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# Impact of Climate Change on the Geographic Scope of Diseases

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Additional information is available at the end of the chapter

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## 1. Introduction

The Intergovernmental Panel on Climate Change [1] has shown that the climate will change, and provides different scenarios on what may happen and when, while the Millennium Ecosystem Assessment [2] has shown how the functioning of ecosystem services contribute to the maintenance or otherwise of human health. It is recognized that the main brunt of the effects of anthropogenic climate change will be borne by communities in developing countries, particularly in Africa. For example, the IPCC forecasts that some parts of Africa will become warmer and wetter, whereas others will become drier, and there will be higher frequencies of storms and floods. Changes in the distributions and amounts of rainfall will ensue, as water is a fundamental human requirement that also influences human wellbeing and health, and understanding how water systems will change is a pre-requisite to understanding potential future changes in disease epidemiology.

A possible scenario that could involve the spread of diseases into non-endemic regions is with the migration of infected people in places where vectors are already present but, as yet, there is no disease. For instance in South Africa, there are three newly described forms of *S. damnosum* (vectors of onchocerciasis) present, with one (the Pienaars form) [3] identified from sites where man-biting is known. This habit appears to be spreading in South Africa in the Johannesburg area and two separate sections of the Orange river [4]. Furthermore, it is known that there is extensive migration into South Africa (both legal and illegal) of people from countries where onchocerciasis is endemic. Such migration is likely to expand with increasing environmental degradation and poverty.

This chapter discusses how future climate change scenarios could influence disease transmission and distribution, for better or for worse; by delving into the past, present and future effects of climate on diseases using malaria as an example. It will also elaborate on the environmental factors affecting disease agents, the effects of climate on the physiological

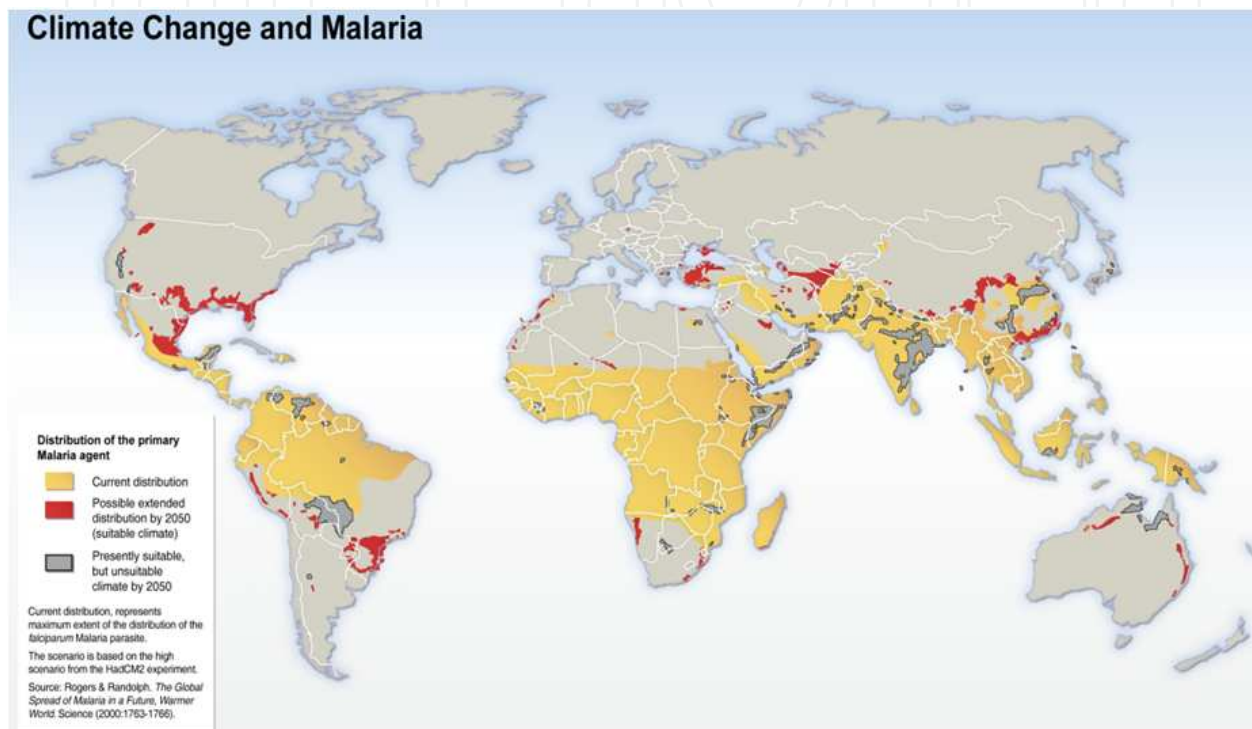
processes of pathogens and the host-pathogen interactions, vector diversity and vector borne diseases, as well as transmission zones related to disease distribution. Finally, the chapter examines the effects of climate change on migration and how this may impact disease distribution.

## 2. Changing patterns of disease distribution with climate: Past, present and future situation

The geographic distribution of diseases has revealed changes over history [5]. Several factors can be linked to the changes in disease distribution, but of interest to this review on climate change and disease distribution, are global warming, agricultural colonization, deforestation and reforestation. Changes in climate, either at the micro (country) or macro-geographical (continent) level influence the survival, reproduction, transmission of disease agents and vectors, and their interaction with the geophysical factors associated with climate: primarily precipitation, humidity, ambient and water temperature. A considerable range of diseases, including cholera [6], lymphatic filariasis [7] and tick-borne encephalitis [8] are affected by changing environmental conditions. However, alongside schistosomiasis and dengue, malaria is one of the diseases that has mostly been influenced by climate change events [9-11] and provides us with adequate information required to understand the roles that climate plays in driving disease prevalence and distribution.

*Plasmodium falciparum* malaria has had a long evolutionary history with humans [12-14], dating back to 10,000 years ago [15, 16], coinciding with human population growth and the change from hunter-gather's behavior to that of agriculture [17, 18]. The plausible distribution of malaria prior to intervention programs [19, 20], was believed to reach latitudinal extremes of 64° north and 32° south [21], at the turn of the 20th century. The 20th century, however, was undoubtedly a period of global climate change [1], and, using this era as a backdrop, many empirical and biological climate-malaria models [8, 10, 22-24] have sought to predict the future impact of climate change on the distribution of this disease. Climate variables such as temperature, humidity and rainfall affect the incidence and distribution of malaria, through changes in the vector and parasite life cycles and behavior [25, 26]. Studies in the Amazon Basin suggest that precipitation drives malaria incidence. However, this relationship varies in the uplands where more precipitation corresponds with more malaria, and is negative in areas dominated by wetlands and large rivers [27]. In determining the climate effects on malaria transmission, studies on *Anopheles stephensi* have shown that temperature affects sporogonic development of *P. falciparum* by altering the kinetics of ookinete maturation [28]. Ookinete development and blood meal digestion are lengthened as temperatures decrease from 27-21°C. Nevertheless, low temperatures (21-27°C) do not appear to significantly influence infection rates or densities of either ookinetes or oocysts. On the other hand, high temperatures (30 and 32°C) appeared to significantly impact parasite densities and infection rates by interfering with developmental processes occurring between parasite fertilization and ookinete formation, especially during zygote and early ookinete maturation [27]. Paaijmans and colleagues [29] further showed that the

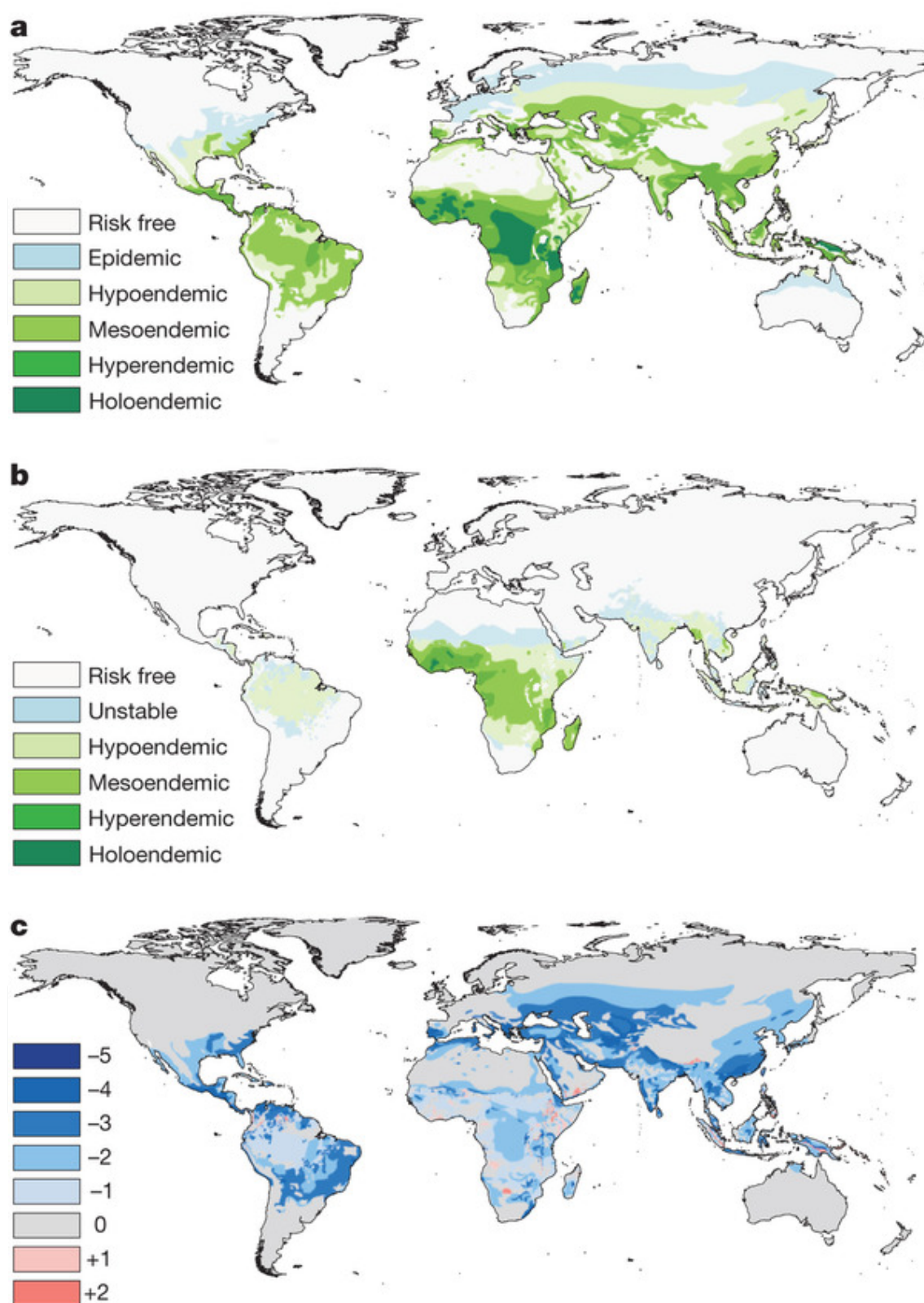
influence of climate on malaria transmission depends on daily temperature variations, with temperature fluctuations around low mean temperatures acting to speed up rate processes, whereas fluctuations around high mean temperatures act to slow down the processes. Despite all the evidence of climatic effects on malaria transmission, evidence-based maps of contemporary malaria endemicity [20] permit a re-evaluation of the changes in global epidemiology of the disease, when compared to the pre-intervention era [19] and enable an assessment of the observed changes in range and endemicity to those proposed to occur in response to climate change and observed under existing public health interventions [31].



**Figure 1.** Climate change and malaria, scenario for 2050 (UNEP/GRID 2005). With climate conditions changing in the future, due to increased concentrations of carbon dioxide in the atmosphere, conditions for pests also change. The primary malaria agent, the *falciparum* malaria parasite, will be able to spread into new areas, as displayed in this map, by 2050 using the Hadley CM2 high scenario. Other areas, not displayed in the map, will be uninhabitable by the parasite.

Based on the biology of the vectors and their absence in regions that at present are too cool for their survival [10, 32], climate-malaria models depict an increase in the geographic range of the disease [33, 34], as temperature and global environmental conditions worsen. Figure 1, reproduced from UNEP/GRID 2005, represents the global distribution predictions of malaria by 2050. The changing climate conditions, due to increased concentrations of carbon dioxide in the atmosphere, will result in heat being trapped herein and thus modify the conditions for malaria vectors and *P. falciparum*, and malaria will be able to spread into new areas. However, despite the influence of climate on the distribution of the vectors, the predicted climate-malaria models hold true only if no actions are taken. Figure 2 reproduced from Gething *et al.* [31], represents the changing global malaria endemicity since the turn of the 20th century. Despite the global temperature increases in the 20th century [1, 35] the





**Figure 2.** a) Pre-intervention endemicity (approximately 1900) as defined in Lysenko and Semashko [19]. b) Contemporary endemicity for 2007 based on a recent global project to define the limits and intensity of current *P. falciparum* transmission [30] and c) Change in endemicity class between 1900 and 2007. Negative and positive values denote reduction and increase respectively in endemicity. (Source: [31]).

figure depicts a marked, global decrease in the range and intensity of malaria transmission. The study also revealed that comparison of global-scale climate patterns with the historical and contemporary patterns of malaria endemicity, pointed out a disconnection in climate–malaria relationship over the twentieth century, indicating that non-climatic factors act as confounders of this relationship over time. Most malaria-climate models assume that: 1. all other factors affecting the distribution of malaria (such as disease and vector control efforts) either remain constant or have a relatively negligible effect; and 2. the link between climate and the global distribution and intensity of malaria is effectively immutable: an empirical climate–malaria relationship observed at one time period will be preserved even under changing climate and disease scenarios [31]. As such, contemporary endemicity maps provide a poor baseline for empirically-based predictions of future climate effects and for the climate-malaria predictions to hold true, the climate change effects must exceed the counteracting socio-economic development [36] and aggressive malaria control efforts [37–39].

### 3. Environmental factors affecting disease agents

Disease agents are affected by environmental factors, notably rainfall, humidity, and temperature. These, in turn, influence the incidence, distribution and spread of water-borne diseases. Water-borne and sanitation-related diseases are major contributors to global disease burden and mortality [40], especially in developing countries and in children under the age of 5 years. The main agents of these diseases are mostly viruses, bacteria, and parasitic worms [41]. Being mostly linked with water and sanitation, these diseases such as Cholera, typhoid, schistosomiasis, guinea worm and toxoplasmosis are prone to weather fluctuation events that affect their survival and dispersal through this medium.

Various studies have shown that many disease outbreaks associated with water are preceded by heavy rainfall events [42–48]. An example of this phenomenon is with the cryptosporidiosis outbreak associated with borehole extracted groundwater, where heavy rainfall led to running water from, fecally polluted, cattle grazing fields leaking into the borehole water supply [44]. Other studies have shown correlations between rainfall and the likelihood of detecting *Giardia* or *Cryptosporidium* oocysts in river water [49] and pathogenic enteric viruses in water [50], as well as coliform re-growth in water distribution systems, presumably because of increased nutrients in water [51].

Flooding, following heavy rainfall events, also poses substantial risk to the spread of infectious diseases –especially in developing countries where cases of leptospirosis, Hepatitis E, malaria and diarrheal diseases have been reported [52–57]. Most often, rainfall- and flood-associated outbreaks often come about as a result of contaminated water seeping into groundwater or water treatment systems [58] or the provision of suitable breeding grounds for disease vectors, in the case of malaria [59, 60].

Of all the climate change events, temperature poses the greatest threat to the spread of infectious diseases, as the increase in temperature has been linked to the blooming of

various planktonic species [61-63] and pathogens [64, 65]. Planktonic blooms occur mostly in the summer months and the accumulation of toxins either through contact with water or food relating to these, such as blue-green algae [61] and dinoflagellates [63], results in cases being reported around these periods. However, the most evidence of the effect of temperature on risk from waterborne disease is in relation to cholera [64]. Evidence suggests that *Vibrio cholerae* survives in marine waters in a viable but non-cultural form that seems to be associated with algae and plankton [5, 64]. This is supported by observations that revealed that increases in sea-surface temperature, as a result of El-Nino events, have resulted in cholera outbreaks in Asia and South America [65, 67, 68].

Temperature does not only affect the growth of enteric pathogens, but also affects the spread of many viral, rickettsial, bacterial and parasitic diseases that can be transmitted by vectors [69]. Gubler and colleagues [25] list a range of possible mechanisms that may affect the risk of transmission of vector-borne diseases as a result of temperature changes. These mechanisms include the increase or decrease in vector survival, changes in rate of vector population growth, changes in feeding behavior, changes in susceptibility of vector to pathogens, changes in incubation period of pathogen, changes in seasonality of vector activity and changes in seasonality of pathogen transmission. These changes will be discussed in subsequent sections.

#### **4. Climate change effects on pathogen strains, host-pathogen interactions and their physiological processes**

Pathogen strain may play a significant role in the host-pathogen or vector-pathogen interactions. This section will try to reveal the latitudinal and temperamental changes on the diversity in pathogens and their interaction with their hosts and vectors.

Seasonal disease variations may come about as the result of many factors, including seasonally heightened host susceptibility resulting from seasonal stressors [70], changes in contact rates resulting from school terms in the case of childhood diseases [71] and seasonal changes in pathogen transmission rates resulting from climate variation [72]. Temperature, which is the main climate change driver, can affect disease by altering the susceptibility of hosts, the virulence of pathogens and the growth rates of both hosts and pathogens, which can in turn influence host pathology and disease emergence [73, 74]. The direct influences of climate variables (e.g. geography, temperature and rainfall) on the geographical range, growth rates of pathogens, variations in pathogen transmission rates and disease incidence is an active area of research in disease ecology [23, 64, 75, 76].

Cholera is an infectious disease endemic to many developing countries. It is caused by the bacterium *Vibrio Cholerae*. In Bangladesh, strains within the 01 serogroup of cholera are designated as being either of the "Classical" or the "El Tor" biotype, and both biotypes are affected by differing seasonal signatures with pronounced seasonality in disease incidence [72, 77]. The Classical strain was believed to be the dominant strain until the 1970s, when it was replaced by the El Tor strain which is more resilient to fluctuations in water

quantity and quality, as evidenced by the less pronounced seasonal variability of its reproductive rates [70]. As such, Koelle and colleagues [70] have shown that the cholera strain sensitivity to environmental fluctuations can be considered a phenotypic character trait subject to evolution. Unlike the Classical, El Tor expresses *vps* (Vibrio polysaccharide) genes responsible for producing an exopolysaccharide that allows it to form a biofilm on abiotic surfaces, thus facilitating its environmental persistence [78]. However, the expression of this exopolysaccharide also appears to reduce intestinal colonization, and leads to a reduced infectivity and virulence [79], giving the Classical the advantage of easier infection and virulence. Despite the apparent evolutionary advantages or disadvantages, cholera cases due to both strains decrease during summer, and this is explained as the result of a dilution effect by the monsoon rains and the concurrent reduction in water salinity levels [80].

Small organisms tend to have faster generation times, stronger growth responses to temperature and wider thermal windows [81]. While it is easy to find variations in intra or extracellular bacterial pathogen strains, diversity in extracellular disease agents is not uncommon. For example, in West Africa, two geographic strains of *Onchocerca volvulus* (the parasite causing river blindness) have been observed and described as the forest and savannah forms [82], and these play important roles in the pathogenicity of the disease, as they transmit the severe –blinding form and the less severe, non-blinding onchocerciasis. The existence of geographic strains of parasites, influenced by environmental factors, has also been reported in *Wuchereria bancrofti* [83, 84].

As evidence suggests, climate-driven changes in interspecific interactions may lead to important consequences for host–pathogen relationships and disease emergence [85]. Because temperature patterns control growth and reproduction in a variety of organisms [86], changes in temperature are likely to influence the rate and timing of development of some species more strongly than others [87]. Thus, climate change will likely lead to both direct (i.e., physiological) and indirect (i.e., interspecific interactions) effects on parasite transmission, some of which may increase disease while others will reduce infection or pathology [87].

## 5. Vector diversity and vector borne diseases

The term “vector-borne diseases” describes illnesses in which pathogenic microorganisms such as viruses, bacteria, parasites, and fungi, are transmitted to humans or other animals through the feeding activity of blood- sucking arthropods.

The underlying mechanisms through which climate change may influence the infectivity of vector-borne diseases may be attributed to four main factors, including (i) expanding the range of vectors and hosts into new human populations, (ii) changes in vector or host population density. Furthermore, such changes in host/vector populations, as well as extreme environmental conditions affecting host immuno-competence may impinge on (iii) the frequency of infections, and (iv) pathogen load [88].



Under the second mechanism, high records of rainfall create convenient breeding grounds and increase food availability for vectors such as mosquitoes, ticks, and snails [88]. In South America, for example, following the abundant rainfall that marked the El Niño Southern Oscillation (ENSO) there came about a corresponding increase in *Aedes* mosquito populations [90, 91] while in Senegal and Niger, a lower incidence of malaria was attributed to diminished mosquito populations following reduced amounts of rainfall [92, 93].

Climate change results in the distribution of vectors into non-endemic areas, as well as their proliferation in endemic areas. Many vector-borne diseases are transmitted between a lower end range of 14–18°C and an upper end range of 35–40°C [89]. As lower end temperatures increase, the transmission periods of infectious diseases such as malaria, encephalitis, and dengue fever are enhanced or extended [94]. With malaria, the enhancement of transmission becomes imminent as temperature increases cause adult female mosquitoes to digest blood and feed more frequently, and as warmer waters shorten the incubation period of vector larvae [89]. Between the temperature ranges of 30–32°C, vectorial capacity increases substantially due to a decrease in the extrinsic incubation period, despite a reduction in the vector's survival rate. Mosquito species such as the *Anopheles gambiae*, *Anopheles funestus*, *Anopheles darlingi*, *Culex quinquefasciatus* and *Aedes aegypti* are responsible for transmission of most vector-borne diseases, and are sensitive to temperature changes as immature stages in the aquatic environment and as adults. Increases in water temperatures lead to faster maturation period for larvae [95] and a greater reproduction rate during disease transmission periods. In warmer climates, adult female mosquitoes digest blood faster and feed more frequently [96], thus increasing transmission intensity. Similarly, malaria parasites and viruses complete extrinsic incubation within the female mosquito in a shorter time as temperature rises [97], thereby increasing the proportion of infective vectors. It is also noted that warmer temperatures in temperate regions result in the “overwinter survivorship” of the North American deer mice, which are carriers of hantaviruses, in southwest United States following the 1997 ENSO [98]. On the other hand, extreme temperatures above the upper end generally disfavor the survival rate of vectors and pathogens [95], as witnessed in Senegal, where over the last three decades the incidence of malaria has been reduced by more than 60% [99]. Warming above 34°C generally has a negative impact on the survival of vectors and parasites [95]. Thus, as global temperature conditions worsen, there will be a shift in the occurrence and distribution of vector-borne diseases, as areas at the lower temperature range of disease transmission will see an increase in disease incidence, while areas at the upper temperature will see a decrease.

Stress, attributed to extreme environmental conditions, challenges the tolerance level of the hosts' immune system and makes them more susceptible to pathogen infections and recrudescence [88]. Temperature variations affect pathogen carrying capacity in arthropod vectors, and consequently, the epidemiology of infectious disease.

Moreover, climate change influences the seasonality of infectious disease patterns. Anomalies in weather patterns bring about irregularities in pathogen abundance, survival within both vectors and hosts, or virulence, impairing the accuracy of surveillance systems

in predicting pandemic outbreaks [100]. It is hypothesized that seasonal changes in photoperiod affect vitamin D metabolism [101], and thus immune cell antibacterial and antiviral activity, leading to host immuno-suppression during the winter season. It is also hypothesized that in the northern and southern hemispheres, seasonal changes in photoperiods may bring about changes in flight and feeding activity of arthropod vectors to result in increased transmissibility of pathogens to hosts.

In terms of vector diversity, the environmental factors affecting the distribution of the vectors have the potential to result in their diversification. West Africa is one region with various ecological zones ranging from an extreme of dense forests, through the savannahs, to the other extreme of the Sahara desert. In this region, the *Anopheles gambiae* is highly diversified, with diversity reported in terms of sibling species, chromosomal and molecular forms. Studies have shown that environmental factors may predict the distribution of the observed diversity within this group of mosquitoes [102-104]. An important question, however, is the reason why these differences in the *An. gambiae* are only reported in west Africa and not anywhere else on the continent. The answer to this question may possibly be found in the existence of several ecological zones within this region. Similarly other examples of vector diversity exist as well. Until the discovery of cytotypes, *Simulium damnosum* was believed to be a single species, exhibiting little morphological variation, although differences in behavior and ecology were observed within the same area [105] and also behaved differently in different areas [106]. Other examples of diversity in vectors of diseases can be found in the *Glossina palpalis* group, *Lutzomyia longipalpis* and ixodid ticks.

## 6. Transmission zones related to disease distribution

Climate and temperature zones play an important role in the establishment of disease transmission zones. From the discussions and evidence presented in the sections above, it is becoming clearer that the effects of climate change will lead to the establishment of disease transmission risk areas, which will expand or decrease as the environmental factors become favorable or unfavorable for disease vectors and pathogens.

The spatial distribution of lymphatic filariasis and onchocerciasis can be classified in terms of high and low transmission areas. de Souza and colleagues [104] demonstrated the presence of low, medium and high lymphatic filariasis transmission zones in Ghana, influenced by the presence/absence of efficient vectors and the effects of environmental factors, specifically temperature. Transmission could also be described in terms of hypo/meso/hyperendemicity for onchocerciasis, depending on the level of prevalence and the risks of morbidity [107]. The importance of these transmission zones lies in the disease control options and the possible re-infection of areas previously declared as non-endemic, from areas of high or medium transmission zones.

Perhaps the idea of transmission zones could best be described using the example of meningitis. Meningococcal meningitis (MCM) is an infection of the meninges, caused by the bacteria *Neisseria meningitidis*, that causes high death rates in African communities.

Although epidemics of MCM occur worldwide [108], the conditions associated with the onset of the epidemic in sub-Saharan Africa, and the occurrence in both space and time of MCM cases and climate variability within the Sahel-Sudan area, result in the establishment of a disease transmission zone or belt. The geographical distribution of disease cases is called the “Meningitis Belt” and is more or less circumscribed to the bio-geographical Sahel-Sudan band [109, 110]. The transmission period in West Africa is climate dependent [111] and usually start at the beginning of February -with the harmattan winds, and then disappear in late May -with the onset of rainfall. Thus, the environmental dependability of MCM epidemics enables the establishment of transmission risk models which can be useful for directing surveillance activities.

## 7. Climate change and migration

Historically, the migration patterns of the early modern hominids out of Africa (some 100,000 years ago) was believed to have coincided with climate related events and wet phases in the Sahara/Sahel region of North Africa [112-114]. This early migration may be linked with the possible co-migration of diseases to other parts of the world. Thus the early migration and disease distribution notion can be supported by studies that established that lice (which transmit diseases such as typhus, trench and relapsing fevers) had accompanied their human hosts in the original peopling of the Americas, near the end of the Pleistocene [115], probably as early as 10,000-15,000 years ago. Millions of people are forced to migrate when climate change brings about environmental disasters [116]. It is estimated that by 2050, between 25 million to 1 billion people will have been displaced as a result of climate change [117]. The impact of climate change on the environment comes about as a result of a) climate processes, characterized by the rise in sea levels, salinization of agricultural land, desertification, and drought, and b) climate events, such as flooding, storms, hurricanes, and typhoons [118]. Global climate change affects all parts of the world, however, the world’s poorest regions, particularly in sub-Saharan Africa, are the hardest hit, since they lack adequate social and economic structures to enable them to cope with climate change induced environmental disasters and its associated problems.

Drought threatens agricultural productivity, and thereby, food security and economic stability in these poor world regions, as their economies are heavily reliant on climate-dependent activities such as farming and fishing. Many people are thus forced to travel outside their local communities in search of better conditions. A common pattern of climate migration is for individuals or groups to travel where environmental conditions are favorable. In West Africa, for example, drought seasons compel young men and women to travel outside their communities to engage in paid labor so as to increase the family income, as well as send remittances to support the rest of the family. The distance travelled by these climate migrants is either internal or external, depending on the family’s resources, whereby they are enabled to only move to neighboring cities, or as far as Europe [118]. Typically, aside financial considerations, other factors such as existing social networks, past colonial relationships, and language determine how far migrants would travel. After the 2005

Hurricane Katrina disaster evacuees from the Gulf regions did not “stream across” to neighboring Mexico, but rather sought refuge elsewhere within the United States [118].

Many climate migration effects on disease distribution have been suggested. Over the short term, it has been suggested that climate change forced-migration will make the achievement of the Millennium Development Goals (MDGs) harder [118]. The provision of uninterrupted health services that underlie goals 4 and 5 of the MDGs (reducing child and maternal mortality and combating HIV/Aids, malaria and other diseases), is also likely to be affected as a result of large-scale climate migration [118]. The displacement of populations as a result of climate effects undermines the provision of medical care and vaccination programs; making infectious diseases harder to deal with and more deadly. It is well documented that refugee populations suffer worse health outcomes than settled populations [118]. Another effect of climate change migration is the spread of diseases as a result of overcrowding. One billion people, live in urban slums: in poor quality housing with limited clean water, sanitation and health services [118]. It is estimated that by 2030 this number will rise to 1.7 billion people [119]. This figure may even be higher, as it is estimated that 78 million people may be displaced by climate change by 2030 [120], and 250 million by 2050 [121]. The high population densities and high contact rates could help to spread disease, while health and education services are often inadequate. As an example, the spread of dengue fever in India has been associated with unplanned urbanization [122].

The most imminent impact of climate migration is the introduction of pathogens into new regions, thereby expanding the geographic range of infectious diseases. Northeastern Brazil, for instance, faces the challenge of outbreaks of visceral leishmaniasis as a consequence of migration during drought seasons [123]. Another example can be given of South Africa, where there are three newly described forms of *S. damnosum* present, with one identified from sites where man-biting is known [3]. This biting habit appears to be spreading in South Africa in the Johannesburg area and two separate sections of the Orange river [4]. Furthermore, it is known that there is extensive migration into South Africa (both legal and illegal) of people from countries where onchocerciasis is endemic. For instance, officially, 24,627 Nigerians entered South Africa in 2004, together with many from other onchocerciasis zones such as Cameroon and the Democratic Republic of the Congo (South Africa Statistics Statistical release P0351). Also, the spread of infectious diseases through migration becomes more serious and difficult to control as natives may have low level or non-immune resistance to newly introduced pathogens [124]. The converse may be true, where migrants into endemic regions may lack the resistance against pathogens. Rapid urban growth places heavy burdens on medical facilities, leads to poor sanitation, crowded housing situations, while the increase in human-human contact facilitates the spread of pathogens within urban communities. Nonetheless, climate migration is occurring at a time of unprecedented pressure on natural resources, especially water and arable land, as well as on social and health services [125].

The argument for disease distribution as a result of climate migration may not be attributed to humans alone, but also to animals and their roles in the transmission of zoonotic diseases.

In West Africa, the migration of the pastoral Fulanis has been a result many factors, including climate change [126, 127]. Cattle and livestock play an important role in the transmission of zoonoses including; anthrax, brucellosis, cryptosporidiosis, giardiasis and *E. coli*. Other animals such as wild birds also share with humans the capacity for moving over large distances, travelling across national and intercontinental boundaries. During migratory movements, birds have the potential of carrying and dispersing pathogens that can be dangerous for both humans and of course other animals and birds [128-130]. This spread of pathogens occurs at multiple geographic scales, consequently influencing disease dynamics [131-134]. Many disease pathogens can be spread in this manner and these include; viruses (e.g., West Nile, Sindbis, Newcastle), bacteria (e.g., borrelia, mycobacteria, salmonellae), and protozoa (e.g., cryptosporidia) [128]. However, the highly pathogenic avian influenza (H5N1) pandemic that started in China in 2005 [135] may be considered as the number one disease that opened up the world of migratory birds and the spread of diseases. In discussing the role of migratory birds in disease distribution, it is important to note that as the climate changes, so does the probability of zoonotic diseases increase. For example, studies have revealed that increasing temperatures alter bird migration patterns [136], and this may affect their duration of stay (influenced by food availability and suitable breeding conditions), thereby increasing the chances of transmitting disease pathogens.

## 8. Conclusion

In conclusion, most studies predict an increase in temperature - as ascribed to global warming, carbon dioxide concentration, rainfall, drought, and humidity. These factors influence the complex interactions within the public health triad comprising the environment, human host and disease vectors or pathogens. Although the effects may not be universal, the suitability of new environments will lead to the spread of disease vectors and pathogens into new areas, whereas the unsuitability (due to drought, for instance) may lead people to migrate into suitable areas, thereby introducing diseases in those areas.

## Author details

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## 9. References

- [1] Intergovernmental Panel on Climate Change (2007) Climate Change: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change (eds Solomon S. et al.) (Cambridge Univ. Press, 2007).

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- [2] Millennium Ecosystem Assessment (2005) Ecosystems and Human Well-being: General Synthesis. Washington, DC: Island Press and World Resources Institute.
- [3] Krüger A, Car M., Maegga BTA (2005) Descriptions of members of the *Simulium damnosum* complex (Diptera: Simuliidae) from southern Africa, Ethiopia and Tanzania. *Annals of Tropical Medicine and Parasitology*. 99: 293-306.
- [4] Palmer RW, De Moor FC (1999) *Simulium damnosum* s.l. complex widespread in Southern Africa. *British Simulium Group Bulletin* 14: 10-14.
- [5] Cliff A, Haggett P (1988) Atlas of disease distributions, analytical approaches to epidemiological data. Oxford: Blackwell.
- [6] Pascual M, Bouma M, Dobson AP (2002) Cholera and climate: revisiting the quantitative evidence. *Microbes Infect*. 4(2): 237-245.
- [7] Sattenspiel L (2000) Tropical environments, human activities, and the transmission of infectious diseases. *Am J Phys Anthropol. Suppl* 31:3-31.
- [8] Randolph SE, Rogers DJ (2000) Fragile transmission cycles of tick-borne encephalitis virus may be disrupted by predicted climate change. *Proc R Soc Lond B*. 267: 1741-1744.
- [9] Martens P (1998) Health and Climate Change: Modelling the Impacts of Global Warming and Ozone Depletion. London: Earthscan Publications Ltd.
- [10] Martens P, Kovats RS, Nijhof S, de Vries P, Livermore MTJ, Bradley DJ, Cox J, McMichael AJ (1999) Climate change and future populations at risk of malaria. *Global Environ Change*. 9 (Suppl 1): S89-S107.
- [11] Rogers DJ, Randolph SE (2000) The global spread of malaria in a future, warmer world. *Science*. 289: 1763-1766.
- [12] Wiesenfeld SL (1967) Sickle-cell trait in human biological and cultural evolution. *Science*. 157: 1134-40.
- [13] Coluzzi M (1999) The clay feet of the malaria giant and its African roots: hypotheses and inferences about origin, spread and control of *Plasmodium falciparum*. *Parassitologia*. 41: 277-83.
- [14] Joy DA, Feng XR, Mu JB, Furuya T, Chotivanich K, Krettli AU, Ho M, Wang A, White NJ, Suh E, Beerli P, Su XZ (2003) Early origin and recent expansion of *Plasmodium falciparum*. *Science*. 300: 318-21.
- [15] Hume JCC, Lyons EJ, Day KP (2003) Human migration, mosquitoes and the evolution of *Plasmodium falciparum*. *Trends Parasitol*. 19: 144-49.
- [16] Hart DL (2004) The origin of malaria: mixed messages from genetic diversity. *Nature Reviews Microbiol*. 2: 15-22.
- [17] De Zulueta J (1987) Changes in the geographical distribution of malaria throughout history. *Parassitologia*. 29: 193-205.
- [18] De Zulueta J (1994) Malaria and ecosystems: from prehistory to posteradication. *Parassitologia*. 36: 7-15.
- [19] Lysenko AJ, Semashko IN (1968) Geography of malaria. A medico-geographic profile of an ancient disease. In: Lebedew AW, editor. *Itogi Nauki: Medicinskaja Geografija*. Academy of Sciences, USSR; Moscow. pp. 25-146.
- [20] Russell PF (1956) World-wide malaria distribution, prevalence and control. *Am J Trop Med Hyg*. 5: 937-56.
- [21] Snow RW, Gilles HM (2002) The epidemiology of malaria. In: Warrell DA, Gilles HM, editors. *Essential malariology*. 4th Edn. Arnold; London. pp. 85-106.

- [22] Parham PE, Michael E (2010) Modeling the effects of weather and climate change on malaria transmission. *Environ. Health Perspect.* 118: 620–626.
- [23] Pascual M, Ahumada JA, Chaves LF, Rodo X, Bouma M (2006) Malaria resurgence in the East African highlands: temperature trends revisited. *Proc. Natl. Acad. Sci. U.S.A.* 103(15): 5829–5834.
- [24] van Lieshout M, Kovats RS, Livermore MTJ, Martens P (2004) Climate change and malaria: analysis of the SRES climate and socio-economic scenarios. *Global Environ. Change-Hum. Policy Dimensions* 14: 87–99.
- [25] Gubler DJ, Reiter P, Ebi KL, Yap W, Nasci R, Patz JA (2001) Climate variability and change in the United States: potential impacts on vector- and rodent-borne diseases. *Environmental Health Perspectives.* 109(suppl 2): 223–233.
- [26] Koenraadt CJM, Githeko AK, Takken W (2004) The effects of rainfall and evapotranspiration on the temporal dynamics of *Anopheles gambiae* s.s. and *Anopheles arabiensis* in a Kenyan village. *Acta Tropica.* 90(2): 141–153.
- [27] Olson SH, Gangnon R, Elguero E, Durieux L, Guégan JF, Foley JA, Patz JA (2009). Links between climate, malaria, and wetlands in the Amazon Basin. *Emerging Infectious Diseases.* 15(4): 659–662.
- [28] Noden BH, Kent MD, Beier JC (1995) The impact of variations in temperature on early *Plasmodium falciparum* development in *Anopheles stephensi*. *Parasitology*, 111(5): 539–545.
- [29] Paaijmans KP, Blanford S, Bell AS, Blanford JI, Read AF, Thomas MB (2010) Influence of climate on malaria transmission depends on daily temperature variation. *PNAS* 107(34): 15135–15139.
- [30] Hay SI, Guerra CA, Gething PW, Patil AP, Tatem AJ, Noor AM, Kabaria CW, Manh BH, Elyazar IR, Brooker S, Smith DL, Moyeed RA, Snow RW (2009) A world malaria map: *Plasmodium falciparum* endemicity in 2007. *PLoS Med.* 6(3): e1000048.
- [31] Gething PW, Smith DL, Patil AP, Tatem AJ, Snow RW, Hay SI (2010) Climate change and the global malaria recession. *Nature.* 465: 342–345.
- [32] Martens WJM, Niessen LW, Rotmans J, Jetten TH, McMichael AJ (1995) Potential impact of global climate change on malaria risk. *Environ. Health Perspect.* 103(5), 458–464.
- [33] Martin MH, Lefebvre GM (1995) Malaria and climate: sensitivity of malaria potential transmission to climate. *Ambio* 24: 200–207.
- [34] Lindsay SW, Martens WJM (1998) Malaria in the African highlands: past, present and future. *Bulletin of the World Health Organization.* 76(1): 33–45.
- [35] Small J, Goetz SJ, Hay SI (2003) Climatic suitability for malaria transmission in Africa, 1911–1995. *Proc. Natl Acad. Sci. USA* 100: 15341–15345.
- [36] Beguin A, Hales S, Rocklov J, Astrom C, Louis VR, Sauerborn R (2011) The opposing effects of climate change and socio-economic development on the global distribution of malaria. *Global Environmental Change.* 21: 1209–1214.
- [37] Fillinger U, Ndenga B, Githeko A, Lindsay SW (2009) Integrated malaria vector control with microbial larvicides and insecticide-treated nets in western Kenya: a controlled trial. *Bull. World Health Organ.* 87: 655–665.
- [38] Kleinschmidt I, Sharp B, Benavente LE, Schwabe C, Torrez M, Kuklinski J, Morris N, Raman J, Carter J (2006) Reduction in infection with *Plasmodium falciparum* one year after the introduction of malaria control interventions on Bioko Island, Equatorial Guinea. *Am. J. Trop. Med. Hyg.* 74(6): 972–978.

- [39] Bhattarai A, Ali AS, Kachur SP, Mårtensson A, Abbas AK, Khatib R, Al-Mafazy AW, Ramsan M, Rotllant G, Gerstenmaier JF, Molteni F, Abdulla S, Montgomery SM, Kaneko A, Björkman A (2007) Impact of artemisinin-based combination therapy and insecticide-treated nets on malaria burden in Zanzibar. *PLoS Med.* 4(11): e309.
- [40] Pruss A, Havelaar A (2001) The global burden of disease study and applications in water, sanitation and hygiene. In: Fewtrell L, Bartram J, editors. *Water Quality: Guidelines, Standards and Health*. London: IWA Publishing. pp. 43–59.
- [41] Hunter PR (1997) *Waterborne Disease: Epidemiology and Ecology*. Chichester: Wiley.
- [42] Smith HV, Patterson WJ, Hardie R, Greene LA, Benton C, Tulloch W, Gilmour RA, Girdwood RWA, Sharp JCM, Forbes GI (1989) An outbreak of cryptosporidiosis caused by post-treatment contamination. *Epidemiology and Infection.* 103: 703–715.
- [43] Joseph C, Hamilton G, O'Connor M, Nicholas S, Marshall R, Stanwell-Smith R, Sims R, Ndawula E, Casemore D, Gallagher P, Harnett P (1991) Cryptosporidiosis in the Isle of Thanet: an outbreak associated with local drinking water. *Epidemiology and Infection.* 107: 509–519.
- [44] Bridgman S, Robertson RMP, Syed Q, Speed N, Andrews N, Hunter PR (1995) Outbreak of Cryptosporidiosis associated with a disinfected groundwater supply. *Epidemiology and Infection.* 115: 555–566.
- [45] Willocks L, Crampin A, Milne L, Seng C, Susman M, Gair R, Moulds M, Shafi S, Wall R, Wiggins R, Lightfoot N (1998) A large outbreak of cryptosporidiosis associated with a public water supply from a deep chalk borehole. *Communicable Disease and Public Health.* 1: 239–243.
- [46] Anon (2000) Waterborne outbreak of gastroenteritis associated with a contaminated municipal water supply, Walkerton, Ontario, May– June 2000. *Canada Communicable Disease Report.* 26: 170–173.
- [47] Curriero FC, Patz JA, Rose JB, Lele S (2001) The association between extreme precipitation and waterborne disease outbreaks in the United States, 1948–1994. *American Journal of Public Health.* 91: 1194–1199.
- [48] Miettinen IT, Zacheus O, von Bonsdorff CH, Vartiainen T (2001) Waterborne epidemics in Finland in 1998–1999. *Water Science and Technology.* 43(12): 67–71.
- [49] Atherbolt TB, LeChevallier MW, Norton WD, Rosen JS (1998) Effect of rainfall on *Giardia* and *Cryptosporidium*. *Journal of the American Water Works Association.* 90: 66–80.
- [50] Miossec L, Le Guyader F, Haugarreau L, Pommepuy M (2000) Magnitude of rainfall on viral contamination of the marine environment during gastroenteritis epidemics in human coastal population. *Revue d'Epidemiologie et de Sante Publique.* 48(suppl 2): 2S62–2S71.
- [51] LeChevallier MW, Schulz W, Lee RG (1991) Bacterial nutrients in drinking water. *Applied and Environmental Microbiology.* 57: 857–862.
- [52] Barcellos C, Sabroza PC (2001). The place behind the case: leptospirosis risks and associated environmental conditions in a flood-related outbreak in Rio de Janeiro. *Cadernos de Saude Publica.* 17(suppl): 59–67.
- [53] Easton A (1999) Leptospirosis in Philippine floods. *British Medical Journal.* 319: 212.
- [54] Homeida M, Ismail AA, El Tom I, Mahmoud B, Ali HM (1988) Resistant malaria and the Sudan floods. *Lancet.* 2(8616): 912.

- [55] Novelli V, El Tohami TA, Osundwa VM, Ashong F (1988) Floods and resistant malaria. *Lancet* 2(8624): 1367.
- [56] Shears P (1988) The Khartoum floods and diarrhoeal diseases. *Lancet*. 2: 517.
- [57] McCarthy MC, He J, Hyams KC, el-Tigani A, Khalid IO, Carl M (1994) Acute hepatitis E infection during the 1988 floods in Khartoum, Sudan. *Transactions of the Royal Society of Tropical Medicine & Hygiene*. 88: 177.
- [58] Hunter PR (2003) Climate change and waterborne and vectorborne disease. *J. Appl. Microbiol.* 94(S), 37S–46S.
- [59] Bouma MJ, Dye C, Van der Kaay HJ (1996) Falciparum malaria and climate change in the North West Frontier Province of Pakistan. *Am J Trop Med Hyg.* 55: 131–137.
- [60] Bouma MJ, Proveda G, Rojas W, Chavasse D, Quinones M, Cox J, Patz J (1997) Predicting high-risk years for malaria in Columbia using parameters of El-Nino southern oscillation. *Trop Med Int Health.* 2: 1122–1127.
- [61] Hunter PR (1998) Cyanobacterial toxins and human health. *Journal of Applied Bacteriology*. 84(suppl): 35S–40S.
- [62] Morris Jr JG (1999) Pfiesteria, “the cell from hell”, and other toxic nightmares. *Clinical Infectious Diseases*. 28: 1191–1196.
- [63] Hungerford JM (2001) Seafood toxins. In: Labbe RG, Garcia S, editors. *Guide to Foodborne Pathogens*. New York: John Wiley and Sons. pp. 267–283.
- [64] Colwell R (1996) Global climate and infectious disease: the cholera paradigm. *Science* 274: 2025–2031.
- [65] Lobitz B, Beck L, Huq A, Wood B, Fuchs G, Faruque ASG, Colwell R (2000) Climate and infectious disease: use of remote sensing for detection of *Vibrio cholerae* by indirect measurement. *Proceedings of the National Academy of Science*. 97: 1438–1443.
- [66] Islam MS, Draser BS, Bradley DJ (1990) Long-term persistence of toxigenic *Vibrio cholerae* O1 in the mucilaginous sheath of a blue-green alga, *Anabaena variabilis*. *Journal of Tropical Medicine and Hygiene*. 93: 133–139.
- [67] Pascual M, Rodo X, Ellner SP, Colwell R, Bouma MJ (2000) Cholera dynamics and El-Nino-Southern Oscillation. *Science*. 289: 1766–1769.
- [68] Speelman EC, Checkley W, Gilman RH, Patz J, Caleron M, Manga S (2000) Cholera incidence and El-Nino-related higher ambient temperature. *Journal of the American Medical Association*. 283: 3072–3074.
- [69] Cook GC (1996) *Manson’s Tropical Diseases*, 20th edn. London: WB Saunders
- [70] Koelle K, Pascual M, Yunus M (2005) Pathogen adaptation to seasonal forcing and climate change. *Proc. R. Soc. B.* 2: 971–977.
- [71] Fine PE, Clarkson JA (1982) Measles in England and Wales—I: an analysis of factors underlying seasonal patterns. *Int. J. Epidemiol.* 11: 5–14.
- [72] Spira WM (1981) Environmental factors in diarrhea transmission: the ecology of *Vibrio cholerae* O1 and cholera. In: Holme T, Holmgren J, Merson MH, Mollby R, editors. *Acute enteric infections in children: new prospects for treatment and prevention* Amsterdam: Elsevier.
- [73] Cairns MA, Ebersole JL, Baker JP, Wigington PJ, Lavigne HR, Davis SM (2005) Influence of summer stream temperatures on black spot infestation of juvenile coho salmon in the Oregon Coast Range. *Transactions of the American Fisheries Society*. 134: 1471–1479.



- [74] Raffel TR, Rohr JR, Kiesecker JM, Hudson PJ (2006) Negative effects of changing temperature on amphibian immunity under field conditions. *Functional Ecology*. 20: 819–828.
- [75] Kutz SJ, Hoberg EP, Polley L, Jenkins EJ (2005) Global warming is changing the dynamics of Arctic host–parasite systems. *Proceedings of the Royal Society B-Biological Sciences*. 272: 2571–2576.
- [76] Bradbury J (2003) Beyond the fire-hazard mentality of medicine: the ecology of infectious diseases. *PLoS Biol*. 1: 148–151.
- [77] Glass RI, Becker S, Huq SI, Stoll BJ, Khan MU, Merson MH, Lee JV, Black RE (1982) Endemic cholera in rural Bangladesh, 1966–1980. *Am. J. Epidemiol*. 116: 959–970.
- [78] Reidl J, Klose KE (2002) *Vibrio cholerae* and cholera: out of the water and into the host. *FEMS Microbiol. Rev*. 26: 125–139.
- [79] Watnick PI, Lauriano CM, Klose KE, Croal L, Kolter R (2001) The absence of a flagellum leads to altered colony morphology, biofilm development, and virulence in *Vibrio cholerae* 0139. *Mol. Microbiol*. 39: 223–235.
- [80] Miller CJ, Drasar BS, Feachem RG (1984) Responses of toxigenic *Vibrio cholerae* 01 to physicochemical stresses in aquatic environments. *J. Hyg. Camb*. 93: 475–495.
- [81] Portner HO (2002) Climate variations and the physiological basis of temperature dependent biogeography: systemic to molecular hierarchy of thermal tolerance in animals. *Comp Biochem Physiol A* 132: 739–761.
- [82] Zimmerman PA, Dadzie KY, De Sole G, Remme E, Alley ES, Unnasch TR (1992). *Onchocerca volvulus* DNA Probe Classification Correlates with Epidemiologic Patterns of Blindness. *J Infect Dis* 165: 964–968.
- [83] Kumar NP, Patra KP, Hoti SL, Das PK (2002). Genetic variability of the human filarial parasite, *Wuchereria bancrofti* in South India. *Acta Tropica* 82: 67–76.
- [84] Thangadurai R, Hoti SL, Kumar NP, Das PK (2006). Phylogeography of human lymphatic filarial parasite, *Wuchereria bancrofti* in India. *Acta Tropica* 98: 297–304.
- [85] Gilman SE, Urban MC, Tewksbury J, Gilchrist GW, Holt RD (2010) A framework for community interactions under climate change. *Trends in Ecology and Evolution*. 25: 325–331.
- [86] Stenseth NC, Mysterud A (2002) Climate, changing phenology, and other life history and traits: nonlinearity and match-mismatch to the environment. *Proceedings of the National Academy of Sciences of the United States of America*. 99: 13379–13381.
- [87] Paull SH, Johnson PTJ (2011). High temperature enhances host pathology in a snail–trematode system: possible consequences of climate change for the emergence of disease. *Freshwater Biology*. 56: 767–778.
- [88] Mills JN, Gage KL, Khan AS (2010) Potential influence of climate change on vector-borne and zoonotic diseases: a review and proposed research plan. *Environ Health Perspect*. 118:1507–1514.
- [89] Githeko AK, Lindsay SW, Confalonieri UE, Patz JA (2000) Climate change and vector-borne diseases: a regional analysis. *Bulletin of the World Health Organization*. 78:9
- [90] Anyamba A, Linthicum KJ, Tucker CJ (2001) Climate-disease connections: Rift Valley fever in Kenya. *Cad Saude Publica*. 17(suppl):133–140.



- [91] Linthicum KJ, Anyamba A, Tucker CJ, Kelley PW, Myers MF, Peters CJ (1999) Climate and satellite indicators to forecast Rift Valley fever epidemics in Kenya. *Science*. 285:397–400.
- [92] Julvez J, Mouchet J, Michault A, Fouta A, Hamidine M (1997) The progress of malaria in Sahelian eastern Niger. An ecological disaster zone. *Bull Soc Pathol Exot*. 90:101–104.
- [93] Mouchet J, Faye O, Juivez J, Manguin S (1996) Drought and malaria retreat in the Sahel, West Africa. *Lancet*. 348:1735–1736.
- [94] Patz JA, Riesen WK (2001) Immunology, climate change and vector-borne diseases. *TRENDS in Immun*. 22:4.
- [95] Rueda LM, Patel KJ, Axtell RC, Stinner RR (1990) Temperature-dependent development and survival rates of *Culex quinquefasciatus* and *Aedes aegypti* (Diptera: Culicidae). *Journal of Medical Entomology*. 27: 892-898.
- [96] Gillies MT (1953) The duration of the gonotrophic cycle in *Anopheles gambiae* and *An. funestus* with a note on the efficiency of hand catching. *East African Medical Journal*. 30: 129-135.
- [97] Turell MJ (1989) Effects of environmental temperature on the vector competence of *Aedes fowleri* for Rift Valley fever virus. *Research in Virology*. 140: 147-154.
- [98] Yates TL, Mills JN, Parmenter CA, Ksiazek TG, Parmenter RR, Vande Castle JR, Calisher CH, Nichol ST, Abbott KD, Young JC, Morrison ML, Beaty B, Dunnum JL, Baker RJ, Salazar-Bravo J, Peters CJ (2002) The ecology and evolutionary history of an emergent disease: hantavirus pulmonary syndrome. *Bioscience* 52:989–998.
- [99] Faye O, Gaye O, Fontenille D, Hébrard G, Konate L, Sy N, Herve JP, Toure YT, Diallo S, Molez JF (1995) Malaria decrease and drought in the Niayes area of Senegal. *Sante' 5*: 299–305.
- [100] Fisman DN (2007) Seasonality of infectious diseases. *Annu. Rev. Public Health*. 28:127–43.
- [101] Cannell JJ, Vieth R, Umhau JC, Holick MF, Grant WB, Madronich S, Garland CF, Giovannucci E (2006) Epidemic influenza and vitamin D. *Epidemiol. Infect*. 134:1129–40.
- [102] Matthews SD, Meehan LJ, Onyabe DY, Vineis J, Nock I, Ndams I, Conn JE (2007) Evidence for late Pleistocene population expansion of the malarial mosquitoes, *Anopheles arabiensis* and *Anopheles gambiae* in Nigeria. *Medical and Veterinary. Entomology*. 21: 358-469.
- [103] Yawson AE, Weetman D, Wilson MD, Donnelly J (2007) Ecological zones rather than molecular forms predict genetic differentiation in the malaria vector *Anopheles gambiae* s.s. in Ghana. *Genetics*. 175: 751-761.
- [104] de Souza D, Kelly-Hope L, Lawson B, Wilson M, Boakye D (2010) Environmental factors associated with the distribution of *Anopheles gambiae* s.s in Ghana; an important vector of lymphatic filariasis and malaria in West Africa. *PLoS ONE*. 5(3):e9927.
- [105] Service MW (1982) Importance of vector ecology in vector disease control in Africa. *Bull. Soc. Vector Ecol*. 7: 1-13.
- [106] Freeman P, de Meillon B (1953) Simuliidae of the Ethiopian Region. *British Museum of Natural History, London*, 224 pp.
- [107] Katabarwa MN, Eyamba A, Chouaibou M, Enyong P, Kuete T, Yaya S, Yougouda A, Baldiagai J, Madi K, Andze GO, Richards F (2010) Does onchocerciasis transmission take place in hypoendemic areas? A study from the North Region of Cameroon. *Trop. Med. Int. Health*. 15: 645–652.

- [108] Molesworth AM, Cuevas LE, Connor SJ, Morse AP, Thomson MC (2003) Environmental Risk and Meningitis Epidemics in Africa. *Emerging Infectious Diseases*. 9(10): 1287-1293.
- [109] Lapeyssonnie L (1963) [The meningococcal meningitis in Africa] (in French). *Bull World Health Organ*. 28-3: 114 p.
- [110] Cheesbrough JS, Morse AP, Green SDR (1995) Meningococcal meningitis and carriage in western Zaire: A hypoendemic zone related to climate? *Epidemiol Infect*. 114: 75–92.
- [111] Sultan B, Labadi K, Guegan JF, Janicot S (2005) Climate drives the meningitis epidemics onset in West Africa. *PLoS Med* 2(1): e6.
- [112] Castaneda IS, Mulitza S, Schefuß E, dos Santos RAL, Damste' JSS, Schouten S (2009) Wet phases in the Sahara/Sahel region and human migration patterns in North Africa. Available: [www.pnas.org/cgi/doi/10.1073/pnas.09057711106](http://www.pnas.org/cgi/doi/10.1073/pnas.09057711106).
- [113] Carto SL, Weaver AJ, Hetherington R, Lam Y, Wiebe E (2009) Out of Africa and into an ice age: On the role of global climate change in the late Pleistocene migration of early modern humans out of Africa. *J Hum Evol* 56:139–161.
- [114] Osborne AH, Vance D, Rohling EJ, Barton N, Rogerson M, Fello N (2008) A humid corridor across the Sahara for the migration of early modern humans out of Africa 120,000 years ago. *Proc Natl Acad Sci USA* 105:16444 –16447.
- [115] Raoult D, Reed DL, Dittmar K, Kirchman JJ, Rolain JM, Guillen S, Light JE (2008) Molecular Identification of Lice from Pre-Columbian Mummies. *Journal of Infectious Diseases* 197 (4): 535-543
- [116] Lonergan S (1998) The role of environmental degradation in population displacement. *Environmental Change and Security Project Report*. 4: 6.
- [117] Stern N (2006) The economics of climate change: the Stern review. *Cambridge University Press*, Cambridge, 56.
- [118] Brown O (2008) Migration and climate change. *International Organization for Migration*. Available: [http://www.iisd.org/pdf/2008/migration\\_climate.pdf](http://www.iisd.org/pdf/2008/migration_climate.pdf). Accessed 2012 Feb 29.
- [119] Sclar ED, Garau P, Carolini G (2005) The 21st century health challenge of slums and cities. *The Lancet* 365: 901-903.
- [120] Global Humanitarian Forum (2009): The Anatomy of a Silent Crisis. Geneva: Global Humanitarian Forum
- [121] Christian Aid (2007): Human tide: The real migration crisis. Christian Aid Report. London. <http://www.christianaid.org.uk/Images/human-tide.pdf>. Accessed: 6/11/2012).
- [122] Shah I, Deshpande GC, Tardeja PN (2004) Outbreak of dengue in Mumbai and predictive markers for dengue shock syndrome. *J. Trop. Pediatrics*, 50: 301-305.
- [123] Franke CR, Ziller M, Staubach C, Latif M (2002) Impact of the El Niño Oscillation on Visceral Leishmaniasis, Brazil. *Emerging Infectious Diseases*. 8(9): 914-7
- [124] Population Action International (2011) Why population matters to infectious diseases and HIV/AIDS. Available: [http://populationaction.org/wpcontent/uploads/2012/02/PAI-1293-DISEASE\\_compressed.pdf](http://populationaction.org/wpcontent/uploads/2012/02/PAI-1293-DISEASE_compressed.pdf). Accessed 2012 Feb 29.
- [125] International Centre for Migration, Health and Development (2010) Climate change, migration, and health. A changing universe. Available: <http://icmhd.wordpress.com/2010/08/19/climate-change-migration-and-health/>. Accessed 2012 Feb 29.

- [126] Adebayo AG (1991) Of Man and Cattle: A Reconsideration of the Traditions of Origin of Pastoral Fulani of Nigeria. *History in Africa*, Vol. 18, pp. 1-21
- [127] Morrissey J (2009) *Environmental Change and Forced Migration: A State of the Art Review*, Refugee Studies Centre, Oxford Department of International Development, Queen Elizabeth House, University of Oxford, Oxford.
- [128] Jourdain E., Gauthier-Clerc M., Bicout D., Sabatier P (2007) Bird Migration Routes and Risk for Pathogen Dispersion into Western Mediterranean Wetlands. *Emerging Infectious Diseases* 13(3): 365-372.
- [129] Reed KD, Meece JK, Henkel JS, Shukla SK (2003) Birds, migration and emerging zoonoses: West Nile virus, Lyme disease, influenza A and enteropathogens. *Clin Med Res.* 1:5–12.
- [130] Hubalek Z (2004) An annotated checklist of pathogenic microorganisms associated with migratory birds. *J Wildl Dis.* 40:639–59.
- [131] Kilpatrick AM, Chmura AA, Gibbons DW, Fleischer RC, Marra PP, Daszak P. (2006) Predicting the global spread of H5N1 avian influenza. *Proc Natl Acad Sci U S A* 103: 19368–19373.
- [132] Marra PP, Griffing S, Caffrey C, Kilpatrick AM, McLean R, Band C, Saito E, Dupuis AP, Kramer L, Novak R (2004) West Nile virus and wildlife. *Bioscience* 54: 393–402.
- [133] Altizer S, Bartel R, Han BA (2011) Animal migration and infectious disease risk. *Science* 331: 296–302.
- [134] Gunnarsson G, Latorre-Margalef N, Hobson KA, Van Wilgenburg SL, Elmberg J, Olsen B, Fouchier RA, Waldenström J (2012) Disease Dynamics and Bird Migration—Linking Mallards *Anas platyrhynchos* and Subtype Diversity of the Influenza A Virus in Time and Space. *PLoS ONE* 7(4): e35679.
- [135] Normile D (2006). Avian influenza. Evidence points to migratory birds in H5N1 spread. *Science* 311:1225.
- [136] Hurlbert AH, Liang Z (2012) Spatiotemporal Variation in Avian Migration Phenology: Citizen Science Reveals Effects of Climate Change. *PLoS ONE* 7 (2): e31662