Chapter 5
Global Warming and Trans-Boundary Movement of Waterborne Microbial Pathogens

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5.1 Introduction

Potential ramifications of climate change, as they relate to waterborne pathogens (primarily viruses, bacterial and parasitic protozoa), are the focus of this chapter. It seems clear that climate change will impact on waterborne pathogens in various ways (Rose et al. 2001), pertinent to transboundary issues are: (1) increases in intense storm events (increasing sewage/animal waste flows into waterways/aquifers) (Charron et al. 2004; Schijven and de Roda Husman 2005; Yang and Goodrich 2009; De Toffol et al. 2009; Richardson et al. 2009); (2) warmer surface water temperatures or salinity changes (for increased autochthonous pathogen growth) (Niemi et al. 2004; Koelle et al. 2005; Lebarbenchon et al. 2008); and (3) changes in food production, as most obvious in animal diseases (Lightner et al. 1997; Rapoport and Shimshony 1997), but also of concern with zoonoses and from changes in social behavior (Schwab et al. 1998; Nancarrow et al. 2008; CDC 2009a). When considering trans-boundary effects on waterborne pathogens, it is therefore the flow of pathogens in surface water (fresh and marine) and in groundwater, as well as in the varying ways water is used/reused in association with human activities (e.g., food production) that are the trans-boundary issues discussed in this chapter (examples in Table 5.1). Changes in infectious and vector-borne diseases associated with rising sea levels, losses of habitat, international travel etc. are not addressed in this chapter.

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Table 5.1 Examples of pathogen effects associated with climate change

Pathogen group	Agent	Food	Water	Indirect effects	Direct effects
Enteric viruses	Hepatitis A Enterovirus Norovirus	Shellfish	Ground water	Storms can increase transport from fecal sources	Survival increases with reduced temperature and sunlight
Bacteria Cyanobacteria Dinoflagellates	*Vibrio vulnificus*, *V. parahaemolyticus, V. cholerae* non-01	Shellfish	Recreation Wound infections	Enhanced zooplankton blooms	Salinity and temperature related growth
Parasitic protozoa	*Cyclospora* *Cryptosporidium* *Giardia*	Fruits and vegetables	Recreational and drinking water	Storms can increase transport from fecal sources	Temperature associated maturation of *Cyclospora*

Adapted from Rose et al. (2001)

5.1.1 Areas of Potential Impact by Climate Change

One of the largest effects of climate change is likely to be reflected in changes in water resource use, which will need to account for water's equally important roles in electricity production/greenhouse gas production (King and Webber 2008) and ecological service provision (Corvalan et al. 2005; Keath and Brown 2009) so as to provide more sustainable water services into the future. A likely consequence of these changed services is an increase in the use of water fit-for-purpose. For example, where there is municipal water supply, not treating all to drinking water quality, give that less than 10% is required in the home for that purpose (Rathjen et al. 2003). Rather, for other urban and irrigation needs, there will be increased use of recycled wastewater streams for toilet flushing, garden/crop irrigation and cloths washing, so reducing the demand on traditional urban water resources (possibly up to 70%) and keeping environmental water to support ecological services and reduce trans-boundary effects of water pollution.

At a regional scale and in developed regions, climate change is already having a profound impact on water decisions within Australia (WSAA 2008), and is expected to have major impacts in many other regions. For example, Californian water resources are projected to significantly change with respect to snowpack, river flows, and sea levels. By 2050, it is predicted that the Sierra snowpack will decline by 25%, which is an important source of urban, agricultural, and environmental water (California Department of Water Resources 2009). More variable weather patterns may also result in increased dryness in the southern regions of California. The sea level has risen about 7 inches at the Golden Gate Bridge in the last century, and continued sea level rise could threaten many coastal communities, as well as the sustainability of the Sacramento-San Joaquin Delta that supplies 25 million Californians with drinking water. As a consequence, water reuse has to increase, most likely via a

second non-potable supply pipe to homes (Okum 2002) and/or through wastewater irrigation (direct or via aquifer storage and recovery) (Kracman et al. 2001; Dillon et al. 2009), which will open up new ecological niches for waterborne pathogens.

An example of the possible effects of climate change on waterborne pathogens in developing regions can be seen in the increases in diarrheal disease during El Niño periods in Peru. For each 1°C increase in temperature, hospital admission increased by 8% (95% CI 7–9%), with an additional 6,225 cases of diarrheal disease recorded (Checkley et al. 2000). In Fiji, diarrheal disease appears to increase by 3% (95% CI 1.2–5.0%) per 1°C temperature increase, noting also that a significant increase in diarrhea rates occurred if rainfall was either higher or lower than average conditions (Singh et al. 2001). Overall in developing regions, water, sanitation and hygiene-related disease currently account for some 5.5% of total disability adjusted life years (DALYs) lost (Prüss-Üstün et al. 2008). Changes in diarrheal disease has been the main metric used in WHO reports on possible climate change impacts related to waterborne disease (McMichael et al. 2004; Campbell-Lendrum and Woodruff 2006). What is not clear from most reports, however, are the trans-boundary effects, let alone the raft of other diseases unrelated to diarrhea (e.g., see Table 5.2 and

Table 5.2 Major potential pathogens/indicators in aquatic environments

Group of organism	Source(s)	Symptom(s)	Survival
Viruses			$T_{99.9}$
Adenovirus	Human feces	C Co F G H R	50 d
Astrovirus	Human feces	G	Unknown
Calicivirus (inc. Norovirus)	Human feces	G	Weeks–months
Coronavirus	Human feces	G	Unknown
Coxsackie A and B	Human feces	B C D E-M F H R S	2 d–46 wk
Echovirus	Human feces	C E-M F G R P.S	2 d–46 wk
Hepatitis A	Human feces	H	25 d at 25°C–677 d at 4°C
Hepatitis E	Pig/human feces[1]	H A	Unknown
Poliovirus	Human feces	C F E-M P R	2–130 d
Reovirus	Human feces	None known	>4 d
Rotavirus	An./human feces[1]	G	2–34 d
Bacteria			T_{90}
Aeromonas spp.	An./human feces	G S W	"Indigenous"
Campylobacter jejuni	An./human feces	G-F	Poor
Toxigenic *E. coli*	An./human feces	G, kidney failure	5 h–2 d
Thermotolerant coliforms/*E. coli*	An./human feces	Fecal indicator	2 h–2 d
Fecal streptococci	An./human feces	Fecal indicator	2 h–12 d
Legionella spp.	Biofilms/amoebae	R	"Indigenous"
Mycobacterium avium complex	Freshwater/ biofilms	R, weight loss	"Indigenous"
Mycobacterium marinum	Sea water	S W Granuloma	"Indigenous"

(continued)

Table 5.2 (continued)

Group of organism	Source(s)	Symptom(s)	Survival
Salmonella spp.	An./human feces	G-F	12 h–5 d
Shigella spp.	An./human feces	Bloody diarrhea	<15 to >70 d
Tropheryma whipplei	Human feces	G,	Unknown
Vibrio spp.	Seawater, feces	G W	"Indigenous"/<6d
Yersinia enterocolitica	An./human feces	Appendicitis-like G	Days–weeks
Protozoa			
Cryptosporidium parvum/C. hominis	An./human feces	Watery diarrhea F	Months
Entamoeba histolytica	Feces	G/dysentery	Unknown
Giardia intestinalis	An./human feces	G/bloating	Weeks
Helminths[2]			
Ascaris spp.	An./human feces	Roundworm	Weeks–months
Taenia spp.	An./human feces	Tapeworm	Weeks
Dinoflagellates			
Alexandrium spp.	Ballast/sea water	PSP	"Indigenous"
Gambierdiscus toxicus	Sea water	Ciguatera shellfish poisoning	"Indigenous"
Gymnodinium spp.	Ballast/sea water	PSP	"Indigenous"
Pfiesteria piscicida	Sea water	Fish kills and human illness	"Indigenous"

Adapted from McNeill (1985), Evison (1988), Hallegraeff (1992), Chung and Sobsey (1993), Gantzer et al. (1998), Fenollar et al. (2009), Lathrop et al. (2009)

[1]Enteric viruses from humans cause most waterborne viral infections (i.e., animal viruses from the same group/family do not infect humans and visa versa, with possible exceptions of porcine hepatitis E and bovine Rotavirus and Norovirus)

[2]Helminths are largely an issue for direct contact with fecal matter/fecally-contaminated foods and typically less important as waterborne pathogens

An. animal source, largely mammals/birds that may yield human-infectious strains

A abortion; *C* carditis; *Co* conjunctivitis, *F* fever; *D* diabetes; *E-M* encephalitis-meningitis; *G* gastroenteritis; *G-F*gastro + fever; *H* hepatitis; *P* paralysis; *PSP* paralytic shellfish poisoning; *R* respiratory infection; *S* skin infection; *W* wound infection

T_{90} or $T_{99.9}$ times for 1 or 3 $\log_{10}$ reduction in numbers respectively at 10–25°C

Niklasson et al. 1998; Blinkova et al. 2009). For example, given the increase in aquaculture produce from developing regions, what may be the impact on countries that purchase these products for increased diarrhea and other disease endpoints?

Globally, some 70% of environmental water withdrawals are used in agriculture (Millennium Assessment Board 2005). The need to reduce the total demand but feed the world is probably the biggest global water issue, and the Israelis are leading the world in demonstrating one solution via drip irrigation of treated municipal wastewaters (Oron et al. 2001). Given the globalization of food products, however, numerous disease outbreaks have been recorded for other situations when crops eaten raw are spray irrigated with poorly treated water (Rose et al. 2001; Jay et al. 2007; CDC 2009b). The latter is of particular concern with zoonotic pathogens in

surface water (Bharti et al. 2003; Fayer 2004; Bednarska et al. 2007; Mattison et al. 2007; Moulin-Schouleur et al. 2007; Zell et al. 2008; Banyai et al. 2009; Robertson 2009; Rutjes et al. 2009), which includes viruses, bacteria and parasitic protozoa (Table 5.2).

5.1.2 Pathogen Dynamics and Problematic Identifications

Before going into details on the range of waterborne and water-based pathogens of concern, two points are important to note. Firstly, pathogens are dynamic in their ability to evolve and change in their potential to be human pathogens, as exemplified by seasonal changes in flu viruses. Secondly, as we use better methods (largely molecular-based) difficult to culture or non-culturable pathogens are being identified which were previously missed. Hence, it would be fair to say that there are many pathogens yet to be identified today (Rosario et al. 2009; Victoria et al. 2009), let alone what may evolve tomorrow, in part reflecting new environmental conditions.

As an example of the difficulty in describing human pathogens one can look at members of the important waterborne parasitic protozoan genus *Cryptosporidium*. Most human illness is thought to be due to *C. hominis* and *C. parvum* (cattle genotype), yet several other *Cryptosporidium* species or genotypes: *C. meleagridis*; *C. felis*; *C. canis*; *C. suis*; *C. muris*; *C. andersoni*; *C. hominis* monkey genotype; *C. parvum* (mouse genotype); *C. parvum* (pig genotype II) and *Cryptosporidium* rabbit genotype have caused human illness (Kváč et al. 2009). So how to target the right species? In a similar way but at the strain level within a species, *Escherichia coli* O157:H7 has been the focus of method development and study, due to numerous water- and food-borne outbreaks. Yet focusing on O157:H7 strains appears to be at the detriment of missing the even more important non-O157 shiga toxin-producing *E. coli* (Bettelheim 2007; Lathrop et al. 2009). The situation is further complicated in *E. coli*, which is probably better described as pangenomic (i.e. not a single isolated species, but one that shares many genes amongst a broader range of related members) that includes the six known pathovars, each of which may have separately inherited particular virulence factors (Rasko et al. 2008).

5.2 The Waterborne Pathogens

Waterborne pathogens are defined as disease-causing organisms excreted in feces/urine and ingested/inhaled with water. They are often referred to as being transmitted via the fecal-oral route (Ashbolt et al. 2001). All of these pathogens can persist to varying degrees in the aquatic environment, but rarely grow outside the host organism(s) they come from. Hence they are introduced and pass through the water environment as allochthonous members. Most waterborne pathogens that infect humans come from human excreta, other mammals or birds (Table 5.2).

In contrast to the fecally-borne pathogens, there are a number of water-based pathogens generally unrelated to fecal contamination, but loosely referred to as waterborne. Examples of these autochthonous or indigenous pathogens are various *Legionella*, *Mycobacterium* and *Helicobacter* spp. native to freshwaters, and *Vibrio vulnificus*, *V. parahaemolyticus* and *V. cholerae* in saline waters. The bacterium that causes cholera (*V. cholerae*) is a good example of a species with members that are transmitted by the fecal-oral route, but that also have a natural life-cycle associated with marine zooplankton (Blokesch and Schoolnik 2007). It also seems that *Legionella pneumophila* serogroup 1, and similar respiratory pathogens, are accidental human pathogens, with various amoebae possibly acting as their main environmental host (Lau and Ashbolt 2009). Unfortunately our lung macrophages are very similar host cells to amoebae, and these *Legionella*-like intracellular pathogens can also parasitize our lung macrophages (Thomas et al. 2008).

5.3 Changing Habitats

In diverse regions around the world, enteric (gastrointestinal) diseases show evidence of significant seasonal fluctuations, e.g.,

- In Scotland and Sweden, *Campylobacter* infections are characterized by short peaks in the spring (associated with snowmelt periods)
- In Bangladesh, cholera outbreaks occur during the monsoon season
- In Peru, *Cyclospora* infections peak in the summer and subside in the winter

Therefore, further extension of "seasonal" effects under climate change is likely to yield further peaks in waterborne diseases. Climatic-related peaks are also common with various autochthonous (indigenous) pathogens, such as marine *Vibrio vulnificus*. Highest concentrations of *V. vulnificus* and increases in shellfish-borne human disease have been recorded in Florida following heavy rainfall associated with El Niño events (Lipp et al. 2001). It appears that reduced salinity due to increased freshwater inputs rather than temperature is the key factor increasing the competitive advantage of *V. vulnificus*.

5.3.1 Which Water-Based (Autochthonous) Microbes Are Pathogens?

A common feature of the autochthonous (and allochthonous) bacterial pathogens, is that not all strains of pathogenic species are effective human pathogens. For example, in a three-year study of environmental and clinical *Vibrio vulnificus* isolates, the more important biotype 3 sub-species represented about 21% of the aquaculture pond isolates versus 86% of clinical cases (Broza et al. 2009). Huge quantities of aquacultural produce are now exported around the world for direct human consumption as well as animal feedstock – their global significance to disease is largely unknown. Indeed, it is often unclear where foodstock or feeds have come from.

However, what is clear is the uptake and release of ship ballast waters being responsible for the reintroduction of cholera into South America in the early 1990s (McCarthy and Khambaty 1994; WHO 2003), and ballast waters in general continue to be a problem for introductions of various toxic dinoflagellates (algae), cyanobacteria and *V. parahaemolyticus* (Myers et al. 2003; Tang and Dobbs 2007).

Identifying what are important biotypes is also at the heart of the issue with trying to determine the clinical significance of the common occurrence of *Mycobacterium avium* complex mycobacteria (Falkinham III 2009) and *Helicobacter pylori* (stomach ulcer and cancer bacterium) (Kawaguchi et al. 2009). Water appears to be a likely vehicle for the exposure of people to these pathogens, but it is currently unclear if water is the primary source or much less important. Interestingly, various mycobacteria seem to be selected in chlorinated waters, possibly due to their relatively slow growth rates and biofilm-forming habitat.

5.3.2 Transfer of Virulence and Antibiotic Resistance Genes

5.3.2.1 *V. cholerae* as a Model

Understanding the ecology of *V. cholerae* ; i.e., its ability to uptake virulence factors from bacteriophages (viruses to bacteria), growth in association with marine plankton and how it is impacted by climatic conditions, has served as a good model for trans-boundary waterborne pathogens and possible climate change impacts (Lipp et al. 2002). The marine life-cycle of *V. cholerae* is now well established and illustrated in Fig. 5.1.

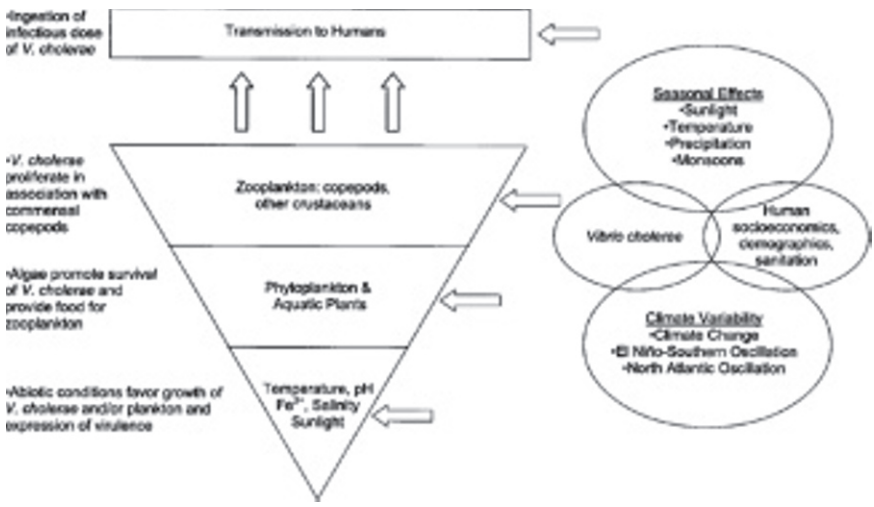

Fig. 5.1 Hierarchical model for environmental cholera transmission. From Lipp et al. (2002) with permission from the publishers

The cholera toxin (CT), which is responsible for the classic symptom of profuse diarrhea, is encoded by a lysogenic bacteriophage designated CTX Phi (includes six toxin genes which also occur on a plasmid) (Faruque et al. 1998). *V. cholerae*, requires two coordinately regulated factors for full virulence, cholera toxin (CT) and toxin-coregulated pill (TCP, surface organelles required for intestinal colonization and the site for phage attachment). Hence, the emergence of toxigenic *V. cholerae* involves horizontal gene transfer, in vivo gene expression and follows phage seasonality. In marine waters *V. cholerae* becomes resistant to the phage, yet in the intestine it remains susceptible and hence, maintains its virulence (Zahid et al. 2008). Also, *V. cholerae* is commensal to phytoplankton and their consumers zooplankton, notably copepods, as such it is also a vector-borne disease. Interestingly, growth of *V. cholerae* on the chitinous exoskeletons of copepods molts induces competence for natural transformation, a mechanism for intra-species gene exchange (Blokesch and Schoolnik 2007).

A further point of some controversy is that toxigenic *V. cholerae* are rarely isolated from the aquatic environment between cholera epidemics, due to their presumed presence in a dormant stage, i.e., active but nonculturable (ABNC) form (Colwell et al. 1996). Nonetheless, the aquatic biofilms rather than surrounding seawater, have proved to be a source of culturable *V. cholerae*, even in non-epidemic periods in Bangladesh (Zahid et al. 2008).

5.3.2.2 Integrons and Antibiotic Resistance

The last example of trans-boundary pathogen concern provided in this chapter relates to the insidious perfusion of antibiotic resistant genes in the environment. Most *β-Proteobacteria* (members of Gram-negative bacteria that includes many pathogens and non-pathogens) contain integrons. Class 1 integrons are central players in the worldwide problem of antibiotic resistance, because they can capture and express diverse resistance genes. In addition, they are often embedded in promiscuous plasmids and transposons, facilitating their lateral transfer into a wide range of pathogens and environmental bacteria (Gillings et al. 2008).

Hence, Gillings et al. (2008) have promoted the need to understand the origin of integrons as important for the practical control of antibiotic resistance and for exploring how lateral gene transfer can seriously impact on, and be impacted by, human activities. They have shown that class 1 integrons are common in nonpathogenic soil and freshwater *β-Proteobacteria* in the absence of antibiotic resistance genes, yet are almost identical to the core of the class 1 integrons now found in pathogens, suggesting that environmental *β-Proteobacteria* were the original source of these genetic elements. Because these elements appear to be readily mobilized, their lateral transfer into human commensals and pathogens was inevitable, especially given their intersect with the human food chain. The strong selection pressure imposed by the human use of antimicrobial compounds then ensured their fixation and global spread into new species (Hardwick et al. 2008; Gillings et al. 2009). Hence, changing food production practices influenced by population

growth, water resources and climate change will further impact on our loss of efficacy in antibiotics. Newer treatment systems for wastewater are also not completely effective at removing resistance genes (Bockelmann et al. 2009) and intensive animal facilities are a likely hotspot for exchange of antibiotic resistance genes (Kozak et al. 2009; Toomey et al. 2009).

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