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Impact of global change on transmission of human infectious diseases

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Global change, which refers to large-scale changes in the earth system and human society, has been changing the outbreak and transmission mode of many infectious diseases. Climate change affects infectious diseases directly and indirectly. Meteorological factors including temperature, precipitation, humidity and radiation influence infectious disease by modulating pathogen, host and transmission pathways. Meteorological disasters such as droughts and floods directly impact the outbreak and transmission of infectious diseases. Climate change indirectly impacts infectious diseases by altering the ecological system, including its underlying surface and vegetation distribution. In addition, anthropogenic activities are a driving force for climate change and an indirect forcing of infectious disease transmission. International travel and rural-urban migration are a root cause of infectious disease transmission. Rapid urbanization along with poor infrastructure and high disease risk in the rural-urban fringe has been changing the pattern of disease outbreaks and mortality. Land use changes, such as agricultural expansion and deforestation, have already changed the transmission of infectious disease. Accelerated air, road and rail transportation development may not only increase the transmission speed of outbreaks, but also enlarge the scope of transmission area. In addition, more frequent trade and other economic activities will also increase the potential risks of disease outbreaks and facilitate the spread of infectious diseases.

global change, infectious disease, natural factors, human activities

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Since the 20th century, the global incidence of infectious diseases has had a series of ups and downs. At the beginning of the century, influenza pandemics were prevalent; in mid century, the incidence of all types of infectious diseases was relatively low. However, since the 1970s, new infectious diseases, including AIDS, severe acute respiratory syndrome (SARS), highly pathogenic avian influenza A (HPAI H5N1), Ebola hemorrhagic fever, legionellosis and Lyme disease, have emerged. Certain non-prevalent diseases, including tuberculosis, cholera, schistosomiasis, plague

and sexually transmitted diseases have reemerged, followed by biological attacks and infectious diseases caused by human activities. Various infectious diseases have unprecedented impacts on human health, social stability and economic development. Nearly 15 million people worldwide die from these diseases every year, accounting for 25% of all deaths (Morens et al., 2004). Morbidity and mortality bring heavy economic burdens to developing countries (Guerrant et al., 1999). Infectious diseases, together with war and famine, still rank first on the list of the greatest threats to human survival (Morens et al., 2004; Binder et al., 1999).

Under the influence of global change in both the natural

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and social environment, the emergence and transmission patterns of infectious diseases have changed. Natural factors, especially global climate change, will directly or indirectly impact the transmission process of many such diseases. Global warming will also cause the rise of sea level and sea surface temperature, which increase the incidence of water-borne diseases including cholera and poisoning of shellfish products (McMichael et al., 1996; Patz et al., 1995). Research indicates that climate change, including increases of temperature and rainfall, more frequent deluges and storms, sea level rise, and environmental deterioration such as that caused by settlement in refugee camps, could cause outbreaks and prevalence of cholera (Tong et al., 2000). Global climate change will influence the spread of insect-borne infectious diseases, through its influence on the geographic distribution change of insect vectors, increase of propagation velocity, invasion strength of insect vectors and by shortening the incubation period of pathogens. Insect-borne infectious diseases strongly affected by climate change include malaria, schistosomiasis, dengue fever, viral encephalitis and others (McMichael et al., 1996; Patz et al., 1995). Extreme temperature, strong rainfall and natural disasters related to weather can directly cause death, adverse health effects and disease. Climate change indirectly affects human health through the following: Altering sources of infection, which causes increased emergence of infectious diseases and expansion of their geographic distributions; impacting grain yield, which causes emergence of dystrophic diseases; population movements, resulting from sea level rise that causes increasing incidence of infectious and mental diseases; by affecting air quality, which increases incidence of respiratory infectious diseases; by influencing society, the economy and population, which produces wide-ranging public health problems (<http://www.unep.org/annualreport/2011/>).

Socioeconomic factors are important in emergence, development and variation of infectious diseases (Jones et al., 2008). First, land use, the human living environment, frequent trade and increased tourism are believed to be important drivers for the re-emergence and devastation of infectious diseases (Patz et al., 2004; Taylor et al., 2001; Weiss et al., 2004; Woolhouse et al., 2005), and create many social problems. For example, antibiotic abuse will lead to pathogen emergence of drug-tolerant persisters and variants (as in malaria, dengue, tuberculosis, cholera and influenza). Land cover change may induce prevalence of infectious diseases; for example, cultivating wastelands and deforestation has led to the emergence and spread of hemorrhagic fevers. Second, changes in the style of human activity have contributed to the spread of infectious diseases. These changes include frequent population migration, an increase in erotic services and multiple sex partners, illegal trade, industrialization of food, insufficient heat treatment in mechanized food production, and careless disinfection.

1 Natural environment and infectious diseases

Under the impetus of global natural and human activities, the emergence and transmission patterns of infectious diseases have changed (Figure 1). Natural factors, including temperature, humidity, rainfall, vegetation and land use directly or indirectly influence the outbreak and spread of many such diseases. Among these factors, the effects of climate change cover the widest range and have greater magnitude. Human activities are not only the major driver of climate change, but also indirect drivers of infectious disease spread.

1.1 Climate change and infectious diseases

1.1.1 Meteorological factors and infectious diseases

Meteorological factors affect infectious disease via three aspects: pathogen, host (Kuhn et al., 2005) and transmission route.

(i) Effects of meteorological factors on pathogens. Temperature and humidity can directly impact reproduction of pathogens and their survival time in the environment (Zhang et al., 2008). Temperature has a significant effect on viruses. First, most viruses, bacteria and parasites have threshold temperatures to survive. For example, the threshold temperature for survival of the malaria parasite *Plasmodium falciparum* is 18°C (MacDonald, 1957), and the spread of malaria is limited to the range 16–33°C (otherwise the spore cannot reproduce). The optimal condition for malaria spread is high humidity and temperature, within 20–30°C (Khasnis et al., 2005). The threshold temperatures for the plasmodia *P. falciparum* and *P. vivax* to survive in malaria mosquitoes are 18°C and 15°C (Duane et al., 2001), respectively, whereas the threshold temperature for Japanese encephalitis virus survival is 20°C (Mellor et al., 2000). Many pathogens, such as *Vibrio cholerae* and hepatitis E virus are restricted to some tropical areas, also because of temperature limitation (Hunter, 2003). In addition, temperature can affect the evolution of viruses, thereby resulting in outbreaks of new infectious diseases. For example, global warming will cause the evolution of influenza virus and extensive outbreaks of influenza (Aimone, 2010; Brown, 2010; Gibbs et al., 2010; Tang et al., 2010). New influenza viruses are emerging, threatening the health and safety of human and other species. Studies have shown a certain correlation between global temperature and nucleoproteins. Global change would impact the evolution of arbovirus to some degree (Gould et al., 2009), and also affect change patterns of emerging diseases. Moreover, temperature can influence viral infectivity and risk. A study in Peru showed that 1°C rise in temperature would increase the probability of severe diarrhea by 5% (Checkley et al., 2000). Another study in Australia showed positive correlation between temperature and cases of *Salmonella* infection (D'Souza

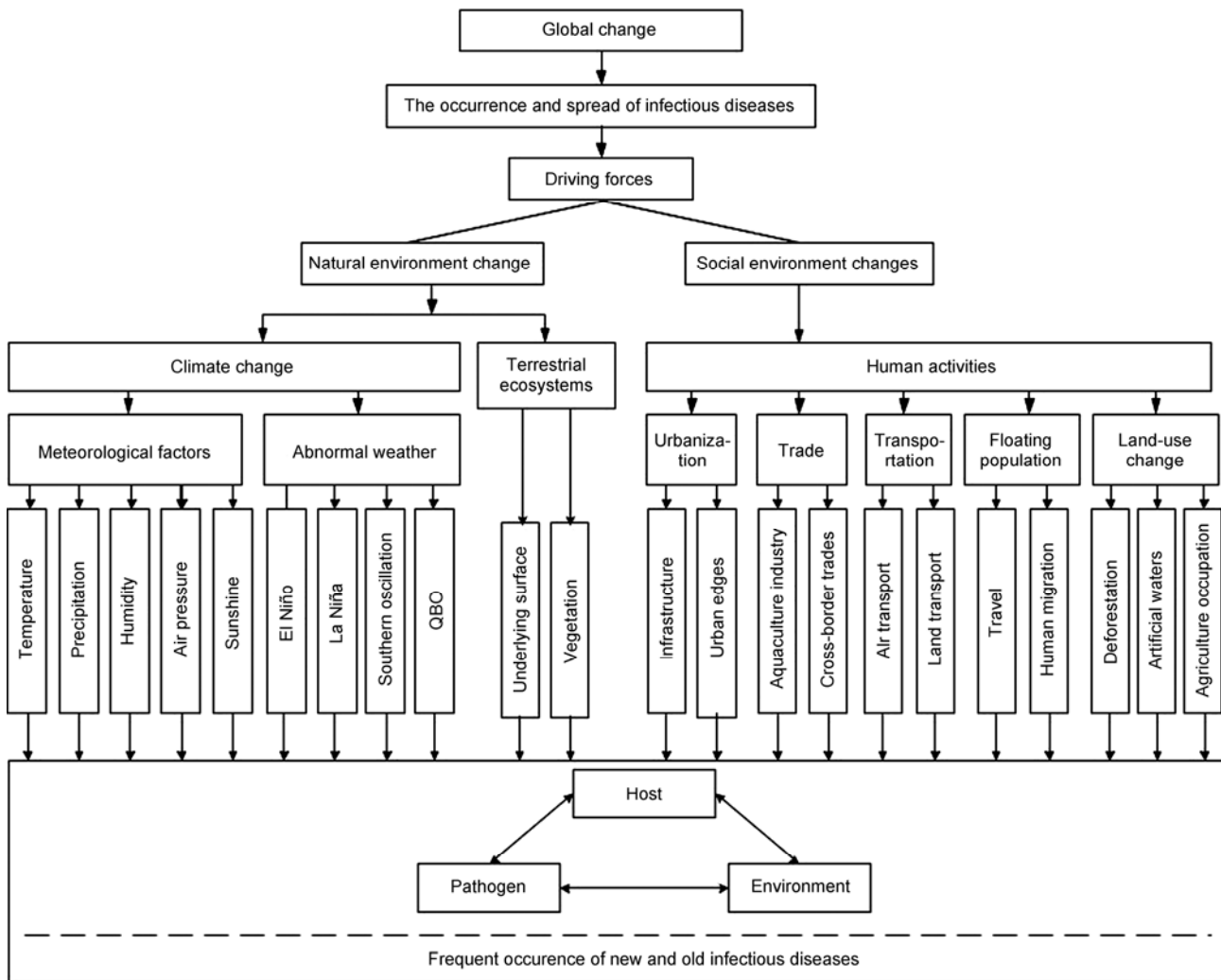


Figure 1 Relationship between global change and outbreak and transmission of infectious diseases.

et al., 2004).

(ii) Effects of meteorological factors on host. Climate change affects infectious disease hosts, via aspects including: (1) Spatiotemporal distribution of arthropods; (2) life cycle characteristics of arthropods; (3) spread pattern of related arboviruses; (4) spread efficiency from arthropod to vertebrate (Gould et al., 2009). Arthropod vectors are poikilothermic animals that are sensitive to change of climatic factors. Meteorological conditions influence the survival and reproduction rate of disease vectors, along with their habitat, distribution and quantity, spatiotemporal pattern of annual carrier activity and development, thus influencing pathogen survival and reproductive rate in disease vectors (Lafferty, 2009). Variations in carrier populations show positive correlation with temperature and humidity, and are also related to rainfall and sunshine, which are greater during periods of disease prevalence (Rogers et al., 2006). Geographic distribution and change of insect host species are closely related to patterns of temperature, rainfall and humidity. Temperature rise can accelerate entomic metabolism,

increase spawning quantity and blood circulation frequency (Mellor et al., 2000). Global warming and climate change have the potential to significantly impact the hydrosphere and fragile atmosphere (Zell, 2004), and especially species diversity, human health and infectious disease distribution (Harvell et al., 1999; Intergovernmental Panel on Climate Change, 2001). One such threat is increasing contact between humans and vector- or water-borne diseases. Global warming is beneficial to the spread of malaria (Khasnis et al., 2005). The effect of rainfall is also significant; it indirectly modulates the life cycle of insects through influencing humidity. A wetter environment is much more beneficial for insect reproduction, which thereby increases the geographic distribution and abundance of seasonal insect vectors (Kuhn et al., 2005). Moreover, mosquito hosts are very sensitive to climate. Their reproductive and death rates are influenced by climate (Zell, 2004).

Mild meteorological conditions are favorable for reproduction of ticks, and modify the distribution of Crimean-Congo hemorrhagic fever (Ergonul, 2006). Outbreaks of

this disease in Turkey are connected with mild temperatures in the preceding spring (Cazorla et al., 2003). Observations in recent years have shown that the number of rodents is also affected by climate, since warm and wet winters and springs can increase their number (Kausrud et al., 2007). Climate change aside, this number has the potential to grow in temperate areas, causing more contact between humans and rodents and greater risk of pathophoresis, especially in urban areas. In some European countries, damage to health infrastructure and the lack of health care knowledge among the public has increased the incidence of plague (Kausrud et al., 2007). A mild climate favors increase in the number of rodent species. However, harsh climate conditions such as heat waves may cause rodents to feed indoors, resulting in more contact with humans (Kausrud et al., 2007). Fluctuation in the number of main hosts is related to cases of plague (Davis et al., 2004). Climate change in central Asia is favorable for the spread of plague. According to prediction, 1°C temperature increase would heighten the number of diseases in hosts caused by *Yersinia pestis* by 50% (Stenseth et al., 2006). Plague epidemics in central Asia are common, and also impact European countries (Akiev et al., 1976). In Sri Lanka, drought has caused river to shift course and river water accumulation, which provides numerous breeding sites for local mosquito vectors and leads to frequent occurrence of malaria. Usually, in El Niño Southern Oscillation (ENSO) years, the northeast monsoon in that country brings sufficient rainfall, but not so the southwest monsoon. In recent years, the reemergence of malaria in certain locations may be related to ENSO. In recent decades, the longer propagation period of falciparum malaria in Pakistan has been connected with seasonal temperature increase in later parts of an ENSO year, which has a high temperature overall. Temperature can directly impact plasmodium growth and the mosquito life cycle. Sufficient rainfall is favorable for mosquito breeding, and suitable climate conditions can strengthen mosquito invasiveness. In 1987, malaria was very prevalent in Rwanda, mainly because of temperature increase (especially for temperature minima) and successional rainfall (Bouma et al., 1994). Temperature influences the growth, development, reproduction and death of *Schistosoma* and *Oncomelania*, and also contact between humans and infected water (Tong et al., 2000). In most cases, *Schistosoma* infection does not occur at low temperature (lower than 9°C). However, infection probability increases with temperature, maximizing within 24–27°C. However, temperatures in excess of 39°C can cause kill *Oncomelania* and reduce the *Schistosoma* infection rate (McMichael et al., 1996). The distribution of *Oncomelania* is also affected by rainfall (McMichael et al., 1996). Temperature has an important influence on the spread of dengue fever. With its increase, incubation of dengue virus in mosquitoes shortens, and these bite humans more frequently. Moreover, the geographic distribution of the mosquito that transmits dengue fever virus may be ex-

tended (McMichael et al., 1996; Patz et al., 1995). Global warming is likely to increase the incidence of food-borne diseases. For example, during the period 1982 to 1991, the incidence of food-borne disease in England was closely related to average temperature; this correlation had an average threshold temperature above 7.5°C (Tong et al., 2000).

(iii) Effects of meteorological factors on transmission. Climate change can influence the occurrence and spread of infectious diseases through its effect on transmission. For example, climate change can cause air pollution and shortages of food and clean water (Debono et al., 2012), leading to changes of high-incidence areas and outbreak patterns, thereby affecting the occurrence and spread of infectious disease.

Climate change can alter the spread of infectious diseases by influencing the water environment. Taking red tides as an example, global warming provides excellent conditions for algae growth. Algae in warm waters and sewage ditches contain many toxins, which may be related to an increased incidence of liver cancer in recent years. With the continuing rise in sea surface temperature, liver cancer has spread to coastal areas in high altitude and low temperature zones (Luber et al., 2009). Red tide outbreaks in drinking water sources present a huge threat to public health. Moreover, eating contaminated fish from red tide waters indirectly impacts human health (Morris et al., 1982). Climate change and over-exploitation of resources will aggravate the shortage of clean water in Africa and Southeast Asia (Intergovernmental Panel on Climate Change, 2007). Shortage of clean water may bring a series of waterborne infectious disease outbreaks. One study of children under the age of five from less developed and developing countries showed that low rainfall was closely related to outbreaks of illnesses such as diarrhea (Lafferty, 2009).

Climate change can alter the spread of infectious disease by influencing food. Climate change, including changes in temperature, rainfall and soil moisture, alter or relocate grain-producing areas (McMichael, 2001). People in such areas tend to be dystrophic, which is more common in developing countries. Thus, hunting wild animals to survive, along with poor health and sanitary conditions, increase risks of infectious disease in residents of these areas (U.S. Global Change Research Program, 2001). Model studies have also shown that decreased grain production caused by global climate change would increase the number of those living in poverty by 5%–10% (Parry et al., 2004); large number of refugees and increased transient populations would cause outbreaks of a series of infectious diseases (McMichael et al., 2006). Increases in average temperature will result in an obvious increase in the number of people infected with *Salmonella* and *Campylobacter* (D'Souza et al., 2004). Diseases caused by food sources can also kill vulnerable people (Hall et al., 2002). In 10 countries of mainland Europe, cases of *Salmonella* food poisoning have risen

to nearly 30% (Kovats et al., 2004). Investigations in England have also found a close connection between the incidence of food poisoning and temperature of the preceding 2–5 weeks (Bentham et al., 2001).

Climate change can alter the spread of infectious diseases by influencing air quality. One study focused on the potential effects of climate change on air pollution over the next 50 years in the eastern United States. The study stated that the ozone-related death toll would increase by 4.5% in 2050 relative to that in 1990 (Hogrefe et al., 2004). Climate change may alter geographic range and time of pollen and spore spread, thereby causing prevalence of anaphylactic diseases including hay fever and asthma (Beggs, 2004).

1.1.2 *Abnormal weather events and infectious diseases*

Abnormal weather events have significant effects on mosquito-borne, rat-borne and tick-borne diseases (Zell, 2004). Disease outbreaks rise significantly during El Niño events, such as incidence of diarrhea in Peru (Checkley et al., 2000), African horse sickness in South Africa (Baylis et al., 1999), and dengue fever in the South Pacific (Hales et al., 1996; Epstein, 1999). The increased hantavirus activity in the Four Corners region of the southwest United States in 1997 and 1998, as well as periodic epidemics of Rift Valley fever in Eastern Africa, are related to El Niño events (Epstein, 1999). Cases of human infection in Colorado of the U.S. are also related to El Niño (Hjelle et al., 2000), so we may conclude that the increase of rainfall owing to El Niño leads to a rise in the number of rodents. Such rise intensifies intraspecific competition, and increased contact between humans and rats strengthens hantavirus activity (Zell, 2004). El Niño is also related to outbreaks of cholera. The most catastrophic flood disasters in a century occurred in 1991 and 1998 in China; cholera was extensively prevalent in 1998, but not in 1991. The flood of 1991 was caused by El Niño, and it produced a decrease in sea surface temperature and seawater salinity in the southeastern waters of China, which is unfavorable for the prevalence of cholera. Flooding in 1998 was caused by La Niña, and it led to abnormal rise in sea surface temperature in southeastern waters, which was favorable for the growth and reproduction of *Comma bacillus* (Wang et al., 2000). The outbreak and prevalence of malaria in Ecuador, Peru and Bolivia in 1983 were related to heavy rains and accompanying ENSO.

The quasi-biennial oscillation (QBO) of stratospheric westerly winds at the equator is associated with tropospheric weather patterns such as change of atmospheric pressure caused by the Southern Oscillation. Recently, it was proven that Ross River virus found in Queensland, Australia had significant seasonal change, with peaks in summer and autumn. Heavier summer rainfall in southeast Queensland had a strong relationship with occurrence of the QBO in the west. The QBO can affect virus activity by influencing the local environment (Zell, 2004).

1.1.3 *Meteorological disasters caused by abnormal weather and infectious diseases*

Drought is found to have a close relationship with outbreaks of some infectious diseases. In 1993, an abnormal occurrence of deadly SARS broke out in New Mexico, Colorado, Utah and Arizona in the United States. A *Bunyaviridae* Hantavirus pathogen was named Sin Nombre virus (Nichol et al., 1993; Wenzel, 1994). Later, it was found that this virus was spread by the deer mouse. During previous outbreaks of hantavirus in spring and summer of 1993, locally abnormal winter rainfall caused a long drought, which resulted in a dramatic 10-fold increase in rodent numbers in the early part of the outbreak (Engelthaler et al., 1999). Owing to shortages of food, deer mice invaded human settlements to forage, thus introducing the virus and causing the outbreak of Hantavirus pulmonary syndrome (Engelthaler et al., 1999). Severe drought can lead to a shortage of water supply, so residents use water from pools with no stream-flow, secondary supplied water and water from long-term storage. Once the water resources are contaminated, outbreaks of intestinal infectious diseases can readily occur (Yang, 2007; Feng, 2000). Drought can cause pollutants to accumulate in dams and blue-green algae to massively propagate, releasing toxins in dams and lakes.

Rainstorms are also connected with the occurrence of some infectious diseases. The bunyavirus Rift Valley Fever Virus (RVFV) shows symptoms in humans, including fever and encephalitis accompanied by retinal inflammation, and first broke out in 1912 (Gonzalez-Scarano et al., 1996). This virus mainly harbors on the body surface of mosquitoes, primarily *Aedes* and *Culex* (Wilson et al., 1994). Rift Valley fever occurs after downpours, with abnormal sea temperatures in the eastern equatorial Pacific and western equatorial Indian Ocean (Linthicum et al., 1999). In addition to rise in sea surface temperature, rainfall causes many grassland depressions in East Africa, which provide breeding sites for immature mosquitoes. There are large numbers of infected grain-sized eggs in these floodplain grasslands, which are the origin of Rift Valley fever outbreaks. Rift Valley fever disappears with ending of the rainy season and decrease in number of mosquitoes (Zell, 2004). Research into the effects of flood disasters on infectious diseases has been carried out domestically. After these disasters, deterioration of environmental hygiene and increased hazardous exposure in crowds always causes outbreaks and prevalence of many infectious diseases. Xianning in Hubei Province suffered flooding in 1998. Investigation of this showed that respiratory infectious disease was the principal disease during the early and late flood stage and effector phase after flooding, while intestinal infectious disease was the main one during the flood period (Zhou, 2000). Insect-borne disease mainly occurs in the disaster period, whereas natural focus infection disease occurs during the later flood stage and effector phase after flood. Effects of flood disaster on infectious diseases include: (1) Effects on schistosomiasis related to

the spread of snails to surrounding areas via floodwater, flottage and other means, causing an increased incidence of schistosomiasis infection (Xie et al., 1999; Zhang et al., 2003, 2004); (2) effects on leptospirosis related to the fact that it is a zoonotic disease caused by pathogenic *Leptospira*, with rats and pigs as principal sources of infection. Most scholars believe that the prevalence of *Leptospira* in disaster areas after flooding depends on the carrier rate of infection sources (Ren et al., 2005). It has been shown that rat density is positively correlated to leptospirosis incidence (Pan et al., 2003); (3) effects on hemorrhagic fever with renal syndrome (HFRS) related to the fact that it is a natural focus infection disease, with rats as the major disease host. When humans and rats cohabitate, the likelihood of contact and infection increases. Local outbreaks or prevalence of HFRS readily occur. Sometimes, flooding may reduce the number of rats and thereby HFRS incidence. Since there is not enough time or no place for rats to move or escape to in flood disaster areas, some rats drown, which results in reduced numbers of hosts and disease incidence. However, some rats move to unaffected high altitudes or bordering land, where the number of infection sources increases, resulting in more contact between humans and rats and increased incidence of disease (Chen et al., 1999; Chen, 1999); (4) effects on intestinal infectious disease related to the fact that water supply facilities and sanitary fixtures like lavatories are flooded or inundated during flood disasters, leading to pollution of these and other water sources by garbage, excrement and the like. This directly produces outbreaks and prevalence of many intestinal infectious diseases, among which infectious diarrhea has a high-frequency (Chen et al., 1998; Cheng et al., 1999); (5) effects on malaria related to the connection between its incidence and rainfall (Wen et al., 2003). Flooding can increase epidemics in malarious areas (Zhang et al., 2004). In addition, torrential rains or rainstorms may transport sewage and waste water into sources of drinking water and dams, thereby causing diseases (Thomas et al., 2006). Recent focuses of model studies are on simulating variations of safety and usability of drinking water caused by climate change (Ferguson et al., 2007).

Heat waves are also frequently related to infectious diseases. Outbreaks of such diseases in southeast Romania from 1996 to 1997 were very similar to those that broke out in Israel in 2000. Both were related to heat waves caused by high temperature in early summer (Rogers et al., 2006).

1.2 Terrestrial ecosystem and infectious diseases

The outbreak of infectious diseases is related to types of underlying surfaces. Previous studies have indicated that appearance of seasonal wetlands is associated with outbreaks of HPAI H5N1 on the Indian subcontinent (Adhikari et al., 2009). The first case of avian influenza in Romania was in the Danube Delta, the largest wetland in Europe. One

of the most important factors sustaining transmission of avian influenza virus is that it may survive in water without a host. Water can increase the spread of excrement and saliva, propagate virus even lacking hosts, and redistribute viruses among different hosts (Gilbert et al., 2008). The occurrence of HPAI H5N1 is closely related to the distribution of water and wetlands in China, India and Pakistan (Adhikari et al., 2009; Fang et al., 2008; Biswas et al., 2009). Global transmission of H5N1 through wild bird migration, poultry transport and the interface of interaction between wild and domestic birds, has been determined for improved understanding (Liang et al., 2010).

The occurrence of infectious disease is associated with the vegetation ecosystem. In the Middle East and northeast Africa, HPAI H5N1 has occurred in areas with greater seasonal variation of Normalized Difference Vegetation Index (NDVI) values (Williams et al., 2009). In Europe, HPAI H5N1 occurrence is also closely related to NDVI (Si et al., 2010). Based on NDVI time series data, this occurrence in Africa and the Middle East is correlated with differences in phenological characteristics of plants (Williams et al., 2008, 2009). This results from the fact that plant distribution affects the food sources of waterfowl, thereby altering their distribution and movement.

Many kinds of natural factors together influence the occurrence and spread of infectious diseases. Research indicates that influences on HPAI H5N1 occurrence among wild birds in Europe were increased NDVI in December, intermediate NDVI in March and at lower elevations, increased minimum temperatures and decreased precipitation in January (Si et al., 2010). It is believed that this occurrence is mainly impacted by food sources, increased temperature and decreased precipitation (Si et al., 2010). Global warming and climate change may significantly modify the hydrosphere through heavy rainfall, floods, storms, heat waves and droughts. Research found that falciparum malaria in northwest Pakistan is related to continuous rainfall in September and October, and continuing high temperatures in November and December (Bouma et al., 1996).

2 Human activity and infectious disease

Human activities influence the emergence and transmission of infectious diseases via the following aspects (Figure 1).

2.1 Growing population mobility

2.1.1 International travel

With the development of transportation over almost a century, human travel has transitioned from interzonal to international. Over the last 50 years, the number of international travelers has risen by 1300% (Mavroidi, 2008). According to statistics, there are nearly one million international travelers daily and one million per week traveling between de-

veloped and developing countries (Garrett, 1996), representing nearly 700 million trips annually (Gossling, 2002). Ecotourism and adventure tourism have seen the highest growth rate at 10% each year since 1985, and often involve activities with high risk of disease transmission (Chomel et al., 2007). Increased tourism, business travel, and immigration have contributed to the dissemination of pathogens (Arguin et al., 2009). Existing reported diseases include HIV, legionellosis, *Cyclospora cayentanensis*, cholera, viral hemorrhagic fevers, transmissible spongiform encephalopathies, dengue, malaria, schistosomiasis, leptospirosis, tuberculosis, dysentery and others (Ostroff et al., 1998). According to transmission media, infectious diseases basically can be divided into water-borne, food-borne (Swaminathan et al., 2009), airborne and zoonotic diseases. Based on the Geosentinel Surveillance Network, statistics of 17353 infected travelers visiting developing countries indicated that tourism, visits with family and friends, and business travel respectively accounted for 59%, 15% and 14% of all trips. In such travel, malaria is the disease with the highest mortality rate and diarrhea is the most frequent, followed by dengue and typhoid fever (Freedman et al., 2006; Hill, 2006). African tick bite fever has been reported to have caused 350 cases attributed to intercontinental travel (Jensenius et al., 2004). Dengue is a tropical infectious disease, transmitted by *Aedes aegypti* and *Aedes albopictus*, which has spread globally by infected travelers becoming a new infection source. Leishmaniasis rapidly spread in developed and non-epidemic countries over the last two decades of the 20th century, for which one important factor was rapidly growing international travel (Antinori et al., 2005; Pavli et al., 2010; Pérez-Ayala et al., 2009). Furthermore, global crowd-gathering events, such as the Olympic Games, World Expo or World Cup, not only increase the number of travel destinations, but also attract many travelers and accordingly increase the disease risk. For instance, according to evaluation of cumulative disease trend from the Geosentinel Surveillance Network, the Beijing Olympic Games was considered to have potential risks for disease outbreaks.

2.1.2 Population migration from rural areas to cities

With the accelerated development of urbanization, a shift in population migration from rural areas to cities and suburbs has changed global patterns of infectious diseases. It is estimated that nearly 40% of the increased urban population in developing countries come from rural areas (Leon, 2008). The “floating population” often suffers poor health care situations and high residential densities, which readily cause infectious disease outbreaks and further spread disease during large population movements. Taking China as an example, there were only 6.57 million who were considered floating in the third census, representing 0.65% of the total population. In the fifth census in 2000 and a 1% population sampling survey in 2005, the floating population reached 121 million and 147 million, respectively, constituting

9.55% and 11.26% of total population. During the SARS event in 2003 and influenza A (H1N1) pandemic in 2009, the floating population was the vulnerable group, easily susceptible to infection and difficult to monitor (Zeng et al., 2009; Zeng, 2009). Research on health surveys of the floating population in Beijing indicated that there was a higher risk of disease and greater mental stress for people migrating from rural areas to cities than for other groups (Chen, 2011). Similar surveys showed that the incidence of viral hepatitis, syphilis, measles, dysentery, mumps and infectious diarrhea in floating populations is higher than in local residents (Yang et al., 2007). China’s Ministry of Health, the WHO and UNICEF conducted a baseline survey of children’s health care conditions in Hangzhou and Beijing at the end of 2006. The survey showed that the incidence of diarrhea in children in the floating population was 16.5% and 13.3%, and that of cough was 34.2% and 30.4% in the respective cities. These rates are all higher than those in poor rural areas in 1998 (Huang et al., 2008). There were similar situations in other developing countries, such as Africa, Asia, and Latin America. Outbreaks of leishmaniasis, which used to be confined to rural areas, occurred in many cities as a result of tremendous rural population immigration (Jeronimo et al., 1994; Werneck et al., 2002). Other forms of cutaneous leishmaniasis broke out in densely populated cities in western and central Asia (Ashford, 2000). In recent years, cholera outbreak in the Tanzanian capital of Dar es Salaam has resulted from large floating poor populations and outdated health care support facilities (Penrose et al., 2010).

2.2 Rapid urbanization

Urbanization and health is another focus of current research on socioeconomic factors, and it is one of six research issues of global environmental change and health in the Earth System Science Partnership (ESSP; Confalonieri et al., 2006). Since the beginning of the 20th century, England became the first country with urban population exceeding rural population; by 2007, over half the global population was living in cities. Moreover, according to a UN forecast, urban population in 2050 will reach 6.3 billion (Alirol et al., 2011). The 20th and 21st centuries represent an important stage of urbanization, and an era with obvious problems of infectious disease and health in cities. With urbanization development, cities expand outward, floating populations increase and urban populations concentrate. Rapid urbanization is often accompanied by poverty and deterioration of living environment, and people’s needs exceed the service capacity. The shift in population from rural to urban has been altering global patterns of infectious diseases and mortality (Hay et al., 2005).

2.2.1 Lagging urban infrastructure

The rapid expansion of cities has greatly modified the living

environment for various sources of infection. Sudden changes of environment increase biological attacks and the possibility of infection events, thus augmenting the probability of spreading infectious disease (Wu et al., 2010). Moreover, poor infrastructure, water supply, drainage, and sewage treatment systems facilitate breeding for some vectors (Sutherst, 2004). For example, rapid urbanization caused a global outbreak of dengue fever in the late 20th century. Su et al. (2005) found that urbanization may change types and dominance of major vectors of dengue fever. In addition, Lin et al. (2005) believed that urbanization may cause vectors to breed indoors. Water pollution caused by urban sewage often provides good breeding areas for *Culex*, causing outbreaks of diseases such as lymphatic filariasis (Maciel et al., 1996), systemic lupus erythematosus, Rift Valley fever and other diseases. Taking China as an example, in the next 20 years there will be 400 million peasants migrating to cities. This implies huge demand for health care and related facility construction along with potentially huge risks, as inferred from lessons in Asian, African and Latin American countries.

2.2.2 Disease spread risks in suburbs

With the expansion of cities, both developed and developing countries face the same problems, in that city edges or suburbs are often the first to impinge on undeveloped areas, such as forests and wastelands. Humans often lack immunity when moving into a new area or environment. Moreover, human impacts may bring new pathogens and vectors into such areas (McMichael et al., 2006; Ashford, 2000; Charrel et al., 2007; Harrus et al., 2005; Patz et al., 2000). Diseases related to this that have been reported include yellow fever, trypanosomiasis, Kyasanur Forest disease, malaria and leishmaniasis (Patz et al., 2000; Molyneux, 2003).

2.3 Continuous changes in land use

Land use change, including agricultural encroachment, deforestation, road and dam building, wetland transformation, mining and urban expansion, has led to a series of disease outbreaks and changed the transmission pattern of many endemic diseases (Patz et al., 2004). Land use change can influence diseases indirectly by changing the habitats and behavioral habits of wild animals and livestock. This increases the possibility of human exposure to pathogens and vectors, reduces regional biodiversity, increases the dominance of infected species, and provides breeding areas for vector-borne disease hosts.

2.3.1 Deforestation

Deforestation has accelerated since the early 20th century, with 101724 km² of forest cleared each year. Forest disappears gradually at a rate of 0.3%, and tropical forests disappear at 23% annually (Wolfe et al., 2000). This disappearance destroys the original ecosystem, and the land is trans-

formed into pastures, farmland, plantations or wasteland, which are low-biodiversity environments. Further, the formation of many fragmented habitats has a “marginal” (edge) effect, which increases the opportunity for human contact with new pathogens or wild animals (Patz et al., 2004). For example, cases of malaria (Yasuoka et al., 2007), onchocerciasis (Wilson et al., 2002), Lyme disease (Killilea et al., 2008), cutaneous leishmaniasis (Chaves et al., 2008) and other vector-borne diseases and corresponding vector populations are rising. Population dominance of vectors in forests is also changing. With the effects of increased light, enlarged wasteland areas and increased surface ponding, the population of photophilic vectors has increased in forests. Recent studies indicate an increase in cases of SARS, Ebola virus, Nipah virus and some bat-host viral pathogens (Leroy et al., 2005; Looi et al., 2007). There is a growing sense that HIV, falciparum malaria and other zoonotic diseases are results of increased human exposure to wild animals (Keele et al., 2009; Rich et al., 2009).

2.3.2 Artificial waters

Dams, paddy field irrigation and other water conservancy facilities furnish breeding areas for vector-borne infectious disease hosts, and increase risks of expanding the habitats of intermediate hosts for schistosomiasis (Seto et al., 2002; Xu et al., 2004). With population movement, extensive development of new irrigated areas, and dam building, the incidence and geographic distribution of schistosomiasis is shifting (Chitsulo et al., 2000). Neighborhood relationships and hydrologic connectivity has been quantified to assess the effect of inter-village parasitic transport on disease transmission and control (Xu et al., 2006). After completion of the Aswan Dam in Egypt, local *Schistosoma haematobium* was gradually replaced by *Schistosoma mansoni* as a result of ecological changes. The completion of the Jama dams on the Senegal River and a dam on the Bafing River exacerbated an *S. mansoni* epidemic in North Senegal (Southgate et al., 2001). The Three Gorges Dam is also believed to enlarge the snail habitat and further spread schistosomiasis in southern China (Li et al., 2000). Furthermore, changes to surface water bodies can impact the regional ecological environment and dominance of vectors of certain vector-borne diseases. Artificial waters have flooded breeding areas of Simuliidae, which transmit onchocerciasis, but provided breeding areas for *Anopheles* and *Planorbis*, which respectively transmit malaria and schistosomiasis. Dam spillways become new breeding areas of *Simulium*, which have triggered epidemics of onchocerciasis in West Africa.

2.3.3 Large-scale agricultural encroachment

Nearly half of the world's land is used for agricultural production, which consumes two-thirds of fresh water resources (Horriggan et al., 2002). The low biodiversity and fragile ecosystems of agricultural land contribute to the occurrence of vector-borne diseases. In developing countries,

traditional agricultural irrigation and intensive irrigation canals have become ideal habitats for *Culex* and snails. Typical irrigated farming generates breeding areas for *Culex* (in particular *Culex tritaeniorhynchus*, a vector of Japanese encephalitis virus) and snails. In western Kenya, *Anopheles arabiensis* is common during rice growing season, whereas *Anopheles funestus* is common in the mature rice season. *Anopheles gambiae* appears after the rainy season, whereas in the dry season, *Anopheles arabiensis* dominates. Research in Tanzania and Kenya found that excessive agricultural encroachment and resistance vectors are keys for outbreak and reemergence of malaria (Bodker et al., 2000; Shanks et al., 2000).

2.4 Rapid development of transportation

2.4.1 Air transportation

Currently, the speed of cross-regional spread of infectious diseases is faster than at any time in history because of rapid and efficient air transportation, which has been significant in boosting long-distance epidemic transmission. In a study of the SARS event of 2003, Olsen et al. believed that the epidemic was transmitted by SARS patients who were in the incubation period and was spread globally by air transport (Breugelmans et al., 2004; Olsen et al., 2003). Wilder-Smith et al. analyzed records of flights to Singapore and believed that the spread of SARS on airplanes was overestimated (Wilder-Smith, 2003a; 2003b; 2004). Vogt et al. (2006) collected information on the passengers and crew on seven flights heading to the U.S. carrying SARS patients, and the results of their retrospective study indicated that the risk of SARS transmission on aircraft has not been overestimated. Although risk of the spread of SARS by air transport remains controversial, a consensus has been reached that epidemic diseases can be effectively spread in aircraft (Roy et al., 2004). This has attracted attention from scholars for taking epidemic prevention and control measures at airport customs and immigration centers (Wilder-Smith, 2003b; 2004). Similarly, in a study on the 2009 H1N1 pandemic, Khan found that in almost all countries accessible to Mexicans, confirmed cases of H1N1 have emerged, and the rate of spread is astonishingly high (Khan et al., 2009). Based on an investigation of H1N1 cases on a airplane flight, Baker found that close contact with symptomatic patients poses a higher risk of infection. After the passengers departed the aircraft, subsequent exposure measurement became inefficient and difficult (Baker et al., 2010). Thus, in-flight measurement of the potential risks of H1N1 transmission was proposed (Wagner et al., 2009). As an efficient means for spreading infectious diseases, air transport is also of concern regarding its influence on risks of transmission of other infectious diseases, including tuberculosis (Abubakar, 2010; Dowdall et al., 2010; Kornlyo-Duong et al., 2010), malaria (Bradley, 1989; Tatem et al., 2006), plague (Pascali, 1982), yellow fever

(Oliva, 1979), cholera (Rondle et al., 1978), dengue fever, Norwalk virus (Kirking et al., 2010), and epidemic meningococcal diseases (Rachael et al., 2009). Modes of transmission during air travel and transport include via droplet transmission, interpersonal contact, vector-borne and zoonotic disease hosts entering the cabin, water and food contamination, and others. There were an estimated 36 billion air travelers in 2010. This means that an outbreak or epidemic of disease anywhere in the world could spread to other regions in only a few hours.

2.4.2 Highway and railway transportation

Highways are the main conduits for interregional and regional traffic. Highways carry more passengers and transport to more destinations than any other mode of transport. Taking China as an example, in 2009 its highways carried 2779081 passengers, more than 100 times the number by water transport and civil aviation combined, and about 18 times that of railway traffic (Bureau of Statistics of China, 2010). H1N1 pandemics spread in cities of all sizes in China, with an initial epidemic stage featuring the spread of imported cases, followed by secondary cases from initial imported cases, and a late stage in which the pandemic spreads from metropolitan areas to small cities and from eastern to western areas (Zeng et al., 2009). In the epidemic process, rapid movement of large populations is mainly realized through road traffic, so this traffic is an important risk factor for the secondary spread of infectious disease in various countries. Further, long-term freight is mainly transported by road traffic. Highway freight volume in 2009 was 21278.34 million tons, accounting for 75.3% of total freight (China Statistics Bureau, 2010). Some zoonoses are readily diffused to larger areas via shipping. For example, highly pathogenic avian influenza (HPAI) virus can generate increased human and animal infection in more areas, via agricultural products shipped from other areas. Fang et al. (2009) showed that during a SARS event, the transportation network of highways and railways is important in spreading an epidemic across mainland China. Close correlation between SARS distribution and a ring road in the Beijing urban area has been reported (Wang et al., 2006; Wang et al., 2008). Fang and Cao found a close association between HPAI outbreaks and minimum distance to the nearest national highway in China (Fang et al., 2008; Cao et al., 2010). However, results of similar studies in Europe did not show this association (Si et al., 2010).

2.5 Global trade and economy

At the start of the 20th century, with development of the transport network, strengthening of international cooperation and division of labor, the rise of the World Trade Organization (WTO) and other trade organizations, global trade increased tremendously. International merchandise trade increased 3- to 4-fold from 1980 to 2000, with the

main contribution from Asia (up to a 5-fold increase) (Sutherst, 2004). For nearly a century, Asia has been considered the epidemic focus of new and old infectious diseases, with repeated devastating events (Guan et al., 2002; Li et al., 2004; Smith et al., 2006). Frequent economic and trade exchange increases the possibility of infectious disease outbreaks. Both Asian and other countries are likely to face the problem of long-distance spread of pathogens and diffusion of new viruses along livestock trade routes.

2.5.1 *Aquaculture industry*

Since 1997, HPAI H5N1 epidemics have broken out in poultry in Asia (Li et al., 2004; Smith et al., 2006; Chen et al., 2006), and this has resulted in enormous damage to the poultry industry in Southeast Asia (Gilbert et al., 2008). According to WHO statistics, as of March 2011, HPAI H5N1 has spread to more than 63 countries, resulting in 535 people infected and 316 deaths (http://www.who.int/csr/disease/avian_influenza/country/cases_table_2011_03_25/en/index.html). According to technical documents published by the United Nations Environment Programme Convention on Migratory Species (UNEP/CMS), the source of HPAI H5N1 was intensively-fed poultry, and virus mutation resulted from these extreme rearing environments. Poultry production in Asian countries can be classified into four categories, namely, backyard farming, free range, greenhouse cultivation and enclosure breeding. According to an investigation by Songserm et al. (2006) in Thailand, all the above methods, except for enclosure breeding, pose risks of H5N1 viral infection. A follow-up study by Biswas and Gilbert showed a higher risk of infection in backyard farming and free range breeding, and infected migratory birds and water containing the virus may have infected poultry (Biswas et al., 2009; Gilbert et al., 2006). According to statistics, about 80% of poultry production is by backyard farming in Asian and African countries (Aini, 1990; Permin et al., 2002). Through 2005, there were 140 million birds infected with the H5N1 virus, resulting in about a \$10 billion loss in Asian countries (Gilbert et al., 2008; Food and Agriculture Organization, 2005). Effective epidemic control methods include large-scale containment killings. As of 2010, more than 260 million chickens and other poultry-birds have been killed worldwide, producing economic losses totaling about \$20 billion (<http://www.fao.org/news/story/en/item/41287/icode/>). The poultry industry in China has an annual output of nearly 15 billion birds, including about 5.6 billion chickens, 760 million ducks and 300 million geese (Martin et al., 2011). These are all sensitive to HPAI H5N1 (Keele et al., 2009), and the mortality rate is near 100% upon infection (Alexander, 2000, 2007; Webster et al., 1987). Losses from an epidemic outbreak over a large area would be immeasurable.

2.5.2 *Disease spread risks of international trade*

Globally, there were about 350 million live plants and wild-

life species traded yearly (Karesh et al., 2007). Approximately 4 million live birds are transported annually, and most of these are from Southeast Asian countries (Karesh et al., 2005). It is generally agreed that the main mode of transmission for avian flu is via various forms of poultry trade, which includes legitimate trade and illegal transactions (Alexander, 2000; Capua et al., 2006; Olsen et al., 2006; Van et al., 2005; Wang et al., 2006). Studies have shown that the major risk of avian flu in Europe is from bird migration, and the main risk in Asia is from the poultry trade (Kilpatrick et al., 2006). There are greater risks in wildlife trade, including induced epidemic outbreaks in areas with crowds of people, livestock epidemics, and strong negative impacts on international economies, trade, the livestock industry, and national wildlife ecosystems (Karesh et al., 2005). Since the mid-1990s, outbreaks of infectious diseases, including mad cow disease (BSE), foot-and-mouth disease (FMD), avian flu, swine flu and others, have caused losses of about \$80 billion to the global economy and trade industries (Karesh et al., 2005). At the beginning of 2003, the UN Food and Agriculture Organization stated that more than a third of meat traded was embargoed worldwide. Wild animals are considered the source of more than 70% of emerging infectious diseases (Kuiken et al., 2005), and diffusion of a series of new viruses has been triggered by various forms of trade. HIV is believed to be derived from human feeding on non-human primates (Gao et al., 1999). The Ebola virus is transmitted via human contact with great apes (Leroy et al., 2004), SARS coronavirus is induced by international trade in small carnivores (Bell et al., 2004), and monkeypox in the U.S. originates from imports of exotic pet rodents from Ghana (Guarner et al., 2004). Chytridiomycosis, derived from the African clawed frog that has been commercially traded, has eliminated nearly 30% of amphibians worldwide (Weldon et al., 2004).

2.6 **Other human activities**

Emergence and reemergence of a given type of infectious disease may be related to socioeconomic pressures, such as collapse of the public health system, lack of disease control strategies, and disturbances resulting from ecological and demographic changes (Taubes, 1997; Gubler, 1998; Reiter, 2001). Other human activities, including population growth, intensive agricultural development and changes in vaccine technology, are likely to alter the evolution and spread of influenza virus (Vandegrift et al., 2010). In addition, population age and size, and demographic components, such as numbers of workers or students, can affect the spread of influenza (Stefano et al., 2010). Influenza is closely related to age structure of the population. Avian and seasonal flu have different features across the population age distribution. Specifically, victims tend to be the young and the elderly. Children and young adults are major flu victims. Statistics on influenza A (H5N1) virus infection in February 2008

indicated that the average age of cases is 18 years.

3 Discussions and conclusions

3.1 Discussion

3.1.1 *Interaction between natural and human factors*

Natural factors affect human activities; climate change can directly influence human behaviors such as by seasonal and other migration, and summer and winter lifestyles. Human behavior directly affects disease transmission. Seasonal outbreaks of avian influenza in Europe show that people are spending more time on outdoor activities in winter (Halstead, 1996). Alternately, in temperate regions, a growing number of summer gastroenteritis cases in developing countries have been connected with the fact that people prefer to picnic or cook outdoors because of increasingly higher temperatures (Altekruse et al., 1998). Rainfall, especially of the heavy variety, can increase the frequency and intensity of drinking water contamination. Climate can affect water sources and sanitation, water shortages can increase the probability of people using contaminated water sources, and these factors may add to cases of intestinal infectious diseases (Zhang et al., 2008). Human activities also change natural factors, which indirectly influence the occurrence and transmission of infectious diseases. Research (Zell, 2004) shows that the spread of infectious disease is affected by the spread or location change of pathogen-infected hosts or insects, and sometimes by both; the role of human activity cannot be ignored in this process. Tang and colleagues analyzed climate change and health statistics of 45 countries in Africa and 113 countries elsewhere. Their research shows that income level can regulate the negative influence of climate change over an average life span. With increased income, the impact of climate change on average life expectancy is gradually decreased. This impact is greater in African or developing countries than in others (especially developed ones), and the effect of climate change on health is much greater than on economic levels (Tang et al., 2010).

3.1.2 *Human adaptation activities and infectious diseases*

Long-distance travel, such as to escape heat or cold, acts as an efficient carrier of pathogens. Upon entering a new area or environment, people often become vulnerable to pathogens because of a lack of immunity. In addition, human intervention introduces new pathogens and vectors to the area, disturbs ecological systems, and causes certain endemic diseases to become epidemic across continents and oceans. Population migration caused by extreme weather or meteorological disasters is often accompanied by infectious disease outbreaks. For example, heavy rain and storms can contaminate drinking water sources, expand the habitats of vector-borne infectious disease hosts, or increase frequency

of contact with humans. During weather-related disasters, low population immunity levels and poor health conditions, along with the gathering of large numbers of people, readily cause outbreaks of epidemics and infectious diseases. It is believed that climate change can put tremendous pressure on worldwide supplies of food and clean water, particularly in Africa and Southeast Asia. This would further stimulate farmland encroachment, deforestation, dam building and other activities, thereby reducing local biological diversity, destroying the ecological environment, and producing rampant vector-borne diseases and zoonosis.

3.2 Conclusions

Under the impetus of global change in both natural and human activities, the occurrence and spread of infectious disease have been altered. Natural factors, especially climatic forcing, influence the outbreak and spread of many infectious diseases. Climate factors, including temperature, precipitation, humidity and sunshine, can affect pathogens, hosts and disease vectors, thereby influencing the occurrence and spread of disease. Furthermore, extreme weather events, such as El Niño/La Niña-Southern Oscillation and the QBO, are related to the transmission of infectious diseases. Droughts, floods and other meteorological disasters are also closely related to the occurrence and spread patterns of various such diseases. The spread of these diseases is associated with land cover type; for example, the first cases of avian influenza are often associated with wetlands and water. Further, vegetation distribution can modify outbreaks, through influencing the food sources of host animals. Many natural factors often work together to affect disease transmission. For example, global warming may significantly impact the hydrosphere via heavy rainfall, floods, storms, heat waves and droughts.

Human activity is an indirect driving force affecting the spread of infectious diseases. Among these activities, international travel and population migration from rural areas to cities are root causes of disease spread. Rapid urbanization accompanied by lagging urban infrastructure, and high risks of infectious disease resulting from urban land use change will alter the pattern of disease and death. Land use changes including agricultural encroachment, deforestation, road and dam building, wetland transformation, mining and urban expansion, have triggered a series of disease outbreaks and changed the transmission pattern of many endemics. Rapid development of aviation, road and railway transportation, not only speeds up the rate of disease spread, but also expands its range. In addition, frequent trade increases the possibility of outbreaks of infectious diseases, and provides the means for long-distance spread of pathogens and diffusion of new pathogens along livestock trade routes. Human activities, including demographic characteristics and health systems, can affect the spread of infectious diseases.

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- Abubakar I. 2010. Tuberculosis and air travel: A systematic review and analysis of policy. *Lancet Infect Dis*, 10: 176–183
- Adhikari D, Chettri A, Barik S K. 2009. Modelling the ecology and distribution of highly pathogenic avian influenza (H5N1) in the Indian subcontinent. *Curr Sci*, 97: 73–78
- Aimone F. 2010. The 1918 influenza epidemic in New York City: A review of the public health response. *Public Health Rep*, 125: 71–79
- Aini I. 1990. Indigenous chicken production in Southeast Asia. *World Poultry Sci J*, 46: 51–57
- Akiev A K, Yemelyanov P E, Labunets N F. 1976. European suslik as a possible carrier of plague in natural foci in eastern Europe. *J Hyg Epidemiol Microbiol Immunol*, 20: 82–90
- Alexander D J. 2000. A review of avian influenza in different bird species. *Vet Microbiol*, 74: 3–13
- Alexander D J. 2007. An overview of the epidemiology of avian influenza. *Vaccine*, 25: 5637–5644
- Alirol E, Getaz L, Stoll B, et al. 2011. Urbanisation and infectious diseases in a globalised world. *Lancet Infect Dis*, 11: 131–141
- Altekruse S F, Swerdlow D L, Wells S J. 1998. Factors in the emergence of food borne diseases. *Vet Clin North Am Food Anim Pract*, 14: 1–15
- Antinori S, Gianelli E, Calattini S, et al. 2005. Cutaneous leishmaniasis: An increasing threat for travellers. *Clin Microbiol Infect*, 11: 343–346
- Arguin P M, Marano N, Freedman D O. 2009. Globally mobile populations and the spread of emerging pathogens. *Emerg Infect Dis*, 15: 1713–1714
- Ashford R W. 2000. The leishmaniasis as emerging and re-emerging zoonoses. *Int J Parasitol*, 30: 1269–1281
- Baker M G, Thornley C N, Mills C, et al. 2010. Transmission of pandemic A/H1N1 2009 influenza on passenger aircraft: Retrospective cohort study. *Br Med J*, 340: c2424
- Baylis M, Mellor P S, Meiswinkel R. 1999. Horse sickness and ENSO in South Africa. *Nature*, 397: 574
- Beggs P J. 2004. Impacts of climate change on aeroallergens: Past and future. *Clin Exp Allergy*, 34: 1507–1513
- Bell D, Robertson S, Hunter P R. 2004. Animal origins of SARS coronavirus: Possible links with the international trade in small carnivores. *Philos Trans R Soc Lond B Biol Sci*, 359: 1107–1114
- Bentham G, Langford I H. 2001. Environmental temperatures and the incidence of food poisoning in England and Wales. *Int J Biometeorol*, 45: 22–26
- Binder S, Levitt A M, Sacks J J, et al. 1999. Emerging infectious diseases: Public health issues for the 21st century. *Science*, 284: 1311–1313
- Biswas P K, Christensen J P, Ahmed S S U, et al. 2009. Debnath. Risk for infection with highly pathogenic avian influenza virus (H5N1) in backyard chickens, Bangladesh. *Emerg Infect Dis*, 15: 1931–1936
- Bodker R, Kisinza W, Malima R, et al. 2000. Resurgence of malaria in the Usambara mountains, Tanzania, an epidemic of drug-resistant parasites. *Glob Change Hum Health*, 1: 134–153
- Bouma M J, Dye C, Van der Kaay H J. 1996. Falciparum malaria and climate change in the northwest frontier province of Pakistan. *Am J Trop Med Hyg*, 55: 131–137
- Bouma M J, Sondorp H E, Van der Kaay H J. 1994. Climate change and periodic epidemic malaria. *Lancet*, 343: 1440
- Bradley D J. 1989. Current trends in malaria in Britain. *J R Soc Med*, 82(Suppl 17): 8–13
- Breugelmans J G, Zucs P, Porten K, et al. 2004. SARS transmission and commercial aircraft. *Emerg Infect Dis*, 10: 1502–1503
- Brown I H. 2010. Summary of avian influenza activity in Europe, Asia, and Africa, 2006–2009. *Avian Dis*, 54: 187–193
- Cao C X, Xu M, Chang C Y, et al. 2010. Risk analysis for the highly pathogenic avian influenza in mainland China using meta-modeling. *Chin Sci Bull*, 5: 4165–4175
- Capua I, Marangon S. 2006. Control of avian influenza in poultry. *Emerg Infect Dis*, 12: 1319–1324
- Cazorla C, Enea M, Lucht F, et al. 2003. First isolation of Rickettsia slovaca from a patient, France. *Emerg Infect Dis*, 9: 135
- Charrel R N, de Lamballerie X, Raoult D. 2007. Chikungunya outbreaks—The globalization of vectorborne diseases. *N Engl J Med*, 356: 769–771
- Chaves L F, Cohen J M, Pascual M, et al. 2008. Social exclusion modifies climate and deforestation impacts on a vector-borne disease. *PLoS Negl Trop Dis*, 2: e176
- Checkley W, Epstein L D, Gilman R H, et al. 2000. Effects of El Niño and ambient temperature on hospital admissions for diarrhoeal diseases in Peruvian children. *Lancet*, 355: 442–450
- Chen H X, Li Q L. 1999. The effect of hemorrhagic fever with renal failure caused by flood and waterlogging in China (in Chinese). *Chin Pub Health*, 15: 666–667
- Chen H X. 1999. The impact and control measures of flood and drought disasters on hemorrhagic fever with renal syndrome (in Chinese). *Chin Pub Health*, 15: 665
- Chen H, Smith G J D, Li K S, et al. 2006. Establishment of multiple sub-lineages of H5N1 influenza virus in Asia: Implications for pandemic control. *Proc Natl Acad Sci USA*, 103: 2845–2850
- Chen J. 2011. Internal migration and health: Re-examining the healthy migrant phenomenon in China. *Soc Sci Med*, 72: 1294–1301
- Chen S L, Zhu H B. 1998. The impact on the diarrhoea epidemics of the floods based on logistic regression (in Chinese). *J Environ Health*, 15: 166–168
- Cheng F, Jiao M X, Zheng J S, et al. 1999. The influence of flood disaster on infectious diseases and prevention measures in 1998 in Hubei Province. *Chin Pub Health* (in Chinese), 15: 510–511
- Chitsulo L, Engels D, Montresor A, et al. 2000. The global status of schistosomiasis and its control. *Acta Trop*, 77: 41–51
- Chomel B B, Belotto A, Meslin F X. 2007. Wildlife, exotic pets, and emerging zoonoses. *Emerg Infect Dis*, 13: 6–11
- Confalonieri A, McMichael A. 2006. Global environmental change and human health. *ESSP Report No. 4*
- Davis S, Begon M, De Bruyn L, et al. 2004. Predictive thresholds for plague in Kazakhstan. *Science*, 304: 736–738
- Debono R, Vincenti K, Calleja N. 2012. Risk communication: Climate change as a human-health threat, a survey of public perceptions in Malta. *Eur J Public Health*, 22: 144–149
- Dowdall N P, Evans A D, Thibeault C. 2010. Air Travel and TB: An airline perspective. *Travel Med Infect Dis*, 8: 96–103
- D'Souza R M, Becker N G, Hall G. 2004. Does ambient temperature affect food-borne disease? *Epidemiology*, 15: 86–92
- Duane J G, Paul R, Kristie L E, et al. 2001. Climate variability and change in the United States: Potential impacts on vector-borne and rodent-borne diseases. *Environ Health Perspect*, 109: 223–233
- Engelthaler D M, Mosley D G, Cheek J E, et al. 1999. Climatic and environmental patterns associated with hantavirus pulmonary syndrome, Four Corners region, United States. *Emerg Infect Dis*, 5: 87–94
- Epstein P R. 1999. Climate and health. *Science*, 285: 347–348
- Ergonul O. 2006. Crimean-Congo haemorrhagic fever. *Lancet Infect Dis*, 6: 203–214
- Fang L Q, De Vlas S J, Feng D, et al. 2009. Geographical spread of SARS in mainland China. *Trop Med Int Health*, 14: 14–20
- Fang L Q, De Vlas S J, Liang S, et al. 2008. Environmental factors contributing to the spread of H5N1 avian influenza in mainland China. *PLoS ONE*, 3: e2268
- Feng J X. 2000. An investigation report about diarrhea outbreak caused by severe drought (in Chinese). *Anthol Med*, 19: S118–119
- Ferguson C M, Croke B F, Beatson P J, et al. 2007. Development of a process-based model to predict pathogen budgets for the Sydney drinking water catchment. *J Water Health*, 5: 187–208
- Food and Agriculture Organization. 2005. A Global Strategy for the Progressive Control of Highly Pathogenic Avian Influenza (HPAI). Rome: Food and Agriculture Organization
- Freedman D O, Weld L H, Kozarsky P E, et al. 2006. Spectrum of disease and relation to place of exposure among ill returned travelers. *N Engl*

- J Med, 354: 119–130
- Gao F, Bailes E, Robertson D L, et al. 1999. Origin of HIV-1 in the chimpanzee *Pan troglodytes troglodytes*. *Nature*, 397: 436–441
- Garrett L. 1996. The return of infectious disease. *Foreign Affairs*, 75: 66–79
- Gibbs E P, Anderson T C. 2010. Equine and canine influenza: A review of current events. *Anim Health Res Rev*, 11: 43–51
- Gilbert M, Xiao X, Pfeiffer D U, et al. 2008. Mapping H5N1 highly pathogenic avian influenza risk in Southeast Asia. *Proc Natl Acad Sci USA*, 105: 4769–4774
- Gilbert M, Chaitaweesub P, Parakamawongsa T, et al. 2006. Free-grazing ducks and highly pathogenic avian influenza, Thailand. *Emerg Infect Dis*, 12: 227–234
- Gonzalez-Scarano F, Nathanson N. 1996. Bunyaviridae. In: Fields B N, Knipe D M, Howley P M, eds. *Virology*. 3rd ed. Philadelphia: Lipincott-Raven Publishers.
- Gossling S. 2002. Global environmental consequences of tourism. *Global Environ Change*, 12: 283–302
- Gould E A, Higgs S. 2009. Impact of climate change and other factors on emerging arbovirus diseases. *Trans R Soc Trop Med Hyg*, 103: 109–121
- Guan Y, Peiris J S, Lipatov A S, et al. 2002. Emergence of multiple genotypes of H5N1 avian influenza viruses in Hong Kong SAR. *Proc Natl Acad Sci USA*, 99: 8950–8955
- Guarner J, Johnson B J, Paddock C D, et al. 2004. Monkeypox transmission and pathogenesis in prairie dogs. *Emerg Infect Dis*, 10: 426–431
- Gubler D J. 1998. Resurgent vector-borne diseases as a global health problem. *Emerg Infect Dis*, 4: 442–450
- Guerrant R L, Blackwood B L. 1999. Threats to global health and survival: The growing crises of tropical infectious diseases—Our “unfinished agenda”. *Clin Infect Dis*, 28: 966–986
- Hales S, Weinstein P, Woodward A. 1996. Dengue fever epidemics in the South Pacific: Driven by El Niño Southern Oscillation? *Lancet*, 348: 1664–1665
- Hall G V, D’Souza R M, Kirk M D. 2002. Foodborne disease in the new millennium: Out of the frying pan and into the fire? *Med J Aust*, 177: 614–618
- Halstead S B. 1996. Human factors in emerging infectious disease. *WHO EMRO*, 2: 21–29
- Harrus V, Baneth G. 2005. Drivers for the emergence and re-emergence of vector-borne protozoal and bacterial diseases. *Int J Parasitol*, 35: 1309–1318
- Harvell C D, Kim K, Burkholder J M, et al. 1999. Emerging marine diseases—Climate links and anthropogenic factors. *Science*, 285: 1505–1510
- Hay S I, Guerra C A, Tatem A J, et al. 2005. Urbanization, malaria transmission and disease burden in Africa. *Nat Rev Microbiol*, 3: 81–90
- Hill D R. 2006. The burden of illness in international travelers. *N Engl J Med*, 354: 115–117
- Hjelle B, Glass G E. 2000. Outbreak of hantavirus infection in the Four Corners region of the United States in the wake of the 1997–1998 El Niño–Southern Oscillation. *J Infect Dis*, 181: 1569–1573
- Hogrefe C, Lynn B, Civerolo K, et al. 2004. Simulating changes in regional air pollution over the eastern United States due to changes in global and regional climate and emissions. *J Geophys Res*, 109: 2627–2638
- Horrihan L, Lawrence R S, Walker P. 2002. How sustainable agriculture can address the environmental and human health harms of industrial agriculture. *Environ Health Perspect*, 110: 445–456
- Huang A Q, Pan X P, Du Q, et al. 2008. An analysis of morbidities and risk factors of diarrhea and cough among urban migrant children (in Chinese). *Chin J Woman Child Health Res*, 19: 1–3
- Hunter P R. 2003. Climate change and waterborne and vector-borne disease. *J Appl Microbiol*, 94: 37S–46S
- Intergovernmental Panel on Climate Change. 1996. In: Houghton J T, Meira Filho L G, Callander B A, et al., eds. *Climate Change 1995: The Science of climate change*. Cambridge: Cambridge University Press.
- Intergovernmental Panel on Climate Change. 2001. In: McCarthy J J, Canziani O F, Leary N A, et al., eds. *Climate Change 2001: Impacts, Adaptation and Vulnerability*. Chapter 9: Human Health. Contribution of WorkingGroup II to the Third Assessment Report of the Intergovernmental Panel on Climate Change (IPCC). Cambridge: Cambridge University Press
- Intergovernmental Panel on Climate Change. 2007. In: Parry M, Canziani O, Palutikof J, et al., eds. *Climate Change 2007: Impacts, Adaptation and Vulnerability*. Cambridge: Cambridge University
- Jensenius M, Fournier P E, Raoult D. 2004. Rickettsioses and the international traveler. *Clin Infect Dis*, 39: 1493–1499
- Jeronimo S M, Oliveira R M, Mackay S, et al. 1994. An urban outbreak of visceral leishmaniasis in Natal, Brazil. *Trans R Soc Trop Med Hyg*, 88: 386–388
- Jones K E, Patel N G, Levy M A, et al. 2008. Global trends in emerging infectious diseases. *Nature*, 451: 990–993
- Karesh W B, Cook R A, Bennett E L, et al. 2005. Wildlife trade and global disease emergence. *Emerg Infect Dis*, 11: 1000–1002
- Karesh W B, Cook R A, Gilbert M, et al. 2007. Implications of wildlife trade on the movement of avian influenza and other infectious diseases. *J Wildl Dis*, 43: 55–59
- Kausrud K L, Viljugrein H, Frigessi A, et al. 2007. Climatically driven synchrony of gerbil populations allows large-scale plague outbreaks. *Proc Biol Sci*, 274: 1963–1969
- Keele B F, Jones J H, Terio K A, et al. 2009. Increased mortality and AIDS-like immunopathology in wild chimpanzees infected with SIVcpz. *Nature*, 460: 515–519
- Khan K, Arino J, Hu W, et al. 2009. Spread of a novel influenza A (H1N1) virus via global airline transportation. *N Engl J Med*, 361: 212–214
- Khasnis A, Nettleman M. 2005. Global warming and infectious disease. *Arch Med Res*, 39: 689–696
- Killalea M E, Swee A, Lane R S, et al. 2008. Spatial dynamics of Lyme disease: A review. *Eco Health*, 5: 167–195
- Kilpatrick A M, Chmura A A, Gibbons D W, et al. 2006. Predicting the global spread of H5N1 avian influenza. *Proc Natl Acad Sci USA*, 103: 19368–19373
- Kirking H L, Cortes J, Burrer S, et al. 2010. Likely transmission of norovirus on an airplane, October 2008. *Clin Infect Dis*, 50: 1216–1221
- Kornylo-Duong K, Kim C, Cramer E H, et al. 2010. Three air travel-related contact investigations associated with infectious tuberculosis, 2007–2008. *Travel Med Infect Dis*, 8: 120–128
- Kovats R S, Edwards S J, Hajat S, et al. 2004. The effect of temperature on food poisoning: A time-series analysis of salmonellosis in ten European countries. *Epidemiol Infect*, 132: 443–453
- Kuhn K, Campbell-Lendrum D, Haines A, et al. 2005. Using climate to predict infectious disease epidemics. *World Health Organization*
- Kuiken T, Leighton F A, Fouchier R A, et al. 2005. Public health. Pathogen surveillance in animals. *Science*, 309: 1680–1681
- Lafferty K D. 2009. The ecology of climate change and infectious diseases. *Ecology*, 90: 888–900
- Leon D A. 2008. Cities, urbanization and health. *Int J Epidemiol*, 37: 4–8
- Leroy E M, Kumulungui B, Pourrut X, et al. 2005. Fruit bats as reservoirs of Ebola virus. *Nature*, 438: 575–576
- Leroy E M, Rouquet P, Formenty P, et al. 2004. Multiple Ebola virus transmission events and rapid decline of central African wildlife. *Science*, 303: 387–390
- Li K S, Guan Y, Wang J, et al. 2004. Genesis of a highly pathogenic and potentially pandemic H5N1 influenza virus in eastern Asia. *Nature*, 430: 209–213
- Li Y S, Sleight A C, Ross A G, et al. 2000. Epidemiology of *Schistosoma japonicum* in China: Morbidity and strategies for control in the Dongting Lake region. *Int J Parasitol*, 30: 273–281
- Liang L, Xu B, Chen Y, et al. 2010. Combining spatial-temporal and phylogenetic analysis approaches for improved understanding on global H5N1 transmission. *PLoS ONE*, 5: e13575.
- Lin L H, Chen W J, Ma Y H, et al. 2000. Analysis on relationship between characteristic of bleeding in house and dengue epidemic. *Chin Publ Health*, 16: 610
- Linthicum K J, Anyamba A, Tucker C J, et al. 1999. Climate and satellite indicators to forecast Rift Valley fever epidemics in Kenya. *Science*, 285: 397–400
- Looi L M, Chua K B. 2007. Lessons from the Nipah virus outbreak in

- Malaysia. *Malays J Pathol*, 29: 63–67
- Luber G, Prudent N. 2009. Climate change and human health. *Trans Am Clin Climatol Assoc*, 120: 113–117
- MacDonald G. 1957. *The Epidemiology and Control of Malaria*. London: Oxford University Press
- Maciel A, Rocha A, Marzochi K B F, et al. 1996. Epidemiology study of bancroftian filariasis in Recife, northeastern of Brazil. *Mem Inst Oswaldo Cruz*, 91: 449–455
- Marano C, Freedman D O. 2009. Global health surveillance and travelers' health. *Curr Opin Infect Dis*, 22: 423–429
- Martin V, Pfeiffer D U, Zhou X, et al. 2011. Spatial distribution and risk factors of highly pathogenic avian influenza (HPAI) H5N1 in China. *PLoS Pathog*, 7: e1001308
- Mavroidi N. 2008. Transmission of zoonoses through immigration and tourism. *Vet Ital*, 44: 651–666
- McMichael A J, Haines A, Slooff R, et al. 1996. Climate change and human health. WHO. Geneva
- McMichael A J, Woodruff R E, Hales S. 2006. Climate change and human health: Present and future risks. *Lancet Infect Dis*, 367: 859–869
- McMichael A J. 2001. Impact of climatic and other environmental changes on food production and population health in the coming decades. *Proc Nutr Soc*, 60: 195–201
- Mellor P S, Leake C J. 2000. Climatic and geographic influences on arboviral infections and vectors. *Rev Sci Tech OIE*, 19: 41–54
- Molyneux D H. 2003. Common themes in changing vector-borne disease scenarios. *Trans R Soc Trop Med Hyg*, 97: 129–132
- Morens D M, Folkers G K, Fauci A S. 2004. The challenge of emerging and re-emerging infectious diseases. *Nature*, 430: 242–249
- Morris J, Lewin P, Smith C W, et al. 1982. Ciguatera fish poisoning: Epidemiology of the disease on St. Thomas, U.S. Virgin Islands. *Am J Trop Med Hyg*, 31: 574–578
- National Statistics Bureau. 2010. *China Statistical Yearbook 2010* (in Chinese). Beijing: China Statistics Press
- Nichol S T, Spiropoulou C F, Morozunov S, et al. 1993. Genetic identification of a novel hantavirus associated with an outbreak of acute respiratory illness in the southwestern United States. *Science*, 262: 615–618
- Oliva F. 1979. Current status of yellow fever in the world and the importance of aircraft in its possible spread. *Minerva Med*, 70: 2573–2582
- Olsen B, Munster V J, Wallensten A, et al. 2006. Global patterns of influenza A virus in wild birds. *Science*, 312: 384–388
- Olsen S J, Chang H L, Cheung T Y, et al. 2003. Transmission of the severe acute respiratory syndrome on aircraft. *N Engl J Med*, 349: 2416–2422
- Ostroff S M, Kozarsky P. 1998. Emerging infectious diseases and travel medicine. *Infect Dis Clin North Am*, 12: 231–241
- Pan H M, Cheng D M, Shi Y N, et al. 2003. The influence of flood disasters to the leptospirosis epidemic (in Chinese). *Chin J Nat Med*, 5: 73–75
- Parry M L, Rosenzweig C, Iglesias A, et al. 2004. Effects of climate change on global food production under SRES emissions and socio-economic scenarios. *Glob Environ Change*, 4: 53–67
- Pascali C. 1982. The role of aircraft in the epidemiology of plague. *Minerva Med*, 73: 2083–2088
- Patz J A, Daszak P, Tabor G M, et al. 2004. Unhealthy landscapes: Policy recommendations on land use change and infectious disease emergence. *Environ Health Perspect*, 112: 1092–1098
- Patz J A, Epstein P, Burke T, et al. 1995. Global climate change and emerging infectious diseases. *JA MA*, 275: 217–223
- Patz J A, Graczyk T K, Geller N, et al. 2000. Effects of environmental change on emerging parasitic diseases. *Int J Parasitol*, 30: 1395–1405
- Pavli A, Maltezou H C. 2010. Leishmaniasis, an emerging infection in travelers. *Int J Infect Dis*, 14: e1032–e1039
- Penrose K, de Castro M C, Werema J, et al. 2010. Informal urban settlements and cholera risk in Dar es Salaam, Tanzania. *PLoS Negl Trop Dis*, 4: e631
- Pérez-Ayala A, Norman F, Pérez-Molina J A, et al. 2009. Imported leishmaniasis: A heterogeneous group of diseases. *J Travel Med*, 16: 395–401
- Permin A, Pedersen G. 2002. The need for a holistic view on disease problems in free-range chickens. In: *Characteristics and Parameters of Family Poultry Production in Africa*. Vienna: IAEA. 9–13
- Population Census Office of the State Council, The National Bureau of Statistics Population Statistics Division of Science and Technology and Society. 1993. *China-National Population Census 1990* (in Chinese). Beijing: China Statistics Press
- Population Census Office of the State Council, The National Bureau of Statistics Population Statistics Division of Science and Technology and Society. 2002. *China-National Population Census 2000* (in Chinese). Beijing: China Statistics Press
- Rachael T, Schubert K, Hellenbrand W, et al. 2009. Risk of transmitting meningococcal infection by transient contact on aircraft and other transport. *Epidemiol Infect*, 137: 1057–1061
- Reiter P. 2001. Climate change and mosquito-borne disease. *Environ Health Perspect*, 109: 141–161
- Ren J, Gu L L, Liu H, et al. 2005. Study on a monitoring program regarding leptospirosis in some fore-and-after flood-affected areas along large rivers in Anhui province (in Chinese). *Chin J Epidemiol*, 26: 690–693
- Rich S M, Leendertz F H, Xu G, et al. 2009. The origin of malignant malaria. *Proc Natl Acad Sci USA*, 106: 14902–14907
- Rogers D J, Randolph S E. 2006. Climate change and vector-borne diseases. *Adv Parasit*, 62: 345–81
- Rondle C J, Ramesh B, Krahn J B, et al. 1978. Cholera: Possible infection from aircraft effluent. *J Hyg (Lond)*, 91: 361–371
- Roy C J, Milton D K. 2004. Airborne transmission of communicable infection—The elusive pathway. *N Engl J Med*, 350: 1710–1712
- Shanks G D, Biomndo K, Hay S I, et al. 2000. Changing patterns of clinical malaria since 1965 among a tea estate population located in the Kenyan highlands. *Trans R Soc Trop Med Hyg*, 94: 253–255
- Si Y, Wang T, Skidmore A K, et al. 2010. Environmental factors influencing the spread of the highly pathogenic avian influenza H5N1 virus in wild birds in Europe. *Ecol Soc*, 15: 26
- Smith G J, Fan X H, Wang J, et al. 2006. Emergence and predominance of an H5N1 influenza variant in China. *Proc Natl Acad Sci USA*, 103: 16936–16941
- Songserm T, Jam-on R, Sae-Heng N, et al. 2006. Domestic ducks and H5N1 influenza epidemic, Thailand. *Emerg Infect Dis*, 12: 575–581
- Southgate V R, Tchuem Tchuenté L A, Sène M, et al. 2001. Studies on the biology of schistosomiasis with emphasis on the Senegal river basin. *Mem Inst Oswaldo Cruz*, 96(Suppl): 75–78
- Stefano M, Marco A. 2010. The role of population heterogeneity and human mobility in the spread of pandemic influenza. *Proc Biol Sci*, 277: 557–565
- Stenseth N C, Samia N I, Viljugrein H, et al. 2006. Plague dynamics are driven by climate variation. *Proc Natl Acad Sci USA*, 103: 13110–13115
- Su A F, Pei Z C, Fu J C, et al. 2005. Analysis of distribution and population density changes of *Aedes aegypti* the transmission vector of dengue fever in Haikou city. *China Trop Med*, 5: 1394–1395
- Sutherst R W. 2004. Global change and human vulnerability to vector-borne diseases. *Clin Microbiol Rev*, 17: 136–173
- Swaminathan A, Torresi J, Schlagenhauf P, et al. 2009. A global study of pathogens and host risk factors associated with infectious gastrointestinal disease in returned international travellers. *J Infect*, 59: 19–27
- Tang J W, Shetty N, Lam T T. 2010. Features of the new pandemic influenza A/H1N1/2009 virus: Virology, epidemiology, clinical and public health aspects. *Curr Opin Pulm Med*, 16: 235–241
- Tatem A J, Rogers D J, Hay S I. 2006. Estimating the malaria risk of African mosquito movement by air travel. *Malar J*, 5: 57
- Taubes G. 1997. Global warming: Apocalypse not. *Science*, 278: 1004–1006
- Taylor L H, Latham S M, Woolhouse M E. 2001. Risk factors for human disease emergence. *Philos Trans R Soc Lond B Biol Sci*, 356: 983–989
- Thomas K M, Charron D F, Waltner-Toews D, et al. 2006. A role of high impact weather events in waterborne disease outbreaks in Canada,

- 1975–2001. *Int J Environ Health Res*, 16: 167–180
- Tong S L, Lu Y. 2000. Global climate change and infectious disease (in Chinese). *Chin J Dis Control Prev*, 4: 17–19
- US Global Change Research Program. 2001. *Climate Change Impacts on the United States: The Potential Consequences of Climate Variability and Change*. Cambridge: Cambridge University Press
- Van Borm S, Thomas I, Hanquet G, et al. 2005. Highly pathogenic H5N1 influenza virus in smuggled Thai eagles, Belgium. *Emerg Infect Dis*, 11: 702–705
- Vandegrift K J, Sokolow S H, Daszak P, et al. 2010. Ecology of avian influenza viruses in a changing world. *Ann N Y Acad Sci*, 1195: 113–128
- Vogt T M, Guerra M A, Flagg E W, et al. 2006. Risk of severe acute respiratory syndrome-associated coronavirus transmission aboard commercial aircraft. *J Travel Med*, 13: 268–272
- Wagner B G, Coburn B J, Blower S. 2009. Calculating the potential for within-flight transmission of influenza A (H1N1). *BMC Med*, 7: 81
- Wang J F, Christakos G, Han W G, et al. 2008. Data-driven exploration of ‘spatial pattern-time process-driving forces’ associations of SARS epidemic in Beijing, China. *J Public Health (Oxf)*, 30: 234–244
- Wang J, McMichael A J, Meng B, et al. 2006. Spatial dynamics of an epidemic of severe acute respiratory syndrome in an urban area. *Bull World Health Organ*, 84: 965–968
- Wang L J, Wei C Y. 2000. Research and prospects on meteorological epidemiology (in Chinese). *Chin J Epidemiol*, 21: 311–312
- Wang M, Di B, Zhou D H, et al. 2006. Food markets with live birds as source of avian influenza. *Emerg Infect Dis*, 12: 1773–1775
- Webster R G, Rott R. 1987. Influenza virus A pathogenicity: The pivotal role of hemagglutinin. *Cell Res*, 50: 665–666
- Weiss R A, McMichael A J. 2004. Social and environmental risk factors in the emergence of infectious diseases. *Nat Med*, 10: S70–76
- Weldon C, du Preez L H, Hyatt A D, et al. 2004. Origin of the amphibian chytrid fungus. *Emerg Infect Dis*, 10: 2100–2105
- Wen L, Xu D Z, Wang S Q, et al. 2003. Epidemics of malaria in Hainan province and modeling malaria incidence with meteorological parameters (in Chinese). *Chin J Dis Control Prev*, 7: 520–524
- Wenzel R P. 1994. A new hantavirus infection in North America. *N Engl J Med*, 330: 1004–1005
- Werneck G L, Rodrigues L, Santos M V, et al. 2002. The burden of *Leishmania chagasi* infection during an urban outbreak of visceral leishmaniasis in Brazil. *Acta Trop*, 83: 13–18
- Wilder-Smith A, Leong H N. 2004. A case of in-flight transmission of severe acute respiratory syndrome (SARS): SARS serology positive. *J Travel Med*, 11: 130
- Wilder-Smith A, Paton N I, Goh K T. 2003a. Experience of severe acute respiratory syndrome in Singapore: Importation of cases, and defense strategies at the airport. *J Travel Med*, 10: 259–262
- Wilder-Smith A, Paton N I, Goh K T. 2003b. Low risk of transmission of severe acute respiratory syndrome on airplanes: The Singapore experience. *Trop Med Int Health*, 8: 1035–1037
- Williams R A J, Fasina F O, Peterson A T. 2008. Predictable ecology and geography of avian influenza (H5N1) transmission in Nigeria and West Africa. *T Roy Soc Trop Med H*, 102: 471–479
- Williams R A J, Peterson A T. 2009. Ecology and geography of avian influenza (HPAIH5N1) transmission in the Middle East and north-eastern Africa. *Int J Health Geogr*, 8: 47
- Wilson M D, Cheke R A, Flasse S P, et al. 2002. Deforestation and the spatio-temporal distribution of savannah and forest members of the *Simulium damnosum* complex in southern Ghana and south-western Togo. *Trans R Soc Trop Med Hyg*, 96: 632–639
- Wilson M L, Chapman L E, Hall D B, et al. 1994. Rift Valley fever in rural northern Senegal: Human risk factors and potential vectors. *Am J Trop Med Hyg*, 50: 663–675
- Wolfe N D, Eitel M N, Gockowski J, et al. 2000. Deforestation, hunting and the ecology of microbial emergence. *Glob Change Hum Health*, 1: 10–25
- Woolhouse M E, Gowtage-Sequeria S. 2005. Host range and emerging and reemerging pathogens. *Emerg Infect Dis*, 11: 1842–1847
- Wu J Y, Lun Z R, James A A, et al. 2010. Dengue Fever in mainland China. *Am J Trop Med Hyg*, 83: 664–671
- Xie C Y, Yang H M, Qiu L, et al. 1999. The analysis of *Oncomelania* diffusion after the Yangtze River flood in Nanjing in 1998 (in Chinese). *J Pract Paras Dis*, 7: 187
- Xu B, Gong P, Biging G, et al. 2004. Snail density prediction for schistosomiasis control using IKONOS and ASTER images. *Photogramm Eng Remote Sens*, 70: 1285–1294
- Xu B, Gong P, Seto S E, et al. 2006. A spatial temporal model for assessing the effects of inter-village connectivity in schistosomiasis transmission. *AAAG*, 96: 31–46
- Yang H. 2007. Effect of extraordinarily serious drought on epidemic tendency of infectious diseases (in Chinese). *Occup Health*, 17: 15
- Yang Y Y, Guo X W, Gan Y D, et al. 2007. The incidence of infectious diseases in floating population in 2006, Daxing District, Beijing (in Chinese). *Capital J Public Health*, 2007, 1: 256–258
- Yasuoka J, Levins R. 2007. Impact of deforestation and agricultural development on anopheline ecology and malaria epidemiology. *Am J Trop Med Hyg*, 76: 450–460
- Zell R. 2004. Global climate change and emergence/re-emergence of infectious disease. *Int J Med Microbiol*, 293: 16–26
- Zeng G, Zhang L J. 2009. How does China response A/H1N1 influenza pandemic more effectively (in Chinese). *Chin J Epidemiol*, 30: 653–655
- Zeng G. 2009. Several critical issues about response to A (H1N1) influenza in China (in Chinese). *Chin J Epidemiol*, 30: 421–423
- Zhang L Z, Shen Y Z, Fan H J. 2004. The epidemic of malaria by flooding in Anhui Province and prevention measures (in Chinese). *Chin J Parasit Dis Control*, 17: 11
- Zhang S Q, Chen J S, Wang W, et al. 2003. Longitudinal observation on the epidemic situation of schistosomiasis after levee break by flood (in Chinese). *J Trop Dis Parasitol*, 1: 200–204
- Zhang S Q, Wang T P, Ge J H, et al. 2004. Influence on the diffusion of snail by flooding in Anhui province (in Chinese). *J Trop Dis Parasitol*, 2: 90–93
- Zhang Y, Bi P. 2008. Commentary on the relationship between climate change and infectious diseases (in Chinese). *Chin J Health Educ*, 24: 781–783
- Zhou G F. 2000. Discussion of characteristic of acute infectious diseases after the floods and the prevention measures (in Chinese). *Hubei J Prev Med*, 11: 72–73