UNITED NATIONS
ECLAC

Economic Commission for Latin America and the Caribbean
Subregional Headquarters for the Caribbean

LIMITED
LC/CAR/L.320
22 October 2011
ORIGINAL: ENGLISH

AN ECONOMIC ASSESSMENT OF THE IMPACT OF CLIMATE CHANGE ON THE HEALTH SECTOR IN MONTSERRAT

This document has been reproduced without formal editing

Acknowledgement

The Economic Commission for Latin America and the Caribbean (ECLAC) Subregional Headquarters for the Caribbean wishes to acknowledge the assistance of Dominic Moran, consultant, in the preparation of this report.

Executive Summary

Climate change has the potential to impact on global, regional, and national disease burdens both directly and indirectly. Projecting and valuing these health impacts is important not only in terms of assessing the overall impact of climate change on various parts of the world, but also in terms of ensuring that national and regional decision-making institutions have access to the data necessary to guide investment decisions and future policy design. This report contributes to the research focusing on projecting and valuing the impacts of climate change in the Caribbean by projecting the climate change-induced excess disease burden for two climate change scenarios in Montserrat for the period 2010 - 2050, and by estimating the monetary value associated with this excess disease burden.

The diseases initially considered in this report are variety of vector and water-borne impacts and other miscellaneous conditions; specifically, malaria, dengue fever, gastroenteritis/diarrheal disease, schistosomiasis, leptospirosis, ciguatera poisoning, meningococcal meningitis, and cardio-respiratory diseases. Disease projections were based on derived baseline incidence and mortality rates, available dose-response relationships found in the published literature, climate change scenario population projections for the A2 and B2 IPCC SRES scenario families, and annual temperature and precipitation anomalies as projected by the downscaled ECHAM4 global climate model.

Monetary valuation was based on a transfer value of statistical life approach with a modification for morbidity. Using discount rates of 1%, 2% and 4%, results show mean annual costs (morbidity and mortality) ranges of $0.61 million (in the B2 scenario, discounted at 4% annually) – $1 million (in the A2 scenario, discounted at 1% annually) for Montserrat. These costs are compared to adaptation cost scenarios involving increased direct spending on per capita health care. This comparison reveals a high benefit-cost ratio suggesting that moderate costs will deliver significant benefit in terms of avoided health burdens in the period 2010-2050. The methodology and results suggest that a focus on coordinated data collection and improved monitoring represents a potentially important no regrets adaptation strategy for Montserrat. Also the report highlights the need for this to be part of a coordinated regional response that avoids duplication in spending.

Table of Contents

I. VECTOR-BORNE DISEASES 4
A. INTRODUCTION 4
B. MALARIA 5
1. Introduction & the Caribbean context 5
2. Systematic literature search 6
3. Entomological models 7
C. DENGUE FEVER 9
1. Introduction & the Caribbean context 9
2. Systematic literature search 10

II. WATER & FOOD-BORNE DISEASES 11
A. INTRODUCTION 11
B. GASTROENTERITIS 11
1. Introduction 11
2. Systematic literature search 12
C. SCHISTOSOMIASIS 13
1. Introduction 13
2. Systematic literature search 13
D. LEPTOSPIROSIS 14
1. Introduction 14
2. Systematic literature search 15
E. CIGUATERA POISONING 16
1. Introduction & the Caribbean context 16
2. Systematic literature search 17

III. MENINGOCOCCAL MENINGITIS 18
A. INTRODUCTION & THE CARIBBEAN CONTEXT 18
B. SYSTEMATIC LITERATURE SEARCH 18

VI. CARDIOVASCULAR & RESPIRATORY DISEASES 19
A. INTRODUCTION & THE CARIBBEAN CONTEXT 19
2. Systematic literature search 20

V. MALNUTRITION & EXTREME EVENTS 21
A. INTRODUCTION 21
B. MALNUTRITION 21
1. Drivers of Malnutrition 21
2. The impact of climate change 22
3. Caribbean context 22
C. EXTREME EVENTS 23

I. COUNTRY CONTEXT – MONTSERRAT 24

II. METHODOLOGY 25

III. HIGHLIGHTS FROM THE PRELIMINARY STAGES 30
A. COMPILATION OF THE HEALTH DATA 30
B. BACKGROUND TO THE CLIMATE MODEL 31
1. Modeled baseline temperature & precipitation 31
2. The scenarios - A2 & B2 32
C. THE DOSE RESPONSE RELATIONSHIPS 33
1. Malaria dose-response relationship 34
2. Gastroenteritis dose-response relationship 35
3. Cardio-Respiratory dose-response relationship 35
4. Explanations for the other conditions 36

IV. SUMMARY RESULTS FROM THE PROJECTION STAGES 37
A. BASELINE MORTALITY & INCIDENCE RATES 37

B. POPULATION PROJECTIONS 38
C. EXCESS DISEASE INCIDENCE 39

V. VALUATION OF THE EXCESS STATISTICAL LIFE BURDEN 40
A. INTRODUCTION 40
B. VALUING STATISTICAL LIVES & THE USE OF BENEFITS TRANSFER 41
1. Statistical lives 41
2. Benefits transfer 42
C. DERIVING THE STATISTICAL LIFE TIME SERIES 43
D. APPLYING THE STATISTICAL LIFE TIME SERIES 44

VI. DISCUSSION 49
A. STUDY OUTCOMES: 49
B. ADAPTATION OPTIONS: 51
C. COST-BENEFIT ANALYSIS 51
1. Mitigation costs 51
2. Adaptation costs 52

REPORT CONCLUSION 54

ANNEX I - EQUATIONS FROM METHODS TABLE 56

ANNEX II - MALARIA LITERATURE SEARCH PAPERS 57

ANNEX III - DENGUE FEVER LITERATURE SEARCH PAPERS 60

ANNEX IV - GASTROENTERITIS LITERATURE SEARCH PAPERS 63

ANNEX V - SCHISTOSOMIASIS LITERATURE SEARCH PAPERS 64

ANNEX VI - LEPTOSPIROSIS LITERATURE SEARCH PAPERS 65

ANNEX VII - CIGUATERA POISONING LITERATURE SEARCH PAPERS 66

ANNEX VIII - MENINGOCOCCAL MENINGITIS LITERATURE SEARCH PAPERS 67

ANNEX IX - TEMPERATURE, CLIMATE, RESPIRATORY & CARDIOVASCULAR DISEASE LITERATURE SEARCH PAPERS 68

REFERENCES 71

List of tables

Table 1: Cardiovascular and Respiratory diseases as defined in the International Classification of Diseases and Related Health Problems, 10th Revision (2007)……...19

Table 2: Methodology Table……..27

Table 3: Summary of the baseline health data compiled for Montserrat……..31

Table 4: Dose-response relationships used to project disease incidence with climate change…….34

Table 5: The baseline incidence and mortality rates derived from the baseline population and health data……...38

Table 6: The projected total excess disease burden for Montserrat covering the period 2010-2050……..40

Table 7: Summary statistics showing the range of statistical life burden anomalies for Montserrat……..46

Table 8: Detailed VSL anomaly results as broken down by type of impact, climate change scenario, discount rate, and disease……...47

Table 9: Estimated present value treatment cost for A2 and B2……...52

Table 10: Present value adaptation costs……..53

List of figures

PART I:

Figure 1: The vector-borne disease transmission cycle……..….5

Figure 2: Malaria entomological model including some of the human dynamics……..……8

Figure 3: The main steps in the *Schistosoma* life cycle transmission cycle, environmental drivers, and non-environmental drivers……...14

Figure 4: Copy of figure 2 from Storck and others (2008) showing overlapped time series of reported cases of leptospirosis and rainfall for the Caribbean Island of Guadeloupe……..15

Figure 5: Copy of figure 3 from Slack and others (2006) showing overlapped average rainfall and average leptospirosis incidence by month for Innisfail health district in the state of Queensland, Australia…….16

Figure 6: Copy of figure 2 from Chateau-Degat et. al. (2005) showing an actual time series of dinoflagellate densities, as well as a modeled time series of dinoflagellate densities……17

Figure 7: Copy of figure 3 from Chateau-Degat et. al. (2005) showing actual and predicted cases of ciguatera poisoning. ……..18

PART II:

Figure 8: Demographic breakdown by gender and age category for Montserrat…….24

Figure 9: Mean baseline monthly temperature and precipitation covering the period 1960 1990 for Montserrat as simulated by the regional downscaling of the ECHAM4 GCM…….32

Figure 10: ECHAM 4 down-scaled A2 and B2 precipitation and temperature projection anomalies 2010-2050……..33

Figure 11: Montserrat Population Data (Baseline & Projected)……...39

List of Acronyms

BT	Benefits Transfer
CAREC	Caribbean Epidemiology Centre
CBA	Cost-Benefit Analysis
CDB	Caribbean Development Bank
CDC	U.S. Centers for Disease Control & Prevention
CIA	U.S. Central Intelligence Agency
CPI	Consumer Price Index
DCPP	Disease Control Priorities Project
DF	Dengue Fever
DHF	Dengue Hemorrhagic Fever
ECHAM	European Centre Hamburg Model (global climate model)
ECHAM4	European Centre Hamburg Model (global climate model) 4
ECLAC	Economic Commission for Latin America and the Caribbean
FAO	Food and Agriculture Association
FS	Food Security
GCM	Global Climate Model
GDP	Gross Domestic Product
GE	Gastroenteritis
GIS	Geographic Information System
HADLEY	Hadley Centre for Climate Prediction and Research
HADAM3	Hadley Centre Atmospheric Global Climate Model 3
IPCC	Intergovernmental Panel on Climate Change
MARA	Mapping Malaria Risk in Africa model
MM	Meningococcal Meningitis
NASA	National Aeronautics and Space Administration
NHS	United Kingdom National Health Service
PAHO	Pan American Health Organization
PM	Particulate Matter Air Pollution
PPP	Purchasing Power Parity
PRECIS	“Providing Regional Climates for Impact Studies” Caribbean initiative
PTSD	Post Traumatic Stress Disorder
PV	Present Value
RS	Remote Sensing
SIDS	Small Island Developing States
SST	Sea Surface Temperature
UN	United Nations
UNDP	United Nations Development Programme
UNESCO	United Nations Educational, Scientific and Cultural Organization
VSL	Value of a statistical life
WB	World Bank
WHO	World Health Organization
WOK	ISI Web of Knowledge
WTP	Willingness-to-Pay

I. 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).

The places most immediately vulnerable to the effects of climate change are small island developing states (SIDS) which tend to be tropical and comparatively remote with less developed infrastructure, a greater vulnerability to natural disasters, and economies that are particularly sensitive to external shocks (ECLAC, 2007; Lewsey and others, 2004; Simpson and others, 2009). This is particularly true for those islands in the Eastern Caribbean because these islands are largely volcanic, something which has forced development into the coastal zones, thereby making it susceptible to the effects of sea level rise, hurricanes, heat waves, droughts, and floods. However, in addition to being especially vulnerable to these more charismatic categories of climate change impact, SIDS are also vulnerable to the impacts that climate change can have on disease (Ebi and others, 2006).

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 and others, 2004). Caribbean SIDS have the potential to be particularly vulnerable to the impacts of climate change on health because they tend to experience a "dual" disease burden of having many endemic and environmentally-sensitive disease vectors as well as human populations burdened from high rates of cardio-respiratory diseases (DCPP, 2006). Trying to understand and model disease incidence in response to climate change is therefore of particular importance in the Caribbean. Furthermore, because incidence of mortality and disease have economic ramifications, it is also important to try and value the climate change-induced disease burden so that it can be appropriately factored into national and regional decision-making regarding investment decisions and resource allocation (especially adaptation spending).

The purpose of this report is not only to investigate the potential disease burden due to climate change for Montserrat but also to derive a monetary value of that disease burden. This monetary value can then stand comparison with the costs of potential adaptation scenarios. The timeline chosen for this study (2010 – 2050) reflects the immediate relevance of climate change and covers a long enough time period to be able to investigate the impacts of climate change without extending analysis further forwards in time than international organizations like the United Nations (UN) are willing to project certain socio-economic variables like national population growth.

The literature review supporting this research was completed in two parts. Firstly, major World Health Organization (WHO) publications were consulted for discussions on: 1) the types of disease most sensitive to climate change; 2) the range of best practice methods available for projecting disease burdens given certain climate change scenarios and models

and; 3) the range of potential weaknesses that can be encountered during this type of research. Following this first stage of the literature review, the baseline health data were compiled, and the quality, type, and availability of these data in combination with the first stage of the literature review informed not only the selection of diseases included in this study, but also the design of the method employed (an updateable dose response-based framework for rapid integrated assessment in situations with substantial data uncertainty). As time series analysis for site-specific derivations of climate-disease dose-response relationships could not be performed on the baseline health data available (see section 7), the second phase of the literature review had two foci: 1) the undertaking of systematic searches to find any published dose-response literature that might be acceptably utilized in this study; 2) to discuss various disease modeling methods that exist in the literature that, despite not being directly applicable in this study, may be suitable given sufficient investment in regional and national-level monitoring for shorter-term disease prediction efforts in the Caribbean.

The dose-response relationships identified during the second phase of the literature review were used to project disease incidence (see Part II, section 2 for method), and following this disease projection, the non-market component of the total economic impact caused by the projected disease impacts were valued using a benefits transfer (BT), willingness-to-pay (WTP)-derived value of statistical life (VSL) estimate scaled to the income of each island. As discussed in subsequent sections, the VSL, WTP-based approach was utilized because the data available for Montserrat was insufficient to support the derivation and analysis of the more health impact assessment metrics of Disability-Adjusted Life Years (DALYs) or Quality-Adjusted Life Years (QALYs). Additional costs, benefits, and issues of efficiency are not addressed in this study.

The magnitude of the resulting impact provides the basis for a benefit cost comparison with adaptation costs relevant to the disease scenarios. Ahead of a decision on specific adaptation measures this report cannot set out definitive benefit-cost ratios.

This report is intended to fit into a larger research framework focused on understanding and projecting the physical and economic impacts of climate change on the Caribbean. This larger research framework includes the following publications covering a wide variety of climate change-related assessments: (Angelesand others, 2007; Blanco and Hernández, 2009; Bueno and others, 2008; CDB, 2008; Centella, 2009; Contreras-Lisperguer and de Cuba, 2008; ECLAC, 2007, 2010; Haites and others, 2002; ECLAC, 2010a; Lewsey and others, 2004; Peterson and others, 2002; Simpson and others, 2009; Singh, 1997; ECLAC, 2010b; Toba, 2009; Vergara, 2009).

The remainder of this report is divided into two parts:

Part I contains five sections, each of which focuses on a particular category of disease or health impact that is sensitive to climate change, and four of which focus on the results from the dose-response relationship searches mentioned previously. A general introduction to each category of disease considered in this study is provided, as are more specific analyses of the state of knowledge regarding the relationship between environmental change and each specific disease. Attention is also paid to Caribbean-focused research that was found, as this literature provides the context for this study. This section provides considerable detail since this information is relevant to education/outreach, decision making, and adaptation planning and the longer term improvement of the climate- health impact picture. Ultimately the method designed for use in this study was necessarily driven by the data availability and quality.

Part II presents the results of disease projection and valuation research, and consists of six sections that cover the country context, the methodology used, results from each main stage in the methodology, and a discussion of these results.

The literature review is presented in its entirety in Part I (for coherence), and a discussion/rationale of the method implemented can be found in Part II, section 2.

Readers who are unfamiliar with the recent literature on the health impact of climate change, and who are unfamiliar with the Caribbean context for the diseases considered, are encouraged to use Part I as an introduction to this field of research prior to consulting Part II.

PART I

I. VECTOR-BORNE DISEASES

A. 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 and others, 2008; Kovats and others, 2003e). 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 and others, 2008; Githeko and others, 2000; Kovats and others, 2003e; Kuhn and others, 2005).

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. 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 and others, 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 and others, 2000; Martens and others, 1995; Martens and others, 1997; McMichael and others, 2004).

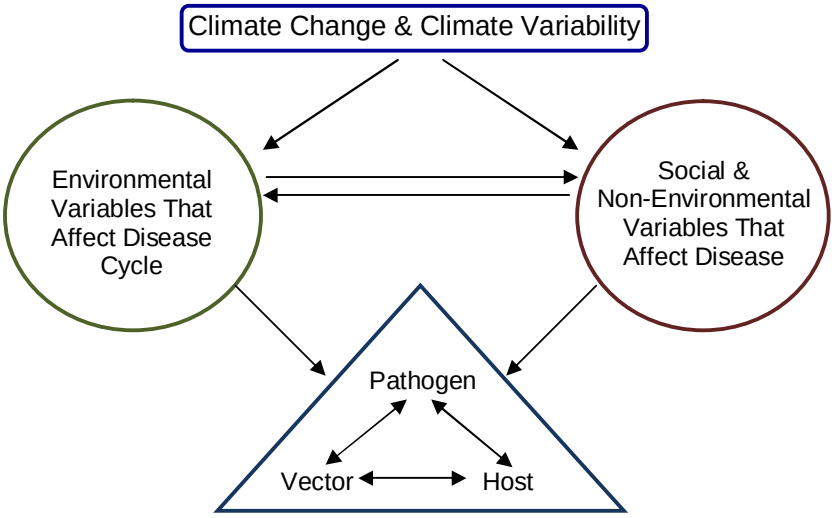
Within areas already conducive to vector survival, temperature increases which remain below the upper physiological threshold for any given vector or pathogen, will accelerate the development and maturation of both vectors and pathogens, as well as the metabolism rate of adult vectors (Githeko and others, 2000; Patz and others, 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 and others, 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 and others, 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 and others, 2000; Kovats and others, 1999; McMichael and others, 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 behavior 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 and others, 2000; Martens and others, 1997; McMichael and others, 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 behavior, and may in the short-term trump the effects of background precipitation or temperature on vector population dynamics (Githeko and others, 2000; Martens and others, 1997; McMichael and others, 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 and others, 2000; Martens and others, 1997; McMichael and others, 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, occur in the Carribean, and for which at least some datasets are available.

B. MALARIA

1. Introduction & the Caribbean context

Despite a significant contraction since 1870 in the geographic distribution of malaria, the disease is currently the most prevalent and significant infectious vector-borne disease in the world (Baron, 2009; van Lieshout,., 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 and others, 2003e; Martens and others, 1997; Martens and others, 1995; van Lieshout and others, 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 and others, 2008; Craig and others, 2004b; van Lieshout and others, 2004).

Consideration of malaria in the context of climate change in the Caribbean is particularly interesting because of its near total eradication between 1958 and 1962, and recent resurgence in multiple countries. Rawlins and others (2008) analysed the incidence of malaria from 1980 – 2006 and found that during this period, there were 897 imported cases in the Caribbean subregion, and three countries - Guyana, Suriname, and Belize – documented more than 875,000 autochthonous cases during this time period. There were also numerous cases of autochthonous malaria reported from several non-endemic islands during this period. This study emphasized that virtually all of the Caribbean countries are at risk for the re-establishment of endemic malaria as a result of these countries having near optimal environments for *Anopheles* vector populations. This optimality is demonstrated by the presence of multiple *Anopheles* species per country, human populations with low immunity, geographic proximity to locations with endemic malaria, increasing chloroquine resistance in the region, and easy travel between countries (Rawlins and others, 2008).

2. Systematic literature search

Given the work by Rawlins and others (2008), and given that malaria is considered highly sensitive to climate change at the global scale (Kovats and others, 2003e; Kuhn and others, 2005; Martens and others, 1995; Martens and others, 1997; Martens, Willem J.M., and others, 1995), 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 the second part of this report. Papers included for consideration in this process were:

1) the significant WHO publications on climate change and disease (Kovats and others, 2003d; Kuhn and others, 2005; McMichael and others, 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 modeling of malaria.[1]

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 modeling techniques and to anticipating the impact that climate change will have on malaria. Early efforts to capture the impact of environmental variables modeled epidemic potential based on a variety of variables (vector abundance, human population size, vector survival probability, vector biting frequency pathogen incubation period, and other entomological variables), and estimated that by 2100 global tropical epidemic potential would increase significantly compared to that in the absence of climate change (Martens and others, 1997; Martens and others, 1995). However, this could not be tied directly to malaria transmission because, among other reasons, human population immunity was not included in the model (Martens, Willem J.M. and others, 1995). Consequently, the two major WHO publications instead used the validated Mapping Malaria Risk in Africa (MARA) model to estimate, by global region, the percent increase in the population at risk of contracting malaria (Campbell-Lendrum and others, 2003; McMichael and others, 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.

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

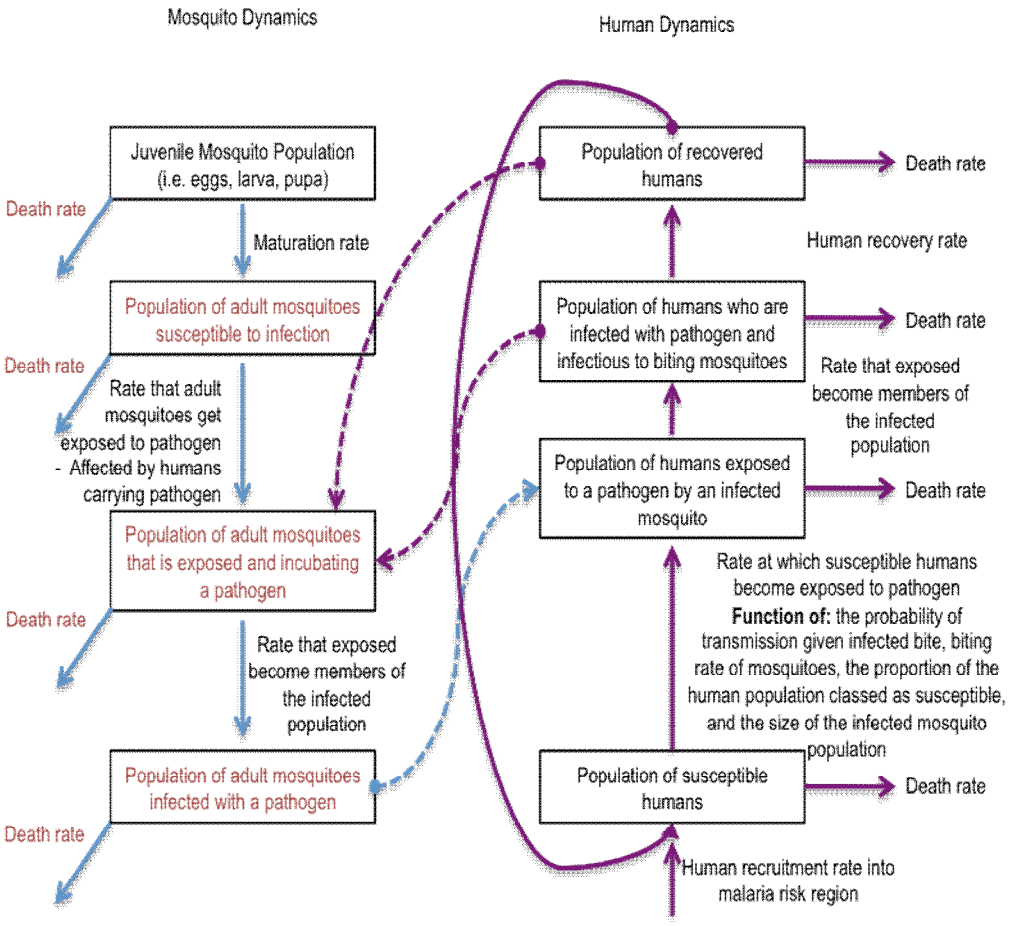
The post-2000 papers sub-set emphasized the following approaches to modeling and predicting malaria risk, potential, and transmission:

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

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 behavioral 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 and others, 1997; Paaijmans and others, 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 behavioral 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)



Source: Data compiled by author

4. The role of non-environmental variables

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 and others, 2010; Craig and others, 2004b; Gerthing and others, 2010; Stratton and others, 2008; Wandiga and others, 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 the suggestion of Lafferty (2009) that malaria and infectious disease risk zones will shift, rather than expand over the next century (Epstein, 2010; Lafferty, 2010; Lafferty, 2009; Ostfeld, 2009; Pascual and Bouma, 2009). These

conflicting perspectives demonstrate the extent to which reconciliation is needed between, on the one hand, modeling 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 behavior and socio-economic development.

This debate is particularly relevant in the context of projecting malaria incidence in the Caribbean where there is a history, including recent examples, of the non-environmental variables significantly decreasing the incidence of malaria from what might otherwise be expected given the environmental suitability of the region to malaria transmission.

C. DENGUE FEVER

1. Introduction & the Caribbean context

In the context of climate change, the study of dengue fever (DF), and its more deadly sibling dengue hemorrhagic fever (DHF),[2] shares a number of strong similarities with the study of malaria. As with malaria, dengue fever 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 the world's most important vector-borne disease (Fuller and others, 2009; Kovats and others, 2003e). 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 and others, 2008; Martens and others, 1997; McMichael and others, 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 dengue fever as on malaria (Githeko and others, 2000; Jetten and Focks, 1997; Martens and others, 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 the lifecycle may be almost completely shielded in certain places from the effects of climate change-induced temperature and precipitation changes (Fuller and others, 2009; Githeko and others, 2000; Jansen and Beebe, 2010; Martens and others, 1997). Therefore, despite the overlapping entomological characteristics between *Aedes* and *Anopheles* mosquitoes, this urbanization points to an extremely important relationship between human-based, non-environmental variables and DF incidence, this should significantly affect dengue fever modeling efforts.

In the Caribbean, most DF incidence occurs on a concentrated set of islands. During the period 1980 – 2001 the Caribbean recorded 43,000 cases of DF, all but 3,000 of which occurred from 1990-2000, and all but 2% of which occurred in Trinidad and Tobago, Barbados, and Jamaica. These cases occurred despite the *Aedes* vectors having largely been eradicated along with the *Anopheles* vectors in the late 1950's.

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., and others, 2004; Amarakoon and others, 2008; Ebi and others, 2006; Fuller and others, 2009; Johansson and others, 2009a; Johansson and others, 2009b; Jury, 2008). Despite their utility in local

[2] 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 .

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, as well as to the continued susceptibility of the Caribbean to the re-establishment of persistent vector-borne disease (Amarakoon A., and others, 2004; Amarakoon and others, 2008; Fuller and others, 2009)

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 modeling
   (Amarakoon and others, 2008; Barbazan and others, 2010; Degallier and others, 2010; Focks and Barrera, 2006; Hopp and Foley, 2001, 2003; Yang and others, 2009a, 2009b)
2) Statistical or time series
   (Amarakoon A., and others, 2004; Brunkard and others, 2008; Halide and Ridd, 2008; Hu and others, 2010; Hurtado-Díaz and others, 2007; Johansson and others, 2009a; Johansson and others, 2009b; Jury, 2008; Lu and others, 2009; Luz and others, 2008; Wu and others, 2007)
3) Global scenarios
   (Hales and others, 2002);
4) GIS and/or RS –based analyses
   (Fuller and others, 2009; Kolivras, 2010)
5) Socio-economic analysis
   (Jansen and Beebe, 2010; Ooi and Gubler, 2009; Tseng and others, 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 dengue fever transmission is climate insensitive (Sutherst, 2004), and the others continue to focus on the entomological sensitivity of the dengue vectors to climate (Yang and others, 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 and others, 2005)

The content of this literature when combined with the inconsistent relationships found in the Caribbean between DF and environmental variables lends credence to the idea suggested by Ebi and others (2006) that the location-specific socio-economic variables and behavioral/control practices will likely continue to exacerbate the impact of climate change in. 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.

II. WATER & FOOD-BORNE DISEASES

A. 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 and others, 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 (Ebiand others, 2008; Kovats and others, 1999; Kuhn and others, 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 and others, 2008; Kovats and others, 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 and others, 2008; Kovats and others, 1999; Kovats and others, 2003f; McMichael and others, 2004).

The primary outcome of contact with the aforementioned environmentally sensitive pathogens is gastroenteritis/diarrhoeal disease, for which there is a relatively strong data set in Montserrat, making it an important condition to consider in the context of projecting and valuing disease incidence with climate change. In addition to gastroenteritis, the following conditions demonstrate sensitivity to environmental change, are relevant in the Eastern Caribbean, have at least some baseline data in Montserrat, and are therefore considered further in the subsequent sections: schistosomiasis, leptospirosis, and ciguatera poisoning.

B. GASTROENTERITIS

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. 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 and others, 2008; Kovats and others, 2003f; McMichael and others, 2004).

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 (Kovatsand others, 2003f; Kuhn and others, 2005; McMichael, and others, 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 and others (2000) and Singh and others (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 and others, 2000; Singh and others, 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 and others, 2004)
2) Forecasting methods to predict age-stratified diarrhoea in Mali (Medina and others, 2007)
3) Temperature and relative humidity to predict all-age GE incidence in Japan (Onozuka and others, 2010)
4) Monthly rainfall and temperature to predict diarrhea incidence in children under age five at a global level (Lloyd and others, 2007)

These papers reveal that increasing temperatures, to varying degrees across age, space and time, tend to increase gastroenteritis 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 such as the state of sanitation infrastructure, and cultural patterns like hygiene practices that should, considering gastroenteritis is primarily water and food-borne, influence incidence of gastroenteritis across space and time.

C. SCHISTOSOMIASIS

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).[3] 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 and others, 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 host snail life cycles to environmental variables (Mangal and others, 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).

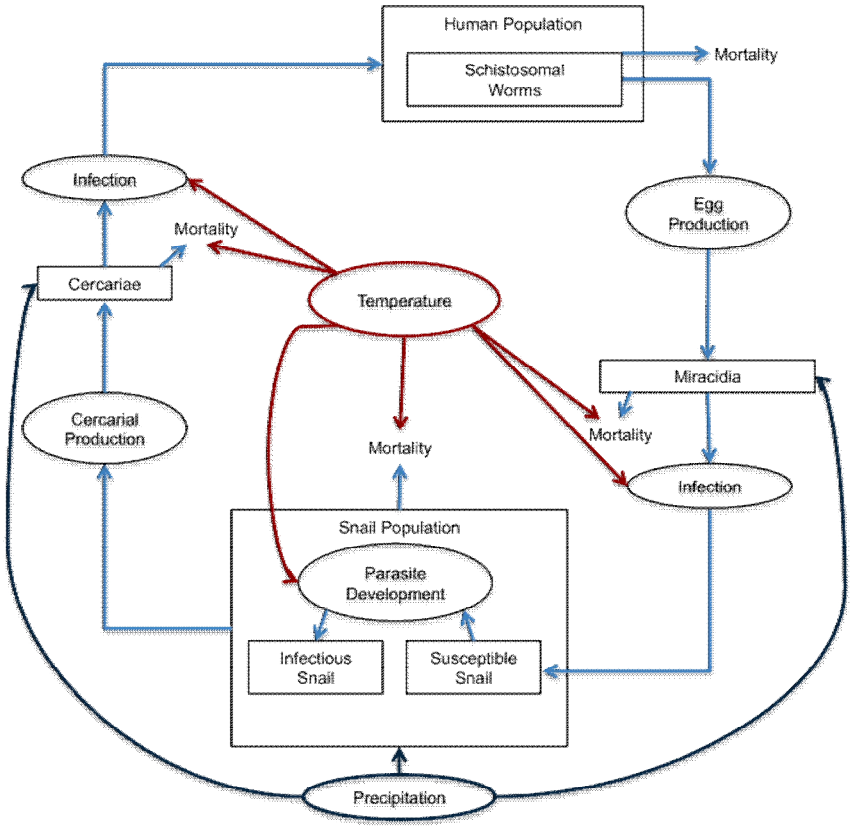
2. Systematic literature search

As was undertaken for the diseases considered previously, a literature search beyond the primary WHO sources already specified (as well as Martens and others (1997), Sutherst (2004) which were identified previously) to try and identify the current state of knowledge regarding the modeling 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 modeling that attempted to capture the types of dynamics shown in figure 3 (Liang, and others, 2007; Liang and others, 2005; Mangal and others, 2008; Martens, W.J.M., and others, 1995; Martens and others, 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 and others, 2008; Martens and others, 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, Martens, W.J.M. and others, 1995). Liang and others (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.

[3] 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, W.J.M., and others, 1995), 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.



Source: Data compiled by author

D. LEPTOSPIROSIS

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 there is contact 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 and others, 2001; Storck and others, 2008; WHO, 2003b). Because the symptoms of infection are so variable 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 and others, 2009).

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 and others, 2006; Storck and others, 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 and others, 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, and others, 2008). Their efforts treated leptospirosis incidence as a disease demonstrating threshold behavior that is driven by precipitation.

Figure 4: This is a copy of figure 2 from Storck and others (2008) showing overlapped time series of reported cases of leptospirosis and rainfall for the

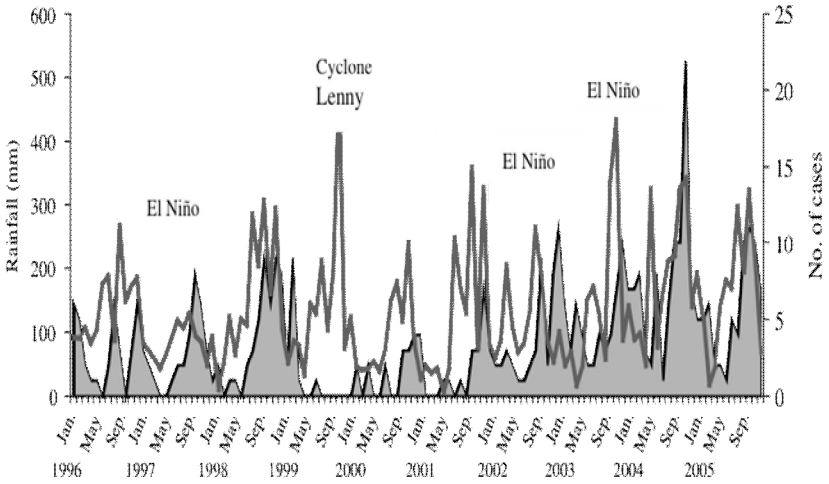
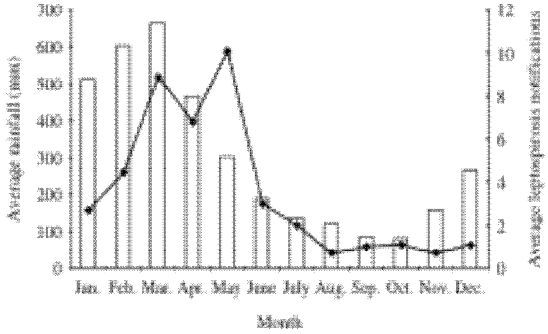


Fig. 2. Monthly rainfall (—) and leptospirosis cases (▒) for 1996–2005.

Source: Data compiled by author

Figure 5: This is a copy of figure 3 from Slack and others (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.



Source: Data compiled by author

E. CIGUATERA POISONING

1. Introduction & the Caribbean context

Ciguatera poisoning is a condition caused by the ingestion of either ciguatoxins or maitotoxins. These are some of the most toxic substances in the world, and they enter the human food chain through the herbivorous reef fish that feed on the dinoflagellates that produce these toxins (Fleming, 2010; Kovats and others, 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 colourless, tasteless, odourless, heat-stable, and acid-stable and therefore cannot be detected in fish or removed prior to consumption (Fleming and others, 2006; Fleming, 2010; Jaykus and others, 2008; Kipping and others, 2006; Ting and Brown, 2001).

Ciguatera poisoning is important to consider in this study because the population dynamics of the ciguatoxin-producing dinoflagellate species are sensitive to changes in sea surface temperatures (SST) (Jaykus and others, 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 and others, 2006; Fleming, 2010; Jaykus and others, 2008; Tester and others, 2010).

Ciguatera poisoning occurs year round in 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 in the Caribbean: *Coolia monotis*, *Prorocentrum belizeanum*, *Prorocentrum lima*, *Prorocentrum mexicanum*, *Prorocentrum hoffmannianum*, *Ostreopsis lenticularis*, and *Ostreopsis siamensis*. Consequently, it is also common

for individual fish in the Caribbean to carry multiple ciguatoxins (Faust, 2009; Tester, and others, 2010; Tosteson, 2004).

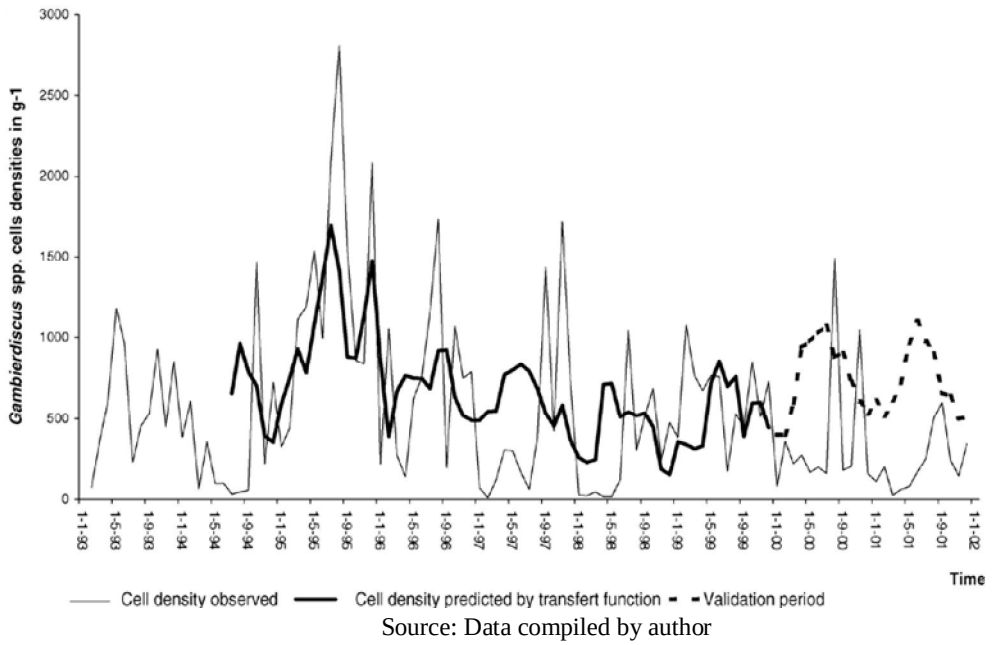
2. Systematic literature search

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 and others, 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.

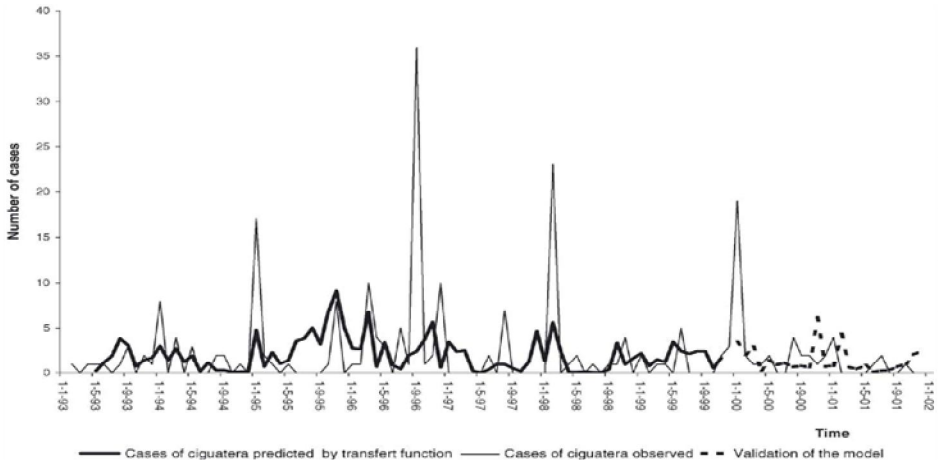
Only one paper attempted to model ciguatera poisoning incidence (Chateau-Degat and others, 2005). Their two-step model formally connected SST and dinoflagellate density (with a lag of 17 months), and dinoflagellate density and disease incidence (with a lag of 3 months). The results of their model demonstrate 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 and others (2005) showing an actual timeseries of dinoflagellate densities, and a modeled time series of dinoflagellate densities.



Source: Data compiled by author

Fig 7: This is a copy of figure 3 from Chateau-Degat and others (2005) showing actual and predicted cases of ciguatera



Source: Data compiled by author

III. MENINGOCOCCAL MENINGITIS

A. INTRODUCTION & THE CARIBBEAN CONTEXT

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, and others, 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 and others, 2008). This theory may have relevance 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).

B. 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 and others, 2003; Palmgren, 2009; Thomson, Madeleine C., and others, 2006; Yaka and others, 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 and others, 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 remain unclear, and has never been successfully modeled to date (Palmgren, 2009; Roberts, 2008; Thomson, Madeleine, C. and others, 2006).

VI. CARDIOVASCULAR & RESPIRATORY DISEASES

A. INTRODUCTION & THE CARIBBEAN CONTEXT

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 and others, 2003; Ebi and others, 2008; McMichael and others, 2004). Adults who suffer from pre-existing cardiovascular and respiratory diseases, the elderly, children, outdoor laborers, 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 and others, 2008; Hales and others, 2003; Luber and Prudent, 2009).

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

Source: Data compiled by author

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 and others, 2008). However, there has been some evidence that tropical climates exhibit different (i.e. non-U-shaped) relationships between temperature and mortality (Kovats and others, 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 and others, 2010). This is true regionally for the Caribbean as well. In 2000, cardiovascular and respiratory diseases held six of the top ten spots for leading causes of death among CAREC member countries (CAREC, 2005; Freeman and others, 1996; PAHO, 2009). As is the case with most regions currently suffering a significant burden from cardiovascular and respiratory diseases, the Caribbean started to experience increased incidence of these diseases following an epidemiological transition. This transition saw increased calalorie availability, decreased vector-borne and water and food-borne illness compared to historic levels, and a significant dietary shift comprising increased sugary, salty and fatty food intake (Albert and others, 2007; Cunningham-Myrie and others, 2008).

2. Systematic literature search

WOK searches[4] 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 from a location that closely matched the climatic conditions of Montserrat. 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 and others, 2009; Hajat and Kosatky, 2009; Kovats and Hajat, 2007). Although a full critical review of this literature and its ramifications is beyond the scope of this study, 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 and others, 2002; Hales and others, 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 at summer-temperature morality relationships instead of, or separately from, all-year relationships (Chung, and others, 2009; Vaneckova, and others, 2008; Zanobetti and Schwartz, 2008). Some attempted to analyze the relationship between short-term heat

[4] 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).

waves and mortality, rather than, or in addition to, longer-term trends (Díaz and others, 2002; Hajat and others, 2006; Medina-Ramón and Schwartz, 2007; Rey and others, 2007; Saez and others, 1995).

Still other studies at least partially stratified their results by age (Bell and others, 2008; Davis and others, 2004; Díaz and others, 2002; Hales and others, 2000; Medina and others, 2007; Saez and others, 1995; Sheridan and others, 2009; Vaneckova and others, 2008). There was 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 and others, 2000; McMichael and others, 2008; Vaneckova and others, 2008). While there is evidence that PM and ground-level ozone increase mortality both independently from, and synergistically with temperature (Daniels and others, 2000; Ebi and others, 2008; HEI International Scientific Versight Committee, 2004; Kovats and others, 2003a; UNDP/World Bank Energy Sector Management Assistance Programme, 2004), this effect varies by location, as does its synergism with temperature (Eb, and others, 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.

V. MALNUTRITION & EXTREME EVENTS

A. INTRODUCTION

This section addresses two other routes through which climate change could impact on the disease burden in Montserrat: malnutrition and extreme events. Full systematic reviews of these topics are beyond the scope of this study, as is more than a very basic attempt at the projection of their impacts on the future disease burdens. However, each of these topics deserves at least a brief discussion in the context of this study because they are capable of making the future populations of Montserrat more susceptible to the diseases already discussed in the preceding four sections (Caulfield and others, 2004; McMichael and others, 2004). Malnutrition is addressed in section 5.2 and extreme events in section 5.3. These sections mark the end of the Part I of this study.

B. MALNUTRITION

1. Drivers of Malnutrition

Malnutrition results from a lack of food security (FS), and even though the world produces more than enough calories for the global population, more than one billion people are currently food insecure (Burke and Lobell, 2010). As Burke and Lobell (2010) and Schmidhuber and Tubiello (2007) discuss, FS is multifaceted and depends not only on the global production of food calories, but also on the local availability of those calories, the accessibility of those calories to individuals, the stability of individuals' access to food supplies, and the nutritional content of the food to which individuals have access (Burke and Lobell, 2010; Schmidhuber and Tubiello, 2007). These are factors that have significant non-agricultural drivers including development patterns in other sectors, socio-economic variables, demographic trends, local, national and international policies, and technology availability (Brown and Funk, 2008; Morton, 2007). That some research estimates that as much as 50% of global malnutrition incidence is the result of non-agricultural drivers of FS (Brown and Funk, 2008), is testament to the importance of these other drivers of FS.

Although important to recognize, the complexity of the determinants of FS makes it both difficult to measure and difficult to model (Burke and Lobell, 2010). Increasing globalization and uncertainty regarding climate change, both of which will affect the ecology and social systems driving food production, only add to the difficulty of projecting malnutrition under climate change (Godfray and others, 2010; Morton, 2007; Schmidhuber and Tubiello, 2007). Some researchers are optimistic about the ability of the world to adapt food production systems to conditions created by climate change, and

argue that the non-environmental drivers of FS can over-ride any problems created by global warming. However, what is put forward as being required for this to happen is a radical overhaul in the way food production is perceived, organized, and undertaken across the world. Godfray and others (2010) argue that in order to meet the world's growing population's calorific demands between now and 2050 that a global strategy will have to be created and implemented to ensure sufficient levels of coordination and resource use across the world go into producing the world's food supply. This opinion is seconded by those supporting the creation of an integrated approach to agricultural systems, in which the cross-linkages between agricultural sectors and non-agricultural sectors, both within and across nation, are recognized and factored into food systems planning (Ericksen and others, 2009).

2. The impact of climate change

This is particularly important because at a global level climate change has the potential to impact on all the sectors that also impact on agricultural production. Over the course of the 21st century, climate change will likely alter the availability and distribution of water resources, as well as growing seasons, the geographic range of agricultural pests, the frequency, location, and intensity of extreme events, and trends in both ENSO timing and duration. Through these impacts, climate change will likely also affect agricultural commodity prices (Brown and Funk, 2008; Dupont and Thirlwell, 2009; Howden and others, 2007; Tubiello and others, 2007). Even if global food production volumes do not decrease in the near or medium future, as has been suggested by Brown and Funk (2008) and Dupont and Thirlwell (2009), the types of changes that global warming can bring about still have the potential to decrease the FS of poorer countries. Therefore, despite being an extremely tall order, global and regional integration of agricultural sector planning with planning for other sectors will help to minimize this effect by increasing awareness of the ways in which agricultural production and food safety are affected by climate change and by helping guide any site-specific strategies, policy changes, or infrastructure creation necessary to improve and maintain food safety (Godfray and others, 2010).

3. Caribbean context

In terms of the future of FS in the Caribbean, even though specific projection of malnutrition is well beyond the scope of this study, it is important to mention the work of Kendall and Petracco (2009). They discuss trends in Caribbean agriculture and suggest a shift in focus to help not only maintain FS in the future, but also to promote the profitability of Caribbean agricultural systems. Their research indicates that Caribbean food production is vulnerable to the impacts of climate change because of the regional emphasis on agricultural exports. The revenues from the region's agricultural exports have not only declined in terms of the percent of GDP they constitute, but have also failed to cover the costs of food imports to the region, making all the Caribbean countries except Guyana, Jamaica, Suriname, and Belize reasonably dependent on food imports. They point out that this makes the Caribbean particularly susceptible to any future climate change-induced instability in both global agricultural production and commodity prices (Kendall and Petracco, 2009).

The way forward that Kendall and Petracco (2009) propose is for Caribbean countries to target the significant import market that exists within the region through the production of organic agricultural products while at the same time expanding non-agricultural exports. To what extent this suggestion is relevant to any particular Caribbean nation depends on a wide variety of factors that cannot be addressed here. However, a cursory investigation assessing the extent to which the problem identified by Kendall and Petracco (2009) holds true for Montserrat revealed the relevance of this discussion. In Montserrat 33% of employment is in the agricultural sector, and approximately 352 hectares are under cultivation (Simpson and others, 2009). Although the relative balance of the value of agricultural imports and exports is not available through Food and Agricultural Organization (FAO) for Montserrat, the strategy proposed by Kendall and Petracco (2009) at least warrants consideration in the context of planning for, and adapting to, the future impact of climate change.

C. EXTREME EVENTS

The main categories of climate sensitive extreme events are: 1) instances of extremely high and extremely low temperatures (a discussion of which can be found in the references in Appendix VII); 2) drought; 3) floods; 4) hurricanes. The later three of these all affect ground cover, agriculture, water quality, and erosion rates (Hales and others, 2003; Lewsey and others, 2004). These events can cause mortality, physical injury, as well as increased risk of respiratory diseases, water-borne diseases, mental illness such as post-traumatic stress disorder (PTSD), anxiety, and depression, and exposure to harmful chemicals (Ebi and others, 2008; Hales and others, 2003; Kovats and others, 2003c). The burden of disease from extreme events can be significant. PTSD, for example, has been documented in as much as 60% of individuals who experience these events (Gelea and others, 2005), indicating that a significant mental health burden can exist alongside other health burdens in the wake of natural disasters.

In the Caribbean, tens of millions of people are affected by natural disasters (and the ensuing health burdens) each decade (Hales and others, 2003). Vulnerability to these events is partially a function of disaster intensity and partially a function of infrastructure, topography, population density, and the presence or absence of early warning systems and emergency response plans (McMichael and others, 2004).

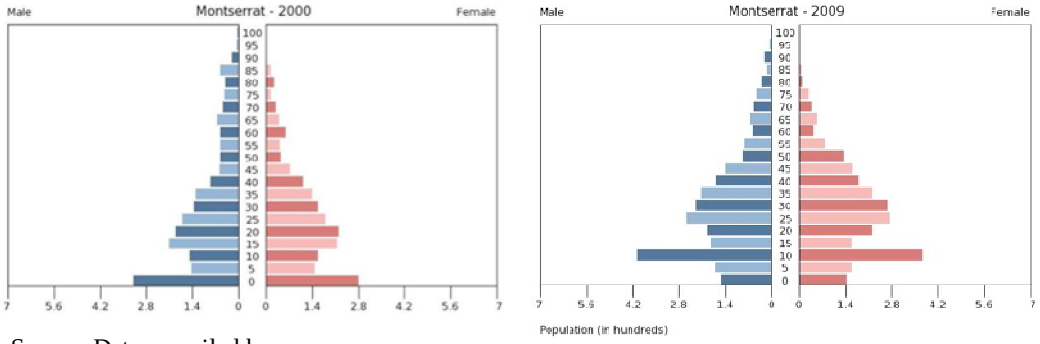
Of these disasters, hurricanes are particularly relevant to the Eastern Caribbean. While it is anticipated that climate change could affect the location of hurricanes as well as increase their size, frequency, and intensity (Contreras-Lisperguer and de Cuba, 2008), this effect remains ill-defined and uncertain (Ebi and others, 2008; Ebi and others, 2006; Lugo, 2000). If they do increase in frequency, however, coastal settlements will be increasingly vulnerable from physical damage to cities and infrastructure, increased coastal erosion, and increased salt-water intrusion of island aquifers (Lugo, 2000; Simpson and others, 2009). Any accompanying flooding could result in water supply contamination and increased disease incidence and would compound the physical and structure damage caused by wind, rain, and waves.

PART II
APPLICATION OF DISEASE BURDEN PROJECTIONS& STATISTICAL LIFE VALUATIONS

I. COUNTRY CONTEXT – MONTSERRAT

Montserrat is located in the Lesser Antilles, and has an area of 102 km^2. The island's population is primarily of African descent, has a relatively low birth rate and long life expectancy, and appears to be moving towards a stable population distribution (figure 11;PAHO, 2007, 2010). Although Montserrat has a long history of having a relatively stable total population (Ebanks, 1989), the population distribution shown in figure 8 is the consequence of a series of eruptions from the Soufrière Hills volcano which resulted in the semi-permanent evacuation of two-thirds of the population, as well as the creation of a permanent exclusion zone covering a significant portion of the island (Fenton, 2010b; PAHO, 2007).

Figure 8: Demographic breakdown by gender and age category for Montserrat in 2000 (left) and 2009 (right). From: (U.S. Census Bureau, 2010)



Source: Data compiled by author

Accurate population figures were extremely difficult to track the result of the emergency, as many residents left to spend varying periods abroad. The best population estimates show a drop from 10,402 in 1994 to 5,600 in 1997. By January 1998, the population had decreased to 3,483. There were 150 live births in 1994, 126 in 1995, and 128 in 1996. Age and sex breakdowns are not available for the period under review. The Island's Sustainable Development Plan (2008 -2020) targets a population of 9000 by 2020. Under this scenario, health exposures are likely to be more important than at present.

Montserrat provides good vaccine coverage and is considered a middle-income country, though the health sector receives substantial donor support, and suffers from a shortage of trained staff (PAHO, 2010).

The eruption has affected every aspect of Montserrat's economy, politics, development, and overall living conditions. The Sustainable Development Plan targets growth potential in tourism, banking/financial services and communication.

II. METHODOLOGY

A variety of publications discuss best-practice approaches to health impact assessment in the context of climate change (Ebi, 2008b; Kovats and others, 2003d; Kuhn and others, 2005; McMichael, and others, 2004; Murray and others, 2003; Sussman and others, 2008; Sutherst, 2004; WHO, 2003a). Separate from the actual methods suggested by these publications, the overall approach outlined in these documents requires the following:

1) Estimating climate variables under climate change
2) Identifying the time line for the study
3) Identifying potential areas of impact
4) Identifying areas of impact that can be quantified
5) Quantifying those impacts
6) Comparing those impacts to a baseline representing a world without climate change
7) Valuing this excess disease burden

The publications cited above also identify a variety of potential areas of methodological weakness including:

1) Transferring dose-response relationships from one location to another
2) Failing to model socio-economic variables (including institutional change)
3) Failing to model adaptation (both autonomous and planned) and mitigation in disease projections
4) Failing to model technological advancement
5) Using linear relationships with only one independent variables
6) Failing to account for extreme events
7) Not having sufficient baseline data

All these potential weaknesses relate to the fact that disease incidence is affected by a complex system that includes coupled human and environmental drivers, and that changing any piece of this complex system has the potential to affect disease incidence in both unexpected and nonlinear ways. Simplifying any such system excessively generates the possibility that key variables and feedback loops will be overlooked and the results therefore unrepresentative of the system they are supposed to model.

While these are very valid potential weaknesses and are both recognized and acknowledged, many could not be avoided in the context of this study, primarily because of significant data constraints.

Therefore, as was mentioned in the introduction, this research designed and implemented a dose response-based rapid integrative assessment approach to project and value the excess disease burden caused by the A2 and B2 climate change scenarios. This approach does follow the basic steps outlined above, but deviates from that literature in that it supports analysis efforts that are data-constrained.[5]

Table 2 (and Annex 1),[6] detail the steps of the methodology developed, and illustrate how they were carried out for Montserrat. These steps are divided into stages relating to preliminary assumptions, baseline projections, climate change scenario projections and endpoint valuation. The

[5] These analyses require such substantial amounts of time and data that, despite their obvious desirability, they are unrealistic given the data availability in many locations around the world, including in this case, Montserrat.
[6] As this same method was applied to both Saint Lucia and Montserrat, the table contains information on both countries

preliminary assumptions relate to the choice of time periods for the study, global warming scenarios, target populations data and selected health endpoints. The baseline details relate the calculation of baseline or business as usual morbidity and mortality rates, which are then recalculated using relevant epidemiological information for comparison under the selected warming scenarios. The endpoint valuation stage details the approach to assign monetary values to cases of morbidity and mortality under the baseline and counterfactual warming scenarios. This stage also details the discounting assumptions required for reporting present values. It is important to note that, as a consequence of being so data-constrained (both in terms of health data and environmental data), statistical modeling and regression methods could not be used. The study therefore assumed that disease incidence would change under climate change scenarios according to linear dose-response relationships identified in published literature and UN population projections, while holding all other socio-economic variables, environmental and technological variables implicitly constant.

There are two key aspects aspect of this methodology is that make it particularly well suited to this type of assessment: 1) It is well-suited for iterative research efforts as the quantitative results can be quickly updated given new or revised information; 2) By facilitating the rapid appraisal of the available data in each phase of the assessment, this approach highlights both the existing data gaps that need to be filled, and those variables that are most strongly driving the quantitative results.

Table 2: Details of the methodology and its implementation in this study. For additional details on the equation variables, see Annex I.

Step Group	General Step	Implementation in This Study	Generic Formula
Preliminary	1. Define the baseline time period	1960-1990: Selected by ECLAC	
	2. Define the projection time period	2010-2050: Chosen to cover a medium-long term period	
	3. Pick the climate emission scenarios that will be utilized	SRES A2 & B2: Selected by ECLAC	
	4. Collect disease incidence and mortality data. Include data on the health impacts of environmentally-sensitive extreme events (droughts, floods, fires, cyclones, and other storms) *Note: Some data may have to be inferred from other data	Montserrat: 1980-2009, Extreme Events 1960-2009 Saint Lucia: 1980-2009, Extreme Events 1960-2009 *Note: - Some mortality data was inferred from data showing zero incidence, or from regional mortality data apportioned by population - Gastroenteritis >5 baseline data was inferred using the ratio of <5 to >5 incidence data during the 1994-2000 period for Montserrat and the 1993-1999 period for Saint Lucia	
	5. Select diseases for inclusion in the study based on data from step 4 and broader climate change health literature	Malaria, Dengue Fever, Gastroenteritis, Schistosomiasis, Leptospirosis, Ciguatera Poisoning, Meningococcal Meningitis, Cardiovascular/Respiratory diseases	
	6. Collect climate model data for projection time period (step 2). Calculate the annual modeled climate anomaly time series.	Selected and Provided by ECLAC: ECHAM4 downscaled climate temperature and precipitation model	Projected Annual Data–Mean Baseline Data
	7. Search the literature for dose-response/exposure-response relationships connecting the step 6 climate variables to the step 5 diseases.	1. Consulted the following: (Ebi, 2008a; Ebi and others, 2008; Ebi and others, 2006; Kovats and others, 2003; Kuhn and others, 2005; McMichael and others, 2004; Murray and others, 2003; Sussman and others, 2008; Sutherst, 2004; WHO, 2003) 2. Conduced ISI Web of Knowledge and Ovid Medline searches for peer-reviewed, English-languages articles and reviews using the following search parameters: "Ciguatera AND Sea Surface Temperature," "Ciguatera AND Caribbean," "Ciguatera AND Climate," "Cardiovascular Morbidity AND Climate," "Cardiovascular Mortality AND Climate," "Heat-related Morbidity AND Climate," "Temperature Morbidity AND Climate," "Heat-Related Mortality AND Climate," "Leptospirosis AND Temperature," "Leptospirosis AND Precipitation," "Leptospirosis AND Climate," "Respiratory Morbidity AND Climate," "Respiratory Mortality AND Climate" "Neisseria meningitidis AND Climate," "N. meningitidis AND Climate," "Meningococcal AND Climate," "Meningitis AND Climate," "Schistosomiasis AND Prediction," "Schistosomiasis AND Climate," "Gastroenteritis AND Forecast," "Gastroenteritis AND Climate," "Diarrh* AND Climate," "Dengue AND Climate," "Malaria AND Climate,"	
	8. Identify population projections for the scenarios from step 3	A2: Best match is UN Constant Fertility Variant (2008) B2: Best match is UN Medium Variant (2008) *Note: Both were linearly interpolated between UN 5 year estimates	
Baseline/ Reference Projection	9. Collect census data covering the baseline-present period.	Montserrat & Saint Lucia: Total population: 1960-2010 Saint Lucia <5's: 1980-2010	
	10. Calculate the average baseline mortality and incidence rates for each of the step 5 diseases using the step 9 and step 4 data. Repeat for relevant extreme event data.	Calculated: $\overline{BIR_x} = \left(\frac{\sum BI_x}{\sum BPop} \right)$ $\overline{BMR_x} = \left(\frac{\sum BM_x}{\sum BPop} \right)$ *Note: - Baseline data summed over the 1980-1990 period (For extreme events, 1960-1990) - Only used years in the baseline interval where both health and population data existed - For Saint Lucia, 3 incidence rates were calculated for gastroenteritis: all, <5s, >5s	For each step 5 disease, and any extreme event data, over the relevant baseline period, calculate: ∑Step 4/∑Step 9
	11. Calculate the yearly reference population projections for the step 2 time period using regressions on the step 9 data. *Note:	Regression Time period utilized: Montserrat & Saint Lucia: Total population: 2001-2010 Saint Lucia: >5's: 2006-2010	

	For countries that have a markedly changed population growth pattern, perform regressions on the recent trends	<5's: (Total pop) – (>5 pop) for each year 2010-2050	
Baseline/ Reference Projection	12. Project the reference incidence and mortality burden for each step 5 condition using steps 10 and 11 for each year in the step 2.	Calculated: $RIB_{xi} = (\overline{BIR_x}) * (RPop_i)$ $RMB_{xi} = (\overline{BMR_x}) * (RPop_i)$	For each step 2 year, and each step 5 disease, calculate: Step 10*Step 11
Climate Change Scenario Projection	13. Create a time series of modified annual incidence and mortality rates covering the step 2 projection period and using the step 10 baselines rates for each step 3 scenario according to the step 7 dose-response relationships and the step 6 climate anomaly data.	Calculated: $PIR_{yxi} = (\overline{BIR_x}) * C$ $PMR_{yxi} = (\overline{BMR_x}) * C$	For each step 2 year, each step 3 scenario, and each disease with a step 7 dose-response relationship, calculate: Step 10*(1+(Step 7*Step 6))
	14. Project the incidence and mortality disease burden for each step 3 scenario using the step 13 rates and the step 8 population projections.	Calculated: $PIB_{yxi} = (PIR_{yxi}) * (SPop_{yi})$ $PMB_{yxi} = (PMR_{yxi}) * (SPop_{yi})$	For each step 2 year, each step 3 scenario, and each disease with a step 7 dose-response relationship, calculate: Step 13*Step 8
	15. Estimate the disease burden for diseases for which no dose-response relationship was found. Use the step 10 incidences and mortality rates and the step 3 population projections. * Note: This will capture only the effect of population changes	Calculated: $EIB_{yxi} = (\overline{BIR_x}) * (SPop_{yi})$ $EMB_{yxi} = (\overline{BMR_x}) * (SPop_{yi})$	For each step 2 year, each step 3 scenario, and each step 5 disease without a dose-response function, calculate: Step 10*Step 7
	16. Calculate the annual incidence and mortality anomalies for each step 3 scenario by subtracting the step 12 reference projections from the step 14/15 projections.	Calculated: $PIA_{yxi} = PIB_{yxi} - RIB_{xi}$ $\quad EIA_{yxi} = EIB_{yxi} - RIB_{xi}$ $PMA_{yxi} = PMB_{yxi} - RMB_{xi}$ $\quad EMA_{yxi} = EMB_{yxi} - RMB_{xi}$	For each step 2 year, each step 3 scenario, and each step 5 disease, calculate: Step 14 - Step 12 & Step 15 - Step12
Valuation	17. Based on the baseline data, pick valuation units.	A willingness-to-pay & benefits transfer-based value of statistical life (VSL) burden was used as the data was insufficient for disability adjusted life years (DALYs) derivation.	
	18. Collect recent PPP-adjusted GDP per capita data	Montserrat: US$3,400 (2002) Saint Lucia: US$10,900 (2009) (CIA World Factbook, 2010c)	
	19. Search for studies containing VSLs derived from environmental and/or health-focused studies	Selected mean US Health VSL: see Appendix 2 (Lindhjem and others, 2010): VSL_{2005}=4,808,000 PPP-adjusted US$	
	20. Collect the PPP-adjusted GDP per capita data for the step 19 reference country to match the years identified in step 18.	2002: US$37,600 2009: US$46,000 (CIA World Factbook, 2010c)	
	21. Inflate or deflate the step 19 VSL using the step 19 reference country consumer price index (CPI) data to match the years of the step 18 GDP data.	Utilized online calculator: http://www.bls.gov/data/inflation_calculator.htm VSL_{2002}=$4,428,874.55 VSL_{2009}=$5,281,586.77	For each year identified in steps 18 and 19, and using the step 19 VSL value, calculate: $VSL_{Step\ 19}$*($CPI_{Step\ 18}$/CPI_{Step19})
	22. Scale the step 21 VSLs to the income of the study sites using the information in steps 18, 20 and 21	Calculated: $VSL_{M\,2002} = \frac{\$3,400}{\$37,600} * \$4,428,874.55 = \$400,483.34$ $VSL_{SL\,2009} = \frac{\$10,900}{\$46,000} * \$5,281,586.77 = \$1,251,506.43$	For each study site, calculate: (Step 18/Step 20)*Step 21
	23. If necessary, scale the step 22 VSLs to match the initial step 2 projection year using CPI data	Utilized online calculator: http://www.bls.gov/data/inflation_calculator.htm $VSL_{M\,2010} = \$485,323.51$ $VSL_{SL\,2010} = \$1,271,772.09$	For each step 22 VSL, calculate: Step 22*($CPI_{Step\ 2}$/$CPI_{Step\ 22}$)

	24. Define an inflation effect for the step 23 VSLs, and project a VSL time series through the step 2 projection period	Calculated 2% annual inflation: $VSL_i = VSL_{2010} * (1.02)^i$ $i : 0\ (2010) - 40\ (2050)$	For each step 23 VSL, inflationary rate, and year n, calculate: Step 23*$(1+r)^n$
	25. Calculate the VSL mortality anomaly for each year in the step 2 projection period, and for each of the step 5 diseases included in step 16, using the step 24 VSL time series.	Calculated: $PVSL_{(M)yxi} = VSL_i * PMA_{yxi}$ $EVSL_{(M)yxi} = VSL_i * EMA_{yxi}$	For each step 2 year, and each step 5 disease with projected mortality data, calculate: Step 24*Step 16
Valuation	26. Find the relevant average life span information	Montserrat: 72.76 years Saint Lucia: 76.45 years (CIA World Factbook, 2010a, b)	
	27. For each step 5 disease for which incidence data was projected, look up the average duration of the disease. Use this information to approximate what fraction of the step 26 average life span this duration constitutes. (Note: This approach was utilized both because no WTP-based studies focusing on morbidity from with these diseases were found, and because the data was insufficient to support a cost of illness approach).	**Life Fractions** Montserrat Saint Lucia Gastroenteritis - Overall Pop 0.000188 0.000179 - <5 0.000113 0.000108 - >5 0.000264 0.000179 Leptospirosis 0.003436 0.003270 Ciguatera Poisoning 0.006872 0.006540 Malaria (1 year of illness) 0.013743 0.013080 Dengue Fever 0.000377 0.000358 Schistosomiasis 0.068719 0.065402 Respiratory Diseases (influenza) 0.000188 0.000179 Extreme Events (1 month) 0.001145 0.001090 (CDC, 2010; Every Day Health, 2009a, b; Fleming, 2010; Galea and others, 2005; *Inteli*Health, 2008; NHS, 2005; Tol, 2008) *Note: This requires assuming that the statistical life burden of these non-fatal conditions always scales linearly to the life fraction constituted by each condition	For each step 5 disease with projected incidence data, calculate: Duration/Step 26
	28. Project the step 16 incidence anomaly VSL fractions for each step 2 year using steps 27 and 24.	Calculated: $PVSL_{(I)yxi} = VSL_i * LF_x * PIA_{yxi}$ $EVSL_{(I)yxi} = VSL_i * LF_x * EIA_{yxi}$	For each step 2 year and each step 5 disease with incidence anomaly data, calculate: Step 24*Step 17*Step 16
	29. For each projection year, sum the VSL data (incidence and mortality) across all diseases to generate a cumulative VSL burden time series for each study site. Discount this cumulative time series if desired/required. .	Calculated: $VSL_{(Total)yxi} = VSL_{(I)yxi} + VSL_{(M)yxi}$ $VSL_{(Cumulative\ Total)yi} = \sum^{All\ x's} VSL_{(Total)yxi}$ $PV_{yi} = \frac{\sum_0^i VSL_{(Cumulative\ Total)yi}}{(1+r)^i}$	Across all diseases within each year, *i*, calculate: A_i=Step 28 + Step 25 Sum(A_i)/$(1+r)^i$

Source: Data compiled by author

III. HIGHLIGHTS FROM THE PRELIMINARY STAGES

A. COMPILATION OF THE HEALTH DATA

The baseline health data were compiled for Montserrat from a variety of sources (CAREC, 2002-2009, 2005, 2008; EM-DAT, 2010; Fenton, 2010a; PAHO, 1998, 2007, 2010). Based on the climate change health impacts literature, and these baseline data, the diseases discussed in Part I of this report were selected for inclusion in this study. The number of people affected and killed by extreme events and malnutrition were also considered.

Health data were available for the period 1980–2009 (see Table 3) meaning that only 10 years of data existed within the baseline window of 1960–1980. The data covered a variety of conditions and causes of mortality, but were inconsistently stratified by gender or age. Furthermore, many of the available datasets for these conditions were incomplete across the baseline time period, and for the majority of these conditions, the direct mortality data were virtually non-existent. Consequently, most of the mortality data had to be inferred from a combination of sources. For years in which no incidence of a disease was recorded, it was inferred that no deaths resulted from that disease either. In the case of deaths from dengue fever, meningococcal meningitis, malnutrition, cardiovascular diseases, and respiratory diseases, the baseline mortality data were at least partly inferred using baseline population data and regional, linearly interpreted mortality rates (CAREC, 2005).

Applying Caribbean-wide mortality rates required assuming that pooled mortality rate for the region matches the mortality rates for these countries. In the absence of other information, this is the best available estimation for these figures. Also, it is important to note that the mortality rates used to model dengue fever mortality are actually the pooled mortality rates for a broad (and undefined) class of vector-borne diseases, and so may not exactly mimic rates of dengue fever mortality. However, it was deemed an acceptable proxy considering that, given the morbidity data available, dengue fever incidence appears to be the most prominent vector-borne disease on both of these islands.

Finally, in instances where just two overlapping sources disagreed, the data from the more recent source were favoured.

Table 3: This table gives an overview of the dates for which data were available. The dates in bracket show the year range, and details are provided below on any extra steps needed to finalize the data set

Diseases/Conditions		Morbidity	Mortality
Malaria		(1980 – 2009) *Except: 1996, 1997, 2006*	(1980 – 2009) *Except: 1995 – 1997, 2006* *All Mortality Data Inferred From Morbidity*
Dengue Fever		(1980 – 2009) *Except: 2006*	(1985 – 2009) *Except: 2002, 2003, 2006, 2008* *Modeled: 1986 – 1992, 1994 – 2000* *Recorded: 1983 – 1985* *Inferred From Morbidity: 1980 – 1993, 1997 - 1999, 2001, 2004 – 2005, 2007*
Gastroenteritis (all cause)	<5	(1980 – 2009) *Except: 1981 - 1982*	
	>5	(1980 – 2009) *Approximated:* 1980, 1983 – 1990 *Recorded:* 1994 - 2009	
Schistosomiasis		(1999 – 2005)	(1999 – 2005) *All Inferred From Morbidity*
Leptospirosis		(1980 – 2009) *Except: 1986 – 1988, 2006*	(1980 – 2009) *Except: 1986 – 1988, 2006* *All Mortality Data Inferred From Morbidity*
Ciguatera Poisoning		(1980 – 2009)	
Meningococcal Meningitis* * *Recorded mortality data includes other types of meningitis and contradicts meningococcal morbidity data and so is not utilized*		(1980 – 2007) *Except: 2006*	(1980 – 2007) *Modeled: 1985 – 2000* *Inferred From Morbidity: 1980 – 1999, 2001 – 2005, 2007*
Cardiovascular Diseases			(1985 – 2006) *Modeled: 1985 – 2000* *Recorded: 1999 - 2006* *Inferred From Morbidity: 1995 - 1998*
Respiratory Disease		(1980 – 2009) *Inferred From Recorded Data on Incidence of Acute Respiratory Infections, Pneumonia, and Influenza (<5 & >5)*	(1985 – 2005) *Modeled: 1985 – 2000* *Recorded: 1992 – 1997, 2000 - 2005*
Malnutrition			(1985 – 2003) *Modeled: 1985 – 2000* *Inferred From Morbidity: 1997 - 2003*
Extreme Events		(1960 – 2009)	(1960 – 2009)

Source: Data compiled by author

B. BACKGROUND TO THE CLIMATE MODEL

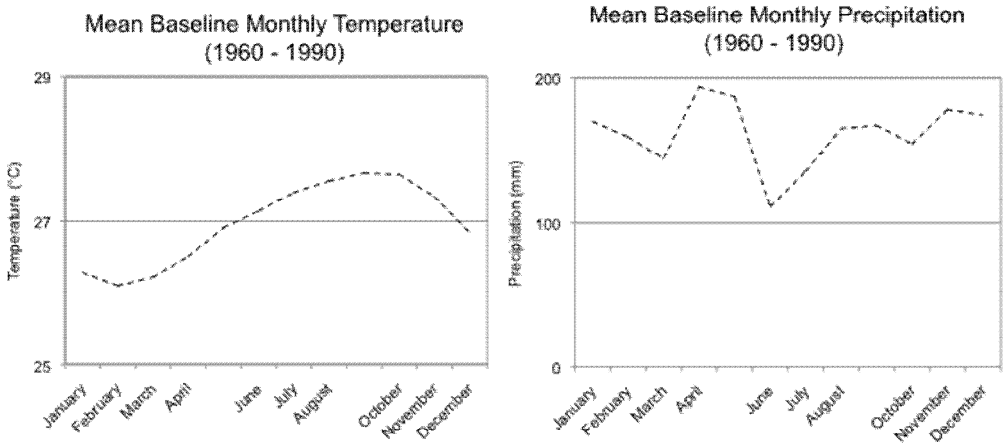
1. Modeled baseline temperature & precipitation

The climate in this region is marked by reasonably constant temperatures throughout the year, as demonstrated by the baseline mean monthly temperature data provided for use in this study (see figure 13). As is described in Centella (2009), these baseline data were generated prior to conduct of this study through the process of downscaling global climate models (GCM) and averaging the results from model runs covering the baseline period of 1960 – 1990. These are the baseline data to which future temperature increases are compared, and the process of downscaling was performed as a part of the "Providing Regional Climates for Impact Studies" (PRECIS) Caribbean initiative in order to generate data sets with higher spatial resolution than is possible at a global level. Experiments using the PRECIS regional climate modeling system utilized information from several GCMs in order to produce multiple versions of down-scaled data (Centella, 2009). The down-scaled climate data provided for use in this study came from the PRECIS experiments that utilized the ECHAM4 and

HADAM3 atmospheric GCMs. Because the ECHAM4 model covered the period 2010-2050, it was the model utilized in this study.[7] The specific 0.5° x 0.5° grid box utilized was that which most closely matched the latitude and longitude estimates for the island.[8]

The ECHAM mean baseline temperature data are reasonably coherent with the annual pattern temperature observed in the Eastern Caribbean. The same cannot be said for the modeled baseline precipitation data in this particular grid box, however, as it fails to capture the existence of the distinct wet and a distinct dry season (see figure 9).[9]

Figure 9: Mean baseline monthly temperature and precipitation covering the period 1960 – 1990 for Montserrat as simulated by the regional downscaling of the ECHAM4 GCM



Source: Data compiled by author

3.2.2

2. The scenarios - A2 & B2

These baseline temperature and precipitation data apply to both the A2 and B2 scenarios. These scenarios represent two families of emissions scenarios specified by the IPCC and they project a rise in the global mean temperature by 2099 of 3.4°C and 2.4°C, respectively.

The A2 family describes a world in which there is an emphasis on family and community. Fertility rates decline slowly, and wealth and technological advances remain unequally distributed around the world. Government strategies, energy use patterns, and environmental concern are heterogeneous, rather than internationally coordinated. The population growth described in this scenario most closely matches the United Nations (UN) *World Population Prospects: The 2008 Revision* constant fertility variant (i.e. 11.2 billion people by 2050), and the greenhouse gas emissions, which reach roughly 140 Gt CO_2-equivalent/year by 2100, are at the top of the range modeled.

In contrast, the B2 scenario family is one in which the world emphasizes the environment, social equality and welfare, and sustainability, especially at a local and community-based level. Technological diffusion makes the world slightly less heterogeneous in terms of technological

[7] ECHAM4 is a GCM developed by the Max Planck Institute for Meteorology in Hamburg
HADAM3 is a GCM developed by the UK Hadley Centre

[8] Data were received from Dr. Abel Centella (abel.centella@insmet.cu)
The Montserrat file name used is: point_298-16.5.csv

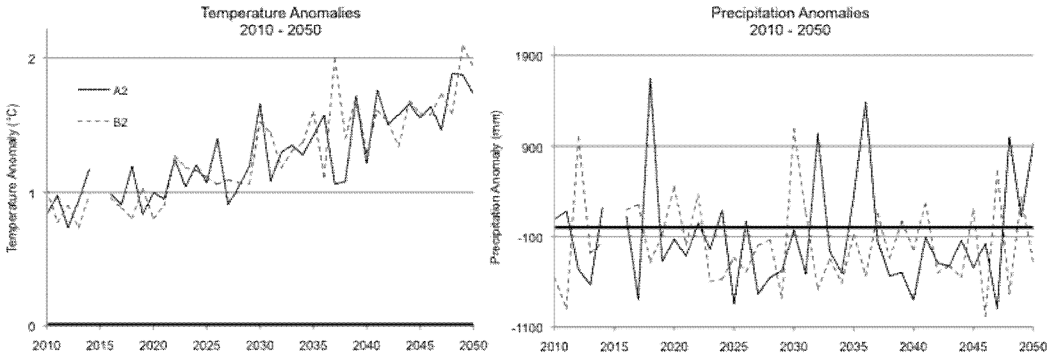
[9] It is important to note that the ECHAM precipitation data were approximately a factor of ten too small to be realistically in units of mm*month^{-1}, see . It was therefore decided to treat it as if it were in units of cm*month^{-1} and convert it to mm*month^{-1}. Attempts to verify these units did not receive a reply on the timeline available for this project.

changes and advances than in A2, but community innovation is emphasized more than global technology diffusion. The population growth described in this scenario most closely matches the United Nations (UN) *World Population Prospects: The 2008 Revision* medium variant (i.e. 9.4 billion people by 2050), and emissions fall in the middle of the modeled range, reaching approximately 60 Gt CO_2-equivalent/year by 2100.

Neither of these scenarios involves a significant effort to reduce dependence from fossil fuels and energy use remains "high" in A2 and "medium" in B2 (IPCC, 2001a, 2001b, 2001c, 2001d, 2001e, 2007; UN, 2008).

The projected down-scaled ECHAM precipitation and temperature anomalies are shown in figure 10. The precipitation anomalies show substantial annual variability without a clear trend with time, where as the temperature anomalies show much less variability and are noticeably increasing with time.

Figure 10: ECHAM4 down-scaled A2 and B2 Precipitation and Temperature Anomalies 2010-2050



Source: Data compiled by author

C. THE DOSE RESPONSE RELATIONSHIPS

The search results described in Part I returned a wide variety of dose-response relationships from the literature. However, only four were found suitable for this study (see Table 4). They pertained to malaria morbidity and mortality (Tol, 2008), gastroenteritis morbidity (Lloyd and others, 2007; Singh and others, 2001), and joint cardio-respiratory mortality (Hashizume and others, 2009). With the exception of the Lloyd and others (2007) dose-response relationship, all of these relationships relied exclusively on temperature changes to predict changes in disease incidence.

Table 4: This table shows the dose response relationships that were deemed useable in this study.

Diseases		Morbidity Dose-Response Relationship		Mortality Dose-Response Relationship	
		Relationship	Source	Relationship	Source
Malaria		0.015 days illness/↑1˚C	Tol (2008)	0.033 deaths/↑1˚C	Tol (2008)
Gastro.	**<5**	↑4%/↓10 mm rain	Lloyd and others (2007)		
	>5	↑3%/↑1˚C	Singh and others (2001)		
	Total Pop	↑3%/↑1˚C			
Cardiovascular Diseases				↑3.2%/↑1˚C	Hashizume and others (2009)
Respiratory Diseases					

Source: Data compiled by author

Before going into detail on the selection of relationships shown above, it is important to note that one key assumption made with these relationships is that they could be applied to annual temperature and precipitation anomalies even though they were not derived using data in this form. For instance, the gastroenteritis relationships shown in this table relate to a monthly percent change in disease incidence per unit temperature or precipitation change in the previous month. As the baseline gastroenteritis data were not consistently available in monthly increments, these relationships had to be generalized to changes occurring over time increments of one year.

Similarly, it is important to mention that the lack of relationships including precipitation should not be interpreted as implying that precipitation does not actually affect disease incidence. Rather it reveals the great extent to which these complex disease-environment systems have had to be simplified. It also demonstrates that the published relationships found either do not include precipitation in the simpler dose-response relationship calculations, or do not publish enough information about the precipitation relationships to make them immediately transferable to a new location, as is the case with the model of leptospirosis incidence in Reis and others (2008) and the precipitation half of the simultaneous temperature-precipitation modeling done by Singh and others (2001).[10]

1. Malaria dose-response relationship

The selection of the dose response relationship for malaria was highly informed by the literature discussed in Part I, section 1.2 and the debate surrounding the Lafferty (2009) publication. Entomological models were deemed unsuitable for use in this study for two reasons: 1) They required information on a great variety of environmental variables, none of which were available for this research; 2) They don't take into account the role that social and economic systems have on vector-borne disease transmission. The short-term regression and GIS-based models were similarly deemed unsuitable because they were mainly designed to focus on short term forecasting rather than longer-term projections. Although a weakness in terms of their applicability in this study, these models may have great potential for monitoring, predicting, and responding to future malaria outbreaks.

Several papers did attempt to address socio-economic factors in the context of malaria transmission, but were not useable in this study. Coleman and others (2010) used principal component analysis to assess the role played by various house-hold levels, non-environmental

[10] Singh and others (2001) discuses modeling the effects of temperature and precipitation simultaneously, but correspondence requesting that full relationship, rather than just the temperature side of the relationship did not receive a reply on the timeline afforded to this project.

variables on disease risk. Craig and others (2004) investigated the role that non-environmental variables are playing in the transmission of malaria in South Africa, and found associations between seasonal malaria cases, drug resistance and relative measures of HIV infection. Stratton and others (2008) provided a literature review of efforts to model malaria, and van Lieshout and others (2004) assessed the additional population at risk of transmission under various climate change scenarios. While these approaches are a fairly novel departure from the entomological modeling, none of them provided relationships that could be used, given the baseline health and climate projection data available for this study.

Tol (2008), however, presented exactly the kind of relationship required for this study. The dose response relationships featured in this publication focused on particular, well-defined regions and utilized the integrated assessment model *FUND*, rather than an entomological model. It is important to note, however, that Toll (2008) presents the total increase in the number of deaths and the number of days of illness per increase in degree Celsius for the Caribbean as a region, rather than for individual countries. In order to derive country-specific dose-response relationships from this larger, cumulative Caribbean relationship, it had to be assumed that Montserrat would contribute to the regional malaria incidence and mortality burden according to the proportion of the regional population that Montserrat represents. This also required assuming that the relationship derived fro Tol (2008) remains equally valid for the scenarios considered here (A2 and B2) as for the scenarios considered in the original publication.

2. Gastroenteritis dose-response relationship

Given the global importance of gastroenteritis incidence, and given that consensus has not been reached on the modeled relationships, it is interesting that the literature search performed a) did not return more studies for consideration, and b) did not reveal an active debate regarding the most appropriate way to model gastroenteritis using environmental variables as was seen in the literature for other diseases.

Despite the paucity of publications, however, Singh and others (2001) and Lloyd and others (2007) were selected for use in this study. These dose-response relationships were selected because they utilized environmental variables that were available for use in this study, and because they focused on all-cause gastroenteritis. The two studies focusing on hospital admissions in Peru - Checkley and others (2000) and Lama and others (2004) - were not chosen out of a concern that modeling hospital admissions might fail to capture the environment- gastroenteritis dynamic related to the more common, less severe instances of gastroenteritis. Additionally, Onozuka and others (2010) was not chosen because it was thought that the Pacific island data used in Singh and others (2001) would be a better environment-disease-adult-incidence proxy for Montserrat, while Medina and others (2007) was excluded because it was unclear how their model could be adapted to another location and because of the particular age-stratification of their data.

3. Cardio-Respiratory dose-response relationship

As discussed in Part I, significant volumes of research have focused on deriving relationships between temperature changes and cardio-respiratory mortality. However, many of these studies were not suitable for use in this study.

1) Heat-wave focused relationships were not utilized in this study because these relationships are not designed to model the impact of the long-term graduate temperature changes (Martens, 1997).
2) Age-stratified relationships, despite their intuitive appeal, were not utilized in this study because the baseline data available for this study are insufficiently stratified by age to apply one of these dose-response relationships.
3) Dose response relationships that attempted to take the effect of PM and ground-level ozone increases on mortality into account could not explicitly be included in this study either. There

is evidence that PM and ground-level ozone increase mortality both independently from, and synergistically with temperature (Daniels and others, 2000; Ebi and others, 2008; HEI International Scientific Versight Committee, 2004; Kovats and others, 2003a; UNDP/World Bank Energy Sector Management Assistance Programme, 2004). However, because the relationship between pollution and mortality varies by location, as does its synergism with temperature (Eb, and others, 2008; UNDP/World Bank Energy Sector Management Assistance Programme, 2004), and because pollution modeling was not included in the climate scenario data provided for this study, it was decided that there was no valid way to justify transferring this extra layer of complexity in addition to transferring a temperature-mortality relationship.

4) None of the many relationships have been derived from major cities around the globe could be utilized because the urban heat island effect can be significant in such environments, and because Montserrat's population is only a fraction of the size of many of these cities.
5) Finally, because the population of Montserrat resides predominantly at sea level, dose-response relationships from markedly higher altitudes were avoided out of a concern that such relationships may not be a good proxy for the temperature variations felt by populations living at sea level in the tropics.

Given these constraints, the relationship that most closely matched the requirements discussed previously for this study is the all-cause,[11] all-age, no-threshold relationship derived from data pertaining to rural Bangladesh as published in 2009 (Hashizume and others, 2009).

It is also worth highlighting that the dose response relationship connecting respiratory and cardiovascular diseases to temperature does not reference a threshold temperature. Because the temperature variation throughout the seasons in Montserrat is relatively small, it is reasonable that a relationship that does not have particular hot and cold thresholds that mark the initiation of increases in mortality (as have been found in temperate regions that experience much greater annual temperature swings) be used. Excessively hot or cold days, however, are still likely to cause greater than average respiratory and cardiovascular distress, and possibly fatalities, that cannot be taken into account using the relationship shown above.

4. Explanations for the other conditions

Dengue Fever: Although some models showed promise for short term predictions of outbreaks – something that highlights the importance of environmental and disease monitoring – no dose-response relationships have been published for dengue fever that could link temperature and precipitation projections to increased incidence of disease. As discussed in Part I, this is a likely consequence of the urbanization of the DF vector, as this reliance on human structures and patterns can trump the impact that temperature and precipitation change might otherwise have on the dengue fever vector.

Schistosomiasis: No dose response relationships could be found connecting temperature and/or precipitation to schistosomiasis incidence. Entomological models were found, but they could not be used in this study for the following reasons: 1) They require information on variables that were not available for this study; 2) none of these entomological models took account of socio-economic variables. Because these variables can dramatically affect the contact that humans have with infected water, and thereby can dramatically affect transmission/contraction of the disease (Kovats and others, 2003e).

[11] Although this risks slight double-counting in terms of the effects of temperature on other diseases, all-cause mortality was considered to be a good proxy for cardiovascular and respiratory mortality because these two categories of disease constitute the primary causes of mortality in both the Caribbean and Bangladesh

Leptospirosis: Only one paper found by the literature search presented a dose response relationship linking leptospirosis incidence to precipitation (Coedço and others, 2008). However, unresolved questions regarding the scale of rainfall used and the formal dose-response equation in combination with other research - (Reis and others, 2008) - that highlights the role that 1) local sanitation infrastructure and 2) exposure to rodent populations each play in transmission, prevented the use of Coedço and others (2008) in this study.

Ciguatera Poisoning: Chateau-Degat and others (2005) did present a 2-stage relationship between SST and ciguatera poisoning. This model had potential because low-level air temperature can serve as a proxy for SST. However, this model failed to capture the peaks in ciguatera poisoning, something which undermined its transferability to the Caribbean. Similarly important in terms of the transferability of this model to the Caribbean is the comparability of the dinoflagellate populations and the ciguatoxins in the Pacific to those in the Caribbean. There is strong evidence that both the dinoflagellate communities producing the ciguatoxins, and the toxins themselves, are different in the Caribbean to the Pacific (Fleming, 2010), meaning that they could have different reactions to changes in SST and different impacts on the local human populations. Therefore, despite the appeal and potential of this model, it could not be transferred for use in this study.

Men. Meningitis: These papers identified through the literature search do give credence to the idea that MM is sensitive to environmental variables that may be altered with climate change. However, these papers also make it clear that there is insufficient causal evidence regarding the environmental mechanisms that influence any of the serogroups of MM to be able to use the outputs of climate change models to project a changing MM disease burden.

IV. SUMMARY RESULTS FROM THE PROJECTION STAGES

A. BASELINE MORTALITY & INCIDENCE RATES

The disease data relating to the period 1980 – 1990 were used in combination with each country's population data to calculate baseline morbidity and mortality rates for each of the discussed diseases. The 1980 - 1990 time frame was chosen because it overlaps with the 1960 – 1990 baseline time period in the climate model data, which was chosen because data in this time period are much less likely than later data to have been affected by anthropogenic climate change. The only exception to this is the gastroenteritis data for people over the age of five, all of which occurred after this period. Estimates for gastroenteritis occurring in this age group during the baseline period were calculated using the average of the ratio of the above age 5 data to the under age 5 cases for the time period 1994 – 2000, as applied to the baseline under age 5 data. Although this introduces additional uncertainty, this was done in order to be able to apply dose response relationships later in this section. The baseline incidence and mortality rates calculated are shown in table 5 below:

Table 5: This table shows the baseline incidence and mortality rates derived from the baseline population and health data. All rates are presented as values per 100,000 people. Grey cells indicate no available data.

Diseases		Incidence Rates	Mortality Rates
Malaria		0	0
Dengue Fever		0	0
Gastroenteritis	<5		
	>5		
	Total Pop	1894.58	
Schistosomiasis			0
Leptospirosis		3.32	0
Ciguatera Poisoning		115.11	
Meningococcal Meningitis		0	0
Cardio. Diseases			265.50
Respiratory Disease		1668.14	
Malnutrition			14.17
Extreme Events		3373.43	3.08

Source: Data compiled by author

These baseline rates are important for two reasons: 1) they define the disease-population relationships that serve as a reference that is assumed to be unaffected by climate change; 2) these are the rates that are adjusted using the dose-response relationships to project disease incidence under climate change.

Importantly, the dose response relationship chosen for malaria is unique in that it does not rely upon adjusting the baseline rate. Instead it connects disease burdens directly to temperature changes as modeled in the A2 and B2 scenarios.

Recall from section 3.3.1 the relevant information on malaria incidence for the baseline is based on the findings in Tol, 2008, which provided a regional incidence that could then be pro rated to the Montserrat population and then forecast forward using the dose-response relationship in the same study, which is the most amendable dose-response function for our purposes. This is why a burden has been projected despite the absence of the disease in our Montserrat baseline window. This is not implausible since we note that the disease has previously been present in Montserrat and the conditions for the vector are favourable. Re introduction (albeit at low projected infection) is a potential scenario, since regional population movements make it possible for the infected vector or individual to be present on the island.

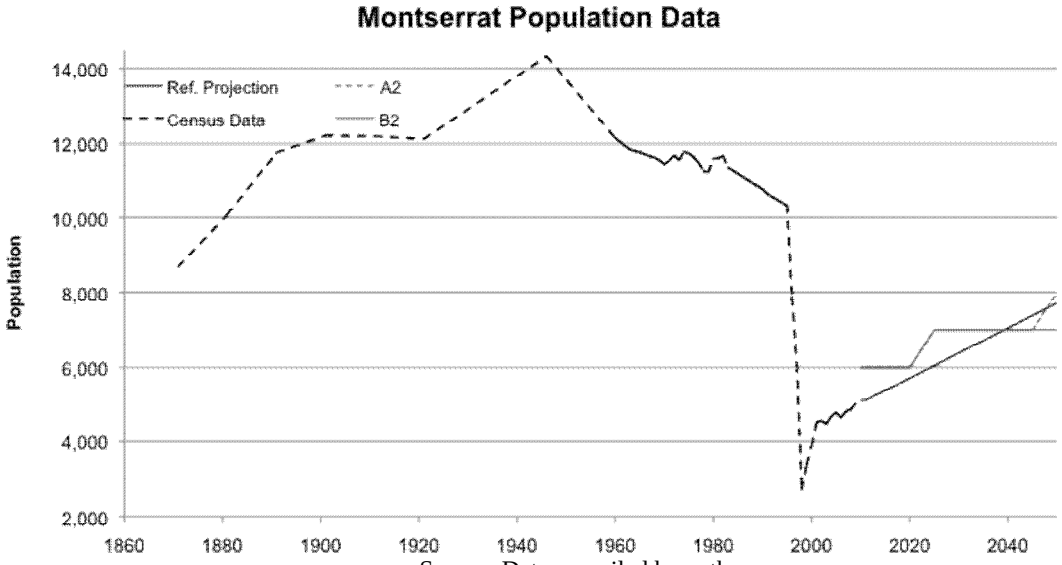
With the exception of malaria, any condition that has a zero baseline rate will be projected to have zero future as well, meaning that they will not contribute anything to the valuation of the projected excess disease burden in the A2 and B2 scenarios. However, as it is possible for diseases to be re-introduced to islands in the Caribbean, and as it is possible that other conditions like MM, leptospirosis, and schistosomiasis may have been under-reported in the baseline period, these rates of zero should not be interpreted to indicate that this disease will not be present in the future.

B. POPULATION PROJECTIONS

In order to project disease burdens, population had to be projected forwards in time through 2050. The population projections for the A2 and B2 scenarios were sourced from the UN projection variants that most closely match these climate scenarios. The reference population projection assumes that current population trends in Montserrat have not yet been affected by climate change, and therefore represents a counterfactual population scenario to the A2 and B2 population projections. The reference projection for the total population was generated by using a linear regression on the 2001-2010 census

data (Fenton, 2010b). Importantly, no age-stratified information was available to break the A2 and B2 projections, into the under-5 and over-5 age categories. This means that all projections are based only on total population for Montserrat. Figure 11 shows the baseline population data, as well as the reference, A2 and B2 population time series for Montserrat.

Figure 11: Montserrat Population Data (Baseline and Projected).
The line that extends up until 2010 is the total population census data. The darker solid line that follows most immediately on from the census data is the total population reference projection. The lighter grey lines are the A2 (dashed line) and B2 (solid line) projections. The A2 projections show a greater population growth at the end of the period in question, but for the majority of the projection period is the same as the B2 population projection.



Source: Data compiled by author

C. EXCESS DISEASE INCIDENCE

Disease burdens were projected for the reference, A2, and B2 scenarios as explained in table 2. Projecting those diseases for which dose response relationships had been found involved altering the baseline morbidity/mortality rates according to the dose response relationship and the climate model anomaly data. Projecting those diseases for which no dose response relationship could be found involved multiplying the baseline morbidity/mortality rates by the projected population values in order to generate a conservative estimate of the disease burden caused by these conditions. Following these projections, the excess disease burden for each year in the 2010-2050 time period was calculated by subtracting the projected reference disease burden from each of the projected climate change scenario disease burdens.

Although the projected impact for Montserrat appears to be relatively small, with no more than 500 extra cases and 100 extra deaths resulting from any of the diseases considered, it is worth reiterating that these numbers depend on the assumptions made throughout the steps shown in table 2, and are burdened by the uncertainties inherent to the data. It is also worth reiterating that this projection of excess disease implicitly holds most socio-economic variables constant over the 2010-2050 period. Time series of projected disease incidence and cumulative disease incidence graphs are available in Appendix IX, and these results agree generally with Amarakoon, D., and others (2004).

Table 6: The projected total excess disease burden 2010-2050.
Values <1 show 2 decimal places. Values >1 are Rounded to the nearest whole number. Unless otherwise indicated, the units are number of cases (morbidity) or deaths (mortality). All rates are presented as values per 100,000 people

2010-2050 Total Mean Disease Burden Anomaly		**Morbidity Anomaly**		**Mortality Anomaly**	
Diseases		**A2**	**B2**	**A2**	**B2**
Malaria		0.76	0.76	2	2
Dengue Fever		0	0	0	0
Gastroenteritis	<5				
	>5				
	Total Pop	484	427		
Schistosomiasis					
Leptospirosis		0.50	0.37	0	0
Ciguatera Poisoning		17	14		
Meningococcal Meningitis		0	0	0	0
Cardio. Diseases				70	62
Respiratory Disease		253	203		
Malnutrition				2	2
Extreme Events (# affected, deaths)		511	410	0.47	0.37

Source: Data compiled by author

V. VALUATION OF THE EXCESS STATISTICAL LIFE BURDEN

A. INTRODUCTION

Climate change will lead to a combination of damages that can be measured in terms of a combination of both market and non-market values. As a prior to adaptation decisions using cost-benefit analysis (CBA), it is necessary to convert the predicted health burden into a consistent metric that can be compared with cost.[12] This section concentrates on the valuation of the specific non-market values related to excess mortality and morbidity, which provides the benefit estimate that can conversely be interpreted as an avoided cost (or return) to any prospective adaption costs in terms of pre emptive health care intervention.

A common approach to health care impact assessment is use of the WHO's Disability Adjusted Life Years as a consistent metric of health gains or losses. Because of data limitations for Montserrat (i.e. a lack of data on impacts stratified by age), this report was unable to undertake a calculation of non-monetized measures using DALYs. As an alternative a monetary approach was adopted, where these costs can be quantified using benefits transfer values for a statistical life (VSL).

A CBA focusing on trade-offs between specific healthcare adaptation policy options is the ultimate objective of this study. This section proceeds with a brief discussion of the VSL concept and of benefits transfer (BT). This is followed by the application of the VSL concept to the valuation of the excess climate change-induced disease burden that was projected in section 7. The section culminates in the estimation of the aforementioned excess, non-market, climate change-induced human health costs for Montserrat for the time period 2010 – 2050. The final part of the section includes a discussion of the results.

[12] Or be expressed as cost per health unit

B. VALUING STATISTICAL LIVES & THE USE OF BENEFITS TRANSFER

1. Statistical lives

At its most basic, the concept behind valuing a statistical life is that individuals (and whole societies) can and do place a value (both implicitly and explicitly) on changes to the statistical risk of death experienced in day-to-day settings. This happens both because health risks affect individual and social welfare, and because individuals and societies have to make economic trade offs due to income constraints (Lindhjem and others, 2010; Marquez, 2006).

In some sectors of analysis, revealed preference methods can be utilized to assess what real human behavior demonstrates about various groups' valuation of mortality risks. However, this type of analysis focuses on situations where data documenting real income-safety trade offs has already been generated, meaning that consideration of the same type of trade off in the context of environment- or policy-driven changes to future health risks (like climate change and adaptation) requires that stated preference methods be used to elicit the valuation (Lindhjem and others, 2010).

As Lindhjem and others (2010) explain, the VSL derived from these stated preference studies represents the marginal value of mortality risk reduction and is defined as the *rate* at which people are willing and able to trade income for a reduction in a statistical mortality risk without a decrease in welfare or utility, as shown in equation (1) below (where WTP represents the willingness-to-pay for the change in mortality risk, R, found in the denominator of the equation).

$$VSL = \frac{\partial(WTP)}{\partial(R)} \qquad (1)$$

Performing this calculation with regards to the reduction in mortality risk that corresponds to one fewer statistical persons dying in a certain activity/context within a given population makes the identified rate at which people are willing to trade individual income for that statistical mortality reduction equivalent to the social welfare value of a single statistical life (in that population and in that risk context). Therefore, the VSL applied in a given context does not actually represent the monetized *worth* of a living human being, but rather is a measure of the *rate* at which real human beings value marginal statistical mortality reductions in particular contexts in relation to income. This is therefore an inferred value.

This distinction is key to the utilization of a nationally derived VSL in a CBA framework because it means that inclusion of the VSL in the CBA allows decision-making to consider the costs of implementing health-related policies alongside quantified, non-zero, non-market, and very real social preferences pertaining to the mortality-related outcomes of those policies, which would otherwise be implicitly valued at zero and have no impact on policy decisions.

This also means that the VSL derived from stated preference studies need not be the same in every context. Within one location, or one population, for instance, the average stated rate at which people are willing to trade their income for a statistical reduction in mortality risk on the highway may not be the same as the average stated rate at which those same people are willing to trade their income for a statistical reduction in the risk of dying as a result of food poisoning or cancer or flying in an airplane. Population-specific variables such as culture, political system, values, perception of risk, and existing infrastructure can affect the extent to which the VSL calculated for each of these activities may differ from each other within a given population (Ready and Navrud, 2006).

Importantly, however, just because it is theoretically possible for the VSL calculated for different categories of mortality risk within one population to vary, does not mean that this is inherently the best explanation for any variation seen in published VSLs for a particular nation. As several analyses

focusing on meta-regression reveal, study design and various methodological decisions often have a significant impact on the end VSL generated by research efforts. Among other methodological variables, how stated preferences are elicited (ie over the phone, in person, on paper, etc), the use of any visual aids, the order in which various risk reduction scenarios are considered, and the vocabulary used to describe the hypothetical choices that are presented to study participants can all bias the final VSL (Barton, 2002; Bellavance and others, 2009; Lindhjem and Navrud, 2008; Lindhjem and others, 2010; Mrozek and Taylor, 2002).

This means that careful consideration of methodological variables is important when attempting to evaluate the extent to which different VSL numbers actually represent different social preferences in different risk contexts (i.e. highway safety, food safety, environmental hazards, etc.) versus the extent to which they represent different methodological choices made by researchers.

Another important ramification of stated preference methodology, which focuses on the creation of a hypothetical market in which WTP is used to value nonmarket goods, is that because the VSL in stated preference studies is derived from a budget- (and purchasing power) constrained WTP, the VSL generated by research will vary from location to location throughout the world and will generally be higher in countries with higher national and per capita incomes (Lindhjem, and others, 2010; Ready and Navrud, 2006).

Although at a superficial level this may seem to indicate that the lives of those in richer contexts are worth more than the lives of those living in poorer contexts,[13] all that this actually indicates that the people living in richer places, and countries with wealthier governments, can afford, in an absolute sense, to trade more money without a reduction in economic welfare, for a given reduction in mortality risk, than can people living in poorer places or countries with poorer governments. In turn, this has ramifications for the ways in which primary study VSL results can be transferred for application in different geographical contexts when primary stated preference studies cannot be undertaken.

2. Benefits transfer

The transfer of a VSL from one country to another (one form of international benefits transfer) requires adjustment for the differences in the income of the study and application site in terms of inflation through time, currency, and purchasing power in order to make the transferred VSL relevant in the decision-making context of the application site (Czajkowski and Ščasný, (2010); Ready and Navrud, 2006; Stellin and Candido, 2006). This can be done in a couple of ways, depending on whether what is transferred is a unit VSL or a VSL-generating function. A benefits transfer function generally takes the form of a linear combination of multiple explanatory variables pertaining to study design, socio-economic variables, and the good being valued, and adapting a benefits transfer function from one location to the other typically requires assuming that the relative impact of all these variables (i.e. the coefficients) on the dependent variable (VSL) is the same in each location.

Thus the framework of the function stays intact across all locations, and the numerical value of the independent variables is specified on a location-by-location basis. Income can either be taken explicitly into account in the function as one of the independent variables, or will implicitly be taken into consideration through the site-specific values of the other socio-economic variables included in the equation (Lindhjem and others, 2010; Ready and Navrud, 2006).

However, while conceptually appealing, utilizing such functions can generate a false sense of accuracy in the results, as not every function is capable of transferring to every location equally well. If, for instance, for a particular VSL-generating function, the assumption that the relationship between the independent variables and the dependent variable is constant with location being false, then that particular function will be non-transferable to other locations (Brouwer and Bateman, 2005).

[13] This misinterpretation caused heated debate in an early IPCC assessment.

Similarly, if the range or variability of the original explanatory data is narrow, then transferring the resulting function to a context in which the independent variables are either outside the range of the original data, or more variable than the original data would likely generate a large transfer error and could therefore mislead decision-makers (Ready and Navrud, 2006).

Also, although functions have the potential to have significant explanatory power as a consequence of utilizing more than one independent variable, no single function includes all the possible explanatory variables, which means that transferring a function from one context to another where a particular variable is important, but not represented as such in the transfer function, will also yield inaccurate results (Stellin and Candido, 2006). Meta-analysis of multiple studies containing VSL derivations has the potential to get around some of these problems by statistically pooling data from a wide variety of sources and re-deriving explanatory relationships, but there is not yet a consensus on their utility in terms of generating benefit transfer functions (Johnston and Rosenberger, 2010; Lindhjem and Navrud, 2008; Lindhjem and others, 2010).

Transferring a unit value for the VSL from one country to another involves scaling that value by the ratio of the purchasing power parity (PPP)-adjusted per capita incomes of the application and original study site. The purpose of this is to ensure that the purchasing power of individual nations is taken into account when transferring a VSL from one location to another. This method assumes that the VSL varies exactly proportionally with income, something that may or may not be true. Some authors advocate raising the ratio of the PPP-adjusted incomes to a number representing the income elasticity of WTP as a way of altering this assumption, but justifying a value other than one for the income elasticity of WTP is difficult, especially in instances where the unit VSL is being transferred between locations with a significant income disparity (Czajkowski and Ščasný, (2010); Ready and Navrud, 2006). Importantly, as is the case with the transfer functions, the transfer of unit values can result in significant errors if the original study conditions and the transfer site conditions are not well matched (Stellin and Candido, 2006).

Given all of this, and considering that international benefits transfer had to be utilized in this study, it was deemed that the most appropriate source for either a unit VSL to transfer or a transfer function for this study was the recent meta-analysis by Lindhjem and others (2010), which incorporated nearly every stated preference study to date on environment-, health-, and travel-risk related VSLs.

C. DERIVING THE STATISTICAL LIFE TIME SERIES

Lindhjem and others (2010) was chosen as the source of the VSL estimate because it is recent, because it does not require age-stratification for the VSL estimation, and because it appears to be the most exhaustive treatment to date of VSL estimates derived from environmental and health-related studies. The transfer method utilized was the income-weighted unit value approach discussed earlier, and the mean value for the US Health VSL estimates contained in Appendix two of Lindhjem and others (2010) - 4,808,000 PPP-adjusted 2005 US$ - was ultimately chosen as the figure subjected to income-adjustment. This number was chosen because it was derived from 24 studies performed in a country with readily available PPP-adjusted GDP data, and because it was also close to being the overall average out of all the mean health VSL values presented in Appendix two of Lindhjem and others (2010).

In order to calculate the VSL estimate for Montserrat, $4,808,000 was the US health VSL had to be deflated using the US Bureau of Labor and Statistics CPI calculator to 2002 levels, [14] and the following income-adjustment (assuming an income elasticity of one) was performed using data available from the *CIA World Factbook* (CIA, 2003; CIA World Factbook, 2010):

[14] It was deemed appropriate to use the US CPI to adjust this figure because the CPIs had been used by the authors originally to inflate / deflate VSL estimates to 2005 levels

$$VSL_M = \frac{PPP - Adjusted\ 2002\ Per\ Capita\ GDP_M}{PPP - Adjusted\ 2002\ Per\ Capita\ GDP_{USA}}\ X\ \$4,428,875 \quad (4)$$

$$VSL_M = \frac{\$3,400}{\$37,600}\ X\ \$4,428,875 = \$400,483\ in\ 2002\ US\$ \quad (5)$$

The VSL estimate was then scaled to 2010 levels using the CPI calculator, and from this, a VSL time series was generated, assuming an annual inflation of 2% from 2010 – 2050.[15]

D. APPLYING THE STATISTICAL LIFE TIME SERIES

The VSL time series was multiplied with the time series of mean mortality and morbidity anomalies discussed previously in order to generate the statistical life valuation of the excess disease due to climate change under the A2 and B2 climate change scenarios.[16] In addition to including those diseases that were projected using dose-response relationships, this part of the analysis also included those diseases for which no dose response relationship was available. The contribution of these diseases to the VSL burden could estimated using the A2 and B2 scenario population projections and the baseline mortality and morbidity rates. Mortality was valued straightforwardly by multiplying the number of deaths each year, for each disease, by the corresponding VSL estimate from the time series.

Morbidity was valued, but in somewhat of an *ad hoc* way as commensurate WTP values for these conditions could not be found and the goal was to generate an overall VSL-based estimate for the excess impact of climate change on health.

The value of morbidity was assumed to be proportional to the average fraction of the average life expectancy taken up by one non-fatal bout of each disease. The average duration was found for each of the following: schistosomiasis, DF, gastroenteritis (children), gastroenteritis (adults), respiratory influenza, post-disaster mental health trauma, ciguatera poisoning, and malaria. These durations were found to be, respectively, five years (NHS, 2005), ten days (CDC, 2010), three days (Every Day Health, 2009b), seven days (Every Day Health, 2009a)[17], five days (*Inteli*Health, 2008), one month (Galea and others, 2005), six months (Fleming, 2010), and one year.[18]

These illness durations were divided into the *CIA World Factbook* listed life expectancies for Montserrat and then multiplied by the VSL estimate time series to generate estimates for the yearly "partial" VSL value represented by each of these conditions. These "partial" VSLs were then multiplied by the incidence time series for the corresponding disease to complete the valuation. The morbidity and mortality valuation results were summed together within each year and discounted in reference to years after 2010 using discount rates of 1%, 2%, and 4%, as was requested for this analysis. The results of this process are shown in tables 7 and 8.

Table 7 provides an overview of the extra VSL burden resulting from climate change under each of the two scenarios considered.

Table 8 provides a detailed breakdown of the results shown in table 7.

---

[15] This assumption came from the following online projections for inflation

[16] Only the mean disease projection time series was utilized because the lower and upper bounds shown in the figures in section 7 only represent the uncertainty in the dose response functions

[17] In the case of Montserrat where gastroenteritis incidence was not stratified by age, the average of the child and adult partial VSLs was used (i.e. 5 days out of the average life span)

[18] The morbidity values in from Tol (2008) represent the number of diseased *years* experienced per degree Celsius increase in small island states, not malaria incidence per se. Thus the fraction of an average life that 1 year represents was found and multiplied by the VSL to generate the "partial" VSL value for malaria incidence.

Reading down the columns in table 8, the results are first separated into that VSL burden caused by morbidity and mortality. Within each of these categories, the results are further categorized by the climate change scenarios utilized (A2 and B2). Within the results for each climate change scenario, the results are divided by discount rate (1%, 2%, or 4%).

Reading across the rows in table 8, results are presented for each of the diseases discussed earlier. The following disease abbreviations are used:

- GE: Gastroenteritis
- Schist: Schistosomiasis
- Cig. Poison. Ciguatera Poisonong
- Men. Men. Meningococcal Meningitis
- Cardio. Diseases Cardiovascular Diseases
- Resp. Diseases Respiratory Diseases
- Malnut. Malnutrition

Within each disease, there are 3 rows (labeled 1, 2, &3) that represent the following:

- Row 1: The cumulative present value of the statistical life burden
- Row 2: The mean annual value of this statistical life burden derived from Row 1
- Row 3: The mean annual statistical life burden per capita derived from Row 1

Essentially, these rows present the same category of information as is shown in the rows on table 7, but are presented for each disease, discount rate, climate scenario, and category of impact (morbidity vs. mortality)
Gray boxes in table 8 indicate that no projections could be made. Boxes with zeros and diagonal lines through them indicate that a zero value has been projected purely because zero incidence/mortality occurred during the baseline period. It is important to note that a disease having zero incidence/mortality in the baseline does not necessarily mean that there will be zero incidence of this disease or mortality from this disease in the future.

It is also worth re-iterating here that the quantitative results of this study should be treated as a starting point, rather than an ending point, and that the method presented in this report can be used iteratively to systematically target data gaps. This will change the results presented in these tables, and should improve the confidence in the numbers generated by this process.

Table 7: VSL anomaly burden range 2010-2050.
This table shows the maximum range of the mean extra VSL burden generated by climate change under two climate change scenarios. The top number in each cell shows the lower bound calculated for that particular category of impact. Each lower bound was calculated using the 4% discount rate. The bottom number in each cell shows the upper bound, which was calculated using the 1% discount rate. The overall VSL impacts are smaller under scenario B2 than A2. The numbers in red form the range presented in the abstract to this report.

Mean Cumulative VSL Anomaly Burden 2010-2050	**A2**			**B2**		
	Morbidity	**Mortality**	**Total**	**Morbidity**	**Mortality**	**Total**
Range Type	**4% DR – 1% DR**	**4% DR – 1% DR**	**4% DR – 1% DR**	**4% DR – 1% DR**	**4% DR – 1% DR**	**4% DR – 1% DR**
Cumulative PV Range	336,957 – 464,724	26,889,115 – 42,277,446	27,226,072 – 42,742,170	300,182 – 350,974	24,872,598 – 36,196,082	25,172,780 – 36,547,056
Mean Annual Range	8,218 – 11,335	655,832 – 1,031,157	664,051 – 1,042,492	7,322 – 8,560	606,649 – 882,831	613,970 – 891,392
Mean Annual Range per Capita	1 – 2	97 – 153	98 – 154	1 – 1	91 – 132	92 – 133

Source: Data compiled by author

Table 8: Detailed VSL anomaly results as broken down by type of impact (mortality vs. morbidity), climate change scenario (A2 vs. B2), and discount rate (1% vs. 2% vs. 4%). Thisbreak down is shown for each disease considered, and within each disease results are shown for the cumulative present SL impact, the mean annual SL impact, and the mean annual per capita SL impact. Because the mortality burden for cardiovascular and respiratory diseases was calculated with those two categories merged, they are merged in this table as well. See preceding text for further information.

Montserrat Diseases		Morbidity Anomaly SL Values						Mortality Anomaly SL Values					
		A2			B2			A2			B2		
		1% DR	2% DR	4% DR	1% DR	2% DR	4% DR	1% DR	2% DR	4% DR	1% DR	2% DR	4% DR
Malaria	1	$6,732	$5,059	$3,336	$6,448	$5,101	$3,337	$1,019,956	$809,753	$533,920	$1,032,133	$816,479	$243,181
	2	$155	$123	$81	$157	$124	$81	$24,877	$19,750	$13,022	$25,174	$19,914	$13,029
	3	$0.02	$0.02	$0.01	$0.02	$0.02	$0.01	$3.68	$2.92	$1.93	$3.77	$2.98	$1.95
Dengue Fever	1	0	0	0	0	0	0	0	0	0	0	0	0
	2	0	0	0	0	0	0	0	0	0	0	0	0
	3	0	0	0	0	0	0	0	0	0	0	0	0
GE	< 5												
	> 5												
	T	1	$52,011	$44,259	$33,229	$44,324	$38,980	$30,685					
		2	$1,269	$1,079	$810	$1,081	$951	$748					
		3	$0.19	$0.16	$0.12	$0.16	$0.14	$0.11					
Schist													
Lepto.	1	$931	$839	$688	$688	$672	$610	0	0	0	0	0	0
	2	$23	$20	$17	$17	$16	$15	0	0	0	0	0	0
	3	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	0	0	0	0	0	0
Cig. Poison.	1	$64,541	$58,122	$47,713	$47,683	$46,605	$42,275						
	2	$1,574	$1,418	$1,164	$1,163	$1,137	$1,031						
	3	$0.23	$0.21	$0.17	$0.17	$0.17	$0.15						

Men. Men.	1	0	0	0	0	0	0	0	0	0	0	0	0
	2	0	0	0	0	0	0	0	0	0	0	0	0
	3	0	0	0	0	0	0	0	0	0	0	0	0
Cardio. Diseases	1							1	$39,849,662	$33,838,456	$25,314,441	$34,123,854	$29,905,592	$23,416,267
	2													
	3							2	$971,943	$825,328	$617,425	$832,289	$729,405	$571,128
Resp. Disease	1	$25,625	$23,077	$18,944	$18,932	$18,504	$16,785							
	2	$625	$563	$462	$462	$451	$409	3	$144	$122	$91	$125	$109	$85
	3	$0.09	$0.08	$0.07	$0.07	$0.07	$0.06							
Malnut.	1							$1,156,357		$1,041,355	$854,851	$854,310	$835,009	$757,433
	2							$28,204		$25,399	$20,850	$20,837	$20,366	$18,474
	3							$4.17		$3.76	$3.09	$3.12	$3.05	$2.76
Extreme Events	1	$315,244	$283,892	$233,048	$232,900	$227,638	$206,490	$251,470		$226,461	$185,902	$185,785	$181,587	$164,717
	2	$7,689	$6,924	$5,684	$5,680	$5,552	$5,036	$6,133		$5,523	$4,534	$4,531	$4,429	$4,4017
	3	$1.14	$1.02	$0.84	$0.85	$0.83	$0.75	$0.91		$0.82	$0.67	$0.68	$0.66	$0.60

VI. DISCUSSION

A. STUDY OUTCOMES

As was the case with the disease projections, data contributing to table 8 show considerable variability in their yearly time series. As was the case with the disease incidence time series, this is the direct result of changes in both the year-to-year interaction of the projected reference population with the A2/B2 population sizes, and the climate anomaly data. The PV trend, however, is smoother for each disease. This indicates that while the rate of cumulative incidence is not projected to be constant with time, the overall trend of increasing cumulative disease burden (and therefore increasing VSL costs) continues throughout the period in question.

Importantly, the only times when the projected all-cause yearly VSL burden is projected to be negative are those times when the reference population exceeds the A2/B2 population. This highlights the influential role that population projections play in this study as well as a need for further research considering the extent to which future population numbers will be independent of climate change, particularly in small tropical islands. Ideally, studies that focus on populations whose growth is completely independent of climate change should control for population growth in order to reveal the isolated impact of climate change on health risks. However, given the small area of these islands, the fact that most of their population lives on the coast in the areas most vulnerable to all the anticipated effects of climate change, and that these tropical islands are likely to be impacted earlier and more severely by climate chance than are more temperature places, this study opted to treat the projected population as being at least partially dependent on, and a consequence of climate change, and so did not strictly control for its effects on either disease incidence or the VSL burden felt by the island.

The PPP-adjusted US$ values shown in tables 7 and 8, reveal a nontrivial impact, especially considering that the statistical life costs represent only the value of the indirect, nonmarket costs of human life part of the total economic costs that will be incurred by each of these fatalities or incidence of disease.[19] Note also that the baseline health data may have been under-reported, and that only four dose-response relationships were available. This all suggests that these numbers most likely represent a lower bound figure on the economic costs that could be incurred, given all the assumptions behind the disease projection efforts, in terms of human health as a result of climate change in the absence of adaptation.

The extent to which they actually are lower bound figures, however, depends on several variables, including the VSL time series utilized in the projections. The unit VSL transfer was performed without undertaking a formal statistical evaluation of its suitability for transfer due to time constraints on this study. It is therefore possible that a transfer error was generated as a consequence of this decision. Additionally, modeling the VSL, with a hypothetical inflation effect, and three significantly different discount rates for such an extended time period generate variation in the results. On this time scale, a different choice of base year, discount rate, initial VSL estimate, or projected inflation rate would noticeably change ranges presented in tables 7 and 8. The impact of the discount rate, for instance, is clear from the data in Table 7. This is a direct result of the fact that the VSL loses ~80% of its undiscounted wealth by 2050 when discounted at a rate of 4%, and it has potential

[19] Examples of the direct costs were found for a handful of the diseases considered here. See http://www.dcp2.org/page/main/BrowseInterventions.html & Ebi (2008a), "Adaptation Costs for Climate Change-Related Cases of Diarrhoeal Disease, Malnutrition, and Malaria in 2030," *Globalization and Health* 4: 9, Markandya and Chiabai (2009), "Valuing Climate Change Impacts on Human Health: Empirical Evidence From the Literature," *International Journal of Environmental Research and Public Health* 6: 759 - 786. However, the dcp2.org data was mostly displayed in units of cost per DALY avoided, which made it unusable in this study. Also, as requests for data on the actual expenditures for these various conditions in Montserrat proved unsuccessful, and given that the Caribbean as a region contains countries with widely varying purchasing power, it was deemed that there wasn't enough information to justify transferring one set of the other direct costs to this study.

negative ramifications not only in terms of the decision-making rules utilized in CBA, but also in terms of intergenerational equity (Marquez, 2006). The sensitivity of the results in tables 7 and 8 to the assumptions made highlights the importance of making ones assumptions clear in economic valuation efforts.

These results could also be significantly changed by improved baseline data, improved population projections, more location-specific dose-response functions covering a wider variety of conditions, and revised climate model data. For instance, DF was assigned a fairly small statistical life impacts in this analysis. This is largely due to the baseline data indicating a negligible incidence rate (that itself was the result of previous intense eradication efforts), and of the available modeling tools being complicated and ill suited to long-term predictions. Consequently, the absence of a significant burden from these diseases in the valuations in this section cannot be taken to indicate that the disease burden from these diseases will necessarily be negligible.

Considering the island's suitability for vector borne diseases, effort should still be put into investigating currently functioning examples of early warning/forecasting systems for vector-borne diseases like dengue fever, as well as surveillance programs, separate from the context of these results and any other long term modeling efforts. See: (Brunkard and others, 2008; Fuller and others, 2009; Halide and Ridd, 2008; Hu and others, 2010; Kuhn and others, 2005; Luz and others, 2008; Sutherst, 2004). The same idea holds true for diseases like leptospirosis and ciguatera poisoning that did not get recorded in the baseline data, that are notoriously under-reported, that do exist in noticeable quantities outside the baseline data range, and for which diagnosis is difficult.

Another important feature of this valuation effort is that it was unable to explicitly or implicitly account for air pollution and short-term heat waves, despite their likely significance during this time period (Ebi and others, 2008; McMichael and others, 2004), and so may under-represent the cardio-respiratory related statistical life costs of climate change. Conversely, it is also possible that the statistical life burden represented here due to gradual temperature change (cardiovascular and respiratory mortality) is significantly over-stated. The reason behind this is that there is the potential for the rate of increase in mean annual temperature to be matched by the rate of physiological adaptation to increased mean temperatures. In the absence of any additional projections of the health impacts of extreme heat events, such autonomous physiological adaptation would significantly decrease the statistical life burden discussed here, as shown in figure 21. This would be particularly true if the physiological adaptation to increases in temperature was accompanied by behavioral adaptation designed to decrease cardiovascular and respiratory health risks (Albert and others, 2007; Cunningham-Myrie and others, 2008; Freeman and others, 1996; Kovats and others, 2003b; Laaidi and others, 2006; Leary, 1999; Lewis, and others, 2000; McMichael and others, 2004).

Two final sources of uncertainty worth mentioning are: 1) the value assigned to the projected morbidity; 2) the assumption of stable social preferences with time. With regards to morbidity, in the absence of stated preference studies confirming the order of magnitude of these numbers, it is possible that the morbidity valuations are greater than they should be for the given amount of disease projected by this study. Regarding the assumption of stable social preferences, it is worth mentioning that while the it had to be assumed that the preferences of Montserrat were captured by the VSL estimates chosen, and that these preferences remained constant with time, it is not necessarily the case that social preferences will remain unchanged in the Caribbean once the impacts of climate change begin to become more noticeable. Any shift in social preferences will necessarily imply a shift in the rate at which either of these island populations is willing to trade income for a health risk reduction. This is something that would change the VSL deemed most appropriate for use in these islands, and would consequently affect the valuation results of studies like this one. Frequent re-evaluation of economic valuation efforts, therefore, will be necessary to ensure that social welfare and social preferences are not only recognized, but also kept up to date in the context of decision-making in the decades to come.

B. ADAPTATION OPTIONS:

As noted, the principal aim of deriving damage projections is to compare these with potential adaptation costs of measures designed to obviate impacts.

Adaptation can be classified in a number of ways. Hallegate (2009) suggest five categories (i) selecting "no-regret" strategies that yield benefits even in absence of climate change; (ii) favouring reversible and flexible options; (iii) buying "safety margins" in new investments; (iv) promoting soft adaptation strategies, including long-term prospective; and (v) reducing decision time horizons.

Within the categories of Hallegattes (2009) it is possible to make further distinctions between direct and indirect adaptations, and between public versus private responsibility. The first distinction relates to the efficiency of intervening in healthcare relative to the costs of adaptation through changes in exposure that may arise from under investment in other sectors. Examples here are the availability and storage of water or the design of regulations on housing design. The second (public- private) distinction relates to the need for government to act in circumstances where information failures lead to sub optimal private behaviors. As noted, the key public function would be to maintain and improve disease surveillance activities, but other examples include the improvement of basic hygiene and lifestyle advice.

Not all of these are immediately relevant to priorities in Montserrat. However, the data gaps identified in this report are significant in that they suggest an initial low cost adaptation in terms of monitoring and data improvement to facilitate integrated health assessment of this nature. It is important to have the best understanding of the nature of the adaptation need, and the analysis presented here suggests scope for improvement. This study suggests that national and regional monitoring initiatives fall within the 'no regrets' category of action that would be worth undertaking even in the absence of climate effects (and that will likely be critical to responding to any future climate effects). A regional focus may be most efficient in detecting disease patterns and movements, especially considering the fluidity of population movements between the different Caribbean islands.

At this stage the quantification of adaptation costs is somewhat speculative, and it is difficult to say much about the success of improved national and regional monitoring, or the likelihood and impact of household/individual behavioral changes. What is possible for Montserrat to do, however, is to set out the range of treatment costs for the impacts identified in this report. Although strictly speaking these do not represent adaptations, as successful adaptation involves prevention rather than cure, they can be added to the VSL.

C. COST-BENEFIT ANALYSIS

The damage cost scenarios derived in the main sections of this report provide a basis for a cost-benefit calculation comparing the benefit (i.e. the monetary value of projected health damages that can be avoided) to a cost of adapting an appropriate adaptation pathway. It may also be instructive to calculate the extent of treatment (or 'mitigation') costs in the event that no adaptation other than treatment is the policy response. This notional treatment cost estimate can also be compared to the cost of an adaptation pathway.

1. Mitigation costs

This section places the damage cost estimates in the context of data on mitigation actions. Recall that mitigation is simply concerned with the cost of treating health impacts as they occur according to the projections developed in the earlier part of this study. Note that this implies treatment of impacts rather than cure, the latter associated with a more targeted program of adaptations to obviate the health impacts occurring. If treatment of cases takes place in the A2 and B2 scenarios, these are the costs that are likely to be incurred.

In table 9 relevant health anomaly data have been combined with treatment cost estimates for the most significant impact categories derived in the damage study. Treatment costs have been derived from the international data as summarized by Markandya and Chiabai (2009). A review of estimates included in this study shows considerable divergence in terms of globally reported treatment costs, which it was not possible to reconcile. Accordingly, while there are considerable differences in treatment methods for each of the relevant Internationally Classification of Diseases (ICD) codes, the study adopted an assumed standard cost per case of $300 (2010) rising at 10% per annum to 2050. This value will be an under-estimate for some case while over estimating the cost for others. Unsurprisingly, highest aggregate treatment cost burdens will be associated with gastroenteritis and cardio and respiratory complications.

Table 9 Estimated present value treatment cost for A2 and B2 relative to baseline $US present value 2010-2050

Discount Rate	A2				B2			
	Cardio and respiratory impacts	Malaria	Dengue	Gastro.	Cardio and respiratory impacts	Malaria	Dengue	Gastro
1%	42,167	441	33,601	228,930	19,417	437	9,495	204,471
2%	1,821	351	28,051	192,367	16,677	346	9,591	176,048
4%	13,281	233	20,140	140,893	12,542	226	8,889	132,913

Source: Data compiled by author

2. Adaptation costs

At the time of writing there are no comprehensive 'global' studies of the health impacts or adaptation costs. Nor have any case studies of health adaptation costs have been published in high-income countries. However, many countries, including the UK, have implemented health adaptation measures in the form of heat-wave plans (Department of Health, 2008). The costs of these plans can be estimated. Heat early-warning systems are relatively inexpensive, unless they include active measures that are implemented following an alert. The reported costs for European countries range from €200,000 to €6 million per year (WHO Regional Office for Europe, 2008). Structural interventions are more expensive. France spent more than €150 million in 2004 on providing additional staff and cool rooms in residential homes for the elderly (Michelon, Magne and Simon-Delaville, 2005). Mortality in this group doubled during the 2003 heat wave.

In low- and middle-income countries, a recent review for the World Health Organization found few examples of studies estimating the costs of adaptation (Markandya and Chiabai, 2009). An unpublished study in South Africa quantified adaptation costs as the prevention costs of the additional burden of malaria cases due to climate change to 2025 (van Rensburg and Blignaut, (2002). However, several such case studies are currently being undertaken in African and Asian countries.

A reasonable assumption is that adptation needs for Montserrat are similar to those considered for St Lucia[20]. But these adaptation have been developed independently of this study and consequently without a focus on prioritization with reference to either the cost-effectiveness of individual (or combined) interventions, or the likely economic burdens predicted by this study. Understandably there is a considerable focus on adapting to extreme events, but overall the study does not provide an indicative costing of necessary interventions relative to the baseline of current spending. Notwithstanding this drawback for our purposes, the classification by Vitalis. and Ragunanan[21] suggests categories that overlap in terms of the likely effectiveness of measures (i.e. not all measures are necessary to undertake).

In terms of the endpoints outlined in table 9 this study suggests an adaptation strategy based on monitoring and public information, i.e. relatively low cost "no regrets" interventions. In the absence of more detailed baseline spending and unmet adaptation needs, this study suggests that a comprehensive program could be developed based on a modest increase in per capita healthcare spending ($220 in 2007 (PAHO 2010). This study considers that there is limited value in discriminating between the needs for both (A2 and B2) scenarios. As a baseline, an assumption is that approximately 10% of this per capita value is notionally targeting the main endpoints considered in table 9. This is likely to be an over-estimate, but this will bias our results in the correct direction if the study is subsequently comparing them with the impact costs. The study then assumes this adaptation cost to grow by 3 percent per annum (again as a baseline). The alternative adaptation scenarios increase this cost by 5, 7 and 10 percent. Hence the present value adaptation costs are derived from the difference between the baseline and the counterfactual increases.

If these levels of spending are indeed effective in targeting the endpoints covered in this study, a benefit-cost calculation can be derived from comparing the cumulative PV ranges (or central point) of the range for A2 or B2 from Table 7, with the PV adaptation costs from Table 10. A cursory comparison suggests that adaptation is highly efficient (i.e. benefits greatly outweigh adaptation costs). Comparing adaptation costs to treatment (or mitigation) costs (Tables 9 &10) suggests a more marginal comparison. However, note that the analysis has been highly conservative in our treatment of the latter. In reality, treatment costs are likely to be significantly higher than the standard $300 per episode cost used in this calculation.

Note that in health cost benefit studies there is often a suggestion that costs be discounted at a higher rate than benefits (Brouwer and others 2005), which would make the comparison suggested here even more favorable

Table 10 Present value adaptation costs $US 2010-2050

Discount rate	**5% increase**	**7%**	**10%**
1%	28,552	39,971	68,922
2%	22,457	31,440	54,212
4%	14,707	20,589	35,502

Source: Data compiled by author

[20] Vitalis, E. and M. Ragunanan (2011) Vulnerability & Adaptation Assessment – Health Sector, Sustainable Development and Environment Division, St Lucia

[21] Legislative or regulatory, Public education or communication, Surveillance and monitoring, Ecosystem intervention, Infrastructure development, Technological/engineering
Medical intervention, Research/further information

To provide some context, the adaptation costs can also be compared to the annual revenue projection for Glendon hospital of $1.8 million according to Health Statistics Handbook for Montserrat (1999-2009)[22]. Alternatively they represent around 0.1% of GDP of national Gross Domestic Product approximately $US 60 million[23] (2009).

VII. CONCLUSION

This study applied epidemiological and economic information to generate the first projections for Montserrat of the excess health burden that could, in the absence of adaptation efforts, result from climate change over the next forty years. The study monetary damage costs are then compared to potential adaptation costs, revealing a favorable benefit-cost ratio. Put simply, adaptation spending under our assumptions represents a good investment.

Although reliant on the assumptions discussed throughout this report, and subject to uncertainty resulting from climate model projections, limited health baseline data, and linear dose-response functions, these efforts provide an update-able and revisable framework for the consideration of the non-market health costs resulting from climate change. This study also demonstrates the potential for climate change to add a substantial burden to the future health systems on Montserrat, something that that will only compound their vulnerability to the other anticipated impacts of climate change.

However, given consistent attention to global health research efforts, as well as to the development and *maintenance* of consistent prevention policies, these health impacts are not likely to be insurmountable in the context of Montserrat (Ebi et. al., 2008; (Kovats and others, 2003d). As has already been discussed, for example, various locations in the world have developed forecasting systems for DF and other vector-borne diseases that could be mimicked and implemented either nationally or regionally within the Caribbean. Combining such macro-level policies (for which the costs of development could possibly be shared internationally) with cheap micro-level behavioral changes, such as those advocated by (Tun-Lin and others, 2009), seems to have a reasonable potential for pre-empting the re-establishment of DF and other vector-borne epidemic cycles on these two islands.

Similarly, although temperature has the very real potential to generate significant excess mortality (be it through heat waves, gradual temperature increases, or both), the power of temperature to increase mortality largely depends on the population of Montserrat being a) caught off guard when it comes to the effects of heat exposure, and b) widely plagued by the cardiovascular and respiratory conditions that temperature exacerbates. Here too, then, a mix of macro-level efforts to maintain an education and warning system can be combined with micro-level behavioral changes, such as improving the quality of ones diet, to relieve at least part of the threat that climate change poses to human health. The same principle applies for water and food-borne diseases, and the pairing of sanitation infrastructure with individual hygiene habits.

As Kovats and others (2003d) discusses, however, the ability to adapt through changes like those just mentioned will depend, among other things, on the availability of local resources, the maintenance of local infrastructure, the ability to spread and manage risk, public awareness, and political will. What this means in terms of adapting Caribbean health sectors to climate change is that the ability of each country to successfully implement health-related adaptation will be tightly interwoven with the impact of climate change on all the other national and regional sectors, as well as on local attitudes towards the magnitude of the problem. Ultimately, therefore, the ability to

[22] This publication does not specify whether this is US or EC dollars.
[23] Montserrat Statistics Department, 2010

efficiently and successfully adapt a particular health sector to the threats posed by climate change will depend not on the theoretical potential for health risks to be overcome, but also on the consideration afforded, and resources allocated to, the health sector in comparison to other sectors. Consequently, the availability of health data outside the health sector that is intentionally geared towards being included in decision-making contexts will become increasingly vital, as will the creation and maintenance of health information systems as described in (DCPP, 2007).

This report has made a number of adaptation cost assumptions to set the damage cost estimates in the context of an adaptation cost-benefit analysis (CBA). The CBA suggests that limited spending on monitoring and information provision would be highly efficient. However Montserrat should ideally invest some research capacity in defining its current health spending baseline and in identifying adaptation unmet needs.

Thus, although dependent on a large variety of specific assumptions, and despite revealing a number of areas worthy of further research, the results of this study are important because the framework utilized and the analyses undertaken have generated examples of data that are not only specifically geared for inclusion in a formal decision-making context, but are also intended to contribute to the overall process of adapting Montserrat to the future risks associated with climate change.

ANNEX I - EQUATIONS FROM METHODS TABLE

Step	Equation Number	Equation	Variable Explanations (Cumulative)
10	A.1	$\overline{BIR_x} = \left(\frac{\sum BI_x}{\sum BPop} \right)$	x: Disease (Step 5) BIR: Mean Baseline Incidence Rate BMR: Mean Baseline Mortality Rate BI: Annual Baseline Incidence BM: Annual Baseline Deaths BPop: Annual Baseline Population
	A.2	$\overline{BMR_x} = \left(\frac{\sum BM_x}{\sum BPop} \right)$	
12	A.3	$RIB_{xi} = (\overline{BIR_x}) * (RPop_i)$	*i*: Projection Year 0 (2010) → 40 (2050) RIB: Annual Reference Incidence Burden RMB: Annual Reference Mortality Burden RPop: Reference Population Projection
	A.4	$RMB_{xi} = (\overline{BMR_x}) * (RPop_i)$	
13	A.5	$PIR_{yxi} = (\overline{BIR_x}) * C$	y: Relevant Climate Change Scenario PIR: Projected Incidence Rate PMR: Projected Mortality Rate C: Adjustment Factor For BIR & BMR, $C=1+(DR_{x+*}CA_{yi})$ DR: Dose-Response % Change CA: Climate Anomaly Data
	A.6	$PMR_{yxi} = (\overline{BMR_x}) * C$	
14	A.7	$PIB_{yxi} = (PIR_{yxi}) * (SPop_{yi})$	PIB: Projected Annual Incidence Burden PMB: Projected Annual Mortality Burden SPop: Projected Annual Scenario Population
	A.8	$PMB_{yxi} = (PMR_{yxi}) * (SPop_{yi})$	
15	A.9	$EIB_{yxi} = (\overline{BIR_x}) * (SPop_{yi})$	EIB: Estimated Annual Incidence Burden EMB: Estimated Annual Mortality Burden (i.e. no DR available)
	A.10	$EMB_{yxi} = (\overline{BMR_x}) * (SPop_{yi})$	
16	A.11	$PIA_{yxi} = PIB_{yxi} - RIB_{xi}$	PIA: Projected Annual Incidence Anomaly EIA: Estimated Annual Incidence Anomaly PMA: Projected Annual Mortality Anomaly EMA: Estimated Annual Mortality Anomaly
	A.12	$PMA_{yxi} = PMB_{yxi} - RMB_{xi}$	
	A.13	$EIA_{yxi} = EIB_{yxi} - RIB_{xi}$	
	A.14	$EMA_{yxi} = EMB_{yxi} - RMB_{xi}$	
25	A.15	$PVSL_{(M)yxi} = VSL_i * PMA_{yxi}$	$PVSL_{(M)}$: Projected Annual VSL Mortality Anomaly $EVSL_{(M)}$: Estimated Annual VSL Mortality Anomaly
	A.16	$EVSL_{(M)yxi} = VSL_i * EMA_{yxi}$	
28	A.17	$PVSL_{(I)yxi} = VSL_i * LF_x * PIA_{yxi}$	$PVSL_{(I)}$: Projected Annual VSL Incidence Anomaly $EVSL_{(I)}$: Estimated Annual VSL Incidence Anomaly LF: Approximated Life Fraction
	A.18	$EVSL_{(I)yxi} = VSL_i * LF_x * EIA_{yxi}$	
29	A.19	$VSL_{(Total)yxi} = VSL_{(I)yxi} + VSL_{(M)yxi}$	$VSL_{(Total)}$: Total Incidence & Mortality VSL Burden For a Given Disease $VSL_{(I)}$: Will either be $PVSL_{(I)}$ or $EVSL_{(I)}$ Depending on the Disease Data $VSL_{(M)}$: Will either be $PVSL_{(M)}$ or $EVSL_{(M)}$ Depending on the Disease Data $VSL_{(Cumulative\ Total)}$: The sum of each $VSL_{(Total)}$ in a given year PV_{yi}: Discounted Present Value of the $VSL_{(Cumulative\ Total)}$ up to year *i* r: Discount Rate
	A.20	$VSL_{(Cumulative\ Total)yi} = \sum^{All\ x's} VSL_{(Total)yxi}$	
	A.21	$PV_{yi} = \frac{\sum_0^i VSL_{(Cumulative\ Total)yi}}{(1+r)^i}$	

ANNEX II - MALARIA LITERATURE SEARCH PAPERS

Abellana, Rosa, and others 2008. Spatio-Seasonal Modeling of the Incidence Rate of Malaria in Mozambique. *Malaria Journal* 7: 228.

Bhattacharya, Sumana, and others 2006. Climate Change and Malaria in India. *Current Science* 90: 369 - 375.

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

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

Coleman, Marlize, and others 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., and others 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.

Craig, M.H., and others 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.

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.

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

Gething, Peter W., and others 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.

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

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.

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

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

Leonardo, L.R., and others 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.

Lindsay, Steven W., and others 2010. Assessing the Future Threat from Vivax Malaria in the United Kingdom Using Two Markedly Different Modelling Approaches. *Malaria Journal* 9: 70.

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.

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

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

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

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

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

Ruiz, Daniel, and others 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.

Stratton, Leanne, and others 2008. The Persistent Problem of Malaria: Addressing the Fundamental Causes of a Global Killer. *Social Science & Medicine* 67: 854 - 862.

Teklehaimanot, Hailay D., and others 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.

Thomson, Madeleine C., and others 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., and others 2006. Malaria Early Warnings Based on Season Climate Forecasts from Multi-Model Ensembles. *Nature* 439: 576 - 579.

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

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

Wandiga, Shem O., and others 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.

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

ANNEX III - DENGUE FEVER LITERATURE SEARCH PAPERS

Amarakoon, Dharmaratne, and others 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.

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

Barbazan, P., and others 2010. Modelling the Effect of Temperature on Transmission of Dengue. *Medical and Veterinary Entomology* 24: 66 - 73.

Brunkard, Joan M., and others 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.

Degallier, Nicolas, and others 2010. Toward an Early Warning System for Dengue Prevention: Modeling Climate Impact on Dengue Transmission. *Climatic Change* 98: 581 - 592.

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

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

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

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

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.

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

Hu, Wenbiao, and others 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., and others 2007. Short Communication: Impact of Climate Variability on the Incidence of Dengue in Mexico. *Tropical Medicine and International Health* 12: 1327 - 1337.

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

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

Johansson, Michael A., and others 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.

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

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

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

Lu, Liang, and others 2009. Time Series Analysis of Dengue Fever and Weather in Guangzhou. *BMC Public Health* 9: 395.

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

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

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

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

Wu, Pei-Chih, and others 2007. Weather as an Effective Predictor for Occurrence of Dengue Fever in Taiwan. *Acta Tropica* 103: 50 - 57.

Yang, H.M., and others 2009. Assessing the Effects of Temperature on Dengue Transmission. *Epidemiology and Infection* 137: 1179 - 1187.

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

ANNEX IV - GASTROENTERITIS LITERATURE SEARCH PAPERS

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

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

Lloyd, Simon J., and others 2007. Global Diarrhoea Morbidity, Weather and Climate. *Climate Research* 34: 119 - 127.

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

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

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

ANNEX V - SCHISTOSOMIASIS LITERATURE SEARCH PAPERS

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

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

Yang, Guo-Jing, and others 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.

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.

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

ANNEX VI - LEPTOSPIROSIS LITERATURE SEARCH PAPERS

=Codeço, Claudia Torres, and others 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.

=Slack, A.T., and others 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. Herrmann, and others 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.

=Tassinari, Wagner S., and others 2008. Detection and Modelling of Cast Clusters for Urban Leptospirosis. *Tropical Medicine and International Health* 13: 503 - 512.

ANNEX VII - CIGUATERA POISONING LITERATURE SEARCH PAPERS

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

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

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

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

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

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

ANNEX VIII - MENINGOCOCCAL MENINGITIS LITERATURE SEARCH PAPERS

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

Harrison, Lee H., and others 2009. Global Epidemiology of Meningococcal Disease. *Vaccine* 27S: B51 - B63.

Molesworth, Anna M., and others 2003. Environmental Risk and Meningitis Epidemics in Africa. *Emerging Infectious Diseases* 9: 1287 - 1293.

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.

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

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

Sultan, Benjamin, and others 2005. Climate Drives the Meningitis Epidemics Onset in West Africa. *PLoS Medicine* 2: 0043 - 0049.

Sultan, B., and others 2005. Meningitis and Climate in West Africa (Summary). *PLoS Medicine* 2: 0003.

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

Yaka, Pascal, and others 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.

ANNEX IX - TEMPERATURE, CLIMATE, RESPIRATORY & CARDIOVASCULAR DISEASE LITERATURE SEARCH PAPERS

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

Ayres, J.G., and others 2009. Climate Change and Respiratory Disease: European Respiratory Society Position Statement. *European Respiratory Journal* 34: 295 - 302.

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

Bell, Michelle L., and others 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.

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

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

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

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

[9] Díaz, J., and others 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.

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

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.

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

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

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

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

Gosling, Simon N., and others 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.

Hales, Simon, and others 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.

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

Hajat, Shakoor, and others 2006. Impact of High Temperatures on Mortality: Is There an Added Heath Wave Effect? *Epidemiology* 17: 632 - 638.

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

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.

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

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

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

Laaidi, Mohamed, and others 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.

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

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

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.

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

Medina, Daniel C., and others 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.

Patz, Jonathan A., and others 2000. The Effects of Changing Weather on Public Health. *Annual Review of Public Health* 21: 271 - 307.

Patz, Jonathan A., and others 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.

Rey, Grégoire, and others 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.

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

Sheridan, Scott C., and others 2009. Trends in Heat-Related Mortality in the United States, 1975 - 2004. *Natural Hazards* 50: 145 - 160.

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

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

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., R. Stennett and A. Chen. 2004. *Climate Variability and Disease Patterns in Two South Eastern Caribbean Countries*, Port of Spain.

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.

Angeles, M.E., J.E. Gonzalez, D.J.E. III and J.L. Hernández. 2007. Predictions of Future Climate Change in the Caribbean Region Using Global General Circulation Models. *International Journal of Climatology* 27: 555 - 569.

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*

Barton, D.N. 2002. The Transferability of Benefit Transfer: Contingent Valuation of Water Quality Improvements in Costa Rica. *Ecological Economics* 42: 147 - 164.

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.

Bellavance, F., G. Dionne and M. Lebeau. 2009. The Value of a Statistical Life: A Meta-Analysis with a Mixed Effects Regression Model. *Journal of Health Economics* 28: 444 - 464.

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.

Blanco, J.T. and D. Hernández. 2009. *The Potential Costs of Climate Change in Tropical Vector-Borne Diseases – a Case Study of Malaria and Dengue in Colombia*, The World Bank: Latin America and the Caribbean Region,

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

Brouwer, R. and I.J. Bateman. 2005. Benefits Transfer of Willingness to Pay Estimates and Functions for Health-Risk Reductions: A Cross-Country Study. *Journal of Health Economics* 24: 591 - 611.

Brouwer, W.B.F., L.W. Niessen, M.J. Postma and F.F.H. Rutten. 2005. Need for Differential Discounting of Costs and Health Effects in Cost Effectiveness Analyses. *British Medical Journal* 331: 446-448.

Brown, M.E. and C.C. Funk. 2008. Food Security under Climate Change. *Science* 319: 580 - 581.

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.

Bueno, R., C. Herzfeld, E.A. Stanton and F. Ackerman. 2008. *The Caribbean and Climate Change: The Costs of Inaction*, Stockholm Environment Institute - US Center Global Development and Environment Institute, Tufts University, Boston.

Burke, M. and D. Lobell. 2010. 'Climate Effects on Food Security: An Overview.' In *Climate Change and Food Security: Adapting Agriculture to a Warmer World* edited by D. Lobell and M. Burke, 13 - 30. Dordrecht: Springer.

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.

CAREC. 2002-2009. CAREC Surveillance Report Caribbean Epidemiology Centre (http://www.carec.org). Available At: http://new.paho.org/carec/index.php?option=com_content&task=view&id=138&Itemid=337. Accessed On: 2 July 2010.

CAREC. 2005. *Leading Causes of Death and Mortality Rates (Counts and Rates) in Caribbean Epidemiology Centre Member Countries (Cmcs): 1985 1990 1995 2000*, Pan American Health Organization/World Health Organization (PAHO/WHO),

CAREC. 2008. Morbidity Review of Communicable Diseases in CAREC Member Countries 1980-2005 Caribbean Epidemiology Centre. Available At: http://new.paho.org/carec/?option=com_content&task=view&id=137&Itemid=304. Accessed On: 2 July 2010.

Caulfield, L.E., S.A. Richard and R.E. Black. 2004. Undernutrition as an Underlying Cause of Malaria Morbidity and Mortality in Children Less Than Five Years Old. *American Journal of Tropical Medicine and Hygiene* 71: 55 - 63.

CDB. 2008. *Position Paper: Responding to Climate Change in the Caribbean Development Bank and Its Borrowing Member Countries*, Caribbean Development Bank,

CDC. 2010. Dengue Epidemiology. U.S. Center for Disease Control,. Available At: http://www.cdc.gov/dengue/epidemiology/index.html. Accessed On: 26 August 2010.

Centella, A. 2009. *Regional Climate Modeling in the Caribbean*, Instituto de Meteorología de Cuba, Havana.

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.

CIA. 2003. United States (2002 GDP - Per Capita (PPP)). United States Central Intelligence Agency,. Available At: http://www.umsl.edu/services/govdocs/wofact2003/geos/us.html. Accessed On: 22 August 2010.

CIA World Factbook. 2010a. Montserrat. United States Central Intelligence Agency. Available At: https://www.cia.gov/library/publications/the-world-factbook/geos/mh.html. Accessed On: August 21 2010.

CIA World Factbook. 2010b. Saint Lucia United States Central Intelligence Agency. Available At: https://www.cia.gov/library/publications/the-world-factbook/geos/st.html. Accessed On: August 21 2010.

CIA World Factbook. 2010c. Saint Lucia (2009 GDP - Per Capita (PPP)), United States (2009 GDP - Per Capita (PPP)), & Montserrat (2002 GDP - Per Capita (PPP)). United States Central Intelligence Agency. Available At: https://www.cia.gov/library/publications/the-world-factbook/fields/2004.html. Accessed On: 22 August 2010.

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.

Contreras-Lisperguer, R. and K. de Cuba. 2008. *The Potential Impact of Climate Change on the Energy Sector in the Caribbean Region: An Effort to Understand How Climate Change May Impact the Productivity of Existing and Future Energy Production Systems in the Caribbean*, Organization of American States,

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.

Czajkowski, M. and M. Ščasný. (2010). Study on Benefit Transfer in an International Setting. How to Improve Welfare Estimates in the Case of the Countries' Income Heterogeneity? . *Ecological Economics* In press

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.

DCPP. 2006. *Noncommunicable Diseases: Noncommunicable Diseases Now Account for a Majority of Deaths in Low- and Middle-Income Countries.* Disease Control Priorities Project. http://www.dcp2.org/file/58/DCPP-NCD.pdf.

DCPP. 2007. Want Value for Money? Invest in Information: Comprehensive Health Information Systems Are Essential and Affordable. Disease Control Priorities Project,. Available At: http://www.dcp2.org/file/82/DCPP-InfoforDecision.pdf. Accessed On: 5 August 2010.

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.

Department of Health. 2008. *Heatwave Plan for England - Protecting Health and Reducing Harm from Extreme Heat and Heatwaes.*, Department of Health, London.

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.

Dupont, A. and M. Thirlwell. 2009. A New Era of Food Insecurity? *Survival* 51: 711 - 98.

Ebanks, G.E. 1989. Stable Population Size: Montserrat. *Canadian Studies in Population* 16: 217 - 236.

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.

Ebi, K.L. 2008b. Healthy People 2100: Modeling Population Health Impacts of Climate Change. *Climatic Change* 88: 5 - 19.

ECLAC. 2007. *Addressing the Impacts of Climate Change: Focus on the Caribbean*, LC/CAR/L.149. Economic Commission for Latin America and the Caribbean,

ECLAC. 2010. *Review of the Economics of Climate Change (Recc) in the Caribbean Project: Phase I Climate Change Profiles in Select Caribbean Countries*, LC/CAR/L.250. Economic Commission for Latin America and the Caribbean: Subregional Headquarters for the Caribbean,

EM-DAT. 2010. *Em-Dat: Reported Disasters in St. Lucia and Montserrat from 1900 - 2010*. EM-DAT: The OFDA/CRED International Disaster Database at the Université Catholique de Louvain. http://www.emdat.be.

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

Ericksen, P., J.S.I. Ingram and D.M. Liverman. 2009. Food Security and Global Environmental Change: Emerging Challenges. *Environmental Science & Policy* 12: 373 - 377.

Every Day Health. 2009a. Gastroenteritis in Adults. Harvard Health Publications. Available At: http://www.everydayhealth.com/health-information/gastroenteritis-in-adults-expected-duration.aspx. Accessed On: 26 August 2010.

Every Day Health. 2009b. Gastroenteritis in Children. Harvard Health Publications. Available At: http://www.everydayhealth.com/health-information/gastroenteritis-in-children-expected-duration.aspx. Accessed On: 26 August 2010.

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

Fenton, A. 2010a. *Email: Montserrat Ciguatera Poisoning Incidence.*

Fenton, A. 2010b. *Email: Population Data Request.*

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.

Freeman, V., H. Fraser, T. Forrester, R. Wilks, J. Cruickshank, C. Rotimi and R. Cooper. 1996. A Comparative Study of Hypertension Prevalence, Awareness, Treatment and Control Rates in St Lucia, Jamaica and Barbados. *Journal of Hypertension* 14: 495 - 501.

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.

Gelea, S., A. Nandi and D. Vlahov. 2005. The Epidemiology of Post-Traumatic Stress Disorder after Disasters. *Epidemiologic Reviews* 27: 78 - 91.

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.

Godfray, H.C.J., J.R. Beddington, I.R. Crute, L. Haddad, D. Lawrence, J.F. Muir, J. Pretty, S. Robinson, S.M. Thomas and C. Toulmin. 2010. Food Security: The Challenge of Feeding 9 Billion People. *Science* 327: 812 - 818.

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.

Haites, E., D. Pantin, M. Attzs, J. Bruce and J. MacKinnon. 2002. *Assessment of the Economic Impact of Climate Change on Caricom Countries*, Toronto.

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.

Hallegatte, S. 2009. Strategies to Adapt to an Uncertain Climate Change. *Global Environmental Change* 19: 240-247.

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.

Howden, S.M., J.-F. Soussana, F.N. Tubiello, N. Chhetri, M. Dunlop and H. Meinke. 2007. Adapting Agriculture to Climate Change. *Proceedings of the National Academy of Sciences* 104: 19691 - 19696.

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.

Hutchinson, S. 2010. *Review of the Economics of Climate Change in the Caribbean Phase II - the Water Sector*, LD/CAR/L.260. Economic Commission for Latin America and the Caribbean: Subregional Headquarters for the Caribbean,

*Intel*iHealth. 2008. Flu (Influenza). Aetna InteliHealth Inc.,. Available At: http://www.intelihealth.com/IH/ihtIH/c/9339/10198.html. Accessed On: 26 August 2010.

IPCC. 2001a. 4.2.1 Storylines. Available At: http://www.ipcc.ch/ipccreports/sres/emission/index.php?idp=91. Accessed On: 30 July 2010.

IPCC. 2001b. 4.3.2 A2 Storyline and Scenario Family. Available At: http://www.ipcc.ch/ipccreports/sres/emission/index.php?idp=94. Accessed On: 30 July 2010.

IPCC. 2001c. 4.3.4 B2 Storyline and Scenario Family. Available At: http://www.ipcc.ch/ipccreports/sres/emission/index.php?idp=95 Accessed On: 30 July 2010.

IPCC. 2001d. 4.4.2 Translation of Storylines into Scenario Drivers. Available At: http://www.ipcc.ch/ipccreports/sres/emission/index.php?idp=97 Accessed On: 30 July 2010.

IPCC. 2001e. 4.4.3 Population Prospects Footnote 20. Available At: http://www.ipcc.ch/ipccreports/sres/emission/index.php?idp=99 Accessed On: 30 July 2010.

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.

Johnston, R.J. and R.S. Rosenberger. 2010. Methods, Trends, and Controversies in Contemporary Benefit Transfer. *Journal of Economic Surveys* 24: 479 - 510.

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.

Kendall, P. and M. Petracco. 2009. The Current State and Future of Caribbean Agriculture. *Journal of Sustainable Agriculture* 33: 780 - 797.

Kipping, R., H. Eastcott and J. Sarangi. 2006. Tropical Fish Poisoning in Temperature Climates: Food Poisoning from Ciguatera Toxin Presenting in Avonmouth. *Journal of Public Health* 28: 343 - 346.

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. 'Disasters: Floods and Windstorms.' In *Methods of Assessing Human Vulnerability and Public Health Adaptation to Climate Change: Health and Global Environmental Change Series No. 1* edited by 62 - 67. Copenhagen: World Health Organization.

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

Kovats, S., K.L. Ebi and B. Menne. 2003e. '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. 2003f. '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.

Laaidi, M., K. Laaidi and J.-P. Besancenot. 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.

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.

Leary, N.A. 1999. A Frameowrk for Benefit-Cost Analysis of Adaptation to Climate Change and Climate Variability. *Mitigation and Adaptation Strategies for Global Change* 4: 307 - 318.

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.

Lewis, L., B. Roach and E. Haynes. 2000. A Low-Cost Approach to Public Health Education Using Multimedia Packages. *Journal of Telemedicine and Telecare* 6: S2:41 - S2:43.

Lewsey, C., G. Cid and E. Kruse. 2004. Assessing Climate Change Impacts on Coastal Infrastructure in the Eastern Caribbean. *Marine Policy* 28: 393 - 409.

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.

Lindhjem, H. and S. Navrud. 2008. How Reliable Are Meta-Analyses for International Benefit Transfers? *Ecological Economics* 66: 425 - 435.

Lindhjem, H., S. Navrud and N.A. Braathen. 2010. *Valuing Lives Saved from Environmental, Transport and Health Policies: A Meta-Analysis of Stated Preference Studies*, ENV/EPOC/WPNEP(2008)10/FINAL. Paris.

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.

Lugo, A.E. 2000. Effects and Outcomes of Caribbean Hurricanes in a Climate Change Scenario. *The Science of the Total Environment* 262: 243 - 251.

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.

Markandya, A. and A. Chiabai. 2009. Valuing Climate Change Impacts on Human Health: Empirical Evidence from the Literature. *International Journal of Environmental Research and Public Health* 6: 759 - 786.

Marquez, P. 2006. Cost Benefit Analysis, Value of a Statistical Life and Culture: Challenges for Risk Regulation. University of Munich. Available At: http://mpra.ub.uni-muenchen.de/2632/ Accessed On: 15 July 2010.

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.

McMichael, A.J., P. Wilkinson, R.S. Kovats, S. Pattenden, S. Hajat, B. Armstrong, N. Vajanapoom, E.M. Niciu, H. Mahomed, C. Kingkeow, M. Kosnik, M.S. O'Neill, I. Romieu, M. Ramirez-Aguilar, M.L. Barreto, N. Gouveia and B. Nikiforov. 2008. International Study of Temperature, Heat and Urban Mortality: The 'Isothurm' Project. *Environmental Epidemiology* 37: 1121 - 1131.

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.

Michelon, T., P. Magne and F. Simon-Delaville. 2005. 'Lessons of the 2003 Heat Wave in France and Action Taken to Limit the Effects of Future Heat Waves.' In *Extreme Weather Events and Public Health Responses* edited by W. Kirch, B. Menne and R. Bertollini, 131-140. Verlag: Springer.

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.

Morton, J.F. 2007. The Impact of Climate Change on Smallholder and Subsistence Agriculture. *Proceedings of the National Academy of Sciences* 104: 19680 - 19685.

Mrozek, J.R. and L.O. taylor. 2002. What Determines the Value of Life? A Meta-Analysis. *Journal of Policy Analysis and Management* 21: 253 - 270.

Murray, C.J.L., M. Ezzati, A.D. Lopez, A. Rodgers and S.V. Hoorn. 2003. Comparative Quantification of Health Risks: Conceptual Framework and Methodological Issues. *Population Health Metrics* 1: 1.

NASA. 2002. NASA New Start Index Inflation Calculator. National Aeronautics and Space Administration,. Available At: http://cost.jsc.nasa.gov/inflation/nasa/inflateNASA.html. Accessed On: 25 August 2010.

NHS. 2005. Schistosomiasis (Bilharzia). U.K. National Health Service. Available At: http://www.cks.nhs.uk/patient_information_leaflet/schistosomiasis_bilharzia. Accessed On: 26 August 2010.

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.

PAHO. 1998. 'Montserrat.' In *Health in the Americas, 1998 Edition, Volume II* (II), edited by 372 - 374 Pan American Health Organization.

PAHO. 2007. 'Montserrat.' In *Health in the Americas, 2007, Volume II-Countries* (II), edited by 486 - 493. Pan American Health Organization.

PAHO. 2009. *Health Information and Analysis Project: Health Situation in the Americas: Basic Indicators 2009*. Pan American Health Organization.

PAHO. 2010. *Health Systems Profile: Montserrat 2008*, Pan American Health Organization/World Health Organization, Washington, DC.

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

Parham, P.E. and E. Michael. 2010. Modeling the Effects of Weather and Climate Change on Malaria Transmission. *Environmental Health Perspectives* 118: 620 - 626.

Parry, M., N. Arnell, P. Berry, D. Dodman, S. Fankhauser, C. Hope, S. Kovats, R. Nicholls, S. Satterthwaite, R. Tiffin and T. Wheeler. 2009. Assessing the Costs of Adaptation to Climate Change: A Review of Unfccc and Other Estimates. IIED. Available At: http://pubs.iied.org/pdfs/11501IIED.pdf. Accessed On: 15 May 2011.

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.

Peterson, T.C., M.A. Taylor, R. Demeritte, D.L. Duncombe, S. Burton, F. Thompson, A. Porter, M. Mercedes, E. Villegas, R.S. Fils, A.K. Tank, A. Martis, R. Warner, A. Joyette, W. Mills, L. Alexander and B. Gleason. 2002. Recent Changes in Climate Extremes in the Caribbean Region. *Journal of Geophysical Research* 107: 16-1 - 16-9.

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

Rawlins, S.C., A. Hinds and J.M. Rawlins. 2008. Malaria and Its Vectors in the Caribbean: The Continuing Challenge of the Disease Forty-Five Years after Eradication from the Islands. *West Indian Medical Journal* 57: 462 - 469.

Ready, R. and S. Navrud. 2006. International Benefit Transfer: Methods and Validity Tests. *Ecological Economics* 60: 429 - 434.

Reis, R.B., G.S. Ribeiro, R.D.M. Felzemburgh, F.S. Santana, S. Mohr, A.X.T.O. Melendez, A. Queiroz, A.C. Santos, R.R. Ravines, W.S. Tassinari, M.S. Carvalho, M.G. Reis and A.I. Ko. 2008. Impact of Environment and Social Gradient on *Leptospira* Infection in Urban Slums. *PLoS Neglected Tropical Diseases* 2: e228.

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.

Schmidhuber, J. and F.N. Tubiello. 2007. Global Food Security under Climate Change. *Proceedings of the National Academy of Sciences* 104: 19703 - 19708.

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.

Simpson, M.C., D. Scott, M. New, R. Sim, D. Smith, M. Harrison, C.M. Eakin, R. Warrick, A.E. Strong, P. Kouwenhoven, S. Harrison, M. Wilson, G.C. Nelson, S. Donner, R. Kay, D.K. Geldhill, G. Liu, J.A. Morgan, J.A. Kleypas, P.J. Mumby, T.R.L. Christensen, M.L. Baskett, W.J. Skirving, C. Elrick, M. Taylor, J. Bell, M. Rutty, J.B. Burnett, M. Overmas, R. Robertson and H. Stager. 2009. *An Overview of Modeling Climate Change Impacts in the Caribbean Region with Contribution from the Pacific Islands*, Barbados.

Singh, B. 1997. Climate-Related Global Changes in the Southern Caribbean: Trinidad and Tobabo. *Global and Planetary Change* 15: 93 - 111.

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.

Sookram, S. 2010. *Regional Report on the Impact of Climate Change on the Tourism Sector*, LD/CAR/L.263. Economic Commission for Latin America and the Caribbean: Subregional Headquarters for the Caribbean,

Stellin, G. and A. Candido. 2006. *The Transfer of Benefit Measures: The Applicability of Conditions and the Results*. Center or Interational Food and Agricultural Policy. http://ageconsearch.umn.edu/bitstream/6627/2/cp06st01.pdf.

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.

Sussman, F.G., M.L. Cropper, H. Galbraith, D. Godschalk, J. Loomis, G. Luber, M. McGeehin, J.E. Neumann, W.D. Shaw, A. Vedlitz and S. Zahran. 2008. 'Effects of Global Change on Human Welfare.' 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, (ed.), K.L. Ebi, A.E. Grambsch, F.G. Sussman, T.J. Wilbanks and (Authors), 111 - 168. Washington, DC: U.S. Environmental Protection Agency.

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.

Ting, J.Y.S. and A.F.T. Brown. 2001. Ciguatera Poisoning: A Global Issue with Common Management Problems. *European Journal of Emergency Medicine* 8: 295 - 300.

Toba, N. 2009. *Potential Economic Impacts of Climate Change in the Caribbean Community*, The World Bank: Latin America and the Caribbean Region,,

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.

Tubiello, F.N., J.-F. Soussana and S.M. Howden. 2007. Crop and Pasture Response to Climate Change. *Proceedings of the National Academy of Sciences* 104: 19686 - 19690.

Tun-Lin, W., Lenhart, V.S. Nam, E. Rebollar-Téllez, A.C. Morrison, P. Barbazan, M. Cote, J. Midega, F. Sanchez, P.M.-S. Kroeger, M.B. Nathan, F. Meheus and M. Petzold. 2009. Reducing Costs and Operational Constraints of Dengue Vector Control by Targeting Productive Breeding Places: A Multi-Country Non-Inferiority Cluster Randomized Trial. *Tropical Medicine and International Health* 14: 1143 - 1153.

U.S. Census Bureau. 2010. International Data Base. U.S. Census Bureau,. Available At: http://www.census.gov/ipc/www/idb/country.php. Accessed On: 22 August 2010.

UN. 2008. World Population Prospects: The 2008 Revision. Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat Available At: http://esa.un.org/unpp/index.asp. Accessed On: 1 August 2010.

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.

van Rensburg, J.J.J. and J.N. Blignaut. Year. The Economic Impact of an Increasing Health Risk Due Ot Global Warming. Forum for Economics and the Environment - First Conference Proceedings. Cape Town. pp. 17-127.

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.

Vergara, W. 2009. *Assessing the Potential Consequences of Climate Destabilization in Latin America (Latin America and Caribbean Region Sustainable Development Working Paper 32)*, The World Bank: Latin America and the Caribbean Region,

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.

Vitalis, E. and M. Ragunanan. 2011. *Vulnerability & Adaptation Assessment - Health Sector* Sustainable Development and Environment Division

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: 2010. 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.

WHO. 2006. Mortality Country Fact Sheet 2006: Bangladesh. WHO. Available At: http://www.who.int/whosis/mort/profiles/mort_searo_bgd_bangladesh.pdf. Accessed On: August 1 2010.

WHO. 2007. International Statistical Classification of Diseases and Related Health Problems, 10th Revision. Available At: http://apps.who.int/classifications/apps/icd/icd10online/. Accessed On: 1 August 2010.

WHO Regional Office for Europe. 2008. *Improving Public Health Responses to Extreme Weather/Heat-Waves - EuroHEAT Technical Summary*, WHO Regional Office for Europe, Copenhagen.

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.