

# **CLIMATE CHANGE AND HUMAN INFECTIOUS DISEASES (REVIEW)**

By

El Samra GH

*Department of Occupational and Environmental Medicine, Faculty of Medicine, Cairo University.*

*El Samra GH:gelsamra991@gmail.com*

## **Abstract**

The Intergovernmental Panel on Climate (IPCC) has concluded that climate change has significant effects on human health. In order to understand how climate change impacts infectious diseases; it is essential to understand the pathways of disease transmission. Diseases transmitted directly from source of infection to the affected individual are least affected by environmental factors. If diseases are transmitted indirectly through a physical vehicle or a biological vector, climate change will have significant effect on disease transmission through the effect on the vehicle or the vector and the pathogen. The effect of climate change varies from one region to the other. Moreover, vulnerability to the effects of climate change varies from one individual to the other, from one group to the other and from one country to the other. Disease transmission is also affected by socio-economic factors, globalization of travel and trade, land use, unplanned urbanization, population growth, and cultural attitudes, among other factors.

Diseases transmitted via direct pathways include air-borne diseases like influenza, tuberculosis, measles or sexually transmitted diseases. Food-borne diseases and water-borne diseases like cholera and diarrhoeal diseases are transmitted indirectly, via physical vehicle.

Mosquitoes are the best known biological disease vectors. Diseases transmitted indirectly through mosquitoes can be transmitted either by mosquito-human-mosquito pathway such as malaria, dengue, yellow fever, chikungunia, zika and lymphatic filariasis; or by mosquito-animal-mosquito pathway such as west Nile virus disease, Rift valley fever and Saint Louis encephalitis.

Diseases transmitted indirectly via biological vectors also include tick-borne diseases such as lyme disease, tick-borne relapsing fever, typhus, Q fever and Rocky Mountain spotted fever; sandfly-borne diseases such as leishmaniasis; and rodent-borne diseases such as leptospirosis, hantavirus pulmonary syndrome, tularemia and plague through fleas.

Diseases can also be transmitted indirectly through other vectors like tse-tse fly, black fly, triatomine bug and aquatic snails.

Beginning in mid-1970s, there has been worldwide emergence, resurgence and redistribution of infectious diseases. Definitions of emerging diseases were given as well as examples of climate change impacts on emerging diseases. Examples given included west Nile virus disease, hantavirus pulmonary syndrome, Saint Louis encephalitis, Murray valley fever and Zika.

**Key words:** Infectious diseases, Climate change, Biological vectors, Emerging diseases and Resurgence and redistribution of infectious diseases.

### **Introduction**

Climate change is occurring as a result of imbalance between incoming and outgoing radiations in the atmosphere. As solar radiations reach the atmosphere, some are reflected; part of what reaches the earth is reflected and the rest warms the earth's surface causing the emission of infrared radiations. Some of the emitted infrared radiations passes through the atmosphere; some is reflected back to the earth and some is absorbed by the green house gases - primarily CO<sub>2</sub>, methane and nitrous oxide, among other gases – and is emitted to the earth's surface, which gains more heat, resulting in global warming. The term “climate change” is preferred to “global warming” (Shuman, 2010).

For the past 420000 years, atmospheric carbon dioxide levels have remained within a range of 180 – 280 ppm. Carbon dioxide level is now close to 370 ppm and rising. The

concentration of green house gases in the atmosphere has reached record levels (Epstein, 2002).

This century, warming has been rapid and it is not occurring uniformly. Global temperature has risen at faster rate than any time since records began to be kept in the 1850s; and temperature is expected to increase by another 1.8 – 5.8 °C by the end of this century. The hydrologic cycle is going to be altered since hot air causes more evaporation; warmer air can contain more water vapour than cooler air (air can hold 7% more water vapour for each 1°C it warms) (Shuman, 2010). Also, since 1957, world oceans have accumulated 22 times as much heat as the atmosphere, accelerating the global hydrological cycle (Epstein, 2002 and Epstein, 2010). Some geographic areas will have more rainfall and some more drought; and severe weather events – including heat waves and storms – are expected to be more common. In

2001, IPCC concluded that humans are playing a major role in causing climate change largely through deforestation and the combustion of fossil fuels that produce heat-trapping carbon dioxide .

Because of the rising temperature and the changing rainfall pattern, climate change is expected to have substantial effects on the burden of infectious diseases that are transmitted by insect vectors and through contaminated food and water. In a few instances these effects will be favourable but in most cases they will impose new threats to public health.

Extreme weather events can be associated with clusters of vector-borne and water-borne diseases. Flash floods leave behind mosquito breeding sites, drive rodents from their burrows to human habitation and seed waterways with micro-organisms (such as *E. coli*, *Cryptosporidium* and *Vibrio cholera*) and nutrients that can trigger red tide.

Beginning in mid-1970s there has been world-wide emergence, re-surgence and redistribution of infectious diseases. Changes in biodiversity - that are caused by climate change - have apparently contributed to emergence and re-surgence of some infectious diseases.

The following components are essential for the transmission of infectious diseases: an agent (or pathogen), a host (or vector) and transmission environment (Epstein, 2001). Some pathogens are carried by vectors and some require intermediate hosts to complete their transmission cycle.

Both the infectious agents and the associated vector organisms are devoid of thermostatic mechanisms. Their temperature and fluid levels are, therefore, determined directly by the surrounding climate conditions (climate envelope). The incubation period of a vector-borne infective organism within its vector is very sensitive to changes in environmental temperature. The agent, vector and host are also sensitive to the level of precipitation, relative humidity, sea-level rise, wind and duration of daily sunlight (Patz et al., 2003 and Lindgren and Ebi, 2010). Climate warming tends to favour geographic expansion of several infectious disease vectors at the latitudinal and altitudinal levels, meaning that certain vector-borne diseases may be introduced to regions that have not previously encountered them, and that extreme weather events may help create the opportunities for

more clustered disease outbreaks or outbreaks of diseases at non-traditional place and time (Epstein, 2001). “Climate conditions” determine the geographic and seasonal distribution of infectious diseases and their vectors while “weather” affects timing and intensity of disease outbreaks (Dobson and Carper, 1993 and Kuhn et al., 2005).

The effect of climate change on infectious diseases can also be modified by sociodemographic influences such as migration, transportation, nutrition, population growth, drug resistance, water projects, urbanization ...etc. Climate change is likely to affect human migration as well as mobility of animal hosts, vectors and pathogens. Although the spread of infectious diseases is multicausal, global climate change may be a major contributor.

**Disease classification relevant to climate/health relationship** (Patz et al., 2003):

From the epidemiological point of view, among the most important characteristics for disease classification are: the method of transmission of the pathogen (direct or indirect) and its natural reservoir (animal or human).

## **Directly Transmitted Infectious Diseases:**

### **a. Anthroponoses :**

Directly transmitted anthroponoses include diseases in which the pathogen is normally transmitted directly between two human hosts through physical contact or droplet exposure. These diseases are generally least likely to be influenced by climatic factors since the agent spends little or no time outside the human host. However, they are affected by human behaviour such as poor sanitation or crowding that may result from altered land use caused by climatic change. Examples of directly transmitted anthroponoses include tuberculosis, measles and sexually transmitted diseases like AIDS/ HIV.

### **b. Zoonoses :**

Here the pathogen is also transmitted through physical contact or droplet exposure between animal reservoirs; humans are infected through accidental encounter. The persistence of these pathogens in nature is largely dependent on transmission dynamics between animals and on climate variability. Hantavirus pulmonary syndrome is a directly transmitted zoonosis which is naturally maintained

in rodent reservoirs. Several of today's anthropogenic diseases, e.g. tuberculosis and AIDs/ HIV originally emerged from animals.

### **Indirectly Transmitted Infectious Diseases:**

c. In indirectly transmitted anthroponoses, transmission of pathogen between two human hosts occurs by either a physical vehicle (food or water) or a biological vector (mosquito, tick etc..). Most vectors require a blood meal from the vertebrate host in order to sustain life and reproduce. Indirectly transmitted anthroponoses include malaria and dengue, whereby the respective malaria parasite and dengue virus are transmitted between human hosts by mosquito vectors (vector-borne diseases). Indirectly transmitted water-borne anthroponoses are susceptible to climatic factors because pathogens exist in the external environment during part of their life cycles. Flooding may result in the contamination of water supplies, and the reproduction rate of the pathogen may be influenced by ambient air temperature.

Cholera is an indirectly transmitted water-borne anthroponose. In the marine environment, *Vibrio cholera* resides by attaching to zooplankton. Survival of zooplankton in turn depends on the abundance of their food supply, phytoplankton. Phytoplankton population tends to increase when ocean temperatures are high. As a result of these ecological relationships, cholera outbreaks occur when ocean surface temperature rises (McMichael et al., 1996).

d. In indirectly transmitted zoonoses, the natural cycle of transmission occurs between two non-human vertebrates; humans are infected due to an accidental encounter with an infected vehicle or vector. This class of diseases involves four components in the transmission cycle: The pathogen; biological vector or physical vehicle, animal reservoir and human host. These diseases are highly susceptible to a combination of ecological and climatic factors because of the numerous components in the transmission cycle and the interaction of each of these with the external environment.

**Complex cycles of disease transmission** also exist for several diseases which cannot be classified simply by mode of transmission or natural reservoir. An example is Rift valley fever where the virus naturally causes a zoonotic disease spread among vertebrate hosts by mosquito species *Aedes*. Under flood conditions *Culex* mosquitoes also may feed upon infected hosts. This vector is called a “bridge species” because it feeds on humans also resulting in spread of the virus outside its normal zoonotic cycle (WHO, 2003).

#### **Vulnerability to climate change:**

The Intergovernmental Panel on Climate Change (IPCC) (2001) has defined vulnerability to climate change as the degree to which a system is susceptible to - or unable to cope with the adverse effects of climate variability or change. Vulnerability differs from one individual to another, from one group to another and from one country to another. While population in most countries will be exposed to the hazards of climate change, the risk is greater in lower-income countries because the burden of climate-sensitive diseases is higher and the public health systems are weak. Another trend of importance is population growth (IPCC, 2007).

The following is an overview of the different types of relationships between climate and disease and how climate may impact the transmission of the most important communicable diseases.

#### **I. Air-borne Diseases :**

Certain diseases like influenza, meningococcal meningitis and human respiratory syncytial virus in children normally occur during winter season in temperate regions. This seasonal pattern is probably more due to human behavior (e.g. people crowding indoors during winter) which is affected by cold weather. However, if warmer winter seasons resulted in less indoor crowding, the risk of disease transmission would decrease. On the other hand, hotter summer would contribute to the risk of acquiring legionellosis if the heat causes increased use of cooling systems (Lindgren and Ebi, 2010).

In Sub-Saharan Africa, meningococcal meningitis tends to erupt during the hot and dry season and subside soon after the beginning of the rainy season (WHO, 2016a).

#### **II. Food-borne Diseases :**

Heat waves as well as longer hot summer will increase the risk of outbreaks of food-borne diseases. Many

food-borne pathogens will increase their growth rate at higher temperatures. Studies have shown a linear relationship between ambient temperature and outbreaks of salmonella infection. In general, the link between climate and food-borne outbreaks is probably most often due to improper production, storage, transportation and handling of food at higher ambient temperatures especially if the capacity of freezers, refrigerators and other cold storage devices is exceeded during heat waves. Restaurants and consumers may leave food out for too long in high ambient temperatures at buffet settings or outdoor barbecues, for example. Heavy rainfall may cause irrigation water and agricultural products to be contaminated with pathogens from human excreta or animal material.

### **III. Water-borne Diseases :**

Climate change may increase the risk of water-borne diseases in several ways:

Increased temperature will cause increased growth and survival of certain pathogens/parasites in water sources (including private drinking and recreational water sources and coastal waters) and in water pipes; consequently, may lengthen the seasonality of water-

borne diseases. Enteric diseases: cholera, campylobacter infection, cyclospora etc... have shown seasonal fluctuations in different parts of the world. High ambient temperature has been associated with increased hospital admissions with diarrheal diseases.

Heavy rainfall can cause dissemination and transport of pathogens. It can contaminate fresh water sources by human or animal fecal material and cause leakage of pathogens such as salmonella and cryptosporidium into drinking water sources.

Moreover, flooding and landslides can damage infrastructure (water treatment plants, electrical substations etc...), overload capacity or cause leakage of sewage into water sources.

Drought may have negative effects on both quality and quantity of drinking water. Pollutants in water will be more concentrated. Drought has been associated with increased diarrheal diseases which are likely associated with water storage processes and bad hygiene. Water shortage will increase the risk of water-washed diseases like trachoma, scabies and louse-borne typhus.

Increasing sea surface temperature can influence the viability and growth of enteric pathogens as *Vibrio cholera* by increasing their reservoir's food supply.

#### **IV. Vector-borne Diseases (VBDs):**

Vectors are living organisms that can transmit infectious diseases (vector-borne diseases) between humans or from animals to humans. Many of the vectors are blood-sucking insects, which ingest pathogenic micro-organisms during a blood meal from an infected host (human or animal) and later inject it into a new host during their subsequent blood meal (WHO, 2016<sup>a</sup>). Sustained (or endemic) transmission of vector-borne disease in a particular area depends on an adequate vector population and favourable environmental conditions for vector, host and infective agent; an intermediate host species may be necessary.

Vector-borne diseases account for more than 17% of all infectious diseases. Every year there are more than one billion cases and over 1 million deaths from vector-borne diseases such as malaria, dengue, schistosomiasis, human African trypanosomiasis, leishmaniasis, chagas disease, yellow fever, Japanese encephalitis and

onchocerciasis, globally. More than 2.5 million people in over 100 countries are at risk of contracting dengue alone. Malaria causes more than 400000 deaths every year, globally, most of them are children under 5 years of age. Other vector-borne diseases affect hundreds of millions of people world wide (WHO, 2016a).

Vectors, pathogens and hosts each survive and reproduce within certain optimal climatic conditions and changes in these conditions can modify greatly the properties of disease transmission. Biotic factors such as vegetation; population of the host species; predators, competitors and parasites; and human interventions are also important factors in disease transmission. Climate change will impact various vector-borne diseases differently because of differences in epidemiology and ecology.

#### **Influence of Climatic Factors on VBD Transmission:**

The most influential climatic factors for transmission of vector-borne diseases are temperature and precipitation but sea level rise, wind and day light duration are important factors (WHO, 2003).



☛ *An increase in temperature* can modify the growth of disease-carrying vectors, accelerate their metabolic rate, consequently affecting their nutritional requirements. They feed more frequently, lay more eggs and their biting rate increases. Temperature alters the rate at which vectors come in contact with humans. Vectors may adapt to changes in temperature by expanding and changing their geographic distribution since this is limited by minimum and maximum temperatures.

Extreme temperatures are often lethal to the disease-causing pathogens, but incremental changes in temperature may exert varying effects. Where a vector lives in an environment where the mean temperature approaches the limit of physiological tolerance of the pathogen, a small increase in temperature may be lethal to the pathogen. Alternatively, where the vector lives in an environment of low mean temperature, a small increase in temperature may result in increased development, and replication and shorter incubation period of the pathogen in the vector. Also, changes in temperature regime may alter the length of the transmission season.

☛ *High temperature and relative humidity* prolong the survival of most arthropod vectors.

Increased precipitation may increase the presence of disease vectors by expanding the size of existent larval habitats and creating new breeding sites. In Addition, precipitation may favour most of the metabolic processes of the vector and support growth of food supplies which in turn support a greater population of vertebrate reservoirs. Outbreaks of vector-borne diseases have shown seasonal fluctuation whereby disease transmission was highest in the months of heavy rain and humidity. However, vectors like *Aedes aegypti*, adapted to urban environments, breed in water containers and are relatively unaffected by precipitation.

Flooding causes a decrease of vector population by eliminating larval habitats and creating unsuitable environment for vertebrate reservoirs. Alternatively, insects may use standing water caused by flooding as breeding grounds; and invasions of disease vectors in the aftermath has been reported (WHO, 2016<sup>a</sup>). Flooding may force insect and rodent vectors to seek refuge in houses thus increasing the likelihood of vector-human contact.

Leptospirosis epidemics have been reported following severe flooding.

Drought can cause rivers to slow creating more stagnant pools that are ideal vector breeding habitats. Mosquitoes and ticks can desiccate easily and survival decreases under hot dry conditions.

Introduction or expansion of irrigation systems in areas affected by drought or decreased precipitation would increase the number of breeding sites for mosquitoes (WHO, 2016a).

☛ *Wind* contributes to passive dispersion of flying insects. Wind direction and speed

affect vector distribution; some insect vectors including anopheline mosquitoes and sandflies can thus be dispersed hundreds of kilometers from their original area causing disease emergence in the new areas.

Sea level rise and increased coastal flooding may result in greater quantities of brackish water which would favour vector species, such as *A. albopictus*, which prefer brackish (saline) water for breeding (McMichael et al., 1996).

Distribution of vector-borne diseases is determined by a dynamic complex of environmental and social

factors. Human behavior is likely to be affected by climate change which may increase the interaction with vectors and the diseases they carry. Globalization of travel and trade, unplanned urbanization and environmental challenges such as climate change are having a significant impact on disease transmission. Some diseases are emerging in countries where they were previously unknown.

- **Mosquito-borne Diseases :**

Mosquito – human- mosquito transmission pathway:

- **Malaria:**

The natural ecology of malaria involves malaria parasite infecting successively two types of hosts: humans and the female blood-feeding *Anopheles* mosquitoes. Malaria is caused by 4 species of plasmodium parasite. *Plasmodium vivax* has the broadest geographical range and *Plasmodium falciparum* is the most dangerous. The plasmodium completes its asexual life-cycle in man and then undergoes sexual development and sporogony in the mosquito.

Evidence of the responsiveness of malaria incidence to local climate change includes the observed marked increase in the incidence and

distribution of malaria during the hot wet weather and the annual fluctuations with variation in the annual temperature and precipitation.

Climate change has a mixed effect on malaria transmission. Temperature, precipitation and extreme weather events can affect the viability and distribution of mosquitoes. In some areas, the geographical distribution ranges will contract due to the lack of necessary humidity and water for mosquito breeding. In other areas, the range will expand and the transmission season may be longer. Some higher altitude mosquito species are able to survive in sheltered places during cold spells but most Anopheline mosquito activity stops when the temperature drops below 22 °C. However, the activity of several species that transmit falciparum malaria generally stops when mean winter temperature drops below 16 - 18°C. Warming is expected to increase survival rates of both vectors and parasites in temperate zones and precipitation directly influences the abundance of breeding sites; however, warming without additional precipitation may serve to reduce mosquito longevity and reduce malaria transmission in tropical areas.

Sporogenic development of *P. vivax* ceases below 14 – 16 °C and below 18 - 20°C for *P. falciparum*. A slight increase in the minimum average temperature will greatly shorten the extrinsic incubation period of the parasites (McMichael et al., 1996).

During drought massive numbers of puddles created in drying river beds can lead to vector explosion.

In Africa it is estimated that in 2100, there will be an increasing number of person-months of exposure to malaria by 16 - 28%. Moreover, it is predicted that by the latter half of the 21st century there will be an increase from around 45% to 60% in the proportion of the world population living within the malaria transmission zone; this amounts to 50 – 80 million additional cases of malaria annually (McMichael et al., 1996).

### **Dengue:**

Dengue is a febrile illness caused by one of four distinct viruses. Infection with one strain does not confer immunity to the others. Subsequent infection with another strain is significantly associated with more severe forms of the disease namely dengue hemorrhagic fever and dengue shock syndrome.

The major vector for dengue is the domestic, container-breeding *Aedes aegypti*. The current range of *A. aegypti* is limited by cold weather, epidemic transmission of dengue is seldom sustained below 20°C. Probably under the effect of climate change it has reappeared in areas from which it has been eradicated, and has been reported at higher altitudes. Climate will affect the distribution, lifecycle and population dynamics of the vector and an increase of 3-4°C would double the reproduction rate of dengue virus.

The disease can also be transmitted by *Aedes albopictus*, an opportunistic dengue vector, which can tolerate cold weather and breeds readily in urban and rural environments, whereas *A. aegypti* is more restricted to cities. *A. albopictus* is found in Asia and has reached the United States, Mexico, the Caribbean, parts of Australia, parts of Africa and Brazil and is spreading still further (McMichael et al., 1996). Climate change will further increase the potential geographic distribution of the vector in Europe. However, for disease transmission to be established, long periods with temperatures that are high enough are required (Lindgren and Ebi, 2010).

### **Yellow Fever:**

Yellow fever is a viral infection confined to tropical climate zones outside Asia and the Pacific. Urban yellow fever is transmitted between humans by *Aedes aegypti*, but the urban cycle starts only if the virus is imported from monkeys in the jungle where the virus is transmitted via other mosquito species among monkeys (emerging disease) (McMichael, 1996).

Climate warming may increase the currently low risk of urban epidemics in East Africa and the Americas. The extrinsic incubation of the virus is influenced by temperature. The survival of *Aedes* mosquitoes is heavily influenced by human behavior particularly the storage of drinking water in small vessels especially during drought.

### **Chikungunya :**

Chikungunya is an emerging viral disease, first described during an outbreak in southern Tanzania in 1952. Beginning in the 1960s, periodic outbreaks have been reported in Asia, Africa and the Indian sub-continent. Since 2005, following several decades of inactivity, the disease has re-emerged and caused larger outbreaks

in Asia, Africa and the Americas. In 2007, an outbreak was reported in Italy and outbreaks of the disease have since been reported in France and Croatia. In Africa outbreaks were reported in the democratic republic of Congo, in the Gabon and in southern Tanzania (ter Meulen, 2010 and WHO, 2016b). The disease is transmitted from man to man by the infected female mosquito bites. Most commonly the mosquitoes involved are *Aedes aegypti* (in tropics and subtropics) and *Aedes albopictus* (in temperate and cold temperate regions). In Africa, several other mosquito vectors have been implicated and there is evidence that some animals may act as reservoirs.

Re- emergence of Chikungunia appears to follow the weather patterns that are associated with climate change.

During drought conditions, *A. aegypti* thrives in water storage containers, and in 2004 Kenya experienced an outbreak after a long period of drought. During rainy periods, however, mosquito reproduction relies on small pools and ponds; central Thailand experienced an epidemic of Chikungunia six weeks following heavy rainfall.

Mosquito-human-mosquito transmission pathway involves also Zika; and Lymphatic Filariasis which is transmitted mainly by *Culex* mosquitoes but also by *Anopheles* and *Aedes* mosquitoes (WHO, 2017).

*Mosquito-animal – mosquito transmission pathway:*

#### **West Nile virus (WNV):**

West Nile virus circulates in a mosquito-bird-mosquito cycle with many bird species involved. Humans are dead-end hosts. An outbreak was reported in Romania in 1996 and the disease is now reportable at the EU level in minor outbreaks and has recently been introduced in the USA. The vector is *Culex pipiens*. Studies show that climatic conditions that favour high population densities of both birds and mosquitoes in the same location precede major outbreaks (Lindgren and Ebi, 2010). A relatively small increase in temperature causes a marked decrease in the incubation period of the virus in the mosquito (Epstein, 2005; Lindgren and Ebi, 2010 and ter Meulen, 2010).

#### **Rift valley fever:**

Rift valley fever is an African disease where the virus naturally causes a zoonotic disease spread

among vertebrate hosts by mosquito species *Aedes*. Under flood conditions, *Culex* mosquitoes also may feed upon infected hosts. The vector is called a "bridge species" because it feeds on humans also resulting in spread of the virus outside its normal zoonotic cycle (Patz et al., 2003 and Lindgren and Ebi, 2010).

#### **Ross River Virus (RRV) Disease:**

Ross River virus causes a non-fatal polyarthrititis which ranges from mild to severe debilitating disease. The virus is maintained in a primary mosquito-mammal- mosquito cycle involving primarily kangaroos and wild rodents and the disease is transmitted mainly through bites of mosquito species *Culex australicus*. Although the disease is little known outside Australia, it is nearly found in all parts of the continent. Very large outbreaks were most often associated with above average rainfall. Sea level rise that floods saltwater marches and coastal wet lands, the habitat of saltwater mosquitoes, can result in outbreaks of the disease. Warmer temperatures generally enhance transmission of the disease. The virus occasionally "spills over" to human populations through a human- mosquito cycle, when a high

level of the virus is circulating in the environment (Woodruff and Bambrick, 2008).

#### **Dirofilariasis :**

The definitive mammalian hosts for *Dirofilaria* are primarily domestic dogs and wild canines. The infective agent is a nematode, *Dirofilaria immitis* and *Dirofilaria repens* worms. Humans are infected with *Dirofilaria* through mosquito bites. Several species of mosquitoes are capable of transmitting the disease including *Aedes* and *Anopheles*. In the definitive host the worm produces microfilariae which, when ingested by the mosquito vector, develop into larvae which are ready to infect another host during a blood meal. Humans and several other mammals are accidental hosts that play no role in disease transmission.

Human dirofilariasis typically manifests as either subcutaneous nodules or lung disease where worms dying in the pulmonary artery branches can produce granulomas that can be seen in X-ray examination as coin shadows.

Changes in disease incidence are attributed to the effect of temperature on the parasite itself, on the density

of the vector population ( and the emergence of *Aedes albopictus* as a competent vector ) and to changes in human exposure. Recent research demonstrates that temperature dictates the development of *Dirofilaria* larvae in the vector with a threshold below which development will not proceed and consequently determines the seasonal occurrence of the disease in temperate latitudes (ter Meulen, 2010). *Dirofilariae* are increasingly recognized worldwide as human pathogens.

Mosquito-animal-mosquito transmission pathway includes also Saint Louis encephalitis, Murray valley encephalitis; and Japanese encephalitis which is transmitted by *Culex* mosquitoes (CDC,2015) .

- **Tick-borne Diseases (TBDs) :**

Ticks are widely distributed around the world including both temperate and tropical regions. They tend to flourish more in countries with warm and humid climates and are more active during warmer months although this varies in different geographic areas. Increases in the length of the tick activity seasons are correlated with the number of days of milder winters. Changes in temperature and precipitation patterns and in the length and weather in different seasons

will affect the geographical distribution, seasonality and incidence of tick-borne diseases.

Tick abundance decreases with altitude, higher altitudes are cooler (Gray et al., 2009).

For *Ixodes scapularis* ticks that transmit Lyme disease, a suitable microclimate for development and survival includes : suitable host availability; temperature fluctuations between 10 and +35°C with tolerance to the extremes for only brief periods, and a constant relative humidity not less than 80% in the air and near saturation in the soil. Increasing day light and high temperature affect tick behavior and increase the host questing activity and biting rates of ticks. Ticks are very sensitive to desiccation; severe drought significantly lowers tick density (Estrada-Pena et al., 2012).

Tick survival strongly depends on climate conditions found on the ground which may be very different from local ambient conditions. Climate change will impact the number of infected ticks by affecting vegetations. Areas with woods, bushes and high grass or leaf litter are likely to have more ticks. In warmer weather, fallen leaves provide a suitable microclimate for survival of free-living tick stages.

In contrast to tick species that are dependent on the host for their survival, species that spend most of their lives off the host are more sensitive to external conditions caused by climate change (Olson and Patz, 2010).

Climate change impact on TBDs is further complicated by the way it alters host diversity, abundance and dispersal; the development cycle of the pathogen; and the suitability of habitat for both tick and host. Climate change affects the survival and abundance of tick-maintenance hosts such as deer; and

pathogen reservoir hosts such as birds and rodents.

The magnitude of the effect of climate change on TBD transmission in an endemic area depends also on ecological as well as socio-economic factors; human migration and settlement; ecosystems and biodiversity; migrating patterns of birds; land use and land cover; changed human cultural and behavioral patterns and immunity of the population. Human response to warm climate encourages outdoor recreational activities such as picnics and allows more contact with ticks.

### **Examples of infectious tick-borne diseases and their pathogens:**

*Bacteria and rickettsia :*

Lyme disease ( Borreliosis ), Tick-borne relapsing fever, Typhus.

*Rickettsial diseases :*

Rocky mountain spotted fever and Q fever, Ehrlicosis anaplasmosis ( formerly human granulocytic ehrlicosis HGE ), Tularemia

*Viruses :*

Tick-borne meningoencephalitis, Crimean-Congo haemorrhagic fever

*Protozoa :*

Babesiosis

Of the more prevalent tick-borne diseases are Lyme disease and tick-borne meningoencephalitis. These are transmitted by ticks *Ixodes ricinus* and *Ixodes persulcatus* .



- **Sandfly-borne diseases :**

- **Lieshmaniasis :**

The most common types of Lieshmania are Lieshmania tropica which causes cutaneous lieshmaniasis (mostly in Europe); and Lieshmania infantum which causes visceral lieshmaniasis (VL) (mostly in the Mediterranean area) , which infects the liver and the spleen and may be fatal. The two Lieshmania species are transmitted by Phlebotomus sandflies.

Lieshmania is found in the tropics, subtropics, southern Europe and the Mediterranean region. Sandflies do not require standing water for breeding. The parasite lives and multiplies in the sandfly; infected animals such as dogs and rodents can maintain the transmission cycle. However, in some parts of the world infected people are required to maintain the cycle (anthroponotic cycle).

Sandflies are very sensitive to temperature and with increasing temperatures the geographical range of sandflies will expand (ter Meulen, 2010). With the more uncontrolled movement of pet dogs the pathogen is now present in many countries.

Leishmaniasis infection rarely causes symptoms in healthy individuals. It has lately emerged as a health problem because of the co-infection of the Leishmania parasite with HIV / AIDS. Co-infection causes a mean survival of 13 months. Treatment of HIV infection reduces the incidence of co-infection but not relapses of VL. The effectiveness of treatment of leishmaniasis is markedly reduced in case of co-infection with HIV. About 400 – 500 cases of leishmania/HIV are reported in Europe every year (Lindgren and Ebi, 2010 and ter Meulen, 2010).

- **Rodent-borne diseases :**

Rodents are involved in the transmission of a number of human diseases. They shed pathogens in their urine, feces or saliva. Standing water contaminated with urine, droppings or saliva of rodents after flooding has been linked to outbreaks of leptospirosis; transmission of the disease occurs by contact with the contaminated water.

Man can develop Hantavirus pulmonary syndrome, neuropathia epidemica or tularemia if he breathes in droplets of air contaminated with the virus (Lindgren and Ebi, 2010). The increased weather variability that accompanies climate change

has contributed to the emergence of Hantavirus pulmonary syndrome in the United States (Epstein, 2001).

In addition, rodents are reservoirs for the pathogens transmitted by fleas (Plague). Climate plays a role in the annual seasonality of Plague; the potential effects of temperature rise and abundant rains associated with climate change on flea survival and reproduction and on rodent prevalence are important factors in the spread of the disease in rodents and subsequently in man (ter Meulen, 2010).

Rodents are affected by seasonal climatic conditions; milder winters, heavy rainfall and prolonged vegetation periods may contribute to increased food availability for rodents. Drought followed by heavy rainfall will boost the conditions leading to a larger rodent population. Prolonged drought may decrease rodent population density. Floods will drive rodents from their burrows to safer places in human dwellings increasing the risk of infection. Changes in land use and specific ecological conditions like the interaction between rodent and predator population are important rodent population modifiers.

- **Diseases Transmitted by Other Vectors :**

- **Human African Trypanosomiasis (Sleeping Sickness):**

The parasite is a trypanosome species and the vector is the blood-feeding tsetse fly which does not depend on a water habitat. Different species of the parasite and vector are implicated. The disease is prevalent in west and central Africa and in south Asia. The number of reported cases is small but the disease is usually fatal if not treated.

Tsetse fly depends on shrub and tree cover and the distribution is negatively affected by desertification and drought. Land use changes can affect the suitability of environmental conditions for the fly through their impact on temperature and relative humidity.

- **American Trypanosomiasis (Chagas Disease) :**

Chagas disease is caused by parasite *Trypanosoma cruzi* and is transmitted by the blood-feeding triatomine bug. In 1996 it was estimated that one hundred million people are currently at risk of the disease and 18 million were infected. The distribution of the disease is limited to south and central America where it is a major public health problem (McMichael et al., 1996).

Triatomine bugs are sensitive to small variations in temperature and humidity. At higher temperature the vector feeds more frequently, has a shortened life cycle, mates more frequently and lays more eggs resulting in an increase in the population density. In hot dry conditions, the bug feeds more to avoid dehydration. Higher temperature could extend the geographic distribution of the vector.

#### **Onchocerciasis ( River Blindness) :**

The disease is prevalent in Latin America and west Africa. The infective agent is the nematode *Onchocerca volvulus* and is transmitted by the Black fly. The nematode damages the skin, the lymphatic system and, in most cases, the eye. Given that the vector depends on fast-flowing river water for successful reproduction and that it can be spread by wind, climate plays an important role in onchocerciasis incidence (WHO, 1985). The disease is prevalent in Latin America and west Africa.

- **Diseases Transmitted by Aquatic Snails :**

#### **Schistosomiasis:**

Schistosomiasis is a water-based infection caused by 5 species of a trematode *Schistosoma*: *S.*

*haematobium* and *S. mansoni*, the ones in Egypt and *S. japonicum*, *S. mekongi* and *S. intercalatum* in other parts of the world. Symptoms vary but include bloody urine and liver disorders. Worldwide prevalence has risen since the middle of the 20th century. Water shortage due to climate change created greater need for expansion of irrigation systems, particularly in arid regions, where specific viable snail populations can find human parasite carriers. The mature parasite's eggs are excreted in urine or stools and often enter surface water where they hatch into larval miracidia capable of infecting the snail ( the intermediate host ). Several species of snails are responsible for carrying infection. The parasite multiplies in the snail and one miracidium produces several hundred cercariae capable of infecting humans.

Temperature influences snail reproduction and growth; schistosome mortality, infectivity and development in the snail and there is increased human-water contact. During winter in many countries the water snails tend to lose their schistosome infection. But if temperature increases, snails may mediate schistosomiasis transmission for a longer period. Mathematical models

indicate that change in temperature may cause infection to spread beyond the current distribution area; however, the transmission potential will decrease in some areas that are currently endemic because temperatures may be too high for the snail and parasite (McMichael, 1996).

Indirect effect of climate change could increase the incidence of Schistosomiasis. Water shortages due to climate change could create a greater need for irrigation, particularly in arid regions. If irrigation systems were extended, snail population would probably increase leading to greater risk of infection. Higher temperatures also increase human water contact.

### **Emerging Infectious Diseases:**

Definition: An emerging infectious disease is:

- an infectious disease that is newly recognized in humans;
- one that has been recognized before, was thought to be under control but is newly appearing in a different population or geographic area than previously affected;
- one that is newly affecting many more individuals;

- one that was previously recognized but is now transmitted by another vector or the route of transmission has been identified recently; or
- one that has developed new attributes (e.g. pathogen virulence, resistance to antibiotics or host susceptibility) (US government, 2014).

Most emerging diseases are caused by changes in “microbial traffic” that is, the introduction of existing agents into, or the dissemination of existing agents among, human populations. These agents may be from other geographic areas, or transmitted from an animal reservoir or from smaller human populations such as indigenous groups living in remote isolated areas where the agent might have been circulating in small silent outbreaks.

The risk of emerging diseases will increase due to changes in survival of pathogens in the environment, changes in the pathway of carriers or vectors and shifting of the distribution ranges of animal species.

Factors contributing to the emergence and resurgence of diseases include environmental change such as climate change; socioeconomic change; health care interventions; changes in

food production and distribution; human behavior; public health infrastructure; microbial adaptation; rapid population growth and high density human settlements; greater human mobility and travel; inappropriate use of pesticides and antibiotics; and political disruption (McMichael et al., 1996).

Climate change, combined with global mobility and forced migration under the effect of extreme weather events or sea level rise, which result in displacement of human populations, will result in previously unforeseen evolution of emerging and resurging infections and their redistribution across the planet.

Deforestation and changes in biodiversity have altered the balance among predators, competitors and prey which helps keep pests and pathogens in check, have apparently contributed to emergence and resurgence of some infectious diseases.

The following are some illustrative examples of emerging diseases and the climatic conditions involved in their emergence:

#### **West Nile Virus (WNV) Disease:**

WNV disease is a viral zoonotic disease maintained in a mosquito-bird-

mosquito cycle; humans being dead-end hosts. WNV was first reported in Uganda in 1937. Significant outbreaks that occurred in Romania in 1996 and in Russia in 1999 followed prolonged droughts and excessive heat. WNV was imported in the USA in 1999, probably via migratory or imported birds; four large outbreaks revealed that drought was a common feature.

During drought, *Culex pipiens*, the primary vector for WNV, thrives in city storm drains, catch basins and unused pools, especially in the water originally rich in organic matter which becomes even richer during drought; and in hot temperatures. Moreover, while mild winters and dry summers favour the breeding of the mosquitoes, mosquito predators (amphibians and dragonflies) can decline in number. Also, during drought, birds congregate around these scanty water sites where the virus can circulate more easily between urban mosquitoes and birds. Mild winters coupled with prolonged droughts and heat waves amplify WNV and shorten its extrinsic incubation period. Rain that follows hot summer may help to unleash crops of *Aedes* mosquito species that may act as additional “bridge vectors” carrying infection to man (Epstein,

2001 and Lindgren and Ebi, 2010).

### **Saint Louis Encephalitis (SLE):**

St. Louis encephalitis is a zoonotic disease, maintained in a mosquito-bird-mosquito cycle. Wild birds are the primary vertebrate hosts. Vectors are *Culex* species mosquitoes. Together with birds that are abundant in urban and sub-urban environments, mosquitoes are involved in periodic amplification of the virus. Birds sustain an unapparent infection but develop viraemia that is sufficient to infect the mosquito vectors. Humans and domestic mammals can acquire infection but are dead-end hosts (CDC, 2009). St. Louis encephalitis was first reported in 1933 in the USA. Several outbreaks were associated with 2 months of drought; however, between 1974 and 1976 and after; outbreaks showed a variable relation to weather. However, once established in a region, summer rains may boost populations of *Aedes japonicus* and other *Aedes* species that function as “bridge vectors” carrying the virus from birds to man (Epstein, 2001).

### **Hantavirus Pulmonary Syndrome (HPS):**

Rats and mice are reservoirs for Hantaviruses. Rodents shed the virus in

their urine, droppings and saliva. The virus may be transmitted to humans when contaminated water is stirred up, aerosolized and drops of these aerosols are breathed in (air-borne transmission). Rodents are affected by seasonal climate conditions. In 1987 to 1993 in the United States changes in land use and prolonged droughts reduced natural predators of rodents (owls, eagles, falcons, snakes etc...). When droughts yielded to intense rain in the winter of 1993, grasshoppers and pinion nuts, on which rodents feed, flourished boosting mouse population tenfold leading to the emergence of a new disease (McMichael et al., 1996 and Epstein, 2001).

### **Murray Valley Encephalitis (MVE):**

MVE virus is endemic in the northern regions of Australia and in Papua New Guinea. The virus is maintained in a mosquito- bird-mosquito cycle. Humans are dead-end hosts where the virus causes permanent neurological disease or death. Water birds are the natural vertebrate reservoirs and the mosquito vector is *Culex annulirostris*. The first epidemics occurred in the south eastern states of Australia in 1917 and 1918 following years of heavy summer monsoon rainfall and flooding

of the Murray-Darling river system. Infected birds or infected mosquitoes migrating from the endemic areas in the north, associated with an increase of the bird and mosquito population in the south, lead to virus overflow infecting humans.

It is uncertain whether the virus was also endemic in the southern states at undetectable levels and became evident with periods of intense breeding of birds and mosquitoes following excessive flooding (Einseidel et al., 2003).

### **Zika :**

Zika virus was first identified in Uganda in 1947 in monkeys. It was later identified in humans in 1952 in Uganda and the United Republic of Tanzania. The disease is transmitted between humans by the female mosquito *Aedes aegypti*; *Aedes albopictus* is also implicated. The virus has gradually spread through the tropics. The first large outbreak was reported in 2007. From 1960s to 1980s, human infections were found across Africa, the Americas, Asia and the Pacific region (WHO, 2016<sup>c</sup>).

Favorable climatic conditions for *A. aegypti* contributed to the explosive spread of Zika in Brazil in 2015. El

Niño weather phenomenon, which originates in the Pacific ocean, tends to increase global temperature for a couple of years by releasing heat from the ocean to the atmosphere. This weather event resulted in exceptionally high temperatures accompanied by severe drought, throughout the second half of 2015. Although precipitation provides essential habitat for the vector, drought can indirectly expand the vector's range; in several locations, range expansion of *Aedes aegypti* was correlated with increase in water storage in household containers during severe drought. It was noted that regions with extreme climatic condition in one month were subject to expansion of the geographic distribution of Zika virus in the subsequent month.

### **References**

1. Centers for Disease Control and Prevention, National Center for Emerging and Zoonotic Infectious Diseases, Division of Vector-borne diseases (2009): Saint Louis Virus Encephalitis. CDC.
2. Dobson A and Carper R (1993): Health and climate change: Biodiversity. *Lancet*; 342: 1096-99.
3. Einseidel L, Ravindran J, Slavotinek J and Gorgon DL (2003): MR Findings in Murray Valley encephalitis. *Amer J Neurol*; 24: 1379-82.
4. Epstein P (2010): The ecology of climate change and infectious diseases: Comment. *Ecology*; 91 (3): 925-8.
5. Epstein P (2005): Climate change and human health. *New Engl J Med*; 353 (4): 1423-6.

6. Epstein P (2002): Climate change and infectious diseases: Stormy weather ahead. *Epidemiol*; 13 (4): 373-6.
7. Epstein P (2001): Climate change and emerging infectious diseases. *Microbes infect*; 3: 747-54.
8. Estrada-Pena A, Ayllon N and de La Fuente J (2012): Impact of climate trends on tick-borne pathogen transmission. *Front Physiol*; 3: 1-12.
9. Government of South Australia (2012): Ross River virus Infection. South Australia Health, Health Topics. SA.
10. Gray J S, Dautel H, Estrada-Pena A, Kahl O and Lindgren E (2009): Effects of climate change on ticks and tick-borne diseases in Europe. *Interdiscip Perspect Infect Dis*; 2009: 1-12.
11. Intergovernmental Panel on Climate Change (IPCC) (2007): Fourth Assessment Report.
12. Intergovernmental Panel on Climate Change (IPCC) (2001): Third Assessment Report.
13. Kuhn K, Campbell-Lendrum D H, Haines A and Cox J (2005): Using Climate to Predict Infectious Disease Epidemics. World Health Organization. Geneva.
14. Lindgren E and Ebi K L (2010): Climate change and communicable diseases in EU member states. Technical Document 1. Climate change-related infectious diseases, Appendix 1, European Center for Disease Prevention and Control. Stockholm.
15. McMichael A J, Haines A, Sloof R and Kovats S (1996): Climate Change and Human Health, Chapter 2, the Climate System. World Health Organization. Geneva.
16. Olson S H and Patz J A (2010): Global Environmental Change and Tick-borne Disease Incidence. The State of The Science Workshop entitled "Critical Needs and Gaps in Understanding Prevention, Amelioration and Resolution of Lyme Disease and Other Tick-borne Diseases: Short-term and Long-term Outcomes". Institute of Medicine Committee on Lyme Disease and Other Tick-borne Diseases. Washington D.C.
17. Patz J A, Githeko AK, McCarty J P, Hussein S, Confalonieri U and Wet N (2003): Climate change and infectious diseases, Chapter 6. In. A J McMichael, D H Campbell-Lendrum, C F Corvalan, K L Ebi, A K Githeko, J D Sheraga and A Woodward. *Climate Change and Human Health, Risks and Responses*. World Health Organization. Geneva.
18. Shuman E K (2010): Global climate change and infectious diseases. *N Eng J Med*; 362: 1061-63.
19. The United States Government and Global Emerging Infectious Disease Preparedness and Response (2014): Emerging infectious diseases. Available at : <http://files.kff.org/attachment/the-u-s-government-global-emerging-infectious-disease-preparedness-and-response-fact-sheet>
20. ter Meulen V (2010): Climate change and infectious diseases in Europe. European Academies, Science Advisory Council.
21. Woodruff R and Bambrick H (2008): Climate change impacts on the burden of Ross River virus disease. Garnaut Climate Change Review. National Center for Epidemiology and Population Health, Australia National University. Sydney. Australia.
22. World Health organization (2017): Lymphatic Filariasis. Media Center. Fact Sheet. WHO. Geneva.
23. World Health Organization (2016a): Vector-borne Diseases. Media Center, Fact Sheet. WHO, Geneva.
24. World Health Organization (2016b): Chikungunya. Media Center, Fact Sheet. WHO, Geneva.
25. World Health Organization (2016c): Zika. Media Center, Fact Sheet. WHO, Geneva.
26. World Health Organization (1985): Review of the work on the onchocerciasis control programme in the Volta River basin area from 1974 – 1984. In: *Ten Years of Onchocerciasis Control in West Africa*. WHO, Geneva.