

Dimensions of Effects of Climate Change on Water-Transmitted Infectious Diseases

Marieta AH Braks¹ and Ana Maria de Roda Husman^{2*}

¹Laboratory for Zoonoses and Environmental Microbiology, Centre for Infectious Disease Control Netherlands, National Institute for Public Health and the Environment (RIVM), BA Bilthoven, The Netherlands

²Divisions Environmental Epidemiology/Veterinary Public Health, Institute for Risk Assessment Sciences, Faculty of Veterinary Medicine, Utrecht University, TD Utrecht, The Netherlands

Abstract

Human pathogens that are water-transmitted may follow various routes, ranging from water ingestion to transmission via insect vectors. Since water-transmitted pathogens are highly influenced by climate and environmental conditions, any climate change may alter the infectious disease burden from exposure to these pathogens. Climate factors determine the number, type, virulence and infectivity of pathogens transmitted through water or vectors that breed in water, and thus may have an impact on resulting infectious diseases. In this perspective, the most important climate factors are temperature, relative humidity, UV radiation, precipitation patterns and water availability.

Some indigenous species of bacteria, amoebas and algae are able to grow in aquatic environments with higher temperatures, whereas enteric bacteria, viruses and parasites that are derived from human or animal faeces are not. Increased UV radiation may result in increased inactivation of enteric pathogens, or alternatively growth, whereas increased precipitation intensity will lead to peak concentrations of these pathogens due to e.g. sewage overflow and runoff. For mosquito borne disease, high spatio-temporal variation and uncertainties in the many variables that determine their emergence preclude general projections in the future.

Climate factors not only affect pathogen behaviour, but also influence human behaviour, thus stressing the need to study both the complexity of pathogen behaviour and social behaviour with respect to expected climate changes.

Keywords: Climate change; Water-transmitted infectious diseases; Pathogen; Vector

Introduction

Global climate change is anticipated to have a wide-ranging impact and possibly adverse effects on human health, which may be direct or indirect. Direct effects include extreme weather conditions, such as floods, and sea level rise, e.g. leading to an increased risk of drowning, and temperature related effects, such as heat waves and smog. An increased frequency or severity of heat waves would cause an increase in mortality and illness. Indirect effects may include change in distribution and incidences of infectious diseases [1]. Initial projections of effects of climate change on health suggested dramatic future increases in the geographic range and incidence of infectious diseases [2-4]. The impact of climate change on the distribution and prevalence of infectious diseases, however, is currently under debate [5,6]. Increasing evidence shows that infectious disease emergence is a complex and dynamic process, which preclude generalisations. In addition, many factors, including policy and intervention measures, can affect the epidemiology of an infectious disease, and some may overshadow the effects of climate [7,8]. On the other hand, climate change may lead to a reduction in the burden of some specific infectious diseases.

While the majority of recent publications acknowledge the complexity and uncertainties of the relation between climate change and infectious disease, the generalized idea that disease transmission is favoured by climate change remains the most appealing working hypothesis and dominates the public debate. In this paper, we focus on the major issues in this discussion with respect to water-transmitted diseases for Western Europe, especially the Netherlands, on the base of examples for which different (minimal, opposite or unknown) effects of climate change are anticipated.

Climate Change and Water-transmitted Infectious Diseases

Infections with water-transmitted pathogens may be asymptomatic, or may result in mild or serious illness in humans [9]. Water-transmitted pathogens may be bacteria, viruses, parasites, amoebas or algae. Pathogens that are water-transmitted may follow various routes, ranging from water ingestion to transmission via insect vectors, and are classified into four different categories according to Bradley [10] (Table 1).

First, water-borne pathogens are passively carried in water bodies. Water-borne diseases are caused by the ingestion of water contaminated by human or animal excreta containing pathogens. Worldwide, water-borne diarrhoeal diseases each year kill an estimated 1.8 million people of which 88% is attributable to the environment, including risks associated with unsafe water, lack of sanitation and poor hygiene [11]. Worldwide, and in Europe, norovirus is found as one of the most prevalent causative agents of gastroenteritis [12]. Cholera (caused by

***Corresponding author:** Ana Maria de Roda Husman, Divisions Environmental Epidemiology/Veterinary Public Health, Institute for Risk Assessment Sciences, Faculty of Veterinary Medicine, Utrecht University, P.O. Box 80, 178/80 175, 3508 TD Utrecht, The Netherlands, Tel: +31302532059/5370; Fax: +31302744434; E-mail: a.m.derodahusman@uu.nl

Received June 05, 2013; **Accepted** July 01, 2013; **Published** July 04, 2013

Citation: Braks MAH, de Roda Husman AM (2013) Dimensions of Effects of Climate Change on Water-Transmitted Infectious Diseases. *Air Water Borne Diseases* 2: 109. doi:[10.4172/2167-7719.1000109](http://dx.doi.org/10.4172/2167-7719.1000109)

Copyright: © 2013 Braks MAH, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Category	Transmission	Climatic determinants	Examples
Water-borne	Ingestion of water contaminated by human or animal faeces or urine containing pathogenic bacteria, viruses or parasites	Water temperature Rainfall/Drought UV radiation	Gastroenteritis, enteric hepatitis, amoebic and bacillary dysentery, cholera, leptospirosis, poliomyelitis, typhoid/paratyphoid fever
Water-washed	Skin, ear or eye contact with contaminated water and poor personal hygiene	Flooding Water temperature Rainfall	Conjunctivitis, trachoma, intestinal helminth infections, leprosy, scabies
Water-based	Organisms that originate in the water or spend part of their life cycle in aquatic animals and come in direct contact with humans in water or by inhalation	Water temperature Relative humidity Rainfall/Drought UV radiation	Dracunculiasis, schistosomiasis, (tricho) bilharziasis, algal diseases, legionellosis
Water-related	Insect vectors breeding in water	Water temperature Air temperature Flooding Relative humidity Rainfall/Drought	Dengue, malaria, West Nile fever, Rift Valley fever

Table 1: Water-transmitted infectious diseases divided into four categories according to their transmission route [10].

Vibrio cholerae) is one of those water-borne infectious diseases that keeps re-emerging in war zones and after natural disasters with 3-5 million affected each year and 100,000-130,000 deaths [13]. Water-washed diseases are the result of contact with contaminated water. One of the most prominent water-washed diseases in the world is trachoma, caused by *Chlamydia trachomatis*, with 300 and 500 million infected people of which approximately 84 million people in need of treatment and about eight million people blinded [14,15]. Trachoma infection occurs most frequently in Africa whereas conjunctivitis (caused by e.g. adenoviruses) is a European example of a water-washed disease. Water-based diseases are caused by organisms that originate in the water or spend part of their life cycle in aquatic animals and come in direct contact with humans in water or by inhalation. In 2000, it was estimated that worldwide at least 200 million people are infected with schistosomiasis, a snail-borne disease, and another 600 million are at risk of infection [16]. Other examples of water-based disease more relevant to Europe include trichobilharziasis, algal diseases and legionellosis. Finally, water-related infectious diseases are caused by pathogens that are transmitted by insect vectors that spend part of their life cycle in water, such as mosquitoes, black flies, and several biting midge species. Examples of human vector borne diseases, among many, are malaria, West Nile fever, and river blindness, whereas blue tongue disease only occurs in animals.

For Western Europe, climate models predict that temperatures will rise, summers will be drier but winter precipitation will increase, and that extreme weather will occur more frequently in the coming decades [17]. For water-transmitted infectious diseases, the increased frequency of heavy rainfall with associated flooding, and the increased temperature are considered the most determinant factors of climate change. In addition, global warming is expected to raise sea levels through the melting of glaciers and polar ice plus the thermal expansion of sea water [17,18]. Growth of specific species of bacteria, amoeba and algae in water may occur under the influence of sunlight and water temperature. On the other hand, these climate factors largely inactivate or kill enteric pathogens that cannot replicate in water but depend on their human or animal host for replication. Other factors such as nutrient levels and dissolved organic carbon also influence the life and death of water-borne pathogens [19]. Climate factors not only affect pathogen behaviour, but also the aquatic environment will be influenced by climate. Rainfall changes the volumes in different water bodies and may cause flooding; air temperature will affect water temperature and evaporation whereas sunlight hours depend on clouding. For water-transmitted infectious diseases in general and water-related diseases specifically, changes in relative humidity

or saturation vapour pressure are additional important determining factors because of the sensitivity of the aerial adult arthropod vectors to it. Though atmospheric temperature and rainfall are regularly recorded by the Intergovernmental Panel on Climate Change, water temperature and relative humidity are not since these are seen as secondary effects of climate change.

Water-borne pathogens

People ingest water during consumption or recreation. Daily drinking water consumption may range from approximately 200 mL in some Western countries to more than 2 L in some (sub) tropical regions [20], also depending on the level of exercise and labour. During water recreation or bathing, water may also be (accidentally) ingested [21,22]. Water-borne pathogens include bacteria, viruses and protozoan parasites. These pathogens merely comprise of enteric bacteria, such as *E. coli* O157, Shigella, and viruses such as adenovirus, enterovirus, norovirus, rotavirus and hepatitis A virus, but also *Staphylococcus aureus* and pathogenic *Leptospira*. Inactivation by increased water temperatures may be less relevant for organisms that create environmentally robust survival structures, such as the protozoan parasites *Cryptosporidium* oocysts and *Giardia* cysts, because they are less temperature sensitive and also more persistent [23]. Of the broad range of pathogens that cause this category of water-transmitted infectious diseases, noroviruses and *Vibrio* bacteria are explicitly discussed in this section.

Noroviruses: Noroviruses belong to the family of the *Caliciviridae*, are 28-35 nm of size, are non-enveloped viruses and contain a positive sense, single-stranded RNA genome of approximately 7.6 kb [24]. The Norovirus genus is divided into five genogroups (genogroup GGI to GGV). The diversity of noroviruses increases continually due to the generation of new variants, with two groups of strains predominating in the past five years [25-29]. Most human pathogenic noroviruses cluster within genogroup (GG) I and GGII. No zoonotic potential could be demonstrated for these two genogroups, therefore they are considered to be solely transmitted between humans. Norovirus infections cause acute gastroenteritis in humans. Symptoms include projectile vomiting, watery non-bloody diarrhoea with abdominal cramps and nausea within 24 to 48 hours after exposure [30]. Infected persons shed large numbers of viral particles to the environment. Transmission occurs through the faecal-oral route. Noroviruses are the primary cause of viral diarrhoea in adults. Besides from contaminated foods, outbreaks originating from contaminated drinking water as well as from recreational water have been described [31-34]. Because no robust cell culture system for the detection of infectious human noroviruses

is available [35], information on the persistence of infectious virus particles in the environment is limited.

Noroviruses cannot grow in aquatic environments, rather at increased water temperatures they will become inactivated [35], probably losing the ability to infect human cells and to replicate, as was shown for e.g. enteroviruses [36]. As a host-dependent pathogen, though stable in warm-blooded animals at 37°C, noroviruses are sensitive to environmental temperatures above e.g. 20°C. Due to the non-enveloped structure of noroviruses, they are presumed to be at least as resistant to environmental degradation and chemical inactivation as other culturable human enteric viruses, such as poliovirus. The effect of increased virus inactivation due to higher water temperatures may be overshadowed by peak concentrations in human pathogenic viruses originating from runoff and sewer overflows, following heavy rainfall events that are expected to occur more often during summers [37]. With rainfall, pathogenic microorganisms of human and animal faecal origin may enter surface waters by discharges of raw and treated wastewater, and runoff from the land of faecal matter from wildlife such as birds, waterfowl and deer, or domestic animals or manure. In this respect, the occurrence of peak concentrations of norovirus has been reported, but in general, peak events appear to be difficult to predict and therefore such measurements are not easily performed [38]. Since norovirus peak concentrations determine the risk of infection, this effect is likely to be of more importance than enhanced virus inactivation.

Vibrio spp.: *Vibrio* species are motile Gram-negative rod shaped bacteria with a single polar flagellum that are common inhabitants of various aquatic environments [39]. Depending on the species, they tolerate a range of salinities from 15-25 ppt and are common in marine environments. They are capable of multiplication in marine water at elevated water temperatures (> 17-20°C). When environmental conditions are unfavourable, *Vibrio* species enter a viable non-culturable state, which enables them to survive such conditions. Many *Vibrio* species cause disease in aquatic animals such as fish, shellfish and marine mammals but also in humans. Human pathogenic *Vibrio* species have been associated with wound infections (*V. alginolyticus*, *V. vulnificus*) and ear infections (*V. alginolyticus*) after exposure to contaminated surface waters, and gastroenteritis (*V. parahaemolyticus*, *V. cholerae* non-O1/O139) after consumption of contaminated food. More serious complications such as septicemia (*V. vulnificus*) have also been reported, although these are rare and mainly occur in people who are immunocompromised or have a chronic liver disease. *V. cholerae* O1/O139 can cause acute watery diarrhoea in humans, which can lead to death, if left untreated [40,41]. *Vibrio* infections as a result of exposure to recreational waters in the Netherlands have been observed during warm summers [42,43] and recent monitoring of Dutch coastal recreational waters has demonstrated the presence of various *Vibrio* species, such as *V. alginolyticus*, *V. parahaemolyticus*, *V. cholerae* non O1/O139 and *V. fluvialis* [44].

Growth patterns of *Vibrio* are highly dependent on water temperature amongst some other environmental conditions. Some algae and amoebas, and bacteria, such as pathogenic *Vibrio*, grow and proliferate in the aquatic environment independent of a host for replication. Their numbers in the water body, either fresh or marine, increase with increasing water temperature.

At the expense of fresh water bodies, an increase in saline and brackish water bodies in coastal areas may be expected due to sea level rise [18]. Such development may create an ideal new habitat for certain *Vibrio* spp. that is highly dependent on salinity for their respective growth rates.

Reduced water availability may result in decreased water volume in surface waters such as recreational water thus resulting in increased concentrations of the pathogens present [37]. However, reduced water levels may result in increased water temperatures and increased sunlight radiation which has an inactivating effect on most pathogens, but to a different extent. A reduced volume of water in lakes or rivers may also change the physiochemical composition of the water, e.g. the availability of nutrients may de- or increase and salinity may rise. The latter may e.g. cause a shift towards proliferation of microorganisms that endure high salt concentrations, such as various human pathogenic *Vibrio* species.

Cholera outbreaks occur seasonally and are associated with monsoon seasons, warm temperatures, heavy rainfall, and increased plankton populations. New major outbreaks of cholera are continuing to occur, especially in the wake of climate changes. During 1998, there was a dramatic increase in the number of cholera cases worldwide compared to 1997, with the total number of cases almost doubling. Colwell and Patz [45] showed the relationship between increased sea-surface temperatures and cholera incidence in Bangladesh. Also temporal dynamics of precipitation coincided, especially when abrupt and heavy, with an exacerbation of the cholera epidemic [46].

Water-washed pathogens

Pathogens causing diseases in this category of water-washed infectious diseases, including *Mycobacterium*, *Chlamydia*, *Shigella*, *Salmonella*, *E. coli*, *adenovirus* and *Pseudomonas*, are transmitted primarily through inadequate supplies of water for basic hygiene practices; thus, exposing individuals to risk through contact of human eyes, ears or skin with contaminated water. Most often in many developing countries, the only source of water available to populations for drinking, bathing, washing clothes, and for animal care is contaminated with pathogens from raw sewage, and human and animal faeces. The survival rates of the pathogenic organisms that cause water-washed diseases are enhanced in warm moist environments, and where situations such as increased flooding facilitate the transportation of the organism, the risk of outbreaks increases. However, lack of water may cause water-washed diseases by poor personal hygiene, and skin or eye contact with contaminated water. In Europe, adenovirus causing conjunctivitis and *Pseudomonas* causing ear infections are common water-washed pathogens and discussed here.

Adenovirus: Adenoviruses are medium-sized (90–100 nm), non-enveloped (naked) icosahedral viruses composed of a nucleocapsid and a double-stranded linear DNA genome. Adenoviruses survive longer in water than enteroviruses and hepatitis A virus, which may in part be due to their double-stranded DNA, and in part by their capsid structure [47]. Viruses of the family *Adenoviridae* infect various species of vertebrates, including humans. There are 51 described serotypes in humans of which one third are associated with human disease, while other infections are asymptomatic [47]. Adenoviruses are responsible for 5-10% of the upper respiratory infections in children, and many infections in adults as well. Children and the immunocompromised are more severely impacted by adenovirus infections than healthy adults [48]. Adenoviruses have been responsible for many recreational water-related outbreaks, more than any other water-borne virus, including a great number of swimming pool related outbreaks [47].

Of the many types of adenoviruses, some (type 40 and 41) are associated with water-borne gastrointestinal illness, whereas other types are associated with respiratory and ocular symptoms. In the Netherlands, adenoviruses appear on the list of viruses with more than

1200 diagnosed cases each year, most likely a large under representation of actual cases [49]. Adenoviruses were frequently detected in Dutch surface waters [50]. They are capable of robust survival in water also at higher water temperatures as predicted in climate change scenarios. Adenoviruses are of equal or greater sensitivity to oxidizing disinfectants compared with other water-borne viruses and are the most resistant pathogens to UV also relevant with respect to an eventual increase in sunlight [47].

Pseudomonas: *Pseudomonas aeruginosa* are aerobic, non-spore-forming, motile, Gram-negative rod shaped bacteria with an optimum growth temperature of 37°C, but they may also grow at 41°C [51]. The rods are approximately 0.5-1×1.5-4 µm in size. *P. aeruginosa* is ubiquitous in water, vegetation and soil. *P. aeruginosa* is capable of using various organic compounds in low concentrations for growth, and can therefore proliferate in aquatic environments. *P. aeruginosa* is an opportunistic pathogen that does not infect healthy individuals, but becomes infectious when the immune system is weakened, e.g. after surgery, during immunosuppressant therapy and antibiotic use. *P. aeruginosa* is resistant to many commonly used antibiotics and is, as such, a feared cause of nosocomial infections. Infections are generally local, affecting wounds, the urinary tract, the respiratory tract, ears, eyes, the intestines and the skin. *P. aeruginosa* multiplies in surface waters when the water temperature exceeds 18-20°C. In swimming pools, the bacterium may proliferate on humid surfaces, but also in places where pool hydraulics are suboptimal. Swimming pool related infections due to *P. aeruginosa* are most often otitis externa and folliculitis; otitis externa is frequently observed after exposure to contaminated surface water. Swimming pool related infections are often associated with insufficient disinfection and cleaning [52,53]. During the summer season, ear complaints associated with *P. aeruginosa* infections as a result of exposure to contaminated surface waters are common in the Netherlands [54]. In drinking water systems, *Pseudomonas* spp. may grow under ambient temperatures with the risk of growth of human pathogenic *Pseudomonas* leading to exposure of humans.

Water-based pathogens

Water-based pathogens are organisms that originate in the water or spend part of their life cycle in aquatic animals leading to infections upon human exposure through contact, by ingestion or by inhalation. Infamous examples include *Dracunculus*, *Schistosoma*, *Trichobilharzia*, *cyanobacteria* and *Legionella* spp.

Trichobilharzia: Swimmers' itch or cercarial dermatitis is the result of the penetration of the human skin by cercariae of bird trematode pathogens of the genus *Trichobilharzia* [55]. These bird schistosomes have a two-host life cycle with freshwater snails as their intermediate host and waterfowl as their final host. Eggs produced by the females embryonate within the host body; fully developed eggs are expelled via faeces or nasal secretions to the aquatic environment. In the water body, the free-swimming larvae infect a suitable snail host, in which the larvae transform to sporocysts that asexually produce cercariae. Cercariae leave the snails into the water and attempt to reach a new host. The free-swimming cercariae exhibit a high affinity for the human skin and try to penetrate the skin resulting in an allergic response. Short exposure and first contact will result in a weak reaction, characterized by development of maculae, or colored markings. Typical symptoms develop after repeated exposure. Shortly after cercarial penetration into the skin a primary itching (like fine prickling) occurs persistently for up to 1 hour and the infection manifests as an itchy maculopapular eruption. Papulae can be encompassed by an erythematous zone that can persist for three days. The itching accompanying the eruption of

papulae can range from negligible to unbearable. After ten days, the papulae usually disappear and leave pigmented spots that can persist for weeks. Severe infections can be accompanied by fever, limb and lymph node swelling, nausea and diarrhea [56]. Outbreaks of presumptive cercarial dermatitis in freshwater lakes are frequently reported, also in the Netherlands [57,58].

In parasites such as *Trichobilharzia*, which depend on intermediate cold-blooded hosts, increased water temperatures accelerate development in freshwater snails and induce increased shedding of cercariae. Moreover, if elevated water temperatures result in more dense algal populations, snails will have excessive feeding opportunities thus resulting in higher numbers of intermediate hosts for *Trichobilharzia*. As a consequence of global warming, bird migration southward decreases and birds become sedentary, resulting in the presence of the parasite's final host for prolonged periods resulting in an extended replication season [59].

Cyanobacteria: Cyanobacteria have some characteristics of bacteria and some of algae. They have the same size as algae, contain blue-green and green pigments and can perform photosynthesis. Due to eutrophication of many water bodies, as a result of human activities, excessive proliferation of cyanobacteria occurs, resulting in extremely high cell densities which may cause considerable nuisance during water recreation due to transparency loss, discoloured water and scum formation. The effects of cyanobacteria on human health are associated with algal toxins. Toxic cyanobacteria are found worldwide in inland and coastal waters [60]. Primarily, marine algal toxins are a problem because they concentrate in shellfish and fish that are eaten by man. However, human exposure may also occur through dermal contact causing 'seaweed dermatitis', water ingestion or inhalation of water droplets. Symptoms related to these exposure routes have primarily been observed in Japan, Australia, New Zealand and the south-eastern states of the USA; cyanobacteria species involved appear not to cause problems in European recreational waters [52]. In freshwater, the most widespread cyanobacterial toxins are microcystins and neurotoxins. Blooming of *Microcystis*, and more recently *Planktothrix* and *Anabaena* is frequently observed in the Netherlands [61]. Internationally, there are numerous reports of lethal poisoning of animals that drank from water with cyanobacteria. Human cases of illness or death were related to exposure to cyanobacterial toxins by renal dialysis and drinking water consumption [60]. However, illness reports related to recreational water are usually anecdotal and there are no publications on large outbreaks in international literature. Moreover, epidemiological studies from all over the world e.g. in Australia, UK, and US, did not find significant differences between people exposed and non-exposed to cyanobacteria in recreational waters, or only noted a trend of more mild health complaints in the exposed compared to the non-exposed at longer duration of water contact and higher cyanobacterial cell counts [62]. There may, however, be long term effects such as liver damage and development of tumours but solid data that demonstrate these effects lack to date [63].

Rising temperatures favour cyanobacteria in several ways. Cyanobacteria generally grow better at higher surface water temperatures (often above 25°C). Warming of fresh surface waters also strengthens the vertical stratification of lakes, reducing vertical mixing. Many cyanobacteria exploit these stratified conditions by forming intracellular gas vesicles, which make the cells buoyant. Buoyant cyanobacteria float upward when mixing is weak and accumulate in dense surface blooms. These surface blooms may even locally increase water temperatures through the intense absorption of

light. The temperatures of surface blooms in the Baltic Sea and in Lake IJsselmeer, the Netherlands, can be at least 1.5°C above those of ambient waters [64]. Both in fresh [65] and marine waters [66], blooms of toxic algae are expected to occur more often at elevated water temperatures. Moreover, as a result of global warming, water temperatures may be higher for prolonged periods, thus lengthening the period for explosive growth of cyanobacteria resulting in extended periods of nuisance blooms that affect recreational water quality.

Legionella: Legionella are Gram-negative, non-spore-forming, motile, aerobic rod shaped bacteria of 0.5-1.0×1.0-3.0 µm in size [67]. The genus comprises over 50 species of which pathogenicity is not always clear; it appears that many pathogenicity factors are involved. Legionella species are ubiquitous in natural and artificial water environments worldwide. Legionella are heterotrophic bacteria that may be free-living, or living within amoebas or biofilms. Legionella proliferate at water temperatures over 25°C; they may be present in high numbers in natural spas using thermal spring water, in poorly maintained hot tubs and whirlpools. In the swimming pool environment, a risk of infection may arise from using showers. Legionella infections occur through inhalation of contaminated aerosols; there is no evidence of person-to-person transmission. *Legionella pneumophila* is the most frequent isolated species that may cause either Legionella-pneumonia or Pontiac-fever, a mild flu-like condition. There are no typical symptoms of Legionella-pneumonia; symptoms are diverse and some infected persons do not develop symptoms [52,68]. Legionella bacteria are frequently detected in tap water distribution systems in the Netherlands [69].

Legionella species proliferate at elevated water temperatures, but need amoebas or other protozoa for their replication. Abundance of Legionella species is not expected to occur in surface water, due to overgrowth of other microorganisms for which environmental growth conditions are more favourable, but Legionella does grow to high levels in drinking water distribution systems that also provide water for showers and whirlpools in swimming pools. Due to increased atmospheric temperature, water used for human consumption, and for interactive water features or fountains may warm in the features' plumbing system thus creating an environment for Legionella proliferation. Rainwater used for drinking or other purposes may also be at risk for the presence of pathogenic *Legionella spp.* [58]. High Legionella numbers in showers, whirlpools, interactive water features and fountains pose health risks through inhalation of aerosols. Increased surface water temperatures, as in outdoor recreational freshwaters, may have minor influence on the occurrence and proliferation of Legionella species; however, warm, humid and showery summer weather was found to be associated with a higher incidence of Legionella disease in the Netherlands [70].

Water-related pathogens

Water-related infectious diseases are caused by pathogens that are transmitted by insect vectors that spend part of their life cycle in water, such as mosquitoes. Vector-borne diseases, in general, are sensitive to climate change, mainly because the bionomics of the arthropod vectors are directly affected by abiotic factors such as ambient temperature and humidity, and therefore also in changes in them. Due to their dependence for reproduction on water bodies, mosquito borne disease are also strongly affected by changes in rainfall patterns causing flooding and droughts, and the rise in sea levels anticipated in climate change scenarios.

Gubler et al. [71] list a range of possible mechanisms whereby changes in temperature have an impact on the risk of transmission of

vector borne diseases. General assumption is that when temperatures increase, transmission of mosquito borne pathogens increases due to shortening of the larval developmental time and decrease of the extrinsic incubation time. The possibility that temperature might negatively affect mosquito (and pathogen) physiology and associated ecology [72] is often ignored, simplified or overlooked. Further, the epidemiology of mosquito borne disease is determined by a complex interplay of multitude factors, many non-climatic, affecting pathogens, mosquitoes and humans [73]. In addition, the fact that many mosquito borne pathogens infect humans, but are circulating naturally among reservoir animal hosts, complicates the picture further [74].

Plasmodium spp.: Since scientific recognition of global warming, the question whether malaria caused by *Plasmodium spp.* parasites will re-emerge in Western Europe, has dominated research into the effect of climate change on public health. Despite a number of reports estimating that the chance is extremely low for Western Europe [75-80], unrest still exists among governments and citizens. Malaria was actually endemic in many locations in Western Europe until the end of the 60's of the last century. In the Netherlands, malaria caused by *Plasmodium vivax* occurred in the coastal areas and was transmitted by the malaria mosquito *Anopheles atroparvus*, a species that preferentially breeds in brackish water bodies and feeds predominantly on animals (zoophilic). This species is incompetent to transmit *P. falciparum*, causative agent of the more severe form of malaria. In the Netherlands, malaria was finally eradicated through intensive use of malaria drugs for children and transmission interruption using indoor DDT rather than the eradication of the anopheline vectors [77]. The increased farmers' use of insecticides against flies and use of fertilizers and phosphates in the environment, however, decreased the mosquito density. The concurrent modernization of husbandry decreased exposure to zoophilic mosquitoes and further interrupted transmission. Recently, the annual number of imported malaria has been declining, even with an increasing number of travellers visiting malaria endemic countries [81]. Projections for the UK [80] show that despite 8-14% increase of local transmission due to temperature change in 2050 does not form a serious threat for reemergence of malaria. During 2009-2011, the occurrence of multiple autochthonous vivax- malaria in Greece was predominantly caused by the recent steady introduction of non-symptomatic gametocyte immigrant workers infecting the local malaria mosquito population [82], rather than induced by climate change.

West Nile virus: In Europe, human West Nile fever cases have been reported from several countries since the 1960's, but it appears that the frequency has accelerated over the past 15 years [83,84]. West Nile virus (WNV) exists in an enzootic cycle between birds and mosquitoes, principally of the *Culex* species. Humans, horses and other mammals can also be infected with WNV, although they are dead-end hosts from which a vector cannot pick up the virus. In Europe, WNV has been isolated from mosquitoes, migrating birds, horses and humans [85]. Linking entomologic and data meteorological have been linked to data in order to model a West Nile fever (WNF) outbreaks in France [86], USA [87], Russia [88], Israel [89]. In general, relatively mild winters, dry spring and summers, heat waves early in the season and wet autumns promote WNF outbreaks [90]. In the USA, the emergence of WNF was mainly determined by bird migration routes [91] and mosquito ecology [92]. Although correlated with relatively warmer weather events [87], increased virus circulation and disease burden of WNF does not occur more frequently in the warmer southern states. In other words, ecological requirements in a location need to be met to sustain an enzootic virus transmission such as sufficient susceptible

bird population, and presence of efficient mosquito vectors. In addition, disease cases have shown to depend on socioeconomics; Mexican and American sides of the same Texan city were unequally hit by WNF [93]. For Europe, the origin of the reemergence of WNF is unclear and currently investigated [94-96].

Conclusions

Predicting the impact of climate change on public health has proven to be very difficult. In part this is due to the uncertainty in predicting the multifactorial local effects of global changes in climate [2]. But even when assuming a certain scenario as a fact, huge uncertainties due to variability and incomplete knowledge over its effect on health remain [97]. It has been found to be very difficult to quantify changes in water-transmitted infectious diseases or even to establish the direction of the changes, either negative or positive effects. Moreover, the assessment of the additional effect of climate change over other global changes on public health has been hampered by absent or low-resolution epidemiological data on current infectious disease burdens. Nevertheless, the demands for solutions, how to mitigate its effects and how to adapt human societies to changes are present and pressing. To this end, developments of detailed and specific projections are needed, instead of building a general theory of climate change and infectious disease around the one-tailed prediction that climate change will increase the problem of infectious diseases.

Acknowledgements

Study carried out by order and for the account of the Ministry of Health, Welfare and Sport. Project V/3300041/01/KW.

References

- Costello A, Abbas M, Allen A, Ball S, Bell S, et al. (2009) Managing the health effects of climate change: Lancet and University College London Institute for Global Health Commission. *Lancet* 373: 1693-1733.
- Hunter PR (2003) Climate change and waterborne and vector-borne disease. *J Appl Microbiol* 94: 37S-46S.
- Epstein PR (2001) Climate change and emerging infectious diseases. *Microbes Infect* 3: 747-754.
- Patz JA, Epstein PR, Burke TA, Balbus JM (1996) Global climate change and emerging infectious diseases. *JAMA* 275: 217-223.
- Reiter P (2008) Climate change and mosquito-borne disease: knowing the horse before hitching the cart. *Rev Sci Tech* 27: 383-398.
- Ostfeld RS (2009) Climate change and the distribution and intensity of infectious diseases. *Ecology* 90: 903-905.
- Lafferty KD (2009) Calling for an ecological approach to studying climate change and infectious diseases. *Ecology* 90: 932-933.
- Lafferty KD (2009) The ecology of climate change and infectious diseases. *Ecology* 90: 888-900.
- WHO (2011) Guidelines for drinking water quality, fourth edition, World Health Organization: Geneva.
- Bradley D, Evison LM, James A, Pescod MB, White AU, et al. (1977) Health aspects of water supplies in tropical countries. In *Water, wastes and health in hot climates*, John Wiley: London.
- WHO Water, Sanitation and Hygiene Links to Health FACTS AND FIGURES.
- Newell DG, Koopmans M, Verhoef L, Duizer E, Aidara-Kane A, et al. (2010) Food-borne diseases - the challenges of 20 years ago still persist while new ones continue to emerge. *Int J Food Microbiol* 139: S3-S15.
- WHO Cholera factsheet 107.
- West SK (2004) Trachoma: new assault on an ancient disease. *Prog Retin Eye Res* 23: 381-401.
- WHO Priority eye diseases.
- Vennervald BJ, Dunne DW (2004) Morbidity in schistosomiasis: an update. *Curr Opin Infect Dis* 17: 439-447.
- IPCC Climate Change (2007) Synthesis Report. Contribution of Working Groups I, II and III to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change Intergovernmental Panel on Climate Change: Geneva.
- Ramasamy R, Surendran SN (2011) Possible impact of rising sea levels on vector-borne infectious diseases. *BMC Infect Dis* 11: 18.
- Porcal P, Koprivnjak JF, Molot LA, Dillon PJ (2009) Humic substances-part 7: the biogeochemistry of dissolved organic carbon and its interactions with climate change. *Environ Sci Pollut Res Int* 16: 714-726.
- Schijven JF, Teunis PF, Rutjes SA, Bouwknegt M, de Roda Husman AM (2011) QMRAspot: a tool for Quantitative Microbial Risk Assessment from surface water to potable water. *Water Res* 45: 5564-5576.
- Schets FM, Schijven JF, de Roda Husman AM (2011) Exposure assessment for swimmers in bathing waters and swimming pools. *Water Res* 45: 2392-2400.
- Schijven J, De Roda Husman AM, (2006) A survey of diving behaviour and accidental water ingestion among Dutch occupational and sport divers to assess the risk of infection with waterborne pathogenic microorganisms. *Environ Health Perspect* 114: 712-717.
- Schijven JF, de Bruin HA, Hassanizadeh SM, de Roda Husman AM (2003) Bacteriophages and Clostridium spores as indicator organisms for removal of pathogens by passage through saturated dune sand. *Water Res* 37: 2186-2194.
- Green KY, Belliot G, Taylor JL, Valdesuso J, Lew JF, et al. (2002) A predominant role for Norwalk-like viruses as agents of epidemic gastroenteritis in Maryland nursing homes for the elderly. *J Infect Dis* 185: 133-146.
- Höhne M, Schreier E (2004) Detection and characterization of norovirus outbreaks in Germany: application of a one-tube RT-PCR using a fluorogenic real-time detection system. *J Med Virol* 72: 312-319.
- Lindell AT, Grillner L, Svensson L, Wirgart BZ (2005) Molecular epidemiology of norovirus infections in Stockholm, Sweden, during the years 2000 to 2003: association of the GGIIb genetic cluster with infection in children. *J Clin Microbiol* 43: 1086-1092.
- Lopman BA, Reacher MH, Vipond IB, Sarangi J, Brown DW (2004) Clinical manifestation of norovirus gastroenteritis in health care settings. *Clin Infect Dis* 39: 318-324.
- Maunula L, Von Bonsdorff CH (2005) Norovirus genotypes causing gastroenteritis outbreaks in Finland 1998-2002. *J Clin Virol* 34: 186-194.
- Reuter G, Krisztalovics K, Vennema H, Koopmans M, Szucs G (2005) Evidence of the etiological predominance of norovirus in gastroenteritis outbreaks--emerging new-variant and recombinant noroviruses in Hungary. *J Med Virol* 76: 598-607.
- McCarthy M, Estes MK, Hyams KC (2000) Norwalk-like virus infection in military forces: epidemic potential, sporadic disease, and the future direction of prevention and control efforts. *J Infect Dis* 181: S387-S391.
- Boccia D, Tozzi AE, Cotter B, Rizzo C, Russo T, et al. (2002) Waterborne outbreak of Norwalk-like virus gastroenteritis at a tourist resort, Italy. *Emerg Infect Dis* 8: 563-568.
- Häfliger D, Hübner P, Lüthy J (2000) Outbreak of viral gastroenteritis due to sewage-contaminated drinking water. *Int J Food Microbiol* 54: 123-126.
- Hoebe CJ, Vennema H, de Roda Husman AM, van Duynhoven YT (2004) Norovirus outbreak among primary schoolchildren who had played in a recreational water fountain. *J Infect Dis* 189: 699-705.
- Nygård K, Vold L, Halvorsen E, Bringeland E, Røttingen JA, et al. (2004) Waterborne outbreak of gastroenteritis in a religious summer camp in Norway, 2002. *Epidemiol Infect* 132: 223-229.
- Duizer E, Schwab KJ, Neill FH, Atmar RL, Koopmans MP, et al. (2004) Laboratory efforts to cultivate noroviruses. *J Gen Virol* 85: 79-87.
- de Roda Husman AM, Lodder WJ, Rutjes SA, Schijven JF, Teunis PF (2009) Long-term inactivation study of three enteroviruses in artificial surface and groundwaters, using PCR and cell culture. *Appl Environ Microbiol* 75: 1050-1057.
- Schijven JF, de Roda Husman AM (2005) Effect of climate changes on waterborne disease in The Netherlands. *Water Sci Technol* 51: 79-87.

38. Westrell T, Teunis P, van den Berg H, Lodder W, Ketelaars H, et al. (2006) Short- and long-term variations of norovirus concentrations in the Meuse river during a 2-year study period. *Water Res* 40: 2613-2620.
39. Thompson FL, Gevers D, Thompson CC, Dawyndt P, Naser S, et al. (2005) Phylogeny and molecular identification of vibrios on the basis of multilocus sequence analysis. *Appl Environ Microbiol* 71: 5107-5115
40. Morris JG Jr (2003) Cholera and other types of vibriosis: a story of human pandemics and oysters on the half shell. *Clin Infect Dis* 37: 272-280.
41. Oliver JD, Kaper JB, (1997) *Vibrio* species. In *Food Microbiology - Fundamentals and Frontiers*. ASM Press: Washington, DC.
42. Schets FM, van den Berg HHJL, Demeulemeester AA, van Dijk E, Rutjes SA, et al (2008) *Vibrio alginolyticus*-infecties na zwemmen in de Oosterschelde. *Nederlands Tijdschrift voor Medische Microbiologie* 16: 26-28.
43. Schets FM, van den Berg HH, Demeulemeester AA, van Dijk E, Rutjes SA, et al. (2006) *Vibrio alginolyticus* infections in the Netherlands after swimming in the North Sea. *Euro Surveill* 11: E061109.
44. Schets FM, van den Berg HH, Marchese A, Garbom S, de Roda Husman AM, (2011) Potentially human pathogenic vibrios in marine and fresh bathing waters related to environmental conditions and disease outcome. *Int J Hyg Environ Health* 214: 399-406.
45. Colwell RR, Patz JA (1998) *Climate, infectious disease and human health: an interdisciplinary perspective.*; American Academy of Microbiology: Washington, USA.
46. de Magny GC, Thiaw W, Kumar V, Manga NM, Diop BM, et al. (2012) Cholera outbreak in Senegal in 2005: was climate a factor? *PLoS One* 7: e44577.
47. Mena KD, Gerba CP (2009) Waterborne adenovirus. *Rev Environ Contam Toxicol* 198: 133-167.
48. Schilham MW, Claas EC, van Zaane W, Heemskerk B, Vossen JM, et al. (2002) High levels of adenovirus DNA in serum correlate with fatal outcome of adenovirus infection in children after allogeneic stem-cell transplantation. *Clin Infect Dis* 35: 526-532.
49. RIVM-Cib (2010) Meldingen uit de virologische laboratoria. *Infectieziektenbulletin* 21.
50. Rutjes SA, De Roda Husman AM, (2007) Optimalisatie van virusdetectie ten behoeve van het Nederlandse Waterleidingbesluit, RIVM Rapport 703719018; National Institute for Public Health and the Environment, Bilthoven.
51. Hoadley AW, Ajello G, Masterson N (1975) Preliminary studies of fluorescent pseudomonads capable of growth at 41 C in swimming pool waters. *Appl Microbiol* 29: 527-531.
52. WHO (2006) *Swimming pools and similar environments* World Health Organization, Geneva.
53. Mena KD, Gerba CP (2009) Risk assessment of *Pseudomonas aeruginosa* in water. *Rev Environ Contam Toxicol* 201: 71-115.
54. Schets FM, van den Berg HHJL, Lodder WJ, Docters van Leeuwen AE, De Roda Husman AM, (2006) Pathogene micro-organismen in zwemwater in relatie tot indicatoren voor fecale verontreiniging, RIVM Rapport 330400001 National Institute for Public Health and the Environment, Bilthoven.
55. Cort WW (1928) Schistosome dermatitis in the United States (Michigan). *JAMA* 90: 1027-1029.
56. Horák P, Kolárová L, Adema CM (2002) Biology of the schistosome genus *Trichobilharzia*. *Adv Parasitol* 52: 155-233.
57. Schets FM, Lodder WJ, van Duynhoven YT, de Roda Husman AM (2008) Cercarial dermatitis in the Netherlands caused by *Trichobilharzia* spp. *J Water Health* 6: 187-195.
58. Schets FM, Lodder WJ, de Roda Husman AM (2010) Confirmation of the presence of *Trichobilharzia* by examination of water samples and snails following reports of cases of cercarial dermatitis. *Parasitology* 137: 77-83.
59. Mas-Coma S, Valero MA, Bargues MD (2009) Climate change effects on trematodiasis, with emphasis on zoonotic fascioliasis and schistosomiasis. *Vet Parasitol* 163: 264-280.
60. Chorus I, Falconer IR, Salas HJ, Bartram J (2000) Health risks caused by freshwater cyanobacteria in recreational waters. *J Toxicol Environ Health B Crit Rev* 3: 323-347.
61. DG Water (2010) *Blauwalgen in Nederland- Landelijk beeld 2005-2009*.
62. Stewart I, Webb PM, Schluter PJ, Shaw GR (2006) Recreational and occupational field exposure to freshwater cyanobacteria--a review of anecdotal and case reports, epidemiological studies and the challenges for epidemiologic assessment. *Environ Health* 5: 6.
63. Funari E, Testai E (2008) Human health risk assessment related to cyanotoxins exposure. *Crit Rev Toxicol* 38: 97-125.
64. Paerl HW, Huisman J (2008) Climate. Blooms like it hot. *Science* 320: 57-58.
65. Roijackers RMM, Lurling M, (2007) *Climate Change and Bathing Water Quality*. Wageningen UR, Wageningen.
66. Peperzak L (2005) Future increase in harmful algal blooms in the North Sea due to climate change. *Water Sci Technol* 51: 31-36.
67. Blackmon JA, Chandler FW, Cherry WB, England AC 3rd, Feeley JC, et al. (1981) Legionellosis. *Am J Pathol* 103: 429-465.
68. WHO (2007) *Legionella and the prevention of legionellosis*. World Health Organization: Geneva.
69. Versteegh JFM, Brandsema PS, van der Aa NGFM, Dik HHJ, de Groot GM, (2007) Evaluatie legionella preventie Waterleidingwet. National Institute for Public Health and the Environment: Bilthoven.
70. Karagiannis I, Brandsema P, VAN DER Sande M (2009) Warm, wet weather associated with increased Legionnaires' disease incidence in The Netherlands. *Epidemiol Infect* 137: 181-187.
71. Gubler DJ, Reiter P, Ebi KL, Yap W, Nasci R, et al. (2001) Climate variability and change in the United States: potential impacts on vector- and rodent-borne diseases. *Environ Health Perspect* 109: 223-233.
72. Paaajmans KP, Blanford S, Chan BH, Thomas MB (2012) Warmer temperatures reduce the vectorial capacity of malaria mosquitoes. *Biol Lett* 8: 465-468.
73. Medlock J, Jameson LJ (2010) Ecological approaches to informing public health policy and risk assessments on emerging vector-borne zoonoses. *Emerg Health Threats J* 3: e1.
74. Weaver SC (2005) Host range, amplification and arboviral disease emergence. *Arch Virol Suppl* 33-44.
75. Takken W, Kager PA, van der Kaay HJ (1999) [A return of endemic malaria to the Netherlands is highly unlikely]. *Ned Tijdschr Geneesk* 143: 836-838.
76. Rogers DJ, Randolph SE (2000) The global spread of malaria in a future, warmer world. *Science* 289: 1763-1766.
77. Verhave JP (2000) The disappearance of Dutch malaria and the Rockefeller Foundation. *Parassitologia* 42: 111-115.
78. Gething PW, Smith DL, Patil AP, Tatem AJ, Snow RW, et al. (2010) Climate change and the global malaria recession. *Nature* 465: 342-345.
79. Lindsay SW, Hole DG, Hutchinson RA, Richards SA, Willis SG (2010) Assessing the future threat from vivax malaria in the United Kingdom using two markedly different modelling approaches. *Malar J* 9: 70.
80. Kuhn KG, Campbell-Lendrum DH, Armstrong B, Davies CR (2003) Malaria in Britain: past, present, and future. *Proc Natl Acad Sci U S A* 100: 9997-10001.
81. van Rijkvorsel GG, Sonder GJ, Geskus RB, Wetsteyn JC, Ligthelm RJ, et al. (2010) Declining incidence of imported malaria in the Netherlands, 2000-2007. *Malar J* 9: 300.
82. Danis K, Baka A, Lenglet A, Van Bortel W, Terzaki I, et al. (2011) Autochthonous *Plasmodium vivax* malaria in Greece, 2011. *Euro Surveill* 16.
83. Calistri P, Giovannini A, Hubalek Z, Ionescu A, Monaco F, et al. (2010) Epidemiology of west nile in europe and in the mediterranean basin. *Open Virol J* 4: 29-37.
84. Danis K, Papa A, Papanikolaou E, Dougas G, Terzaki I, et al. (2011) Ongoing outbreak of West Nile virus infection in humans, Greece, July to August 2011. *Euro Surveill* 16.
85. Hubálek Z, Halouzka J (1999) West Nile fever--a reemerging mosquito-borne viral disease in Europe. *Emerg Infect Dis* 5: 643-650.
86. Ludwig A, Bicout DJ, Chalvet-Monfray K, Sabatier P, (2005) Modélisation de l'agressivité de *Culex modestus*, vecteur potentiel de West-Nile en Camargue, en fonction de données météorologiques. *Environnement, Risques et Santé* 4: 109-113.

87. Reisen WK, Fang Y, Martinez VM (2006) Effects of temperature on the transmission of west nile virus by *Culex tarsalis* (Diptera: Culicidae). *J Med Entomol* 43: 309-317.
88. Platonov AE, Fedorova MV, Karan LS, Shopenskaya TA, Platonova OV, et al. (2008) Epidemiology of West Nile infection in Volgograd, Russia, in relation to climate change and mosquito (Diptera: Culicidae) bionomics. *Parasitol Res* 103: S45-S53.
89. Paz S (2006) The West Nile Virus outbreak in Israel (2000) from a new perspective: the regional impact of climate change. *Int J Environ Health Res* 16: 1-13.
90. Semenza JC, Menne B (2009) Climate change and infectious diseases in Europe. *Lancet Infect Dis* 9: 365-375.
91. Dusek RJ, McLean RG, Kramer LD, Ubico SR, Dupuis AP 2nd, et al. (2009) Prevalence of West Nile virus in migratory birds during spring and fall migration. *Am J Trop Med Hyg* 81: 1151-1158.
92. Bowden SE, Magori K, Drake JM (2011) Regional differences in the association between land cover and West Nile virus disease incidence in humans in the United States. *Am J Trop Med Hyg* 84: 234-238.
93. Reiter P, Lathrop S, Bunning M, Biggerstaff B, Singer D, et al. (2003) Texas lifestyle limits transmission of dengue virus. *Emerg Infect Dis* 9: 86-89.
94. Reiter P, (2010) West Nile virus in Europe: understanding the present to gauge the future. *Euro Surveill* 15: 19508.
95. Lelli R, (2010) West Nile virus in Europe: understanding the present to gauge the future. *Euro Surveill* 15: 19538.
96. Monaco F, Lelli R, Teodori L, Pinoni C, Di Gennaro A, et al. (2010) Re-emergence of West Nile virus in Italy. *Zoonoses Public Health* 57: 476-486.
97. Randolph SE (2009) Perspectives on climate change impacts on infectious diseases. *Ecology* 90: 927-931.

Citation: Braks MAH, de Roda Husman AM (2013) Dimensions of Effects of Climate Change on Water-Transmitted Infectious Diseases. *Air Water Borne Diseases* 2: 109. doi:[10.4172/2167-7719.1000109](https://doi.org/10.4172/2167-7719.1000109)

Submit your next manuscript and get advantages of OMICS Group submissions

Unique features:

- User friendly/feasible website-translation of your paper to 50 world's leading languages
- Audio Version of published paper
- Digital articles to share and explore

Special features:

- 250 Open Access Journals
- 20,000 editorial team
- 21 days rapid review process
- Quality and quick editorial, review and publication processing
- Indexing at PubMed (partial), Scopus, EBSCO, Index Copernicus and Google Scholar etc
- Sharing Option: Social Networking Enabled
- Authors, Reviewers and Editors rewarded with online Scientific Credits
- Better discount for your subsequent articles

Submit your manuscript at: <http://www.omicsonline.org/submission>