

Climatic Factors Under the Tropics

Jacques Reis¹, Pascal Handschumacher², Valerie S. Palmer³,
and Peter S. Spencer³

¹University of Strasbourg, Strasbourg, France ²Aix Marseille University, Marseille, France

³Oregon Health & Science University, Portland, OR, United States

OUTLINE

3.1 Introduction	26	<i>3.5.5 The Tropical Origin of Infectious Diseases</i>	31
3.2 A Physical Geographic Approach: The Climate and Its Characteristics	26	<i>3.5.6 Fungal Proliferation Under the Tropics</i>	31
3.2.1 The Role of the Inter-Tropical Convergence Zone	26	<i>3.5.7 Algal Blooms and Seafood Poisoning</i>	32
3.2.2 Types of Tropical Climate	26	3.6 Weather Events and Extreme Effects of Climate	32
3.3 A Biogeographical Approach: How Climate's Features Determine Natural Biomes and Ecosystems	26	3.7 A Health Geographical Approach: Case Studies of Climatic Features that Modulate Tropical Diseases	33
3.3.1 The Marine Biome	27	3.7.1 Mainly Climatic Determinants	33
3.3.2 Microbial Diversity and Biogeography	27	3.7.2 Complex Relations Between Climate, Soil, and Human Activities	33
3.4 Biology: Adaptation and Acclimation to Tropical Climates	28	3.8 The "Boomerang Effect" Under the Tropics: Health Consequences of Anthropogenic Changes	35
3.4.1 Thermal Tolerance	28	3.8.1 The Bangladesh Story: Persistent Dryness, Safe Drinking Water Availability and Deep-Water Wells	35
3.4.2 Water Availability	28	3.8.2 Agriculture Under the Tropics	35
3.4.3 UVR and Airborne Propagation of Neurotropic Viruses	28	3.8.3 Perturbation of the Global Nutrient Cycles	35
3.4.4 Thermoregulation in Man	29	3.8.4 Climate in Tropical Cities	35
3.4.5 Nutrition	29	3.9 Conclusions	36
3.5 Does the Tropical Climate Favor Pathogens and Infections in Humans?	30	References	36
3.5.1 Biogeography	30	Further Reading	39
3.5.2 Role of Environmental Conditions	30		
3.5.3 Infections of Humans	30		
3.5.4 Seasonality Under the Tropics	31		

3.1 INTRODUCTION

This chapter examines the impact of climatic factors on health and disease in tropical zones. The tropics are the regions surrounding the Equator, delimited in latitude by the Tropic of Cancer in the Northern Hemisphere and the Tropic of Capricorn in the Southern Hemisphere. We examine the physical determinants that define the tropics and their direct and indirect roles in the maintenance of health and induction of disease. We discuss the influence of geographical features on ecosystems and living organisms, interactions that can promote the onset of disease (with cases studies) in different ecosystems, and anthropogenic changes and their impact on the ecosystem and, consequently, on human health. These physical constraints of the climate interfere largely with sociocultural and economical issues (see dedicated chapters), creating specific epidemiological conditions under the tropics.

3.2 A PHYSICAL GEOGRAPHIC APPROACH: THE CLIMATE AND ITS CHARACTERISTICS

The climate system is driven by one energy source: the sun. Three-quarters of this energy is absorbed (by the atmosphere and the Earth's surface), the remaining quarter is reflected back into space.

The solar radiations over planet Earth have a heterogeneous distribution, dependent notably on Earth rotation, latitude, altitude, and surface (land vs water). The non-uniformity of the Earth also contributes to heterogeneous energy distribution, for example, the irregular distribution of continents, topography with mountain barriers, and large water surfaces (oceans, seas). These physical features cause heating differences that generate circulations of air and water masses, which allow heat exchanges between the Earth's zones. In general, tropical zones (air, oceans, land) receive more solar radiation than the poles. The main characteristic of a tropical climate is that the daytime temperature is constantly over 18°C.¹

3.2.1 The Role of the Inter-Tropical Convergence Zone

One of the main determinants of tropical weather and climates is the Inter-Tropical Convergence Zone (ITCZ), "the location where northeast winds in the Northern Hemisphere converge with the southeast winds from the Southern Hemisphere". ITCZ is a

low-pressure zone characterized by a line of cumulus clouds in the tropics.^{2,3}

At the Equator, the ITCZ causes significant rainfall throughout the year. Sometimes, there are two rainy seasons, a long and a short one. Some areas, such as parts of the Amazon Basin in South America, receive 3 or more meters (9 ft) of rain per year. Temperatures range from 25 to 35°C, with the hottest months only 2–3°C higher than those of cooler months. Constant temperature and high humidity are linked with luxuriant vegetation and a great biodiversity of flora, fauna and microbial organisms.

At locations progressively distant from the Equator, seasons become progressively more distinct (i.e., rainy season and dry season); these are linked with seasonal variations in the position of the ITCZ. For example, in Sahelian regions, there is a cold dry season (October–November to March), followed by a hot dry season (April to June) followed by a short rainy season with low rainfall (200–400 mm/year). The dry season is characterized by a continental trade wind blowing from the Northeast (Harmattan), which often brings much dust.

3.2.2 Types of Tropical Climate

Between the Equator and the Tropics, dependent mostly on temperature variability, humidity and precipitation, as well as winds, there are gradients leading from tropical moist to tropical dry. Three main types are described: the Tropical Rainforest or Equatorial, the Tropical Monsoon, and the Tropical Wet and Dry or Savannah.³

At the limit of the tropical area, the hot and arid climatic type is characterized by a huge temperature variation (from a daily temperature of 45°C to nocturnal frost), a scarcity of precipitation (mostly absent) and desertic vegetation. The Sahara Desert in northern Africa receives only 2–10 cm of rain per year, and the Chilean Atacama Desert is famous for its usual dryness. As the vegetal soil's cover is reduced, the permanent wind can erode the earth's surface and increase airborne particulate matter (PM), originating sometimes in major dust storms.

3.3 A BIOGEOGRAPHICAL APPROACH: HOW CLIMATE'S FEATURES DETERMINE NATURAL BIOMES AND ECOSYSTEMS

Functional biogeography brings the link between "the distribution of species and ecosystems across space and time and of the underlying biotic and abiotic factors, mechanisms, and processes".⁴

Biomes are characterized by distinctive plant and animal species that are maintained under regional climatic conditions. Several continental biomes exist in the tropics, including rainforests (Amazonia, Congo), dry deciduous forests (Madagascar, Deccan, Thailand), spiny forests (Madagascar), grasslands and deserts.⁵

Tropical rainforests, thought to be the oldest biome on Earth, comprise only 40% of the world's tropical forests and only 20% of the world's total forests. Flora are organized into four strata depending on the sunlight's access: giant trees constitute the emergent layer. The canopy layer above, formed by the trees' crown and often covered with other plants and tied together with woody vines (lianas), is home to 90% of the organisms found in the rainforest. The understory (under-canopy and shrub layer) allows the growth of young trees and plants that tolerate low light. The forest floor receives less than 2% of the sunlight. In most tropical rainforests, the soils are relatively poor in nutrients, apart from recent volcanic soils.^{6,7} The plant-available soil phosphorus and the dry-season intensity are, in Panama, the strongest predictors for the distribution of more than half of tree species.⁸

Many forested areas in the tropics are not rainforests. Forests that receive irregular rainfall (monsoons followed by a dry season) are moist deciduous forests. Trees in these forests may drop their leaves in the dry season.⁷

Arid zones are characterized by excessive heat and inadequate, variable precipitation and scarce vegetation. In the deserts, vegetation is virtually absent, whereas semi-desertic vegetation includes a mixture of grasses, herbs, and small, short trees and shrubs.⁹ The majority of grasslands (among the 26% of total land area) are located in tropical low-income countries, such as the savannahs of Sahelian countries and shrublands and llanos in South America. Grasses, often 3–6 ft tall at maturity, are the dominant vegetation, with shrubs or bush associated with scarce drought- and fire-resistant trees. Grasses are particularly important for the resident populations since they provide the feed base for grazing livestock.¹⁰ Desertification and vulnerability of water resources, including freshwater,^{11,12} are great concerns in arid zones.

3.3.1 The Marine Biome

Tropical marine biomes also require consideration.¹³ Marine regions cover about three-quarters of the Earth's surface and represent the largest of all ecosystems: they include oceans, coral reefs, and estuaries.¹⁴ The seas have an enormous capacity to store heat.¹⁵ Over 1.3 billion people live on tropical coasts, primarily in low- and middle-income countries.¹⁶ Marine

food chains that support seafood species have been used by human societies for food since earliest times, e.g., in the Red Sea.¹⁷

Tropical marginal seas (TMSs) are natural subregions of tropical oceans containing notably three key ecosystems: coral reefs and emergent atolls, deep benthic systems, and pelagic biomes.¹⁸ In the oceans, two other zones are described: the intertidal and abyssal zones. Coral reefs are widely distributed in warm shallow waters, in the Atlantic Ocean (Wider Caribbean) and the Indo-Pacific (from East Africa and the Red Sea to the Central Pacific Ocean).¹⁵ These tropical reefs shelter one-quarter to one-third of all marine species. This diversity is mainly concentrated in the central Indo-Pacific (the "Coral Triangle") and decreases with increasing distance from the Indo-Australian archipelago.¹⁹

Tropical coastal zones constitute an important ecosystem for humans since they serve as "food factories". The highest primary productivity and the richest fisheries are found within the Exclusive Economic Zones (EEZs), narrow strips of 200 nmi/370 km from coastlines.¹⁷ As shown for the South American coastlines, the influence of the ocean currents, with the phenomenon of upwelling (Humboldt current) and the high environmental variability caused by the El Niño Southern Oscillation (ENSO) and La Niña Southern Oscillation (LNSO), have important roles in the biodiversity of the oceans and coastal lines.²⁰

Some tropical biomes support the highest biodiversity in the world (e.g., tropical forests and coral reefs); others have the lowest rates of biodiversity, like the arid zones.²¹ "Tropical ecosystems support a diversity of species and ecological processes that are unparalleled anywhere else on Earth"²² The relation of the species' richness and a latitudinal gradient in geographical range is called the Rapoport's rule after Eduardo H. Rapoport.²³ However, other factors are also involved, notably geographical (longitude, elevation and depth) and environmental conditions (topography and aridity). One of the mechanisms of generation of this biodiversity is clearly the climate, particularly in the tropics.²⁴

3.3.2 Microbial Diversity and Biogeography

The essential role of microbes in ecosystem homeostasis has been recognized recently with the emergence of a new field of Microbial Ecology.^{25,26} Besides their great abundance (Earth hosts more than an estimated 10^{30} microbial cells), microorganisms are also immensely diverse and constitute about 60% of the Earth's biomass. Bacteria and Archaea have a central role in ecosystem processes. In the soil, they participate

in water purification, soil fertility, decomposition and catalysis, and are involved in the carbon (C), nitrogen (N), sulfur (S), and phosphorus (P) cycles, providing nutrients (N and P) and storage of carbon (up to half of carbon in living organisms).²⁷ Their diversity changes with distance, season, climate, soil texture and other environmental parameters. In arid and semi-arid soils (Israel), the microbial biogeography is determined more by specific environmental factors (moisture, organic matter, and silt/clay content) than dispersal limitation (geographic distances and spatial distribution patterns). As everywhere in the Biosphere, these microorganisms interact constantly with humans, in pathogenic or beneficial ways.²⁸ We discuss microorganisms in desert dust later.

3.4 BIOLOGY: ADAPTATION AND ACCLIMATION TO TROPICAL CLIMATES

A fundamental issue in biology is the way that climate influences the physiology and evolution of organisms, either directly or indirectly. Water availability, temperature, but also radiation (especially ultraviolet radiation (UVR)), are typical direct tropical climatic constraints. Indirect influences on health include those related to nutrition, which is heavily dependent on the type of plant and animal species able to thrive in tropical climates. These, together with the anatomical and physiological adaptations of human to life in the tropics, such as skin pigmentation, are important drivers of ecosystem and human health.

3.4.1 Thermal Tolerance

Thermal tolerance affects plants, viruses,²⁹ insects^{30,31} and marine and terrestrial ectotherms (reptiles).^{32,33} Janzen, in the 1960s, noted that tropical species are exposed to low annual variations in ambient temperature; he concluded that selection had favored organisms with a narrow physiological tolerance to temperature. This leads to smaller areas of biological distribution and increases the turnover of species. Thus, any perturbation can endanger these tropical species because their physiological adaptation is limited.^{32,34}

Seasonality, which involves a greater climatic tolerance and a spatial heterogeneity, also plays a critical role in thermal adaptation.³⁵ Another consequence is the capacity of organisms (e.g., vectors and reservoirs) to spread to higher latitudes. Ability for thermal adaptation is key. To survive at higher latitudes, organisms must be able to withstand greater temporal variability of climate relative to that at lower latitudes.³¹

3.4.2 Water Availability

Rodents from arid and semi-arid habitats live under conditions where the spatial and temporal availability of free water is limited or scarce. For example, South American desert rodents possess structural as well as physiological systems for water conservation.³⁶

Tropical plants are stressed by a dynamic network of interacting stressors, such as availability of water, CO₂, light and nutrients, temperature and salinity. Several strategies have allowed them to adapt to a wide ecophysiological variety of habitats; e.g., some employ a method of carbon fixation adapted to arid conditions; other plants have developed effective physiological (nocturnal stomatal opening for CO₂ uptake and daytime closure of stomata) and biochemical plasticity (crassulacean acid metabolism plants fix CO₂ nocturnally in the dark period) allowing a high rate of responsiveness by readily reversible variations in performance.³⁷

3.4.3 UVR and Airborne Propagation of Neurotropic Viruses

Weather patterns also affect the distribution of viral organisms and the risk for certain human viral diseases. One example is Varicella Zoster Virus (VZV) because it is mainly transmitted via virus-contaminated air. The associated human disease (chickenpox) is a highly contagious acute disorder that may reappear as herpes zoster if the dormant virus in the nervous system is later reactivated. Whereas, in the tropics, primary acute VZV infection occurs in later childhood, in temperate zones most infection occurs before leaving school. Therefore, adults are more susceptible to infection in tropical countries (30%–50%) compared to those in temperate countries (5%–10%). Thus VZV infection is subject to climatic modulation. Rice³⁸ suggests that UVR is the climatic factor responsible for the geographical differences in transmission. Furthermore, he hypothesizes that UVR has been involved in the co-evolution of VZV as humans migrated out of Africa, with loss of the selective advantage of resistance to UVR in the tropics, where UVR is at its highest level. This hypothesis is debated. Different genotypes of VZV segregate geographically into tropical and temperate areas. The UVRs do not explain the distribution of VZV genotypes in different tropical and temperate regions of Mexico.³⁹ Nevertheless, the seasonality of varicella in Perth, Western Australia, which peaks during August–September (Australian Spring when both UVR and temperature are relatively low) and a significant association between VZV infection and UVR were confirmed, as well as the role of the temperature in the VZV transmission.⁴⁰

Human biology, notably skin pigmentation, also shows adaptation to climate and UV radiation. Many genes (MC1R, MATP (SLC45A2), SLC24A5, TYR, DCT, OCA2, KITLG, SLC24A4, and IRF4) are involved in melanogenesis, such that pigmentation-associated gene variants are specific to either Europeans or East Asians.⁴¹ Skin protects against the deleterious effects of UVR and allows photosynthesis of UVR B-dependent vitamin D3. There is a strong correlation between human skin pigmentation and latitude, i.e., UVR levels. Living under high UVR (near the Equator), our ancestors had a rich protective eumelanin skin. Positive selection during evolution led to elimination of the MC1R locus polymorphism and to a continuous purifying selection acting on the same locus. When *Homo sapiens* dispersed out of the tropics, the goals changed. To adapt to low and highly seasonal UVB conditions, the need to maximize cutaneous biosynthesis of pre-vitamin D3 prevailed and drove a depigmentation selection.^{42,43} The role of vitamin D3 has already been illustrated, notably in the pathogenesis of multiple sclerosis.⁴⁴ The deleterious impact (survival and/or reproductive fitness), on which the selection pressure was exercised, is still debated: effect on folate metabolism? Skin cancer?^{42,43,45}

The skin is the first stage of the immune, innate and humoral response. External perturbations, such as UVR, can modify this response and lead to an immune-suppressive effect. Cytokines made by UV-irradiated keratinocytes play an essential role. Keratinocyte-derived interleukin (IL)-10 is responsible for the systemic impairment of antigen-presenting cell function and the UV-induced suppression of delayed-type hypersensitivity.⁴⁶

3.4.4 Thermoregulation in Man

Although Man is a poikilotherm, thermoregulation is a challenge in the tropics. Skin keratinization aids in controlling the transepidermal modulation of water loss. Keratinization and epidermal differentiation genes are under accelerated evolution in the human lineage, driven by environmental selection pressure.⁴⁷

3.4.5 Nutrition

Plants adapted to climatic extremes, such as severe drought and water-logging, comprise an important source of food for many in tropical climes. The same plants may be eaten in temperate regions but consumption patterns differ. In tropical regions subject to severe drought, most food and feed plants become depleted, resulting in disease and death of cattle and other sources of animal protein. Food dependency on

one or more environmentally tolerant plants increases progressively with time, leading to malnutrition and disease. Since many plant species contain chemicals with toxic or neurotoxic potential, those in the tropics who must rely on individual plant species both for subsistence and emergency food, are susceptible to plant-specific neurological disease. Outbreaks of such diseases (e.g., lathyrism, cassavism) are almost always restricted to impoverished rural communities that depend on locally available sources for food and feed, are subject to weather extremes, and at risk for plant losses from these and other causes, such as viral and fungal infection. Toxic human neurological disease may affect such communities seasonally when crop yields are low, food stores have diminished, and protein malnutrition is evolving. By contrast, in temperate climates, where the same plants may be consumed throughout the year, populations remain relatively well nourished and are spared from neurotoxicity because they typically consume a mixed diet of animals and plants, such that the threshold for toxicity for any individual plant species is never exceeded.⁴⁸

The most important example is cassava (*Manihot esculenta*), the tuber and leaves of which are eaten for their carbohydrate and protein content, respectively. The plant is eaten without detectable illness by hundreds of millions of people in the tropics, subtropics and beyond. However, in certain impoverished communities of sub-Saharan Africa and India (inner Kerala) that must subsist on cassava as their sole or major dietary source, outbreaks of irreversible upper motor neuron disease in the form of spastic paraparesis are not infrequent. Local names for cassavism include *konzo* and *mantakassa*, from the Democratic Republic of Congo and Mozambique, respectively. While the culpable neurotoxin has yet to be pinpointed, cassava is protein-poor and contains cyanogenic glucosides that produce cyanide, thiocyanate and cyanate in the consumer.⁴⁹

Another example of great historical importance is grasspea (*Lathyrus sativus*), a remarkably environmentally tolerant and nutritious legume that, with prolonged dietary dependency, results in a neurotoxic upper motor neuron disease (lathyrism). While grasspea in small quantities is widely eaten as a tasty snack in affluent Bangladeshi households, those who are impoverished are heavily dependent on grasspea and develop varying degrees of spastic paraparesis. Today, lathyrism is largely confined to the Ethiopian highlands. The culpable neurotoxin is a plant-specific amino acid, beta-N-oxalylamino-L-alanine (L-BOAA), that acts as an excitant via a specific class of neuronal glutamate receptors. The same receptor has been implicated in cassavism, which might explain the comparable neurologic outcome.^{50,51}

A third example is the cycad (*Cycas* spp.), an exceptionally poisonous largely tropical plant that is resistant to extreme environmental conditions, including drought, fire and cyclones. Nevertheless, humans and animals have used cycads as a source of food and feed especially after tropical cyclones when less hardy plants are destroyed. Animals that eat untreated plant components (leaves, seed) develop a chronic and perhaps progressive neuromuscular disease. Humans that detoxify cycad seed (Australian aborigines) or sago (Ryukyu islanders, Japan) through extensive water leaching are spared illness, while others at high risk for amyotrophic lateral sclerosis and Parkinsonism—dementia complex have used raw cycad seed for medicine or incompletely detoxified seed for food.⁵²

Possible independent influences of dietary habits (nutrition) and chemical environment (related to climate and biomes) have been demonstrated. Lactase persistence in adulthood is a heritable condition providing a physiological advantage, the capacity to digest lactose contained in fresh milk. This enzymatic shift represents the best-known adaptation related to diet. A similar shift has been demonstrated for the enzyme, arylamine *N*-acetyltransferase 2 (NAT2) involved in acetylation (a well-known pharmacogenetic trait), although the environmental causative factor (if any) driving its evolution is as yet unknown. Investigations in the African population of the Sahelian belt showed a clear difference for the activity of NAT2 between nomadic pastoralists and hunter-gatherers (slow acetylators) versus agriculturalists or food-producing populations (sedentary farmers).⁵³

3.5 DOES THE TROPICAL CLIMATE FAVOR PATHOGENS AND INFECTIONS IN HUMANS?

Galiana⁵⁴ proposed the holistic Ecosystem Screening Approach to understand pathogen-associated microorganisms affecting host disease. Actually, pathogens challenge all kinds of living organisms: plants, animals, and humans. The questions to be addressed are numerous.

3.5.1 Biogeography

The distribution of human-associated pathogens seems to follow Rapoport's rule: a study showed a positive correlation between range, size and latitude for 290 human pathogenic species.⁵⁵ Latitudinal gradient, nested species pattern, and Rapoport's rule, are the factors that produce the observed geographical distribution of human pathogenic species.⁵⁵ However, there is no tropical effect for parasite species. Kamiya⁵⁶ has shown that the rich diversity of parasite species

across animal, plant, and fungal hosts does not correlate with, and even runs counter to, Rapoport's rule. Parasite diversity tends to be greater as one moves further from the Equator.⁵⁶

3.5.2 Role of Environmental Conditions

"The life cycles and transmission of many infectious agents—including those causing disease in humans, agricultural systems and free-living animals and plants—are inextricably tied to climate".⁵⁷ The life cycle of parasites is influenced by environmental factors. For example, the development, survival, distribution, and migratory behavior of free-living helminth larvae on pasture are primarily weather-related. Eggs hatch and develop more readily at higher temperatures, and optimal temperature allows for larval activity and thus motility. Moisture affects motility and must be present to prevent desiccation and death of developing larvae. Rainfall favors larval dispersal. As larvae migrate deep into the soil, the soil type has a major effect on their ability to migrate.⁵⁸ Endohelminths (mainly trematodes) additionally are influenced by temperature, water salinity, pH, oxygen content, water mineral content (hardness), light (linked with depth/water pressure), UVRs, and desiccation.⁵⁹

3.5.3 Infections of Humans

The impact of climatic conditions on human pathogens depends on several features: free persistence outside the host (parasites), transmission via a biological vector or a non-biological physical vehicle (water, soil, etc.) and, commonly, involvement of natural reservoirs (mice, rodents, small mammals, deer, birds, fish, zooplankton).^{57,60} Based on their natural host and transmissibility, epidemiologists classify infectious diseases into anthroponoses (i.e., human to animal) or zoonoses (animal to human). In some cases, a pathogen can spread outside its normal zoonotic cycle and affect humans, e.g., Rift Valley fever, when floods trigger *Culex* mosquitoes to feed on both infected ungulate hosts and then on humans.

Climatic factors act on different targets: the life cycle of the pathogen, its hosts and reservoirs. For example, the rates of replication, development, transmission, and mortality of a pathogen and many vectors depend on temperature and humidity. Both pathogens (protozoa, bacteria, viruses, etc.) and their associated vector organisms (snails, mosquitoes, ticks, sandflies, fleas and flies, etc.) live in a limited range of climatic conditions, the "climate envelope", which allows continuation of their natural life cycle. Besides temperature, climatic and geographic factors, important modulating

factors include: precipitation, sea level elevation, wind and duration of sunlight, estuary flow, and water salinity in marshes. Patz⁶⁰ reviewed extensively the impact of these different factors on the life cycles of pathogens. An increasing concern addresses the ecosystems where the pathogens (Ecosystem Screening Approach), vectors and reservoirs reside. Ecophysiological approaches must consider multiple host species and parasite developmental stages that have a different sensitivity to climatic factors.⁵⁷

3.5.4 Seasonality Under the Tropics

The seasonality of infections, first described by Hippocrates in *Air*, is well known.⁶⁰ However, the topic is still debated and mysterious, mainly because of its complexity (climatic factors but also runoff, increased snowmelt, floods).^{61–63} Complexity is related to the numerous interacting factors and their variability and strength in the environment in which they occur. Different clusters of seasonal determinants have been studied: human activities; host susceptibility (related to the endocrine-immune systems variability); vitamin D levels; melatonin; epithelia's mucosal integrity and pathogen survival and transmissibility (directly under the influence of climatic factors that define seasons, temperature, humidity, and precipitation) depending of course on their type of environment (e.g., sewage, aerosol, droplets, etc.), changes in vector abundance, and natural reservoirs.^{61,62,64} Seasonal patterns in parasitism have been shown: the long dry season may limit development and survival of parasite stages in the environment and, as a result, host contact and parasite transmission. Seasonal increased disease risk with increasing temperature and/or rainfall occurs for Japanese encephalitis, Chikungunya, West Nile viruses, Rift Valley fever, yellow fever and other mosquito-borne viral diseases. The ENSO is responsible for fluctuations of numerous arthropod-borne viral diseases, including Rift Valley fever, Ross River virus infection and dengue (Fisman, 2012). The Noumea study (New Caledonia) showed that “the epidemic dynamics of dengue were essentially driven by climate during the last forty years. Specific conditions based on maximal temperature and relative humidity thresholds were determinant in outbreaks occurrence”. As dengue fever affects at least 500,000 patients leading to 25,000 deaths yearly, the forecast operational model of outbreaks would be helpful in term of Public Health.⁶⁵

3.5.5 The Tropical Origin of Infectious Diseases

In the Tropics, seasonality is mainly responsible for population dynamics of vectors, hosts and sometimes, pathogenic agents. For example, malaria, even though it

reached its maximum extent in the world during the so-called “Little Ice Age”, is mainly transmitted in tropical areas and during the rainy season. These climatic conditions allow us to distinguish two major areas of transmission: stable and unstable. Stable malaria transmission areas are located in equatorial and rainy regions of the tropics while unstable transmission areas are located in dry parts of the tropics.⁶⁶ These degrees of stability are determined following the duration of the transmission season and have a crucial importance in degrees of disease severity. In the stable areas, immunological mechanisms will, after some time, lead to a protection due to permanent exposure whereas seasonal transmission will lead each year to loss of immunity between rainy seasons and most frequent severe forms of the disease.

On the contrary, very wet conditions and high clay soils can be unfavorable environmental conditions for the larva of the Tsetse fly because they permit development of their predators, such as termites (clay soils) or fungi (wet conditions).⁶⁷

Seasonality can also be the reflection of human activities associated with climate characteristics. In the Casamance region in southern Senegal, agriculture uses excessive phytosanitary products because of exposure to numerous pests. In 2005, the Bignona Department was confronted with an alarming digestive and neurological syndrome characterized by high lethality (58%). The syndrome affected three villages, especially in the rainy season. The study showed a link between the syndrome and the use of pesticides or the presence of stocks treated by these products in the houses.⁶⁸

Another example of the seasonal interplay between human activity and an environmental agent is the “acute encephalitis syndrome” that seasonally affects children in northern regions of Bangladesh, India, and Vietnam in communities where lychee fruit is cultivated. While this often-fatal disease has been attributed to an unknown virus, pesticide, fruit coloring, or heat stroke, the actual cause is most probably an amino acid in lychee fruit pulp that blocks gluconeogenesis in the consumer. Hungry, poorly nourished children who eat the more toxic unripe lychee fruit develop an acute illness (seizures, coma, death) resulting from severe hypoglycemia that is readily treatable with dextrose infusion. A similar acute hypoglycemic encephalopathy occurs in West African and Jamaican children who consume unripe ackee fruit (*Blighia sapida*), which contains hypoglycin A, the higher homolog of the hypoglycemic agent α -(methylenecyclopropyl) glycine in *Litchi sinensis*.^{69,70}

3.5.6 Fungal Proliferation Under the Tropics

Climatic conditions also affect the concentration of mycotoxins, including neurotoxic mycotoxins, in food.

Warmer weather, heat waves, increased precipitation and drought will have various region-dependent impacts on mycotoxin production. Overall mycotoxin production will vary with temperature, rainfall and crop production. In China, there is a differential seasonal incidence of moldy sugarcane poisoning, which results in an acute encephalopathy, with putaminal necrosis and persistent dystonia in survivors.⁷¹

Plants are susceptible to infection by fungi that produce secondary metabolites (mycotoxins) with neurotoxic potential in humans and the animals on which they depend for food and transport. Climatic conditions modulate fungal infection and growth both before and after harvest, and during storage and transport. Again, the same organism may flourish in both temperate and tropical climes, but exposure may be controlled by human intervention in the former but not the latter. For example, the ergot-generating fungus *Claviceps purpurea*, food contamination of which can cause extremity gangrene, dystonic and convulsive disorders, was once widespread in Europe and Russia (last major outbreak in 1938) but is now reported only in Ethiopia. Other mycotoxins with immunosuppressive potential, such as aflatoxin, are generally found in higher concentrations in food materials grown in tropical climes. Outbreaks of food-associated aflatoxicosis resulting in acute hepatic encephalopathy have occurred in Malaysia.⁷²

Whether and how exposure to these mycotoxins increases risk for other infectious diseases has not been addressed. A recent environmental, nutritional and case control study of Nodding Syndrome, a pediatric epileptic disorder associated with infection with the nematode *Onchocerca volvulus*, found an association with prior measles infection and food dependency on moldy maize prior to onset of head nodding. The authors suggested the nematode infection was secondary to measles immunosuppression, while Nodding Syndrome may be a post-measles disorder akin to subacute sclerosing panencephalitis in which central nervous system measles virus activation is triggered by heavy dietary exposure to immunosuppressive mycotoxins.⁷³

3.5.7 Algal Blooms and Seafood Poisoning

Harmful algal blooms (HABs) are increasing in frequency and intensity worldwide, perhaps in association with climate change. Toxic blue–green algae flourish in warm waters, increase their growth rate with higher levels of carbon dioxide, and thrive on nutrients in water run-off associated with heavy rainfalls.⁷⁴ Cyanobacteria, which are found in fresh, estuarine and marine waters, are composed of a variety of non-toxic

and toxic strains, the latter releasing a range of toxic and neurotoxic substances into water, including microcystins, cylindrospermopsins, anatoxins and saxitoxins, the latter being responsible for Paralytic Shellfish Poisoning. Other algal neurotoxins known to cause human neurological illness include domoic acid, maitotoxin and ciguatoxin. Ciguatoxin, which accumulates to toxic levels in large predatory reef fish, is one of the most common causes of human neurotoxic illness (ciguatera food poisoning) worldwide. Additionally, most genera of cyanobacteria elaborate beta-*N*-methylamino-L-alanine (L-BMAA), a low-potency neurotoxin of interest in relation to the presence of the amino acid in cycads linked to western Pacific amyotrophic lateral sclerosis and Parkinsonism–dementia complex.

3.6 WEATHER EVENTS AND EXTREME EFFECTS OF CLIMATE

Extreme weather events, including heat waves, droughts and floods, dust winds and tropical cyclones, have occurred since time immemorial, even if climate change is increasing their occurrence. These different events can be mild or catastrophic depending on the climatic zones and the biomes involved.

In the tropics, ocean-atmospheric phenomena (sea surface temperature anomalies associated with large-scale air circulation changes) are involved in many extreme weather events. The Pacific Ocean with its dominant mode of climate variability, the El Niño–Southern Oscillation (ENSO), increases the risk of droughts, floods and tropical cyclones and infection risk from certain vector- and rodent-borne diseases (e.g., malaria and dengue).⁷⁵ In Australia, the “Big Dry” (2003–2012) was related to the Indian Ocean Dipole (IOD), the mode of variability in the tropical Indian Ocean; it is also involved in rainfalls in countries surrounding the tropical Indian Ocean.⁷⁶ Disasters can affect the biomes and destroy ecosystems (e.g., the 1997–98 ENSO-related drought in Sabah, Borneo).⁷⁷ The material consequences from fires, dwelling collapse, landslide, and mud torrent are supplemented by human displacement, traumatic injury, drowning, food shortages and famine, poisoning of water supplies and food, chemical dissemination, waterborne diseases⁷⁸ and increased infection.⁷⁹ The impact of these events can be evaluated by different indices, such as the Climate Change Vulnerability Index (CCVI)⁸⁰ and the Germanwatch Global Climate Risk Index (CRI).⁸¹ These also take into account socioeconomic factors, such as poor development, poor sanitation, lack of preparedness, etc. The health issues are

in the neurological field and include: injuries and head trauma, anxiety, and emotional stress (especially in the elderly), and post-traumatic stress disorder.^{82,83}

Heat waves will become more frequent, more intense, and longer-lasting with the advance of global warming triggered by climate change. Exposure to extreme heat can have lethal consequences ranging from heat rash and heat cramps to heat exhaustion and heat stroke. Those at great risk are the elderly, infants and children, those with chronic illness, and city dwellers who reside in dwellings that lack air conditioning. Body temperature that rise to 105°F or more can result in delirium, convulsions, coma and death. Only scarce epidemiological data are available in tropical countries even if heatwaves occur more and more. During the European heat wave in 2003, an estimated 35,000 people died from stroke, heart attack, lung disease, and other causes exacerbated by heat.⁸⁴ In Pakistan in 2005, temperatures as high as 49°C (120°F) caused the deaths of about 2000 people from dehydration and heat stroke, mostly in Sindh province and its capital city, Karachi. The heat wave also claimed the lives of zoo animals and countless agricultural livestock. The event followed a separate heat wave in neighboring India that killed 2500 people in May 2015.⁸⁵

Desert winds aerosolize tons of soil-derived dust, notably in the tropical deserts of Africa and Asia. At a regional level, sand winds are responsible for the high concentrations of particulate matter (PM) in deserts. Desert dust is seasonally or constantly injected into the atmosphere and winds facilitate its transoceanic and transcontinental dispersal. The estimated annual quantity of desert dust subject to regional or global airborne migration is 0.5–5.0 billion tons. As dust and airborne PM serve as the vehicles for pathogens, they pose environmental challenges that extend well beyond tropical zones.^{86,87} Dust clouds allow pathogens to be disseminated beyond their usual geographical range and thereby pose a wider risk to human health.⁸⁸ Bacterial abundance is correlated with the dust events, with >10-fold higher concentrations on severe dust days. Airborne microorganisms impact indigenous microbial communities.⁸⁹

3.7 A HEALTH GEOGRAPHICAL APPROACH: CASE STUDIES OF CLIMATIC FEATURES THAT MODULATE TROPICAL DISEASES

3.7.1 Mainly Climatic Determinants

3.7.1.1 Epidemic Meningococcal Meningitis

Every year, West African countries within the Sahelo-Saharan band are afflicted with major meningococcal

meningitis (MCM) disease outbreaks, which affect up to 200,000 people, mainly young children, in one of the world's poorest regions. The timing of the epidemic year, the dry months of February to May and the spatial distribution of disease cases throughout the "Meningitis Belt", strongly indicate a close linkage between the life cycle of the causative MCM agent (the throat bacterium *Neisseria meningitidis*) and climate variability. While dry throats are speculated to increase risk for bacterial infection and transmission, the mechanisms responsible for the observed patterns are still not clearly identified.⁹⁰ The strong seasonality could be due to changes in temperature, humidity, and dust. The amount of dust is particularly high in this part of the world thanks to the Harmattan, a strong wind that comes in from the north-east. The Harmattan picks up dust as it blows over desert regions like the Bodélé Depression, a dried-up lakebed in central Chad that is the largest dust source on Earth. The resulting dust storms are so thick that they can block out sunlight for several days.⁹¹

In Mali, an approach based on the construction of an index reflecting the movement of the lower layers of the atmosphere, has illustrated the temporal correspondence between the evolution of the Harmattan and the occurrence of cases of MCM.⁹⁰

However, a study conducted at the local level in west central Senegal, which compared 6 years (3 epidemic and 3 non-epidemic years) showed that the occurrence of sandstorms, haze, and blowing sands appeared not to correlate with the emergence of epidemic episodes. Only a decrease of the relative humidity (<30%) during a continuous period over 10 days distinguished MCM-epidemic and non-epidemic years significantly.⁹² The humidity decrease seems to be necessary but certainly not sufficient to trigger epidemic MCM, as two non-epidemic years include a succession of several days with a relative humidity below 30%. The relatively short duration of the study, coupled with the failure to take account of the immunological status of the population, force us to remain cautious about the findings. However, it appears that the southward spread of desert should amplify the occurrence of phenomena associated with the Harmattan; a dynamics survey of MCM over long period is needed.

3.7.2 Complex Relations Between Climate, Soil, and Human Activities

3.7.2.1 Climate Impact on Tropical Endemic Diseases Vectors Ecology: Case Studies of Malaria and Human African Trypanosomiasis in Africa

Human African trypanosomiasis (HAT) or sleeping sickness is caused by a flagellated protozoan parasite

of the genus *Trypanosoma*: the species *T. gambiense* occurs in West Africa and *T. rhodesiense* in East Africa. Transmission to humans (and animals) is due to a vector, a fly of the genus *Glossina*. The Sudan-Guinea equatorial climate zone favors the ecosystem where this vector lives and acts. Endemic HAT has undergone a remarkable evolution during the 20th century: its historical spots were drastically reduced due to a combination of colonial health policies, population growth and pressure on forest areas and urbanization effects. However, sleeping sickness remains at a low level, especially in the Guinea mangrove coast and western Ivory Coast. Moreover, HAT has taken advantage of the multiple conflicts and political instability that occur in the continent to re-emerge, for example in the Democratic Republic of Congo, Angola, Uganda, Central African Republic, Cameroon, Sudan, or Ivory Coast.^{93,94}

The climate change appears here more or less restrictive in contrast to anthropogenic space transformation and territories health management, which appear to be crucial determinants. This can also be observed with malaria, whose present distribution area (characterized by stability or endemic and instability or epidemic zones) might spread northward, following the hypothesis of nowadays.

Malaria and its often deadly meningeal syndromes are mainly due to *Plasmodium falciparum*, which is mostly prevalent in Africa (between the Sahelian and the equatorial zones) but also affects Southeast Asia and Latin America. However, historically, this disease was not only rampant in the Mediterranean but also spread over North Europe and North America.⁹⁵ The maximum extension of this disease northwards happened at the end of the Little Ice Age for example, indigenous cases were diagnosed in Canada or Scandinavia during the 19th century! The decline and disappearance of malaria arose from environmental sanitation and the widespread use of quinine curative treatment. Improvement in health systems delivered the coup de grâce, thereby allowing eradication in malaria areas of Europe and North America. The present global warming could lead to an extension of the malaria-endemic area to the countries of the North only if health systems collapse. Of course, individual sporadic cases might still occur and be detected; thus, the major risk determinant in the countries of the North is not the climate but rather political, economic, and social pressures. The main malarial risk linked with climate change concerns the endemic areas of malaria in the countries of the South by extending transmission seasons and modifying altitudinal limits of disease extension.⁹⁶

3.7.2.2 The Amazon Basin, Soil Chemical Composition, Soil–Air Interface, Climate Dependency, and Environmental (Surface Water) Contamination by Mercury

Human poisoning by methylmercury (MetHg), the highly bioavailable form of mercury that readily crosses the blood–brain and placental barriers, is characterized by neurological signs in adults and neurodevelopmental effects in children. Thus, the populations at risk are mainly young children and pregnant women.⁹⁷

Although gold mining and panning are often involved in the mercury pollution of rivers (because of the use of this metal to amalgamate gold), it is not the only cause of pollution. A significant part comes from natural causes (earth's crust degassing, erosion and landslide) or other human activities (stubble-burning, farming on steep slopes) contributing to pollute the environment. Natural mercury sources provide for 60% of the mercury carried by the Amazon basin's rivers in Bolivia.⁹⁸ These processes are likely to lead to an environmental contamination only in mercury-containing soil areas. However, it is this pollution process that is the most efficient in the rainy tropics and equatorial areas where chemical processes producing oxysoils, characterized by high mercury content^{99–101} can occur. But not all exposed populations in affected areas are contaminated in the same way. Studies have shown there is a differential exposure in human communities linked with the availability of alternative resources. Thus, access to commercial activities allowed the diversification of their food supply and their food consumption modification. Two activities have been identified: selling of their own agricultural products in surrounding town markets and illegal extraction and trade of wood in remote areas far from regulatory centers. However, the main food of recently settled Indian populations remains potentially mercury-contaminated fish. Thus, on a small scale, a theoretical equal exposure to mercury leads to variations based on human activities, social behavior, commercial links, and ethnicity.¹⁰²

In French Guiana, where exposure to mercury is mainly due to gold extraction, a study showed concordant results with the importance of diet and food consumption. The factors contributing the most to explaining the high level of contamination of a population of 500 individuals consulting in 13 health centers were linked to consumption of fresh fish and livers from game, more than residence in the proximity of a gold-mining community.¹⁰³ But each rainy season may increase pollution of the lowerlands and the rivers because of the process of deforestation followed by land abandonment contributing to increasing human population exposure.¹⁰⁴

3.8 THE “BOOMERANG EFFECT” UNDER THE TROPICS: HEALTH CONSEQUENCES OF ANTHROPOGENIC CHANGES

Human activity has imposed many changes on planet Earth. Many ecosystems have been modified, for example by the introduction of agriculture, industrialization, and the creation of cities. While these anthropogenic activities have dramatically supported population growth, they have also had adverse environmental effects on human health. Here we examine some of these “boomerang effects” and their causes.

3.8.1 The Bangladesh Story: Persistent Dryness, Safe Drinking Water Availability and Deep-Water Wells

Perhaps the greatest manmade environmental disaster is traceable to the goal of reducing outbreaks of infectious diarrhea among impoverished Bangladeshi people dependent on bacteria-laden well water. In the 1980s and 1990s, a third of the country's wells were replaced by tube wells drawing on deep aquifers that yielded water contaminated with arsenic.^{105,106} Half of the new wells had an arsenic concentration of 50 mg/L, far above the World Health Organization (WHO) acceptable standard (<10 mg/L). This exposed 35–77 million people to daily doses of arsenic, a proven cause of skin lesions, cancers and neurological effects. While the goal of preventing waterborne infectious disease was laudable, the resulting chronic waterborne intoxication illustrates why diverse expertise must be considered before such far-reaching decisions are made. Unfortunately, tube-wells have sometimes failed to protect against gastrointestinal diseases in Bangladesh, despite regular use of tube-well water for drinking.¹⁰⁷

3.8.2 Agriculture Under the Tropics

Climate has an important influence on soil characteristics and vegetation and thus determines an area's suitability for agriculture, farming practice, and the type of crops that can be grown. Climates affect agriculture in four different ways: by solar radiation, temperature, precipitation and wind. Indirectly, climate influences agriculture by its effects on soil formation. The diverse types of tropical climate correspond with great variation to the agricultural potential of different parts of the tropics, named agro-ecological zones. “Three worlds of the tropics” can be distinguished in Africa: the humid tropics or rainforest zone, the sub-humid tropics or savannah zone and the semiarid or Sahel zone, which extends into subtropical latitudes.

Even if all the types of tropical agriculture depend on atmospheric conditions, climatic and especially rain variability make tropical agriculture often a risky business, and many famines in the tropics are related to drought. As crops react differently to drought stress, selecting the right crop diminishes the risk of dryland farming. In dry and seasonal dry regions, water shortage and low soil fertility are the most important constraints on crop production.

Global projections describe continued but geographically disproportionate expansion of the human population based on differential regional fertility rates, such that almost 25% of humans will reside in Africa by 2050. By 2025, the Food and Agriculture Organization predicts that 480 million people in Africa could be living in areas with very scarce water, and that as climatic conditions deteriorate, 600,000 km² of land currently classed as moderately constrained will become severely limited. Global warming is predicted to have a general negative effect on plant growth due to the damaging effect of high temperatures on plant development, with predictable results for the availability of animal feed and human food. The increasing threat of climatological extremes, including very high temperatures, might lead to catastrophic reduction of crop productivity, widespread famine, and heavy dependence on environmentally tolerant plants such as cassava and grasspea (*vide supra*).^{108,109}

3.8.3 Perturbation of the Global Nutrient Cycles

Along with climate change, nutrient enrichment of water bodies, caused by the widespread use and runoff of fertilizers, is one of the most profound changes in the Earth's ecosystems. Acting in synergy with the climate, this excess of nutrients, phosphorus and nitrogen favors the growth and proliferation of many organisms involved in the emergence of human (vector-borne infections) and wildlife diseases (HABs, coral reef diseases).¹¹⁰

3.8.4 Climate in Tropical Cities

More than the half of the world's population is now urban and many of the rapidly expanding cities are located in the tropics notably sub-Saharan Africa (e.g., Lagos, Kinshasa, Kampala).^{111,112} Cities have some particular climatic features due to several factors: the urban solar energy's cycle; the effect of urbanism (heat circulation); evapotranspiration and anthropogenic heat sources. There is a clear temperature difference (up to 10°C) between cities and surrounding rural areas, a gradient termed the urban heat island

(UHI).¹¹³ UHI intensities are generally lower in the tropics as compared to those of comparable cities in temperate zones. There is a double fluctuation in temperature associated with diurnal changes and seasonal variation.^{114,115} Cities induce health effects related directly to the temperature (UHI) and to the related production of ozone. The relation between outdoor temperature and mortality risk for many cities is well established.¹¹³

3.9 CONCLUSIONS

Climate scientists have shown an expansion over the past few decades of the tropics both northward and southward, thus confirming several climate models. While there is uncertainty about the causes and mechanisms, the forecast is a widening of tropical climes in the future, notably related to climate change. This will cause fundamental shifts in ecosystems and in human settlement.^{115,116} There is also a huge change in demography (population increase) in many countries, notably in Africa, which is associated with a constant urbanization. Climate change in these conditions could have major negative consequences.

Diverse expertise must be combined to analyze climate trends and thereby predict impacts on biological systems, including human health. There is precedence in veterinary health when, in the 1970s, the science of Ecopathology emerged to address the biological, physical, human, and economic causal elements of disease among livestock. That heuristic concept led to a search for previously unexplored issues, including habitat, hygiene, herd movements, climate and husbandry.¹¹⁷ Relevant here is the “One Health” approach that seeks to address the emergence of human disease from wildlife and livestock populations in multiple regions of the world. “More than 60% of human infectious diseases are caused by pathogens shared with wild or domestic animals.”¹¹⁸ The “Ecosystem approaches to health” or “Ecohealth” extends “One Health” in an ecological approach to the interactions between ecosystems, society and health of animals and humans. “One World One Health” became a protected trademark in 2009.¹¹⁹ Although there are some nuances between these concepts,¹²⁰ their importance is huge, with major practical applications in public health management^{121,122} and in facing the health consequences of climate change.¹²³ Of course, a holistic approach that addresses the whole biological complexity of “Life on Earth” is needed to address the manifold causes of neurological disease in the tropics and beyond.

References

1. Ritter ME. *The physical environment: an introduction to physical geography*. <www.earthonlinemedia.com/ebooks/>; 2006. Accessed 27.07.16.
2. National Weather Service. *Inter-tropical convergence zone*. <www.srh.noaa.gov/jetstream/tropics/itcz.html>. Accessed 31.11.16.
3. The British Geographer. *Tropical revolving storms: Cuba 2008*. <<http://thebritishgeographer.weebly.com/the-climate-of-tropical-regions.html>>. Accessed 31.11.16.
4. Violle C, Reich PB, Pacala SW, Enquist BJ, Kattgei J. The emergence and promise of functional biogeography. *PNAS*. 2014;111:13690–13696.
5. Osborne PL. *Tropical Ecosystems and Ecological Concepts*. 2nd ed. Cambridge: Cambridge University Press; 2012.
6. Internet Geography. *Tropical rainforest*. <www.geography.learnontheinternet.co.uk/topics/rainforest.html#structure>; 2015. Accessed 27.07.16.
7. Conservatory of Flowers. *Tropical ecosystems*, update 07/2014. <www.conservatoryofflowers.org/sites/default/files/Tropical%20Ecosystem.pdf>; 2014. Accessed 27.07.16.
8. Condit R, Bettina MJ, Engelbrecht BMJ, Delicia Pino D, Pérez R, Turner BL. Species distributions in response to individual soil nutrients and seasonal drought across a community of tropical trees. *PNAS*. 2013;110:5064–5068.
9. FAO. *Arid Zone Forestry: A Guide for Field Technicians*. Rome: FAO; 1989. <www.fao.org/docrep/t0122e/t0122e03.htm>. Accessed 27.07.16.
10. Boval M, Dixon RM. The importance of grasslands for animal production and other functions: a review on management and methodological progress in the tropics. *Animals*. 2012;6:748–762.
11. Al-Kalbani MS, Martin F, Price MF, Abahussain A, Ahmed M, O'Higgins T. Vulnerability assessment of environmental and climate change impacts on water resources in Al Jabal Al Akhdar, Sultanate of Oman. *Water*. 2014;6:3118–3135.
12. Gain AK, Giupponi C, Renaud FG. Climate change adaptation and vulnerability assessment of water resources systems in developing countries: a generalized framework and a feasibility study in Bangladesh. *Water*. 2012;4:345–366.
13. Corlett RT. Where are the subtropics? *Biotropica*. 2013;0:1–3, The Association for Tropical Biology and Conservation.
14. University of California Museum of Paleontology. *The marine biome*. <www.ucmp.berkeley.edu/exhibits/biomes/marine.php>. Accessed 27.07.16.
15. UNEP. *Vital Water Graphics: an Overview of the State of the World's Fresh and Marine Waters*. 2nd ed. Nairobi, Kenya: UNEP; 2008. <<http://www.unep.org/dewa/vitalwater/>>. Accessed 27.07.16.
16. Sale PF, Agardy T, Ainsworth CH, Feist BE, Bell JD, Christie P, et al. Transforming management of tropical coastal seas to cope with challenges of the 21st century. *Mar Pollut Bull*. 2014;85:8–23.
17. Price ARG. The marine food chain in relation to biodiversity. *Scientific World Journal*. 2001;1:579–587.
18. McKinnon AD, Williams A, Young J, Ceccarelli D, Dunstan P, Brewin RJ, et al. Tropical marginal seas: priority regions for managing marine biodiversity and ecosystem function. *Ann Rev Mar Sci*. 2014;6:415–437.
19. Plaisance L, Caley MJ, Brainard RE, Knowlton N. The diversity of coral reefs: what are we missing? *PLoS One*. 2011;6:e25026. Available from: <http://dx.doi.org/10.1371/journal.pone.0025026>.
20. Miloslavich P, Klein E, Diaz JM, Hernandez CE, Bigatti G, Campos L. Marine biodiversity in the Atlantic and Pacific coasts of South America: knowledge and gaps. *PLoS One*. 2011;6:e14631. Available from: <http://dx.doi.org/10.1371/journal.pone.0014631>.

21. Gaston KJ. Global patterns in biodiversity. *Nature*. 2000;405: 220–227.
22. Bawa KS, Kress WJ, Nadkarni NM, Lele S. Beyond paradise—meeting the challenges in tropical biology in the 21st century. *Biotropica*. 2004;36:437–446.
23. Stevens GC. The latitudinal gradient in geographical range: how so many species coexist in the tropics. *Am Nat*. 1989;133:240–256.
24. Erwin DH. Climate as a driver of evolutionary change. *Curr Biol*. 2009;19:R575–R583.
25. Guerrero R. Microbial ecology comes of age. *Int Microbiol*. 2002;5:157–159. Available from: <http://dx.doi.org/10.1007/s10123-002-0093-9>.
26. Escalante AE, Pajares S. The coming of age of microbial ecology. In: Benítez M, Miramontes O, Valiente-Banuet A, eds. *Frontiers in Ecology, Evolution and Complexity*. Mexico City: Coplt-arXives; 2014:1–12.
27. Allison SD, Martiny JBH. Resistance, resilience, and redundancy in microbial communities. *PNAS*. 2008;105:11512–11519.
28. Womack AM, Bohannan BJM, Green JL. Biodiversity and biogeography of the atmosphere. *Philos Trans R Soc B*. 2010;365: 3645–3653.
29. Knies JL, Kingsolver JG, Burch CL. Hotter is better and broader: thermal sensitivity of fitness in a population of bacteriophages. *Am Nat*. 2009;173:419–430.
30. Frazier MR, Huey RB, Berrigan D. Thermodynamics constrains the evolution of insect population growth rates: “warmer is better”. *Am Nat*. 2006;168:512–520.
31. Addo-Bediako A, Chown SL, Gaston KJ. Thermal tolerance, climatic variability and latitude. *Proc R Soc Lond B*. 2000;267:739–745.
32. Ghalambor CK, Huey RB, Martin PR, Tewksbury JJ, Wang G. Are mountain passes higher in the tropics? Janzen’s hypothesis revisited. *Integr Comp Biol*. 2006;46:5–17.
33. Sunday JM, Bates AE, Dulvy NK. Thermal tolerance and the global redistribution of animals. *Nat Clim Change*. 2012;2:686–690.
34. Rozner H. Survival of the flexible. *Nature*. 2013;494:22–23.
35. Bonebrake TC, Deutsch CA. Climate heterogeneity modulates impact of warming on tropical insects. *Ecology*. 2012;93:449–455.
36. Bozinovic F, Gallardo P. The water economy of South American desert rodents: from integrative to molecular physiological ecology. *Comp Biochem Physiol C Toxicol Pharmacol*. 2006;142:163–172.
37. Lüttge U. Ability of crassulacean acid metabolism plants to overcome interacting stresses in tropical environments. *AoB Plants*. 2010;2010:plq005. Available from: <http://dx.doi.org/10.1093/aobpla/plq005>.
38. Rice PS. Ultra-violet radiation is responsible for the differences in global epidemiology of chickenpox and the evolution of varicella-zoster virus as man migrated out of Africa. *Virol J*. 2011;8:189–195. Available from: <http://dx.doi.org/10.1186/1743-422X-8-189>.
39. Vaughan G, Rodríguez-Castillo A, Cruz-Rivera MY, Ruiz-Tovar K, Ramírez-González JE, Rivera-Osorio P. Is ultra-violet radiation the main force shaping molecular evolution of varicella-zoster virus? *Virol J*. 2011;8:370–373. Available from: <http://dx.doi.org/10.1186/1743-422X-8-370>.
40. Korostil IA, Regan DG. Varicella-Zoster virus in Perth, Western Australia: seasonality and reactivation. *PLoS One*. 2016;11:e0151319. Available from: <http://dx.doi.org/10.1371/journal.pone.0151319>.
41. Jeong C, Di Rienzo A. Adaptations to local environments in modern human populations. *Curr Opin Genet Dev*. 2014;29:1–8.
42. Jablonski NG, Chaplin G. Human skin pigmentation as an adaptation to UV radiation. *PNAS*. 2010;107:8962–8968.
43. Jablonski NG, Chaplin G. Human skin pigmentation, migration and disease susceptibility. *Philos Trans R Soc B*. 2012;367:785–792.
44. Dumas M, Preux PM. Is tropical neurology specific? In: Preux PM, Dumas M, eds. *Neuroepidemiology in Tropical Health*. 1st ed. Amsterdam: Elsevier/Academic Press; 2016:1–10.
45. Greaves M. Was skin cancer a selective force for black pigmentation in early hominin evolution? *Proc Biol Sci*. 2014;281:20132955. Available from: <http://dx.doi.org/10.1098/rspb.2013.2955>.
46. Ullrich SE. Does exposure to UV radiation induce a shift to a Th2-like immune reaction? *Photochem Photobiol*. 1996;64:254–258.
47. Gautam P, Chaurasia A, Bhattacharya A, Grover R, Indian Genome Variation Consortium R, Mukerji M. Population diversity and adaptive evolution in keratinization genes: impact of environment in shaping skin phenotypes. *Mol Biol Evol*. 2014;32: 555–573.
48. Spencer PS, Ludolph AC, Kisby GE. Neurologic diseases associated with use of plant components with toxic potential. *Environ Res*. 1993;62:106–113.
49. Tshala-Katumbay DD, Spencer PS. Toxic disorders of the upper motor neuron system. In: Eisen A, Shaw P, eds. *Handbook of Clinical Neurology: Motor Neuron Disorders and Related Diseases*. Vol. 82. Edinburgh: Elsevier; 2007:353–372.
50. Spencer PS. Lathyrism. In: Vinken PJ, Bruyn GW, Klawans HL, eds. *Handbook of Clinical Neurology*, Part 2. Vol. 21. Amsterdam: Elsevier Science Publishers; 1995:1–20.
51. Reis J, Spencer PS. Lathyrism. In: Chopra J, Sawhney MS, eds. *Neurology in Tropics*. 2nd ed. New Delhi: Elsevier India; 2015: 369–378.
52. Spencer PS, Gardner E, Palmer VS, Kisby GE. Environmental neurotoxins linked to a prototypical neurodegenerative disease. In: Aschner M, Costa L, eds. *Environmental Factors in Neurodevelopment and Neurodegenerative Disorders*. New York: Elsevier; 2015:212–237.
53. Podgorná E, Issa Diallo I, Vangenot C, Sanchez-Mazas A, Sabbagh A, Černý V. Variation in NAT2 acetylation phenotypes is associated with differences in food producing subsistence modes and ecoregions in Africa. *BMC Evol Biol*. 2015;15:263. Available from: <http://dx.doi.org/10.1186/s12862-015-0543-6>.
54. Galiana E, Marais A, Mura C, Industri B, Arbiol G, Ponchet M. Ecosystem screening approach for pathogen-associated microorganisms affecting host disease. *Appl Environ Microbiol*. 2011;77: 6069–6075.
55. Guernier V, Guégan JF. May Rapoport’s rule apply to human associated pathogens? *Ecohealth*. 2009;6:509–521. Available from: <http://dx.doi.org/10.1007/s10393-010-0290-5>.
56. Kamiya T, O’Dwyer K, Nakagawa S, Poulin R. What determines species richness of parasitic organisms? A meta-analysis across animal, plant and fungal hosts. *Biol Rev Camb Philos Soc*. 2014;89:123–134. Available from: <http://dx.doi.org/10.1111/brev.12046>.
57. Altizer S, Ostfeld RS, Johnson PTJ, Kutz S, Harvell CD. Climate change and infectious diseases: from evidence to a predictive framework. *Science*. 2013;341:514–519. Available from: <http://dx.doi.org/10.1126/science.1239401>.
58. Stromberg BE. Environmental factors influencing transmission. *Vet Parasitol*. 1997;72:247–264.
59. Pietrock M, Marcoglies DJ. Free-living endohelminth stages: at the mercy of environmental conditions. *Trends Parasitol*. 2003;19: 293–299.
60. Patz JA, Githeko AK, McCarty JP, Hussein S, Confalonieri U, deWet N. Climate change and infectious diseases. In: McMichael AJ, Campbell-Lendrum DH, Corvalan CF, Ebi KL, Scheraga JD, Woodward A, eds. *Climate Change and Human Health: Risks and Responses*. Geneva, Switzerland: WHO; 2003:103–132.
61. Grassly NC, Fraser C. Seasonal infectious disease epidemiology. *Proc Biol Sci*. 2006;273:2541–2550. Available from: <http://dx.doi.org/10.1098/rspb.2006.3604>.
62. Naumova EN. Mystery of seasonality: getting the rhythm of nature. *J Public Health Policy*. 2006;27:2–12.

63. Fisman D. Seasonality of viral infections: mechanisms and unknowns. *Clin Microbiol Infect.* 2012;18:946–954.
64. Fares A. Factors influencing the seasonal patterns of infectious diseases. *Int J Prev Med.* 2013;4:128–132.
65. Descloux E, Mangeas M, Menkes CE, Lengaigne M, Leroy A, Tehei T. Climate-based models for understanding and forecasting Dengue epidemics. *PLoS Negl Trop Dis.* 2012;6:e1470. Available from: <http://dx.doi.org/10.1371/journal.pntd.0001470>.
66. Mouchet J, Carnevale P, Coosemans M, Julvez J, Manuin S, Richard-Lenoble D. *Biodiversité du Paludisme Dans le Monde*. Montrouge, France: John Libbey Eurotext; 2004.
67. Handschumacher P, Schwartz D. Do pedo-epidemiological system exists? In: Landa ER, Feller C, eds. *Soil and Culture*. New York: Springer, IRD; 2010:355–368.
68. Touré K, Coly M, Toure D, Fall M, Sarr MD, Diouf A. Investigation of death cases by pesticides poisoning in a rural community, Bignona, Senegal. *Epidemiol.* 2011;1:105. doi:10.4172/2161-1165.1000105. <<http://www.omicsonline.org/investigation-of-death-cases-by-pesticides-poisoning-in-a-rural-community-bignona-senegal-2161-1165.1000105.php?aid=2193>>. Accessed 06.11.16.
69. Anonymous. Toxic hypoglycemic syndrome – Jamaica, 1989–1991. *MMWR.* 1992;41:53–55.
70. Spencer PS, Palmer VS, Mazumder R. Probable toxic cause for suspected lychee-linked viral encephalitis [letter]. *Emerg Infect Dis.* 2015;21:904–905. Available from: <http://dx.doi.org/10.3201/eid2105.141650>.
71. Ming L. Moldy sugarcane poisoning—a case report with a brief review. *J Toxicol Clin Toxicol.* 1995;33:363–367.
72. Lye MS, Ghazali AA, Mohan J, Alwin N, Nair RC. An outbreak of acute hepatic encephalopathy due to severe aflatoxicosis in Malaysia. *Am J Trop Med Hyg.* 1995;53:68–72.
73. Spencer PS, Mazumder R, Palmer VS, Lasarev MR, Stadnik RC, King P. Environmental, dietary and case-control study of Nodding Syndrome in Uganda: a post-measles brain disorder triggered by malnutrition? *J Neurol Sci.* 2016;369:191–203. Available from: <http://dx.doi.org/10.1016/j.jns.2016.08.023>.
74. EPA USA. *Nutrient pollution*. <www.epa.gov/nutrientpollution/climate-change-and-harmful-algal-blooms>. Accessed 06.11.16.
75. Kovats RS. El Nino and human health. *Bull World Health Organ.* 2000;9:1127–1135.
76. Ummenhofer CC, England MH, McIntosh PC, Meyers GA, Pook MJ, Risbey JS. What causes southeast Australia's worst droughts? *Geophys Res Lett.* 2009;36:L04706. Available from: <http://dx.doi.org/10.1029/2008GL036801>.
77. Walsh RP, Newbery DM. The ecoclimatology of Danum, Sabah, in the context of the world's rainforest regions, with particular reference to dry periods and their impact. *Philos Trans R Soc Lond B Biol Sci.* 1999;354:1869–1883.
78. Davies GI, McIver L, Kim Y, Hashizume M, Iddings S, Chan V. Water-borne diseases and extreme weather events in Cambodia: review of impacts and implications of climate change. *Int J Environ Res Public Health.* 2015;12:191–213. Available from: <http://dx.doi.org/10.3390/ijerph120100191>.
79. Doocy S, Dick A, Daniels A, Kirsch TD. The human impact of tropical cyclones: a historical review of events 1980–2009 and systematic literature review. *PLoS Curr.* 2013;16:5. Available from: <http://dx.doi.org/10.1371/currents.dis.2664354a5571512063ed29d25ffbc74>.
80. Verisk Maplecroft. *Climate Change Vulnerability Index (CCVI)*. <<https://maplecroft.com/about/news/ccvi.html>>. Accessed 30.07.16.
81. Kreft S, Eckstein D, Dorsch L, Fischer L. *Global Climate Risk Index 2016 Who Suffers Most From Extreme Weather Events? Weather-Related Loss Events in 2014 and 1995 to 2014*. Bonn, Germany: Germanwatch; 2015. <www.germanwatch.org/en/cr/>. Accessed 27.07.16.
82. Stanke C, Kerac M, Prudhomme C, Medlock J, Murray V. Health effects of drought: a systematic review of the evidence. *PLoS Curr.* 2013;5:pii. Available from: <http://dx.doi.org/10.1371/currents.dis.7a2cee9e980f91ad7697b570bcc4b004>.
83. Ahern M, Kovats RS, Wilkinson P, Few R, Matthies F. Global health impacts of floods: epidemiologic evidence. *Epidemiol Rev.* 2005;1:36–46. Available from: <http://dx.doi.org/10.1093/epir-ev/mxi004>.
84. McGregor GR, Bessemoulin P, Ebi K, Menne B. *Heatwaves and Health: Guidance on Warning-System Development*, WMO-No. 1142. Geneva, Switzerland: World Meteorological Organization and World Health Organization; 2015.
85. Wikipedia. 2015 Pakistan heat wave. <https://en.wikipedia.org/wiki/2015_Pakistan_heat_wave>. Accessed 06.11.16.
86. Díaz J, Tobías A, Linares C. Saharan dust and association between particulate matter and case-specific mortality: a case-crossover analysis in Madrid (Spain). *Environ Health.* 2012;11:11. Available from: <http://dx.doi.org/10.1186/1476-069X-11-11>.
87. Goudie AS. Desert dust and human health disorders. *Environ Int.* 2014;63:101–113. Available from: <http://dx.doi.org/10.1016/j.envint.2013.10.011>.
88. Griffith DW. Atmospheric movement of microorganisms in clouds of desert dust and implications for human health. *Clin Microbiol Rev.* 2007;20:459–477. Available from: <http://dx.doi.org/10.1128/CMR.00039-06>.
89. Yamaguchi N, Park J, Kodama M, Ichijo T, Baba T, Nasu M. Changes in the airborne bacterial community in outdoor environments following Asian dust events. *Microbes Environ.* 2014;29: 82–88. Available from: <http://dx.doi.org/10.1264/jsm2.ME13080>.
90. Sultan B, Labadi K, Guegan JF, Janicot S. Climate drives the meningitis epidemics onset in West Africa. *PLoS Med.* 2005;2:e6.
91. Shirber M. *Climate conditions help forecast meningitis outbreaks*; March 24, 2014. <<http://climate.nasa.gov/news/1054/climate-conditions-help-forecast-meningitis-outbreaks/>>.
92. Mbaye I, Handschumacher P, Chippaux J-P, Diallo A, Ndione JA, Paul P. Influence du climat sur les épidémies de méningite à méningocoque à Niakhar (Sénégal) de 1998 à 2000 et recherche d'indicateurs opérationnels en santé publique. *Environ Risques Santé.* 2004;3:219–226.
93. Berrang-Ford L, Jamie L, Breau S. Conflict and human African trypanosomiasis. *Soc Sci Med.* 2011;72:398–407.
94. Courtin F, Jamonneau V, Duvallet G, Camara M, Kaba D, Solano P. Un siècle de “trypano” en Afrique de l'Ouest. Communication affichée lors des journées du centenaire de la SPE. *Bull Soc Pathol Exot.* 2008;101:287–289.
95. Hay SI, Guerra CA, Tatem AJ, Noor AM, Snow RW. The global distribution and population at risk of malaria: past, present and future. *Lancet Infect Dis.* 2004;4:327–336. doi:10.1016/S1473-3099(04)01043-6.
96. Yamana TK, Bomblies A, Eltahir EAB. Climate change unlikely to increase malaria burden in West Africa. *Nat Clim Change.* 2016;6:1009–1015.
97. Marques RC, Bernardi JVE, Dórea JG, Leão RS, Malm O. Mercury transfer during pregnancy and breastfeeding: hair mercury concentrations as biomarker. *Biol Trace Elem Res.* 2013;154:326–332.
98. Maurice-Bourgoin L., Aalto R., Guyot JL, 2002, Sediment-associated mercury distribution within a major Amazon tributary: century-scale contamination history and importance of flood plain accumulation, *The Structure, Function and Management Implications of Fluvial Sedimentary Systems* (Proceedings of an international symposium, Alice Springs, Australia, September 2002). IAI IS Publ. no. 276, pp.161-168.

99. Lechler PJ, Miller JR, Lacerda LD, Vinson D, Bonzongo JC, Lyons WB. Elevated mercury concentrations in soils, sediments, water, and fish of the Madeira River basin, Brazilian Amazon: a function of natural enrichments? *Sci Total Environ*. 2000;260:87–96.
100. Roulet M, Lucotte M, Saint-Aubin A, Tran S, Rhéault I, Farella N. The geochemistry of Hg in Central Amazonian soils developed on the Alter-do-Chao formation of the lower Tapajos river valley, Para state, Brazil. *Sci Total Environ*. 1998;223:1–24.
101. Do Valle CM, Santana GP, Augusti R, Egreja Filho FB, Windmüller CC. Speciation and quantification of mercury in Oxisol, Ultisol, and Spodosol from Amazon (Manaus, Brazil). *Chemosphere*. 2005;58:779–792.
102. Tschirhart C, Handschumacher P, Laffly D, Bénéfice E. Resource management, networks and spatial contrasts in human mercury contamination along the Rio Beni (Bolivian Amazon). *Hum Ecol*. 2012;40:511–523.
103. Cordier S, Grasmick C, Pasquier-Passelaigue M, Mandereau L, Weber JP, Jouan M. Mercury exposure in French Guiana: levels and determinants. *Arch Environ Health*. 1998;53:299–303.
104. Wantzen K, Mol JH. Soil erosion from agriculture and mining: a threat to tropical stream ecosystem. *Agriculture*. 2013;3:660–683.
105. Yu WH, Harvey CM, Harvey CF. Arsenic in groundwater in Bangladesh: a geostatistical and epidemiological framework for evaluating health effects and potential remedies. *Water Resour Res*. 2003;39:1146. Available from: <http://dx.doi.org/10.1029/2002WR001327>.
106. Chakraborti D, Rahman MM, Mukherjee A, Alauddind M, Hassane M, Dutta RH. Groundwater arsenic contamination in Bangladesh—21 Years of research. *J Trace Elem Med Biol*. 2015;31:237–248.
107. Islam MS, Siddika A, Khan MN, Goldar MM, Sadique MA, Kabir ANMH. Microbiological analysis of tube-well water in a rural area of Bangladesh. *Appl Environ Microbiol*. 2001;67:3328–3330.
108. Uni-Goettingen. *Tropical Agroecosystem Function SS08 U2&U11—climatic factors & permanent cropping with annuals*; 4–23. <www.uni-goettingen.de/de/363593.html>. Accessed 27.07.16.
109. Ramirez-Villegas J, Thornton PK. *Climate change impacts on African crop production*. CCAFS working paper no. 119. Copenhagen, Denmark: Research Program on Climate Change, Agriculture and Food Security. <www.ccafs.cgiar.org>; 2015. Accessed 27.07.16.
110. Johnson PTJ, Townsend AR, Cleveland CC, Glibert PM, Howarth RW, McKenzie VJ, et al. Linking environmental nutrient enrichment and disease emergence in humans and wildlife. *Ecol Appl*. 2010;20:16–29.
111. Butsch C, Sakdapolrak P, Saravanan VS. Urban health in India. *Int Asianforum*. 2012;43:13–32.
112. Roth M. Review of urban climate research in (sub) tropical regions. *Int J Climatol*. 2007;27:1859–1873. Available from: <http://dx.doi.org/10.1002/joc.1591>.
113. Rydin Y, Bleahu A, Davies M, Dávila JD, Friel S, De Grandis G, et al. Shaping cities for health: complexity and the planning of urban environments in the 21st century. *Lancet*. 2012;379:2079–2108.
114. Reiner Jr RC, Smith DL, Gething PW. Climate change, urbanization and disease: summer in the city. *Trans R Soc Trop Med Hyg*. 2015;109:171–172. Available from: <http://dx.doi.org/10.1093/trstmh/tru194>.
115. Seidel DJ, Fu Q, Rander WJ, Reichler TJ. Widening of the tropical belt in a changing climate. *Nat Geosci*. 2008;1:21–24. Available from: <http://dx.doi.org/10.1038/ngeo.2007.38>.
116. Lu J, Deser C, Reichler T. Cause of the widening of the tropical belt since 1958. *Geophys Res Lett*. 2009;36:L03803. Available from: <http://dx.doi.org/10.1029/2008GL036076>.
117. Anonymous. *Ecopathology: the influence of environment on disease*. Spore. 1994;49:6.
118. Karesh WB, Dobson A, Lloyd-Smith JO, Lubroth J, Dixon MA, Bennett M, et al. Ecology of zoonoses: natural and unnatural histories. *Lancet*. 2012;380:1936–1945.
119. Zinsstag J, Schelling E, Waltner-Toews D, Tanner M. From “one medicine” to “one health” and systemic approaches to health and well-being. *Prev Vet Med*. 2011;101:148–156.
120. Roger F, Caron A, Morand S, Pedrono M, de Garine-Wichatitsky M, Chevalier V, et al. One health and ecohealth: the same wine in different bottles? *Infect Ecol Epidemiol*. 2016;6:1–4. Available from: <http://dx.doi.org/10.3402/iee.v6.30978>.
121. Asakura T, Mallee H, Tomokawa S, Moji K, Kobayashi J. The ecosystem approach to health is a promising strategy in international development: lessons from Japan and Laos. *Globalization Health*. 2015;11:1–8. Available from: <http://dx.doi.org/10.1186/s12992-015-0093-0>.
122. Leung Z, Middleton D, Morrison K. One Health and Ecohealth in Ontario: a qualitative study exploring how holistic and integrative approaches are shaping public health practice in Ontario. *Public Health*. 2012;126:358. <<http://www.biomedcentral.com/1471-2458/12/358>>
123. Patz JA, Hahn MB. Climate change and human health: a One Health approach. *Curr Top Microbiol Immunol*. 2013;366:141–171. Available from: http://dx.doi.org/10.1007/82_2012_274.

Further Reading

- Courtin F, Jamonneau V, Duvallet G, Garcia A, Coulibaly B, Doumenge JP, Cuny G, Solano P. Sleeping sickness in West Africa (1906–2006): changes in spatial repartition and lessons from the past. *Trop Med Int Health*. 2008;13:334–344.
- Greaves M. Response to Jablonski and Chaplin. *Proc Biol Sci*. 2014;281:20140940. Available from: <http://dx.doi.org/10.1098/rspb.2014.0940>.
- Mbaye I, Sy MD, Handschumacher P. *Contraintes biologiques, gestion agricole et risque éco-toxicologique dans le district de Bignona (Région de Ziguinchor/Sénégal): Pourquoi une vulnérabilité hétérogène? Natures Tropicales, enjeux et perspectives*. Bordeaux: Presses Universitaires; 2012:57–68.