

## REVIEW ARTICLE

# Climate Change and Emerging Zoonotic Diseases

Tin Tin Myaing

## Abstract

Globalization and climate change have had an unprecedented worldwide impact on emerging and re-emerging animal diseases and zoonoses. Climate variability resulting from naturally occurring climate phenomena such as El Niño, La Niña, and global monsoons, are associated with extreme weather events that lead to changes in the patterns of tropical rainfall. Thus disrupt natural ecosystems by providing more suitable environments for survival of pathogenic bacteria, viruses, and fungi and favor them to move into new areas thus zoonotic diseases are emerging. Climate change can alter bird migration patterns, changes in populations of waterfowl species, influence avian influenza virus transmission cycle. Vector borne diseases are especially susceptible to changing environmental conditions due to the impact of temperature, humidity and demographics of vectors. Transmission of Chikungunya virus and transmission of West Nile virus was directly linked to climate conditions. Lyme borreliosis and Tick-borne encephalitis in EU occurred due to weather changes. There are also reports of changes in the geographical distribution of sand flies, a vector of Leishmania species. Malaria and dengue fever also show significant seasonal patterns whereby transmission is highest in the months of heavy rainfall and humidity. Rift Valley fever virus is a zoonotic disease, spread among vertebrate hosts by the mosquito species *Aedes*. The most important vector of bluetongue virus in Africa and Asia appears under warming temperatures and changes in humidity. Evidence of water contamination following heavy rains has been documented for cryptosporidium, giardia, and *E.coli*. Outbreaks of human disease are often associated with increases in rodent populations after heavy rainfall or during floods, a good example for Leptospirosis. Hantavirus is a zoonotic virus of rodents and has emerged as a human pathogen due to human-induced landscape alterations and climatic changes influencing population dynamics of the rodent reservoir hosts. Public education and awareness to reduce exposure to disease, extreme weather alerts and metrological data collection may be implemented. Researchers should continue to expand their knowledge of how climate and weather influence health outcomes. Physical, biological, health, and social scientists must collaborate to better understand what makes certain people and animals' community more vulnerable to the health impacts of climate variability and change, and how people adapt to emerging zoonotic disease threats. An integrated approach to epidemiological, entomological and environmental data collection, analysis and enhancing zoonotic disease awareness should be reinforced.

University of Veterinary Science, Yezin, Nay Pyi Taw, Myanmar

Presented as an oral presentation at the 12th KKU Veterinary Annual Conference, Khon Kaen, June 2011.

E mail: dr.tintinmyaing@gmail.com

## Introduction

Climate variability resulting from naturally occurring climate phenomena such as El Niño, La Niña, and global monsoons, are associated with extreme weather events that lead to changes in the patterns of tropical rainfall. There was evidence that the increasing occurrence of tropical infectious diseases in the mid latitudes is linked to global warming. Warmer temperatures are already enabling insects and microorganisms to invade and reproduce in areas where once they could not survive in severely low temperatures and seasonal chills. Disruption of natural ecosystems by providing more suitable environments can favor the survival of some pathogenic bacteria, viruses, and fungi movement into new areas. Thus the pathogens are become to adapt, finding new niches and jumping across species into new hosts. Climatic factors such as increased temperature, increased or decreased precipitation, and sea level rise may impact on the emergence or reemergence of infectious diseases. Climate-induced changes in the geographic distribution and biologic behavior of arthropod vector-borne and rodent-borne infectious disease. Global warming found to be favored the spread of the mosquito into the temperate areas. Climate change factors that may expand the range and increase the reproductive potential of rodent populations include increased rainfall, warmer temperatures, and climatic extremes. Outbreaks of human disease are often associated with increases in rodent populations after heavy rainfall or during floods. Insect-borne diseases are now present in temperate areas where the vector insects were non existent in the past. Humans are also at an increased risk from insect-born diseases such as malaria, dengue, and yellow fever. Climate driven and other changes in landscape structure and texture, plus more general factors, may create favorable ecological niches for emerging diseases. Temperature and humidity levels directly affect the feeding activities of these insects, as well as their reproductive success, survival rates and ability to transmit disease. Low winter temperatures kill many exposed ticks and insects and the diseases they carry, so that cycles of disease transmission are interrupted and need to be restarted in spring.

Flea-borne infections are emerging or re-emerging throughout the world, and their incidence is still rising. Flea-borne diseases could re-emerge in epidemic form because of changes in vector-host ecology due to environmental and human behavior modifications. While local environmental changes are frequent, global climate change may influence parameters of flea development, distribution, and disease transmission on a much larger scale. For many fleas, temperature and humidity are crucial for development and survival [1]. The warmer temperatures predicted through most climate change scenarios could lead to an increased expansion of flea vectors into the northern hemispheres [2].

Climate change may also affect diseases spread by snails or by water, such as schistosomiasis and cholera, because changes in rainfall will have an impact on flow of rivers and levels of lakes; melting of polar ice may raise the sea level and inundate coastal and delta regions. If the agent and reservoir are

survived in the newly warmer climate, the agent can be expected to multiply more rapidly, and if the reservoir is an arthropod or snail, it too will develop more rapidly [3]. Freshwater snails, intermediate hosts for Fasciolosis, a disease that affects millions of herbivorous animals and can also affect humans can now be observed in areas above 4200 meters in the highlands of Peru and Bolivia as milder temperatures and altered environment conditions are more favourable to their survival. Fascioliasis is a particular problem where environmental conditions favour the intermediate host, lymnaeid snails. These conditions include low lying wet pasture, areas subject to periodic flooding, and temporary or permanent bodies of water [4].

### Animal Health Influenced Factors

Animal health may be affected by climate change in four ways: heat-related diseases and stress, extreme weather events, adaptation of animal production systems to new environments, and emergence or re-emergence of infectious diseases, especially vector-borne diseases critically dependent on environmental and climatic conditions [5]. The time period between a vector feeding on an infected host and being able to transmit the infection onward to a susceptible host is called the extrinsic incubation period (EIP). The EIP lengthens at lower temperatures. In colder areas, some short-lived vectors, such as mosquitoes and biting midges, tend to die before the EIP is complete and transmission does not occur [6,7]. A small rise in temperatures can produce a 10-fold increase in a mosquito population causing an increase of malaria cases hence, malaria is now occurring in several Eastern European countries as well as in the highland areas of countries like Kenya where historically cooler climatic conditions had prevented the breeding of populations of disease-carrying mosquitoes.

### Climatic Sensitivity on Virus Transmission

#### *Chikungunya virus*

Climate changes favored the exposure of Chikungunya virus (CHIKV) due to increased population of the mosquito vectors and consequently facilitated CHIKV emergence in some geographical areas [8, 9]. Chikungunya virus (CHIKV) is an Alphavirus from the Togaviridae family that shares the same vectors responsible for spreading dengue virus, namely *Aedes aegypti* and, to a lesser extent, *Aedes albopictus*, which are the common peridomestic mosquito species in the Southeast Asia region. Since 2004, several million indigenous cases of Chikungunya virus disease occurred in Africa, the Indian Ocean, India, Asia and, recently, Europe. The virus, usually transmitted by *Aedes aegypti* mosquitoes, has now repeatedly been associated with a new vector, *Ae. Albopictus* [10]. A notable recent example is the chikungunya virus epidemic that swept through the Indian Ocean region beginning in 2006 and which is believed to have infected >2 million persons. CHIKV infections occurred in Kuala Lumpur, Malaysia in 1998. The first sporadic outbreak of chikungunya virus (CHIKV) infection occurred between late 1998 and early 1999 in Port Klang, Kuala Lumpur. CHIKV is endemic [11] may play a substantial role in the spread of the virus to humans, although the effect of a warmer climate has also been reported

[12]. As a whole, such a climate abnormality of prolonged drier-than average conditions may be critical in introducing new viruses into the country [13]. Chikungunya disease outbreak occurred in Italy at late summer 2007 where favourable climate condition for virus replication resulted 200 cases there. Asian tiger mosquitoes (*Ae. albopictus*) are now established in Southern Europe and are better adapted to lower temperatures than the primary vector (*Aedes aegypti*) of the Chikungunya and dengue viruses [14]. A single-nucleotide polymorphism (SNP) in the virus genome accelerated its replication in the relatively common mosquito *Aedes albopictus*, usually a poorer host than *Ae. aegypti* mosquitoes [15].

#### *Dengue hemorrhagic fever virus*

Dengue hemorrhagic fever is found usually in children as a severe form and most commonly occurred in Asia. It is complicated by hemorrhage, shock and sometimes death. Dengue viruses are transmitted and replicates by mosquitoes. Dengue virus is belonged to the family Flaviviridae which in addition to dengue is comprised of yellow fever. Increased warming temperature at night is especially favorable to *Ae. aegypti* while *Ae. albopictus* has been permitted to withstand at freezing condition. The northern form of the mosquito is triggered by shortened periods of sunlight to enter diapauses, a physiological state of the egg that makes the egg resist cold temperature and delays hatching until the spring. Thus, the mosquito is able to survive freezing. Dengue hemorrhagic fever cases in Thailand in 1990 numbered more than 300,000 [16]. Humidity can greatly influence transmission of vector-borne diseases, particularly for insect vectors. Mosquitoes and ticks can desiccate easily and survival decreases under dry conditions. Saturation deficit has been found to be one of the most critical determinants in climate/disease models, for example, dengue fever [17,18] and Lyme disease models [19]. The potential for vector-borne zoonotic transmission to adapt to vector-borne human-to-human transmission is exemplified historically by dengue virus and *Plasmodium* spp., and more recently by Zika virus [20] and probably *P. knowlesi* [21].

#### *Nipah virus (NiV)*

Globally, the rate of tropical deforestation is highest in Asia [22]. In 1997 and 1998 more than 10 million acres of virgin forest burned in Borneo and Sumatra, set ablaze by humans but exacerbated by a severe El Niño Southern Oscillation event drought. It implicated the effects of the El Niño/Southern Oscillation (ENSO) phenomenon in 1997/98 posed to the emergence of NiV [23]. Malaysia had experienced a severe drought that directly preceding those outbreaks resulting from the El Niño conditions. The situation was aggravated by the excessive haze produced by the aggressive slash-and-burn deforestation activities in Indonesia. The haze and habitat loss are thought to have caused a mass exodus of “flying fox” fruit bats (*Pteropus*) searching for food, resulting in an unprecedented encroachment on cultivated fruit trees [23]. This series of environmental and human events may have affected the natural habitat of the pteropid bats, forcing their migration and subsequent encroachment into fruit orchards surrounding the pig-farming area, resulting in the unanticipated introduction of NiV from its natural host to pigs as the amplifying host [24].

### *West Nile Virus*

The transmission of West Nile virus, a zoonotic pathogen, can be directly linked to climate conditions. Birds act as reservoirs for the virus and mosquito vectors pass it from birds to humans. About 20% of infected humans will become ill and about one in 86 people will develop life-threatening symptoms. Temperature and humidity influence the distribution and density of many arthropod vectors and influence the incidence and northern range of vector borne diseases as West Nile virus [25]. Although birds, particularly crows and jays, infected with West Nile virus become ill or die, most infected birds survive and become reservoirs for the virus. Intensity of the virus fluctuates across the four seasons, peaking in summer and declining in fall and winter when the mosquitoes become dormant. Warmer, wetter summers will increase mosquito populations, while warmer winters will allow the virus to migrate [26]. The introduction of West Nile virus into the United States in 1999 was a dramatic example, as was the recent introduction of Usutu virus to Europe from Africa in migrating birds [27].

### *Avian Influenza virus H5N1*

A good example of a pandemic zoonotic disease that could be impacted by climate change is avian influenza [28]. Avian influenza viruses occur naturally in wild birds without producing any illnesses, however, a highly pathogenic strain of the disease named H5N1 is currently a major concern because it can affect humans and cause high case fatality. The first clinical respiratory illness of H5N1 avian influenza occurred in Hong Kong in 1997, when 18 human cases were reported during a poultry outbreak. It broke the species barrier to infect humans, cats, and tigers. So far it has affected many countries in Asia, Africa, and Europe. This emerging situation is mainly because severe winter conditions and droughts, occasioned by climate change can disrupt the normal migration pathways of wild birds and thereby bring both wild and domestic bird populations into greater contact at remaining water sources. So climate change influence the avian influenza virus transmission cycle and directly affect virus survival outside the host [28]. Climate change can alter bird migration patterns, changes in populations of waterfowl species, influence avian influenza virus transmission cycle. The EU has already experienced during early 2006 that very cold weather in some areas causing feed scarcity and unusual freezing of open waters forced wild waterfowl to change their flyways which has led to the introduction of highly pathogenic avian influenza of the H5N1 subtype into the EU[29]. Continuous evolution of the H5N1 virus has been suggested by changes in the internal gene constellation, expanded host range, increased pathogenicity, and greater environmental stability [30,31].

### *Rift Valley Fever (RVF)*

Rift Valley fever where the virus is primarily a zoonotic disease, spread among vertebrate hosts by the mosquito species *Aedes*. Primarily under flood conditions, *Culex* mosquitoes may feed upon infected ungulate hosts. This vector is referred to as a bridge species because it feeds on humans also, resulting in spread of the virus outside its normal zoonotic cycle [32]. Rift Valley Fever (RVF), an important zoonotic viral disease of sheep and cattle, is transmitted by *Aedes* and *Culex* mosquitoes.

Epizootics of RVF are associated with periods of heavy rainfall and flooding [33-35]. In east Africa, with the combination of heavy rainfall following drought associated with ENSO [35,36]. ENSO-related floods in 1998, following drought in 1997, led to an epidemic of RVF and some other diseases outbreak in the Kenya/Somalia border area, causing the deaths of more than 2000 people and two-thirds of all small ruminant livestock [37].

#### *Lyme disease*

Changes in tick distribution have also been observed due to climatic changes. The limit of tick distribution in the EU is shifting northwards and also to higher altitudes; plus, the shift towards milder winters may lead to an expansion of the tick population and, consequently, to the human exposure to Lyme borreliosis and Tick-borne encephalitis. There are also reports of changes in the geographical distribution of sand flies, which are a vector of Leishmania species [29]. The density of infected, nymphal, black-legged ticks (*Ixodes scapularis*), the primary risk factor for Lyme disease, has been linked to increasing forest fragmentation in periurban areas, likely due to corresponding increased numbers of the white-footed mouse (*Peromyscus leucopus*), the principal reservoir of the Lyme bacterium (*Borrelia burgdorferi*) [38].

#### *Hanta virus*

Rodents are sources of a number of zoonoses (including Hantavirus, plague and leptospirosis). Hantavirus is a directly transmitted zoonosis that is naturally maintained in rodent reservoirs and can be transmitted to humans at times of increased local abundance of the reservoir [39]. Outbreaks of Hantavirus in the southwestern United States have clearly been linked to El Nino impacts on rodent populations [40].

### Climate Sensitivity on Bacterial Infection

#### *Anthrax*

Anthrax is an acute infectious zoonotic disease of most warm-blooded animals, including humans, with worldwide distribution. The causative bacterium, *Bacillus anthracis*, forms spores able to remain infective for 10-20 years in pasture. Temperature, relative humidity and soil moisture can affect the successful germination of anthrax spores, while heavy rainfall may stir up dormant spores. Outbreaks are often associated with alternating heavy rainfall and drought, and high temperatures [41]. Changes in green-up and precipitation sporadicity in conjunction with rangeland expansion could indicate that some changes in the epidemiology of anthrax could occur such as longer anthrax seasons and an exposure of animals to more areas where *B. anthracis* may exist [42]. Because large anthrax epizootics often appear to occur after specific rain events in association with overall hot, dry summer conditions [43,44] the increasingly sporadic rate of precipitation may also create some changes in the epidemiology of anthrax in the US as well as potentially in Kazakhstan [45].

#### *Blackleg*

Blackleg, an acute infectious clostridial disease, mostly of young cattle, is also spore-forming,

and disease outbreaks are associated with high temperature and heavy rainfall [4]. Many pathogens or parasites, such as those of anthrax, haemonchosis and summer mastitis, may be subject to the opposing forces of higher temperatures promoting pathogen or vector development, and increased summer dryness leading to more pathogen or vector mortality.

### *Leptospirosis*

Leptospirosis remains one of the most common and most zoonotic infections worldwide [46]. In India, leptospirosis is a major health problem obviously related both to the monsoons and poor sanitary conditions, with multiple epidemics reported in recent years [47-51]. Thailand has been the source of a leptospirosis outbreak in the 21st century, subsequent to flooding, and localized mainly in the northeast region of the country [52]. Fears of the emergence of leptospirosis outbreaks following the 2004 tsunami disaster was possibly due to the beneficial effect of salt water mixing with fresh waters [53].

In Bangladesh, cholera outbreaks occur during the monsoon season [54]. Severe epidemics of cholera strike regularly in many parts of the developing world. The timing of these epidemics is partly explained by environmental and ecological conditions that are influenced by climate. In particular a significant reservoir of the cholera-causing organism, *Vibrio cholerae*, appears to reside in marine ecosystems where it attaches to zooplankton [55]. In Peru, cyclospora infections peak in the summer and subside in the winter. In Scotland, campylobacter infections are characterized by short peaks in the spring [56]. The *E. coli* O157:H7 water contamination in Walkerton, Ontario, in 2000 was preceded by a severe rainstorm. This rainfall flushed *E. coli* into the drinking water system through a faulty well, and the contamination was improperly reported by water utility personnel [57].

### *Future works*

Owing to anthropogenic activities, it needs to take widespread scientific agreement that the world's climate is warming at a faster rate than ever before including concomitant changes in precipitation, flooding, winds and the frequency of extreme events such as El Niño. In order to evaluate the risks of the occurrence of emerging zoonotic diseases especially vector borne diseases that are heavily influenced by weather conditions and climate change, weather forecasting and analysis will need to be included in the early warning systems. Disease surveillance must be combined with a network of expert laboratories with the appropriate diagnostic capabilities to carry out testing for still exotic or rare diseases. An integrated approach to epidemiological, entomological and environmental data collection and analysis is essential to enhance and exchange knowledge between international experts. A comprehensive study of mosquito-borne diseases that requires a combination of entomologists, epidemiologists and climatologists to work together to examine the associations of changing vector habitats, disease patterns and climatic factors. Animal disease awareness and preparedness should be noticed for farmers, veterinarians, relevant sectors, including descriptions of the diseases, preventive measures and practical control measures. Incidence data are needed to provide a baseline for epidemiological studies to increase actively global disease surveillance by computer data based system to share the information among the veterinarians. International

collaboration should be taken such as WHO, OIE and FAO and can work together within the framework. Further work needs to be done to determine the role of climate change in the future epidemiology of zoonotic and other diseases.

## References

1. Idir Bitam, Katharina Dittmar, Philippe Parola, Michael F, Whiting, Didier Raoult. Fleas and flea-borne diseases. *Int J Infect Dis.* 2010; 14: e667–e676.
2. Gage KL, Burkot TR, Eisen RJ, Hayes EB. Climate and vectorborne diseases. *Am J Prev Med.* 2008;35:436-450.
3. Shope RE. Impacts of global climate change on human health: Spread of infectious disease. In: *Global climate change: Implications, challenges and mitigation measures*, editor. SK Majumdar, LS Kalkstein, B Yarnal, EW Miller and LM Rosenfeld: Easton PA: The Pennsylvania Academy of Science.1992.p 363-370.
4. Hall HTB. In: *Diseases and Parasites of Livestock in the Tropics.* Longman Scientific and Technical: Harlow, Essex.1988.
5. Forman S, Hungerford N, Yamakawa M, Yanase T, Tsai HJ, Joo YS, et al. Climate change impacts and risks for animal health in *Asia Rev Sci Tech.* 2008;27:581-597.
6. Lines J. The effects of climatic and land-use changes on insect vectors of human disease. In: Harrington, R., Stork, N.E. (Eds.) *Insects in a Changing Environment.* Academic Press: London, 1995:157-175.
7. Reeves WC, Hardy JL, Reisen WK and Milby MM. Potential effect of global warming on mosquito-borne arboviruses. *J Med Entomol.* 1994;31:323-332.
8. Patz JA, Campbell-Lendrum D, Holloway T, Foley JA. Impact of regional climate change on human health. *Nature.* 2005; p 438.
9. Chretien JP, Anyamba A, Bedno SA, Breiman RF, Sang R, Sergon K, et al. Drought-associated chikungunya emergence along coastal East Africa. *Am J Trop Med Hyg.* 2007;76:405-407.
10. de Lamballerie X, Leroy E, Charrel RN, Ttsetsarkin K, Higgs S, Gould EA. Chikungunya virus adapts to tiger mosquito via evolutionary convergence: a sign of things to come? *Virol J.* 2008;27:33.
11. Lam SK, Chua KB, Hooi PS, Rahimah MA, Kumari S, Tharmaratnam M, et al. Chikungunya infection- an emerging disease in Malaysia. *Southeast Asian J Trop Med Public Health.* 2001;32:447.
12. Rogers DJ, Randolph SE. Studying the global distribution of infectious diseases using GIS and RS. *Nat Rev Microbiol.* 2003;1:231-237.
13. EDEC Mission report. Chikungunya in Italy. Joint EDEC/WHO visit for a European risk assessment,17-18 September 2007. European Center for Disease prevention and Control, Stockholm, Sept 2007.
14. Woolhouse M, Gaunt E. Ecological origins of novel pathogens. *Crit Rev Microbiol.* 2007;33:231-242.
15. OFFICE OF SCIENCE AND INNOVATION T7.3: UK. Infectious Diseases: preparing for the future, The Effects of Climate Change on Infectious Diseases of Animals. Available at [www.foresight.gov.uk](http://www.foresight.gov.uk).
16. Chunsuttiwat S. Epidemiology and control of dengue hemorrhagic fever in Thailand. *Southeast Asian J Trop Med Public Health.* 2001;21:684-685.



17. Focks DA. A simulation model of the epidemiology of urban dengue fever: literature analysis, model development, preliminary validation, and samples of simulation results. *Am J Trop Med Hyg.* 1995;53:489–506.
18. Hales S. Potential effect of population and climate changes on global distribution of dengue fever: an empirical model. *Lancet.* 2002;360:830–834.
19. Mount GA. Simulation of management strategies for the blacklegged tick (*Acari Ixodidae*) and the Lyme disease spirochete, *Borrelia burgdorferi*. *J Med Entomol.* 1997;34: 672-683.
20. Duffy MR, Chen TH, Hancock WT, Powers AM, Kool JL, Lanciotti RS, et al. Zika virus outbreak on Yap Island, Federated States of Micronesia. *N Engl J Med.* 2009;360:2536-2543.
21. Cox-Singh J, Davis TM, Lee KS, Shamsul SS, Matusop A, Ratnam S, et al. *Plasmodium knowlesi* malaria in humans is widely distributed and potentially life threatening. *Clin Infect Dis.* 2008;46:165-171.
22. Mayaux P, Holmgren P, Achard F, Eva H, Stibig HJ, and Bran-thomme A. Tropical forest cover change in the 1990s and options for future monitoring. *Philos Trans R Soc Lond B Biol Sci.* 2005;360:373-384.
23. Chua KB, Chua BH, Wang CW. Anthropogenic deforestation, El Niño and the emergence of Nipah virus in Malaysia. *Malays J Pathol.* 2002;24:15-21
24. Kok Keng Tee, Yutaka Takebe, Adeeba Kamarulzaman. Emerging and re-emerging viruses in Malaysia, 1997-2007. *Int J Infect Dis.* 2009;13:307-318
25. Parkinson AJ, Butler JC. Potential impacts of climate change on infectious diseases in the Arctic. *Int J Circumpolar Health.* 2005;64:478-486.
26. Barker IK, Lindsay LR. Lyme Borreliosis in Ontario: Determining the Risks. *Canadian Med Assoc J.* 2000;182:1573-1574.
27. Weissenböck H, Kolodziejek J, Url A, Lussy H, Rebel-Bauder B, Nowotny N. Emergence of Usutu virus, an African mosquito-borne flavivirus of the Japanese encephalitis virus group, central Europe. *Emerg Infect Dis.* 2002;8:652-656.
28. Gilbert M, Slingenbergh J and Xiao X. Climate change and avian influenza. *Rev Sci Tech.* 2008;27:459-466.
29. COMMISSION OF THE EUROPEAN COMMUNITIES. *Human, Animal and Plant Health Impacts of Climate Change.* COM. Brussels, 14.2009. SEC(2009) 41
30. Sims LD, Ellis TM, Liu KK, Dyrting K, Wong H, Peiris M, et al. Avian influenza in Hong Kong 1997-2002. *Avian Dis.* 2003;47:832-838.
31. Horimoto T, Fukuda N, Iwatsuki-Horimoto K, Guan Y, Lim W, Peiris M, et al. Antigenic differences between H5N1 human influenza viruses isolated in 1997 and 2003. *J Vet Med Sci.* 2004;66:303-305.
32. Wilson ML. Ecology and infectious disease. In: *Ecosystem change and public health: a global perspective*, Aron J.L. & Patz, J.A. eds. Baltimore, USA, John Hopkins University Press 2001; pp. 283-324.
33. Davies FG, Linthicum KJ and James AD. Rainfall and Epizootic Rift-Valley Fever. *Bulletin of the World Health Organization.* 1985;63:941-943.
34. Linthicum KJ, Bailey CL, Davies FG and Tucker CJ. Detection of Rift-Valley Fever Viral Activity in Kenya by Satellite Remote-Sensing Imagery. *Sci.* 1987;235:1656-1659.
35. Little PD, Mahmoud H and Coppock DL. When deserts flood: risk management and climatic processes

- among east African pastoralists. *Climate Res.* 2001;19:149-159.
36. Linthicum KJ, Anyamba A, Tucker CJ, Kelley PW, Myers MF and Peters CJ. Climate and satellite indicators to forecast Rift Valley fever epidemics in Kenya. *Sci.*1999;285:397-400
  37. Anyamba A, Linthicum KJ, Mahoney R, Tucker CJ and Kelley PW. Mapping potential risk of Rift Valley fever outbreaks in African savannas using vegetation index time series data. *Photogrammetric Engineering and Remote Sensing* 2002;68:137-145.
  38. Allan BF, Keesing F, and Ostfeld RS. Effect of forest fragmentation on Lyme disease risk. *Conserv Biol.* 2003;17: 267-272.
  39. Parmenter R R. Incidence of plague associated with increased winter-spring precipitation in New Mexico. *Am J Trop Med Hyg.* 1999;61:814-821.
  40. Hjelle B and Glass GE. Outbreak of Hantavirus Infection in the Four Corners Region of the United States in the Wake of the 1997-1998 El Nino-southern Oscillation. *J Infect Dis.* 2000; 181(5): 1569-1573
  41. Parker R, Mathis C, Looper M and Sawyer J. Anthrax and livestock. In: Guide B-120. Cooperative Extension Service, College of Agriculture and Home Economics, University of New Mexico: Las Cruces, New Mexico 2002.
  42. Peterson AT, Soberon J, Sanchez-Cordero V. Conservation of ecological niches in evolutionary time. *Science.* 1999;285:1265-1266.
  43. Turner AJ, Galvin JW, Rubira RJ, Condron RJ, Bradley T. Experiences with vaccination and epidemiological investigations on an anthrax outbreak in Australia in 1997. *J Appl Microbiol.* 1999;87: 294-297.
  44. Parkinson R, Rajic A, Jenson C. Investigation of an anthrax outbreak in Alberta in 1999 using a geographic information system. *Canadian Vet J.* 2003;44:315-318.
  45. Timothy Andrew Joyner, Larissa Lukhnova, Yerlan Pazilov, Gulnara Temiralyeva, Martin E. Hugh-Jones, Alim Aikimbayev, Jason K. Blackburn. Modeling the Potential Distribution of *Bacillus anthracis* under Multiple Climate Change Scenarios for Kazakhstan. Emerging pathogens Institute, Journal online on 9-3-2010, 2010. Journal PloS-One, University of Florida.
  46. Levett PN. Leptospirosis. *Clin Microbiol Rev.* 2001;14:296-326
  47. Sharma S, Vijayachari P, Sugunan AP, Natarajaseenivasan K, Sehgal SC. Seroprevalence of leptospirosis among high-risk population of Andaman Islands, India *Am J Trop Med Hyg.* 2006;74:278-283.
  48. Manocha H, Ghoshal U, Singh SK, Kishore J, Ayyagari A. Frequency of leptospirosis in patients of acute febrile illness in Uttar Pradesh. *J Assoc Physicians India.* 2004;52:623-625.
  49. Karande S, Bhatt M, Kelkar A, Kulkarni M, De A, Varaiya A. An observational study to detect leptospirosis in Mumbai, India, 2000. *Arch Dis Child.* 2003;88:1070-1075.
  50. Jena AB, Mohanty KC, Devadasan N. An outbreak of leptospirosis in Orissa, India: the importance of surveillance. *Trop Med Int Health.* 2004;9:1016-1021.
  51. Bharadwaj R, Bal AM, Joshi SA, Kagal A, Poll SS, Garad G, et al. An urban outbreak of leptospirosis in Mumbai, India. *Jpn J Infect Dis.* 2002;55:194-196.
  52. Tangkanakul W, Smits HL, Jatanasen S, Ashford DA. Leptospirosis: an emerging health problem in Thailand. *Southeast Asian J Trop Med Public Health.* 2005;36:281-288.
  53. Ellis RD, Fukuda MM, McDaniel P, Welch K, Nisalak A, Murray CK, et al. Causes of fever in adults on the

- Thai-Myanmar border. *Am J Trop Med Hyg.* 2006;74:108-113.
54. Colwell RR. Global climate and infectious disease: the cholera paradigm. *Science.* 1996;274(5295): 2025–2031.
55. Huq A. Cholera and global ecosystems. In: *Ecosystem change and public health: a global perspective*, Aaron JL. & Patz JA, eds. Baltimore, USA, The Johns Hopkins University Press, 2001; 327–352.
56. Colwell RR, Patz JA. Climate, infectious disease and health. Washington, DC, USA, *Am Academy Microbiol*, 1998; p22.
57. Connor DRO. Report of the Walkerton Inquiry: Part I. The Events of May 2000 and Related Issues (Toronto: Ontario Ministry of the Attorney General, Queen’s Printer for Ontario, 2002; 4:103-197.