derived from the Greek, zoon (animal) (pronounced as zoo-on) and nosos (disease). Of the 1415 microbial diseases affecting humans, 61% are zoonotic (Taylor et al., 2001) and among emerging infectious diseases, 75% are zoonotic with wildlife being one of the major sources of infection (Daszak et al., 2001). A new virus has been emerging almost every year since last two decades (Woolhouse and Sequeria, 2005). Of 534 zoonotic viruses (belonging to 8 families) identified 120 cause human illnesses with or without the involvement of intermediate host/vectors. In the past 15 years, many zoonotic viral infections...
are of emerging and re-emerging in nature (Wilke and Haas, 1999) and haemorrhagic fever causing viruses transmitted by insect vectors (arboviruses i.e., yellow fever virus) (Khan et al., 1988), rodents i.e., Hanta viruses (Peters and Khan, 2002) and also by direct contact i.e., Filoviruses (Payling, 1996). Thus, they pose a great challenge to both veterinary and public health professionals. It is essential to investigate the complex interactions between pathogens, host, vectors and environment to curtail these infections. This review focuses on description of the important zoonotic viral infections with especially the recently emerging and reemerging diseases and their causes, transmission, clinical manifestations, distribution and preventive measures, to abreast the knowledge on zoonoses.

Transmission

Zoonotic viruses are transmitted to humans either directly or indirectly. Direct transmission involves contact between the infected and susceptible individual (orf), bite (rabies) and handling of the affected animal's tissues or materials (Orf). Indirect transmission involves transmission through the bite of a hematophagous (blood-sucking) arthropod after replicating in the reservoir animal host (Japanese encephalitis, yellow fever). Most viral zoonoses require blood-sucking arthropods for their transmission to humans. Among them, mosquitoes (Equine encephalitis complex) are the most common followed by ticks (Powassan virus), sand flies (Vesicular stomatitis) and midges (bluetongue). The arthropod vector becomes infected when it feeds the blood of a viremic animal. In most of the cases, virus replicates in the arthropod tissues and reaches their salivary glands. The arthropod then transmits the virus to a new susceptible host when it injects infective salivary fluid while taking a blood meal. The extrinsic incubation period (time between ingestion and transmission of the virus) is usually 8 to 12 days. This period depends on the virus, the environment and the vector species involved (Hubalek and Halouzka, 1999). Arthropod-borne viruses generally remain undetected until humans enroach on the natural enzootic focus or until the virus escapes the primary cycle via a secondary vector or vertebrate host. Wild birds are important to public health as they carry various zoonotic pathogens and they either act as reservoir hosts or help in disseminating the infected arthropod vectors (Reed et al., 2003). In addition, bird migration provides a mechanism for the establishment of new endemic foci of disease at great distances from where an infection was acquired (avian influenza). There has been a change in the transmission pattern especially in the occurrence and incidence of diseases due to broadening of host range (Monkey pox and Nipah viruses), high mutation rate (avian influenza, FMD) and anthropogenic environmental changes viz., ecological imbalance and change in agricultural practices (Wilke and Haas, 1999).

Role of Wildlife in Zoonosis

The significance of wild life as animal reservoir for zoonotic viruses has been traced long back with two important ancient diseases such as rabies and West Nile virus and represent as large spectrum of transmission mode (Marr and Calisher, 2003). Of the total emerging diseases, 75% are considered zoonotic with wild life as a major source of reservoir. Recent emerging viral diseases which moved into new species such as AIDS, SARS and avian influenza have a strong evidence of wild life origin due to human encroachment and changed international trade and travel patterns. Commonly the pattern of moving of viral agents from wild animal species to human occurs either as actual transmission being rare (HIV, Influenza A, Ebola and SARS) but will be maintained and has potential of man to man transmission or direct/indirect manner through animal bite and arthropod vectors (rabies, Nipah, West Nile
virus and hantavirus) (Bengis et al., 2004). Many zoonoses with a wildlife origin are spread through insect vectors (Rift Valley fever, equine encephalitis and Japanese encephalitis), whereas, rabies by animal bite and hantaviruses by contact with rodent excreta is common. The outcome in the form of clinical manifestation in humans depends on the transmission pattern of the agent causing the disease. Direct contact and vector bite lead to the formation of rashes and ulcers, whereas, intake of contaminated meat/water lead to digestive tract problems and diseases transmitted by inhalation of infected foci of dust cause pneumonia like illness (Kruse et al., 2004). Wild life are basically involved in epidemiology of the disease which is influenced by other factors such as change in agro-climatic conditions, host abundance, movement of pathogens/vector/animal host including migratory birds and anthropogenic factors. For example, increase in transmission and subsequent spread of Sin Nombre Hantavirus causing Hantavirus Pulmonary Syndrome (HPS) to humans is due to increase in heavy rainfall and host abundance in USA. Increase in the emergence of some wild life diseases result in high potential of emergence of human pathogens as in the case of West Nile virus spread in USA. A potential threat to human health, animal welfare and species conservation from domesticated and wild life is presented equally by emergence of human and wild life pathogens.

**Manifestations of Viral Zoonoses**

Zoonotic infections are broadly grouped into (1) diseases causing no illness, (2) nonspecific viral syndrome and (3) severe illness. The third category of infections is further classified into (1) hemorrhagic fever, (2) encephalitis and/or rash arthralgia, (3) emerging and reemerging and (4) rare zoonotic infections.

**Encephalitis**

The major viral zoonoses, which are associated with encephalitis, are listed in Table 1. They are arthropod borne and belong mostly to five viral families (Rhabdoviridae, Flaviviridae, Togaviridae, Reoviridae and Bunyaviridae). Most of them are transmitted through mosquito or tick bites, except a few which are transmitted through bite of an infected host (rabies). Mosquitoes and ticks are major vectors for this category of infections. They cause symptoms like fever, vomiting, encephalitis, headache and neurological disorders. Some of these infections are confined to a particular country (Colorado tick fever), while others are distributed worldwide (rabies). Prophylactic/therapeutic measures are available for some of the infections, while for others vector elimination is the only means of control. Intense research is required towards the development of vaccines including conventional as well as recombinant. Specific diagnosis of this group of infections is done employing serological tests like Hemagglutination-Inhibition (HI), Complement Fixation (CF) and Virus Neutralization (VN).

**Hemorrhagic Fevers**

Most of the viral zoonoses causing haemorrhagic fevers are reported to be of emerging and reemerging in nature (Murphy, 1998). There are more than 16 zoonotic infections in this category (Table 2) belong mainly to four viral families (Arenaviridae, Bunyaviridae, Flaviviridae, Filoviridae). These infections are often associated with extensive bleeding in human (Lacy and Smego, 1996). Most of them are transmitted upon vector bite. The common vectors are mosquitoes and ticks. Vaccines are not available for majority of the infections and therefore, control relies on supportive treatment. Control of vector is the main means of control. Chemotherapy is available for some of the infections (Crimean-Congo haemorrhagic
<table>
<thead>
<tr>
<th>SI #</th>
<th>Zoonotic virus</th>
<th>Taxonomic status</th>
<th>Transmission</th>
<th>Vectors</th>
<th>Symptoms</th>
<th>Distribution</th>
<th>Prophylactics/Therapeutics</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Colorado tick/fever</td>
<td>F. keddiei</td>
<td>Tick bite and blood transfusion</td>
<td>D. andersoni (Rocky mountain wood tick)</td>
<td>Fever, chills, myalgia, prostration, meningitis and encephalitis and also hemorrhagic fever in children (5%)</td>
<td>USA, Canada</td>
<td>Supportive prevent tick bites</td>
<td>Murphy et al. (1999)</td>
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<tr>
<td></td>
<td>(American mountain</td>
<td>G. Coliviruses</td>
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<td>fever * (multiple genotype</td>
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<td>2</td>
<td>Equine encephalitis</td>
<td>F. Townsendi</td>
<td>Mosquito bite</td>
<td>Mosquitoes (C. meleagris)</td>
<td>Encephalitis</td>
<td>USA, Caribbean Island</td>
<td>Inactivated vaccines</td>
<td>Murphy et al. (1999)</td>
</tr>
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<td></td>
<td>virus complex (EEE, WEE and</td>
<td>G. Alphaviruses</td>
<td></td>
<td>Wild birds (R)</td>
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<td>for both human and horses</td>
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<td>VEE) *</td>
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<td>G. Flavivirus</td>
<td></td>
<td>A. tripper small mammals (R)</td>
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<td>Nakayama strain</td>
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<td>4</td>
<td>California serogroup</td>
<td>F. Bunyavirus</td>
<td>Mosquito bites maintained in eastern chimpanzee, tree squirrels and foxes</td>
<td>C. meleagris</td>
<td>Headache, fever and seizures</td>
<td>Central, Eastern USA</td>
<td>Prevention of central oedema and seizures; avoid mosquito bites; No vaccine</td>
<td>McFarland et al. (2001)</td>
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<td></td>
<td>encephalitis (including La</td>
<td>G. Bunyavirus</td>
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<td>Crosse virus) *</td>
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<td>6</td>
<td>Powassan virus *</td>
<td>F. Flavivirus</td>
<td>Mosquito bites maintained in rodents</td>
<td>Isodose ticks</td>
<td>Neurological signs and convulsions</td>
<td>Eastern Canada and the</td>
<td>Tick bite treatment</td>
<td>CDC (2001)</td>
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<td></td>
<td></td>
<td>G. Flavivirus</td>
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<td>Northern USA Worldwide</td>
<td>Tick control</td>
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<td>7</td>
<td>Rabies virus</td>
<td>F. Blantvirus</td>
<td>Dog bite inhalation Raccoon-reservoir host (USA) Skunk-reservoir horizontal</td>
<td>Bite of infected animal</td>
<td>Neurological disorders</td>
<td>Worldwide</td>
<td>Human diploid cell rabies vaccine (HDCV), Rabies</td>
<td>Krebs et al. (2000) and</td>
</tr>
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<td></td>
<td>(Other members of lysavirus are</td>
<td>G. Lyssavirus</td>
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<td>vaccine absorbed (RVA), Purified chick embryo cell vaccine (PCEC)</td>
<td>Anonymous (1999)</td>
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<td>Duvenhage and EBL2</td>
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<td>associated with human rabies)</td>
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<td>8</td>
<td>Tick-borne encephalitis</td>
<td>F. Flavivirus</td>
<td>Mosquito virus drinking raw milk</td>
<td>Isodose persulcatum</td>
<td>Encephalitis</td>
<td>Eastern Europe to China</td>
<td>Inactivated vaccines for both eastern and western TBE</td>
<td>Dupuis et al. (1999)</td>
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<td></td>
<td>(Eastern and Western) *</td>
<td>G. Flavivirus</td>
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<td>9</td>
<td>West Nile virus</td>
<td>F. Flavivirus</td>
<td>Mosquitoes with wild birds and Culex mosquitoes</td>
<td>Culex mosquitoes</td>
<td>Encephalitis</td>
<td>Africa, Asia, the</td>
<td>Vaccine for horses</td>
<td>Petersen and Roehrig (2001)</td>
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<tr>
<td></td>
<td></td>
<td>G. Flavivirus</td>
<td></td>
<td></td>
<td></td>
<td>Middle East and Europe</td>
<td>No vaccine for humans</td>
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</tr>
<tr>
<td>10</td>
<td>Kunjin virus</td>
<td>F. Flavivirus</td>
<td>Birds-amplifying hosts</td>
<td>Culex mosquitoes</td>
<td>Encephalitis in human and lethal in horses</td>
<td>Australia</td>
<td>No vaccine</td>
<td>Petersen and Roehrig (2001) and Murphy et al. (1999)</td>
</tr>
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<td></td>
<td></td>
<td>G. Flavivirus</td>
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</tbody>
</table>

EEE: Eastern equine encephalitis, WEE: Western equine encephalitis; VEE: Venezuelan encephalitis; R: Reservoir host. *Man to man transmission? Potential for bioterrorism
<table>
<thead>
<tr>
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<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Lymphocytic chorion meningitis (LCM), Lassa fever*, Argentine (Junin virus), Bolivian * (Machupo virus), and Venezuelan, Brazilian (Sabia virus) hemorrhagic fevers.</td>
<td>F: Arenaviridae G: Arenavirina</td>
<td>Direct contact with infected rodents, wild rats and mice (R)</td>
<td>Not reported</td>
<td>Aseptic meningitis and influenza like illness and rarely severe hemorrhagic manifestations</td>
<td>World wide (Europe, Asia, Africa and Americas) and Tacaribe virus found in Trinidad</td>
<td>No safe vaccine available for arenaviruses. Live attenuated vaccine has been developed for Junin virus in Argentina</td>
<td>Maiztegui et al. (1998)</td>
</tr>
<tr>
<td>2</td>
<td>Crimean-Congo hemorrhagic fever*</td>
<td>F: Bunyaviridae G: Nasivirina</td>
<td>Tick bite</td>
<td>Ticks</td>
<td>Fever, haemorrhages</td>
<td>Sub-Saharan Africa, E Europe, Russia, the Middle East, W China</td>
<td>No vaccine is available; Treatment is supportive; Ribavirin-limited success</td>
<td>Hoch et al. (1995)</td>
</tr>
<tr>
<td>5</td>
<td>E. African forest disease</td>
<td>F: Filoviridae G: Ebola virus</td>
<td>Tick bite wild birds and small mammals; reservoirs; monkeys, amplifier hosts</td>
<td>Haemophysalys sanguinipur</td>
<td>Fever, headache, myalgias, prostration, haemorrhagic</td>
<td>Shimpala Dist of Karnataka (India)</td>
<td>No treatment and vaccine; Avoid tick bite and contact with sick monkeys</td>
<td>Murphy et al. (1999) and Pattnaik (2006)</td>
</tr>
<tr>
<td>7</td>
<td>Yellow fever</td>
<td>F: Flaviviridae G: Flavivirus</td>
<td>Mosquito bite</td>
<td>A. aegypti</td>
<td>Inapparent infection to a deadly hemorrhages</td>
<td>Tropical America and Africa</td>
<td>Live attenuated 1/D vaccine</td>
<td>Martin et al. (2001) and Chan et al. (2001)</td>
</tr>
</tbody>
</table>

*Potential for man to man and nosocomial transmission. **Potential for bioterrorism; R: Reservoir host
fever) with a limited success. Though some of these infections have local importance (Kyasanur forest disease), others have global impact (Dengue, Yellow fever). Specific laboratory diagnosis of hemorrhagic fevers usually requires special serological or virological tests like enzyme-linked immunosorbent assays (ELISAs) to detect virus-specific immunoglobulin. Other tests like Haemagglutination Inhibition (HI), Complement Fixation Test (CFT) and Virus Neutralization (VNT) have to be carried out on paired serum samples collected on two occasions i.e., acute and convalescent phases of illness.

Rashes and Arthralgia
A very few viruses are associated with local rashes and arthralgia and almost all belong to Togaviridae family (Table 3). Most of them are transmitted to humans through infected mosquito bites. These vectors are mainly from Aedes and Culex families. No specific treatment is available and control depends on the elimination of vectors. EU countries appear to be free, while other continents are endemic for these infections.

Emerging and Reemerging Zoonoses
The complex interaction between environment/ecology, social, health care, human demographics and behavior influences the emergence and re-emergence of zoonotic viral diseases. Periodic discovery of new zoonoses suggest that the known viruses are only a fraction of the total number that exist in nature. The RNA viruses are capable of adapting to changing environmental conditions rapidly and are among the most prominent emerging pathogens (Ludwig et al., 2003). Mutations are more common in RNA viruses (Influenza) than DNA viruses (Pox). The common mutations are point (insertion/deletion), drift (minor) and shift (major). In addition to these, movement of population, birds, vectors, pathogens and trade contribute to the global spread of emerging infectious diseases (influenza, severe acute respiratory syndrome). Other factors viz., human migration, change in land use pattern, mining (disturbance of ecosystem), coastal land degradation, wetland modification, construction of buildings, habitat fragmentation, deforestation, expansion of agents host range, human intervention in wildlife resources like hiking, camping and hunting also influence on acquiring zoonotic infections from wildlife (Daszak et al., 2001; Bengis et al., 2004; Patz et al., 2004). Cessation of vaccination against smallpox since 1980s, emergence of some genetically related orthopoxviruses has been reported throughout the world i.e., monkey pox (Nalca et al., 2005), buffalo pox (Singh et al., 2007) and Bovine Vaccinia (BV) infections (Fernandes et al., 2009).

Despite successful eradication of some viral diseases (small pox and almost polio in humans and rinderpest in cattle) due to intensive research and dedicated coordinated efforts, modern medicine has failed to control many infectious diseases resulting from emerging and reemerging viruses (Table 4). Some infectious agents already known to be pathogenic have gained increasing importance in recent decades due to change in disease patterns. Several previously unknown infectious agents with a high pathogenic potential have also been identified (Manojkumar and Mrudula, 2006). Several infectious viral agents (DNA and RNA viral families) have been emerged as zoonotic agents (Table 4). They are associated with flu-like signs (Alkhurma virus infection, influenza A) to respiratory (SARS), pox lesions mostly localized distributed over hairless parts of body namely udder, teats, ears and tail (in buffaloes) and fingers and hands (in humans) due to buffalopox (Fig. 1a) or Orf virus infections in affected goats (Fig. 1b), hepatitis (hepatitis E virus), hemorrhagic fevers (Ebola, Marburg and hanta virus infections) and encephalitis (Hendra virus complex). Treatment/prophylaxis is not available to many of these infections. But some of antiviral
### Table 3: Viral zoonotic infections causing rashes and arthralgia

<table>
<thead>
<tr>
<th>SI #</th>
<th>Zoonotic Virus</th>
<th>Taxonomic Status</th>
<th>Transmission</th>
<th>Vectors</th>
<th>Symptoms</th>
<th>Distribution</th>
<th>Prophylactics/Therapeutics</th>
<th>Reference</th>
</tr>
</thead>
</table>
| 1    | Baruch forest virus | F: *Togavirus*  
G: *Alphavirus* | Mosquito bite | Mosquitoes | Florid rash and less common arthritis | Australia | No vaccine; Hygienic practice | Mackenzie et al. (1998) |
| 2    | Chikungunya | F: *Togavirus*  
G: *Alphavirus* | Mosquito bite | *A. aegypti* | Arthralgia, Hemarthrosis | Africa and Asia | No vaccine; Avoid mosquito bite | Carey (1971)  
Schuffenecker et al. (2006)  
Tesh et al. (1999) |
| 3    | Mayaro virus | F: *Togavirus*  
G: *Alphavirus* | Mosquito bite | *Haemagogus sp.* | Fever, headache, backache, myalgia, epigastric pain, chills, nausea, photophobia, arthralgia and maculo-papular rash | Caribbean and South America | No vaccine; Avoid mosquito bite | Gubler (1981)  
Wyss (1982)  
Sarosi et al. (1982)  
Muir et al. (1992)  
Peters (1995)  
Bancroft (2000) |
| 4    | O'nyong-nyong | F: *Togavirus*  
G: *Alphavirus* | Mosquito bite | *Anopheles* and other mosquitoes | Less pronounced fever; lymphadenopathy | East Africa  
(Uganda) | No vaccine; Avoid mosquito bite | Kiwanuka et al. (1999) |
| 5    | Ross valley fever | F: *Togavirus*  
G: *Alphavirus* | Mosquito bite | *Aedes* and *Culex* sp. | Myalgia, polyarthritis, headache, anorexia, nausea, tenosynovitis, less fever | Australia, Southwestern Pacific Islands and Fiji | No vaccine; Avoid mosquito bite | Gubler (1981)  
Wyss (1982)  
Sarosi et al. (1982)  
Muir et al. (1992)  
Peters (1995)  
Bancroft (2000) |
| 6    | Getah virus | F: *Togavirus*  
G: *Alphavirus* | Mosquito bite | Mosquitoes | Mild fever | South East Asia | No vaccine; avoid mosquito bite | Murphy et al. (1999) |

### Table 4: Emerging and re-emerging zoonotic infections

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<tr>
<th>SI #</th>
<th>Zoonotic Virus</th>
<th>Taxonomic Status</th>
<th>Transmission</th>
<th>Vectors</th>
<th>Symptoms</th>
<th>Distribution</th>
<th>Prophylactics/Therapeutics</th>
<th>Reference</th>
</tr>
</thead>
</table>
| 1    | Alkhurma virus infection | F: *Plosvirus*  
G: *Flavivirus* | Mosquito bite Direct contact with affected sheep and goats | Mosquitoes | Flu-like illness with hepatitis, hemorrhagic manifestations and encephalitis | Saudi Arabia | No vaccine; avoidance of tick bite and contact with infected animals | Madiani (2005)  
Charrel et al. (2006) |
| 2    | Avian influenza (H5N1)* | F: *Orthomyxoviridae*  
G: *Orthomyxovirus* (type A) | Direct contact with affected birds; Migratory birds | Not reported | Flu-like illness | Worldwide | Oseltamivir; Combination of Amantadine and Oseltamivir; HS vaccine (safe and potent) | Swerye and King (2003)  
Areechekchai et al. (2006)  
Hayden et al. (2009) |
| 3    | Buffalo pox | F: *Poxivirus*  
G: *Orthopoxvirus* | Direct contact common in human infection Mechanical transmission in animals but not in humans | Not reported | Pox like lesions on ulder, perineum, hairless parts of the body | India, Pakistan, Bangladesh | Live vaccine (Vij 1996) at IVRI, Muktaswar | Kollapure et al. (1997)  
and Singh et al. (2006, 2007) |
| 4    | Hemorrhagic fevers with renal syndrome (HFRS) two types; Korean HFRS and HFRS | F: *Bunyavirus*  
G: *Bunavirus* | Direct contact with infected rodents | Not reported | Hemorrhagic fever, renal and pulmonary syndrome | Korean type in south east Asia and HFRS in Europe, Asia | No vaccine; ribavirin treatment in earlier cases useful; avoid contact with rodents | McCaughhey and Hart (2000) |
<table>
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<tr>
<th>Sl #</th>
<th>Zoonotic virus</th>
<th>Taxonomic status</th>
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<th>Reference</th>
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<tbody>
<tr>
<td>6</td>
<td>Hantavirus pulmonary syndrome (Sin Nombre virus)</td>
<td><em>Bunyaviridae</em></td>
<td>Direct contact with infected deer mice</td>
<td>Not reported</td>
<td>Acute fulminant illness, case fatality rate 30%</td>
<td>North and South America</td>
<td>No vaccine; avoid contact with rodents but recent report on inactivated vaccine being tried out in China</td>
<td>McCaughhey and Hart (2000), Hooper and Li (2001) and Peters and Khan (2002)</td>
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<td>7</td>
<td>Hendra and Nipah (Henipavirus)</td>
<td><em>Paramyxoviridae</em></td>
<td>Hendra: Contact with infected horses Nipah: Contact with infected pigs</td>
<td>Not reported</td>
<td>Fever, pneumonia, encephalitis</td>
<td>Australia, Singapore, Malaysia</td>
<td>No vaccine; Supportive treatment</td>
<td>Barclay and Paton (2000) Chua (2005)</td>
</tr>
<tr>
<td>8</td>
<td>Hepatitis E virus infection (Swine and deer strains transmission to human under investigation)</td>
<td><em>Hepeviridae</em></td>
<td>Consumption of contaminated pork</td>
<td>Not reported</td>
<td>Hepatitis, jaundice</td>
<td>World wide</td>
<td>No vaccine; Supportive treatment</td>
<td>Fauquet (2005) and Geansa and Perdue (2004)</td>
</tr>
<tr>
<td>9</td>
<td>Marburg and Ebola*</td>
<td><em>Filoviridae</em></td>
<td>Direct contact with infected person</td>
<td>Natural reservoir and vector unknown so far</td>
<td>Hemorrhagic fevers</td>
<td>Ebola in humid rain forests in Central and Western Africa whereas Marburg in Central and Eastern Africa Guangdong Province, China, southern China, Hong Kong, Singapore and Canada</td>
<td>No vaccine and treatment</td>
<td>Peters and Duc (1999) and Miranda et al. (1999)</td>
</tr>
<tr>
<td>10</td>
<td>Severe Acute Respiratory Syndrome (SARS)*</td>
<td><em>Coronaviridae</em></td>
<td>Direct contact with infected person</td>
<td>Himalayan civet cat:</td>
<td>Respiratory and enteric symptoms</td>
<td></td>
<td>No vaccine; Control: stringent quarantine and avoid contact with infected person</td>
<td>Tai (2006) and Peiris and Poon (2008)</td>
</tr>
<tr>
<td>11</td>
<td>Monkey pox**</td>
<td><em>Poxviridae</em></td>
<td>Direct contact with infected monkeys and wild rodents</td>
<td>Wild rodents and non-human primates as reservoir; Emerged in USA due to imported wild rodents</td>
<td>Flu like illness, fever, malaise, back pain and rashes similar to smallpox</td>
<td></td>
<td>No licensed therapy but smallpox partially protects. Recently LC16 m8 vaccine virus lacking BSK protein useful in monkeys</td>
<td>Gualino and Eckburg (2004), Nalca et al. (2005), Sajo et al. (2006) and Parker et al. (2007)</td>
</tr>
<tr>
<td>12</td>
<td>New variant Creutzfeldt-Jakob disease (VCJD)</td>
<td>Unclassified</td>
<td>Consumption of infected/contaminated food by BSE</td>
<td>Not reported</td>
<td>Nervous signs</td>
<td>United Kingdom</td>
<td>No vaccine and treatment</td>
<td>Murphy et al. (1999) and Will (2005)</td>
</tr>
</tbody>
</table>

*Potential of man to man transmission. *Potential tool for bioterrorism
<table>
<thead>
<tr>
<th>#</th>
<th>Zoonotic virus</th>
<th>Taxonomic status</th>
<th>Transmission</th>
<th>Vector</th>
<th>Symptoms</th>
<th>Distribution</th>
<th>Prophylaxis/Therapeutics</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Ebolavirus</td>
<td>F. Filoviridae</td>
<td>Ooccluded sp.</td>
<td>Ooccluded sp in animal infection but not reported in human tissues</td>
<td>Conjunctivitis and flu like illness and reported in laboratory workers</td>
<td>Asia, Europe, USA, EU and FDA</td>
<td>Inactivated live attenuated vaccines (USA)</td>
<td>Gould and Hsia (2009)</td>
</tr>
<tr>
<td>2</td>
<td>Borna disease</td>
<td>F. Bornaiviridae</td>
<td>Direct contact with persistently infected animals</td>
<td>Not reported</td>
<td>Mental illness and symptoms of neurological disorders</td>
<td>Central Europe and eastern Asia</td>
<td>No vaccine</td>
<td>kitahara et al. (1985), riggs et al. (1997), and chalmers et al. (2005)</td>
</tr>
<tr>
<td>3</td>
<td>Foot and mouth disease (FMDV)</td>
<td>F. Poxviridae</td>
<td>Close contact with infected animals</td>
<td>Not reported</td>
<td>Fever, vesicular lesions on hands, and increased</td>
<td>Worldwide</td>
<td>Formalin inactivated vaccines for veterinary use</td>
<td>Bausch et al. (1997) and benson (2009)</td>
</tr>
<tr>
<td>4</td>
<td>Monkeypox virus</td>
<td>F. Herpesviridae</td>
<td>Macrophagocytosis Interspecies</td>
<td>VN and CF antibody titre</td>
<td>Persistent latent infection</td>
<td>USA</td>
<td>Vaccines or vaccinia</td>
<td>Ostrowski et al. (1998) and Murphy et al. (1999)</td>
</tr>
<tr>
<td>5</td>
<td>Newcastle disease</td>
<td>F. Paramyxoviridae</td>
<td>Direct inoculation</td>
<td>Not reported</td>
<td>Rabies-like infection with neurological and self-limited</td>
<td>Worldwide</td>
<td>Inactivated and attenuated vaccines for veterinary use</td>
<td>Coppen and hayden (1997) and purpura (2005)</td>
</tr>
<tr>
<td>6</td>
<td>Foot and mouth disease (FMDV)</td>
<td>F. Poxviridae</td>
<td>Direct contact with infected animals</td>
<td>Not reported</td>
<td>Foot like lesions on hairy parts of the body especially on hands, legs, back, etc depending on the virus</td>
<td>Worldwide</td>
<td>Inactivated and attenuated vaccines for veterinary use</td>
<td>batson et al. (1973), malik et al. (1984), CDC (1973), winters et al. (1999), fields et al. (1995), murphy et al. (1999)</td>
</tr>
<tr>
<td>7</td>
<td>Spongiform encephalopathy (SENV)</td>
<td>F. Togaviridae</td>
<td>Mosquito bite</td>
<td>Mosquitoes</td>
<td>Mild fever</td>
<td>USA</td>
<td>No vaccine and treatment</td>
<td>williams et al. (1979), and weidler and ebbesen (1995)</td>
</tr>
<tr>
<td>8</td>
<td>Simian immunodeficiency virus (SIV) and simian T cell leukemia virus</td>
<td>F. Retroviridae</td>
<td>Sexual contact</td>
<td>Not reported</td>
<td>AIDS symptoms with secondary bacterial and fungal complications</td>
<td>USA</td>
<td>No effective vaccine and treatment as far developed</td>
<td>murphy et al. (1999) and marx et al. (2004)</td>
</tr>
<tr>
<td>9</td>
<td>Vesicular stomatitis virus (VSV)</td>
<td>F. Rhabdoviridae</td>
<td>Direct contact with contaminated materials</td>
<td>Rhinovirus like fever in animal infection but not reported in human part</td>
<td>Acute influenza like illness with Fever, vesicular encephalitis, headache and myalgias</td>
<td>South America and north America and northern hemisphere</td>
<td>No vaccine, control through restricted animal movement</td>
<td>fields and hawkins, (1985) and manford et al. (1999)</td>
</tr>
<tr>
<td>10</td>
<td>Simian foamy virus (SFV/SHV)</td>
<td>F. Soliwiridae</td>
<td>Direct contact with non human primates through animal bite</td>
<td>Not reported African green monkeys, chimpanzees, cats, cattle and macaques (R)</td>
<td>Causes plethora of diseases namely multiple sclerosis, gravis disease and</td>
<td>Central Africa</td>
<td>No vaccine, control through avoiding contact with infected animals</td>
<td>switzer et al. (2004) and wolf et al. (2009)</td>
</tr>
</tbody>
</table>
Fig. 1: (a) Buffalo pox infection in human particularly milkers showing characteristic localized ulcerative and vesicular skin lesions on hand and fingers and (b) Orf virus infection in goats showing characteristic proliferative skin lesions on mouth, lips and nose compounds, which are under trial, are found to be effective. For example Ebola and Marburg viruses are inhibited in vitro by Carbocyclic-3-deazaadenosine, a first compound to cure these virus infections (Huggins et al., 1999).

Rare Viral Zoonoses

Several viral infections cause nonspecific febrile illness in humans and occur rarely (Table 5). Many of them are animal pathogens, but often they produce nonspecific febrile illnesses in humans, though, humans are not the primary hosts. However, there is an increasing trend of occurrence of such infections in recent times (Table 5). Transmission of these infections have been reported upon direct contact of human objects with infected animal (FMD particularly serotypes O followed by C and rarely A, buffalo pox, Orf), handling of such organims in the laboratory (bluetongue, Newcastle disease), sexual contact (simian immuno deficiency (SID) virus), bite/scratch (monkey B virus), vectors (semliki forest virus, African horse sickness and louping ill) and food and water (calici viruses such as swine vesicular exanthema, feline calicivirus and rabbit haemorrhagic disease virus (Thiel and Konig, 1999) causing vomition and diarrhoea. Recently, animal rotaviruses and Eyach virus related to Colorado tick fever virus and Oropouche fever virus, an arbovirus (Nunes et al., 2005) similar to dengue fever in Trinidad are reported to cause mild infections in humans. Treatment is not available for most of the human infections, while some of them can be treated with nucleoside analogues like acyclovir or gancyclovir.
Other Zoonosis

Prion diseases are caused by scrapie associated prion protein (PrP\(^\text{Sc}\)), which are proteinaceous infectious agents common in animals and humans. Some of the animal prion diseases are scrapie of sheep, Bovine Spongiform Encephalopathy (BSE) and goats and mink spongiform encephalopathy. Human prion diseases are Creutzfeldt-Jakob Disease (CJD), Kuru, Gertsmann Strausssler Schienker Syndrome (GSS) and fatal familial insomnia. The human disease variant (vCJD) is believed to be a zoonotic disease caused by BSE agent and recently an emerging disease as well (Murphy, 1998). The route of transmission of vCJD is not yet fully proven but it is generally transmitted through exposure to food contaminated by the bovine BSE agent (Will, 2003). Human prion diseases can be classified as sporadic, hereditary or acquired. Acquired form i.e., vCJD is caused by the transmission of infection from human to human or, as a zoonosis, from cattle to human. Transmission of infectious agents between species through xenotransplantation called xenosis (Takeuchi and Weiss, 2000) is another way of introducing viruses from animal to human (porcine endogenous retroviruses). No specific treatment and vaccine is available. Prevention is by avoiding consumption of BSE contaminated or half cooked meat.

Prevention, Control Measures and Perspectives

Effective prevention and control measures can be achieved through proper diagnostics and prophylactic aids to curtail further spread in most of zoonotic viral diseases. Improved sanitary conditions such as proper treatment and disposal of human waste, higher standards for public water supplies, improved personal hygiene procedures and sanitary food preparation are vital to strengthen the control measures. A clear understanding of epidemiology of the diseases with wild life as reservoir namely the virulence and transmissibility of many diseases (human monkey pox, Tana pox and Yaba pox) could help in understanding the severity and thereby to take appropriate measures in eradication of such dreadful diseases. Research should focus on molecular biology of these viruses so as to develop diagnostics and prophylactics in a modern way to combat these infections in short time. To safeguard the public health from pathogens of zoonotic infections, application of skills, knowledge and resources of veterinary public health is essential. It is time to combat viral zoonoses with a combined effort of veterinary and public health specialists. A better understanding of avian migration patterns and their infectious diseases would be useful to forecast disease outbreaks due to emerging zoonotic infections like avian influenza. Further, the control measures for emerging and re-emerging viral pathogens are demanding, as there is population explosion. Novel, highly sensitive and specific techniques comprising genomics and proteomics along with conventional methods would be useful in the identification of emerging and re-emerging viruses, thereby; therapeutic/prophylactic/preventive measures would be applied on time. The first line of measure to control any disease is the surveillance. Control and prevention strategies should be designed based on transmission pattern and characteristics of virus, involvement of vectors, environment and epidemiology of the disease. The European Union (EU) has established a net work termed as Med-Vet-Net to develop a network of excellence for the integration of medical, veterinary and food scientists in order to develop food safety measures and to improve research on the prevention and control of zoonoses, including food-borne diseases. The network will also consider the concerns of consumers and other stakeholders throughout the food chain. Another system the Hazard Analysis and Critical Control Point (HACCP), which is regulated under FDA and it aims at analyzing hazards associated with food and identify preventive and control measures to
check spread of food-borne diseases including viral pathogens. Similarly, sanitary and phyto-sanitary measures (SPS) measures, which are set out with WTO are to be strictly followed to have safe food in order to conserve the health of animal, human and plants due to zoonotic agents.

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REFERENCES


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