LAND ECONOMY WORKING PAPER SERIES

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Number: 63   **Review of the Evidence Linking Climate Change to Human Health for Eight Diseases of Tropical Importance**

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REVIEW OF THE EVIDENCE LINKING CLIMATE CHANGE TO HUMAN HEALTH FOR EIGHT DISEASES OF TROPICAL IMPORTANCE

Corinne Baulcomb

ABSTRACT

As human societies are beginning to feel the early effects of 21st Century climate change, adaptation is becoming an increasingly important area of enquiry across a range of human sectors and activities. This is particularly true for the health sectors of tropical developing countries, as many of these countries will be some of the first to experience the impacts of global warming. Given this, it is important to understand the mechanisms through which climate change may impact on human health, and thus on the social welfare in tropical developing countries and the resourcing requirements of their health sectors. This paper reviews and synthesizes the published literature on the causal links between climate change and human disease for eight diseases of tropical importance: malaria, dengue fever, gastroenteritis, schistosomiasis, leptospirosis, ciguatera poisoning, meningococcal meningitis, and cardio-respiratory disease.

1. INTRODUCTION

Anthropogenic, greenhouse gas-driven climate change is now nearly universally accepted as a global phenomena that will increase global mean temperatures, cause sea levels to rise, and likely change both the frequency and intensity of major storms around the world. The longer it takes to stabilize emission levels, the more likely it is that parts of the planet will suffer irreversible environmental change as a consequence of these emissions. Similarly, while there is strong evidence that human societies are already beginning to feel the effects of climate change, the longer it takes to stabilize emission levels, the more likely those effects are to be extreme (IPCC, 2007).

Climate change has the potential to affect disease and mortality incidence both directly and indirectly across a wide range of conditions the world over. Direct effects are those in which human health is decreased due to natural disasters and extreme events, where as the indirect effects of climate change on human health stem from the alteration by climate change of the complex socio-economic-environmental systems that govern disease transmission (McMichael, et al., 2004). Understanding the nature of these mechanisms through which climate change can impact on human health is therefore vital to adaptation efforts, both at the institutional and individual/household levels.

This paper reviews and synthesizes a significant part of the published literature that focuses on unraveling these causal mechanisms linking climate change and human health. The review was conducted in two phases[1]. During the first phase, major World Health Organization (WHO) publications on the subject were consulted. During the second phase of the review, systematic literature searches were conducted in order to locate papers that specifically focused on deriving, modeling, or attempting to explain dose-response relationships between climatic variables (such as temperature and precipitation), and the following diseases of tropical importance: malaria, dengue fever, gastroenteritis, schistosomiasis, leptospirosis, ciguatera poisoning, meningococcal meningitis, and cardio-respiratory disease.

The results of this review are presented below. Section 2 provides an overview of the climate-health link for vector-borne diseases, and focuses specifically on malaria and dengue fever. Section 3 provides an overview of the climate-health link for water and food-borne diseases, and focuses specifically on gastroenteritis, schistosomiasis, leptospirosis, and ciguatera poisoning. Section 4 focuses solely on meningococcal meningitis, and section 5 focuses on cardio-respiratory disease. Section 6 provides an overall synthesis of the analyzed literature. It is envisaged that this paper would be a useful starting point for those needing to familiarize themselves with the current literature linking climate change to human health.

2. VECTOR-BORNE DISEASE

2.1 INTRODUCTION
Vector-borne diseases are those diseases that are caused by a pathogen transmitted to humans primarily via biting arthropods such as mosquitoes, flies, fleas, and ticks (Ebi, et al., 2008; Kovats, et al., 2003d). At a global scale, prominent vector-borne diseases that are climate-sensitive include: malaria, filariasis, dengue fever, yellow fever, west Nile virus, leishmaniasis, Chagas' disease, Lyme disease, tick-borne encephalitis, plague, varieties of mosquito-borne encephalitis, ehrlichiosis, African trypanosomiasis, and onchocerciasis (Ebi, et al., 2008; Githeko, et al., 2000; Kovats, et al., 2003d; Kuhn, et al., 2005).

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[1] Please note the following: Because the case study countries for the larger project of which this literature review was a part were Caribbean countries, some Caribbean-specific searches were done for some of these diseases.

The core of the vector-borne disease transmission cycle has three components that are all inter-related: the vector, the host (i.e. humans), and the pathogen. Vectors can become infected from biting an infected host, and can then subsequently infect other hosts. This disease transmission cycle, and thus the incidence of vector-borne disease, does not exist in isolation, however. Rather, it is sensitive to environmental change, and therefore to climate change, because of the role that environmental variables – and temperature in particular - play in both the spatial distribution of vectors and in the life cycles of both the vectors and the pathogens. Arthropod-vectors are unable to thermo-regulate, and each vector's geographical and altitudinal distribution is consequently constrained by its species-specific physiological range of temperature tolerances (Githeko, et al., 2000). This means that the projected global temperature increases for the 21st Century have the potential to expand, at a global scale, the geographic ranges of vectors and their pathogens. Those human populations that are currently shielded from certain vector-borne diseases as a consequence of their being situated in areas with an average temperature towards the lower threshold that is suitable for vector survival will thus experience an increased risk of disease incidence as global temperatures increase (Githeko, et al., 2000; Martens, et al., 1995a; Martens, et al., 1997; McMichael, et al., 2004).

Within areas already conducive to vector survival, temperature increases which remain below the upper physiological threshold for any given vector or pathogen, will accelerate the development and maturation of both vectors and pathogens, as well as the metabolism rate of adult vectors (Githeko, et al., 2000; Patz, et al., 2003; Sutherst, 2004). This developmental acceleration can increase both the vector population size and the risk of humans coming into contact with an infectious vector, and thereby the risk of disease transmission (Martens, et al., 1997; Tabachnick, 2010). Importantly, this may also decrease the time it takes for vectors and pathogens to develop resistance to chemical control measures by increasing the number of generations that occur within any given period of time (Martens, et al., 1997).

Within these same areas, precipitation also affects the disease transmission cycle because it governs, at least to a certain extent, the availability and type of vegetation cover, as well as the availability, stability, and suitability of vector breeding sites in a given location throughout the year (Githeko, et al., 2000; Kovats, et al., 1999; McMichael, et al., 2004).

Despite the sensitivity of vectors and vector-borne pathogens to environmental variables, the disease transmission cycle and disease incidence is also highly sensitive to location-specific social, infrastructure, governance, land use, and economic conditions. These non-environmental variables can impact both directly and indirectly on vector behaviour as well as on vector and pathogen survival. For example, government-funded vector control or vaccination programs can significantly decrease the vector population, or human susceptibility to disease, respectively, thereby reducing disease incidence (Githeko, et al., 2000; Martens, et al., 1997; McMichael, et al., 2004).

Additionally, although local environments do impact on human societies, there are also important social actions/variables that affect the disease cycle through their impact on the environment. Good examples of this include irrigation and deforestation, both of which alter both local vegetation types/ground cover and the location and availability of local water resources. These alterations change vector behaviour, and may in the short-term trump the effects of background precipitation or temperature on vector population dynamics (Githeko, et al., 2000; Martens, et al., 1997; McMichael, et al., 2004). Importantly, non-environmental variables also often strongly affect the contact patterns

between humans and vectors, and the standing level of immunity in a human population to infection (Githeko, et al., 2000; Martens, et al., 1997; McMichael, et al., 2004). Finally, as Tabachnick (2010) discusses, both the environmental and the non-environmental variables which drive aspects of the disease transmission cycle will increasingly be affected and driven by climate change and climate variability, making the full system of drivers and impacts both relatively complicated and location and vector-specific (see figure 1).

Figure 1:   Conceptualization of the full system of variables that affect the vector-borne disease transmission cycle and disease incidence. Adapted from (Tabachnick, 2010)
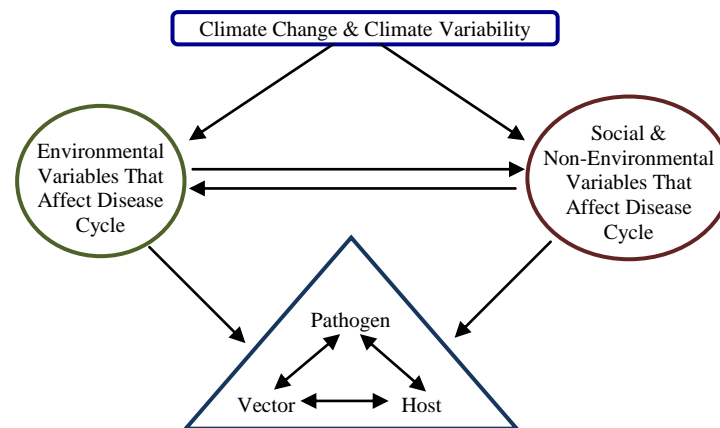
The only vector-borne diseases considered more fully here are malaria and dengue fever (DF)/dengue hemorrhagic fever (DHF), both of which are classified as sensitive to climate change, and occur in many tropical developing countries.

2.2   MALARIA

2.2.1 INTRODUCTION
Despite a significant contraction since 1870 in the geographic distribution of malaria, malaria is currently the most prevalent vector-borne disease in the world, and the world's most significant infectious disease (Baron, 2009; van Lieshout, et al., 2004). It is caused by one of four species of protozoa that can be transferred to humans by any of the 70 varieties of *Anopheles* mosquitoes. Two of the four species of protozoa – *Plasmodium vivax* and *Plasmodium falciparum* – are responsible for more than 90% of the world's malaria cases with *P. vivax* occurring over the greatest geographic range, and *P. falciparum* causing the most severe illness (Kovats, et al., 2003d; Martens, et al., 1995b; Martens, et al., 1997; van Lieshout, et al., 2004). In endemic areas, children, the elderly, and immune-compromised individuals tend to be most vulnerable because of the lack of an immune system response to the protozoa (Abellana, et al., 2008; Craig, et al., 2004a; van Lieshout, et al., 2004).

2.2.2 SYSTEMATIC LITERATURE SEARCH
Given that malaria is considered highly sensitive to climate change at the global scale (Kovats, et al., 2003d; Kuhn, et al., 2005; Martens, et al., 1995a; Martens, et al., 1995b; Martens, et al., 1997), a literature search was conducted in order to determine the published availability of malaria dose-response functions and disease models that could be applied in climate change impact assessments. Papers included for consideration in this process were:

1) Significant WHO publications on climate change and disease (Kovats, et al., 2003c; Kuhn, et al., 2005; McMichael, et al., 2004; WHO, 2003a)
2) Relevant citations recorded within these documents, and those publications returned from ISI Web of Knowledge (WOK) and Medline (OVID) topic searches for "Malaria AND Climate."

The WOK and Medline (OVID) searches yielded more than 800 non-unique results, from which 52 English-language articles were selected on the basis of abstract contents and title for further investigation. Of these 52 publications, a final selection was made consisting of 32 papers published after 2000 (see Appendix I). This effort was not intended to be formally exhaustive, but was intended to provide a reasonable indication of the current state of knowledge and debate regarding the modelling of malaria.[2]

The results of this search show that although there have been significant efforts to model malaria risk and incidence around the world, consensus is still lacking when it comes both to determining the best modelling techniques and to anticipating the impact that climate change will have on malaria. Early efforts to capture the impact of environmental variables modelled epidemic potential based on a variety of variables (vector abundance, human population size, vector survival probability, vector biting frequency, and pathogen incubation period, and other entomological variables), and estimated that by 2100 global tropical epidemic potential would increase significantly compared to that in absence of climate change (Martens, et al., 1995b; Martens, et al., 1997). However, this could not be tied directly to malaria transmission because, among other reasons, human population immunity was not included in the model (Martens, et al., 1995b). Consequently, the two major WHO publications instead used the validated Mapping Malaria Risk in Africa model (MARA) to estimate, by global region, the percent increase in the population at risk of contracting malaria (Campbell-Lendrum, et al., 2003; McMichael, et al., 2004). Though these studies present relative risk figures, they do not represent disease incidence and are based on broad geographic regions that hold a wide range of environmental and socio-economic conditions.

The post-2000 papers sub-set emphasized the following approaches to modelling and predicting malaria risk, potential, and transmission:

1) **Mathematical models of entomological and biological processes**
(Bhattacharya, et al., 2006; Hoshen and Morse, 2004; Lindsay, et al., 2010; Lou and Zhao, 2010; Paaijmans, et al., 2009; Parham and Michael, 2010; Ruiz, et al., 2006)
2) **Large-scale scenario-driven models**
(Tol, 2008; van Lieshout, et al., 2004)
3) **Statistical short-term seasonal forecasting or regression-based models using environmental data and disease incidence**
(Abellana, et al., 2008; Bi, et al., 2003; Chatterjee and Sarkar, 2009; Craig, et al., 2004b; Dev and Dash, 2007; Gilbert and Brindle, 2009; Jury and Kanemba, 2007; Lindsay, et al., 2010; Teklehaimanot, et al., 2004; Thomson, et al., 2005; Thomson, et al., 2006a; Wandiga, et al., 2010; Wiwanitkit, 2006)
4) **Geographic Information System (GIS) and remote sensing (RS) analysis**
(Leonardo, et al., 2005)
5) **Analysis utilizing socio-economic and behavioural data**
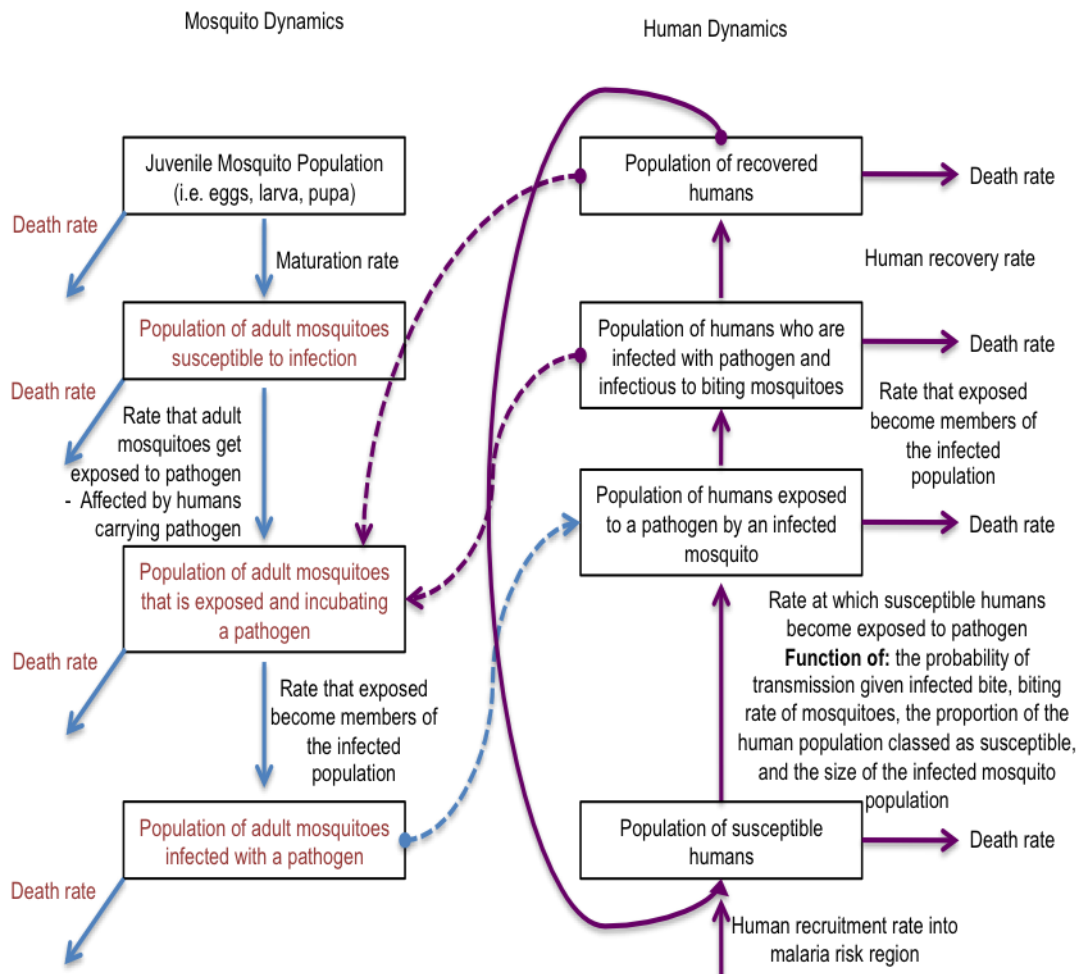(Coleman, et al., 2010; Craig, et al., 2004a; Stratton, et al., 2008).

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[2] Note: This statement on the purpose and limitations of the systematic literature search applies to every disease considered in this report.

Further elaboration on the entomological models found is found in section 2.2.3

2.2.3 ENTOMOLOGICAL MODELS

Of the entomological models found in the more recent papers (all of which implicitly assume the constancy of all socio-economic and behavioural variables), Lou and Zhao (2010) include the most detailed visualization of the entomological cycle for malaria transmission (reproduced sans equations in figure 2 for reference). As shown in this figure, and discussed in Lou and Zhao (2010), key aspects of this cycle, including the duration of the gonotrophic cycle, vector biting frequency, and vector survival rates are sensitive to temperature, and will therefore be sensitive to the effects of climate change. Additionally, although not explicitly taken into account by Lou and Zhao (2010), the length of the protozoan incubation period within the mosquito is also temperature dependent and would have to be included for a complete entomological/process model of malaria transmission (Martens, et al., 1997; Paaijmans, et al., 2009).

Figure 2:   Malaria entomological model including some of the human dynamics. Dashed lines are influence arrows. Solid lines indicate one category of population moving directly into another population category. Red lettering indicates a mosquito variable that is sensitive to temperature. Holding all socio-economic and human behavioural variables constant, the second level on the human side of the diagram would also be sensitive to temperature as a result of being a function of a temperature-sensitive mosquito variable. This would also make the third level on the human side indirectly affected by temperature. Adapted from (Lou and Zhao, 2010)

Mosquito Dynamics

Human Dynamics

Juvenile Mosquito Population (i.e. eggs, larva, pupa)

Death rate

Maturation rate

Population of adult mosquitoes susceptible to infection

Death rate

Rate that adult mosquitoes get exposed to pathogen - Affected by humans carrying pathogen

Population of adult mosquitoes that is exposed and incubating a pathogen

Death rate

Rate that exposed become members of the infected population

Population of adult mosquitoes infected with a pathogen

Death rate

Population of recovered humans

Death rate

Human recovery rate

Population of humans who are infected with pathogen and infectious to biting mosquitoes

Death rate

Rate that exposed become members of the infected population

Population of humans exposed to a pathogen by an infected mosquito

Death rate

Rate at which susceptible humans become exposed to pathogen
Function of: the probability of transmission given infected bite, biting rate of mosquitoes, the proportion of the human population classed as susceptible, and the size of the infected mosquito population

Population of susceptible humans

Death rate

Human recruitment rate into malaria risk region

2.2.4 THE ROLE OF NON-ENVIRONMENTAL VARIABLES

As mentioned previously, socio-economic and human behavioural variables are important in the disease transmission cycle and are frequently not constant with time. Many of the papers evaluated in this literature search explicitly highlight the importance of these non-environmental variables in the incidence of malaria around the world (Coleman, et al., 2010; Craig, et al., 2004a; Gething, et al., 2010; Stratton, et al., 2008; Wandiga, et al., 2010). The complexity of this relationship, and the regional/national variability regarding the extent to which socio-economic variables are capable of trumping the impact of environmental variables continues to fuel an active debate in the literature.

This debate is exemplified by the discussions surrounding Lafferty's 2009 suggestion that malaria and infectious disease risk zones will shift, rather than expand over the next century (Epstein, 2010; Lafferty, 2009, 2010; Ostfeld, 2009; Pascual and Bouma, 2009). These conflicting perspectives demonstrate the extent to which reconciliation is needed between, on the one hand, modelling depicting the extent to which climate change *could*, in theory, increase the geographic range and incidence of malaria through the aforementioned mechanisms, and on the other hand, discussions regarding the likelihood that climate change will *actually* drive increases in malaria given various historic and projected trends in human behaviour and socio-economic development.

This debate is particularly relevant in the context of projecting malaria incidence in countries where substantial effort is being made through behavioural changes to decrease the incidence of malaria from what might otherwise be expected given the environmental suitability of a particular location to malaria transmission.

2.3 DENGUE FEVER

2.3.1 INTRODUCTION

In the context of climate change, the study of dengue fever (DF), and its more deadly sibling dengue hemorrhagic fever (DHF),[3] shares a number of strong similarities with the study of malaria. As with malaria, DF is caused by the transmission of a pathogen (one of four types of flavivirus) to humans by mosquitoes (primarily *Aedes aegypti* and *Aedes albopictus*). Approximately two-thirds of the world's population lives within the geographic range of these vectors, and there are 50-100 million cases annually, making DF a strong rival to malaria for the title of Earth's most important vector-borne disease (Fuller, et al., 2009; Kovats, et al., 2003d). Because mosquitoes transmit the flaviviruses, the entomological mechanisms on which climate change can act are extremely similar to those in the *Anopheles* entomological cycle (Amarakoon, et al., 2008; Martens, et al., 1997; McMichael, et al., 2004). Accordingly, some studies used entomological models similar to those used for malaria, and have concluded that climate change will have a similar impact on the geographic range and transmission potential of DF as on malaria (Githeko, et al., 2000; Jetten and Focks, 1997; Martens, et al., 1997).

However, a significant difference between the malaria and DF is the extent to which the dengue vectors have become urbanized and indoor-dwelling. The extent of this urbanization is so complete that *Aedes* mosquitoes tend to breed exclusively in manmade water storage containers, and its lifecycle may be almost completely shielded in certain places from the effects of climate change-induced temperature and precipitation changes (Fuller, et al., 2009; Githeko, et al., 2000; Jansen and Beebe, 2010; Martens, et al., 1997). Therefore, despite the overlapping entomological characteristics between *Aedes* and

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[3] DF itself is not normally deadly. However, DHF can be when people lack access to medical care. DHF tends to occur when an individual contracts one of the four DF viruses after having previously contracted a different DF virus, as previous contact with any of the DF virus types increases the risk of more extreme responses to subsequent infections (Medline, 2010; WHO, 2010).

Anopheles mosquitoes, this urbanization points to an extremely important relationship between human-based, non-environmental variables and DF incidence, which should significantly affect DF modelling efforts.

Numerous studies on DF in the Caribbean identify cyclical-like variations in disease incidence, and correlations with variables such as temperature, precipitation, sea surface temperature (a proxy for El Niño Southern Oscillation impacts), vegetation indices, and humidity (Amarakoon, A., et al., 2004; Amarakoon, et al., 2008; Ebi, et al., 2006; Fuller, et al., 2009; Johansson, et al., 2009a; Johansson, et al., 2009b; Jury, 2008). Despite their utility in local predictions of DF outbreaks, the relationship between these variables and DF incidence is not constant through time or space. This gives an indication as to the complexity of this disease transmission cycle.

2.3.2 SYSTEMATIC LITERATURE SEARCH
In order to identify potential dose-response relationships between DF and climate, a literature search similar to that conducted for malaria was undertaken for dengue. The same major WHO publications were considered, as were some of the references contained there-in. Medline (OVID) and WOK searches for "Dengue" AND "Climate" revealed more than 400 non-unique results, from which 33 English-language were selected for further investigation on the basis of abstract contents and title. Of these 33 publications, a final selection was made consisting of 25 papers published no earlier than 2000. Two additional, Caribbean-specific sources were located through the research process and were also considered (see Appendix II).

The approaches utilized by the papers found during the literature search are similar to those found in the malaria search, with publications detailing the following approaches:

1) **Entomological/process-based modelling**
   (Amarakoon, et al., 2008; Barbazan, et al., 2010; Degallier, et al., 2010; Focks and Barrera, 2006; Hopp and Foley, 2001, 2003; Yang, et al., 2009a, 2009b)
2) **Statistical or time series**
   (Amarakoon, A., et al., 2004; Brunkard, et al., 2008; Halide and Ridd, 2008; Hu, et al., 2010; Hurtado-Díaz, et al., 2007; Johansson, et al., 2009a; Johansson, et al., 2009b; Jury, 2008; Lu, et al., 2009; Luz, et al., 2008; Wu, et al., 2007)
3) **Global scenarios**
   (Hales, et al., 2002);
4) **GIS and/or RS –based analyses**
   (Fuller, et al., 2009; Kolivras, 2010)
5) **Socio-economic analysis**
   (Jansen and Beebe, 2010; Ooi and Gubler, 2009; Tseng, et al., 2009).

This search effort revealed a similar debate to that found in the malaria literature. Some researchers focus on the role of socio-economic variables and argue that that DF *transmission* is climate insensitive (Sutherst, 2004), and the others continue to focus on the *entomological* sensitivity of the dengue vectors to climate (Yang, et al., 2009a, 2009b). Taken together, the body of literature consulted indicates that:

1) The urbanization of *Aedes* vectors largely shields these vectors from the entomological effects of temperature
2) That DF incidence is extremely sensitive to control measures
3) DF is extremely sensitive to standing population immunity
(Jansen and Beebe, 2010; Kuhn, et al., 2005)

The content of this literature lends credence to the idea suggested by Ebi et al. (2006) that the location-specific socio-economic variables and behavioural/control practices will likely continue to trump the impact of climate change for this disease. This also strongly supports the idea that attentive control measures combined with public health awareness of DF could nullify, or at least markedly offset, the impact that climate change-induced temperature increases could have had in the absence of the urbanization of the vector.

3. WATER & FOOD-BORNE DISEASE

3.1 INTRODUCTION

Water and food-borne diseases are those diseases transmitted to humans through physical contact with, inhalation of aerosolized particles from, or ingestion of contaminated sources of water and food. The pathogens that generate the diseases that fall into this category include viruses, bacteria, and parasites, and as is the case with vector-borne diseases, the most vulnerable groups are young children, the elderly, and anyone whose immune system is compromised (Ebi, et al., 2008). Examples of water and food-borne pathogens that are significant at a global level include species of rotavirus, the hepatitis A and E viruses, members of the norovirus family, species of bacteria within the *Campylobacter*, the *Shigella*, and the *Salmonella* genera, including *Salmonella typhi,* and protozoa found within both the *Cryptosporidium* and *Giardia* genera (Ebi, et al., 2008; Kovats, et al., 1999; Kuhn, et al., 2005). This category also includes, but is not limited to, diseases caused by harmful algal blooms and the toxins they generate, diseases caused by aquatic bacteria in the *Vibrio* genus such as *V. parahaemolyticus*, *V. vulnificus*, and *V. cholerae*, and diseases caused by aquatic amoebae like *Naegleria fowleri* (Ebi, et al., 2008; Kovats, et al., 1999; Luber and Prudent, 2009).

Although the specific reactions to changes in environmental conditions vary by pathogen, in general, the potential impacts of climate change on these can be summarized as follows: increasing temperatures can lead to expanded geographic and altered seasonal/temporal ranges of these pathogens, as well as decreased development and/or replication times and increased pathogen population growth (except in the case of viruses where temperatures higher than particular thresholds result in virus inactivation). In the absence of well-defined, reliable sanitation practices and infrastructure, both precipitation increases and decreases can result in an increased loading of local water resources with pathogens. This excess of pathogens can then be passed on to humans through contact with or consumption of the contaminated water, or through the consumption of food that came into contact with the contaminated water. Importantly, precipitation and temperature changes affecting coastal environments can also drive changes in the coastal aquatic bacteria populations as a result of increased or decreased surface water salinity (Ebi, et al., 2008; Kovats, et al., 1999; Kovats, et al., 2003e; McMichael, et al., 2004).

The primary outcome of contact with the aforementioned environmentally sensitive pathogens is gastroenteritis/diarrheal disease, for which there are often reasonably informative data sets. In addition to gastroenteritis, the following conditions demonstrate sensitivity to environmental change, and are often relevant in tropical developing countries: schistosomiasis, leptospirosis, and ciguatera poisoning.

3.2 GASTROENTERITIS

3.2.1 INTRODUCTION
Gastroenteritis (GE) – the largely non life-threatening inflammation of the gastrointestinal tract – causes bouts diarrhea and is caused by a large number of viruses, bacteria, and parasites which are transmitted to humans via contact with contaminated food and water, as previously described. Because of the strong sensitivity to environmental change of many of the causative agents of GE, researchers anticipate that the impact of climate change on GE will be highly significant (Ebi, et al., 2008; Kovats, et al., 2003e; McMichael, et al., 2004).

2.2.2 SYSTEMATIC LITERATURE SEARCH
Despite the global importance of GE, and despite the sensitivity of many GE-causing pathogens to temperature and precipitation, the literature search performed for GE yielded far fewer relevant and appropriate results than did the literature searches focusing on malaria and DF. In addition to the GE-related parts of the major WHO publications included in the malaria and DF literature searches (Kovats, et al., 2003e; Kuhn, et al., 2005; McMichael, et al., 2004; WHO, 2003a), Medline (OVID) and WOK searches were performed for papers published after 2001 using the terms "Diarrh* AND climate," "Gastroenteritis AND climate," and "Gastroenteritis AND forecast."

These search terms were used, rather than more pathogen-specific terms, because the available baseline health data tended not to distinguish between most GE-causing pathogens. Thus, although this search, like the others, was not intended to be formally exhaustive, it was intended to reveal dose-response relationships connecting environmental variables to all-cause GE. From the results yielded by this search, four studies were selected for further investigation along with the two studies cited in the WHO publications. All six studies are listed in Appendix III.

The two papers discussed in the WHO documents are Checkley et al. (2000) and Singh et al. (2001), the former of which looked at the impacts of the 1997/1998 El Niño summer temperature increases and relative humidity changes on the number of hospital admissions for children under the age of 10 in Peru, and the later of which used data covering 1986 – 1994 to investigate the simultaneous effects of precipitation and temperature on the average annual incidence of adult GE on 18 Pacific islands (Checkley, et al., 2000; Singh, et al., 2001). The other four papers involved using the following:

1) Temperature and cholera presence to predict adult hospital admissions due to diarrhea in Lima, Peru (Lama, et al., 2004)
2) Forecasting methods to predict age-stratified diarrhea in Mali (Medina, et al., 2007)
3) Temperature and relative humidity to predict all-age GE incidence in Japan (Onozuka, et al., 2010)
4) Monthly rainfall and temperature to predict diarrhea incidence in children under age five at a global level (Lloyd, et al., 2007)

These papers reveal that increasing temperatures, to varying degrees across age, space and time, tend to increase GE incidence (or hospitalizations). Precipitation (and humidity) also affect incidence, though the sign of this relationship is inconsistent across these studies. It is surprising that these studies did not place a lot of emphasis on socio-economic conditions like the state of sanitation infrastructure, and cultural patterns like hygiene practices that should, considering GE is primarily water and food-borne, influence incidence of GE across space and time.

3.3 SCHISTOSOMIASIS

3.3.1 INTRODUCTION

Schistosomiasis is a disease caused by parasitic worms within the genus *Schistosoma*, three of which - *S. mansoni*, *S. japonicum*, and *S. haematobium* – cause the majority of cases globally. Transmission occurs when the free-swimming schistosomiasis larvae (called *cercoria*) penetrate the skin of humans, travel through the human circulatory system looking for a mate, and then utilize humans as host for reproductive purposes (Webber, 2005; Yang, 2006).[4] Globally, close to 800 million people live in areas where they are at risk from infection, and 76 countries consider schistosomiasis endemic, something that translates into a significant burden of disease (Yang, 2006). While people can become infected at any age, outdoor workers are especially vulnerable. Also, it is common for children in endemic areas to acquire an infection which develops in terms of symptoms and intensity, if left untreated, until the children are approximately age 15 when the intensity of the disease begins to decrease (Webber, 2005). Infection can cause an enlarged liver, an enlarged spleen, bloody urine, and fever (Webber, 2005; Zhou, et al., 2008). Although the life cycle is somewhat involved (see figure 3), as is the case with malaria, the sensitivity of schistosomiasis to climate change is a direct result of the sensitivity of the worm and intermediate (i.e. snail) host life cycles to environmental variables (Mangal, et al., 2008), which in turn makes the regions on the fringe of endemic areas some of the most vulnerable in terms of the effects of climate change on schistosomiasis (Sutherst, 2004).

3.3.2 SYSTEMATIC LITERATURE SEARCH

As was undertaken for the diseases considered previously, a literature search beyond the primary WHO sources already specified (as well as Martens et al. (1997), Sutherst (2004) and Martens et al. (1995a) which were identified previously) to try and identify the current state of knowledge regarding the modelling of climatic influences on schistosomiasis. Medline (OVID) and WOK searches for "Schistosomiasis AND Climate" and "Schistosomiasis AND Prediction" revealed more than 150 non-unique English language sources, five of which were selected for further investigation, as was a related PhD report (see Appendix IV).

As was the case with the GE literature search, this yielded a surprisingly small pool of papers, and interestingly, all but one of these five papers focused on entomological modelling that attempted to capture the types of dynamics shown in figure 3 (Liang, et al., 2005; Liang, et al., 2007; Mangal, et al., 2008; Martens, et al., 1995a; Martens, et al., 1997). This stands in contrast to the literature for the diseases considered previously, which showed a reasonable balance between 1) the statistical and time series approaches that both explicitly and implicitly contain socio-economic and cultural variable, and 2) the mathematical models depicting entomological relationships. While these papers indicate that, to a point, increasing temperature can increase infection rates and the per person worm burden (Mangal, et al., 2008; Martens, et al., 1997), there is a point beyond which increasing temperatures increases the mortality in various parts of the pathogen and host life cycles, which translates into decreased infection (Martens, et al., 1995a). Liang et al. (2005) also point out that location-specific features of the life cycle are important enough to schistosomiasis dynamics, that it is difficult to transfer a model to a different site and calibrate it effectively.

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[4] Thus, although an earlier stage in the parasite life cycle requires species-specific snails as intermediate hosts (something that results in schistosomiasis being classified as a zoonotic disease), this project classifies the disease as water-borne.

Figure 3:   The main steps in the *Schistosoma* life cycle, as recreated from (Martens, et al., 1995a), are

shown below. The red arrows indicate phases that are directly impacted by temperature changes, while the dark blue arrows indicate the stages of influence for precipitation. The *cercariae* and *miracidia* are free-living, aquatic stages in the life cycle.
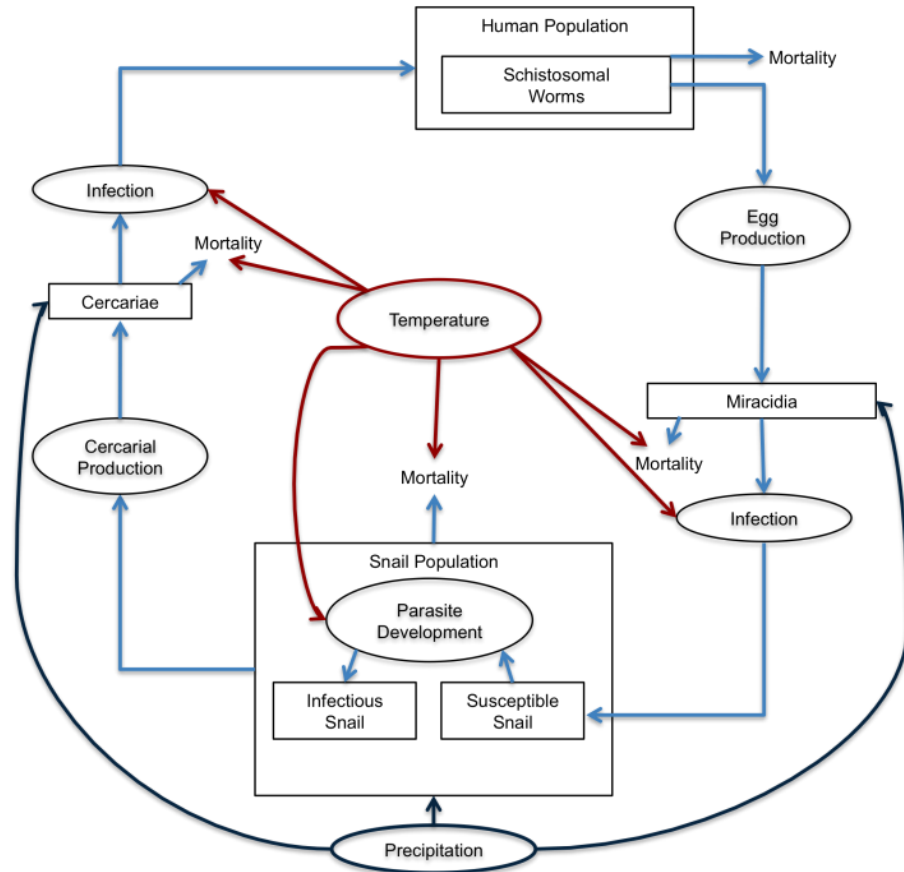
3.4 LEPTOSPIROSIS

3.4.1 INTRODUCTION

Leptospirosis is caused by more than 200 serovars of the more than 16 species of bacteria in the genus *Leptospira*, which are common around the world (Levett, 2001). Transmission to humans occurs when contact occurs between the bacteria - which are released into the environment through the bodily fluids of various mammals, reptiles, and amphibians - and human mucus membranes, waterlogged skin, or broken skin.

Once transmission occurs, the bacteria target the kidneys, liver, lungs, and cerebrospinal fluid of its host, and can generate an incredible range of symptoms mimicking other conditions including, but not limited to, influenza, malaria, typhoid fever, hepatitis, food poisoning, renal failure, DF/DHF, and chronic fatigue syndrome (Flannery, et al., 2001; Storck, et al., 2008; WHO, 2003b). Because the symptoms of infection are so variables and can last for years, because most cases are relatively mild and non-fatal, and because confirming leptospirosis requires a lab-based test, leptospirosis is extremely under-

diagnosed around the world (Levett, 2001). Importantly, immunity is achieved only on a per-serovar basis, meaning the risk of infection is likely to remain high throughout life for those in the most vulnerable groups (i.e. children, outdoor laborers/farmers, sewer workers, veterinarians, food-prep workers, leisure participants, soldiers, and lab staff) (WHO, 2003b).

Transmission of leptospirosis to humans is sensitive to environmental change in two respects: 1) Land use patterns can make certain environments more conducive to populations of the mammals, and particularly the rodents, that carry the *Leptospira* bacteria. This can increase the prevalence of the bacteria in the environment. 2) Precipitation events wash the bodily fluids containing the bacteria into local bodies of water, thereby concentrating them and increasing the likelihood of human contact with them (Victoriano, et al., 2009).

3.4.2 SYSTEMATIC LITERATURE SEARCH

A literature search was performed in Medline (OVID) and WOK for "Leptospirosis AND Climate," "Leptospirosis and Temperature," and "Leptospirosis and Precipitation." The search results yielded comparatively few results, and while several papers were found discussing leptospirosis in general, only four papers were selected for further investigation for a dose-response relationship (see Appendix V).

Of these papers, two graphically show clear coincidence between changes in precipitation and reported leptospirosis incidence through time (Slack, et al., 2006; Storck, et al., 2008). Their figures are included below for reference as figure 4 and 5. However, while both these papers indicate a lag time of about a month between the start of sustained rainfall and the onset of leptospirosis, neither included dose-response relationships between precipitation and incidence data. Similarly, while another paper identifies a background rate of leptospirosis in developing countries of 10-100/100,000 people, and derives statistical clusters of disease incidence with time, no dose-response relationship is presented (Tassinari, et al., 2008). The final paper considered, however, did formalize a nonlinear relationship between severe leptospirosis incidence in Salvador, Brazil and the previous week's total rainfall (Codeço, et al., 2008). Their efforts treated leptospirosis incidence as a disease demonstrating threshold behaviour that is driven by precipitation.

Figure 4: This is a copy of figure 2 from Storck et al. (2008) showing overlapped time series of reported cases of leptospirosis and rainfall for the Caribbean Island of Guadeloupe.
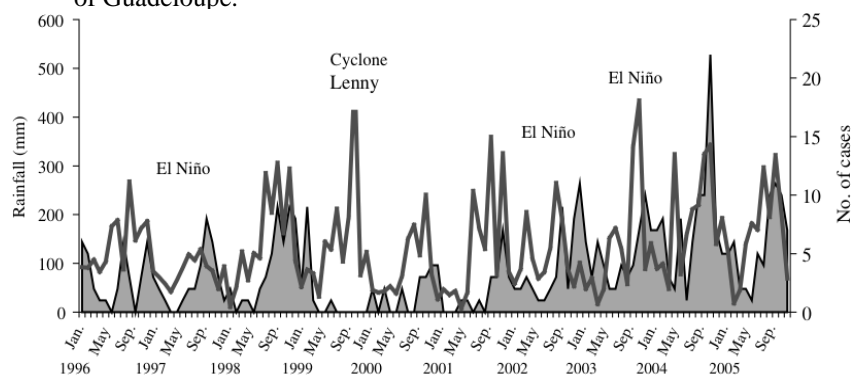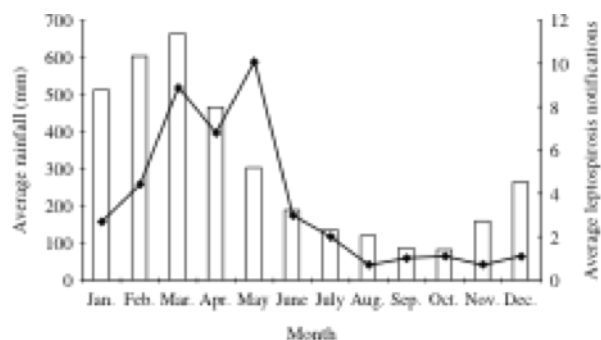

Fig. 2. Monthly rainfall (—) and leptospirosis cases (■) for 1996–2005.

Figure 5:   This is a copy of figure 3 from Slack et al. (2006) showing overlapped average
             rainfall and average leptospirosis incidence by month for Innisfail health district in
             the state of Queensland, Australia. The data used spanned 1998 – 2004.

3.5 CIGUATERA POISONING

3.5.1 INTRODUCTION

Ciguatera poisoning is a condition caused by the ingestion of either ciguatoxins or
maitotoxins. These are some of the most toxic substances on Earth, and they enter the
human food chain through the herbivorous reef fish that feed on the dinoflagellates that
produce these toxins (Fleming, 2010; Kovats, et al., 1999). Ciguatoxins (the most important
cause of ciguatera poisoning) are lipid-soluble and harmless to fish, and this results in them
becoming increasingly concentrated in the fatty tissues of fish that feeding at higher trophic
levels in the marine food chain. Furthermore, they are colorless, tasteless, odorless, heat-
stable, and acid-stable and therefore cannot be detected in fish or removed prior to
consumption (Fleming, et al., 2006; Fleming, 2010; Jaykus, et al., 2008; Kipping, et al.,
2006; Ting and Brown, 2001).

Ciguatera poisoning is important to consider in this project because the population
dynamics of the ciguatoxin-producing dinoflagellate species are sensitive to changes in sea
surface temperatures (SST) (Jaykus, et al., 2008) and because within several hours of
consuming tainted fish, 73-100% of people will start to experience gastrointestinal
symptoms, which are commonly followed by cardiovascular symptoms as well as
debilitating neurologic symptoms. In instances where these symptoms are non-fatal, the
neurologic symptoms can continue for years after the initial exposure. Additionally, for six
months following the initial poisoning certain foods such as fish, ethanol, caffeine, and nuts
can re-trigger the initial symptoms of the poisoning. Due to the fact that these symptoms
overlap significantly with other algae-produced toxins, diagnosis can be difficult.
Consequently, while highly significant, ciguatera poisoning is significantly under-reported
not only in endemic areas, but also on a global level (Fleming, et al., 2006; Fleming, 2010;
Jaykus, et al., 2008; Tester, et al., 2010).

Ciguatera poisoning often occurs year round in places like the Caribbean, and the link
between SST-driven ciguatoxin production and incidence of ciguatera is extremely
complex. In addition to the common genera of dinoflagellate responsible for the production
of ciguatoxins – *Gambierdiscus* – the following dinoflagellate populations also produce
ciguatoxins: *Coolia monotis*, *Prorocentrum belizeanum*, *Prorocentrum lima*, *Prorocentrum*

mexicanum, *Prorocentrum hoffmannianum*, *Ostreopsis lenticularis*, and *Ostreopsis siamensis*. Consequently, it is also common for individual fish to carry multiple ciguatoxins (Faust, 2009; Tester, et al., 2010; Tosteson, 2004).

3.5.2 SYSTEMATIC LITERATURE SEARCH

The literature search was performed in Medline (OVID) and WOK for: "Ciguatera AND Climate," "Ciguatera AND Sea Surface Temperature," and "Ciguatera AND Caribbean." Of the 135 results, 15 were selected for further inspection and of those only 5 were included in the final round of paper selection. An additional in-press article discussing the state of ciguatera poisoning in the Caribbean was located through a Google search for ciguatera incidence data (Tester, et al., 2010). These 6 papers are listed in Appendix VI.

Three of these articles discussed ciguatera in the Caribbean directly. Even though they did not yield dose-response relationships, they did provide important information on the Caribbean-specific pattern of ciguatera poisoning that demonstrates that while ciguatera poisoning incidence is likely going to be sensitive to climate change-induced changes in SST, it will also be incredibly difficult to predict.

Only one paper attempted to model ciguatera poisoning incidence, and did so using data from French Polynesia (Chateau-Degat, et al., 2005). Their two-step model formally connected SST and dinoflagellate density (with a lag of 17 months), and dinoflagellate density and disease incidence (with a lag of 3 months). The results of their model demonstrate there is potential with regards to modeling and predicting ciguatera poisoning in response to changes in SST (see figures 6 and 7) However, this model fails to capture the peaks in both dinoflagellate density and ciguatera incidence. This indicates either that SST is not the only explanatory variable worth considering, or that problems of under-diagnosis limit predictive power of the model (or, more likely, that some combination of both these limitations is present in their data). This significantly limits the utility of their model in its current form.

Figure 6:   This is a copy of figure 2 from Chateau-Degat et al. (2005) showing an actual time series of dinoflagellate densities, and a modelled time series of dinoflagellate densities.
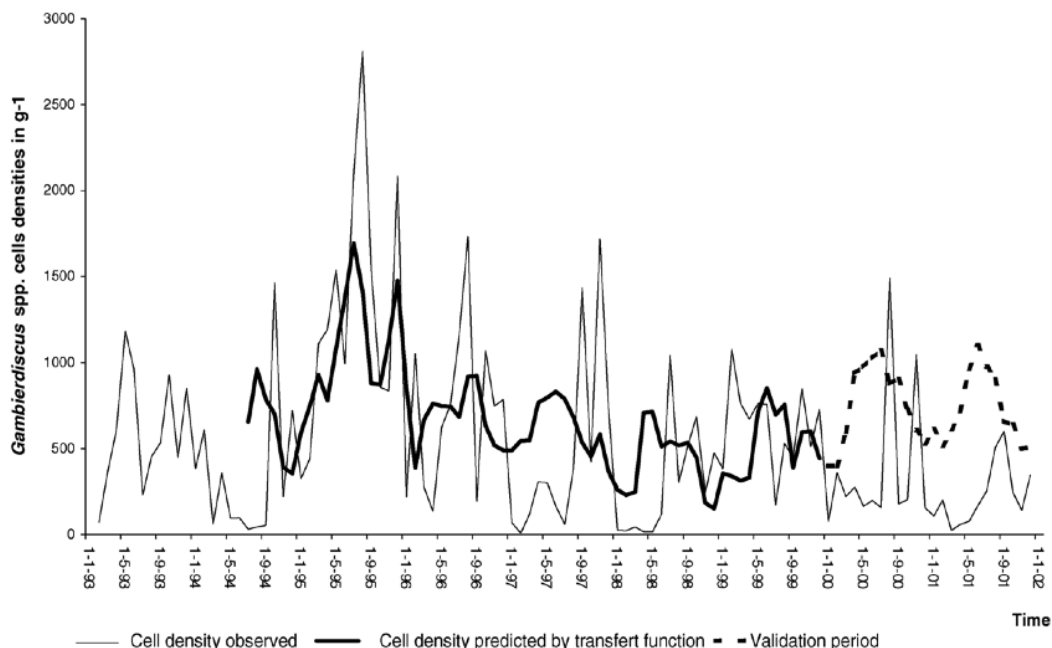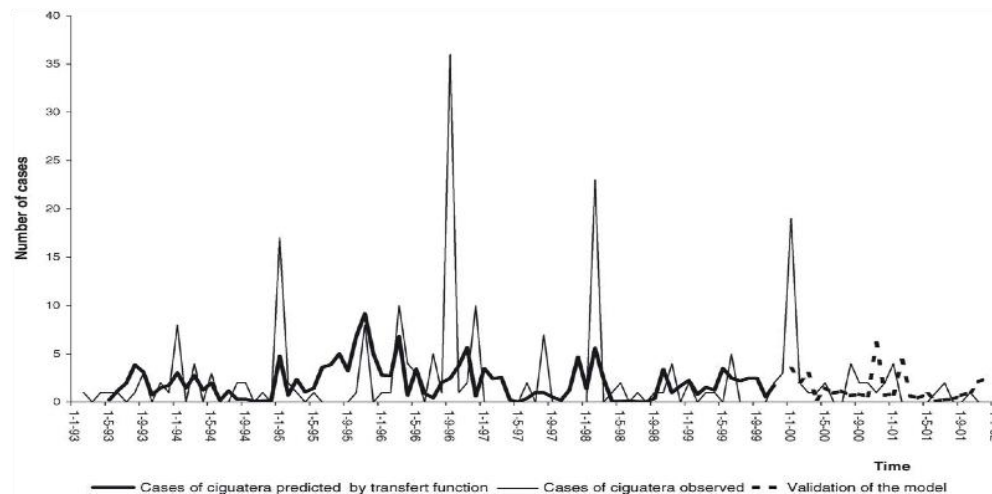
Figure 7:   This is a copy of figure 3 from Chateau-Degat et al. (2005) showing actual and
                predicted cases of ciguatera poisoning. This also fails to capture the extreme
                values of incidence.

4. MENINGOCOCCAL MENINGITIS

4.1 INTRODUCTION

Predominantly caused by six of the thirteen serogroups of the air-borne bacteria *Neisseria meningitidis* (A, B, C, W135, X, Y), meningococcal meningitis (MM) is a seriously debilitating disease. As many as 17% of patients who do receive treatment still die from MM. Furthermore, as many as 20% of MM survivors are left with permanent impairment or disability as a result of having contracted MM (Boyne, 2001; Kuhn, et al., 2005; Palmgren, 2009). Considering that the most vulnerable groups are children and young persons, outbreaks of MM have the potential for significant long-term economic and social consequences (Roberts, 2008).

One important theory of MM transmission focuses on the damage caused to the mucus membranes of the upper respiratory by the air-borne dust that abounds in the African meningitis belt during the driest months of the year (Roberts, 2008; Yaka, et al., 2008). This theory may have relevance in the tropical developing countries west of Africa, such as those in the Caribbean. Not only does the region have a distinctly dry season, but it also receives significant quantities of the same dust implicated in the initiation of African MM epidemics. This dust adds to respiratory stress caused by localized pollution and volcanic activity and may therefore have a role to play in MM transmission (Prospero and Lamb, 2003).

4.2 SYSTEMATIC LITERATURE SEARCH

Medline (OVID) and WOK searches for "Meningitis and Climate," "Meningococcal AND Climate," "Neisseria meningitidis AND climate," "N. meningitidis AND Climate" yielded 51 English-language articles on WOK, from which eleven were short-listed for further

investigation, and more than 400 results on Medline, from which 15 were short-listed. Of these 26, a final ten were selected for further investigation (Appendix VII).

This search revealed that a wide variety of environmental variables have been connected, with various lag times, to MM incidence including dust, wind speed, absolute humidity, land cover, precipitation, cold cloud duration, and soil type (Molesworth, et al., 2003; Palmgren, 2009; Thomson, et al., 2006b; Yaka, et al., 2008). None of the studies presented transferable dose-response relationships, however, and several present evidence not only that MM epidemiology varies globally in relation to environmental variables, but also that definitive causal links between the environment and MM epidemics have not yet been identified (Harrison, et al., 2009; Palmgren, 2009; Roberts, 2008). Part of the reason for this is the fact that the effects of carriers and vaccination in *N. meningitidis* transmission remains unclear, and has never been successfully modelled to date (Palmgren, 2009; Roberts, 2008; Thomson, et al., 2006b).

5. CARDIO-RESPIRATORY DISEASE

5.1 INTRODUCTION

There is strong evidence suggesting that climate change will impact on the disease burden associated with cardiovascular and respiratory conditions (for lists, see table 1) because of the sensitivity of human cardiovascular and respiratory systems to temperature change. Increases in temperature increase blood viscosity. In turn, this can trigger heart attacks, strokes, and other vascular events. Temperature changes can also increase ones heart rate, cause constriction of the bronchial tubes, and exacerbate both concurrent acute and chronic respiratory conditions (Campbell-Lendrum, et al., 2003; Ebi, et al., 2008; McMichael, et al., 2004). Adults who suffer from pre-existing cardiovascular and respiratory diseases, the elderly, children, outdoor labourers, and the mentally ill are most vulnerable to this category of impact. Additionally, individuals who lack access to air conditioning are more at risk, as are those individuals who reside in cities and are exposed to the 'urban heat island effect,' as both these factors exacerbate the effects of temperature increases (Ebi, et al., 2008; Hales, et al., 2003; Luber and Prudent, 2009).

Although much research has been done to define dose-response relationships for mortality and temperature, much of this research has been done in temperate climates where the relationship between temperature and mortality is J-shaped or U-shaped (Ebi, et al., 2008). However, there has been some evidence that tropical climates exhibit different (i.e. non-U-shaped) relationships between temperature and mortality (Kovats, et al., 2003b).

The importance of this potential health impact is largely a consequence of the fact that cardiovascular diseases are currently the global leading cause of death (Chiu, et al., 2010). Most regions currently suffering a significant burden from cardiovascular and respiratory diseases started to experience increased incidence of these diseases following an epidemiological transition. This transition involves an increased availability of calories paired with both decreased vector-borne and water and food-borne illness compared to historic levels, and a significant increase in the consumption of sugary, salty and fatty foods intake (Albert, et al., 2007; Cunningham-Myrie, et al., 2008).

Table 1: Cardiovascular and Respiratory Diseases as defined in the *International Classification of Diseases and Related Health Problems, 10th Revision* (2007). Available From (WHO, 2007)

Cardiovascular Diseases		Respiratory Diseases	
ICD Code	Name	ICD Code	Name
I00-I02	Acute Rheumatic Fever	J00-J06	Acute Upper Respiratory Infections
I05-I09	Chronic Rheumatic Heard Diseases	J09-J18	Influenza and Pneumonia
I10-I15	Hypertensive Diseases	J20-J22	Other Acute Lower Respiratory Infections
I20-I25	Ischaemic Heart Diseases	J30-J39	Other Diseases of the Upper Respiratory Tract
I26-I28	Pulmonary Heart Diseases of Pulmonary Circulation	J40-J47	Chronic Lower Respiratory Diseases
I30-I52	Other forms of Heart Disease	J60-J70	Lung Diseases Due to External Agents
I60-I69	Cerebrovascular Diseases	J80-J84	Other Respiratory Diseases Principally Affecting the Intersitium
I70-I79	Diseases of Arteries, Arterioles, and Capillaries	J85-J86	Suppurative and Necrotic Conditions of the Lower Respiratory Tract
I80-I89	Diseases of Veins, Lymphatic Vessels and Lymph Notes, not Elsewhere Classified	J90-J94	Other Diseases of Pleura
I95-I99	Other and Unspecified Disorders of the Circulatory System	J95-J99	Other Diseases of the Respiratory System

5.2 SYSTEMATIC LITERATURE SEARCH

WOK searches[5] for "Temperature Morbidity AND Climate," "Heat-related Morbidity AND Climate," "Heat-Related Mortality AND Climate," "Cardiovascular Mortality AND Climate," "Cardiovascular Morbidity AND Climate," "Respiratory Morbidity AND Climate," and "Respiratory Mortality AND Climate" collectively returned more than 500 non-unique English language results, from which 52 were selected for further investigation. From these, a final 38 articles, including a few review papers, were consulted for dose-response relationships (see Appendix VII). It is important to note that in the selection of these 38 papers, many papers which focused solely on temperate areas were excluded as an *a prioi* decision had been made to try and find a dose-response relationship relevant to a tropical location. Thus, this literature search is not formally exhaustive of all dose-response relationships connecting mortality to temperature.

Detailed summaries and critical reviews of many of the articles selected (as well as some of the studies excluded) can be found elsewhere (Basu and Samet, 2002; Gosling, et al., 2009; Hajat and Kosatky, 2009; Kovats and Hajat, 2007). Although a full critical review of this

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[5] The WOK searches were performed first and as so many relevant results were returned, and as the literature search was not intended to be formally exhaustive, the searches were not duplicated on Medline (OVID) in this case.

literature and its ramifications is beyond the scope of this paper, it is important to highlight some key aspects of the literature. The first feature worth mentioning is that the dose-response functions captured by this literature search varied significantly from a 1% change in mortality per 1°C increase to more than a 50% change per 1°C increase, depending on the location, the conditions considered, the particular details of the study design, and the data utilized (Díaz, et al., 2002; Hales, et al., 2000). This demonstrates the importance of trying to find as close a match as is possible when transferring, rather than deriving, a dose-response relationship between temperature and mortality.

Another feature worth mentioning is that in the vast majority of these studies, there was no breakdown by specific diseases, and mortality data was either analyzed without regard to cause of death, or was broken down into broad categories such as cardiovascular, respiratory, or cardio-respiratory. The primary reason given for this is the general paucity of disease-specific mortality data. Although one can only work with the data that is available, this does somewhat run the risk of double counting the impacts of climate change on other temperature-sensitive conditions in places where mortality from the conditions is significant. It is also important to mention that while most of these studies utilized time series analysis to generate the dose-response relationships, they did not all do so in the same way. Some, for instance, looked summer-temperature morality relationships instead of, or separately from, all-year relationships (Chung, et al., 2009; Vaneckova, et al., 2008; Zanobetti and Schwartz, 2008). Some attempted to analyze the relationship between short-term heat waves and mortality, rather than, or in addition to, longer-term trends (Díaz, et al., 2002; Hajat, et al., 2006; Medina-Ramón and Schwartz, 2007; Rey, et al., 2007; Saez, et al., 1995).

Still other studies at least partially stratified their results by age (Bell, et al., 2008; Davis, et al., 2004; Díaz, et al., 2002; Hales, et al., 2000; Medina, et al., 2007; Saez, et al., 1995; Sheridan, et al., 2009; Vaneckova, et al., 2008). There were also some research that explicitly included particulate-matter air pollution (PM) and ozone as either explanatory variables or covariates in at least one of dose-response relationships generated (Hales, et al., 2000; McMichael, et al., 2008; Vaneckova, et al., 2008). While there is evidence that PM and ground-level ozone increase mortality both independently from, and synergistically with temperature (Daniels, et al., 2000; Ebi, et al., 2008; HEI International Scientific Versight Committee, 2004; Kovats, et al., 2003a; UNDP/World Bank Energy Sector Management Assistance Programme, 2004), this effect varies by location, as does its synergism with temperature (Ebi, et al., 2008; UNDP/World Bank Energy Sector Management Assistance Programme, 2004). Finally, it is important to mention that most of the relationships revealed in this literature search focused on large cities. Given that the urban heat island effect can be significant, these relationships are essentially non-transferable to more rural locations.

6. SYNTHESIS & CONCLUSION

It is clear from this review that there are a wide variety of promising approaches currently being pursued around the world to try and unravel the causal mechanisms that link climate change to human health. However, it is also clear from this review that there are many unresolved methodological debates within the literature and that there is some conflict between approaches that try and derive long-term dose-response for health impact projections and those that are best suited for near term disease monitoring to facilitate rapid health sector or governmental response. Each will have a role to play in the future of disease and health management, but they serve different purposes and there is a risk that too great a focus on one may come at the cost of undermining the continued development of the other. Finally, for many of these diseases there has been insufficient integration of

the environmental drivers of disease with the non-environmental drivers of disease. This is an area critically in need of additional future, and warrants significant attention in the future. After all, the non-environmental variables are the ones that humans will have the best opportunity to be able to target and control with adaptation efforts over the next 100 years, not the environmental-variables.

References

Abellana, R., C. Ascaso, J. Aponte, F. Saute, D. Nhalungo, A. Nhacolo and P. Alonso. 2008. Spatio-Seasonal Modeling of the Incidence Rate of Malaria in Mozambique. *Malaria Journal* 7: 228.

Albert, J.L., P.M. Samuda, V. Molina, T.M. Regis, M. Severin, B. Finlay and J.L. Prevost. 2007. Developing Food-Based Dietary Guidelines to Promote Healthy Diets and Lifestyles in the Eastern Caribbean. *Journal of Nutrition Education and Behavior* 39: 343 - 350.

Amarakoon, A., A. Chen, S. Rawlins and M. Taylor. 2004. Dengue Epidemics - Its Association with Precipitation and Temperature, and Its Seasonality in Some Caribbean Countries. *West Indian Medical Journal* 53 60.

Amarakoon, D., A. Chen, S. Rawlins, D.D. Chadee, M. Taylor and R. Stennett. 2008. Dengue Epidemics in the Caribbean-Temperature Indices to Gauge the Potential for Onset of Dengue. *Mitigation and Adaptation Strategies for Global Change* 13: 341 - 357.

Barbazan, P., M. Guiserix, W. Boonyuan, W. Tuntaprasart, D. Pontier and J.-P. Gonzalez. 2010. Modelling the Effect of Temperature on Transmission of Dengue. *Medical and Veterinary Entomology* 24: 66 - 73.

Baron, E.J. 2009. A Nutty Idea for Controlling the Spread of Malaria. *Travel Medicine Advisor*

Basu, R. and J.M. Samet. 2002. Relation between Elevated Ambient Temperature and Mortality: A Review of the Epidemiologic Evidence. *Epidemiologic Reviews* 24: 190 - 202.

Bell, M.L., M.S. O'Neill, N. Ranjit, V.H. Borja-Aburto, L.A. Cifuentes and N.C. Gouveia. 2008. Vulnerability to Heat-Related Mortality in Latin America: A Case-Crossover Study in São Paulo, Brazil, Santiago, Chile and Mexico City, Mexico. *International Journal of Epidemiology* 37: 796 - 804.

Bhattacharya, S., C. Sharma, R.C. Dhiman and A.P. Mitra. 2006. Climate Change and Malaria in India. *Current Science* 90: 369 - 375.

Bi, P., S. Tong, K. Donald, K.A. Parton and J. Ni. 2003. Climatic Variables and Transmission of Malaria: A 12-Year Data Analysis in Shuchen County, China. *Public Health Reports* 118: 65 - 71.

Boyne, L. 2001. Meningococcal Infection. *Nursing Standard* 16: 47 - 55.

Brunkard, J.M., E. Cifuentes and S.J. Rothenberg. 2008. Assessing the Roles of Temperature, Precipitation, and Enso in Dengue Re-Emergence on the Texas-Mexico Border Region. *salud pública de méxico* 50: 227 - 234.

Campbell-Lendrum, D.H., C.F. Corvalán and A. Prüss-Ustün. 2003. 'How Much Disease Could Climate Change Cause?' In *Climate Change and Human Health: Risks and Responses* edited by A.J. McMichael, D.H. Campbell-Lendrum, C.F. Corvalán,

K.L. Ebi, A.K. Githeko, J.D. Scheraga and A. Woodward, 133 - 158. Geneva: World Health Organization.

Chateau-Degat, M.-L., M. Chinain, N. Cerf, S. Gingras, B. Hubert and É. Dewailly. 2005. Seawater Temperature, *Gambierdiscus* Spp. Variability and Incidence of Ciguatera Poisoning in French Polynesia. *Harmful Algae* 4: 1053 - 1062.

Chatterjee, C. and R.R. Sarkar. 2009. Multi-Step Polynomial Regression Method to Model and Forecast Malaria Incidence. *Plos One* 4: E4726.

Checkley, W., L.D. Epstein, R.H. Gilman, D. Figueroa, R.I. Cama, J.A. Patz and R.E. Black. 2000. Effects of El Nino and Ambient Temperature on Hospital Admissions for Diarrhoeal Diseases in Peruvian Children. *The Lancet* 355: 442 - 450.

Chiu, M., P.C. Austin, D.G. Manuel and J.V. Tu. 2010. Comparison of Cardiovascular Risk Profiles among Ethnic Groups Using Population Health Survey between 1996 and 2007. *CMAJ* 182: E301 - E310.

Chung, J.-Y., Y. Honda, Y.-C. Hong, X.-C. Pan, Y.-L. Gui and H. Kim. 2009. Ambient Temperature and Mortality: An International Study in Four Capital Cities of East Asia. *Science of the Total Environment* 408: 390 - 396.

Codeço, C.T., S. Lele, M. Pascual, M. Bouma and A.I. Ko. 2008. A Stochastic Model for Ecological Systems with Strong Nonlinear Response to Environmental Drivers: Application to Two Water-Borne Diseases. *Journal of the Royal Society Interface* 5: 247 - 252.

Coleman, M., M. Coleman, M.L.H. Mabaso, A.M. Mabuza, G. Kok, M. Coetzee and D.N. Durrheim. 2010. Household and Microeconomic Factors Associated with Malaria in Mpumalanga, South Africa. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 104: 143 - 147.

Craig, M.H., I. Kleinschmidt, D.L. Sueur and B.L. Sharp. 2004a. Exploring 30 Years of Malaria Case Data in Kwazulu-Natal, South Africa: Part II. The Impact of Non-Climatic Factors. *Tropical Medicine and International Health* 9: 1258 - 1266.

Craig, M.H., I. Kleinschmidt, J.B. Nawn, D.L. Sueur and B.L. Sharp. 2004b. Exploring 30 Years of Malaria Case Data Kwazulu-Natal, South Africa: Part I. The Impact of Climatic Factors. *Tropical Medicine and International Health* 9: 1247 - 1257.

Cunningham-Myrie, C., M. Reid and T.E. Forrester. 2008. A Comparative Study of the Quality and Availability of Health Information Used to Facilitate Cost Burden Analysis of Diabetes and Hypertension in the Caribbean. *West Indian Medical Journal* 57: 383 - 392.

Daniels, M.J., F. Dominici, J.M. Samet and S.L. Zeger. 2000. Estimating Particulate Matter-Mortality Dose-Response Curves and Threshold Levels:An Analysis of Daily Time-Series for the 20 Largest US Cities. *American Journal of Epidemiology* 152: 397 - 406.

Davis, R.E., P.C. Knappenberger, P.J. Michaels and W.M. Novicoff. 2004. Seasonality of Climate-Human Mortality Relationships in US Cities and Impacts of Climate Change. *Climate Research* 26: 61 - 76.

Degallier, N., C. Favier, C. Menkes, N. Lengaigne, W.M. Ramalho, R. Souza, J. Servain and J.-P. Boulanger. 2010. Toward an Early Warning System for Dengue Prevention: Modeling Climate Impact on Dengue Transmission. *Climatic Change* 98: 581 - 592.

Dev, V. and A.P. Dash. 2007. Short Communication: Rainfall and Malaria Transmission in North-Eastern India. *Annals of Tropical Medicine & Parasitology* 101: 457 - 459.

Díaz, J., R. García, F.V.d. Castro, E. Hernández, C. López and A. Otero. 2002. Effects of Extremely Hot Days on People Older Than 65 Years in Seville (Spain) from 1986 - 1997. *International Journal of Biometeorology* 46: 145 - 149.

Ebi, K.L., N.D. Lewis and C. Corvalan. 2006. Climate Variability and Change and Their Potential Health Effects in Small Island States: Information for Adaptation Planning in the Health Sector. *Environmental Health Perspectives* 114: 1957 - 1963.

Ebi, K.L., J. Balbus, P.L. Kinney, E. Lipp, D. Mills, M.S. O'Neill and M. Wilson. 2008. 'Effects of Global Change on Human Health.' In *Analyses of the Effects of Global Change on Human Health and Welfare and Human Systems: A Report by the U.S. Climate Change Science Program and the Subcommittee on Global Change Research* edited by J.L. Gamble, K.L. Ebi, A.E. Grambsch, F.G. Sussman and T.J. Wilbanks, 39 - 87. Washington, DC: U.S. Environmental Protection Agency.

Ebi, K.L. 2008a. Adaptation Costs for Climate Change-Related Cases of Diarrhoeal Disease, Malnutrition, and Malaria in 2030. *Globalization and Health* 4: 9.

Epstein, P. 2010. The Ecology of Climate Change and Infectious Diseases: Comment. *Ecology* 91: 925 - 928.

Faust, M.A. 2009. *Ciguatera-Causing Dinoflagellates in a Coral-Reef-Mangrove Ecosystem, Belize*, National Museum of Natural History, Smithsonian Institution, Washington, DC.

Flannery, B., M.M. Pereira, L. de Freitas Velloso, C. de Castro Carvalho, L.G. de Codes, G. de Saboia Orrico, C.M.R. Dourado, L.W. Riley, M.G. Reis and A.I. Ko. 2001. Referral Pattern of Leptospirosis Cases During a Large Urban Epidemic of Dengue. *American Journal of Tropical Medicine and Hygiene* 65: 657 - 663.

Fleming, L.E., K. Broad, A. Clement, E. Dewailly, S. Elmir, A. Knap, S.A. Pomponi, S. Smith, H.S. Gabriele and P. Walsh. 2006. Oceans and Human Health: Emerging Public Health Risks in the Marine Environment. *Marine Pollution Bulletin* 53: 545 - 560.

Fleming, L.E. 2010. Ciguatera Fish Poisoning. Woods Hole Oceanographic Institution. Available At: http://www.whoi.edu/science/B/redtide/illness/ciguatera_fish_poisoning.html Accessed On: 13 July 2010.

Focks, D.A. and R. Barrera. 2006. *Dengue Transmission Dynamics: Assesssment and Implications for Control*, World Health Organization, Geneva.

Fuller, D.O., A. Troyo and J.C. Beier. 2009. El Niño Southern Oscillation and Vegetation Dynamics as Predictors of Dengue Fever Cases in Costa Rica. *Environmental Research Letters* 4: 8.

Gething, P.W., D.L. Smith, A.P. Patil, A.J. Tatem, R.W. Snow and S.I. Hay. 2010. Climate Change and the Global Malaria Recession. *Nature* 465: 342 - 346.

Gilbert, M. and R. Brindle. 2009. El Niño and Variations in the Prevalence of *Plasmodium Vivax* and *P. Falciparum* in Vanuatu. *Transactions of the Royal Society of Tropical Medical and Hygiene* 103: 1285 -1287.

Githeko, A.K., S.W. Lindsay, U.E. Confalonieri and J.A. Patz. 2000. Climate Change and Vector-Borne Diseases: A Regional Analysis. *Bulletin of The World Health Organization* 78: 1136 - 1147.

Gosling, S.N., J.A. Lowe, G.R. McGregor, M. Pelling and B.D. Malamud. 2009. Associations between Elevated Atmospheric Temperature and Human Mortality: A Critical Review of the Literature. *Climatic Change* 92: 299 - 341.

Hajat, S., B. Armstrong, M. Baccini, A. Biggeri, L. Bisanti, A. Russo, A. Paldy, B. Menne and T. Kosatsky. 2006. Impact of High Temperatures on Mortality: Is There an Added Heath Wave Effect? *Epidemiology* 17: 632 - 638.

Hajat, S. and T. Kosatky. 2009. Heat-Related Mortality: A Review and Exploration of Heterogeneity. *Journal of Epidemiology and Community Health*

Hales, S., C. Salmond, G.I. Town, T. Kjellstrom and A. Woodward. 2000. Daily Mortality in Relation to Weather and Air Pollution in Christchurch, New Zealand. *Australian and New Zealand Journal of Public Health* 24: 89 - 91.

Hales, S., N.d. Wet, J. Maindonald and A. Woodward. 2002. Potential Effect of Population and Climate Changes on Global Distribution of Dengue Fever: An Empirical Model *The Lancet* 360: 830 - 834.

Hales, S., S.J. Edwards and R.S. Kovats. 2003. 'Impacts on Health of Climate Extremes.' In *Climate Change and Human Health: Risks and Responses* edited by A.J. McMIchael, D.H. Campbell-Lendrum, C.F. Corvalán, K.L. Ebi, A.K. Githeko, J.D. Scheraga and A. Woodward, 79 - 102. Geneva: World Health Organization.

Halide, H. and P. Ridd. 2008. A Predictive Model for Dengue Hemorrhagic Fever Epidemics. *International Journal of Environmental Health Research* 18: 253 -265.

Harrison, L.H., C.L. Trotter and M.E. Ramsay. 2009. Global Epidemiology of Meningococcal Disease. *Vaccine* 27S: B51 - B63.

Hashizume, M., Y. Wagatsuma, T. Hayashi, S.K. Saha, K. Streatfield and M. Yunus. 2009. The Effect of Temperature on Mortality in Rural Bangladesh - a Population-Based Time-Series Study. *International Journal of Epidemiology* 38: 1689 - 1697.

HEI International Scientific Versight Committee. 2004. *Health Effects of Outdoor Air Pollution in Developing Countries of Asia: A Literature Review. Special Report 15.*, HEI Special Report 15. Health Effects Institute, Boston.

Hopp, M.J. and J.A. Foley. 2001. Global-Scale Relationships between Climate and the Dengue Fever Vector, *Aedes Aegypti*. *Climatic Change* 48: 441 - 463.

Hopp, M.J. and J.A. Foley. 2003. Worldwide Fluctuations in Dengue Fever Cases Related to Climate Variability. *Climate Research* 25: 85 - 94.

Hoshen, M.B. and A.P. Morse. 2004. A Weather-Driven Model of Malaria Transmission. *Malaria Journal* 3: 32.

Hu, W., A. Clements, G. Williams and S. Tong. 2010. Dengue Dever and El Niño/Southern Oscillation in Queensland, Australia: A Time Series Predictive Model. *Occupational and Environmental Medicine* 67: 307 - 311.

Hurtado-Díaz, M., H. Riojas-Rodríguez, S.J. Rothenberg, H. Gomez-Dantés and E. Ciguentes. 2007. Short Communication: Impact of Climate Variability on the Incidence of Dengue in Mexico. *Tropical Medicine and International Health* 12: 1327 - 1337.

IPCC. 2007. *Climate Change 2007: Synthesis Report*, Valencia.

Jansen, C.C. and N.W. Beebe. 2010. The Dengue Vector *Aedes Aegypti*: What Comes Next. *Microbes and Infection* 12: 272 - 279.

Jaykus, L.-A., M. Woolridge, J.M. Frank, M. Miraglia, A. McQuatters-Gollop, C. Tirado, R. Clarke and M. Friel. 2008. *Climate Change: Implications for Food Safety*, Food and Agriculture Organization of the United Nations (FAO), Rome.

Jetten, T.H. and D.A. Focks. 1997. Potential Changes in the Distribution of Dengue Transmission under Climate Warming. *American Journal of Tropical Medicine and Hygiene* 57: 285 - 297.

Johansson, M.A., F. Dominici and G.E. Glass. 2009a. Local and Global Effects of Climate on Dengue Transmission in Puerto Rico. *PLoS Neglected Tropical Diseases* 3: e382.

Johansson, M.A., D.A.T. Cummings and G.E. Glass. 2009b. Multiyear Climate Variability and Dengue - El Niño Southern Oscillation, Weather, and Dengue Incidence in Puerto Rico, Mexico, and Thailand: A Longitudinal Data Analysis. *PLoS Medicine* 6: e1000168.

Jury, M.R. and A.D. Kanemba. 2007. A Climate-Based Model for Malaria Prediction in Southeastern Africa. *South African Journal of Science* 103: 57 - 62.

Jury, M.R. 2008. Climate Influence on Dengue Epidemics in Puerto Rico. *International Journal of Environmental Health Research* 18: 323 - 334.

Kolivras, K.N. 2010. Changes in Dengue Risk Potential in Hawaii, USA, Due to Climate Variability and Change. *Climate Research* 42: 1 - 11.

Kovats, R.S., M.J. Bouma and A. Haines. 1999. *El Niño and Health*, WHO/SDE/PHE/99.4. World Health Organization: Sustainable Development and Health Environments, Geneva.

Kovats, R.S. and S. Hajat. 2007. Heat Stress and Public Health: A Critical Reivew. *Annual Review of Public Health* 29: 41 - 55.

Kovats, S., K.L. Ebi and B. Menne. 2003a. 'Air Pollution.' In *Methods of Assessing Human Vulnerability and Public Health Adaptation to Climate Change: Health and Global Environmental Change Series No. 1* edited by 59 - 62. Copenhagen: World Health Organization.

Kovats, S., K.L. Ebi and B. Menne. 2003b. 'Direct Effects of Heat and Heat-Waves.' In *Methods of Assessing Human Vulnerability and Public Health Adaptation to Climate Change: Health and Global Environmental Change Series No. 1* edited by 54 - 58. Copenhagen: World Health Organization.

Kovats, S., K.L. Ebi and B. Menne. 2003c. *Methods of Assessing Human Vulnerability and Public Health Adaptation to Climate Change*, World Health Organization, Copenhagen.

Kovats, S., K.L. Ebi and B. Menne. 2003d. 'Vector-Borne Diseases.' In *Methods of Assessing Human Vulnerability and Public Health Adaptation to Climate Change: Health and Global Environmental Change Series No. 1* edited by 68 - 84. Copenhagen: World Health Organization.

Kovats, S., K.L. Ebi and B. Menne. 2003e. 'Waterborne and Foodborne Diarrhoeal Disease.' In *Methods of Assessing Human Vulnerability and Public Health Adaptation to Climate Change: Health and Global Environmental Change Series No. 1* edited by 85 - 87. Copenhagen: World Health Organization.

Kuhn, K., D. Campbell-Lendrum, A. Haines and J. Cox. 2005. *Using Climate to Predict Infectious Disease Epidemics*, World Health Organization, Geneva.

Lafferty, K.D. 2009. The Ecology of Climate Change and Infectious Diseases. *Ecology* 90: 888 - 900.

Lafferty, K.D. 2010. The Ecology of Climate Change and Infectious Diseases: Reply. *Ecology* 91: 928 - 929.

Lama, J.R., C.R. seas, R. León-Barúa, E. Gotuzzo and R.B. Sack. 2004. Environmental Temperature, Cholera, and Acute Diarrhoea in Adults in Lima, Peru. *Journal of Health, Population, and Nutrition* 22: 399 - 403.

Leonardo, L.R., P.T. Rivera, B.A. Crisostommo, J.N. Saroll, N.C. Bantayan, W.U. Tiu and N.R. Bergquist. 2005. A Study of the Environmental Determinants of Malaria and Schistosomiasis in the Philippines Using Remote Sensing and Geographic Information Systems. *Parassitologia* 47: 105 - 114.

Levett, P.N. 2001. Leptospirosis. *Clinical Microbiology Reviews* 14: 296 - 326.

Liang, S., R.C. Spear, E. Seto, A. Hubbard and D. Qiu. 2005. A Multi-Group Model of *Schistosoma Japonicum* Transmission Dynamics and Control: Model Calibration and Control Prediction. *Tropical Medicine and International Health* 10: 263 - 278.

Liang, S., E.Y.W. Seto, J.V. Remais, B. Zhong, C. Yang, A. Hubbard, G.M. Davis, X. Gu, D. Qiu and R.C. Spear. 2007. Environmental Effects on Parasitic Disease

Transmission Exemplified by Schistosomiasis in Western China. *Proceedings of the National Academy of Sciences* 104: 7110 - 7115.

Lindsay, S.W., D.G. Hole, R.A. Hutchinson, S.A. Richards and S.G. Willis. 2010. Assessing the Future Threat from Vivax Malaria in the United Kingdom Using Two Markedly Different Modelling Approaches. *Malaria Journal* 9: 70.

Lloyd, S.J., R.S. Kovats and B.G. Armstrong. 2007. Global Diarrhoea Morbidity, Weather and Climate. *Climate Research* 34: 119 - 127.

Lou, Y. and X.-Q. Zhao. 2010. A Climate-Based Malaria Transmission Model with Structured Vector Population. *SIAM Journal on Applied Mathematics* 70: 2023 - 2044.

Lu, L., H. Lin, L. Tian, W. Yang, J. Sun and Q. Liu. 2009. Time Series Analysis of Dengue Fever and Weather in Guangzhou. *BMC Public Health* 9: 395.

Luber, G. and N. Prudent. 2009. Climate Change and Human Health. *Transactions of the American Clinical and Climatological Association* 120: 113-117.

Luz, P.M., B.V.M. Mendes, C.T. Codeço, C.J. Struchiner and A.P. Galvani. 2008. Time Series Analysis of Dengue Incidence in Rio De Janeiro, Brazil. *American Journal of Tropical Medicine and Hygiene* 79: 933 - 939.

Mangal, T.D., S. Paterson and A. Fenton. 2008. Predicting the Impact of Long-Term Temperature Changes on the Epidemiology and Control of Schistosomiasis: A Mechanistic Model. *Plos One* 3: e1438.

Martens, W.J.M., T.H. Jetten, J. Rotmans and L.W. Niessen. 1995a. Climate Change and Vector-Borne Diseases: A Global Modelling Perspective. *Global Environmental Change* 5: 195 - 209.

Martens, W.J.M., L.W. Niessen, J. Rotmans, T.H. Jetten and A.J. McMichael. 1995b. Potential Impact of Global Climate Change on Malaria Risk. *Environmental Health Perspectives* 103: 458 - 464.

Martens, W.J.M. 1997. Climate Change, Thermal Stress and Mortality Changes. *Social Science & Medicine* 46: 331 - 344.

Martens, W.J.M., T.H. Jetten and D.A. Focks. 1997. Sensitivity of Malaria, Schistosomiasis and Dengue to Global Warming. *Climatic Change* 35: 145 - 156.

McMichael, A.J., D. Campbell-Lendrum, S. Kovats, S. Edwards, P. Wilkinson, T. Wilson, R. Nicholls, S. hales, F. Tanswer, D.L. Sueur, M. Schlesinger and N. Andronova. 2004. 'Global Climate Change.' In *Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attributable Fo Selected Risk Factors* (2), edited by M. Ezzati, A.D. Lopez, A. Rodgers and C.J.L. Murray, 1543 - 1649. Geneva: World Health Organization.

Medina, D.C., S.E. Findley, B. Guindo and S. Doumbia. 2007. Forecasting Non-Stationary Diarrhea, Acute Respiratory Infection, and Malaria Time-Series in Niono, Mali. *Plos One* 2: e1181.

Medina-Ramón, M. and J. Schwartz. 2007. Temperature, Temperature Extremes, and Mortality: A Study of Acclimatisation and Effect Modification in 50 US Cities. *Occupational and Environmental Medicine* 64: 827 - 833.

Molesworth, A.M., L.E. Cuevas, S.J. Connor, A.P. Morse and M.C. Thomson. 2003. Environmental Risk and Meningitis Epidemics in Africa. *Emerging Infectious Diseases* 9: 1287 - 1293.

Onozuka, D., M. Hashizume and A. Hagihara. 2010. Effects of Weather Variability on Infectious Gastroenteritis. *Epidemiology and Infection* 138: 236 - 243.

Ooi, E.-E. and D.J. Gubler. 2009. Global Spread of Epidemic Dengue: The Influence of Environmental Change. *Future Virology* 4: 571 - 580.

Ostfeld, R.S. 2009. Climate Change and the Distribution and Intensity of Infectious Diseases. *Ecology* 90: 903 - 905.

Paaijmans, K.P., A.F. Read and M.B. Thomas. 2009. Understanding the Link between Malaria Risk and Climate. *Proceedings of the National Academy of Sciences* 106: 13844 - 13849.

Palmgren, H. 2009. Meningococcal Disease and Climate. *Global Health Action* 2:

Pascual, M. and M.J. Bouma. 2009. Do Rising Temperatures Matter? *Ecology* 90: 906 - 912.

Patz, J.A., A.K. Githeko, J.P. McCarty, S. Hussein, U. Confalonieri and N.d. Wet. 2003. 'Climate Change and Infectious Diseases.' In *Climate Change and Human Health: Risks and Responses* edited by A.J. McMIchael, D.H. Campbell-Lendrum, C.F. Corvalán, K.L. Ebi, A.K. Githeko, J.D. Scheraga and A. Woodward, 103 - 132. Geneva: World Health Organization.

Prospero, J.M. and P.J. Lamb. 2003. African Droughts and Dust Transport to the Caribbean: Climate Change Implications. *Science* 302: 1024 - 1027.

Rey, G., E. Jougla, A. Fouillet, G. Pavillon, P. Bessemoulin, P. Frayssinet, J. Clavel and D. Hémon. 2007. The Impact of Major Heat Waves on All-Cause and Cause-Specific Mortality in France from 1971 - 2003. *International Archives of Occupational and Environmental Health* 80: 615 - 626.

Roberts, L. 2008. An Ill Wind, Bringing Meningitis. *Science* 320: 1710 - 1715.

Ruiz, D., G. Poveda, I.D. Vélez, M.L. Quiñones, G.L. Rúa, L.E. Velásquez and J.S. Zuluaga. 2006. Modelling Entomological-Climate Interactions of *Plasmodium Falciparum* Malaria Transmission in Two Colombian Endemic-Regions: Contributions to a National Malaria Early Warning System. *Malaria Journal* 5: 66.

Saez, M., J. Sunyer, J. Castellsagué, C. Murillo and J.M. Antó. 1995. Relationship between Weather Temperature and Mortality: A Time Series Analysis Approach in Barcelona. *International Journal of Epidemiology* 24: 576 - 582.

Sheridan, S.C., A.J. Kalkstein and L.S. Kalkstein. 2009. Trends in Heat-Related Mortality in the United States, 1975 - 2004. *Natural Hazards* 50: 145 - 160.

Singh, R.B.K., S. Hales, N.d. Wet, R. Raj, M. Hearnden and P. Weinstein. 2001. The Influence of Climate Variation and Change on Diarrheal Disease in the Pacific Islands. *Environmental Health Perspectives* 109: 155 - 159.

Slack, A.T., M.L. Symonds, M.F. Dohnt and L.D. Smythe. 2006. The Epidemiology of Leptospirosis and the Emergence of *Leptospira Borgpetersenii* Serovar Arborea in Queensland, Australia, 1988-2004. *Epidemiology and Infection* 134: 1217 - 1225.

Storck, C.H., D. Postic, I. Lamaury and J.M. Perez. 2008. Changes in Epidemiology of Leptospirosis in 2003-2004, a Two El Niño Southern Oscillation Period, Gueadeloupe Archipelago, French West Indies. *Epidemiology and Infection* 136: 1407 - 1415.

Stratton, L., M.S. O'Neill, M.E. Kruk and M.L. Bell. 2008. The Persistent Problem of Malaria: Addressing the Fundamental Causes of a Global Killer. *Social Science & Medicine* 67: 854 - 862.

Sutherst, R.W. 2004. Global Change and Human Vulnerability to Vector-Borne Diseases. *Clinical Microbiology Reviews* 17: 136 - 173.

Tabachnick, W.J. 2010. Challenges in Predicting Climate and Environmental Effects on Vector-Borne Disease Episystems in a Changing World. *The Journal of Experimental Biology* 213: 946 - 954.

Tassinari, W.S., D.C.P. Pelligrini, C.B.P. Sá, R.B. Reis, A.I. Ko and M.S. Carvalho. 2008. Detection and Modelling of Cast Clusters for Urban Leptospirosis. *Tropical Medicine and International Health* 13: 503 - 512.

Teklehaimanot, H.D., J. Schwartz, A. Teklehaimanot and M. Lipsitch. 2004. Weather-Based Prediction of *Plasmodium Falciparum* Malaria in Epidemic-Prone Regions of Ethiopia II. Weather-Based Prediction Systems Perform Comparably to Early Detection Systems in Identifying Time for Interventions. *Malaria Journal* 3: 44.

Tester, P.A., R.L. Feldman, A.W. Nau, S.R. Kibler and R.W. Litaker. 2010. Ciguatera Fish Poisoning and Sea Surface Temperatures in the Caribbean Sea and the West Indies. *Toxicon* In Press: 13.

Thomson, M.C., S.J. Mason, T. Phindela and S.J. Connor. 2005. Use of Rainfall and Sea Surface Temperature Monitoring for Malaria Early Warning in Botswana. *American Journal of Tropical Medicine and Hygiene* 73: 214 - 221.

Thomson, M.C., F.J. Doblas-Reyes, S.J. Mason, R. Hagedorn, S.J. Connor, T. Phindela, A.P. Morse and T.N. Palmer. 2006a. Malaria Early Warnings Based on Season Climate Forecasts from Multi-Model Ensembles. *Nature* 439: 576 - 579.

Thomson, M.C., A.M. Molesworth, M.H. Djingarey, K.R. Yameogo, F. Belanger and L.E. Cuevas. 2006b. Potential of Environmental Models to Predict Meningitis Epidemics in Africa. *Tropical Medicine and International Health* 11: 781 - 788.

Tol, R.S.J. 2008. Climate, Development and Malaria: An Application of *Fund*. *Climatic Change* 88: 21 - 34.

Tosteson, T.R. 2004. Caribbean Ciguatera: A Changing Paradigm. *Revista de Biologia Tropical* 52: 5.

Tseng, W.-C., C.-C. Chen, C.-C. Chang and Y.-H. Chu. 2009. Estimating the Economic Impacts of Climate Change on Infectious Diseases: A Case Study on Dengue Fever in Taiwan. *Climatic Change* 91: 123 - 140.

UNDP/World Bank Energy Sector Management Assistance Programme. 2004. *South Asia Urban Air Quality Management Briefing Note No. 11: Health Impacts of Outdoor Air Pollution.* UNDP/World Bank Energy Sector Management Assistance Programme. http://www.undp.org.cu/eventos/aprotegidas/Briefing_Note_No_11.pdf.

van Lieshout, M., R.S. Kovats, M.T.J. Livermore and P. Martens. 2004. Climate Change and Malaria: Analysis of the Sres Climate and Socio-Economic Scenarios. *Global Environmental Change* 14: 87 - 99.

Vaneckova, P., P.J. Beggs, R.J.d. Dear and K.W.J. McCracken. 2008. Effect of Temperature on Mortality During the Six Warmer Months in Sydney, Australia, between 1993 and 2004. *Environmental Research* 108: 361 - 369.

Victoriano, A.F.B., L.D. Smythe, N. Gloriani-Barzaga, L.L. Cavinta, T. Kasai, K. Limpakarnjanarat, B.L. Ong, G. gongal, J. Hall, C.A. Coulombe, Y. Yanagihara, S.-i. Yoshida and B. Adler. 2009. Leptospirosis in the Asia Pacific Region. *BMC Infectious Diseases* 9: 147.

Wandiga, S.O., M. Opondo, D. Olago, A. Githeko, F. Githui, M. Marshall, T. Downs, A. Opere, C. Oludhe, G.O. Ouma, P.Z. Yanda, R. Kangalawe, R. Kabumbuli, J. Kathuri, E. Apindi, L. Olaka, L. Ogallo, P. Mugambi, R. Sigalla, R. Nanyunja, T. Baguma and P. Achola. 2010. Vulnerability to Epidemic Malaria in the Highlands of Lake Victoria Basin: The Role of Climate Change/Variability, Hydrology and Socio-Economic Factors. *Climatic Change* 99: 473 - 497.

Webber, R. 2005. *Communicable Disease Epidemiology and Control.* CABI Publishing.

WHO. 2003a. *Climate Change and Human Health: Risks and Responses*, World Health Organization, Geneva.

WHO. 2003b. *Human Leptospirosis: Guidance for Diagnosis, Surveillance and Control*, World Health Organization, Geneva.

Wiwanitkit, V. 2006. Correlation between Rainfall and the Prevalence of Malaria in Thailand. *Journal of Infection* 52: 227 - 230.

Wu, P.-C., H.-R. Guo, S.-C. Lung, C.-Y. Lin and H.-J. Su. 2007. Weather as an Effective Predictor for Occurrence of Dengue Fever in Taiwan. *Acta Tropica* 103: 50 - 57.

Yaka, P., B. Sultan, H. Broutin, S. Janicot, S. Philippon and N. Fourquet. 2008. Relationships between Climate and Year-to-Year Variability in Meningitis Outbreaks: A Case Study in Burkina Faso and Niger. *International Journal of Health Geographics* 7: 34.

Yang, G. 2006. Potential Impact of Climate Change and Water Resources Development on the Epidemiology of Schistosomiasis in China. PhD, Swiss Tropical Institute, Universität Basel Available At: http://edoc.unibas.ch/478/1/DissB_7523.pdf. Accessed On: 26 July 2010.

Yang, H.M., M.L.G. Macoris, K.C. Galvani, M.T.M. Andrighetti and D.M.V. Wanderley. 2009a. Assessing the Effects of Temperature on Dengue Transmission. *Epidemiology and Infection* 137: 1179 - 1187.

Yang, H.M., M.L.G. Macoris, K.C. Galvani, M.T.M. Andrighetti and D.M.V. Wanderley. 2009b. Assessing the Effects of Temperature on the Population of *Aedes Aegypti*, the Vector of Dengue. *Epidemiology and Infection* 137: 1188 - 1202.

Zanobetti, A. and J. Schwartz. 2008. Temperature and Mortality in Nine US Cities. *Epidemiology* 19: 563 - 570.

Zhou, X.-N., G.-J. Yang, K. Yang, X.-H. Wang, Q.-B. Hong, L.-P. Sun, J.B. Malone, T.K. Kristensen, N.R. Bergquist and J. Utzinger. 2008. Potential Impact of Climate Change on Schistosomiasis Transmission in China. *American Journal of Tropical Medicine and Hygiene* 78: 188 - 194.

APPENDIX I
MALARIA LITERATURE SEARCH PAPERS

[1]   Abellana, Rosa, et al. 2008. Spatio-Seasonal Modeling of the Incidence Rate of Malaria in Mozambique. *Malaria Journal* 7: 228.

[2]   Bhattacharya, Sumana, et al. 2006. Climate Change and Malaria in India. *Current Science* 90: 369 - 375.

[3]   Bi, Peng, et al. 2003. Climatic Variables and Transmission of Malaria: A 12-Year Data Analysis in Shuchen County, China. *Public Health Reports* 118: 65 - 71.

[4]   Chatterjee, Chandrajit and Ram Rup Sarkar. 2009. Multi-Step Polynomial Regression Method to Model and Forecast Malaria Incidence. *Plos One* 4: E4726.

[5]   Coleman, Marlize, et al. 2010. Household and Microeconomic Factors Associated with Malaria in Mpumalanga, South Africa. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 104: 143 - 147.

[6]   Craig, M.H., et al. 2004. Exploring 30 Years of Malaria Case Data Kwazulu-Natal, South Africa: Part I. The Impact of Climatic Factors. *Tropical Medicine and International Health* 9: 1247 - 1257.

[7]   Craig, M.H., et al. 2004. Exploring 30 Years of Malaria Case Data in Kwazulu-Natal, South Africa: Part Ii. The Impact of Non-Climatic Factors. *Tropical Medicine and International Health* 9: 1258 - 1266.

[8]   Dev, V. and A.P. Dash. 2007. Short Communication: Rainfall and Malaria Transmission in North-Eastern India. *Annals of Tropical Medicine & Parasitology* 101: 457 - 459.

[9]   Epstein, Paul. 2010. The Ecology of Climate Change and Infectious Diseases: Comment. *Ecology* 91: 925 - 928.

[10] Gething, Peter W., et al. 2010. Climate Change and the Global Malaria Recession. *Nature* 465: 342 - 346.

[11] Gilbert, M. and R. Brindle. 2009. El Niño and Variations in the Prevalence of *Plasmodium Vivax* and *P. Falciparum* in Vanuatu. *Transactions of the Royal Society of Tropical Medical and Hygiene* 103: 1285 -1287.

[12] Hoshen, Moshe B. and Andrew P. Morse. 2004. A Weather-Driven Model of Malaria Transmission. *Malaria Journal* 3: 32.

[13] Jury, M.R. and A.D. Kanemba. 2007. A Climate-Based Model for Malaria Prediction in Southeastern Africa. *South African Journal of Science* 103: 57 - 62.

[14] Lafferty, Keving D. 2009. The Ecology of Climate Change and Infectious Diseases. *Ecology* 90: 888 - 900.

[15] Lafferty, Kevin D. 2010. The Ecology of Climate Change and Infectious Diseases: Reply. *Ecology* 91: 928 - 929.

[16] Leonardo, L.R., et al. 2005. A Study of the Environmental Determinants of Malaria and Schistosomiasis in the Philippines Using Remote Sensing and Geographic Information Systems. *Parassitologia* 47: 105 - 114.

[17] Lindsay, Steven W., et al. 2010. Assessing the Future Threat from Vivax Malaria in the United Kingdom Using Two Markedly Different Modelling Approaches. *Malaria Journal* 9: 70.

[18] Lou, Yijun and Xiao-Qiang Zhao. 2010. A Climate-Based Malaria Transmission Model with Structured Vector Population. *SIAM Journal on Applied Mathematics* 70: 2023 - 2044.

[19] Moreno, Ana Rosa. 2006. Climate Change and Human Health in Latin America: Drivers, Effects, and Policies. *Regional Environmental Change* 6: 157 - 164.

[20] Ostfeld, Richard S. 2009. Climate Change and the Distribution and Intensity of Infectious Diseases. *Ecology* 90: 903 - 905.

[21] Paaijmans, Krijn P., et al. 2009. Understanding the Link between Malaria Risk and Climate. *Proceedings of the National Academy of Sciences* 106: 13844 - 13849.

[22] Parham, Paul Edward and Edwin Michael. 2010. Modeling the Effects of Weather and Climate Change on Malaria Transmission. *Environmental Health Perspectives* 118: 620 - 626.

[23] Pascual, Mercedes and Menno J. Bouma. 2009. Do Rising Temperatures Matter? *Ecology* 90: 906 - 912.

[24] Ruiz, Daniel, et al. 2006. Modelling Entomological-Climate Interactions of *Plasmodium Falciparum* Malaria Transmission in Two Colombian Endemic-Regions: Contributions to a National Malaria Early Warning System. *Malaria Journal* 5: 66.

[25] Stratton, Leanne, et al. 2008. The Persistent Problem of Malaria: Addressing the Fundamental Causes of a Global Killer. *Social Science & Medicine* 67: 854 - 862.

[26] Teklehaimanot, Hailay D., et al. 2004. Weather-Based Prediction of *Plasmodium Falciparum* Malaria in Epidemic-Prone Regions of Ethiopia Ii.

Weather-Based Prediction Systems Perform Comparably to Early Detection Systems in Identifying Time for Interventions. *Malaria Journal* 3: 44.

[27] Thomson, Madeleine C., et al. 2005. Use of Rainfall and Sea Surface Temperature Monitoring for Malaria Early Warning in Botswana. *American Journal of Tropical Medicine and Hygiene* 73: 214 - 221.

[28] Thomson, M.C., et al. 2006. Malaria Early Warnings Based on Season Climate Forecasts from Multi-Model Ensembles. *Nature* 439: 576 - 579.

[29] Tol, Richard S. J. 2008. Climate, Development and Malaria: An Application of *Fund*. *Climatic Change* 88: 21 - 34.

[30] van Lieshout, M. , et al. 2004. Climate Change and Malaria: Analysis of the Sres Climate and Socio-Economic Scenarios. *Global Environmental Change* 14: 87 - 99.

[31] Wandiga, Shem O., et al. 2010. Vulnerability to Epidemic Malaria in the Highlands of Lake Victoria Basin: The Role of Climate Change/Variability, Hydrology and Socio-Economic Factors. *Climatic Change* 99: 473 - 497.

[32] Wiwanitkit, Viroj. 2006. Correlation between Rainfall and the Prevalence of Malaria in Thailand. *Journal of Infection* 52: 227 - 230.

[1]   Amarakoon, Dharmaratne, et al. 2008. Dengue Epidemics in the Caribbean-Temperature Indices to Gauge the Potential for Onset of Dengue. *Mitigation and Adaptation Strategies for Global Change* 13: 341 - 357.

[2]   Amarakoon, AMD, et al. 2004. Dengue Epidemics - Its Association with Precipitation and Temperature, and Its Seasonality in Some Caribbean Countries. *West Indian Medical Journal* 53 60.

[3]   Barbazan, P., et al. 2010. Modelling the Effect of Temperature on Transmission of Dengue. *Medical and Veterinary Entomology* 24: 66 - 73.

[4]   Brunkard, Joan M., et al. 2008. Assessing the Roles of Temperature, Precipitation, and Enso in Dengue Re-Emergence on the Texas-Mexico Border Region. *salud pública de méxico* 50: 227 - 234.

[5]   Degallier, Nicolas, et al. 2010. Toward an Early Warning System for Dengue Prevention: Modeling Climate Impact on Dengue Transmission. *Climatic Change* 98: 581 - 592.

[6]   Focks, Dana A. and Roberto Barrera. *Dengue Transmission Dynamics: Assesssment and Implications for Control*, World Health Organization, Geneva.

[7]   Fuller, D.O., et al. 2009. El Niño Southern Oscillation and Vegetation Dynamics as Predictors of Dengue Fever Cases in Costa Rica. *Environmental Research Letters* 4: 8.

[8]   Hales, Simon, et al. 2002. Potential Effect of Population and Climate Changes on Global Distribution of Dengue Fever: An Empirical Model *The Lancet* 360: 830 - 834.

9]   Halide, Halmar and Peter Ridd. 2008. A Predictive Model for Dengue Hemorrhagic Fever Epidemics. *International Journal of Environmental Health Research* 18: 253 -265.

[10]   Hopp, Marianne J. and Jonathan A. Foley. 2001. Global-Scale Relationships between Climate and the Dengue Fever Vector, *Aedes Aegypti*. *Climatic Change* 48: 441 - 463.

[11]   Hopp, Marianne J. and Jonathan A. Foley. 2003. Worldwide Fluctuations in Dengue Fever Cases Related to Climate Variability. *Climate Research* 25: 85 - 94.

[12]   Hu, Wenbiao, et al. 2010. Dengue Dever and El Niño/Southern Oscillation in Queensland, Australia: A Time Series Predictive Model. *Occupational and Environmental Medicine* 67: 307 - 311.

[13]   Hurtado-Díaz, M., et al. 2007. Short Communication: Impact of Climate Variability on the Incidence of Dengue in Mexico. *Tropical Medicine and International Health* 12: 1327 - 1337.

[14] Jansen, Cassie C. and Nigel W. Beebe. 2010. The Dengue Vector *Aedes Aegypti*: What Comes Next. *Microbes and Infection* 12: 272 - 279.

[15] Johansson, Michael A., et al. 2009. Local and Global Effects of Climate on Dengue Transmission in Puerto Rico. *PLoS Neglected Tropical Diseases* 3: e382.

[16] Johansson, Michael A., et al. 2009. Multiyear Climate Variability and Dengue - El Niño Southern Oscillation, Weather, and Dengue Incidence in Puerto Rico, Mexico, and Thailand: A Longitudinal Data Analysis. *PLoS Medicine* 6: e1000168.

[17] Jury, Mark R. 2008. Climate Influence on Dengue Epidemics in Puerto Rico. *International Journal of Environmental Health Research* 18: 323 - 334.

[18] Kolivras, Korine N. 2010. Changes in Dengue Risk Potential in Hawaii, USA, Due to Climate Variability and Change. *Climate Research* 42: 1 - 11.

[19] Kurane, Ichiro. 2009. The Emerging and Forecaseted Effect of Climate Change on Human Health. *Journal of Health Science* 55: 865 - 869.

[20] Lu, Liang, et al. 2009. Time Series Analysis of Dengue Fever and Weather in Guangzhou. *BMC Public Health* 9: 395.

[21] Luz, Paula M., et al. 2008. Time Series Analysis of Dengue Incidence in Rio De Janeiro, Brazil. *American Journal of Tropical Medicine and Hygiene* 79: 933 - 939.

[22] Ooi, Eng-Eong and Duane J. Gubler. 2009. Global Spread of Epidemic Dengue: The Influence of Environmental Change. *Future Virology* 4: 571 - 580.

[23] Rohani, Pejman. 2009. The Link between Dengue Incidence and El Niño Southern Oscillation. *PLoS Medicine* 6: e1000185.

[24] Tseng, Wei-CHun, et al. 2009. Estimating the Economic Impacts of Climate Change on Infectious Diseases: A Case Study on Dengue Fever in Taiwan. *Climatic Change* 91: 123 - 140.

[25] Wu, Pei-Chih, et al. 2007. Weather as an Effective Predictor for Occurrence of Dengue Fever in Taiwan. *Acta Tropica* 103: 50 - 57.

[26] Yang, H.M., et al. 2009. Assessing the Effects of Temperature on Dengue Transmission. *Epidemiology and Infection* 137: 1179 - 1187.

[27] Yang, H.M., et al. 2009. Assessing the Effects of Temperature on the Population of *Aedes Aegypti*, the Vector of Dengue. *Epidemiology and Infection* 137: 1188 - 1202.

APPENDIX III
GASTROENTERITIS LITERATURE SEARCH PAPERS

[1]   Checkley, William, et al. 2000. Effects of El Nino and Ambient Temperature on
      Hospital Admissions for Diarrhoeal Diseases in Peruvian Children. *The Lancet* 355:
      442 - 450.

[2]   Lama, Javier R., et al. 2004. Environmental Temperature, Cholera, and Acute
      Diarrhoea in Adults in Lima, Peru. *Journal of Health, Population, and Nutrition* 22:
      399 - 403.

[3]   Lloyd, Simon J., et al. 2007. Global Diarrhoea Morbidity, Weather and Climate.
      Climate Research 34: 119 - 127.

[4]   Medina, Daniel C., et al. 2007. Forecasting Non-Stationary Diarrhea, Acute
      Respiratory Infection, and Malaria Time-Series in Niono, Mali. *Plos One* 2: e1181.

[5]   Onozuka, D., et al. 2010. Effects of Weather Variability on Infectious Gastroenteritis.
      Epidemiology and Infection 138: 236 - 243.

[6]   Singh, Reena B.K., et al. 2001. The Influence of Climate Variation and Change on
      Diarrheal Disease in the Pacific Islands. *Environmental Health Perspectives* 109:
      155 - 159.

[1]   Mangal, Tara D., et al. 2008. Predicting the Impact of Long-Term Temperature
       Changes on the Epidemiology and Control of Schistosomiasis: A Mechanistic
       Model. *Plos One* 3: e1438.

[2]   Liang, Song, et al. 2005. A Multi-Group Model of *Schistosoma Japonicum*
       Transmission Dynamics and Control: Model Calibration and Control Prediction.
       Tropical Medicine and International Health 10: 263 - 278.

[3]   Liang, Song, et al. 2007. Environmental Effects on Parasitic Disease Transmission
       Exemplified by Schistosomiasis in Western China. *Proceedings of the National
       Academy of Sciences* 104: 7110 - 7115.

[4]   Yang, Guo-Jing, et al. 2006. A Growing Degree-Days Based Time-Series Analysis for
       Prediction of *Schistosoma Japonicum* Transmission in Jiangsu Province, China.
       American Journal of Tropical Medicine and Hygiene 75: 549 - 555.

[5]   Yang, Guojing. 2006. Potential Impact of Climate Change and Water Resources
       Development on the Epidemiology of Schistosomiasis in China. PhD Thesis, Swiss
       Tropical Institute, Universität Basel Available At:
       http://edoc.unibas.ch/478/1/DissB_7523.pdf. Accessed On: 26 July 2010.

[6]   Zhou, Xiao-Nong, et al. 2008. Potential Impact of Climate Change on Schistosomiasis
       Transmission in China. *American Journal of Tropical Medicine and Hygiene* 78:
       188 - 194.

APPENDIX V
LEPTOSPIROSIS LITERATURE SEARCH PAPERS

[1]   Codeço, Claudia Torres, et al. 2008. A Stochastic Model for Ecological Systems with Strong Nonlinear Response to Environmental Drivers: Application to Two Water-Borne Diseases. *Journal of the Royal Society Interface* 5: 247 - 252.

[2]   Slack, A.T., et al. 2006. The Epidemiology of Leptospirosis and the Emergence of *Leptospira Borgpetersenii* Serovar Arborea in Queensland, Australia, 1988-2004. *Epidemiology and Infection* 134: 1217 - 1225.

[3]   Storck, C. Herrmann, et al. 2008. Changes in Epidemiology of Leptospirosis in 2003-2004, a Two El Niño Southern Oscillation Period, Gueadeloupe Archipelago, French West Indies. *Epidemiology and Infection* 136: 1407 - 1415.

[4]   Tassinari, Wagner S., et al. 2008. Detection and Modelling of Cast Clusters for Urban Leptospirosis. *Tropical Medicine and International Health* 13: 503 - 512.

APPENDIX VI
CIGUATERA POISONING LITERATURE SEARCH PAPERS

[1]   Chateau-Degat, Marie-Ludivine, et al. 2005. Seawater Temperature, *Gambierdiscus* Spp. Variability and Incidence of Ciguatera Poisoning in French Polynesia. *Harmful Algae* 4: 1053 - 1062.

[2]   Faust, Maria A. *Ciguatera-Causing Dinoflagellates in a Coral-Reef-Mangrove Ecosystem, Belize*, National Museum of Natural History, Smithsonian Institution, Washington, DC.

[3]   Hales, Simon, et al. 1999. Ciguatera (Fish Poisoning), El Niño, and Pacific Sea Surface Temperatures. *Ecosystem health* 5: 20 - 25.

[4]   Okolodkov, Yuri B., et al. 2007. Seasonal Changes of Benthic and Epiphytic Dinoflagellates in the Veracruz Reef Zone, Gulf of Mexico. *Aquatic Microbial Ecology* 47: 223 - 237.

[5]   Tester, Patricia A., et al. 2010. Ciguatera Fish Poisoning and Sea Surface Temperatures in the Caribbean Sea and the West Indies. *Toxicon* In Press: 13.

[6]   Tosteson, T.R. 2004. Caribbean Ciguatera: A Changing Paradigm. *Revista de Biologia Tropical* 52: 5.

APPENDIX VII

MENINGOCOCCAL MENINGITIS LITERATURE SEARCH PAPERS

[1]   Boyne, Lorna. 2001. Meningococcal Infection. *Nursing Standard* 16: 47 - 55.

[2]   Harrison, Lee H., et al. 2009. Global Epidemiology of Meningococcal Disease. *Vaccine* 27S: B51 - B63.

[3]   Molesworth, Anna M., et al. 2003. Environmental Risk and Meningitis Epidemics in Africa. *Emerging Infectious Diseases* 9: 1287 - 1293.

[4]   Mueller, Judith E. and Bradford D. Gessner. 2010. A Hypothetical Explanatory Model for Meningococcal Meningitis in the African Meningitis Belt. *International Journal of Infectious Diseases* 14: e553 - e559.

[5]   Palmgren, Helena. 2009. Meningococcal Disease and Climate. *Global Health Action* 2:

[6]   Roberts, Leslie. 2008. An Ill Wind, Bringing Meningitis. *Science* 320: 1710 - 1715.

[7]   Sultan, Benjamin, et al. 2005. Climate Drives the Meningitis Epidemics Onset in West Africa. *PLoS Medicine* 2: 0043 - 0049.

[8]   Sultan, B., et al. 2005. Meningitis and Climate in West Africa (Summary). *PLoS Medicine* 2: 0003.

[9]   Thomson, Madeleine C., et al. 2006. Potential of Environmental Models to Predict Meningitis Epidemics in Africa. *Tropical Medicine and International Health* 11: 781 - 788.

[10]   Yaka, Pascal, et al. 2008. Relationships between Climate and Year-to-Year Variability in Meningitis Outbreaks: A Case Study in Burkina Faso and Niger. *International Journal of Health Geographics* 7: 34.

APPENDIX VIII
TEMPERATURE, CLIMATE, RESPIRATORY & CARDIOVASCULAR DISEASE LITERATURE SEARCH PAPERS

[1]   Almeida, Sofia P., et al. 2010. Effects of Apparent Temperature on Daily Mortality in Lisbon and Oporto, Portugal. *Environmental Health* 9: 12.

[2]   Ayres, J.G., et al. 2009. Climate Change and Respiratory Disease: European Respiratory Society Position Statement. *European Respiratory Journal* 34: 295 - 302.

[3]   Basu, Rupa and Jonathan M. Samet. 2002. Relation between Elevated Ambient Temperature and Mortality: A Review of Epidemiologic Evidence. *Epidemiologic Reviews* 24: 190 - 202.

[4]   Bell, Michelle L., et al. 2008. Vulnerability to Heat-Related Mortality in Latin America: A Case-Crossover Study in São Paulo, Brazil, Santiago, Chile and Mexico City, Mexico. *International Journal of Epidemiology* 37: 796 - 804.

[5]   Curriero, Frank C., et al. 2002. Temperature and Mortality in 11 Cities in the Eastern United States. *American Journal of Epidemiology* 155: 80 - 87.

[6]   Chung, Joo-Youn, et al. 2009. Ambient Temperature and Mortality: An International Study in Four Capital Cities of East Asia. *Science of the Total Environment* 408: 390 - 396.

[7]   Davis, Robert E., et al. 2004. Seasonality of Climate-Human Mortality Relationships in Us Cities and Impacts of Climate Change. *Climate Research* 26: 61 - 76.

[8]   Dear, Keith. 2009. Commentary: On Hashizume Et Al.'S 'the Effect of Temperature on Mortality in Rural Bangladesh'. *International Journal of Epidemiology* 38: 1697 - 1699.

[9]   Díaz, J., et al. 2002. Effects of Extremely Hot Days on People Older Than 65 Years in Seville (Spain) from 1986 - 1997. *International Journal of Biometeorology* 46: 145 - 149.

[10] El-Zein, Abbas, et al. 2004. A Time-Series Analysis of Mortality and Air Temperature in Greater Beirut. *Science of the Total Environment* 330: 71 - 80.

[11] Farajzadeh, M. and M. Darand. 2009. Analyzing the Influence of Air Temperature on the Cardiovascular, Respiratory and Stroke Mortality in Tehran. *Iranian Journal of Environmental Health Science & Engineering* 6: 261 - 170.

[12] Josefina, Silvia, et al. 2008. Epidemiology of Asthma Mortality in Cuba and Its Relation to Climate, 1989 - 2003. *MEDICC Review* 10: 14 - 29.

[13] Golden, Jay S., et al. 2008. A Biometeorology Study of Climate and Heat-Related Morbidity in Phoenix from 2001 - 2006. *International Journal of Biometeorology* 52: 471 - 480.

[14] Gosai, Ashmita, et al. 2009. Climate and Respiratory Disease in Auckland, New Zealand. *Australian and New Zealand Journal of Public Health* 33: 521 - 526.

[15] Gosling, Simon N., et al. 2009. Associations between Elevated Atmospheric Temperature and Human Mortality: A Critical Review of the Literature. *Climatic Change* 92: 299 - 341.

[16] Gosling, Simon N., et al. 2009. Climate Change and Heat-Related Mortalty in Six Cities Part 2: Climate Model Evaluation and Projected Impacts from Changes in the Mean and Variability of Temperature with Climate Change. *International Journal of Biometeorology* 53: 31 - 51.

[17] Hales, Simon, et al. 2000. Daily Mortality in Relation to Weather and Air Pollution in Christchurch, New Zealand. *Australian and New Zealand Journal of Public Health* 24: 89 - 91.

[18] Hajat, Shakoor and Tom Kosatky. 2009. Heat-Related Mortality: A Review and Exploration of Heterogeneity. *Journal of Epidemiology and Community Health*

[19] Hajat, Shakoor, et al. 2006. Impact of High Temperatures on Mortality: Is There an Added Heath Wave Effect? *Epidemiology* 17: 632 - 638.

[20] Hashizume, Masahiro, et al. 2009. The Effect of Temperature on Mortality in Rural Bangladesh - a Population-Based Time-Series Study. *International Journal of Epidemiology* 38: 1689 - 1697.

[21] Kalkstein, Laurence S. and J. Scott Greene. 1997. An Evaluation of Climate/Mortality Relationships in Large U.S. Cities and the Possible Impact of a Climate Change. *Environmental Health Perspectives* 105: 84 - 93.

[22] Kim, Ho, et al. 2006. High Temperature, Heat Index and Mortality in 6 Major Cities in South Korea. *Archives of Environmental & Occupational Health* 61: 265 - 270.

[23] Kinney, Patrick L., et al. 2008. Approaches for Estimating Effects of Climate Change on Heat-Related Deaths: Challenges and Opportunities. *Environmental Science & Policy* 11: 87 - 96.

[24] Kovats, R. Sari and Shakoor Hajat. 2007. Heat Stress and Public Health: A Critical Reivew. *Annual Review of Public Health* 29: 41 - 55.

[25] Laaidi, Mohamed, et al. 2006. Temperature-Related Mortality in France, a Comparison between Regions with Different Climates from the Perspective of Global Warming. *International Journal of Biometeorology* 51: 145 - 153.

[26] Martens, W.J.M. 1998. Climate Change, Thermal Stress and Mortality Changes. *Social Science & Medicine* 46: 331 - 344.

[27] Martens, W.J.M. 1998. Health Impacts of Climate Change and Ozone Depletion: An Ecoepidemiologic Modeling Approach. *Environmental Health Perspectives* 106: 241 - 251.

[28] McGeehin, Michael A. and Maria Mirabelli. 2001. The Potential Impacts of Cimate Variability and Change on Temperature-Related Morbidity and Mortality in the United States. *Environmental Health Perspectives* 109: 185 - 189.

[29] McMichael, Anthony J., et al. 2008. International Study of Temperature, Heat and Urban Mortality: The 'Isothurm' Project. *Environmental Epidemiology* 37: 1121 - 1131.

[30] Medina, Daniel C., et al. 2007. Forecasting Non-Stationary Diarrhea, Acute Respiratory Infection, and Malaria Time-Series in Niono, Mali. *Plos One* 2: e1181.

[31] Medina-Ramón, M. and J. Schwartz. 2007. Temperature, Temperature Extremes, and Mortality: A Study of Acclimatisation and Effect Modification in 50 Us Cities. *Occupational and Environmental Medicine* 64: 827 - 833.

[32] Patz, Jonathan A., et al. 2000. The Effects of Changing Weather on Public Health. *Annual Review of Public Health* 21: 271 - 307.

[33] Patz, Jonathan A., et al. 2000. The Potential Health Impacts of Climate Variability and Change for the United States: Executive Summary of the Report of the Health Sector of the U.S. National Assessment. *Environmental Health Perspectives* 108: 367 - 376.

[34] Rey, Grégoire, et al. 2007. The Impact of Major Heat Waves on All-Cause and Cause-Specific Mortality in France from 1971 - 2003. *International Archives of Occupational and Environmental Health* 80: 615 - 626.

[35] Saez, Marc, et al. 1995. Relationship between Weather Temperature and Mortality: A Time Series Analysis Approach in Barcelona. *International Journal of Epidemiology* 24: 576 - 582.

[36] Sheridan, Scott C., et al. 2009. Trends in Heat-Related Mortality in the United States, 1975 - 2004. *Natural Hazards* 50: 145 - 160.

[37] Vaneckova, Pavla, et al. 2008. Effect of Temperature on Mortality During the Six Warmer Months in Sydney, Australia, between 1993 and 2004. *Environmental Research* 108: 361 - 369.

[38] Zanobetti, Antonella and Joel Schwartz. 2008. Temperature and Mortality in Nine Us Cities. *Epidemiology* 19: 563 - 570.