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Epidemiology, control, and prevention of emerging zoonotic viruses

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Abstract: Zoonoses are infections in humans transmitted by animal pathogens or animal infections transmitted to humans. Viruses are the main etiological agents of emerging or re-emerging zoonoses. This chapter will discuss the most relevant foodborne and waterborne viral zoonotic infections along with their specific etiological agent, the issue of global and local infections, climate change, clinical manifestations and epidemiology and possible control and prevention measures.

Key words: zoonoses, epidemiology, viral emerging diseases, control and prevention, global and local issues, foodborne, waterborne.

20.1 Introduction

Zoonosis, from the Greek *zoon* (animal) and *nosos* (disease), is defined as infections that are transmitted from animals (wild and domestic) to humans and from humans to animals, through several different routes: (i) direct contact; (ii) via intermediate vectors such as mosquitoes and ticks; or (iii) through food- and waterborne infections (Hubalek, 2003).

The vast majority of recognized human pathogenic viruses have been circulating for a long time in the environment and also among the population. Their evolution is related to human evolution and *vice versa*, resulting in an equilibrium in which both are able to coexist, sometimes without consequences for human health. However, the crossing over of the species barrier by some pathogens can have devastating consequences in terms of infections, public health and mortality. Several factors have highly impacted the spread of viral zoonoses throughout the world. Increasing globalization, increased mobility, changes in demography, environmental determinants

such as ecologic and climatologic influences, and intense farming have increased the potential for transmission of pathogenic viruses between animals and humans (Kuiken *et al*., 2005; Osterhaus, 2001). Human and livestock populations have increased globally, causing closer contact between animals and humans. The human impact of changes in ecology and climate, together with faster transportation between countries and regions (faster than the incubation period of most of the infectious diseases), have accelerated the emergence or re-emergence of zoonotic pathogens. 'Emergence' means either the appearance of a newly evolved or a newly recognized pathogen, or the appearance of a known existing pathogen in a geographic area where it has never been found before. 'Re-emergence' is used to refer to pathogens whose presence is already recognized in a given area but which have increased in incidence (locally or globally, as for pandemics) or for pathogens which have seen their potential to cause severe infections in humans increase due to a shift in their ecology allowing, for example, the crossing of barriers from wildlife to domestic animals (as in the case of avian influenza) (Cutler *et al*., 2010).

More than half of the microbial diseases affecting humans (61%) are attributed to zoonotic transmission, with wildlife being one of the biggest sources for the spread of infectious diseases (Daszak *et al*., 2001; Taylor *et al*., 2001).The majority of the viral infections in humans newly identified over the last decade had a zoonotic background (e.g., SARS-Coronavirus, avian influenza), indicating that the influence of animals in human diseases has increased and is continuing to evolve.

20.2 Emerging viruses: geographical factors

Throughout human history, the geographical distribution of diseases has undergone considerable change. Several factors, including progress in urban sanitation, increased standards of living and improvement in personal hygiene, accompanied by the development of vaccines and antibiotics, have helped to reduce the level of infectious diseases during the last two centuries. Anthropogenic activities have greatly influenced the environment. The following list summarizes the anthropogenic factors contributing to changes in zoonosis patterns:

- national and international conflicts;
- travel;
- population growth, stress, social inequality, urbanization;
- trade, import of exotic animals, transboundary animal transport;
- water control and irrigation projects;
- changes in infrastructures (such as air conditioning, cooling towers, among others);
- agricultural practices;

- conditions affecting pathogens directly or indirectly (antibiotic-resistant pathogens);
- climate change; and
- demographic change.

20.2.1 Human mobility and population growth

Since the beginning of human history, population movement has shaped the patterns and the spread of infectious diseases. During migration, not only do people carry with them their genetic make-up, but they may also carry pathogens in or on their bodies and they may take with them disease vectors or reservoir animals. Religious pilgrimages, military manoeuvres, and trade caravans have facilitated the spread of many diseases, such as the plague or smallpox. Military manoeuvres have always been a source of epidemics since soldiers can be brought into new environments with different infectious zoonosis (Haggett, 1994). Populations in occupied areas sometimes have to abandon their villages and cities, and many severe outbreaks have been associated with poor water quality and low levels of sanitation among refugees.

However, for most of human history, populations have stayed relatively isolated. This pattern has changed in recent centuries. People started exploring by sea and looking to discover new worlds. Columbus not only discovered the New World; he and other explorers also played a significant role in the emergence of infectious diseases. By the time the ships arrived at the New World, diseases such as measles, tuberculosis and influenza were widespread throughout Europe. Explorers from infected urban areas brought diseases to the Americas, where they killed thousands of people who had mostly evolved from a very small gene pool and had no immunity to the infectious agents. So when the explorers arrived several epidemics occurred, the first ones always being the most severe. For instance, the population of central Mexico fell by an estimated one-third in the decade following contact with Europeans and one-third to half of the local population of Santo Domingo died following smallpox infection which then spread to other areas in the Caribbean and the Americas. The explorers also paid a high price for discovering the New World, as they became infected with tropical diseases that were not indigenous in Europe.

Global economics and advances in technology have changed the world greatly in recent centuries. These two factors have also contributed to the emergence or re-emergence of infectious diseases and to the spread of zoonotic diseases that would otherwise have been confined to local areas. Advances in technology have speeded up transportation, increasing the global mobility of people, and allowing faster trade in products and services that can be obtained rapidly from anywhere in the world. Every day, around the world, more than 1.4 billion people cross international borders in aeroplanes. According to the World Tourism Organization, in 2011 a total of 980 million people crossed international borders worldwide, an increase of 4.4% compared to 2010, and

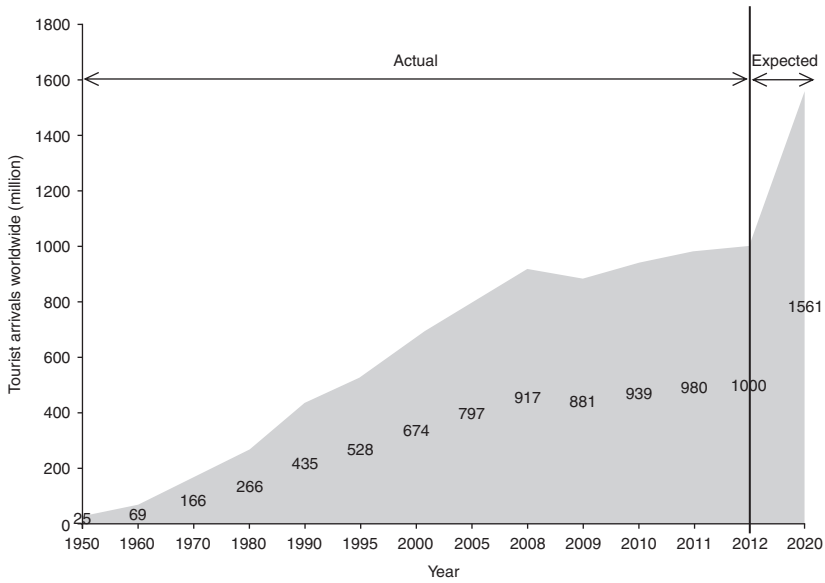

Fig. 20.1 International tourist arrivals (millions). Tourism is the fastest growing industry worldwide over the last century.

it is estimated that by the year 2020 that number will increase to more than 1.5 billion (see Fig. 20.1). Other means of transport carry millions of people, with cruise ships transporting 47 million passengers per year (Wilson, 2003). Today it is possible to travel to virtually any part of the world in a time period that is shorter than the incubation period of most infectious disease agents. Therefore, the traveller acts as a transmitter, a sentinel and a courier of infectious diseases. Wilson (2003) states that 'the traveller can be seen as an interactive biological unit who picks up, processes, carries and drops off microbial genetic material. A traveller can introduce potential pathogens in the absence of signs and symptoms.' Thus, human travel and migration have major consequences for the spread of infectious diseases worldwide.

By the year 2050, it is estimated that world population will increase by 27% from its current 7.0 billion to 8.9 billion. Many different factors – economic, social and political – have caused the human population to start migrating from rural areas to more urban ones. The number of big cities has increased, especially in developing countries where the population has risen steeply. The rapid growth of cities is often related to rapid and unplanned urbanization which can result in the deterioration of air and water quality, inadequate sanitation facilities and services, and the overuse of water resources (Moore *et al.*, 2003). The burden of infectious disease in developing countries with rapid urbanization rates is extremely high, due to both overpopulation and pollution (Crompton and Savioli, 1993). Closer contact between reservoir hosts also results from overpopulation, which then creates a potential risk of the

crossing over of species (Haggett, 1994). In developing countries, access to sanitation in urban areas lacking municipal water supply is still restricted, so people often use common water sources that are faecally contaminated (Moore *et al.*, 2003). The rapid and uncontrolled growth of population in urban areas and the extension of agricultural areas to new environments precipitate the risk of the emergence or re-emergence of an infectious disease.

20.2.2 Trade

However, other factors have influenced the emergence and re-emergence of infectious zoonoses, and continue to do so. Globalization is responsible for an increase in global trade. Through global trade, human pathogens, insect vectors, and their intermediate animal hosts can be disseminated throughout the world. Trade in food raw materials in the globalized world is increasing each year. Animals can be reared and fresh produce grown, and both can be shipped thousands of kilometres from place of origin to point of consumption (Tauxe, 1997). Again, advances in technology have enabled mass production, mass processing and a better food distribution network, making it feasible for food grown in one location to be processed somewhere else and consumed in multiple destinations. As a consequence, unwanted micro-organisms can be transported from one place where they are endemic to another where they may adapt and cause major outbreaks of infectious diseases (Wilson, 1995). Vector insects can also be transported along the distribution chain, adapt to a new environment and infect humans through direct contact or by having an amplifying animal host which then can contaminate environmental waters (see Fig. 20.2).

20.3 Clinical manifestations of some emerging types

The majority of zoonotic transmitted viral infections can be categorized into three different groups: (i) diseases with no illness; (ii) severe illness; and (iii) non-specific syndrome. Several emerging or re-emerging zoonotic viral diseases fall into the second category. Despite research and diverse institutional efforts to eradicate viral diseases such as poliomyelitis and smallpox, many infectious diseases, including several caused by emerging or re-emerging zoonotic viruses, are still uncontrolled and far from being eradicated or understood. Some previously known agents have become more infectious due to alterations in disease patterns (e.g.,Oropouche virus or Chikungunya virus). Others, such as Nipah and Hendra viruses or SARS-Coronavirus, were not discovered and described until very recently, and have a high pathogenic potential (Manojkumar and Mrudula, 2006). They are associated with encephalitis (Nipah virus, tick-borne encephalitis), haemorrhagic fevers (Hantavirus), flu-like signs (avian influenza) and respiratory disease (SARS-Coronavirus).

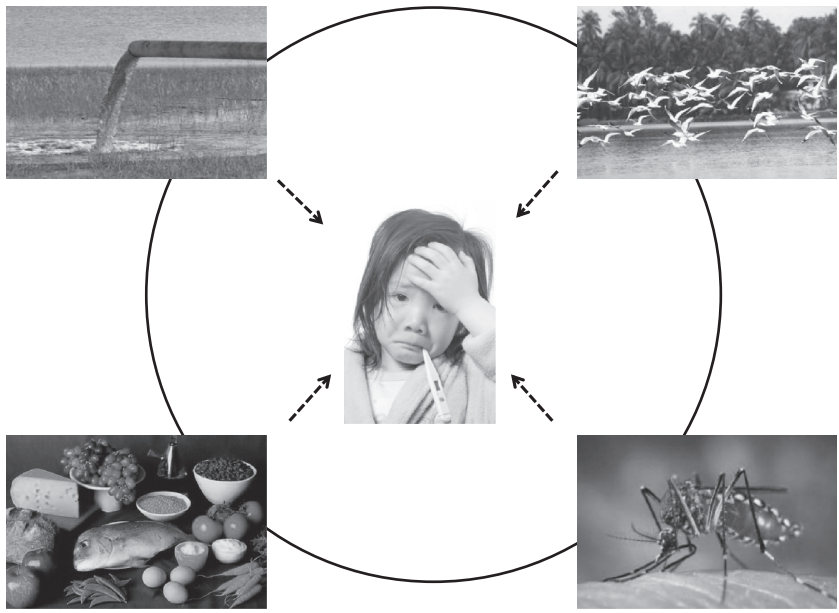

Fig. 20.2 Most common routes of transmission of infectious diseases between animals and humans.

20.3.1 Nipah virus

Nipah virus was discovered recently and belongs to the genus *Henipavirus* in the family *Paramyxoviridae* (Harcourt *et al*., 2000).It is a single-stranded RNA virus named after the village in peninsular Malaysia in which it was isolated for the first time from a human victim (Chua *et al*., 2000). Nipah virus was the aetiological agent responsible for a large outbreak in humans in Malaysia during 1998 and 1999 and for five subsequent outbreaks in Bangladesh between 2001 and 2005 (Chua *et al*., 2000; Hsu *et al*., 2004). The island flying fox, a fruit bat, (*Pteropushypomelanus*) is the primary reservoir for this virus where it persists in low numbers (Chua *et al*., 2002), and it has emerged via domestic animal amplifier hosts. Nipah virus has the ability to replicate uncontrollably in pigs, causing respiratory and/or neurological diseases leading ultimately to death (MohdNor *et al*., 2000). The effects on humans of infection by Nipah virus can range from an asymptomatic infection to fatal encephalitis. The incubation period can vary from four to 45 days. The first stage of the infection is the development of fever, headache, sore throat, myalgia, and vomiting, similar to common 'flu. The infection can then evolve to dizziness, altered consciousness, drowsiness, and some neurological signs indicating acute encephalitis. Moreover, some infected people can also exhibit severe respiratory problems and atypical pneumonia. The most severe cases develop encephalitis and seizures that progress in 24–48 h

to a coma situation. Over 20% of the people who survive acute encephalitis suffer subsequent neurological effects, including personality changes and repeated convulsions. Fatality is estimated in between 40% and 75% of cases, with the percentage depending on the outbreak and on the local authorities' surveillance capacity. For example, of a total of 269 human cases of encephalitis infection with Nipah virus reported in 1999 in Malaysia, 40% (108) were fatal (Malaysian Ministry of Health, 2001).

20.3.2 Tick-borne encephalitis (TBE)

TBE virus is a non-fragmented, single-stranded RNA virus that belongs to the genus *Flavivirus* contained in the *Flaviviridae* family (Mandl *et al*., 1997). TBE is endemic to northern, central and eastern Europe, Russia and the Far East. TBE cases have been reported not only in less developed countries such as Byelorussia, Bulgaria, Lithuania, and Kazakhstan but also in well-developed countries including Austria, Denmark, Germany, and Switzerland, where hygiene and sanitation standards are extremely high (Blaskovic *et al*., 1967; Korenberg and Kovalevskii, 1999; Lindgren and Gustafson, 2001; Ormaasen *et al*., 2001). The three main subtypes of TBE virus are the European, Siberian, and Far-Eastern (Ecker *et al*., 1999). TBE is a vector-borne infectious disease transmitted by the bite of the ticks *Ixodes ricinus* and *Ixodes persulcatus* (eastern Europe). Infections related to the consumption of infected unpasteurized milk and cheese made from such milk have also been reported (Dumpis *et al*., 1999). Infections as a result of the consumption of contaminated dairy products were first identified in the European part of Russia between 1947 and 1951. In these situations, whole families were being infected. This is contrary to what was observed when the transmission was from tick bites, where only single persons but not the whole family were infected. Research has proven that this form of disease was associated with the consumption of goat's milk containing infectious TBE viruses (Korenberg and Pchelkina, 1975; Popov and Ivanova, 1968). Later reports also demonstrated the transmission of TBE virus through the consumption of non-pasteurized milk from sheep and cows (Gresíková *et al.*, 1975; Leonov *et al.*, 1976).

The clinical manifestations resulting from an infection with TBE virus are well-known and a wide range of symptoms can be observed. The Far-Eastern subtype presents a monophasic progression, whereas the European is biphasic (Dumpis *et al*., 1999; Gritsun *et al*., 2003). The incubation period of a TBE virus infection is 7 to 14 days. In the biphasic course, symptoms in the prodromal stage can be mistaken for 'flu, with non-specific moderate fever and myalgia, accompanied by high fever and vomiting (Gritsun *et al*., 2003). In the European subtype, this is often succeeded by an asymptomatic period lasting 2 to 10 days. The disease can then progress to the nervous system, advancing to the second stage, which is characterized by the development of high temperatures and acute central nervous system (CNS) symptoms.

Acute TBE is characterized by encephalitic symptoms in a high percentage of infected persons, ranging from 45% to 56% (Haglund and Günther, 2003). Several symptoms may be observed, including mild meningitis and severe meningoencephalomyelitis, which progress 5–10 days after the remission of the fever. The acute febrile period correlates with the presence of the viraemia. In the second stage, the virus infects the CNS, where it replicates, resulting in the inflammation, lysis and dysfunction of the cells (Dumpis *et al.*, 1999). The second stage of illness can range from 2 to 20 days. During convalescence, problems with concentration and memory, sleep disturbances, and headache following mental or physical stress have been reported.

Patients with severe infections may show a poliomyelitis-like syndrome and an altered state of awareness which may result in a long-term disability (Dumpis *et al.*, 1999; Gritsun *et al*., 2003; Kleiter *et al*., 2007). The Siberian TBE virus subtype is thought to be responsible for severe chronic infections in Siberia and far-eastern Russia (Gritsun *et al*., 2003). Chronic TBE infections can be divided into two forms. In one form, the infected person develops hyperkinesias (which occurs regularly and can develop during the acute stage), and epileptoid syndrome. The second form of chronic TBE relates to long-term sequelae of one of the types of acute TBE, where the neurological symptoms can appear several years after the infected tick bite.

Latvia, the Urals, and the Western Siberian regions of Russia have the highest incidence of TBE viruses with the rate of attack in these regions reaching up to 199 cases per 100 000 inhabitants per year. Mortality rates due to TBE virus infection are extremely high for the Eastern TBE subtype, ranging from 5% to 20%, and extremely low for the Western subtype where it is between 0.5% and 2%.

20.3.3 Highly pathogenic avian influenza (H5N1)

Avian influenza (AI) is caused by a single-stranded negative-sense RNA virus belonging to the *Influenzavirus* A genus in the *Orthomyxoviridae* family (Swayne and Halvorson, 2008). Even though AI viruses are considered specific to given species and rarely cross the species barrier, since 1959 AI viruses subtypes H5, H7, and H9 have managed to cause sporadic human infections (INFOSAN IFSAN, 2004). H5N1 highly pathogenic AI (HPAI) was first isolated from a domestic goose in Guangdong, China in 1996, followed by an outbreak in the live bird markets in Hong Kong the following year. It was during this outbreak that the first human infection occurred and by the end of 1997, 18 people were hospitalized with six of the cases being fatal. Several other H5N1 HPAI outbreaks in humans have been reported since 2003,the probable cause being close contact with contaminated birds (CDC, 2006; WHO, 2006). Surveillance data have reported the detection of H5N1 HPAI virus in imported frozen duck meat, and both on the surface of and inside contaminated eggs (Beato *et al*., 2009; Harder *et al*., 2009;

Tumpey *et al*., 2002). However, although there is experimental evidence that the ingestion of uncooked poultry blood or meat has transmitted the H5N1 HPAI to carnivorous animals, there is no direct established link between the consumption of contaminated poultry products and human infection (CDC, 2007; WHO, 2005; Writ. Comm. Second WHO Consult. Clin. Aspects Hum. Infect. Avian Influenza A Virus, 2008). Contaminated matrices such as water, and poultry faeces used as fertilizers or fish feed, have been suspected to be the source of transmission of H5N1 HPAI viruses when no direct contact with poultry or poultry products existed (de Jong *et al*., 2005; Kandun *et al*., 2006). Human-to-human transmission of AI virus is relatively low but has occurred in Thailand (2004), Vietnam (2004), Azerbaijan (2006), Indonesia (2006), Egypt (2007), and Pakistan (2007), generally after one member of the family has been in direct contact with infected poultry or other infected humans (CDC, 2006; Ungchusak *et al*., 2005; WHO, 2008).

The first symptoms of infection with H5N1 HPAI viruses can occur 2–4 days after the exposure to infected poultry (Beigel *et al*., 2005). Longer incubation periods of more than eight days have also been reported (Beigel *et al*., 2005). It is not known whether and to what extent the virus is shed during this period (Beigel *et al*., 2005; Chotpitayasunondh *et al*., 2005; Kandun *et al*., 2006; Tran *et al*., 2004). The most common symptoms of an infection with H5N1 HPAI virus in humans include fever, shortness of breath, cough, severe respiratory disease, and pneumonia (Beigel *et al*., 2005; Chotpitayasunondh *et al*., 2005; Gilsdorf *et al*., 2006; Ungchusak *et al*., 2005). The virus appears to be the sole aetiological agent in the case of pneumonia, with no evidence of infection by any bacteria in most of the cases. In 2–8 days after the infection with H5N1 virus, patients also develop non-respiratory symptoms such as vomiting, diarrhoea, and abdominal pain (Ungchusak *et al*., 2005; WHO, 2005; Yuen *et al*., 1998), diarrhoea being in some cases the primary evidence of infection (Apisarnthanarak *et al*., 2004; de Jong *et al*., 2005).

H5N1 HPAI virus has been isolated from the cerebrospinal fluid and blood of a patient who developed symptoms of diarrhoea and convulsions, followed by coma (de Jong *et al.*, 2005). It was reported that the sister of this patient had also died of a similar illness just two weeks before, suggesting an existing predisposition to this type of disease (de Jong *et al*., 2005). As is observed for seasonal influenza virus, infection of the CNS appears to be rare (Morishima *et al*., 2002). The clinical pathway of an infection with H5N1 HPAI virus usually involves a rapid progression to the lower tract, at which point the patient needs to be mechanically ventilated (Beigel *et al*., 2005; Chotpitayasunondh *et al*., 2005; Tam, 2002; Tran *et al*., 2004). Acute respiratory distress syndrome (ARDS) has also been associated with the progression to respiratory failure. Several other clinical manifestations of multi-organ failure have been diagnosed, with cardiac and renal dysfunction, pulmonary haemorrhage, ventilator-associated pneumonia, and pneumothorax (Beigel *et al*., 2005; Chotpitayasunondh *et al*., 2005; Gilsdorf *et al*., 2006; Ungchusak *et al*., 2005).The mean time between onset and hospitalization was 4.6 days

(median of 4.0 days), ranging from 0 to 22 days (WHO, 2008). An increase in the case fatality rate was directly correlated to time from illness onset to hospitalization: 12% for 0 days, 47 and 55% for days 1 and 2, respectively, and over 70% for 4–6 days (WHO, 2008).

The WHO epidemiological study of the confirmed cases of H5N1 HPAI infection, conducted between November 2003 and May 2008, revealed a marked demographic incidence (WHO, 2008). Of 383 cases, 52% occurred in patients under the age of 20 years with the vast majority of the patients being under 40. The fatality rate was approximately 63%, infection with the H5N1 HPAI virus being more severe than infection with the seasonal virus. The demographic patterns of H5N1 virus are different to those observed for seasonal influenza. A higher fatality rate was found in the group comprising 10–19-year-olds (78%), in contrast to a low rate in those over 50 years. A possible explanation may be an age-related resistance or the paradigm of risk or exposure behaviour (e.g., close contact with infected poultry).

20.3.4 SARS-coronavirus

Severe acute respiratory syndrome (SARS) is a febrile respiratory illness primarily transmitted by respiratory droplets or close personal contact, and is caused by the SARS-Coronavirus (S → ARS-CoV) (Stockman *et al*., 2006). SARS-CoV is a positive, single-stranded RNA virus belonging to the genus *Coronavirus* within the *Coronaviridae* family. The major clinical features on presentation include persistent fever, chills/rigor, myalgia, dry cough, headache, malaise, dyspnea, sputum production, sore throat, coryza, nausea and vomiting. Dizziness and diarrhoea are less common features. Watery diarrhoea is a prominent extrapulmonary symptom in 40–70% of patients with SARS 1 week into the clinical course of illness (Hui and Chan, 2010).

In February 2003 China reported to the World Health Organization that 305 cases of atypical pneumonia of unknown aetiology had been identified in Guangdong Province since November 2002, and that five people had died. Also in February 2003, a physician from Guangdong Province, ill with atypical pneumonia, visited Hong Kong and stayed overnight in an hotel. The agent that caused his severe acute respiratory syndrome – SARS-CoV – was transmitted to at least ten persons, who subsequently initiated outbreaks in Hong Kong, Singapore, Vietnam, and Canada (Peiris *et al*., 2003). The incubation period after infection was 4.6 days and the mean time from symptom onset to hospitalization varied between 2 and 8 days, decreasing in the course of the epidemic, while the mean time from onset to death was 23.7 days (Hui and Chang, 2010; Leung *et al*., 2004). The final outcome of this outbreak was 8000 probable or confirmed cases and 774 deaths, the overall mortality during the outbreak being estimated at 9.6% (Leung *et al*., 2004).

In the 2002–2003 outbreaks, SARS transmission ceased 4 months after the initiation of global spread in Hong Kong. Subsequently there were three instances of laboratory-acquired infection and one reintroduction from

animals in Guangdong Province (Anderson and Tong, 2010), but none of these occurrences had sufficient secondary human-to-human transmission to generate a threat of a recurrent global outbreak.

The earliest cases of SARS in Guangdong involved employees of exotic meat markets in the province. Research has shown that the majority of the infections occurred in people directly handling animals only recently captured from the wild, and that were to be consumed as delicacies (Graham and Bari, 2010; Li *et al.*, 2006; Shi and Hu, 2008). Moreover, infections identified after the primary SARS epidemic was brought under control were associated with restaurants that prepared and served civet meat (Graham and Bari, 2010; Wang *et al.*, 2005). The culling of civets vastly reduced the number of infected animals in Guangdong marketplaces (Zhong, 2004).

However, several studies revealed that palm civets were simply conduits rather than the fundamental reservoirs of SARS-CoV-like viruses in the wild. SARS-CoV-like RNA sequences and anti-SARS nucleocapsid antibodies were found by several studies in an Old World species of horseshoe bats in the genus *Rhinolophus*, especially in *Rhinolophus sinicus* and *Rhinolophus macrotis* (Lau *et al.*, 2005; Li *et al.,* 2005). Further analyses suggest that Bat-SCoVs and SARS-CoV have been evolving independently, presumably in bat hosts, for a long time (Ren *et al.*, 2006). In addition to masked palm civets and bats, other animal species might have been involved in the evolution and emergence of SARS-CoV. At least seven animal species can harbor SARS-CoV in certain circumstances, including raccoon dog, red fox, Chinese ferret, mink, pig, wild boar, and rice field rat (Li *et al.,* 2005). In conclusion, it is likely that another outbreak of SARS-CoV or a similar Coronavirus may occur in the future. Timely measures should be taken that would restrain the virus from causing an epidemic like the one that occurred in 2003.

20.4 Possible control measures

Effective prevention and control measures for most zoonotic viral diseases can be accomplished by means of adequate diagnoses and prophylaxis. The first step in control of any disease is surveillance. The characteristics and transmission patterns of the virus, along with the understanding of vectors and animal reservoirs, and the environment and epidemiology of the disease, have to be fully considered when control and prevention measures are defined. A better understanding of the epidemiology of the diseases associated with wildlife as reservoirs, including the virulence of the agents and their routes of transmission, would contribute to improving eradication measures for such scourges. In addition, better sanitary conditions, including proper treatment and release of human waste, improvement in public water supplies, proper personal hygiene methods and sanitary food preparation, are of extreme importance in control measures. Improved diagnostics and prophylaxis require research deep inside the molecular biology of each virus. A clear knowledge of the

migration pattern of birds and the diseases they transmit would help to prevent outbreaks of emerging viruses such as H5N1 HPAI. The emergence and re-emergence of viral zoonoses must unite the efforts of two scientific fields that are currently working separately: public health and veterinary studies. Better, highly sensitive and faster detection techniques, including molecular biology methods such as genomics and proteomics working side-by-side with more conventional methodologies, would enable identification of emerging or re-emerging viruses. Rapid detection would facilitate the timely application of therapeutic/prophylactic/preventive measures. Proper vaccination campaigns can help decrease the incidence and spread of infectious diseases. Travellers should obtain information from the appropriate institutions, such as government medical advisory bodies and local health authorities, about the risks they incur, the prophylactic treatments available and the do's and don'ts while visiting a different country with a different environment and potential hazards. Medical institutions and the media should work together to disclose more knowledge on emerging or re-emerging diseases, the transmission routes, and the prophylactic measures available. Public awareness regarding the handling of wild animals in exotic markets and better food handling and cooking procedures would also help prevent outbreaks from occurring.

20.5 Conclusion

Globalization has enabled rapid development, but it has a darker side, accompanied as it is by the spread of emerging and re-emerging viral zoonoses. Changes in mobility patterns, favoured means of transportation, and land use, frequent and fast trade, and other anthropogenic factors increase the possibility of widespread viral disease. The presence of emerging or re-emerging zoonotic viruses can profoundly affect the environment and human health in a world where there is an ever increasing demand for water and food due to population growth. The implementation of effective prevention and control measures, up-to-date and helpful information for travellers, and better and improved sanitary conditions would greatly help in curbing the spread of emerging and re-emerging viral zoonoses.

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