NUI MAYNOOTH

Ollscoil na hÉireann Má Nuad

CLIMATE CHANGE AND HEALTH IN IRELAND: A NATIONAL VULNERABILITY ASSESSMENT

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Dedicated to the memory of my parents,

Andrew and Maureen Cullen

Abstract

In keeping with global trends, the mean annual air temperature in Ireland has increased by 0.5^0C over the last 100 years. This is comparable to the global 0.6 ^{0}C rise. Climate change is expected to impact on health, and will have both direct and indirect impacts. As a result, calls have been made to estimate the impact of climate change on health nationally.

The health impacts of changes in climatic variables on the Irish population were estimated and quantified where possible. Mortality, morbidity and climatic data were obtained from the Central Statistics Office, the Health Protection Surveillance Centre, Met Eireann and the Irish Climate and Analysis Research Group. Temperature outputs were obtained from global climate models modified for Irish conditions, and driven by specified scenarios. An analogue approach was adopted, using curve fitting and modelling techniques to estimate future impacts on health.

Overall reductions are seen in mortality, increasing as time progresses. The reductions are evident in total mortality, specifically in respiratory and cardiovascular mortality, and are greatest in the older population. Changes in the incidence of food-borne disease were estimated, and increases in the future incidence of salmonella, campylobacter and E Coli 0157 were quantified. In areas where increased rainfall and flooding occur, the incidence of waterborne disease may increase, particularly in areas where the water supply is predominantly from private wells, and in areas where water treatment facilities are inadequate. The possibility of increases in other infectious diseases, including malaria is discussed. Increases may also occur in the incidence of allergic diseases, and skin cancer.

Recommendations are made and include the development of an educational campaign to reduce future mortality from heat waves, and measures to reduce the vulnerability of water supplies to the impact of climate change.

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CHAPTER 1 – CLIMATE CHANGE

"Every doctor should know what to expect of the weather"

Hippocrates (a), 400 B.C.

1.1 Introduction

Climate is defined as the 'average weather', and is generally averaged over a time period of thirty years (AR4, 2007). Over longer periods of time, climate has been observed to change, and the processes that cause local or global climatic changes are termed forcings. Forcings can result from either change in the oceans and atmosphere or from changes in the incoming and outgoing solar radiation. They may also be either natural or anthropogenic. Natural forcings may be caused, for example, by volcanic eruptions, which release aerosols into the stratosphere. When these small particles reflect back incoming solar radiation, they result in the earth becoming cooler. Changes in climate are not solely a recent phenomenon, and examination of ice cores and tree rings has shown that climate has periodically changed in the past. Past change in climate were due to such factors as volcanic activity and the removal of carbon dioxide from the atmosphere by plants. These changes typically occurred slowly, a change of 5 ^{0}C occurring over tens of thousands of years. This slow rate of change enabled adaptation of ecosystems to occur. A forcing may also be anthropogenic and such a forcing has resulted in what has been termed the enhanced greenhouse effect. This is a recent (in the last two hundred years), positive forcing, causing the temperature of the earth to rise. It is an accentuation of the greenhouse effect, which is necessary for life to exist on earth.

1.2 The greenhouse effect

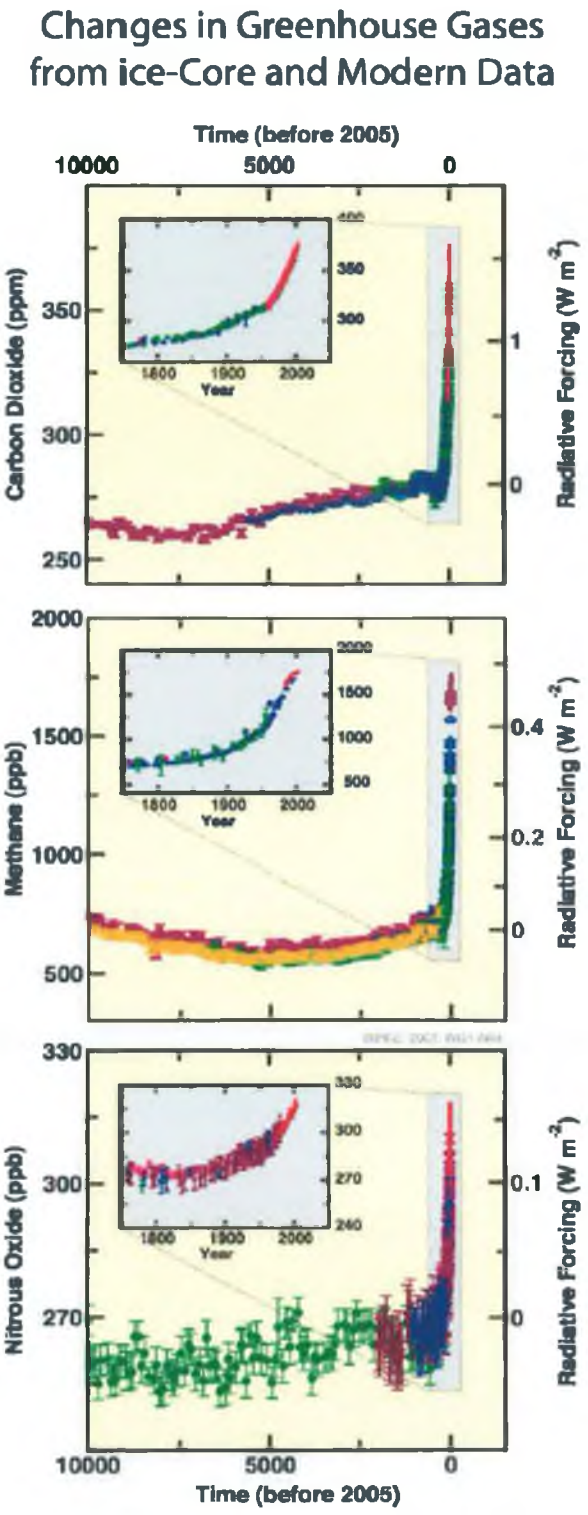
The Irish scientist, John Tyndall, born in 1820 in Leighlinbridge, Co. Carlow, is credited with discovering the greenhouse effect. He described how the effect occurred:

> *"Thus the atmosphere admits of the entrance of the solar heat, but checks its exit; and the result is a tendency to accumulate heat at the surface of the planet"*
>
> (Fleming, 1998)

The greenhouse effect results from the fact that the incoming solar radiation is absorbed by the ground, but is emitted back into the atmosphere at a longer wavelength. This lower energy radiation is absorbed by gases in the atmosphere such as carbon dioxide, nitrous oxides, methane and also water vapour. As a result of these greenhouse gases, the average temperature of the surface of the planet is 15^0C. Without it the average temperature of the earth would be approximately -18^0C, and too cold for life as we know it to exist. The greenhouse effect is therefore one of the life-supporting systems of the planet. However, in recent years, the quantity of these gases in the atmosphere has increased, resulting in the enhanced (and anthropogenic) greenhouse effect.

1.3 Enhanced (anthropogenic) greenhouse effect

The cause of the enhanced greenhouse effect is well established, and results from an increased concentration of greenhouse gases in the atmosphere (Parry and Carter, 1998; IPCC, 2001, AR4, 2007). Since the Industrial Revolution, the chemical composition of the atmosphere has changed, and the concentrations of greenhouse gases, such as carbon dioxide, methane and nitrous oxides in the atmosphere have increased markedly. The changes in these greenhouse asses and their radiative forcings are depicted (Figure 1.1).



(from AR4, 2007)

Figure 1.1 Changes in atmospheric carbon dioxide, methane and nitrous oxides from ice-core data covering the last 10,000 years (large panels) and since 1750 (inset panels). Different colours in ice core data reflect different studies. Atmospheric data are in red.

As a result of the industrial revolution, carbon dioxide concentrations in the atmosphere have increased by over 30% since 1700, largely as a result of increased combustion of fossil fuels (Houghton, 1997), and at present, every year, burning fossil fuels releases carbon dioxide from fossilized plant life, that took 400 years to grow (Dukes, 2003). The carbon cycle however is slow, and the increased carbon dioxide being produced cannot be removed from the atmosphere as quickly as it is entering it. Deforestation is also a significant contributor to the increased concentration of greenhouse gases in the atmosphere, as carbon dioxide is removed from the atmosphere by growing trees. However, from 1990 to 2005, the world lost 3% of its forests (WHO, 2007 (b)). The concentration of methane in the atmosphere has increased by 70% since the Industrial Revolution (AR4, 2007). Methane is a more potent greenhouse gas than carbon dioxide, and is produced in the intestinal tract of ruminant animals, in rice fields, and in landfills sites where anaerobic processes occurs. The concentrations of nitrogen oxides in the atmosphere have increased by almost 20% since the Industrial Revolution (Oceana, 2008). Nitrous oxides are also released from the burning of fossil fuel, and in addition from agricultural practices such as winter ploughing. This enhanced anthropogenic greenhouse effect is commonly referred to as climate change.

1.4 Recent climate trends globally

Climate change is defined as 'any change in climate over time', whether due to natural variation or human activity (AR4, 2007). Interestingly, this definition differs from the definition in the United Nations Framework Convention on Climate Change (UNFCCC), which includes in its definition, a stipulation that the change in climate directly results from human activity. There is a natural variability in the background climate, and detection of climate change is therefore difficult. Nevertheless, the evidence points strongly to the fact that the climate is changing. The fourth assessment report of the International Panel on Climate Change (IPCC) found that warming of the climate system is 'unequivocal' (AR4, 2007).

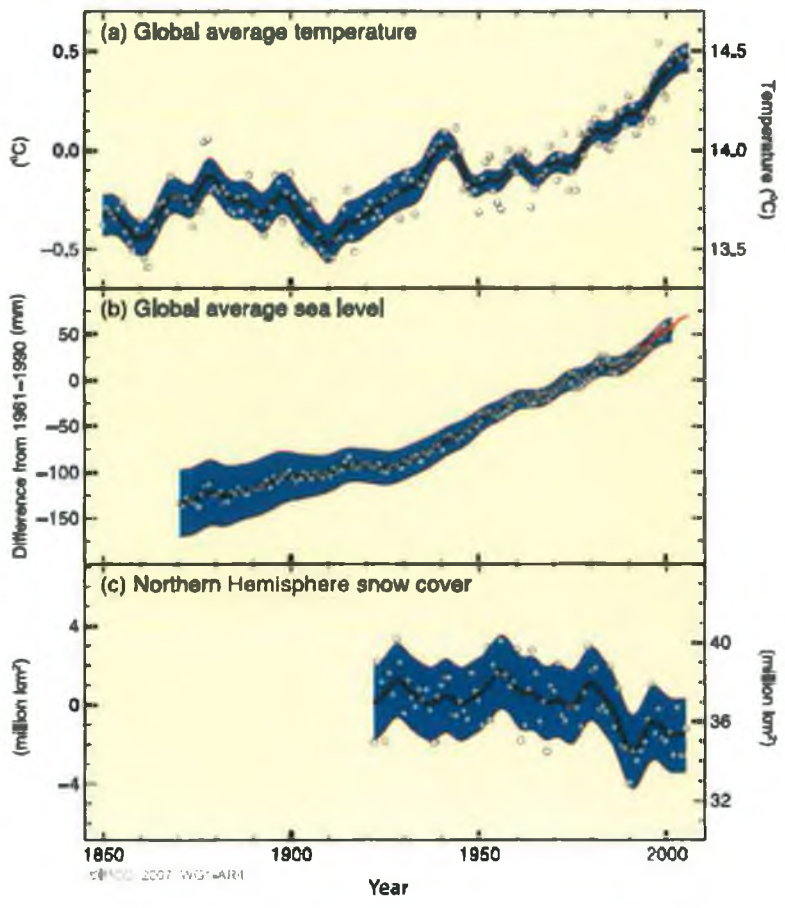
1.4.1 Temperature

Globally, eleven of the last twelve years (1995-2006) rank among the twelve warmest since instrumental records of global surface temperature began in 1850. In addition,

the recent fourth assessment report from the IPCC (AR4, 2007) found that the increase in mean temperature of 0.6 °C reported in the third assessment (noted in the 100 years from 1901 to 2000 (IPCC, 2001), had risen to approximately 0.74 °C in the 100 years from 1906 to 2005. This recent fourth report also found that in the last fifty years, a linear trend showing a mean increase of 0.13^{0}C degrees per decade was almost twice the increase for the previous 100 years (AR4, 2007).

1.4.2 Water resources

Higher temperatures will result in increased oceanic evaporation, and the resulting increased rain will lead to increased runoff and flooding at higher latitudes (AR4, 2007). There appears to be a gradient developing in terms of water resources; while an increase in rainfall of between 10 and 40% has been noted in Northern Europe, in the last century, there appears to be a trend towards less rain in Southern Europe (AR4, 2007), rainfall having fallen by up to 20% in the Mediterranean basin (Eisenreich, 2005). The oceans have been absorbing over 80% of the heat added to the climate system, causing the water to expand and the sea-level to rise. Global average sea levels have risen at a rate of 1.8 mm per year from 1961 to 2003, and at a faster rate of 3.1 mm per year from 1993 to 2003. It is unclear whether the later rate reflects decadal variation or an increasing rate (AR4, 2007). The upward trend in combined global temperatures, sea-levels and reduction in snow cover from 1850 to 2005 is evident (Figure 1.2).



(from AR4, 2007)

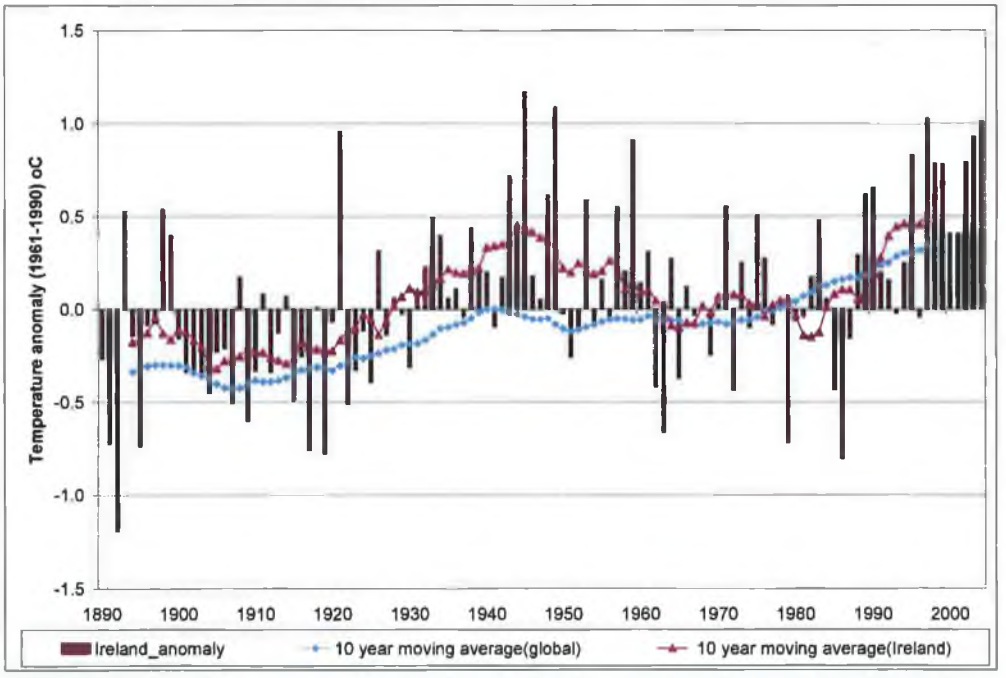
Figure 1.2 Changes in (a) global temperature, (b)global sea-level from tidal gauge data (blue) and satellite data (red), and (c) snow cover in the Northern hemisphere relative to 1961-1990. Smoothed curves reflect decadal average values while circles reflect yearly values. The shaded areas reflect uncertainty values.

1.5 Recent climate trends in Ireland

1.5.1 Temperature

From an analysis of long term Irish meteorological data, Irish temperature trends have been found to have largely followed global trends, but with a lag of several years (Figure 1.3), (Sweeney *et al,* 2002). Similar to global trends, there was a warming trend in Ireland from the beginning of the century, which lasted until the 1940s. This was followed by a cooling period, which persisted up to the start of the 1980s. Following this cooling period, a rapid warming period ensued and this continues. Using a moving average to smooth the data, a mean increase in temperature of 0.51 ^{0}C over the last 100

years in Ireland has been observed (Figure 1.3) (Sweeney *et al,* 2002). Similar to the recent global trend, from 1890 to 2004, an increase of 0.7^0C in mean temperature is also seen(Mc Elwain and Sweeney, 2007). Because the earlier warming trend in the 1930s and 1940s was stronger than at present, 1945 was the warmest year in Ireland in the last century. However, most of this recent warming occurred in the 1990s, and this decade was the warmest decade in Irish instrumental records, with five of the ten warmest years occurring since 1990 (Sweeney *et al*, 2002).



(Mc Elwain and Sweeney, 2007))

Figure 1.3 Global and national temperature anomaly to 2004 (10-year moving average)

The later temperature increase has occurred in a much shorter time scale and to a much greater magnitude, and the rapid warming in the last two decades is significant at the 99% level; in addition, the warming trend which began in Ireland in the early 1980s, continues to be observed at a rate of 0.25^0C per decade (Sweeney *et al*, 2002).

Although Ireland experiences considerable climate variability and the trend is not linear, the highest decadal change has occurred since 1980, with an increase in mean temperatures of 0.42^0C per decade (Mc Elwain and Sweeney, 2007).

1.5.2 Precipitation

Changes in precipitation trends in Ireland are also apparent, with significant increases in winter rainfall having occurred in the north in recent years. A pronounced precipitation gradient appears to be developing over Ireland with Malin Head, in the north showing a 40% increase in the 10 year moving average over the 20th century, with rainfall increasing from 800 mm in 1890 to 1,110mm in 1990 (Mc Elwain and Sweeney, 2007). In addition, 4 of the 5 wettest years in the area occurred in the 1990s. In contrast, in a southerly direction, the summers in the southeast have become drier (Sweeney *et al,* 2002), and analysis of records dating from 1890 in Birr and from 1957 in Rosslare show a decreased rainfall pattern (Mc Elwain and Sweeney, 2007).

1.6 Changes in future climatic parameters

Changes in climatic parameters will depend on the emissions of greenhouse gases. However, even if greenhouse gas emissions were kept at 2000 levels, increases of 0.1^{0}C per decade in mean temperature would still occur, for the next two decades, primarily because of the slow response of the oceans across the ocean-atmospheric interface (AR4, 2007). Future emissions rates have been estimated for a range of greenhouse gas emissions both globally (AR4, 2007) and for Ireland (Sweeney *et al,* 2003).

1.6.1 Changes in future global climatic parameters

Global increases in mean temperatures of 0.2 ^{0}C per decade for the next two decades have been estimated, and average increases in mean temperatures to the end of the century, from different climate models, have been quantified, with mean increases of between 1.8°C to 4.0°C (range 1.1^{0}C to 6.4^{0}C) relative to 1980-1999 levels, being estimated (AR4, 2007). The recent IPCC report found that "it is very likely that hot extremes, heat-waves, and heavy precipitation events will continue to become more frequent" and sea levels could be expected to rise by between 0.18 and 0.59 metres by the end of the century (AR4, 2007). However, an overall decrease in water availability has been predicted in some southern European countries (AR4, 2007).

CHAPTER 2- CLIMATE CHANGE AND HEALTH

"Whoever wishes to investigate medicine properly should proceed thus: in the first place to consider the seasons of the year and what affect each of them produces (for they are not all alike, but differ much from themselves in regard to their changes). Then the winds, the hot and the cold, especially such as are common to all countries, and then such as are peculiar to each locality".

Hippocrates (b)

2.1 Introduction

Although the link between climate and health has been known since the time of ancient Greece, this knowledge was essentially confined to the impact of weather variability on health. It was over two thousand years later, in 1986, that the link between climate and health was recognised internationally. In that year, the World Meteorological Organization, together with the World Health Organization and the United Nations Environment Programme, convened a meeting in Leningrad, to discuss the relationship between natural variations in climate and health (WHO, 1996). Following this, in 1988, the Intergovernmental Panel on Climate Change (IPCC) was established within the UN framework. The aim of this body was to advise national governments on the causes and impacts of climate change. Health was recognized as one of the impacts of climate change by this panel. However, it is interesting to note that from the time of Hippocrates, the influence of 'hot and cold winds', as a specific environmental factor affecting health had been noted.

2.2 Development of links between climate change and health

To date, the IPCC has published four major reports, the First, Second, Third and Fourth Assessment Reports, in 1990, 1995, 2001 and 2007 respectively (IPCC,1990; IPCC 1995; 1PCC, 2001; AR4 2007). The prominence of health, as an impact of climate change, has increased in each successive report, reflecting the growth in knowledge of the relationship between health and climate change. Initially, in 1990, when the First Assessment Report of the IPCC was issued, health was referred to, although briefly.

The report stressed that climate change could cause damage to ecosystems, and result in disturbances of human settlements, enforced migration and economic disruption.

In 1995, more data was available, and the impact of climate change on health was reported in more detail in the Second Assessment Report. The data used came primarily from two sources, studies relating health to climate variability e.g. El Nino (a periodic disruption of the ocean-atmosphere system in the Tropical Pacific), and from extreme climatic events, such as natural disasters and heat waves. The main findings of this second report were that there would be two major health impacts from climate change, namely, an increase in short term mortality from heat waves, which would be offset to an uncertain degree by a reduction in cold-related deaths and an increase in infectious diseases, including vector-borne diseases. The overall assessment was that the likely health impacts would be predominantly adverse, and consequently the need for adaptation was stressed. This report emphasized the multi-factorial nature of health, and acknowledged that social, economic and health care circumstances, among other issues, would mediate the impact of climate change on human health.

Following this Second Assessment Report from the IPCC, an assessment funded by the Environment Programme of the Research Directorate General of the Commission of the European Communities, and produced in association with the University of East Anglia, was issued. This report, an 'Assessment of Potential Effects and Adaptations for Climate Change in Europe' (ACACIA, 2000), provided the basis from which the chapter on the health effects in Europe in the IPCC Third Assessment Report was derived. The report contained a comprehensive assessment of the impact of climate change on health, and also on water resources, ecosystems, coastal zones and marine ecosystems, human settlements, energy and industry. The main conclusions of the Third Assessment Report were again, that although warmer winters and fewer cold spells would decrease cold-related mortality in many temperate countries, overall, negative health impacts would outweigh positive health impacts. Negative impacts would result from changes in the frequencies and intensities of extremes of heat, cold, floods and droughts, and changes in the range of infectious diseases. The report found that the pattern of acclimatization to future climate regimes was difficult to estimate.

The Fourth Assessment Report noted that aside from some study on the impacts of heat waves, little extra research on the health impacts of climate change had been undertaken. This report stressed the vulnerability of people, in particular people living in poorer countries and with a low adaptive capacity to cope. The importance of education, health care and public health infrastructures in mitigating these negative impacts was emphasized (AR4, 2007).

2.3 Estimates of the impacts of climate change on global health

It is now clear that the climate is changing rapidly, and in recent years, much work has been carried out on the impact that this may have on health. Two global estimates of the impact of climate change on health have been identified. Adverse health impacts of climate change were estimated to have been responsible in 2000 for approximately 2.4% of worldwide diarrhoea, 6% of malaria in some middle-income countries, and 7% of cases dengue fever in some industrialized countries (World Health Report, 2002). However, poorer countries suffer disproportionately, and in a comparative study, the global health impact of climate change in Europe as a component of five specific environmental risks was low (Ezzati *et al,* 2002).

Risk	Africa	S.E.Asia	Europe
	Selected Countries M/F	Selected Countries M/F	Selected Countries M/F
Water	207-169	326-327	0-1
Outdoor air	5-5	72-60	12-11
Indoor air	118-101	218-304	0-0
Lead	4-3	38-19	4-2
Climate change	18-18	35-38	0-0

(after Ezzati et al, 2002)

Table 2.1 Mortality (thousands of deaths) by environmental risk per year

However, this assessment was undertaken prior to the heat-wave in Europe of 2003. The World Health Organization (WHO) has also issued reviews of the overall impacts of climate change on health (WHO, 2001(a); WHO, 2004(b)), on heatwaves (WHO, 2004(a)), on the impact of climate on infectious diseases (WHO, 2005), on malaria (WHO, 2001(b)), and on methods to assess vulnerability to the impacts of climate change (WHO 2003(b); WHO 2007(d)).

2.4 National assessments of the impacts on health of climate change

A national impact assessment has been defined as an assessment at national level or below, undertaken by central or regional government, which explicitly addresses climate change and health, and involves formal methods of assessment, e.g. systematic literature review or modelling (Kovats *et al*, 2003(b)).

National investigations into the impact of climate change on health date from 1985, when the first report on future impacts of elevated carbon dioxide on health was published in the USA (White *et al,* 1985). Although limited by a lack of knowledge of the likely specific impacts of climate change, this report was a comprehensive outline of possible impacts of climate change on health, all of which are still being discussed today. The national estimates that have been undertaken since have reached a variety of conclusions, but all are broadly in line with this original report.

The first country in Europe to refer to the health impacts of climate change nationally was the UK (CCIRG, 1996). This report indicated that the incidence of food-borne and water-borne diseases might be expected to increase. In addition, more frequent heat waves would be expected to result increased mortality from cardiovascular and respiratory diseases, and existing health problems could be exacerbated by urban air pollution. An impact assessment of climate change in Wales found that the impacts of climate change on health would be a 'relatively minor challenge' to the health service, although the report noted that people on lower incomes would be disproportionately affected (Farrar and Vaze, 2000). Similarly, a comprehensive assessment of the impacts of both direct and indirect impacts of climate change on health in the UK (UK Department of Health, 2001) concluded that although climate change would have 'a significant effect' on health in the United Kingdom, not all the impacts would be negative, as winter mortality might be expected to reduce substantially.

By 2003, 16 countries had undertaken national health impact assessments, 8 of which were undertaken in tropical countries, and by 2007, eight other countries had undertaken such assessments. These reports are summarized (Table 2.2).

Country	Area of concern
Antigua & Barbuda	Coastal flooding, Hurricanes, Dengue fever
Australia	Change in malaria distribution, Increase in diarrhoea. Increase in heat related deaths
Bhutan	Increase in flooding and water-borne disease
Bolivia	Increase in malaria and leishmania
Cameroon	Possible increase in cholera, malaria, yellow fever, meningitis, malnutrition
Canada	Increase in heat related deaths, increase in vector borne disease. Environmental refugees
Fiji	Increase in dengue. diarrhoea
Finland	Increase in allergic disorders, reduction in winter mortality
Germany	Increased mortality from heat-waves and increased incidence of tick –borne disease
India	Increase in malaria and infectious diseases
Japan	Heat stress, air pollution, malaria
Kiribati	Increase in dengue, diarrhoea and ciguatera fish poisoning
Netherlands	Heat stress, flooding
New Zealand	Possible introduction of mosquito vectors into North Island
Panama	Possible increase in diarrhoea
Portugal	Heat related deaths, food and water borne disease, air pollution, vector and rodent borne disease
Spain	Increase in heat related mortality and infectious diseases
Sri Lanka	Malaria
St. Lucia	Increase in heat –related deaths
Switzerland	Increased mortality from heat-waves and increased incidence of tick –borne disease
Tajikistan	
United Kingdom	Heat related deaths and extreme events
United States	Heat stress, water borne disease, air pollution, flooding, vector borne disease
Zambia	Malaria, schistosomiasis, waterborne disease and malnutrition

(after Kovats et al, 2003(b); AR4, 2007)

Table 2.2 Summary of national assessments and areas of concern

All national assessments identified to date share two common themes, the importance of infrastructure in mitigating adverse health impacts and secondly, the lack of baseline data on which to base future estimates. The lack of baseline data is one factor that has resulted in the majority of national assessments to date being largely qualitative, and

the only countries having performed quantitative assessments of the likely impacts of climate change to date are the UK, Australia and Portugal. These assessments will now be briefly discussed and because their climates and socio-economic situations more readily approximate to the Irish situation, the assessments of the U.S., Canada, and New Zealand will also be considered.

2.4.1 United Kingdom

The United Kingdom assessment (UK Department of Health, 2001), was a quantitative analysis of the health impacts of climate change, together with a discussion of the impacts on health of mitigation policies. The report acknowledged that there were significant uncertainties relating to predicting future health impact of climate change. Nevertheless, a quantitative report was produced for three future time periods. The following conclusions were made for 2050, using an estimate that the emissions of greenhouse gasses would be in the medium to high range:

- A substantial reduction in cold related deaths from the present level of 60-80,000 deaths a year, by 20,000
- An increase in heat related deaths of 2,800 approximately
- An increase in numbers suffering from food-poisoning, of approximately 10,000 per annum
- The possibility of local outbreaks of malaria
- Possible increases in cases of cryptosporidiosis, a water borne disease and an increase in algal blooms
- A significant increase in the risk of major disasters, including winter gales and coastal flooding.
- A reduction in air pollutants in general, but an increase in the levels of ground level ozone.
- An increase of 30,000 cases of skin cancer, possibly reducing to 5,000 with reductions in emissions of chlorofluorocarbons.
- No increase in tick borne diseases
- Possible fall in air pollution levels, with a possible slight increases in ground level ozone

The researchers noted that measures taken to reduce the rate of climate change, such as reduced emissions of fossil fuels, could produce secondary beneficial effects on health and also recommended that an expanded research programme be put in place to further investigate the impacts of climate change on health as a matter of urgency.

2.4.2 Portugal

Portugal completed a quantitative assessment in 2002 (Santos *et al,* 2002), using two different regional climatic models. Using the Hadley model and assuming full acclimatization, the assessment estimated that in Lisbon in the year 2020 there would be a 7% increase in heat related deaths from the present levels of 6 deaths per 1,000, although if no acclimatization was assumed and a less conservative approach adopted, a six fold increase in heat related deaths could be expected. The study did not address changes in cold related deaths. More recent work (Casimiro *et al,* 2006) reported that a more substantial rise in heat related deaths, from between 5.4 and 6 per 100,000 to between 8.5 and 12.1 per 100,000 to a maximum of 29.5 by 2050s could be expected if no adaptation occurs. Similar to the U.K. report, the Portuguese assessment also indicated that there would be a decline in pollution from nitrogen dioxide but an increase in ground level ozone and aeroallergens, increases in the incidence of food and water-borne diseases, increases in both floods and droughts, and increase in the number of days suitable for malaria, dengue, and West Nile virus, although the risk of transmission would be low, under varying specified scenarios, due to socio economic conditions and access to health care. No change was expected in the incidence of Lyme disease as the area where disease transmission may increase (Central and Northern regions) is sparsely populated. An increased risk of leishmaniasis, Mediterranean spotted fever and leptospirosis was predicted. The issue of uncertainty was again addressed; the report noting both the scarcity of environmental and health data in Portugal and also the lack of knowledge of the relationship between climate and health, consequently concluding that no definite conclusions could be reached on the magnitude of change of some of the potential impacts of climate change in Portugal.

2.4.3 Australia

A quantitative assessment of the impact of climate change in Australia was completed in 2002, and the results outlined in Table 2.3 (Mc Michael *et al,* 2002). In the absence of adaptive measures, it was estimated that that there would be a substantial increase in

heat related mortality over the next 50 years, which would outweigh any reduction in winter mortality. Extreme rainfall would result in increases in annual flood related injuries and deaths by approximately 240%. Areas where malaria and dengue would become established were expected to move southwards, if public health infrastructures were not in place. A particularly vulnerable sub-group of the population would be the Aboriginal people, whose living conditions at present are poor. It was expected that the number of diarrhoeal admissions in this population would increase by approximately 10% by 2050.

Health outcome	Population	Exposure	Health impact
Thermal Extreme	10 Australian cities 2 New Zealand cities	Max temp Min temp	Mortality change in over 65s increases range from 8% in 2020 up to 56% by 2050 in Adelaide
Inland Flooding	Australia	Extreme rainfall	By 2020, annual flood related deaths and injuries may increase by 240%
Coastal flooding	Australia New Zealand Pacific island states	Sea level Topography Flood defences	Annual population exposed to coastal flooding
Malaria	Australia	Weekly temp and rainfall	Population living in potential malaria transmission zone
Dengue	Australia New Zealand Pacific island states	Annual vapour pressure	Population living in potential dengue transmission zone
Diarrhoeal Diseases	Central Australia (Indigenous)	Mean temp	Increase in incidence

(after Mc Michael et al, 2002).

Table 2.3 Impacts arising from climate change in Australia and New Zealand

2.4.4 United States

A qualitative report was issued by the United States and the health impacts were assessed individually in the categories outlined (Patz *et al*, 2000). The overriding feature of this report was the uncertainty involved in the assessments. The only definitive projection given was that there might be an increase in water borne diseases, due to deficiencies in watershed protection. The uncertainties were related to the ability of climatic models to accurately project regional-scale impacts, the sensitivity of human health to aspects of weather and climate, and the variable vulnerability of the population to any health risk. In relation to temperature-related health effects, although examples of heat related mortality were cited, the report was unable to determine whether there would be an increase or decrease in the number of people that might be

affected. Although the report found that the distribution of air-borne allergens would change, it stated that integrated air quality modelling studies would be required before the health impact could be assessed. Similarly, the report found that it was not possible to determine whether the incidence of vector diseases would increase or decrease with climate change. The report concluded that the levels of uncertainty preclude any definitive statement on the direction of potential future change for each of these health outcomes and recommended that more research be carried out, in order for more quantitative assessments to be undertaken.

2.4.5 Canada

There has been one non-quantitative assessment undertaken in Canada, in 1997; (Duncan, 2003), which found that increases could be expected in mortality and morbidity from high temperatures, extreme weather events, vector and rodent borne disease including hanta virus, Lyme disease and encephalitis and air pollution. Melting glaciers would lead to an increase in the flooding of coastal wetlands, which could lead to salt water intrusion in groundwater, and flooded treatment plants. Simultaneously, lake levels could decline, leading to water quality problems.

2.4.6 New Zealand

A qualitative assessment in 2001 by the Ministry of the Environment (Woodward and Hales, 2001) found that the direct impacts of climate change in New Zealand were likely to be small. It was estimated that parts of the North Island might become more receptive to dengue fever. Floods and droughts would also have an adverse impact on health. The risk of skin cancer would continue, and the report stated that the recovery of the ozone layer would be hampered by climate change. It was noted that New Zealand might need to provide for people living in the Pacific Islands who would be more severely affected.

2.5 Are health impacts already occurring?

Although there is evidence that climate change is affecting the natural world, as yet, there is very little evidence of this impacting on human health (ACACIA, 2001; Mc Michael *et al,* 2001). Nevertheless, the recent heat-waves in Europe cannot be ruled out (Beniston, 2004), and in addition, the possibility has been raised that a change in the

geographic range of tick borne encephalitis in Sweden (Lindgren *et al*, 2000), and the occurrence of malaria at higher altitudes in East Africa (Pascual *et al*, 2006) are linked to climatic change. However, it is difficult to causally link these changes to climate change, and calls for more frequent and long term sampling have been made (Kovats *et al*, 2001; Patz and Olsen, 2006).

2.6 Conclusion

In conclusion, although many calls have been made for studies on the impact of climate change on health to be undertaken, only a limited number of national assessments have thus far been carried out. Very few have been quantitative in nature, and the majority of these assessments have been qualitative and all are characterized by uncertainties, relating to a lack of baseline data. Nevertheless, it is clear that both direct and indirect impacts may adversely affect health, although vulnerability to these impacts is not uniform and is related to both individual and societal factors. No national assessment has been undertaken on the future health impacts of climate change in Ireland. It is the purpose of this study to attempt to quantify the possible health impacts of climate change, in the Irish context. The methodological considerations necessary to approach these issues will be examined in the next chapter.

CHAPTER 3 METHODOLOGY

3.1 Introduction

The diverse nature of the impacts of climate change on health outlined in Chapter 2 illustrates the wide ranging impacts of climate on health. It also highlights the challenges inherent in determining the future impacts of climate change on health. These difficulties have been acknowledged (Haines *et al,* 1993; Martens, 1998; Patz *et al,* 2000; Mc Michael *et al,* 2001):

> "*even without climate change and ozone depletion, the complexity of influences of various factors upon health effects defies a ready quantitative analysis of effects*"
>
> (Martens, 1998).

Methodologies to assess vulnerability to climate change are not readily available and calls for new methods have been made:

> "*Further development of methods and tools for vulnerability assessment appears warranted, especially for the human dimensions of vulnerability,...*"
>
> (IPCC, 2001)

The most recent IPCC (AR4, 2007) report echoes this:

> *"Little advancement has been made [*since the 2001 report*], in the development of climate-health impact models that project future health effects".*

In this chapter, the limitations of traditional epidemiological approaches will be discussed, and following this, both the data that is available in the Irish context and the methods that will be used to ascertain the impacts of climate change in the Irish context will be discussed.

3.2 Review of current epidemiological techniques

Techniques for the estimation of future health impacts from climate change are not as well developed as those which have been used in traditional epidemiological studies, which tended to focus on a specific agent as a cause of ill health e.g. an infectious micro-organism. On the contrary, the diverse impacts of climate change reflect complex, dynamic interactions between the organism and the environment, leading to specific challenges, and these have been outlined (IPCC, 2001; Mc Michael *et al,* 2001; AR4, 2007). Firstly, many of the health impacts of climate change are complex and non-linear. For example, a change in the incidence of salmonella may be related to a change in poultry practices, coincident with a change in temperature. There are many such confounding factors in the wider 'causal web' (Bernard and Ebi, 2001). Secondly, the nature of an investigation such as the impact of climate change on health entails the use of entire populations, as there is no 'unexposed' group. Studies that investigate populations as a whole, and not individuals, have been criticized because of the 'ecological fallacy', whereby conclusions drawn from populations do not always relate to the individual. Thirdly, adaptation may occur to adverse impacts and thus reduce the impact on health. Finally, although modelling is commonly used in studies of the future impacts of climate change, there are specific challenges associated with the use of models, as there is uncertainty in the simulation of many physical processes and feedbacks, for example, the role of clouds (IPCC 2001).

3.3 Methods currently in use

Despite these challenges, several methods have been used to study the impact of climate change on health (IPCC, 2001; UK Department of Health, 2001; WHO, 2003(b)), and these may be used in combination (Bernard and Ebi, 2001). These methods comprise: analogue studies that use the present climate-health relationship to foreshadow future impacts, with modelling of future health outcomes based on the relationship, predictive modeling of future outcomes, and the use of expert judgment. The disparate impacts on health that may be anticipated with climate change, and frequently, the lack of necessary data, necessitate the use of various methodological approaches in relation to

each expected outcome. Each of the methodological approaches outlined has its own advantages and weaknesses (Table 3.1).

Study	Advantages	Disadvantages
Analogue	Allows quantitative assessment Inexpensive, rapid Computation simpler	Limited application to populations with different vulnerabilities Not integrated Uses secondary data
Modelling	Quantitative Integration of numerous factors possible	Data intensive Complex, many uncertainties Expensive, time consuming Uses secondary data
Expert Judgement	Inexpensive, rapid Not data intensive Involvement of stakeholders possible	Qualitative results Imprecise, may be subjective

(after Santos et al, 2002)

Table 3.1 Advantages and disadvantages of study methods

A particular disadvantage in the Irish context is the lack of morbidity data, which poses a challenge in estimating the temperature-morbidity relationship. The use of a combination of approaches in different parts of the assessment, or at different stages of the analysis, has been recommended (Balbus *et al*, 1998). This approach has been used in other national assessments and is the approach that will be adopted in this study (Table 3.2).

Country	Method used
United Kingdom	Literature review, Expert judgement Predictive modelling
Australia	Predictive modelling
Canada	Literature review Expert judgement,
Netherlands	Literature review, Expert judgement,
New Zealand	Literature review, Predictive modelling
Portugal	Literature review, Predictive modelling,

(after Kovats et al, 2003(b))

Table 3.2 Methodological methods used in national assessments

Despite the difficulties inherent in attempting to establish the impact of climate change on health, such work is nevertheless necessary, in order to both identify those who are

most vulnerable to such effects, and also to prepare ourselves more fully to cope with such events (Martens, 1998).

3.4 Structure of study

The impacts of climate change on health will be divided into the generally accepted categories (Table 2.2), namely:

- temperature related mortality,
- morbidity related to water-borne disease
- morbidity related to food-borne disease
- morbidity related to other indirect effects, such as Lyme disease and skin cancer.

Each of these categories will be discussed in a separate chapter, and a final chapter will summarize the findings and make recommendations. A separate methodological approach will be used for the assessment of each of these impacts, and this will be outlined in the relevant chapter. However, reference will be made in all the chapters to the future temperatures that may be expected and therefore it is necessary to briefly examine how estimates of these temperatures are obtained.

3.5 The estimation of future temperatures

To estimate future changes in climate, two factors are necessary. Firstly, it is necessary to quantify the amounts of greenhouse gases that will be emitted in future time periods. As greenhouse gases are emitted from many sources, scenarios are used to estimate the quantities of future outputs of these gases. Secondly, it is necessary to equate the quantity of greenhouse gases emitted with the impact on climate that may be expected and climate models are used for this purpose.

3.5.1 Scenarios

The varied sources of greenhouse gases have been outlined (Section 1.3). In order to determine the quantity of future emissions of these gases, it is therefore necessary to examine such issues as future energy sources, population growth and land use. Scenarios are used for this purpose, and are defined as 'plausible and often simplified descriptions of how the future may develop' (IPCC, 2001). For the Fourth Assessment

Report, the IPCC used a set of scenarios that included specific assumptions in relation to technological and economic developments in society in the future. These scenarios are called SRES (Special Report on Emission Scenarios), and are grouped into 'four narrative storylines'. Each storyline contains assumptions about such issues as population growth, economic development and energy use, all of which will influence greenhouse gas emissions. The scenarios vary from future conditions that have a global focus as opposed to a more local focus, and also range from a consumerist outlook to a more conservative approach to the use of resources. Scenario A1 represents a world with rapid economic growth, with new and efficient technologies and a population that peaks in mid century. There are three subgroups based on energy sources in this scenario, and not all are primarily fossil fuel based. Scenario B1 reflects a more service orientated economy with a similar population trend as A1, but with greater equity. Scenario A2 reflects a self reliant world with more provincial enterprise and preservation of local identities. The population will continue to grow, but more slowly. Finally, scenario B2 emphasises local solutions for local problems, intermediate levels of economic growth, less rapid technological change and a slowly growing population. These greenhouse gas emission scenarios are used as inputs into global climate models, from which future climate predictions are developed.

3.5.2 Climate models

A climate model is a numerical representation of the climate system, using climatic and atmospheric data to estimate future climatic variables. There have been enormous developments in climate modelling since the first climate model was developed by Richardson in the early part of the twentieth century (Houghton, 1997). Advances in climate modelling are occurring continuously, leading to increased confidence in determining the magnitude of warming (AR4, 2007). The outputs from scenario driven climate change models can be used in vulnerability assessments to climate change (Hulme and Carter, 2002), and have been used in assessments of the health impacts of climate change in Australia (Mc Michael *et al*, 2002) and the UK (Donaldson *et al,* 2001(b)). A major disadvantage however, of models is their coarse resolution; Ireland is represented by only one grid square in the HadCM3 model. This issue has been has been overcome in the Irish context, by downscaling, using a technique which incorporates mesoscale (large scale) predictor variables in an empirical statistical technique linking global climate model output to surface observations (Sweeney *et al,*

2003). This has produced future estimations in Irish meteorological variables from the 1961-1990 average (Sweeney *et al,* 2003). There are uncertainties associated with the use of scenario driven climate change models, relating to such issues as the simulation of physical processes and feedbacks, in particular the role of clouds in climate models (IPCC 2001). The errors associated with such uncertainties may be minimized if the modelled baseline output is subtracted from the future output. This process allows the errors to effectively cancel each other out, and allows the result obtained when the models are subtracted from each other to be attributed to the impacts of climate change. This is the approach that will used in this thesis.

3.5.3 Establishing the present mortality-temperature relationship

Initially, it is necessary to initially establish the present temperature-mortality relationship. However, using a mean national temperature to establish this relationship would minimise peak values and consequently, information on the relationship between the extremes of temperatures and mortality would be less informative. This is important as more extremes of temperature may be expected in the future (AR4, 2007). It was necessary therefore to choose an Irish meteorological station that, while representative of Irish meteorological data, would also experience extremes of temperature and therefore be of benefit in developing the temperature-mortality relationship at extremes of temperature. Kilkenny being inland is not affected by coastal influences, has a more continental climate, and was therefore chosen as this station. In addition, while Irish precipitation patterns vary markedly, temperature displays more seasonal than spatial variation (Goodale *et al*, 1998). When the minimum and maximum temperature values for Kilkenny are compared to the values obtained in two other meteorological synoptic stations, namely Valentia in the South-west and Dublin airport in the East, in the time period, the values noted in Kilkenny are the most extreme (Table 3.3).

Station	Minimum	Maximum
Kilkenny	-13.40	31.4
Valentia	-6.8	28.4
Dublin	-7.9	28.7

Table 3.3 Range of minimum and maximum temperatures 1981-2002

As a result of the larger range of temperatures experienced in Kilkenny, it was possible to ascertain the temperature-mortality relationship at more extreme temperatures. Nevertheless, the temperature-mortality relationship for Kilkenny is comparable to the relationship established for both other stations (Figure 3.1).

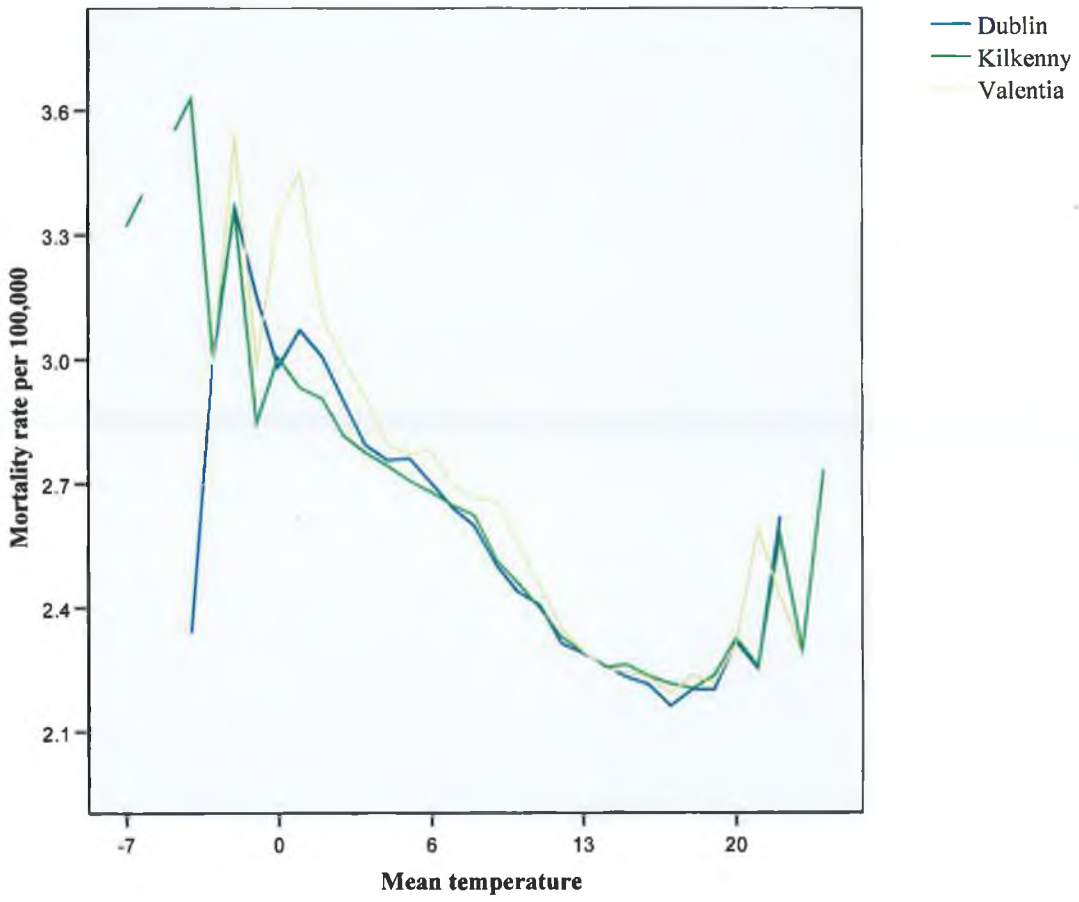


Figure 3.1 Comparison of temperature-mortality relationship in three locations in Ireland

Similarly, when mortality data was calculated in successive three degree bands of mean temperature, the range where total mortality is lowest was 17 to 20 using the Kilkenny data. This temperature range was comparable to that obtained from Valentia and Dublin (Table 3.4).

Station	3 degree Celsius band of mean temperature where total mortality is lowest
Kilkenny	17^0C -20^0C
Valentia	17^0C -20^0C
Dublin	16^0C -19^0C

Table 3.4 Ranges of mean temperature where mortality is lowest

3.6 Data sources

The purpose of this study is to investigate the future impacts of climate change on health in Ireland, and it was therefore necessary to obtain data on mortality and morbidity, and also on both present and future temperatures.

3.6.1 Mortality data

Mortality data was obtained from the Central Statistics Office (CSO) as follows. It is a legal requirement, in Ireland that every death that takes place in the State must be recorded and registered. The medical practitioner certifies the cause of death, and this information along with specified demographic criteria is recorded on the death certificate. The mortality data is classified under the International Classification of Diseases (ICD) system. This is the international standard diagnostic classification for epidemiological and many health management purposes (WHO, 2007(c)). The mortality data is registered centrally in the Registrar's Office and from here, the data is transferred to the CSO. In this office, the data is collated and analysed, and quarterly and annual reports are produced.

For the purposes of this study, daily mortality data was obtained from the CSO for the 22 year time period 1 January 1981 to 31 December 2002, for the following age groups: under 15, 15-64, 65-74, 75-84, 85 years and above, and total age groups. Under the

ICD classification, mortality data was obtained as follows: cancer ICD 140-239, all circulatory diseases ICD 390-459, ischaemic heart disease ICD 410-414, stroke ICD 430-438, respiratory diseases ICD 460-519, accidents and injuries ICD 800-999, and all cause mortality ICD 001-999. Mortality data was also obtained for deaths from heat stroke and hypothermia from the Central Statistics Office for the years 1981-2002.

To take the changing demographic structure into consideration, population data for census years and estimated population data for non-census years for the specified age groups for the years 1981-2002 were obtained from the Information Management Unit of the Department of Health and Children. As mortality due to accidents and injuries is generally accepted as not being temperature related, an age-specific daily mortality rate for the age groups under 14, 15-64, 65-74, 75-84, 85 years and above per 100,000 population was calculated for total mortality, omitting accidents and injuries. The term 'total mortality rate' as used subsequently in this work refers to the all-cause mortality rate per 100,000 population minus mortality resulting from accidents and injuries calculated on this basis.

3.6.2. Morbidity data

Data on the incidence of infectious food-borne and water-borne disease in Ireland is limited. Up to 2004, all infectious food-borne and water-borne disease had been categorized under three headings: food poisoning other than salmonella, gastroenteritis in children less than 2 years, and salmonella. This data is available from 1988. Weekly data on the incidence of salmonella was therefore obtained for the years 1988 to 2005. However from 2004, many more infectious diseases became statutorily notifiable, and for 2004 and 2005, weekly data on the incidence of campylobacter and giardia were also obtained. Although Verocytogenic E. Coli serotype 0157 was not statutorily notifiable until 2004, (when it came under the general VTEC category), a system of enhanced surveillance was in place from 1999 and monthly data is available on this disease from that year. Monthly data on the incidence of cryptosporidiosis was obtained for 2004 and 2005. In all instances, morbidity data were obtained from the National Disease Surveillance Centre, now the Health Protection Surveillance Centre.

3.6.3 Present temperature data

It was necessary to obtain daily temperature data in order to develop both the temperature-mortality relationship for temperature related mortality and the temperature-morbidity relationship for food-borne and water-borne disease. Minimum and maximum temperature values for Kilkenny for the time period 1981-2002 were therefore obtained to ascertain the temperature-related mortality. Temperature data was also obtained for the years 2004-5 in order to ascertain the temperature-morbidity relationship for food-borne and water-borne disease in order to correspond with the time period to which the data obtained pertains.

3.6.4 Future temperature data

Climatological projections for the years 2010 to 2099 were obtained from the results of another project undertaken by researchers at the Department of Geography, NUI Maynooth (Sweeney *et al*, 2003). Three climatic models were used, the Hadley Climate Model (HadCM3), the Canadian Centre for Climate Modelling and Analysis (CCCM) model and the Commonwealth Scientific and Industrial Research Organization (CSIRO) model. Climate change scenarios are generally presented for the years 2020, 2050 and 2080. In this study, three thirty year time periods were examined, namely 2010 to 2039, 2040 to 2069 and 2070 to 2099. The baseline time period was taken as 1961 to 1990. A synopsis of the temperature output from these three models follows, using scenarios A2 and B2, for the Synoptic Weather Station, Kilkenny as this data will be used in the study. Maximum, mean and minimum temperatures will be examined up to the year 2099, and summaries of these temperatures are outlined in the following section.

3.7 Future temperature values

The mean of the temperature outputs from the scenario driven models are depicted in this section, and will be depicted in both graph form and in tables of future temperatures. Maximum, mean and minimum temperatures will be examined separately. The apparent discrepancies seen in the graphs are artefacts of the modelling process. The first column in the table indicates the baseline modelled temperatures from which the future modelled temperatures are based, and does not depict the actual temperature

in this time period. Summer refers to the months of June, July and August, and winter to the months of December, January and February. The term A2 and B2 after the climate model in the first column of the tables refers to the scenario used (Section 3.5.2).

3.7.1 Maximum temperatures

Increases in maximum temperatures are evident (Figure 3.2 and Table 3.5).

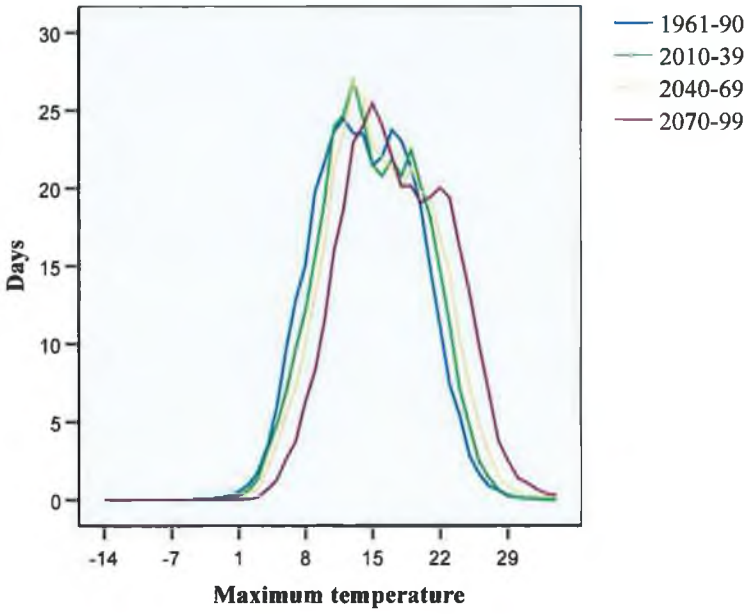


Figure 3.2 Changes in maximum temperature from baseline

	1961-1990	2010-2039	2040-2069	2070-2099
CCM A2	13.5	14.6	16.0	16.7
CCM B2	13.5	14.9	15.5	16.2
CSIRO A2	13.4	14.6	15.6	16.7
CSIRO B2	13.4	15.0	15.8	16.1
HadCM3 A2	14.6	15.0	16.0	17.9
HadCM3 B2	14.6	15.2	15.9	16.7

Table 3.5 Mean annual maximum temperatures in degrees Celsius

An increase in summer maximum temperatures, ranging from 0.6-1.6^{0}C in the earlier part of this century to 2.4^{0}C -4.2 ^{0}C towards the end of the century is anticipated (Table 3.6).

	1961-1990	2010-2039	2040-2069	2070-2099
CCM A2	19.2	20.7	22.3	23.2
CCM B2	19.3	20.7	21.5	22.3
CSIRO A2	19.2	20.3	21.3	22.3
CSIRO B2	19.1	20.7	21.3	21.5
HadCM3 A2	19.2	19.8	21.0	23.4
HadCM3 B2	19.2	19.9	20.9	21.9

Table 3.6 Summer maximum temperatures in degrees Celsius

However, most of the warming to date has occurred in the winter periods, where maximum temperatures have increased more than minimum temperatures (Mc Elwain and Sweeney, 2007). Nevertheless, increases are evident in the number of days when the maximum temperatures exceed the 95th percentile of the baseline value, particularly towards the end of the century (Table 3.7).

	95th percentile Degrees Celsius	2010-2039	2040-2069	2070-2099
CCM A2	21.7	19.9	45.4	60.3
CCM B2	21.7	19.2	32.9	47.1
CSIRO A2	21.8	11.9	30.6	51.5
CSIRO B2	21.5	19.1	32.4	36.9
HadCM3 A2	23.7	3.4	11.2	31.1
HadCM3 B2	22.9	4.1	12.9	21.7

Table 3.7 Mean number of days per year when the maximum temperatures exceed 95th percentile values of the maximum temperature in the 1960-1990 modelled baseline

3.7.2 Mean temperatures

A rise in annual mean temperatures is evident in all scenarios, increasing as time progresses (Figure 3.3).

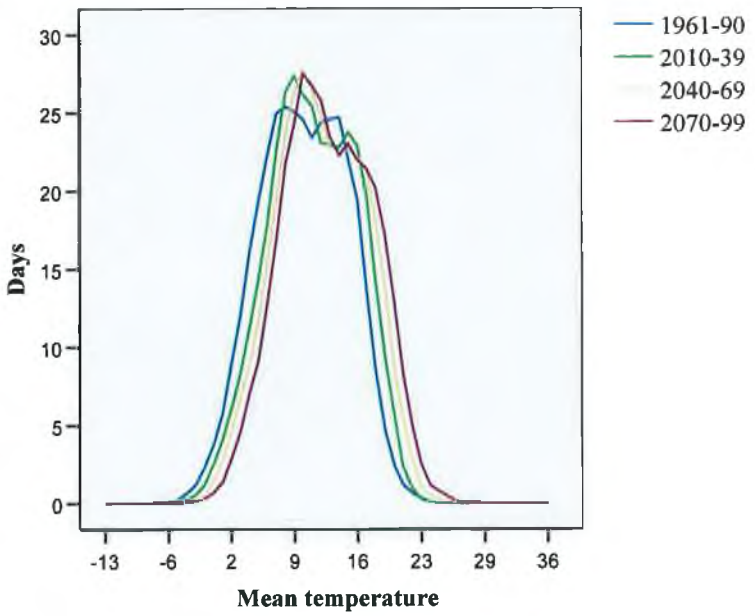


Figure 3.3 Changes in mean temperature from baseline

The increase ranges from 0.3 to 1.5 ^{0}C in the time period 2010 to 2039 to 1.6 to 3.3^{0}C in the time period 2070 to 2099 (Table 3.8).

	1961-1990	2010-2039	2040-2069	2070-2099
CCM A2	9.4	10.6	12.0	12.7
CCM B2	9.6	11.1	11.8	12.4
CSIRO A2	9.4	10.6	11.5	12.6
CSIRO B2	9.5	10.9	11.6	12.1
HadCM3 A2	11.3	11.6	12.6	14.2
HadCM3 B2	11.3	11.9	12.4	13.2

Table 3.8 Change in future annual mean temperatures in degrees Celsius from modelled baseline

A rise in summer mean temperatures is also evident (Table 3.9).

	1961-1990	2010-2039	2040-2069	2070-2099
CCM A2	14.6	16.0	17.4	18.4
CCM B2	15.5	16.7	17.4	17.9
CSIRO A2	14.6	15.6	16.4	17.4
CSIRO B2	14.9	15.9	16.5	16.5
HadCM3 A2	14.6	15.3	16.0	17.9
HadCM3 B2	14.6	15.2	15.9	16.7

Table 3.9 Summer mean temperatures in degrees Celsius

3.7.3 Minimum temperatures

Minimum temperatures are seen to increase throughout the century (Figure 3.4).

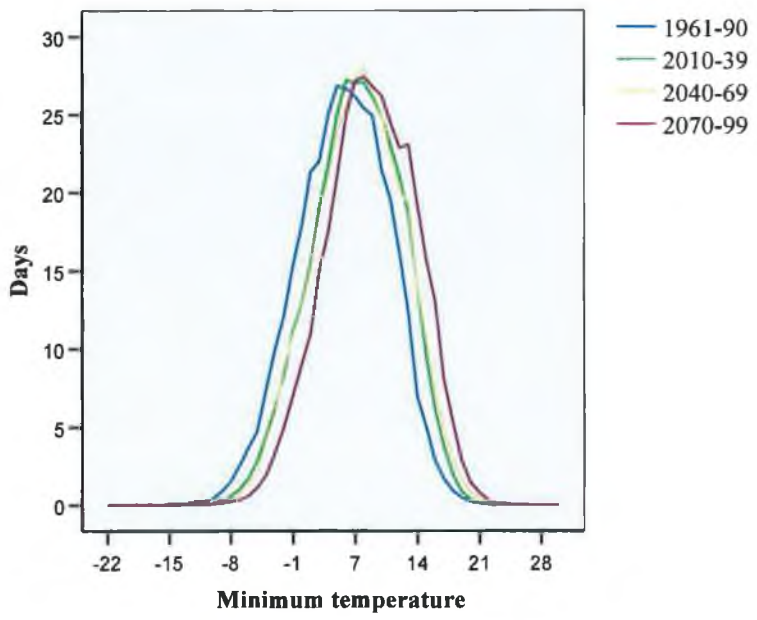


Figure 3.4 Changes in minimum temperature from baseline

In the spring, summer and autumn, minimum temperatures are increasing more than maximum temperatures (Mc Elwain and Sweeney, 2007), and a pattern of increasing annual minimum and winter minimum temperatures is evident in all scenarios, increasing as time progresses (Tables 3.10 and 3.11 respectively).

	1961-1990	2010-2039	2040-2069	2070-2099
CCM A2	5.4	6.5	7.9	8.7
CCM B2	5.6	7.3	8.1	8.6
CSIRO A2	5.4	6.5	7.4	8.6
CSIRO B2	5.4	6.8	7.5	8.0
HadCM3 A2	5.4	5.6	6.4	7.6
HadCM3 B2	5.4	5.9	6.2	6.8

Table 3.10 Mean minimum temperatures in degrees Celsius

	1961-1990	2010-2039	2040-2069	2070-2099
CCM A2	1.0	1.7	3.4	4.1
CCM B2	0.5	1.9	2.8	3.7
CSIRO A2	1.3	1.8	3.7	4.9
CSIRO B2	1.1	2.6	3.8	4.2
HadCM3 A2	1.5	2.3	2.1	2.8
HadCM3 B2	1.6	2.5	1.8	2.2

Table 3.11 Winter minimum temperatures in degrees Celsius

As expected, there is also a progressive decrease in the number of days with minimum temperatures less than the 5th percentiles of the baseline scenarios into the future (Table 3.12).

	Degrees Celsius	2010-2039	2040-2069	2070-2099
CCM A2	-1.5	-8.9	-22.6	-25.5
CCM B2	-2.4	-13.7	-23.8	-26.0
CSIRO A2	-1.3	-11.7	-18.5	-26.7
CSIRO B2	-1.1	-19.4	-22.0	-24.2
HadCM3 A2	-2.7	-1.5	-6.2	-11.6
HadCM3 B2	-2.6	-1.2	-3.6	-8.1

Table 3.12 Change in the number of days per annum when the mean minimum temperatures is less than the 5th percentile of the 1960-1990 modelled baseline

3.8 Changes in precipitation

Substantial changes are expected in relation to water resources; in relation to water quantity, overall increases of 11% in winter rain are predicted for Ireland, with the greatest increase of approximately 20% in the north-west. Winter runoffs will increase in north western areas, in south western and midland areas, and in the Wicklow mountains. The magnitude and frequency of individual flood events may increase in the western half of the country and seasonal flooding may occur over a larger area and persist for longer periods of time (Sweeney *et al,* 2003).

In contrast, reductions in summer precipitation will also occur, and an east-west trend will develop, with maximum drying expected to occur in the east. Major decreases in summer and autumn rainfall in the east and midlands, of approximately 25%, extending to 40% in some areas in the east may be expected. Decreases in winter rainfall of up to

25% may occur in the eastern areas, and significantly, this may not be adequate to recharge the rivers in the Wicklow mountains. This could exacerbate the reduced water storage caused by the reduced summer rainfall. An overall decrease in runoff is predicted for the years 2041 to 2070, with the exception of western Mayo, where a slight increase may occur in a small number of areas. In the time period 2071 to 2090, while the east-west trend in annual runoff will still be evident, drying is expected to be less (Sweeney *et al*, 2003).

3.9 Conclusion

Future climatic scenarios for Ireland have been briefly outlined. Alongside increases in mean temperatures, increases in the number of days with maximum temperatures over the 95th percentile of baseline values and reductions in the number of days when the mean minimum temperatures are less than the 5th percentile of the mean minimum temperature have been noted. Substantial changes in precipitation may also be expected. It is the purpose of this study to investigate the impact of these changes on the health of Irish people and to establish those who are most vulnerable. The future impacts of changes in temperature on mortality will now be investigated, but it will first be necessary to investigate the present impacts of the temperature changes on health and this is the subject of the following chapter.

CHAPTER 4 - IMPACT OF PRESENT TEMPERATURE ON MORTALITY

*"Hot winds ...render them [*inhabitants of cities*] paralytic when exposed suddenly to strong sunshine"*

Hippocrates (b)

4.1 Introduction

As a result of climate change, future temperatures are expected to increase and these changes have been outlined (Chapter 3). Ambient temperatures have an impact on well-being and it has long been known that extremes of temperature can be deleterious to human health. The physiological responses of the body to very hot and very cold temperatures underlie these adverse health impacts, and as they are markedly different, they therefore require separate consideration.

In higher ambient temperatures, the body utilizes several mechanisms to reduce heat, including dilation of the blood vessels in the skin, sweating, the production of hormones such as anti-diuretic hormone and an increase in the respiratory rate. If such mechanisms are not effective, for example, through illness or if conditions of high humidity inhibit sweating, heat-related illness and mortality may occur (Basu and Samet, 2002). The loss of fluid that arises from increased sweating leads to a relative increase in platelets, increasing the risk of myocardial infarction and cerebral thrombosis, i.e. heart attack and stroke. Even moderate dehydration resulting from warmer temperatures can precipitate a myocardial infarct in people with atheromatous arteries (Donaldson *et al*, 2003 (b)). Many diseases, in particular respiratory and cardiovascular diseases are sensitive to the physiological changes that result from exposure to heat, although mortality from heat stroke is rare. The increases in mortality resulting from excessive heat, and in particular arising from the recent heat-waves in Europe is acknowledged in the literature (Vandentorren and Empereur-Bissonnet 1995; Conti *et al,* 2005; Le Tertre *et al,* 2006), for example. The respiratory tract may also be adversely affected by heat, and also by pollutants, particularly ozone.

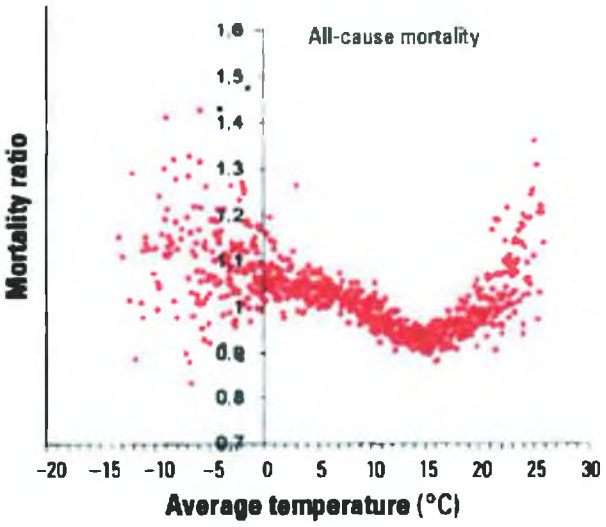
The physiological responses of the body to cold are generally mediated through the sympathetic nervous system, resulting in a constriction of blood vessels in the skin and

a consequent diversion of blood to the centre of the body to conserve heat. This also has the effect of raising both the blood pressure and the heart rate, and results in an increased risk of cardiovascular disease (Bokenes *et al* 2000; Keatinge and Donaldson, 1995; Donaldson *et al,* 1997; Nafstad *et al*, 2001). Cold air also has an adverse impact on the respiratory system, causing the smaller airways in the lungs to narrow, and the increase in respiratory mortality in cold weather is well documented (Eurowinter Group, 1997; Huynen *et al,* 2001; Carder *et al,* 2005, for example).

Age appears to be a significant determinant of susceptibility to temperature related deaths (Aylin *et al,* 2002; Eurowinter Group, 1997; Donaldson *et al*; 2003(b); Goodman *et al,* 2004, for example). Lying between the extremes of both hot and cold temperatures, is a temperature range where mortality is lowest, the minimum mortality band. Prior to the determination of the impacts of future temperature changes on Irish mortality rates, it is necessary to ascertain the present impact of temperature on mortality and also to ascertain the minimum mortality band. This is the purpose of the present chapter.

4.2 Minimum mortality band

It is clear that exposure to both extremes of high temperatures and low temperatures has adverse health implications. Arising from the higher mortalities observed at both ends of the temperature range, it is not surprising, therefore that many studies have described the temperature-mortality relationship as U shaped, with mortality highest at the extremes of temperature and lowest in the concavity of the curve in many European countries (Mackenbach *et al,* 1993; Kunst *et al*, 1993; Ballester *et al,* 1997; Saez *et al*, 1995; Huynen *et al*, 2001; Keatinge *et al*, 2000) (Figure 4.1).



(*Huynen et al*, 2001)

Figure 4.1 Temperature-mortality relationship in the Netherlands (mortality ratio is the number of deaths on day divided by the number of deaths over the study period)

This band of temperature will be referred to as the minimum mortality band or MMB. The establishment of the MMB allows calculation of the relative contributions of cold and heat to mortality. Cold and heat related mortalities have been defined respectively as all deaths occurring on days when temperatures were below or above the temperature range where mortality was at its lowest (Donaldson *et al,* 2001(b)). The present temperature-mortality relationship will now be examined and is the subject of the remainder of this chapter.

4.3. Data sources

4.3.1. Mortality data

Mortality data were obtained for the years 1981-2002 as discussed in Section 3.6.1. Cardiovascular diseases accounts for almost a half of total mortality in Ireland (Table 4.1).

Disease category	ICD 9 Code	Percentage
Respiratory	460-519	15
Cardiovascular (CV):	390-459	48
Ischaemic heart disease	410-414	26 (54% of total CV)
Stroke	430-438	10 (20% of total CV)
Cancer	140-239	24
Accidents and injuries	800-999	5
Other diseases		8

Table 4.1 Causes of mortality 1981-2002

Mortality from diseases generally regarded as being temperature sensitive, namely cardiovascular and respiratory diseases, accounted for almost two thirds of mortality in Ireland in the time period 1981-2002. Mortality from ischaemic heart disease and stroke accounted for three-quarters of the mortality from cardiovascular diseases. Cancer accounted for a quarter of total mortality, and respiratory mortality resulted in 15% of total mortality. There were no deaths in Ireland in the time series that were directly attributable to heatstroke; on the contrary, an average of 20 deaths per year is directly attributed to hypothermia (CSO, 2005).

Mortality, as expected, is highest in the older age categories, with over three quarters of all deaths in the time period occurring in the over 65 age group (Figure 4.2). These deaths occurred principally in people aged between 75 and 84 years, where over a third of the total mortality occurred.

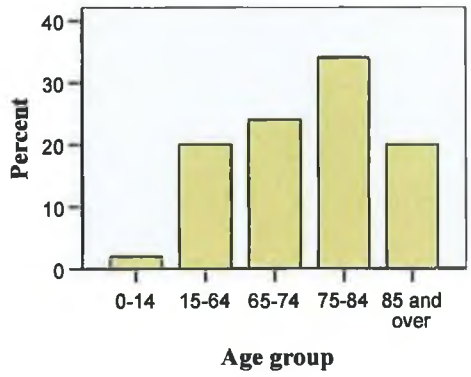


Figure 4.2 Age distribution of mortality data

4.3.2 Meteorological data

Daily maximum and minimum temperature data were obtained as discussed (Section 3.6.3). These daily maximum and minimum temperatures were averaged to produce a daily mean temperature value for each day in the 22 year time period (Hajat *et al,* 2002; Kovats *et al*, 2004) (Figure 4.3 (a) to (c))

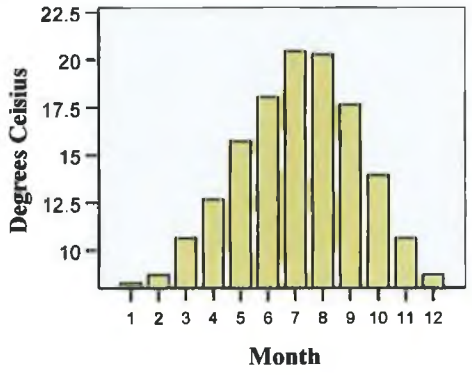


Figure 4.3(a) Range of maximum temperatures per month 1981-2002

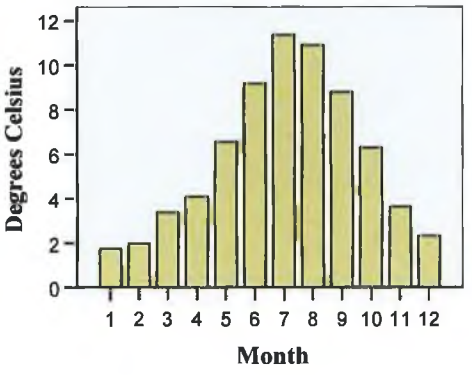


Figure 4.3 (b) Range of minimum temperatures per month 1981-2002

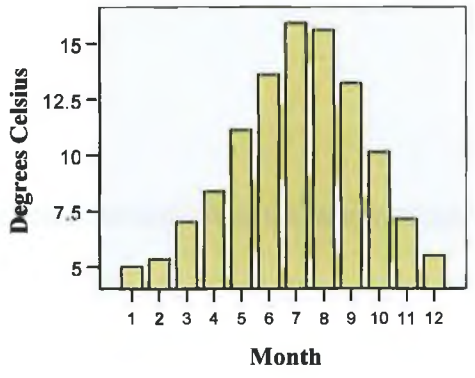


Figure 4.3(c) Range of mean temperatures per month 1981-2002

The mean temperature in the time series was 9.9^{0}C. The maximum temperature in the time series was 31.4^{0}C, and this temperature was reached on 2 days in succession, on the 13th and 14th of July 1983. The lowest temperature was reached on the 12 January 1982, when the minimum temperature was –13.4 ^{0}C.

4.4 Methodology

To assess the present impact of temperature on mortality, the temperature data were sub-divided into three degree bands of temperature, successively overlapping by 1^0C, for example, 11^0C -14^0C, 12^0C -15^0C, etc. for the range of temperatures experienced in the time series. The mortality rates in each band of temperature were determined, and the three degree range of temperature range where mortality was lowest was taken as the minimum mortality band, or MMB. In order to determine the incremental change in mortality above and below this range, the temperature-mortality relationship was established from the mid-point of this range (Mackenbach *et al,* 1993). Prior to establishing the temperature-mortality relationship, it was necessary to take the issue of declining mortality rates and also two temporal issues, namely the delayed impact of cold on mortality and the hastening of very high temperatures on mortality into account. The statistical package used was SPSS Version 14.0.

4.4.1 Declining mortality rates

Total mortality in the time period 1981-2002 has declined markedly, in particular, mortality from cardiovascular disease, and this has been largely attributed to improvements in life style and medical treatments (Shelley *et al*, 1991; Bennett *at al*, 2006).

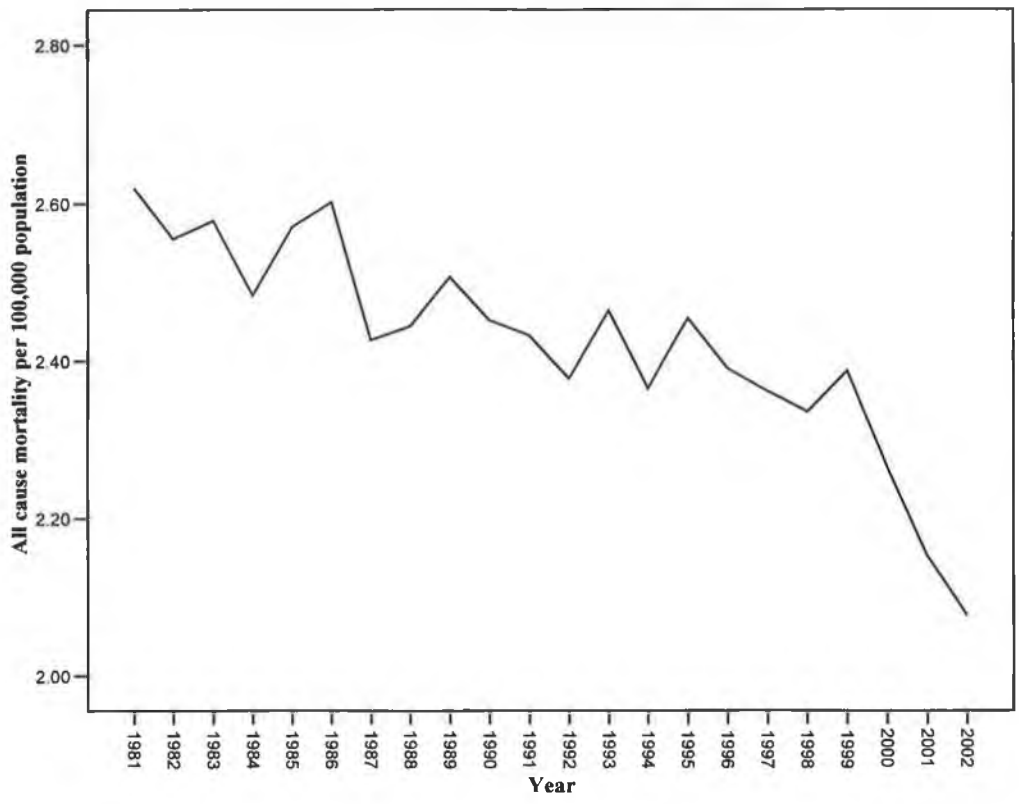
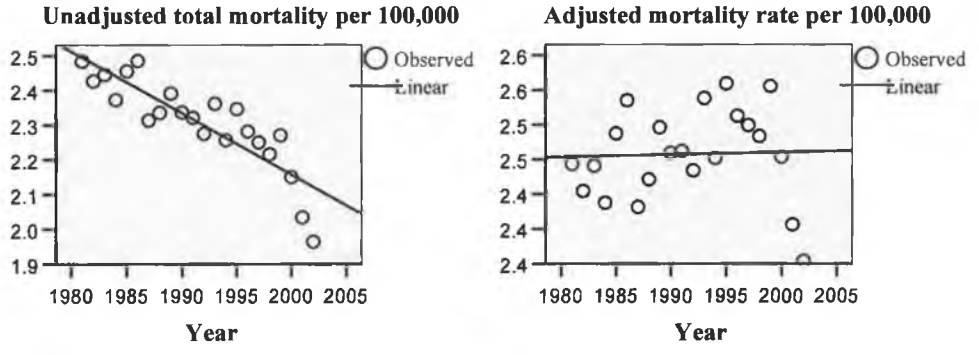


Figure 4.4 Trend in all cause crude mortality rate per 100,000 population 1981-2002 per day

The downward trend in total mortality is evident (Figure 4.4). Similar trends were seen in mortality in the specified age groups, and these are depicted in Appendix A (Figures 1 to 5). Slight increases in mortality are noted in 1986 in the age-groups over 65 and it was most noticeable in the over 85 year age groups. This may possibly have been due to influenza, although it is not possible to be definite in relation to this, as a register of this disease was only initiated in 2004. Influenza is recorded as a cause of death in approximately 40 deaths in Ireland per year (CSO, 2004). However no increase in influenza was noted in the U.K. in this time period (Elliot and Fleming, 2006). Trends in the mortality rates from specific diseases are also outlined in Appendix A (Figures 6 to 10). A decrease in mortality from cardiovascular diseases is evident. Although mortality from cancer also fell in the latter period of the time series, no overall discernible overall trend was evident in mortality from cancer or respiratory disease. In order to more closely identify the temperature-mortality relationship, it was necessary to remove the influence of the downward trends identified from the mortality data. This was achieved by regressing the mortality rates in the specified age groups and for each

disease category against year. To calculate the daily change due to these unrelated factors, the unstandardized regression coefficient b was divided by 365.25 and each daily death rate was adjusted by this factor, multiplied by 1 to 8035 (the number of days in the time-series). The change before and after adjustment for the downward trend in total mortality is shown (Figure 4.5 (a) and 4.5 (b)).



R square 0.756 p<0.001

(a) (b)

Figure 4.5 Total mortality 1981-2002 before (a) and after (b) detrending.

While there has been an increase in mean temperatures over the time period, the increase did not justify detrending, and similar to previous work (Donaldson *et al,* 2001; Mc Michael, *et al,* 2002), the time series was not adjusted.

4.4.2 Lagging

The impact of minimum temperatures occurs after a lag period, and appears to be disease specific, with mortality from cardiovascular disease generally preceding mortality from respiratory disease (Eurowinter Group, 1997; Bull and Morton 1973, for example). A straightforward approach was used to determine the number of days by which the data should be lagged. The mortality data was lagged by successive number of days in an iterative process, and regressed with temperatures, until a peak was reached in order to obtain the highest R square value. The number of days following the days with minimum temperature, on which the R square value peaked, was used, although the difference between individual R square values was low in many instances. In this way, the number of lag days at which the impact of minimum temperature on mortality was maximal was established (Tables 4.2 and 4.3).

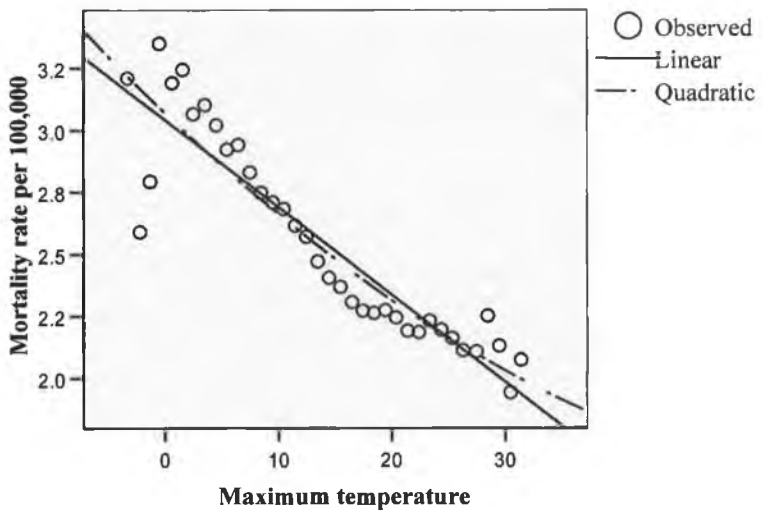
	0	1	2	3	4	6	8	10	11	12	14
14 and under	0.006	0.006	0.006	0.008	**0.009**	0.009	0.009	0.005	0.008	0.008	0.006
15-64	0.034	0.038	0.039	0.040	**0.042**	0.042	0.040	0.039	0.038	0.037	0.035
65-74	0.110	0.126	**0.131**	0.130	0.127	0.124	0.129	0.125	0.121	0.121	0.115
75-84	0.159	0.179	0.193	0.196	0.200	0.206	0.209	0.212	**0.213**	0.212	0.210
Age 85 and over	0.149	0.165	0.180	0.188	0.190	**0.201**	0.194	0.188	0.188	0.196	0.193

Table 4.2 R square values for relationship between minimum temperatures and total mortality in relation to age category and time lag

Days	**0**	**1**	**2**	**3**	**4**	**6**	**8**	**10**	**12**	**14**
Total	0.252	0.284	0.303	0.310	0.312	0.320	**0.321**	0.316	0.314	0.308
CV	0.212	0.232	0.243	0.245	0.245	**0.249**	0.247	0.243	0.237	0.236
IHD	0.135	0.144	**0.149**	0.145	0.148	0.144	0.140	0.136	0.129	0.136
Stroke	0.071	0.083	0.088	0.090	0.090	**0.098**	0.096	0.091	0.090	0.083
Cancer	0.003	0.006	**0.007**	0.007	0.007	0.007	0.005	0.005	0.006	0.004
Resp	0.191	0.206	0.220	0.233	0.241	0.247	0.261	0.257	**0.265**	0.263

Table 4.3 R square values for relationship between minimum temperatures and mortality in relation to disease category and time lag

When the impact of minimum temperatures was being assessed, the mortality data was lagged by the number of days outlined in bold (Tables 4.2 and 4.3). As impact of maximum temperatures is generally regarded to occur immediately, the data was not lagged when the impact of maximum temperatures was being assessed. Interestingly, although the R square values for the quadratic association increased if the mortality data was lagged by 2 days following the hottest days, the shape of the curve appeared to resemble the curve for minimum temperatures (Figure 4.6). This confirmed the decision to use unlagged data when assessing the impact of maximum temperatures.



R square 0.847 p<0.000

Figure 4.6 Association between lagged maximum daily temperature and all cause mortality per 100,000 population 1981-2002

Mortality data is generally not lagged when the impact of mean temperatures is being assessed (Donaldson *et al,* 2001(a), for example), and therefore this data was not lagged.

4.4.3 Harvesting

In contrast to the delayed impact of cold, the adverse impacts of extreme heat may have a 'harvesting' effect on mortality rates, whereby in an already vulnerable population, imminent death is hastened by the physiological stresses resulting from excessive heat. To date, although various methods have been used, no standard method of assessing mortality arising from heat waves has been established (Kovats and Ebi, 2006).

In this study, the impact of high temperatures on mortality patterns will be assessed by examining the overall changes in mortality in the days following the hottest days in the time series when compared to the corresponding time period in the remaining time series.

4.5 Results

The mortality in each successive three degree band of mean temperature was plotted against temperature, for each age group and disease category. The temperature-mortality relationship established will be presented in a graph format, with the equation of the relationship and the associated R square and p values.

Where quadratic equations are used, they will be expressed in the format

$$\text{Mortality} = a + b(t) + c(t)^2$$

Where linear equations are used, they will be expressed in the format

$$\text{Mortality} = a + b(t)$$

where t = temperature.

The turning point of the curve, i.e. the threshold temperature where mortality is lowest, was determined by identifying the 3 degree temperature range where mortality was lowest. The linear temperature-mortality relationship was ascertained above and below the midpoint of this temperature range where possible. The change in mortality per degree change in temperature was expressed as both a change in mortality per 100,000 and also as a change in mortality rate. This latter figure was obtained by dividing the b coefficient of equation by the mean adjusted daily mortality rate for the time period.

4.5.1 Mean temperatures

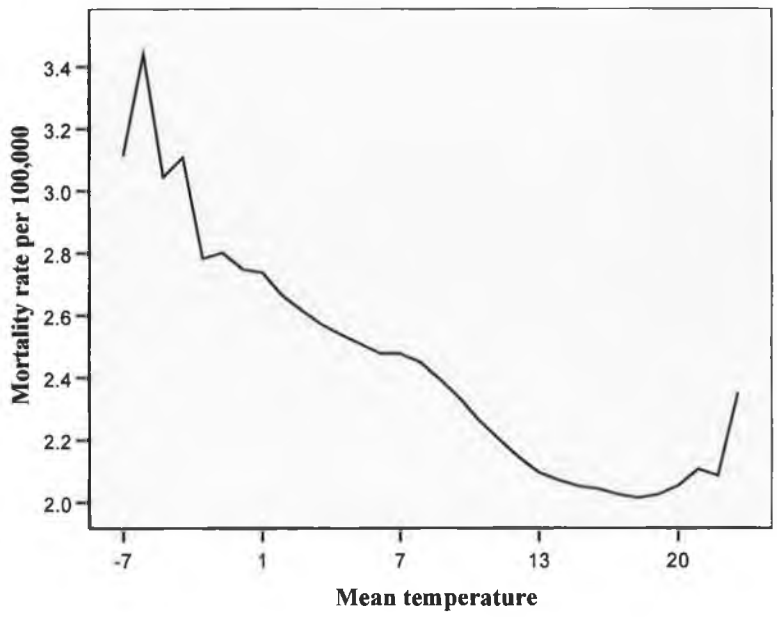
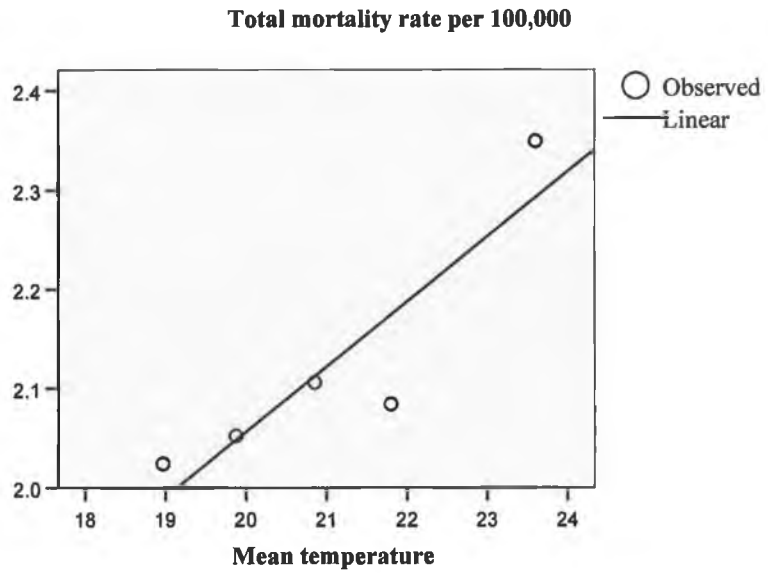


Figure 4.7 Association between daily mean temperature and all cause mortality per 100,000 population 1981-2002

A U-shaped curve is apparent when the relationship between total mortality and mean temperatures is assessed (Figure 4.7). The three degree band of mean temperature where total mortality is lowest was found to lie between 17^{0}C and 20^{0}C. Above 20^{0}C it was not possible to obtain a clear relationship between mortality and temperatures. However, above 18.5^{0}C (the midpoint of this temperature range), a linear relationship is obtained (Figure 4.8(a)).



R square 0.813 p<0.036

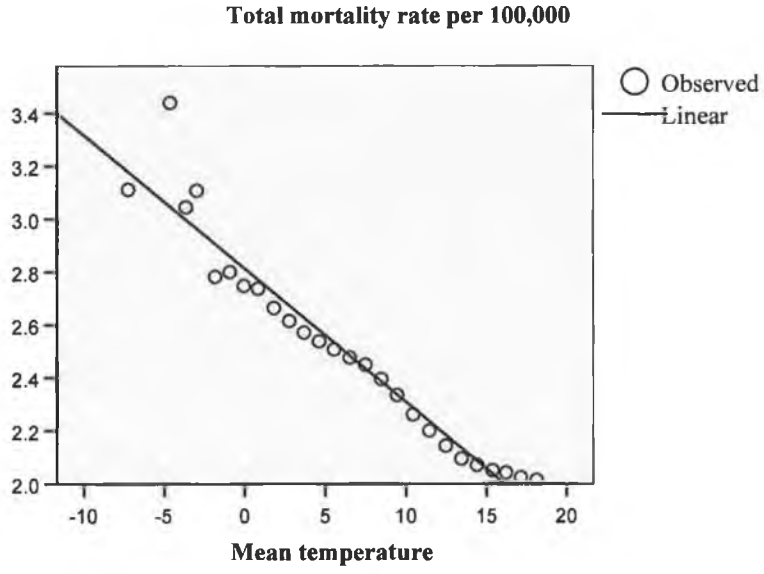
Figure 4.8(a) Relationship between mean temperatures and total mortality at mean temperatures greater than 18.5^{0}C

The equation of the line presenting the relationship between hotter temperatures above 18.5^{0}C and total mortality (Figure 4.6(a)) is represented by the equation

Mortality = 0.743+0.060(t)

Mortality increases by 0.060 per 100,000 per day per 1^{0}C increase at these higher temperature, and by 2.4% over the mean daily mortality of 2.51 per 100,000 in the adjusted time series.

If total mortality is regressed with mean temperatures below and including 18.5^{0}C, a clear linear relationship is also evident (Figure 4.8(b)).



R square 0.935 p<0.000

Figure 4.8 (b) Relationship between mean temperatures and total mortality at mean temperatures less than 18.5^{0}C

This relationship between colder temperatures and total mortality is represented by the equation

Mortality = 2.819-0.051(t)

Mortality increases by 0.05 per 100,000 per day, or by 2.0% over the mean mortality rate is 2.51 per 100,000 per day, per 1^{0}C decrease in temperature below 18.5^{0}C.

In the same way, the three degree range of temperature where mortality was lowest was identified and the incremental changes in mortality per degree change in temperature above and below the midpoint of the three degree band identified, for the specified age groups and disease categories. A similar relationship between mortality and temperature is evident for all age groups over 15 and for all disease categories apart from cancer. These results are outlined in full in Appendix B (Figures 1 to 5 for age-groups and Figures 6 to 11 for disease categories) and summarized (Tables 4.4 and 4.5).

Age Category	Range in brackets) and midpoint of threshold temperature ^{0}C	Increase in mortality per degree fall below midpoint of threshold	Increase in mortality per degree rise above midpoint of threshold	Percent of days with mean temperatures less than the midpoint of threshold
14 and under	(17-20)	**Not apparent**	**6.5%**	**97.6%**
15-64	14.5 (13-15)	0.7% 1.4	1.5% 1.1	79% 81.2%
65-74	19.5 (20-22)	2% 2.4	Not apparent	97% 99.0%
75-84	17.5 (16-18)	2.4% 2.5	3.7% 3.2	96% 95.2%
Over 85	21.5 (20-23)	3.9% 3.9	Not apparent	89% 99.9%
Total	18.5 (17-20)	2% 2	2.8% 2.4	96% 97.6%

Table 4.4 Incremental changes in total mortality in specific age-groups in relation to the threshold temperature and the percentage of days when mean temperatures are below this threshold

Age Category	Threshold Temperature ^{0}C	Increase in mortality per degree fall below threshold	Increase in mortality per degree rise above threshold	Percent of days with mean temperatures less than the temperature where mortality is lowest
CV	22.5 (21-24)	1.9 %	Not apparent	100 %
IHD	21.5(20-23)	2.1%	Not apparent	99.9%
Resp	19.5 (18-21)	4.1%	6.5%	99.0%
Stroke	17.5 (16-19)	2.0%	Not apparent	95.2%

Table 4.5 Incremental changes in mortality from specific diseases in relation to the threshold temperature and the percentage of days when mean temperatures are below this threshold

The threshold temperatures, the incremental changes in mortality in relation to changes above and below this threshold temperature, and the proportion of days when mean temperatures were below this temperature and in mortality in the specified age groups and disease categories are presented (Tables 4.4 and 4.5 respectively). It is clear, that the great majority of days in the time series had mean temperatures less than the threshold temperature where total mortality was lowest. In addition, while incremental increases in mortality were obtained for all age groups and disease categories at temperatures below this threshold, it was not possible to determine incremental increases in mortality for all age groups from higher mean temperatures, and it was only possible to determine incremental changes in mortality from respiratory disease at higher mean temperatures.

To ascertain if the temperature range where mortality was lowest had changed over the time period used to generate the temperature mortality relationship, an indication that acclimatization might have had occurred during the time-frame of the study, the data was divided into both four and two discrete time intervals and the three degree band where total mortality was lowest was ascertained in each time interval (Table 4.6). There was no discernible trend in the temperature range over time, and no evidence that a change had occurred in the temperature range where mortality was lowest.

Time interval	Temperature range Degrees Celsius
1981-1985	16-19
1986-1990	21-24
1991-1996	16-19
1997-2002	18-21
1981-1990	17-20
1991-2002	17-20

Table 4.6 Position of three degree temperature range where mortality was lowest relative to time

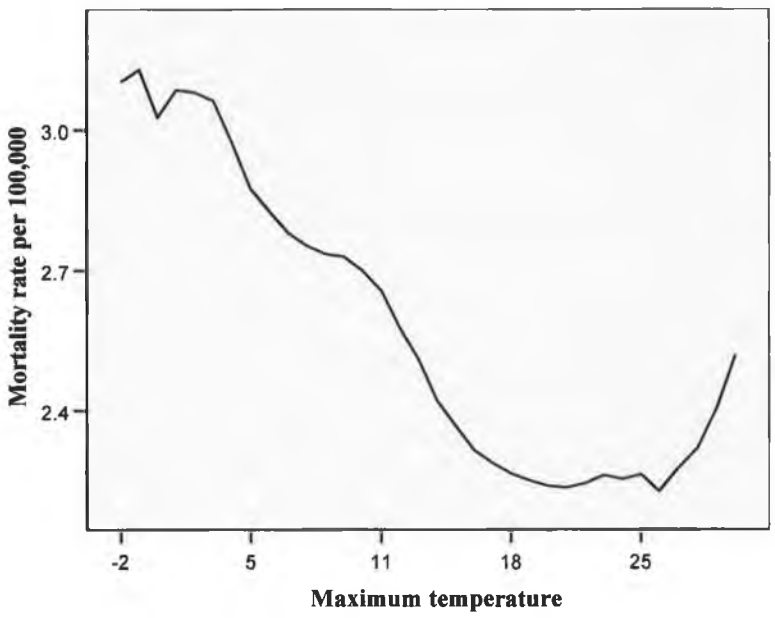
4.5.2 Extremes of temperatures

Maximum and minimum temperatures

Similar to the technique used to determine the impact of mean temperatures on mortality, the mortality rates was ascertained for three degrees of maximum and minimum temperatures.

Maximum temperatures

The characteristic U shape is evident (Figure 4.9(a))



R square 0.917 p<0.05

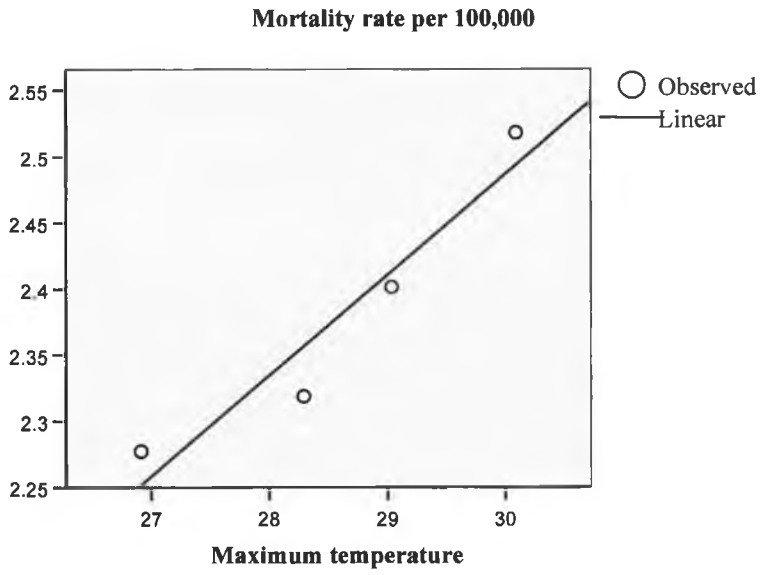
Figure 4.9(a) Association between daily maximum temperature and all cause mortality per 100,000 population 1981-2002

Mortality is lowest in the temperature range 25^0C to 28^0C. At temperatures above 26.5^0C, the midpoint of this range, mortality rises linearly (Figure 4.9(b)).

The relationship is expressed by the equation

Mortality = 0.200+0.076(t)

Mortality increases by 0.076 per 100,000 per day, or by 3.0% per degree rise above 26.5^0C over the mean mortality rate of 2.51 per 100,000.

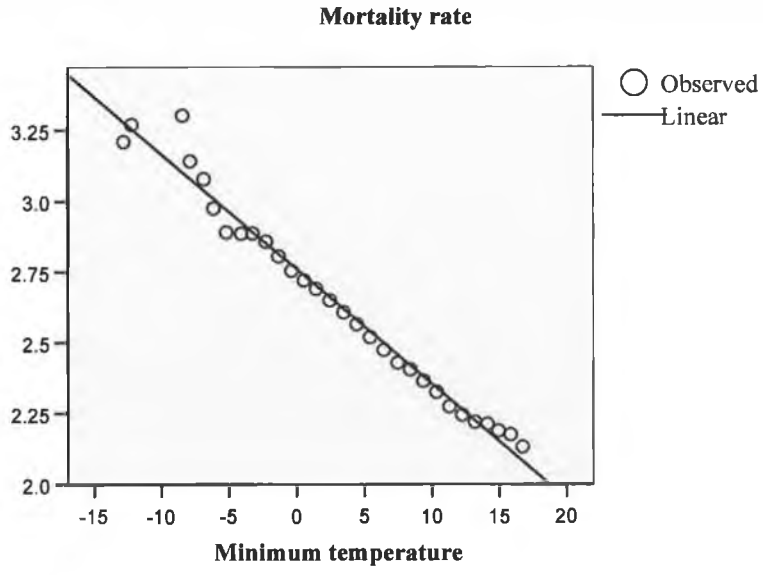


R square 0.917 p<0.05

Figure 4.9(b) Association between daily maximum temperatures above 28.5^{0}C and total mortality per 100,000 population 1981-2002

Minimum temperatures

On the contrary, the relationship with minimum temperatures is linear. No threshold temperature was apparent and mortality falls as minimum temperatures increases.



R square 0.980 p<0.001

Figure 4.10 Association between daily lagged minimum temperatures and total mortality per 100,000 population 1981-2002

The relationship is expressed by the equation

Mortality =2.760-0.041(t)

Mortality increases by 0.076 per 100,000 per day, or by 1.6% per degree fall in minimum temperature above the mean mortality rate of 2.51 per 100,000.

Similar to the procedure adopted for mean temperatures, the three degree band of maximum temperature where mortality was lowest and the threshold temperatures were identified for both maximum and minimum temperatures and the temperature-mortality relationship established with reference to the mid-point of this range temperature in the specified age groups and disease categories. The incremental changes in mortality per degree change in temperature above the maximum temperature threshold and the incremental fall in mortality per degree fall in minimum temperature was identified. The results are depicted in Appendix C, Figures 1 to 5 for age-groups and Figures 6 to 9 for disease categories. The results are summarized (Tables 4.7 and 4.8).

Age Category	Increase in mortality per 1^0C fall in minimum temperatures	Increase in mortality per 1^0C rise above maximum threshold	Maximum temp threshold
14 and under	3.6%	Insufficient days	25-28^{0}C
15-64	0.8%	1.0%	20-23^{0}C
65-74	1.9 %	1.2%	23-26^{0}C
75-84	2.7 %	1.3%	21-24^{0}C
Over 85	2.6 %	Insufficient days	26-29^{0}C
Total	1.6 %	3%	25-28^{0}C

(R square value in brackets, $p < 0.000$)

Table 4.7 Relative contributions of heat and cold to mortality in age categories

Disease Category	Increase in mortality per 1^0C fall	Increase in mortality per 1^0C rise above maximum threshold	Maximum temp threshold
Cardiovascular	1.9%	Insufficient days	15-18^{0}C
Ischaemic heart disease	1.9%	Insufficient days	25-27^{0}C
Stroke	1.3 %	Insufficient days	25-27^{0}C
Respiratory	2.7%	1.5%	20-23^{0}C

(R square values $p<0.05$)

Table 4.8 Relative contributions of heat and cold to mortality in disease categories

The elucidation of the incremental changes in mortality per degree fall in minimum temperatures, for both age categories and disease categories was straightforward, and incremental changes in mortality in these categories were established. However, it was

not possible to quantify incremental increases in mortality per degree rise in maximum temperature in all age groups and disease categories. While it was possible to derive incremental increases in mortality with increasing maximum temperatures for people in age-groups from 15 to 84 years, and total mortality from respiratory disease, it was not possible to derive a linear relationship in the 14 and under age group, and the 85 years and over age category for maximum temperatures, or in relation to mortality from cardiovascular diseases. It is clear that respiratory mortality is sensitive to both extremes of temperature, but appears to be most sensitive to minimum temperatures, as the incremental increase in mortality is greatest with these temperatures.

4.5.3 The impact of the hottest days

The impact of the hottest days was examined, by assessing the impact on mortality of the extremes of maximum temperatures. Mortality rates on days with high temperatures were examined during three specific periods of warmer temperatures, namely on summer (June, July and August) days in the time series, on days when the maximum temperatures exceeded the 99th centile of maximum temperatures, and also on the hottest day in the time-series. In addition, in order to examine the temporal pattern of mortality following the hottest day, mortality patterns both on and during time periods after the hottest day were examined, and compared to the mean mortality rates in the Summer months (June, July and August), excluding the year with the hottest days.

Although mortality rates on days when the maximum temperature is at the 99th percentile of maximum temperatures, are lower than the mean mortality rate in the time series, when the comparison period is confined to the summer months, the mortality rates on this day are higher than the mean mortality rate in summer, in all age groups aside from the age 85 and over age groups (Table 4.9).

Age category	Summer max 19.6^{0}C	99th percentile 27.3^{0}C	Mean mortality rates 1981-2002
14 and under	0.20	0.21	0.22
15-64	0.81	0.83	0.84
65-74	9.51	9.83	10.38
75-84	19.67	20.88	22.69
Over 85	51.57	49.76	60.35
Total	2.24	2.31	2.51

Table 4.9 Impacts of extremes of maximum temperature on mortality in age categories

Similarly, in relation to mortality in disease categories, although mortality rates on days when the maximum temperature is at the 99th percentile of maximum temperatures are lower than the mean mortality rate in the time series, the mortality rates on these days are higher than the mean mortality rate in the summer months in all disease categories, aside from cancer (Table 4.10).

Disease category	Summer mean 19.6^{0}C	99th percentile 27.3^{0}C	Mean mortality rates 1981-2002
Respiratory	0.26	0.29	0.34
Cardiovascular	1.21	1.23	1.34
Ischaemic heart disease	0.66	0.70	0.72
Stroke	0.26	0.27	0.30
Cancer	1.08	1.16	1.10

Table 4.10 Impacts of extremes of maximum temperature on mortality in disease categories

The hottest day in the time series in Ireland occurred on the 13th of July 1983, when the maximum temperature reached 31.4 ^{0}C. High temperatures were experienced throughout Europe during July 1983. The Azores anti-cyclone ridged strongly north-eastwards into Europe and the high temperatures experienced were without precedent for 200 years. New temperature records were established in West Germany (40.2^{0}C) and Austria (39.7^{0}C) (Ratcliffe, 1983). Similarly, in the U.K., July 1983 was the hottest summer for over 300 years (Jackson, 1983). This high temperature of 31.4^{0}C experienced in Ireland was also equalled on the following day. On this day, mortality rates in Ireland were higher than the mean annual mortality rates, and also higher than the mean mortality rates in the summer months of the time series. The increase in total mortality from the very hot day can be seen immediately on the 13th July, the hottest day (Figure 4.11). To assess the change in mortality, mortality on these very hot days was examined, and compared with mortality in June, July and August in the remaining years, when the average maximum temperature was 19.8^{0}C. Substantial increases were evident in the total mortality in each age group on the two hottest days, in particular, mortality in the over 85 year age group increased on these days (Tables 4.11 and 4.12).

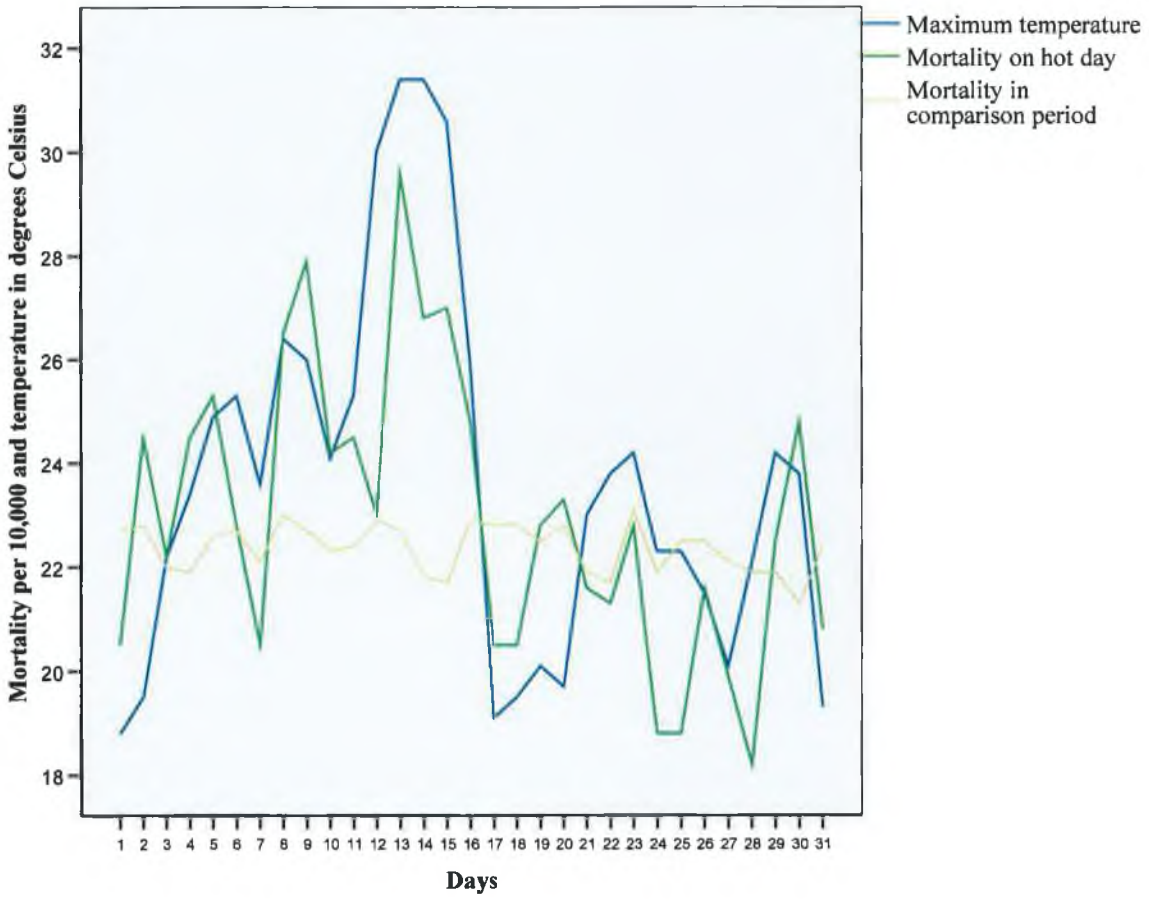


Figure 4.11 Maximum temperatures and total mortality on the hottest day (13th July 1983) and the following day

Although the temperature-mortality relationship was not as clear in the under 14 year age group, and mortality in this age group is low, mortality was also seen to increase substantially in the this age group on very hot days.

Age group	Mortality on hottest days	Mortality in June, July and August 1981-2002 excluding 1983	Percent change in mortality on hottest days
Under 14	0.49*	0.21	+133
15-64	0.98	0.79	+24
65-74	9.78	9.52	+3
75-84	26.11	19.49	+34
Over 85	86.20*	49.86	+73
Total	2.82*	2.22	+27

(Statistically significant at $p < 0.05$, $T_{20} = 2.09$)*

Table 4.11 Change in mortality on hottest day in age categories in comparison with corresponding time period in time series

Mortality in all other diseases was also seen to increase (Table 4.12).

Disease category	Mortality on hottest days	Mortality in June, July and August 1981-2002 excluding 1983	Percent change in mortality on hottest days
Respiratory	0.30	0.24	+25
Cardiovascular	1.47	1.20	+23
Ischaemic heart disease	0.75	0.65	+15
Stroke	0.33	0.26	+27
Cancer	1.17	1.08	+8

Table 4.12 Change in mortality on hottest day in disease categories in comparison with corresponding time period in time series

Using the Student's t-test distribution, mortality rates in the total age group, and specifically in the over 85 year and under 14 year age groups on this day were greater than the mean and 2.09 standard deviations of the comparison time period on the hottest day. Although mortality in all diseases was also seen to increase, none of these increases were statistically significant (Table 4.12).

It is known that very high temperatures may hasten death in those already ill (Whitman *et al,* 1997), a phenomenon known as 'harvesting'. In the days following the hottest day, a fall in mortality can also be seen (Figure 4.9), possibly indicating that harvesting has occurred and that mortality has been hastened in people who may have been very ill at this time. Studies of harvesting have generally used longer periods of time over which this phenomenon is detected (Grize *et al,* 2005; Conti *et al,* 2007, for example). In order to clarify this issue over the short period of time during which the hot temperatures prevailed, mortality patterns on each day in the month of July 1983 were plotted for the age-categories and disease groups, and are depicted in Appendix D (Figures 1-5 and Figures 6-10, respectively). The variation in mortality rates in one day are in contrast to the more even pattern evident when the mean of the comparison period is plotted. Nevertheless a peak is evident in mortality in the 75-84 year age group (Figure 4) and in total mortality from respiratory disease (Figure 9) in the hot period. To quantify the temporal pattern in mortality during this time, mortality rates on the hottest days and in the one week and two week periods following the hot days were ascertained and compared to the mortality rates in the months of June, July and August in the time series (excluding 1983), in age groups and disease categories. Aside from a slight increase in mortality in the 75-84 year old age-group, mortality rates in all other age groups fell from the peak in mortality noted on the hottest days, in the one week period

following the hottest days. Similarly, in the two week time period, mortality had also fallen in all age groups from the rates noted on the hottest days (Table 4.13). The changes in total mortality in the one and two week time period following the hottest days were not significantly different from the mortality rates in the comparison period, using the Student's t-test. This suggests that the statistically significant increase in mortality noted in the total age group, and specifically in the over 85 year and under 14 year age groups on the hottest days may indicate the hastening of imminent mortality, as none of the changes in mortality rates following the hottest days was statistically significant.

Age category	Mortality on hottest days	Mortality rates in the week following and including the hottest days	Mortality in the two weeks following and including the hottest days	Mortality in June, July and August 1981-2002 excluding 1983
Less 14	0.49*	0.22	0.19	0.20
15-64	0.98	0.91	0.86	0.80
65-74	9.78	9.22	9.31	9.53
75-84	26.11	26.30	22.77	19.62
Over 85	86.20*	56.27	58.34	51.68
Total	2.82*	2.46	2.33	2.25

*(* statistically significant at $p < 0.05$, $T_{20} = 2.09$)*

Table 4.13 Mean daily age-specific mortality rates per 100,000 following the hottest days

In relation to disease category, mortality from respiratory disease alone remained higher than the mean mortality rate in the corresponding month in the time series, but again, this increase was not statistically significant (Table 4.14).

Disease category	Mortality on hottest days	Mortality rates in the week following and including the hottest days	Mortality in the two weeks following and including the hottest days	Mortality in June, July and August 1981-2002 excluding 1983
Respiratory	0.30	0.31	0.31	0.26
Cardiovascular	1.47	1.28	1.19	1.21
Ischaemic heart disease	0.75	0.66	0.61	0.66
Stroke	0.33	0.27	0.25	0.26
Cancer	1.17	1.05	1.08	1.08

Table 4.14 Mean daily disease specific mortality rates per 100,000 following the hottest days

4.6 Discussion

4.6.1 Mean temperatures

The relationship between mean temperature and mortality which has been established, is in accordance with the literature, namely, that mortality increases at the extremes of temperature, and that vulnerability increases with age (Hajat *et al,* 2002; Goodman *et al,* 2004; Kovats *et al,* 2004(b); Carder *et al,* 2005).

Colder mean temperatures

The two day and six day lag periods found in the present study for ischaemic heart disease and stroke respectively are comparable to the two day lag noted with ischaemic heart disease and the three to five day lag noted for stroke (Bull and Morton, 1978; Eurowinter Group 1997), and also the one to six day lag noted for mortality from cardiovascular diseases (Carder *et al,* 2005).The impacts of minimum temperature on respiratory disease generally take a longer time to become manifest, and the lag times noted in this study associated with the impact of minimum temperatures (Tables 4.2 and 4.3), also accord with previous work. The 12 day lag found with respiratory disease in this study is comparable with the Eurowinter study (Eurowinter Group, 1997), and with the 13-18 day lag noted with respiratory diseases in Scotland (Carder *et al,* 2005), although it is longer then the 5 day lag noted previously (Bull and Morton 1973). While the impacts of minimum temperatures generally fall after approximately 14 days, although extending up to 18 days from respiratory mortality (Carder *et al,* 2005), the total impacts of cold temperatures on mortality may extend for up to 40 days (Goodman *et al,* 2004).

Cold has been found to be strongly associated with increases in total mortality in this study and the increase in total mortality of 2% per degree fall in mean temperature noted in the present study is comparable with the 2.6% increase in total mortality associated with every 1°C decrease in temperature over the subsequent 40 days in Ireland (Goodman *et al,* 2004), the 2.9% increase in mortality found when temperatures fell below 11°C in Scotland (Carder *et al,* 2005), and the findings of a increase in mortality of 1.37% at temperatures below 18^0C (Eurowinter Group, 1977). A similar

increase in total mortality of 1.4% when temperatures fall below 10 °C was noted in the Netherlands' (Huynen *et al,* 2001).

The vulnerability of older people to colder temperature is evident in the present study, as the incremental increase in mortality per degree fall below the threshold for mean temperature increased with age (Table 4.4), from 1.4% in the 15-64 year age-group to 3.9% in the over 85 year age group. Significantly, the great majority of excess winter mortality (87%) in Ireland occurs in people over 65 years (Healy, 2005). This increase in vulnerability with age is in accordance with both the Irish literature (Moran *et al,* 2000; Goodman *et al,* 2004) and the international literature (e.g. Bull, 1973; Eurowinter Group, 1997; Boulay *et al,* 1999; Huynen *et al,* 2001; Aylin *et al,* 2001; von Rossum *et al,* 2001; Gemmell *et al,* 2002 for example). The increase in mortality over a 40 day period was noted to increase from 1.4% in the under 65 year age group, to 2.8% and 3.0% in the 65-74 and over 75 year age group respectively in a Dublin study (Goodman *et al,* 2004). An increase of 1.5% in the probability of dying in winter for every 1°C reduction in mean winter temperature, in people over the age of 65 was observed in the U.K. (Aylin *et al,* 2001). No clear impact of cold temperatures on children could be discerned, and few studies have investigated this issue, but a summer- winter variation in excess of 200% in the 0-9 age group has been found (Gemmell *et al,* 2000).

In relation to disease categories, respiratory disease appears to be the most sensitive to colder mean temperatures in this study, with increases of 4.1% noted per degree fall in temperature below the threshold. A similar sensitivity of respiratory disease to cold temperatures was again previously seen in Ireland, where an increase in mortality from respiratory disease of 6.7% for every degree fall in minimum temperature over a 40 day period was noted (Goodman *et al,* 2004). These results are also comparable with the 5.2% increase noted in mortality from respiratory disease in the Netherlands at temperatures below 16.5^{0}C (Huynen *et al,* 2001), and the 4.8% increase recorded in Scotland when temperatures fall below 11^{0}C (Carder *et al,* 2005*),* but higher than the 2.1% increase noted in Norway at temperatures less than 10^{0}C (Nafstad *et al* 2001), although as will be seen later, insulation standards in Norway are higher than they have been to date in Ireland. The increase in cardiovascular mortality of 1.9% per degree fall in temperature noted in the present study, is also comparable to the 2.5% increase for every degree fall in minimum temperature over a 40 day period in Ireland (Goodman *et*

al, 2004), the 3.4% increase in mortality from cardiovascular disease when temperatures fall below 11^0C in Scotland, but again higher than the 1.7% increase in mortality from cardiovascular disease when temperatures fell below 10^0C in Norway (Nafstad *et al,* 2001), and the 1.7% increase in mortality from cardiovascular disease when temperatures fall below 10 °C in the Netherlands (Huynen *et al,* 2001). Stroke, with an incremental increase in mortality of 2.0% per degree fall in temperature also appears to be sensitive to colder temperature, and the incidence of stroke has been noted to be higher in winter (Azevedo *et al,* 1995), although in general, mortality from stroke appears to have the lowest seasonal component (Aylin *et al* 2001; Gemmell *et al,* 2000). This work confirms the sensitivity of respiratory and cardiovascular diseases to cold temperatures, established in the literature (Hajat *et al,* 2002; Huynen *et al,* 2001; Kunst *et al,* 1993; Gemmell *et al,* 2000; Eurowinter Group, 1997; Nafstad *et al* l, 2001; Hajat and Haines, 2002; Goodman *et al,* 2004; Paldy *et al,* 2005; Carder *et al,* 2005). Evidence of this impact may be seen after exposure to even mild cooling temperatures (Bokenes *et al* 2000; Keatinge and Donaldson, 1995; Donaldson *et al,* 1997).This sensitivity to cold is related to the impact of cold on diverse physiological processes, including increased blood pressure (Elwood *et al,* 1993; Donaldson and Keatinge, 2002; Donaldson *et al* 1997; Goodwin *et al,* 2001), alterations in the levels of chemicals which increase the tendency of the blood to form clots, including increases in the number of red cells, platelets, fibrinogen, and plasma viscosity (Keatinge *et al,* 1984; Donaldson *et al,* 1997; Elwood *et al* 1993; Woodhouse *et al,* 1994), possibly cholesterol (Elwood *et al,* 1993; Donaldson *et al* 1997; Keatinge *et al,* 1984), and the effect of cold itself (Zhang, 2004). In addition, variability in blood pressure in cold weather may contribute to the high cardiovascular mortality observed in the winter (Jehn *et al,* 2002, Gomez-Angelats *et al,* 2004).

Warmer mean temperatures

The increase in total mortality of 2.4% per 1^0C rise in mean temperature above 17.5^0C in this study is higher than the 0.4% increase in mortality found previously per degree rise in mean temperatures (Goodman *et al,* 2004), although it is comparable to the increase of 3.3% in total mortality per 1^0C rise in mean temperature above 21.5^0C noted in the UK (Hajat *et al,* 2002), and the 1.3% increase in mortality found above 18^0C in London (Pattenden *et al,* 2003). An incremental increase in mortality in the 15-64 and the 75-84 year age groups of 1.1% and 3.2% respectively was identified. An increase in heat related mortality in the 65-74 and over 75 year age groups, of 0.7% and 0.3%

respectively has been noted previously in Ireland (Goodman *et al,* 2004). Interestingly, the incremental increase in total mortality, at 2.4% was higher than the corresponding decrease of 2% as temperatures fell below the threshold. Additionally, mortality in the 75-84 year age group was higher at mean temperatures above the threshold than below it, possibility indicating a particular vulnerability in this age group (Table 4.4), although it may also be partly due to people aged 85 years and over staying inside on very hot days, and thus being shielded from the extreme heat.

In contrast to the incremental changes in mortality at temperatures established below the temperature threshold, it was more difficult to determine the incremental increase in mortality from specific disease categories above the threshold, due to insufficient number of days with such temperatures. However, a 6.5% increase in mortality was noted from respiratory disease per degree rise as mean temperatures rose above 19.5^0C. The sensitivity of respiratory mortality to warmer temperatures in the Irish context has already been established (Goodman *et al,* 2004), where, in people aged over 65, increases mortality from respiratory disease of 0.8% were found in Dublin, per 1^0C rise in mean temperature, although, as in the present study no change in mortality from cardiovascular disease was seen. The sensitivity of respiratory disease to higher mean temperature has also been found elsewhere. In the Netherlands, increases in mortality from respiratory disease (12.8%), heart disease (1.9%), all causes (2.7%), and cancer (0.5%) have been noted in people over 65, when mean temperatures rise above 16.5^0C (Huynen *et al,* 2001). Similarly, in Norway, respiratory mortality increases by 4.7% for every 10^0C rise above the previous 7 days mean temperature (Nafstad *et al,* 2001). In Hungary, at mean temperatures above 23^0C, total mortality increases by 10.6%, however cardiovascular disease appears to be more sensitive than respiratory disease, with increases of 18% and 8.8% respectively (Paldy *et al,* 2005). In the U.K., again respiratory disease is the most temperature sensitive, with increases in mortality from respiratory and cardiovascular disease of 5.5% and 3% respectively per 1^0C rise in mean temperature above 21.5^0C (the 97^{th} percentile) noted (Hajat *et al,* 2002).

The absence of a definite relationship in the present study between cancer and mean temperatures is consistent with the literature (Huynen *et al,* 2001, Eurowinter Group,1997, Mackenbach *et al,* 1993, Van Rossum *et al,* 2001) although a low seasonal variation in cancer deaths in the order of 5% has been found (Gemmell *et al,* 2000).

Minimum mortality band

The range of mean temperature obtained in the present study where total mortality is lowest (17^0C to 20^0C), and above which mortality begins to rise, namely the minimum mortality band or MMB, is higher then the 15.6^0C to 18.6^0C band found in the UK (Donaldson *et al*, 2000), but is comparable to recent work in the UK, where a band of mean temperature between 17^0C and 18^0C was established, above which heat mortality begins to rise (Hajat *et al,* 2007). Similarly, the threshold temperature established in the present study is higher than the mean temperature (May, June, July and August) in Ireland of 14.1^0C. This phenomenon, whereby the mean summer temperature is lower than the temperature ranges where mortality is lowest, has also been noted previously (Keatinge *et al,* 2000) (Table 4.15).

Region	Temperature band of lowest mortality Degrees Celsius	Mean summer Temperature (May to August)
North Finland	14.3 to 17.3	13.5
South Finland	13.3 to 16.3	14.5
Netherlands	17.3 to 20.3	16.1
London	19.3 to 22.3	16.9
Baden-Württemberg	19.0 to 22.0	17.7
North Italy	16.8 to 19.8	20.7
Athens	22.7 to 25.7	24.1

(after Keatinge et al, (2000))

Table 4.15 Temperature ranges where total mortality was lowest in 65-74 age group

The three degree band of mean temperature range at which mortality in the 65-74 year age is at its lowest in Ireland was established in the present study as the range 18^0C to 21^0C. This range is comparable to work undertaken in the same 65-74 age group in the Netherlands and London, being in the mid-range of the temperature ranges (17.3^0C to 20.3^0C, and 19.3^0C to 22.3^0C respectively) previously noted (Table 4.15) (Keatinge *et al*, 2000).

The three degree band of mean temperature where total mortality is lowest established in the present study (17^0C to 20^0C), allows a quantification of cold and heat related mortality to be made. Such mortality is determined by multiplying the mortality rates on days when the mean temperature is above or below the range, by the proportion of days when the mean temperatures are above or below this range (Donaldson *et al,* 2001(b)).

The following formulae may be used: cold related mortality =
Mortality rate on days with mean temperatures below the MMB x Frequency of days with mean temperatures below the MMB

Conversely, heat related mortality =
Mortality rate on days with mean temperatures above the MMB x Frequency of days with these temperatures above the MMB
Total mortality below 17^0C is 2.53 per 100,000, while it is 2.53 per 100,000 at temperatures higher than 20^0C. There are 7,547 days in the time-series of 8,035 days when temperatures were below 17^0C and 49 when temperatures were above 20^0C.

Therefore the heat related mortality per year = $\frac{(2.34 \times 49)\times 365}{8035}$ =5
per 100,000

and cold related mortality per year = $\frac{(2.53 \times 7547)\times 365}{8035}$ =867
per 100,000

Mortality per 100,000	Ireland
Heat related mortality	5
Cold related mortality	867

Table 4.16 Relative contribution of mean temperatures to total mortality

Cold-related mortality clearly exceeds heat-related mortality in the present study. Cold-related mortality also exceeded heat-related mortality in the U.K., where at 1,409 per 100,000 population, it exceeded heat-related mortality at 1.4 per 100,000 (Table 4.17) (Donaldson *et al,* 2001(a)).

Temperature related mortality per million	Ireland	United Kingdom
Heat related	5	1.4
Cold related	867	141

Table 4.17 Present mean temperature related mortality per 100,000 in Ireland and the United Kingdom

The respective cold and heat related mortalities for the regions listed in Table 4.17 have been calculated (data derived from original paper), and compared with the results of the UK assessment and the present study (Table 4.18).

Region	Cold related Mortality per 100,000 (a)	Heat related Mortality (b)	Ratio of (a) to (b)
North Finland	246	30	8
South Finland	138	25	6
Netherlands	135	5	27
London	313	4	78
Baden- Württemberg	194	11	18
North Italy	124	33	4
Athens	253	45	6
UK*	141	1.4	101
Present study**	867	5	173

(data from Keatinge et al, 2000; Donaldson et al, 2001* *and present data***)*

Table 4.18 Comparison of cold and heat related mortalities per 100,000

The results of heat-related mortality in the present study are comparable to figures obtained in London and the Netherlands (Table 4.18), in contrast, it is clear that cold related mortality in Ireland is substantially higher than heat related mortality in all regions assessed. This marked excess of cold related mortality requires further consideration. For a country with an equable climate, Ireland has had, and continues to have higher winter mortality rates than might be expected from the impact of temperature alone. The higher cold related mortality in Ireland, reported in this study, is consistent with earlier work in Ireland (Healy, 2003) and internationally (Curwen, 1990/1; Mc Kee, 1989). Although seasonal mortality in Ireland is not officially calculated on a national basis (Keating, 2005), recent work (Healy, 2003) on seasonal variation in mortality in 14 European countries (estimated by calculating the surplus number of deaths occurring during the winter season compared with the average of the

non-winter seasons), has reported a proportionately lower seasonal variation in mortality in the UK (0.18) than in Ireland (0.21), although both countries had higher seasonal mortalities than the mean of 0.16. This indicates that Irish mortality rates are 21% higher in winter than the mean mortality rate. Cold related mortality in Ireland, with an estimated excess of 2,000 winter time deaths, is one of the highest in Europe, and factors such as poor insulation and fuel poverty have been incriminated (Healy, 2003). Almost a million Irish homes are estimated to be poorly insulated and energy inefficient (Dunphy, 2007). The importance of good quality of housing, heating, nutrition and clothing in counteracting the impact of cold has been stressed in the Irish context (Moran *et al*, 2000; Middleton *et al,* 2000) and internationally (Donaldson *et al,* 2001(a)). The importance of housing standards and insulation can be clearly seen when Ireland and Norway (where high standards of housing insulation prevail), are contrasted. Although the mean January temperature in Norway is lower than Ireland (-1.1^{0}C and 5^{0}C respectively), and although the crude mortality rates are comparable, excess winter mortality rates are over five times higher in Ireland (Clinch and Healy, 2000). In addition, the mean seasonal variation in mortality from heart disease at 35% in Ireland is greater than the 22% variation in Norway (Eng and Mercer, 1998).

The three degree band of mean temperature established for Ireland, where mortality is lowest at 17^{0}C to 20^{0}C was higher then the three degree range of 15^{0}C to 18^{0}C established in the UK study (Donaldson *et al,* 2001(a). As a result, there was an average of 2 days per year in the present Irish time series when the mean temperatures rose above 20^{0}C, in comparison to an average of 13 days per year when mean temperatures in the UK rose above the temperature range of lowest mortality, thus leaving the estimate of heat related mortality open to a wide margin of error. Nevertheless, it is clear that heat related mortality is significantly less then cold related mortality in both countries.

In conclusion, the majority of days in Ireland have mean temperatures below the temperature at which mortality is lowest. The majority of the mortality occurs in the lower temperature region of the temperature-mortality graphs and resulting from the dominant impact of cold on mortality rates, there were insufficient days above the threshold by which the incremental increases in mortality from disease categories at warmer temperatures could be determined.

4.6.2 Extremes of temperature

It is clear that while both extremes of cold and hot temperatures are deleterious to health, the present evidence indicates that minimum temperatures appear to be more deleterious than maximum temperatures. This is sadly underscored by the fact that, notwithstanding the possibility of adverse social conditions contributing, an average of 20 deaths a year occurs from hypothermia, while no deaths have been recorded in Ireland as having been caused by heatstroke due to climatic conditions.

The impact of minimum temperature on mortality is straightforward, and the dominance of the linear relationship observed, facilitates quantification of the incremental increase in mortality per degree fall in temperature in all age groups over 15 and in disease categories (Figure 4.8 and 4.9). The incremental increases in mortality per degree fall in temperature, increases with age, indicating that the vulnerability to cold increases with age. The incremental increase of 0.8% in the 15-64 age group, increased substantially to 2.6% in the over 85 year age group. Total mortality increases by 1.6% per degree fall and respiratory disease appears to be the most sensitive to minimum temperatures, with an incremental increase in mortality of 2.7% per degree fall in minimum temperature.

On the contrary, the elucidation of the impact of maximum temperature is less straightforward, and even at the 99th percentile of maximum temperatures, mortality rates are less than the mean. However, mortality rates were statistically significantly higher on the hottest day. The threshold for maximum temperature, above which total mortality increased was identified at 26.5^{0}C. Above these higher temperatures, total mortality increases by 3% per degree rise, in comparison to the decrease in mortality of 1.6% as minimum temperatures fall. The 3% increase in mortality noted at higher temperatures is comparable to previous work in Australia, where total mortality increases by 3% per degree at maximum temperatures over 28^{0}C (Mc Michael *et al*, 2002). Due to an insufficient number of days with high temperatures, it was not possible to quantify the incremental increase in mortality for age categories or other disease categories, aside from respiratory mortality. Incremental increases of 1.5% in respiratory mortality are evident per degree rise in maximum temperatures above the threshold, in contrast to the 2.7% increase as minimum temperatures decline. The sensitivity of respiratory mortality to extremes of high temperatures has been seen previously. In New Zealand, while total mortality is seen to rise by 1% for every 1^{0}C

rise above maximum temperatures of 20.5^{0}C, respiratory mortality increases by 3% (Hales *et al,* 2000).

4.6.3 Impact of hottest days

The impact of maximum temperatures in this study appears to be strongest on the day of maximum temperature (Figure 4.9), and this confirms the decision to use unlagged data. This is consistent with the literature (Hajat *et al*, 2002; Donaldson *et al,* 2001(b); Goodman *et al,* 2004; Kovats *et al*, 2004(a); Carder *et al,* 2005), although a 1 day (Sartor *et al,* 1995; Ramlow and Kuller, 1990), and a 2 day lag (Whitman *et al,* 1997) have also been reported.

However, the temporal pattern of mortality following a hot day varies in accordance with the different time intervals during which mortality is assessed. Statistically significant increases in mortality occurred on the hottest days in the under 14 and over 85 age groups and in total mortality (Table 4.11 and 4.12), but when the data was reassessed to include mortality rates in the one and two weeks following the hottest days, no statistically significant change in mortality rates was observed (Tables 4.13 and 4.14). This indicates that there is some evidence that the phenomenon of 'harvesting' did not occur in the mortality patterns observed following the hottest days in the under 14, and age 85 and over age –groups, and that the statistically significant increases in mortality that occurred on the hottest days possibly represented direct heat-related mortality. Nevertheless, mortality rates at the 99th percentile of maximum temperatures in the present study were still less than the mean mortality rate in the time series and again confirms the dominant role that minimum temperatures play in the determination of temperature related mortality at present.

4.7 Conclusion

In conclusion, almost two-thirds of mortality in Ireland is sensitive to temperature, in particular mortality from respiratory and cardiovascular diseases. This is consistent with the literature, and demonstrates that warmer mean temperatures are, in general, beneficial to health. In relation to extremes of temperature, the impact of minimum temperatures appears to dominate mortality rates. Incremental increases in mortality are clearly evident as minimum temperatures fall, for all age groups over 15 and for both

respiratory and cardiovascular diseases. The impact of maximum temperatures is immediate. The threshold for adverse impacts on total mortality appears to be 26.5^{0}C and respiratory disease may be the most sensitive disease to the impact rising maximum temperatures. The findings of the present chapter on the current impact of temperature on health are in accordance with the literature and this gives confidence to the validity of future estimates which are based on these findings. The ranges of temperatures that may be expected in Ireland have been outlined in Chapter 2 and it is apparent that the population of Ireland will be subjected to higher temperatures than have been experienced in recent times. The extent to which this may impact on mortality rates is the subject of the following chapter.

CHAPTER 5 FUTURE ESTIMATES OF TEMPERATURE RELATED MORTALITY

"Over the next half century, significant climate change can be expected in Ireland"

(Sweeney *et al,* 2002)

5.1 Introduction

The impacts of future increases in temperature on mortality have been assessed in a number of countries world-wide (Table 2.2). The majority of these assessments have been qualitative, and have estimated that while future climate related impacts would result in an overall reduction in cold-related deaths, higher mortalities could also be expected from higher maximum temperatures, for example, in Canada (Lemmen and Warren, 2004), in Finland (Hassi and Rytkonen, 2005), in the Netherlands (Bresser *et al,* 2005) and in Germany (Zebisch *et al,* 2005). However, very few national assessments to date have quantified the impacts of future increases in temperature on mortality, and none have quantified the changes in incidence that may be expected from specific diseases. Three national assessments have been identified that have undertaken a quantitative analysis on total future mortality rates. The national assessment undertaken in the U.K estimated that there would be 20,000 less cold-related deaths occurring annually resulting from a rise in mean temperatures (Donaldson *et al,* 2001(b)). On the contrary, a 5-41% increase in mortality in temperate Australian cities resulting from the impacts of a rise in extreme temperatures on total mortality was estimated in the national assessment (Mc Michael *et al,* 2002), and in Portugal, again using an temperature-mortality model, increases in annual heat-related mortality from 0.2 to 15 deaths per 100,000 by 2020, and increasing to between 2.1 to 29.6 by the 2050s were estimated, although changes in cold related mortality were not assessed (Dessai, 2002). Although no national assessment identified, quantified the impacts of a rise in mean temperatures on the incidence of specific diseases, one study was found, which noted that a reduction in the incidence of respiratory and cardiovascular diseases could be expected with climate change (Langford and Bentham, 1995).

This chapter will build on the temperature-mortality relationship established in Chapter

4, in order to investigate the impacts of future changes in mean and extremes of temperatures on both total mortality and mortality from specific causes that may be expected to occur in Ireland in coming decades.

5.2 Methodological approach

5.2.1 Mean temperatures

Two approaches have been used to quantify the impact of future mean temperatures on mortality rates. One approach identifies the temperature range in which mortality is lowest, with future changes in mortality ascertained by the relative changes in the number of days with mean temperatures above and below this range (Donaldson *et al,* 2001(b)). A second method has also been used, and is a development on the first. In this method, a temperature-mortality relationship is modelled and the mortality values extrapolated by regression when mortality rates at specified temperatures are not available (Langford and Bentham, 1995; Dessai, 2002). This is the method that will be used in this study.

Daily temperature outputs were obtained from three global climate models, each driven by scenarios A2 and B2 (as discussed in Section 3.5.1). To estimate the impact of future changes in temperature on mortality, the number of days with mean temperatures in each one degree band of mean temperature in the baseline period (1960 to 1990) and in each of the three future time periods (namely 2010-39, 2040-69 and 2070-2099), was quantified. As some of the temperatures that will be experienced in the future time periods are higher than have been previously experienced, a quadratic curve was fitted to the temperature-mortality relationship already established and extrapolated to obtain mortality rates at these higher temperatures. Future estimates of mortality were obtained by multiplying the number of days in each temperature band in future scenarios by the corresponding mortality rate. There are errors associated with the use of models, and it is therefore preferable to subtract the output of the baseline model from the output of the future model, in preference to using actual data for the baseline period. In this way, the impact of the errors is minimized as they are effectively subtracted from each other. When the mortality data obtained from the extrapolated equation using temperature data from the baseline model is subtracted from the mortality data obtained when temperature outputs from future scenarios are used in the equation, the difference in

mortality is ascribed to the impacts of climate change. This therefore resulted in six estimates of future mortality rates. The findings reported will be presented as the mean and a range of the outputs. The complete results for the impacts of future mean temperatures on mortality are detailed in Appendix E. The change that may be expected in future mortality rates are expressed with reference to the baseline model for the years 1961-1990. Results were obtained for the three future time periods, 2010 to 2039, 2040 to 2069 and 2070 to 2099. These changes in mortality are presented as a change in mortality per 100,000 population. The change in mortality rate was also calculated, by dividing the yearly change in the mortality rate by the mean yearly mortality rate in the adjusted mortality in the time series 1981-2002. In this way, future estimates were made of changes in total mortality in relation to age and total mortality from cardiovascular and respiratory diseases.

5.2.2 Extremes of temperatures

The impact of maximum and minimum temperatures on mortality was calculated in a similar manner to that of mean temperatures. The complete results for the impacts of future extremes of temperatures on mortality are detailed in Appendix F. In addition, the number of days with maximum temperatures above the threshold above which mortality rates are seen to rise was determined (Guest et *al,* 1999; Dessai, 2002; Mc Michael *et al,* 2002).

5.3 Results

5.3.1 Impact of increases in mean temperatures

The output of the temperature-mortality relationship established (Figure 4.7) was extrapolated and the mortality rate in each one degree band of mean temperature was established. The number of days with temperatures in each one degree band was estimated for the baseline and future scenarios (Tables 5.1 to 5.5).

Temp ^{0}C	Mort. rate	Temp ^{0}C	Mort. rate	Temp ^{0}C	Mort. rate	Temp ^{0}C	Mort. rate
-13 to <-12	4.06	0 to<1	3.00	13 to<14	2.37	26 to <27	2.49
-12 to <-11	4.04	1 to <2	2.93	14 to <15	2.35	27 to<28	2.52
-11 to <-10	3.93	2 to<3	2.86	15 to<16	2.34	28 to <29	2.56
-10 to <-9	3.82	3 to <4	2.80	16 to <17	2.34	29 to<30	2.61
-9 to <-8	3.71	4 to<5	2.74	17 to<18	2.33	30 to <31	2.66
-8 to <-7	3.61	5 to <6	2.68	18 to<19	2.33	31 to<32	2.71
-7 to <-6	3.51	6 to<7	2.63	19 to <20	2.34	32 to <33	2.77
-6 to <-5	3.41	7 to <8	2.59	20 to<21	2.35	33 to<34	2.83
-5 to <-4	3.31	8 to<9	2.50	21 to <22	2.36	34 to <35	2.89
-4 to <-3	3.24	9 to<10	2.47	22 to<23	2.38	35 to<36	2.99
-3 to <-2	3.15	10 to<11	2.44	23 to <24	2.40	36 to<37	3.04
-2 to <-1	3.07	11 to <12	2.41	24 to<25	2.42		
-1 to <0	3.07	12 to<13	2.39	25 to<26	2.45		

Table 5.1 Total mortality rates associated with future mean temperatures

Temp ^{0}C	Number of days	Temp ^{0}C	Number of days	Temp ^{0}C	Number of days	Temp ^{0}C	Number of days
-12.5	0	0.5	173	13.5	742	26.5	0
-11.5	1	1.5	268	14.5	660	27.5	0
-10.5	0	2.5	361	15.5	584	28.5	0
-9.5	1	3.5	480	16.5	408	29.5	0
-8.5	1	4.5	579	17.5	256	30.5	0
-7.5	1	5.5	669	18.5	140	31.5	0
-6.5	2	6.5	749	19.5	74	32.5	0
-5.5	5	7.5	762	20.5	37	33.5	0
-4.5	8	8.5	753	21.5	20	34.5	0
-3.5	20	9.5	738	22.5	11	35.5	0
-2.5	36	10.5	703	23.5	4	36.5	0
-1.5	70	11.5	732	24.5	2		
-0.5	113	12.5	738	25.5	1		

Table 5.2 Frequency of days with mean temperatures per 1 degree band of mean temperature in the baseline period 1961-1990

Temp ^{0}C	Number of days	Temp ^{0}C	Number of days	Temp ^{0}C	Number of days	Temp ^{0}C	Number of days
-12.5	0	0.5	123	13.5	684	26.5	0
-11.5	0	1.5	183	14.5	713	27.5	0
-10.5	0	2.5	251	15.5	688	28.5	0
-9.5	1	3.5	337	16.5	591	29.5	0
-8.5	1	4.5	429	17.5	428	30.5	0
-7.5	0	5.5	538	18.5	281	31.5	0
-6.5	0	6.5	678	19.5	171	32.5	0
-5.5	3	7.5	789	20.5	71	33.5	0
-4.5	3	8.5	821	21.5	29	34.5	0
-3.5	8	9.5	785	22.5	9	35.5	0
-2.5	17	10.5	763	23.5	4	36.5	0
-1.5	38	11.5	692	24.5	2		
-0.5	77	12.5	690	25.5	1		

Table 5.3 Frequency of days with mean temperatures per 1 degree band of mean temperature in the time period 2010-39

Temp ^{0}C	Number of days	Temp ^{0}C	Number of days	Temp ^{0}C	Number of days	Temp ^{0}C	Number of days
-12.5	0	0.5	141	13.5	678	26.5	3
-11.5	0	1.5	202	14.5	666	27.5	2
-10.5	0	2.5	268	15.5	643	28.5	1
-9.5	0	3.5	352	16.5	643	29.5	1
-8.5	0	4.5	462	17.5	543	30.5	0
-7.5	0	5.5	588	18.5	414	31.5	0
-6.5	1	6.5	709	19.5	256	32.5	
-5.5	1	7.5	789	20.5	164	33.5	
-4.5	3	8.5	834	21.5	81	34.5	
-3.5	8	9.5	792	22.5	30	35.5	
-2.5	16	10.5	740	23.5	15	36.5	
-1.5	43	11.5	682	24.5	9		
-0.5	82	12.5	679	25.5	6		

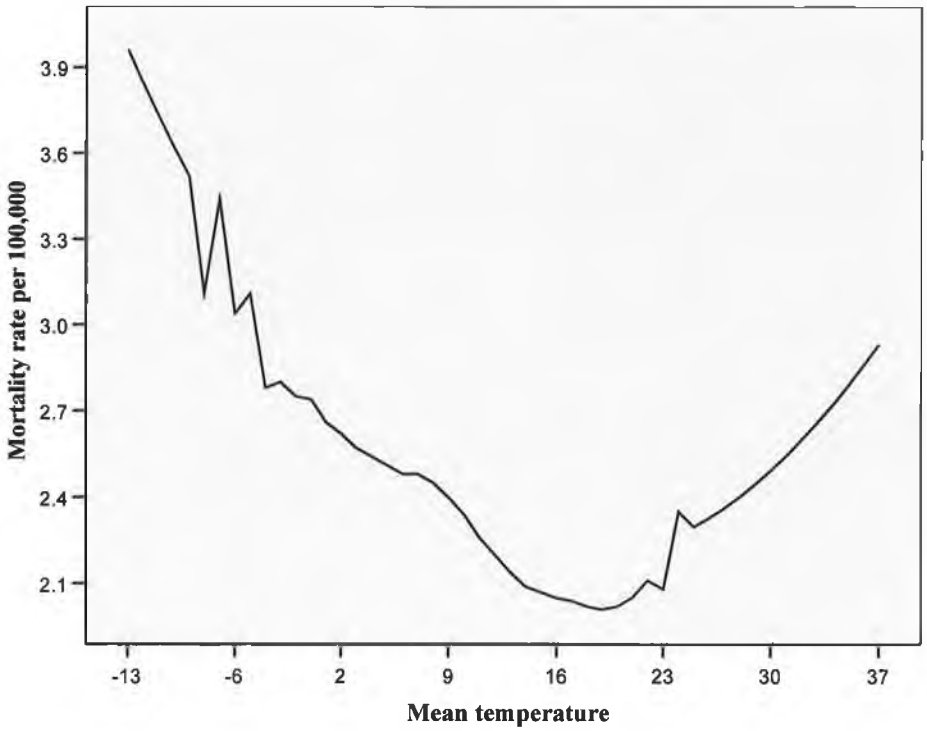
Table 5.4 Frequency of days with mean temperatures per 1 degree band of mean temperature in the time period 2040-69

Temp ^0C	Number of days	Temp ^0C	Number of days	Temp ^0C	Number of days	Temp ^0C	Number of days
-12.5	0	0.5	41	13.5	669	26.5	4
-11.5	0	1.5	86	14.5	691	27.5	3
-10.5	0	2.5	139	15.5	660	28.5	2
-9.5	0	3.5	212	16.5	643	29.5	1
-8.5	0	4.5	275	17.5	606	30.5	1
-7.5	0	5.5	387	18.5	515	31.5	0
-6.5	0	6.5	508	19.5	389	32.5	
-5.5	0	7.5	652	20.5	252	33.5	
-4.5	1	8.5	730	21.5	146	34.5	
-3.5	2	9.5	825	22.5	75	35.5	
-2.5	5	10.5	802	23.5	35	36.5	
-1.5	10	11.5	774	24.5	23		
-0.5	21	12.5	703	25.5	13		

Table 5.5 Frequency of days with mean temperatures per 1 degree band of mean temperature in the time period 2070-99

The changes in mortality will be presented in three ways. Firstly, the extrapolated temperature-mortality relationship will be presented in graph form. The equation of the relationship, and the associated R square and p values will be shown under the graph. Secondly, as the relative changes in future cold and heat related mortality rates resulting from changes in mean temperature are more readily seen in a bar chart, the changes in mortality will be presented in this format. The reference temperature used for these bar charts is the three degree band of mean temperature already identified where mortality is lowest, the minimum mortality band, or MMB, and this temperature range will be shown under the chart. Finally, the more precise, quantitative estimations of future changes in mortality are presented in a table format.

The quadratic relationship representing the mean temperature-total mortality relationship was found to be expressed by the equation $2.786-0.069(t)+0.002(t)^2$ and is depicted (Figure 5.1(a)).



R square = 0.928 p<0.001

Figure 5.1 (a) Extrapolation of mean temperature - total mortality relationship per 100,000

The range of mean temperatures between 17^0C and 20^0 where total mortality is lowest is apparent. Using this as a reference point, the changes that may be expected resulting from mean temperatures that may be expected in the future are depicted (Figure 5.1(b)).

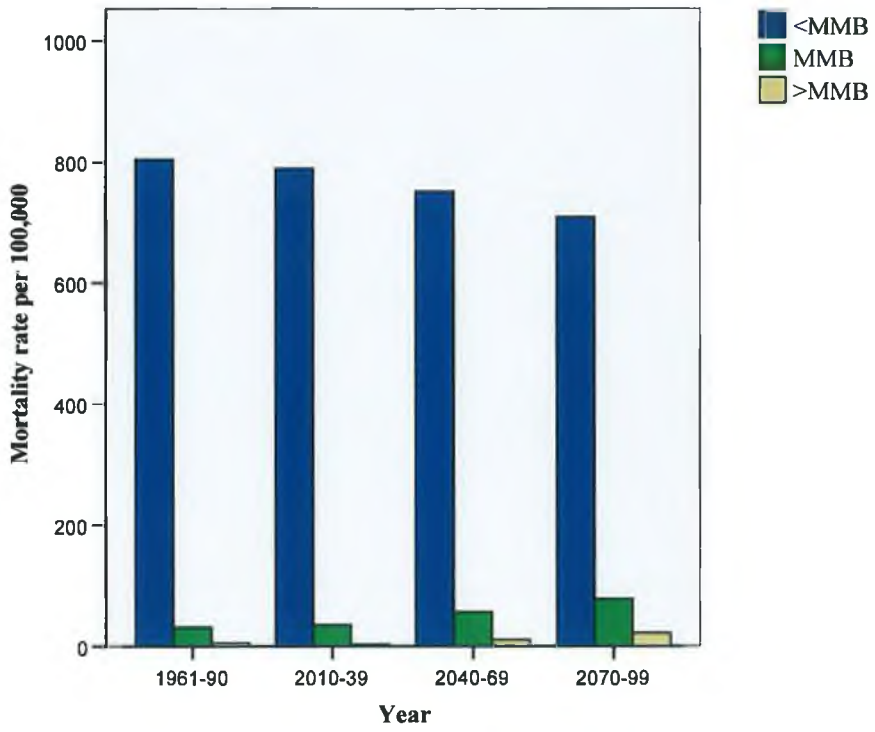


Figure 5.1(b) Impact of change in mean temperature on total mortality per 100,000 per year *(MMB 17^{0}C -20^{0}C)*

Total mortality on days with mean temperatures less than the MMB is seen to fall slightly, through successive future time periods. Mortality on colder days remains substantially higher than mortality occurring on warmer days, which although increased, remains relatively small. These finds are tabulated and summarized (Table 5.6(a)).

Climate model and scenario	2010 – 2039	2040 - 2069	2070 - 2099
CCM A2	-15	-31	-37
CCM B2	-19	-27	-34
CSIRO A2	-16	-28	-42
CSIRO B2	-21	-30	-37
Had3CM A2	-4	-13	-26
Had3CM B2	-8	-12	-19
Mean Range	-14 (-4 to -21)	-24 (-12 to -31)	-33 (-19 to -42)

Table 5.6(a) Mean changes in total mortality per year from the mean mortality rate per 100,000 population, resulting from outputs from three climate models using A2 and B2 scenarios and mean temperatures through three future time periods

Dividing the mean changes in mortality per 100,000 by the mean total mortality rate of 2.51 per 100,000 in the time series (Table 4.11), reductions in mortality of 1.5%, 2.6% and 3.5% in total mortality are found for the time periods 2010-2039, 2040 to 2069 and 2070 to 2099 respectively (5.6(b)).

Climate model and scenario	2010 – 2039	2040 - 2069	2070 - 2099
CCM A2	-1.6	-3.4	-4.0
CCM B2	-2.1	-2.9	-3.7
CSIRO A2	-1.7	-3.1	-4.6
CSIRO B2	-2.3	-3.3	-4.0
Had3CM A2	-0.4	-1.4	-2.8
Had3CM B2	-0.9	-1.3	-2.1
Mean Range	-1.5 (-0.4 to -2.3)	-2.6 (-1.3 to -3.4)	-3.5 (-2.1 to -4.6)

Table 5.6(b) Mean percent changes in total mortality per year from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios and mean temperatures through three future time periods

In a similar manner, the percentage changes in mortality rates from the mean mortality rates in the adjusted time series (detailed in Tables 4.9 and 4.10) were estimated for mortality in the age-specific groups over 15 and from mortality in disease categories. These results are detailed in Appendix E and summarized in Table 5.7 and Table 5.8.

Age group	2010-39 % change	2040-69 % change	2070-99 % change
15-64	-1.0 (-0.3 to -1.3)	-1.7 (-1.0 to -2.3)	-2.2 (-1.3 to -2.6)
65-74	-1.5 (-0.4 to -2.3)	-2.6 (-1.3 to -3.4)	-3.6 (-2.2 to -4.5)
75-84	-1.8 (-0.5 to -2.8)	-3.0 (-1.5 to -4.0)	-4.2 (-5.4 to -11.7)
85 and over	-2.0 (-0.5 to -3.1)	-3.5 (-1.8 to -4.7)	-4.9 (-2.5 to -5.4)

Table 5.7 Mean and range of percent changes in age-specific mortality per year from the mean mortality rate in the adjusted time series resulting from changes in mean temperatures

Reductions in mortality in the 15-64 year age group of 1%, increase to 2% in the age 85 and over year age group, with reductions of 1.5% and 1.8% in the 65-74 and 75-84 age groups respectively in the coming decades are estimated.

Disease category	2010-39 % change	2040-69 % change	2070-99 % change
Cardiovascular	-1.5 (-0.4 to -2.2)	-2.6 (-1.2 to -3.5)	-3.7 (-2.0 to -4.7)
Ischaemic heart disease	-1.4 (-0.4 to -2.3)	-2.5 (-1.1 to -3.4)	-3.4 (-1.9 to -4.6)
Stroke	-1.5 (0 to -2.7)	-2.6 (-0.9 to -3.7)	-3.3 (-1.8 to -4.6)
Respiratory	-3.5 (-0.8 to -4.8)	-6.0 (-3.2 to -8.1)	-8.5 (-4.8 to -10.5)

Table 5.8 Mean and range of percent changes in mortality from disease categories per year from the mean mortality rate in the adjusted time series resulting from changes in mean temperatures

Similarly, mean reductions from the mean mortality rates in the adjusted time series (outlined in Table 4.11), of 1.5% in cardiovascular diseases and 3.5% from respiratory disease in this time period 2010-2039 are also estimated. To estimate the impact of these changes, using the reductions in mortality per 100,000 population, on data from the most recent census (2006), reductions in future mortality by mid-century were obtained.

Age category	Population	2040-2069
15-64	2,909,037	-145
65-74	262548	-257
75-84	157350	-397
85 and over	48028	-371
Total	3463412	-831

Table 5.9 Estimated annual change in total age-specific mortality by mid-century by age based on 2006 population figures resulting from changes in mean temperatures

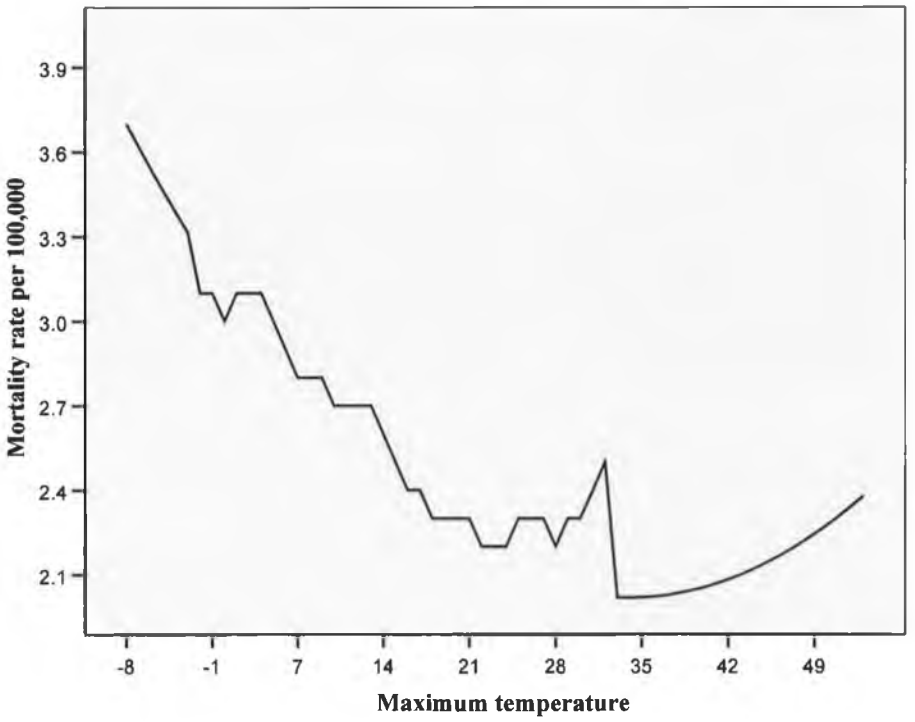
An annual reduction in mortality of approximately 800 by the middle of the century may be expected (Table 5.9).

5.3.2 Impact of increases in maximum and minimum temperatures

Maximum temperatures

In a similar manner to the method used for mean temperatures, the impacts of a rise in daily minimum and maximum temperatures were also estimated. The quadratic relationship that represents the maximum temperature-total mortality relationship is

expressed by the equation $3.141-0.067(t)+0.001(t)^2$. This relationship was extrapolated and is depicted in Figure 5.2(a).



R square = 0.933 p<0.001

Figure 5.2 (a) Extrapolation of maximum temperature- total mortality relationship

A reduction in future mortality rates is evident, with mean reductions in total mortality ranging from 9 per 100,000 in the coming decades, possibly up to 40 per 100,000 the end of the century (Table 5.10(a)).

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1	-18	-24
CCM B2	-4	-11	-19
CSIRO A2	-16	-29	-56
CSIRO B2	-21	-32	-63
Had3CM A2	-4	-14	-30
Had3CM B2	-8	-13	-45
Mean	-9	-20	-40
Range	(-1 to -21)	(-32 to -11)	(-30 to -63)

Table 5.10 (a) Mean changes in total mortality per 100,000 resulting from changes in maximum temperatures using outputs from three climate models utilizing A2 and B2 scenarios and maximum temperatures through three time periods

Dividing the mean changes in mortality per 100,000 by the mean total mortality rate of 2.51 per 100,000 in the time series, reductions in mortality of 1.0%, 2.1% and 4.3% are

found for the time periods 2010-2039, 2040 to 2069 and 2070 to 2099 respectively (Table 5.10(b)).

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-0.1	-1.9	-2.6
CCM B2	-0.5	-1.2	-2.1
CSIRO A2	-1.8	-3.1	-6.1
CSIRO B2	-2.3	-3.5	-6.9
Had3CM A2	-0.4	-1.6	-3.3
Had3CM B2	-0.9	-1.5	-4.9
Mean Range	-1.0 (-0.4 to -2.3)	-2.1 (-1.2 to -3.5)	-4.3 (-2.1 to -6.9)

Table 5.10 (b) Mean percent changes in total mortality per year from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios and maximum temperatures through three future time periods

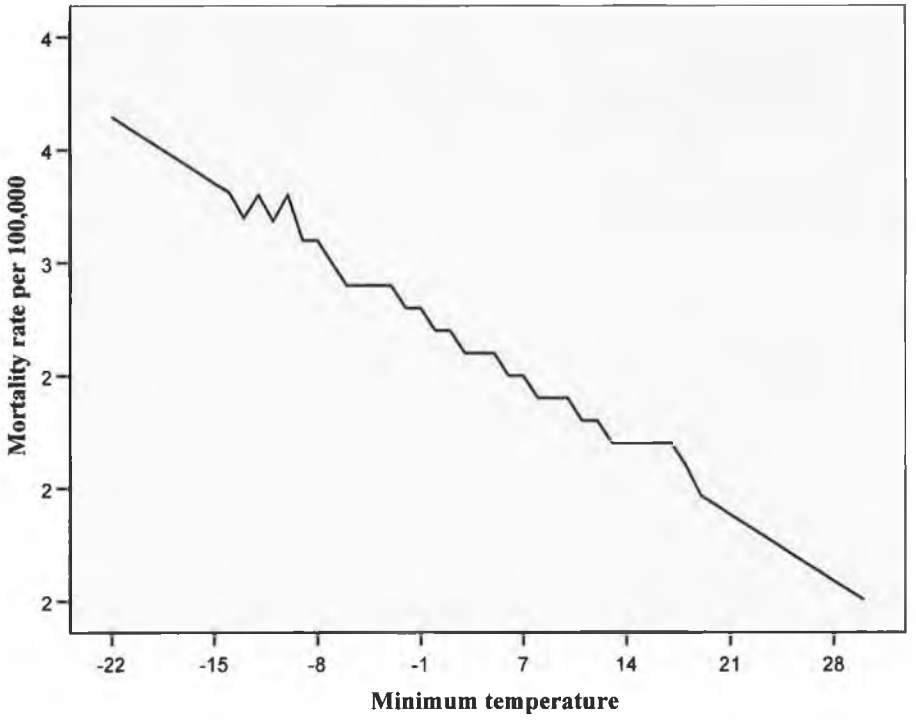
Although reductions are evident in total mortality, resulting from the rise in maximum temperatures, the number of days when maximum temperatures exceed the threshold will also increase (Table 5.10 (c)).

Threshold Temperature 26.5^0C	2010 - 2039	2040 - 2069	2070 - 2099
Mean Range	0 (0 to 1)	3 (1 to 6)	8 (0 to 16)

Table 5.10 (c) Mean and range in the number of days per year when the number of days when maximum temperatures exceed the threshold for total mortality

Minimum temperatures

Similarly, the impact of a rise in daily minimum temperatures was also estimated. The relationship that represented the minimum temperature-total mortality relationship could be expressed by the linear equation 2.760-0.041(t), R square 0.980 or by the quadratic expression $2.7496-0.00422(t)+0.00021(t)^2$, R square 0.978; the quadratic expression is depicted (Figure 5.2(b)).



R square quadratic = 0.978 p<0.001

Figure 5.2 (b) Extrapolation of lagged minimum temperature- total mortality relationship

Again, reductions in future mortality rates are evident, both in rates per 100,000 and reductions from the mean mortality rate in the adjusted time-series (Table 5.11(a) and Table 5.11(b) respectively)).

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-16	-36	-47
CCM B2	-10	-20	-28
CSIRO A2	-2	-14	-44
CSIRO B2	-21	-16	-23
Had3CM A2	-3	-14	-31
Had3CM B2	+7	+2	-5
Mean Range	-7 (+7 to -21)	-16 (+2 to -36)	-30 (-5 to -47)

Table 5.11 (a) Mean changes in total mortality per 100,000 resulting from changes in minimum temperatures using outputs from three climate models utilizing A2 and B2 scenarios through three time periods

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1.7	-3.9	-5.0
CCM B2	-1.1	-2.2	-3.1
CSIRO A2	-0.2	-1.5	-4.7
CSIRO B2	-2.3	-1.7	-2.5
Had3CM A2	-0.3	-1.5	-3.3
Had3CM B2	+0.7	+0.2	-0.6
Mean Range	-0.8 (+0.7 to –1.7)	-1.8 (+0.2 to -3.9)	-3.2 (-0.6 to -5.0)

Table 5.11 (b) Mean percent changes in total mortality per year from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios and minimum temperatures through three future time periods

In a similar manner, the changes in mortality were estimated for mortality in the specified age-specific groups from mortality in disease categories. A summary of the percentage change in mortality rates is presented in age groups (Tables 5.12) and disease categories (Tables 5.13), aside from cancer as it was not possible to elucidate a relationship with extremes of temperature from this disease. The changes per 100,000 population are detailed in Appendix F.

Age category	2010-39 Max temp	2010-39 Min temp	2040-69 Max temp	2040-69 Min temp	2070-99 Max temp	2070-99 Min temp
14 and under	-1.5 (0 to -4.5)	-2.3 (0 to -4.5)	-6.1 (-4.5 to -9.1)	-4.5 (0 to -9.1)	-9.8 (-4.5 to -18.2)	-6.1 (0 to -9.1)
15-64	-2.0 (0 to -4.8)	-1.2 (0 to -3.6)	-3.8 (-2.4 to -6.0)	-2.8 (0 to -6.0)	-7.3 (-3.6 to -11.9)	-5.4 (-1.2 to -8.3)
65-74	-3.2 (-1.3 to -7.5)	-2.7 (+1.8 to -5.3)	-6.5 (-3.6 to 11.1)	-5.6 (+0.7 to -11.9)	-12.9 (-6.4 to -21.0)	-10.0 (+2.4 to -15.3)
75-84	-4.5 (-2.0 to -10.2)	-4.1 (+2.8 to 10.4)	-9.4 (-5.3 to15.4)	-6.9 (+2.2 to -13.6)	-18.9 (-9.2 to -30.5)	-11.1 (-2.6 to -15.5)
85 and over	-5.1 (-0.5 to -11.8)	-4.1 (+3.3 to10.7)	-10.7 (-5.8 to17.5)	-8.5 (+1.1 to -18.7)	-21.5 (-10.3 to34.5)	-15.5 (2.8 to -23.5)

Table 5.12 Mean percent changes in total mortality per year from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios and both maximum and minimum temperatures through three future time periods

Disease category	2010-39 Max temp	2010-39 Min temp	2040-69 Max temp	2040-69 Min temp	2070-99 Max temp	2070-99 Min temp
CV	-3.7 (-7.5 to -25.4)	-2.9 +2.2 to -6.0)	-7.7 (-4.5 to -12.7)	-6.0 (+0.7 to -12.7)	-15.3 (-0.7 to -9.0)	-10.3 (-2.2 to -15.7)
IHD	-3.5 (0 to -8.3)	-3.0 (+1.4 to -6.9)	-7.6 (-4.2 to -12.5)	-5.1 (+1.4 to -11.1)	-17.1 (-11.1 to -25.0)	-9.5 (-1.4 to -13.9)
Resp	-7.4 (0 to -17.6)	-7.4 (+5.9 to -20.6)	-17.2 (-8.8 to -29.4)	-14.2 (+2.9 to -32.4)	-36.3 (-17.6 to -58.8)	-27.5 (-5.9 to -41.2)
Stroke	-3.9 (0 to -10.1)	-2.7 (0 to -6.7)	-7.8 (-3.3 to -13.3)	-6.7 (0 to -13.3)	-15.6 (-6.7 to -23.3)	-12.2 (-3.3 to -16.7)

Table 5.13 Mean percent changes in total mortality per year from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios and both maximum and minimum temperatures through three future time periods

The number of days per year when maximum temperatures may exceed the threshold temperatures for age groups and disease categories was quantified (Tables 5.14 and 5.15 respectively).

Age category	Range and midpoint of threshold temperature	2010-39	2040-69	2070-99
15-64	20-23^{0}C 21.5^{0}C	15 (7 to 21)	31 (19 to 46)	48 (31 to 61)
65-74	23-26^{0}C 24.6^{0}C	3 (2 to 4)	10 (9 to 18)	19 (7 to 33)
75-84	21-24^{0}C 22.5^{0}C	10 (5-16)	23 (15 to 38)	38 (25 to 54)
85 and over	26-29^{0}C 27.5^{0}C	0 (0 to 0)	2 (0 to 3)	3 (0 to 11)
Total	25-28^{0}C 26.5^{0}C	0 (0 to 1)	3 (1 to 6)	8 (0 to 16)

Table 5.14 Mean and range of number of days annually when maximum temperatures exceed threshold temperature in relation to age groups

Related to the threshold identified, a substantial increase in the number of days when the maximum temperatures will exceed the threshold temperatures for the 15-64 age-group, and to a lesser extent, the older age groups is evident.

Disease category	Range and midpoint of threshold temperature	2010-39	2040-69	2070-99
CV	25-28^{0}C 26.5^{0}C	0 (0 to 1)	3 (1 to 6)	8 (0 to 16)
IHD	25-28^{0}C 26.5^{0}C	0 (0 to 1)	3 (1 to 6)	8 (0 to 16)
Stroke	27-30^{0}C 28.5^{0}C	0 (0 to 0)	2 (0 to 3)	3 (0 to 11)
Respiratory	20-23^{0}C 21.5^{0}C	15 (7 to 21)	31 (19 to 46)	48 (31 to 61)

Table 5.15 Mean and range of the number of days annually when maximum temperatures exceed threshold temperature in relation to disease category

There is an increase in the number of days when the threshold at which mortality from respiratory diseases rises, and beginning in the coming decades, and increasing as time progresses. Later in the century, an increase in the number of days when the maximum temperature exceeds the threshold for an increase in mortality from cardiovascular disease is evident.

5.4 Discussion

5.4.1 Increases in mean temperatures

The impact of a rise in daily mean temperatures on Irish mortality rates up to 2099 has been quantitatively assessed. This is the first time such work has been undertaken in Ireland, and it is also the first time that a quantitative assessment have been made in relation to future mortality rates from specific diseases in the context of a national assessment.

The nature of the extrapolated curve of the relationship between total future mortality and mean temperature is as expected, with the U-shaped curve evident, and higher mortality rates occurring on the colder days (Figure 5.1(a)). As a result, the beneficial impacts of warmer temperatures on total mortality are therefore seen to increase progressively throughout the century. Substantial reductions in total mortality are noted, from a mean of 1.5% (14 per 100,000) early in the century rising to 3.5% (33 per 100,000) towards the end of the time period (Tables 5.6 (a) and 5.6 (b)). The lowest reduction in mortality resulting from rising mean temperatures was seen in one model, namely the Hadley3 A2 model, and the largest were seen with the outputs from the CSIRO B2 model. This reduction in mortality rates is evident in all age-groups over 15, and is seen in all scenarios. Mortality in all age groups may therefore be expected to progressively decline as time progresses and future mean temperatures increase. The vulnerability of the older population to cold temperatures has been established, with the incremental changes in mortality per degree fall in temperature increasing as mean temperatures fall (Table 4.4). This sensitivity to cold temperatures is evident in the reductions in mortality that may be expected in the future, which increases with advancing age, from a mean of 3 per 100,000 in the 15-64 year age group, progressively increasing to a mean of 499 per 100,000 in the age 85 and over age group in the time period 2010 to 2039 (Tables 1 and 4, Appendix E). Although the incremental reductions in mortality in the 15-64 age-specific population are low, this is significant, as 84% of the population in 2006 was in this age-group (Table 5.9).

The shape of the temperature – mortality relationship for cardiovascular disease is, as might be expected, similar to the relationship for total mortality, and mortality from this disease shows the greatest reductions in mortality per 100,000 population. Substantial reductions in mortality from this disease may be expected to continue to the end of the century, with reductions in mortality in the region of 7 per 100,000 (1.5%), early in this century, rising to 17 per 100,000 (3.7%) by 2099 (Table 5.8). Proportional reductions in mortality are also evident in both ischaemic heart disease and stroke. The reduction in mortality from cardiovascular disease is significant, as although mortality has been declining from this disease (Figure 4.4(g)), it remains the leading cause of mortality in Ireland. The reductions in respiratory disease, of 4 per 100,000 early in the century and rising to 11 per 100,000, while comparable to the reductions in ischaemic heart disease are also significant as mortality from respiratory disease is less common than mortality from ischaemic heart disease (Table 4.1), and therefore the proportional reductions in mortality are larger. Furthermore, in contrast, no reduction in respiratory mortality has been observed to date (Figure 4.4(k)), and mortality from respiratory disease in Ireland is the highest in Western Europe, with death rates over twice the EU average and recently noted to be greater than from ischemic heart disease (Brennan *et al,* 2008).

Therefore, arising from changes in mean temperatures resulting from climate change, in every age group over 15, progressive and substantial reductions in mortality in every age group may be expected, increasing as time progresses. The reduction in total mortality found in this study is consistent with previous qualitative assessments of the future impacts of changes in mean temperatures in other European countries, (Hassi and Rytkonen, 2005; Bresser *et al,* 2005; Zebisch *et al*, 2005) and in Canada (Lemmen and Warren, 2004), and the USA (Mc Geehin and Mirabelli, 2001). The quantitative results obtained are comparable with the only other quantitative national assessment identified, namely the national assessment undertaken in the U.K. (Table 5.16).

	2010-2039	2040-2069	2070-2099
Present study	-140 (-20 to -190)	-240 (-90 to -260)	-330 (-140 to -340)
UK data	-219	-152 to -364	-461

Table 5.16 Change in mortality per million[1] resulting from rises in mean temperatures: comparison of present results and those of the UK (From Donaldson *et al,* 2001(b))

Although the results of the U.K. assessment (Donaldson *et al,* 2001(b)), were obtained by a different manner, as outlined (Section 5.2.1), it is reassuring to note that they are comparable with the results of the present study, although in the latter part of the century the mortality reductions are higher in the U.K. The reductions in total mortality of 0.4% to 2.3% estimated in this study for the time period 2010-39 are lower than the 8 to 12% reduction in total mortality, estimated by 2030 in earlier work in Australian cities (Guest *et al,* 1999). However the Australian study also included the substantial increase in population that may be expected, in addition to incorporating other climatic indices. Reductions in mortality specifically from respiratory and cardiovascular diseases in future time periods, found in the present study have also been reported previously in the UK (Langford and Bentham, 1995).

The overall beneficial impacts of warmer temperatures on overall mortality can be seen, and this is particularly relevant for Ireland. Ireland has a mortality rate in winter that is higher than almost all of its European counterparts; this has been largely attributed to poor insulation and fuel poverty (Clinch and Healy, 2000). However, during the latter half of the 20th century, winter mortality in many European countries has fallen; this has been attributed to central heating, warmer clothing and improved medical services (Lerchyl, 1998; Donaldson and Keating, 2002). This decline in mortality is continuing in the UK, and there is evidence that the reductions in winter mortality, and in particular from respiratory and cardiovascular diseases, are due to increased wealth (Carson *et al,* 2006), housing and also behavioural factors (Hajat *et al*, 2007). Nevertheless, no evidence was seen of a decline in winter mortality in a preliminary analysis of the data in the Irish time series investigated. This indicates that Ireland's total mortality rates may benefit disproportionately from future increases in mean temperatures. In addition, the

[1] The denominator of 100,000 used in the present study has been converted to per million to facilitate comparison

mean age of the Irish population is getting older. Future population trends have been examined, and forecasts estimate large increases in those aged over 65, rising to almost a quarter of the population being aged over 65 by 2050 (Table 5.17) (CSO, 2004; Eurostat, 2006). This increase in the older population may increase further in the future as a result of milder winters, and result in an increased need for residential care for an older population and increased bed capacity in acute hospitals.

Age Group	2006-2016	2021-2036	2050
14 and under	4%-26%	-9%-31%	16%
15-64	9%-23%	20%-40%	58%
Over 65	8%-47%	70%-166%	23%
Total	8%-26%	23%-51%	36%

(after CSO 2004 and Eurostat)

Table 5.17 Future estimated percentage changes in population

It is clear from experience in warmer European countries that as temperatures increase, people adapt to the warmer climate, and the temperature threshold above which mortality rises is higher in hotter countries (Table 4.16) (Keatinge *et al,* 2000). It might be expected therefore that this reduction in mortality will be counteracted to some extent by acclimatization, and the reductions in mortality estimated may be an over estimation. However, adaptation may be difficult to achieve if mean temperatures continue to rise. Nevertheless, due to the predominant impact of cold temperatures on mortality, it may be expected that Ireland's total mortality rates may improve as a result of changes in mean temperatures resulting from climate change.

5.4.2 Increases in extreme temperatures

Age category

As a result of increasing maximum temperatures, reductions in total mortality were noted, and these reductions in mortality increased as time progresses. The reduction in total mortality in the region of 1% early in the century, rises to 2% by mid-century and possibly to over 5% towards the end of the century (Table 5.10(b)). These reductions in mortality are seen in all age groups, and are most marked in the age 85 and over age-group, where possible reductions of over 300 per 100,000 on current mortality rates may occur in the coming decades (Table 5(a), Appendix F). However, associated with increasing maximum temperatures, there will be an increase in the number of days when the maximum temperatures will exceed the threshold, and this is marked in the 15-64

year age group (Tables 5.14 and 5.15). Apart from the recognized risk factors for heat related mortality of age and pre-existing disease, sensitivity to heat related mortality has been related to vigorous outdoor exercise in hot weather, particularly in younger age groups (Coris *et al,* 2004), and cognisance of this may be needed in health education programmes.

In relation to the impact of minimum temperatures, five of the six scenarios showed a reduction in mortality in all age categories. The reductions in mortality from the five scenarios are evident in all age-groups, and reductions in total mortality in the order of 0.8% early in the century will possibly rise to over 3% by the end of the century (Table 5.11 (b)). However, an increase in mortality was evident with one scenario, namely the Hadley3 B2 scenario, in the first two time periods. Interestingly, both Hadley scenarios record the lowest rises in mean annual and mean winter temperatures in all time periods (Tables 3.8 and 3.9 respectively). However minimum temperatures are generally expected to increase more in summer than in winter, and by the end of the century, increasing minimum temperatures will result in a reduction in mortality in all scenarios, for all age groups and disease categories.

Disease category

In relation to maximum temperatures, the outputs from all climate models indicate a consistent and substantial reduction in mortality from cardiovascular and respiratory diseases (Table 5.13), although there was a substantial number of days when the maximum temperature threshold above which mortality from respiratory disease rises was exceeded (Table 5.15). The relationship with minimum temperatures is more straightforward, and reflects the substantial incremental changes in mortality with decreasing minimum temperatures demonstrated (Table 4.9). Reductions in mortality from respiratory disease and cardiovascular diseases are evident in five of the six scenarios, increasing as time progresses. However, again, with the output from the Hadley3 B2 scenario, an increase in mortality is seen early in the century in mortality from all disease categories, reflecting the adverse impact of colder winter temperatures evident in this modelled output. However by the end of the century, all scenarios indicated a decrease in mortality resulting from rises in future minimum temperatures in each disease category.

In conclusion, arising from the impact of future changes in maximum temperatures, reductions in mortality in all age groups may be expected, increasing as time progresses. Mortality from respiratory disease, ischaemic heart disease and stroke will reduce and rising minimum temperatures will, aside from one scenario (Hadley3 B2), substantially contribute to reductions in mortality in all age and disease categories.

Four previous studies have been identified that investigated the impacts of future maximum temperatures on mortality. In an investigation of the impact of climate change in Australian cities, Guest (Guest *et al,* 1999) reported a decrease in total mortality, which was attributed to a greater reduction in cold mortality. However, more recent work on the future impacts of climate change in Australian cities, incorporated the synergistic impacts of pollution with maximum temperatures, and reported an overall increase in mortality from between 6% to 41% by 2020 and from between 149% to 160% by 2050 (Mc Michael *et al*, 2003). Increases in future mortality rates of up to 15 per 100,000 by 2020 and by 35 per 100,000 in Portugal by 2050 were also estimated, although this might be expected to reduce with acclimatization (Dessai, 2003), however this study did not investigate the impact of cold temperatures on mortality. Similarly, a more recent study reported on the US population, noted that that as this population is fully acclimatized to cold, as a result of central heating, that the rise in heat related mortality will not be outweighed by the decrease in cold - related mortality (Medina-Ramon and Schwartz, 2007). However, it cannot be said that the Irish population is acclimatized to cold temperatures in the light of the dominant impact that cold temperatures have on mortality rates at present.

Although this study indicates that a decrease in overall mortality rates may be expected resulting from increased maximum temperatures, it is clear that many factors modify the impact of maximum temperatures on mortality, including pollution and acclimatization. Nether of these issues were accounted for in the present study. Due to a lack of long term studies, it is uncertain how long acclimatization may take to occur (Dessai, 2003). While short-term acclimatization may take 3-12 days, it may take several years to become fully adapted (WHO, 2004(a)), and even up to three decades has been suggested (Desai, 2003). However adaptation may not be achieved if temperatures continue to rise.

Higher air temperatures may also compromise air quality. Air quality is related to the quantity and nature of pollutants emitted, and the nature of the meteorological variables that impact on pollutant dispersal. Ground level ozone is formed in warm weather, from a combination of nitrogen oxides and volatile organic compounds, and is a respiratory irritant. High temperatures are often associated with increased incidences of ground level ozone, and of particulate pollution, both of which may simultaneously adversely impact health. However, there is uncertainty in the determination of the causation of mortality from pollution and temperature, and it is therefore difficult to predict the impact of climate change on local air pollution concentrations (IPCC, 2001; Basu and Samet, 2002). While work in the US has indicated that temperature acts synergistically with ozone, the effect appears to be modified by local characteristics, such as the use of air-conditioning and exposure to traffic (Ren *et al,* 2007). In addition, the interaction between pollutants and temperature indicates that previous studies may have underestimated the impacts of air temperatures on mortality (Goodman *et al*, 2004). However, mortality is expected to increase with higher levels of ozone, the concentrations depending on meteorological conditions (AR4, 2007). The heat waves recently experienced in Europe have allowed more research on this issue, and a quantification of the relationship to be undertaken. Up to a third of the excess deaths in England and Wales during the first two weeks of August 2003 were associated with the elevated ambient ozone and particulate matter concentrations (Stedman, 2004), and from 28% to 60% of the excess mortality in the Netherlands during the same heat wave may have been caused by ozone and particulate matter (Fischer *et al,* 2004). When temperatures are very high, heat appears to be the primary cause of death, ozone contributing to increased mortality when temperatures are less extreme (Vandentorren and Empereur-Bissonnet, 2005). Short-term acute ozone pollution episodes are not a common feature of air quality in Ireland at present, although they may be in the future (EPA, 2006 (d)), as levels of both precursors of ozone, nitrogen oxides and volatile organic compounds may increase. Although emissions of nitrogen oxides are reducing, largely as a result of cleaner technology, these decreases are being offset by increases in the number of vehicles, and although the levels of nitrogen oxide are currently below the limits set by European Directives, they are substantially higher in urban areas. Secondly, emissions of volatile organic compounds, while falling, at present are higher then the European limit which will come into effect in 2010 (EPA 2006 (d)). Studies to assess the relationship between air pollution and health outcomes are not recommended

to be undertaken as part of the remit of assessments of the impacts of climate change (Kovats *et al*, 2003(b)).

Fewer numbers of days with very low temperatures may be also expected. Low temperatures are associated with poor dispersal of pollutants, when it becomes trapped below warmer air, resulting in a temperature inversion. Pollutants contained in the cold air become trapped and remain in the atmosphere until dispersed by wind. The adverse impact of pollutants and cold temperatures was clearly seen in Dublin, following a ban on coal sales, when a reduction in total mortality of 6%, a fall in respiratory mortality of 15.5% and a fall in cardiovascular mortality of 10.3% was evident when compared to mortality rates six years before the ban (Clancy *et al,* 2002). Levels of sulphur dioxide are also decreasing in Ireland, and measurements of both sulphur dioxide and particulate matter less than 10 microns in size are below European limits (EPA 2006 (d)). Consequently, fewer days when pollutants are not adequately dispersed may occur. Further work in Ireland is indicated to investigate the impacts of air-pollution on mortality on the days with the higher maximum and minimum temperatures that we will undoubtedly experience.

It is difficult to accurately estimate the impact of a future heat wave on Irish mortality rates. It is clear that the impact of the hottest day in the time series initially impacted on mortality (Figure 4.9). Total mortality, and specifically in the under 14 and age 85 and over age-group was seen to increase significantly, on this day (Table 4.12). There was however, evidence of a hastening of imminent mortality (harvesting effect) after the very hot days in the present study, and no statistically significant increase in mortality was evident after 2 weeks (Tables 4.13 and 4.14). The statistically significant increase in mortality in people aged 85 and over on the hottest day is important, and Table 5.14 indicates the substantial number of days when maximum temperatures may be expected to exceed this threshold temperature. Many reasons have been cited for the increase in vulnerability in older people, including the higher prevalence of respiratory and cardiovascular disease in these age groups; furthermore, many older people live on their own, some in poor social and economic circumstances (Semenza *et al,* 1996). The vulnerability of older people to a heat wave was seen in the tragic impact of the heat-wave in France in 2003, which resulted in approximately 15,000 excess deaths, an increase of 55% when compared to mortality data in the years 2000-2002 (Vandentorren and Empereur-Bissonnet, 2005). Mortality was highest in the older population, with

over a 70% increase recorded in the over 75 year age group (Belmin, 2003). Disturbingly, over 60% of people who died from heat stroke related illness in France in 2003 resided in a health care facility, almost half being retirement homes (Vandentorren and Empereur-Bissonnet, 2005). Mortality was rapid, the median length of stay between admission to hospital and death being one day (Vanhems and Gambotti, 2003). Similarly, the majority (97%), of the estimated excess of 2,100 deaths in Portugal were in the over 74 age group (Calado *et al,* 2005; Nogueria *et al,* 2005). In the London heat wave of 2003, mortality in the over 75 age group again sustained the highest increase in mortality rates, with an increase of 22% over the mean 1998-2002 level (Johnson *et al,* 2005 (b)), and over four fifths (85%) of the mortality was suffered by people in the over 75 year age group (Hajat *et al,* 2002; Kovats *et al,* 2004 (a); Johnson *et al,* 2005(a)).

The statistically significant increase in mortality in children on the hottest day is also important. Few studies have investigated the impact of extreme heat on children, and mortality in this age group is generally low in temperate countries. Nevertheless, 6% of mortality from heat-related events in the United States occurred in the under 15 age group and an excess of almost 5% in mortality occurred in this age group in the UK and Wales after the heat wave in 1995 (Rooney *et al,* 1998). However, no evidence of an increase in child mortality attributable to the heat wave experienced in European countries in 2003 was reported (Kosatsky, 2005; Garssen *et al,* 2005; Pirard *et al,* 2005; Kovats and Ebi, 2006), although more research on the impacts of heat on children has been requested (Ishigami *et al,* 2008). More work is indicated to investigate the impact of extremes of temperature on children in Ireland.

However, there are problems encountered in assessing mortality from heat waves (Section 4.3.4). The quantification of 'harvesting' is difficult (WHO (2004(a)) and there is no agreement in the literature on a standardized time interval for such an assessment of mortality. The absence of such a system makes it difficult to quantify the increase in mortality after hot periods (WHO 2004 (b)), although work is being undertaken on this issue (Robinson, 2001). Standardized methods of data collection through all countries would be necessary to allow detailed comparisons, as estimates of changes in mortality resulting from a high temperatures are sensitive to the method used (Ledrans *et al,* 2004). Descriptive studies (Rooney *et al,* 1998; Conti *et al,* 2005), case control (Semenza *et al,* 1996), analytical time series (Hajat *et al,* 2002) and centiles of

maximum temperature (Medina-Ramoz and Schwartz, 2007), have been used. However, little is known about the ratio of deaths which occur immediately after very hot days to the fall in mortality that may occur afterwards (Bresser *et al,* 2005). While there is some evidence that the majority of the mortality that occurs after a very hot period falls into this category (Braga *et al,* 2001), interestingly, there was no evidence of such an effect three weeks after the French heat-wave in 2003 (Vandentorren and Empereur-Bissonnet, 2005), and later work indicates that less than 10% of the mortality may have been hastened in the French heat-wave (Le Tertre *et al,* 2006). However it is difficult to extrapolate the impact of heat waves on the Irish population as even on the days with maximum temperatures at the 99th percentile, mortality rates were lower than the mean.

Undoubtedly, patterns of mortality are unique to each situation and vary according to many issues; for example the effect may be accentuated if there are previous episodes of very warm weather which may weaken the already vulnerable population (Huynen *et al,* 2001), and the implementation of heat wave plans may reduce the effect (Weisskopf *et al,* 2002). In addition, four particular variables are relevant in Ireland. Firstly, the number of older people is expected to increase (Table 5.17). Secondly, there is also a trend to city living in Ireland, and as buildings in cities tend to retain heat at night, people living in cities are disproportionately affected by excessive heat and are at particular risk because of the 'heat island' effect (Conti *et al,* 2005; Grize *et al,* 2005, for example). In addition, this phenomenon is accentuated when minimum temperatures are high; this gives rise to oppressive night time conditions, which afford little relief (Kalkstein, 1993). During the French heat-wave, the short hot nights did not allow the city to cool, and contributed to the mortality increases of 130% in the Paris suburbs (Grynszpan, 2003). This is significant, as minimum temperatures are rising more than maximum temperatures (Sweeney *et al,* 2002). Inhabitants of cities have suffered disproportionately from heat-waves in the past; while an increase in mortality of 17% occurred nationally in the U.K. heat wave of 2003, the increase rose to 42% in London (Johnson *et al,* 2005(b)). Similarly, during the heat wave of 2003 in Switzerland, higher excess mortality was observed in suburban (10.2%) and in urban areas (7.9%) than in rural areas (1%) (Grize *et al,* 2005). Thirdly, social conditions are important mediators of vulnerability to heat waves. The number of older people living alone in Ireland is expected to rise sharply, and as early as 2011, a fifth of older men and almost a third of

older women will be living alone (National Council on Aging and Older People, 2001). For older people who may be living alone, the impact of the urban heat island may be exacerbated by poor social conditions, resulting in an 'urban loneliness island' (Conti *et al,* 2005). Having social contacts in the area were found to be protective (Semenza *et al,* 1996), and excess heat-related mortality in a heat-wave in Chicago varied almost one hundred fold between neighbourhoods, as a result of factors such as housing quality and social interactions (Klinenberg, 2002). In the Italian heat wave of 2003, the greatest excess in mortality was registered in people who scored highly on a deprivation index, which incorporated such factors as unemployment, over crowding and education (Michellozzi *et al,* 2004). Poor housing conditions and the absence of air-conditioning have also been associated with increased mortality in a heat wave in the United Sates (Ramlow and Kuller, 1990), and mortality following the 2003 heat wave in France was noted to be five times less in houses that were well-insulated (Riberon *et al,* 2005). It is uncertain what proportion of buildings in Ireland has air conditioning (Forsythe, 2005). Finally, the most sensitive regions to heat-related mortality appear to be areas where extremely high temperatures occur infrequently or irregularly (Kalkstein and Smoyer, 1993), and Ireland may therefore be in a vulnerable position in relation to unanticipated events. More hot days will occur in the future, and the impacts of such days could be expected to be more severe again, if they occur concurrently. Work undertaken to date indicates that substantial increases may be expected in the number of heat waves lasting more than 6 consecutive days in Ireland (McElwain and Sweeney, 2007), and a significant number of days may be expected, when the threshold for maximum temperature above which mortality rises in both the 15-64 age group and the age groups over 75 and for respiratory mortality is exceeded. This indicates the potential vulnerability of these age-groups to higher adverse temperatures that is difficult to quantify. Unlike a rise in mean temperature, where adaptation may occur over time if mean temperatures stabilize, heat waves, by their nature, are irregular and extreme events, and adaptation therefore could not be expected to occur readily. In addition, it is possible to endure unusually hot days if the nights are cooler, and it is therefore of concern that minimum temperatures are expected to rise more in summer than in winter. Mortality increased in the 15-64 year age group during the heat wave in France in 2003 (Vandentorren and Empereur-Bissonnet, 2005), and as maximum temperatures increase throughout the century in Ireland, vulnerability to these very high temperatures may also increase in younger age groups. Cohen, (Cohen *et al,* 2005*)* in discussing the

impacts of the heat wave in France in 2003, described how people discovered that heat 'could be an aggressor and a threat'. While a reduction will occur in the frequency of colder days, the impact of cold on mortality is still expected to be dominant and would not seem to be outweighed by the possibility of higher maximum temperatures increasing mortality in summer. Nevertheless, it is certain that the older population is vulnerable to heat, and as temperatures continue to rise, measures to reduce future mortality from heat waves are therefore necessary.

5.5 Issues of uncertainty

There are several issues of uncertainty in this study. The temperature-mortality model used, was driven by the predominant impact of cold on mortality, and was not particularly sensitive to the impact of higher maximum temperatures; elementary curve fitting techniques were used to estimate the temperature-mortality relationship, and the threshold temperatures, and although the R square values were satisfactory, more precise estimates might be obtained with more sophisticated techniques. Furthermore, current estimates of future heat-related mortality may be an under-estimate because although the mortality data was lagged when the impact of minimum temperatures was being assessed, the mortality data was not lagged when the impact of mean temperatures was being assessed. In addition, inaccuracies will result from temperatures in the future being higher than those used to establish the temperature-mortality model on which the future estimates are based. There are also several other issues that might be expected to impact on the validity of these results. Firstly, seasonality was not taken into the analysis and recommendations have been made to remove this impact, using statistical techniques (WHO 2003(b)). However, there is much evidence that the phenomenon of seasonality itself is driven by temperature itself, and seasonality has not always been removed in temperature-mortality studies (e.g. Donaldson *et al,* 2001(b); Gemmell *et al,* 2000). It is also not possible to account for the impact of influenza in the analysis, and although there is possible evidence of such an effect in the data-set in 1986, a register of this disease was only initiated on January 1st, 2004. Due to vaccination, the role of influenza as a determinant of excess winter mortality has declined in the U.K; it has been estimated to account for 2.4% of excess winter mortality, and the role of both indoor and outdoor cold stress is regarded as more important (Donaldson and Keating, 2002). In addition, although influenza has been found to be an independent predictor of

mortality, the heat and cold coefficients have not been found to have been altered by the inclusion of influenza (Pattenden *et al,* 2003). Nevertheless, largely due to vaccination and a reduction in the number of new strains, the incidence of influenza has fallen dramatically in Ireland in recent years and influenza is recorded as a cause of death in approximately 40 deaths in Ireland per year (CSO, 2004). Thirdly, as the impact of temperatures from one station was assessed on national mortality data, a certain amount of systematic error may be expected. However, good correlation values were obtained when the temperature data from Kilkenny was correlated with temperatures from other meteorological stations in Ireland. Similarly, the values for the minimum mortality band established here might be expected to be slightly higher in cities such as Dublin or Cork, where the population might be expected to be accustomed to slightly higher temperatures, due to the heat-island effect. However, to counterbalance this, heat wave effects may also be more pronounced in the cities. Fourthly, although socio-economic conditions have a substantial impact on mortality rates, such factors were not taken into account. Mortality in heat waves has been noted to be higher in poorer areas and future work might take such factors into account. Finally, the impact of other meteorological variables such as humidity or wind speed, or levels of air-pollution which might be expected to influence the impact of high temperatures on health were not assessed. Heat related mortality may be expected to be higher in conditions of high humidity, when sweating is impeded, and also in situations where levels of air-pollution are high. This may be an area of future research.

5.6 Conclusion

In conclusion, the impacts of cold temperature greatly outweigh the impacts of hot temperatures on mortality at present. Reductions in mortality may therefore be expected from the increase in mean temperatures resulting from climate change, and Ireland with high excess winter mortality may benefit disproportionately. While future maximum and minimum temperatures will also result generally in an overall reduction in mortality, particularly from respiratory disease, future maximum temperatures may also result in a rise in mortality, and there is evidence that such mortality may not be accounted for by harvesting. However it is possible to reduce mortality from heat waves, and reductions in mortality have been shown to occur following such interventions. These will be discussed in Chapter 9.

CHAPTER 6 - CLIMATE CHANGE AND WATER-RELATED DISEASE

"The issue of water quality is of importance to anyone who uses a tap or a toilet"

(Boyden, 2006)

6.1 Introduction

Water is a fundamental requirement for life on earth, sustaining essential physiological processes. Aside from this vital role, water is also necessary for cleaning, cooking and recreational purposes, all life sustaining processes. Despite its seeming abundance, most of the water on the planet is not immediately available for use, and although water covers almost three-quarters (70%) of the Earth's surface, the majority (over 97%) lies in the ocean. Three quarters of the remainder lies in ice sheets and glaciers, and less than 1% of the total water on the planet on the surface, is in groundwater, the soil, and the atmosphere. Water-related diseases refer to illnesses which are caused either directly or indirectly by the quality or quantity of water (WHO, 1999). As a result, all of the international assessments of the impacts of climate change on water-related diseases identified in the literature to date have been qualitative, for example, in Finland, (Hassi and Rytkonen, 2005), in Portugal (Santos *et al,* 2002), and the Netherlands (Bresser *et al,* 2005), and all have referred to the importance of water infrastructure in determining water quality. An examination of the impact of climate change on the incidence of water-related diseases in Ireland in the future is the subject of the remainder of this chapter. The approach will be as follows: the relationship between water and health will be discussed, followed by consideration of the future impacts of climate change on water-related diseases. Methodological issues relating to the assessment of the impact climate change on water-related diseases in Ireland will be considered and an assessment of the impacts of climate change on water-related disease will then be undertaken.

6.2 Water and health

The global burden of infectious waterborne disease is considerable (Ford, 1999) but unequally distributed (See also Tables 1.1 and 1.2). Over 1 billion people lack access to

safe water supplies and 2.6 billion people lack adequate sanitation, resulting in 3.2 million deaths per year, or 6% of all deaths globally (Corvalan *et al*, 2005). However, water-borne disease is also a problem in European countries, including Ireland. Although sanitation services have improved and outbreaks resulting in high mortality rates are rare, nevertheless, epidemics of serious water-borne diseases have occurred in Ireland in the past, and outbreaks of both cholera and typhoid have incurred major loss of life. Although half a million people were reputed to have died from cholera in Ireland in 1832 (Farrell, 1866), the casualty toll was most probably in the order of 25,000 between 1832 and 1834 (Howe, 1972), and 145 cases of typhoid were reported in one parish in Sligo in 1880 in a 10 month period (Woodhouse, 1880).

While most of the burden of poor water quality today is endured by people living in poorer countries, problems have also been noted in temperate countries, although it is difficult to estimate the prevalence. In Europe to date, no reliable data are available to estimate the burden of such diseases (Ballester and Sunyer, 2000), the available data having been described as 'incomplete and inconsistent' (EEA/WHO 2002). However, estimates of the burden of waterborne disease are considerable; in the United States it is estimated that 9 million people out of the 200 million people who have access to public water supplies contract a waterborne illness every year, most cases however are mild (Rose *et al*, 2001). In the UK, over 4,000 people were estimated to have been affected by outbreaks of waterborne disease, as distinct from individual cases, over a 9 year period (Hunter, 2003). In Ireland, from 2001 to 2005, 18 possible waterborne outbreaks were reported causing over 1,100 people to be affected. This gives a much higher rate than the UK, although the link with water in the Irish situation was circumstantial in some cases (HPSC, 2006(b)).

Although the quality of Irish waters today is under pressure from agricultural, industrial and municipal discharges (Byrne, 2006), both drinking and bathing water parameters in Ireland are tested on a regular basis, and while there are some problems, the overall quality of Irish drinking water in public supplies and bathing water is good (EPA, 2006(a); EPA, 2006(b)). While the quantity of water that is available for drinking is under pressure from increased abstraction in Northern European countries, as yet water availability is not a problem (EEA, 2000). Nevertheless, the potential for strife and unrest exists as surface and ground water become contaminated and the importance of

potable water in sufficient quantities becomes apparent (FSAI, 2006(a)). In addition, warmer waters will encourage micro-organisms to multiply. The changes in rainfall may have more important impacts on human and environmental systems in Ireland than the changes in temperature (Sweeney *et al,* 2003). At present, those most vulnerable to water related diseases are people who have suppressed immune systems (e.g. suffering from HIV/AIDS, people on steroid treatment), the elderly, babies and those whose health is already weakened.

6.3 Impacts of climate change on the water cycle in Ireland

The future impacts of climate change on water resources in Ireland, based on global climate models, downscaled for Ireland have been discussed (Section 3.8) (Sweeney *et al*, 2003). As a result of the impacts of climate change, an increase in the number of episodes of precipitation, particularly in northern and western areas, and an increase in the number of floods may be expected in Ireland. Increased rainfall resulting from climate change, will lead to higher rates of water runoff, which can transport pathogenic bacteria from the land into drinking-water sources, and thus increase the risk of waterborne disease (AR4, 2007; Hunter, 2003). In addition, excessive rainfall or flooding may overwhelm water purification facilities, resulting in increased turbidity. Turbidity is significant, because it may compromise the efficiency of the water purification plant, as the fine particles suspended in the water may shield pathogenic organisms from the disinfection process.

Decreased precipitation will also occur, particularly in Eastern areas, and water availability will also be reduced by increased evaporation and possibly compromised by the incursion of sea water, resulting in the contamination of groundwater ((Sweeney *et al*, 2003; AR4, 2007). There will also be an increased demand for water from increased populations living in warmer climates, by agricultural irrigation and also by increased recreational use of water in summertime (EEA/WHO, 2002; Sweeney *et al,* 2003). A reduction in water supplies may lead to an increase in diseases that are associated with water scarcity. There is a lack of data on the health impacts of restrictions to water (EEA/WHO, 2002). However, decreased ground water levels may lead to an increased dependence on surface waters, which may be more contaminated leading to an increased incidence of water-borne diseases (AR4, 2007).

6.4 Methodological issues

There are particular difficulties encountered when estimating the future changes in the water cycle resulting from climate change. The quality and quantity of water depends on many factors, including agricultural, urban and industrial land use, the depth of the aquifers, water management practices, and, significantly climatic factors (Rose *et al* 2001; EEA/WHO, 2002). The complex interconnections of these factors make it difficult to analyse the specific impact of future climate change on the water-cycle (EEA/WHO, 2002). In addition, precipitation scenarios are also less reliable than temperature scenarios (Sweeney *et al*, 2003). The complexity of the water-cycle, and the many factors that influence water quality, such as agricultural, environmental and socio-economic, militate against a quantitative assessment of the impact of climate change on water-related diseases. Nevertheless, in order to assess the impacts of climate change on water-related diseases, a framework for the classification of water-related diseases was sought. One classification identified, sub-divides such diseases into four non-exclusive categories, namely waterborne, water-based, water-washed and diseases related to insect vectors (Bradley, 2004). A slightly amended version of this classification has been used here in the present study, and the four categories of water - related diseases comprise:

- Water-related diseases associated with contaminated drinking water (this category comprises the largest category),
- Water-related diseases associated with recreational use of contaminated water,
- Water-related diseases associated with compromised cleaning practices resulting from a lack of access to water,
- Water-related insect vectors, such as mosquitoes and tsetse flies. Such diseases are not endemic in Ireland at present (malaria will be discussed in chapter 8)

6.4.1 Data sources

Data availability on diseases associated with water is limited (Table 6.1).

Category	Disease	Data available
Waterborne disease associated with contaminated drinking water	**Cryptosporidiosis, giardia and all serotypes of VTEC**	**Since 2004**
Water-related diseases associated with recreational use	**Leptospirosis and Legionnaire's disease**	**Since 1982**
Water-related diseases associated with compromised cleaning practices	**Scabies, Conjunctivitis**	**Not available**
Water-related insect vectors	**Malaria**	**Discussed in Chapter 8**

Table 6.1 Data availability on water-related diseases

Incidence data on water-related diseases in the first category, i.e. those associated with contaminated drinking water, namely cryptosporidiosis, giardia and all serotypes of VTEC is only available since 2004, when these diseases became legally notifiable. Monthly data on the number of cases of cryptosporidiosis and giardia was obtained for 2004 and 2005 from the Health Protection Surveillance Centre (HPSC). All serotypes of VTEC also became legally notifiable in 2004. An assessment was therefore made of the incidence of these diseases in the years 2004 and 2005 with respect to mean temperature and to rainfall. Temperature data was obtained, as before, from Met Eireann. Similarly, mean monthly rainfall data from Kilkenny, Valentia and Dublin Airport was also obtained for the corresponding years, 2004 and 2005 from Met Eireann.

Data on diseases in the second category, namely diseases associated with bathing and other recreational uses of contaminated water is also limited. Although such diseases as leptospirosis and legionnaire's disease had been notifiable, these are rare diseases and it is therefore difficult to quantitatively estimate the present impact of climate on water-related disease. The range and disparate nature of diseases in the third category, namely those arising from compromised cleaning practices associated with a lack of water (for example conjunctivitis, scabies, and gastroenteritis), also renders it difficult to quantify a relationship with climatic factors. Based on the classification outlined, a discussion of the first three water-related diseases will follow. In addition to data from the HPSC, qualitative information was also gathered from other literature sources, in

order to gain more information on the link between climatic influences and water-related diseases.

6.5 Water-related diseases associated with contaminated drinking water

In temperate countries, the most significant infection associated with drinking contaminated water is cryptosporidiosis (ACACIA, 2000). Other common infections spread by water include giardia and toxic verocytogenic E. Coli (VTEC). However, VTEC infections may also be spread by food, and therefore this organism is also discussed in Chapter 7. A brief synopsis of cryptosporidiosis, giardia and VTEC follows, prior to an assessment of the present and future association with climate. As the first two categories comprise the vast majority of water-related diseases in temperate countries, this will comprise the greater part of this chapter.

6.5.1 Cryptosporidiosis

Introduction

Cryptosporidium is the most significant water-borne disease associated with the public water supply in Western Europe (Acacia, 2000; IPCC, 2001), and is primarily transmitted by the consumption of contaminated water. Infection can also occur as a result of recreational bathing, consumption of contaminated foods, and from animal and person to person transmission. Symptoms develop after a week, the main symptom being watery, non-bloody diarrhoea, which may be profuse and accompanied by abdominal pain. It is often accompanied by anorexia and vomiting in children. Infection may be also asymptomatic. The disease is generally self-limiting in a month or less in people with intact immune systems. However people with suppressed immune systems may be unable to clear this infection from their body, and this may be fatal. Cryptosporidiosis multiplies in the gastrointestinal tract, producing tiny oocysts, 4-6 microns in diameter. These oocysts are very infectious and can survive for long periods in cool, wet environments. Significantly, they are resistant to routine chlorination and are generally removed from the water in the filtration stage of the water treatment process. Children appear to be at high risk of this disease, possibly because they may become exposed during outdoor play. Two Irish surveys both found a prevalence of approximately 4% in the incidence of the organism in cases of diarrhoea in young children (Corbett-Feeney, 1987; Carson, 1989).

Results

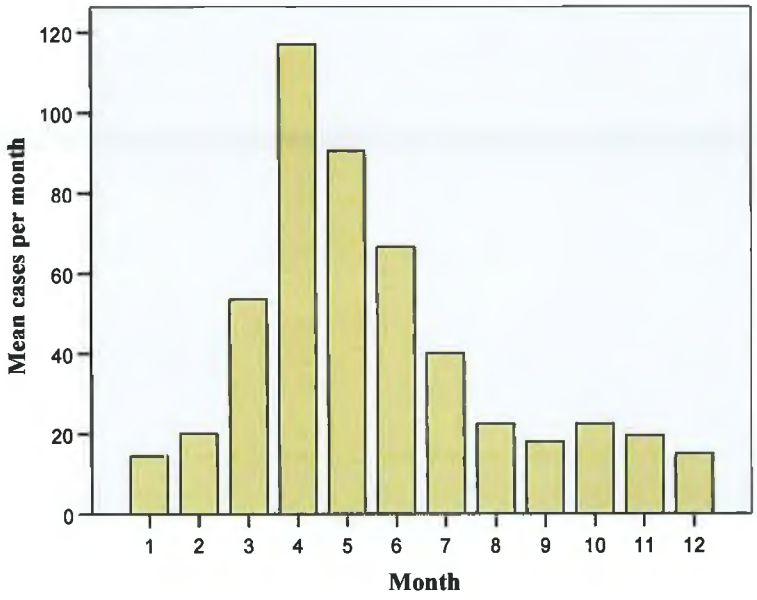
The incidence of cryptosporidiosis in general in Ireland is low. There were 453 cases in 2004, and 546 in 2005. In 2004, over 80% of cases were in children under 15 (Table 6.2).

Age (years)	Number of cases
Less than 1	38
1-4	221
5-9	69
10-41	21
Greater than 14	79
Not known	4
Total	432

(adapted from Garvey and Mc Keon, 2004)

Table 6.2 Age distribution of cryptosporidiosis 2004

A seasonal incidence is noted in the incidence of cryptosporidiosis in 2004 and 2005 and the highest incidence is recorded in April, with over a half of the cases in 2004 and 2005 being reported in the months of April, May and June (Figure 6.1).



(adapted from HPSC 2005(a) and 2006(b))

Figure 6.1 Monthly distribution of cases of cryptosporidiosis 2004-5

Although an increased incidence of cryptosporidiosis is associated with heavy rainfall, the heaviest rainfall in the three stations assessed occurred in October (Figure 6.2) and incidence of the disease is seen to peak in April (Figure 6.1).

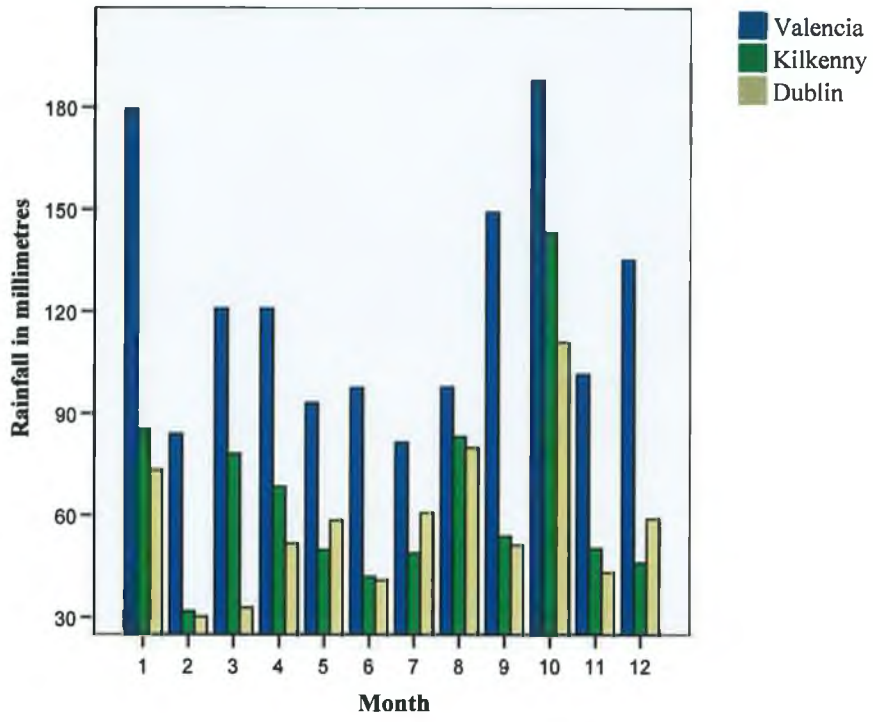


Figure 6.2 Mean monthly rainfall in millimetres 2004-2005

No link with mean monthly temperature is apparent (Figure 6.3).

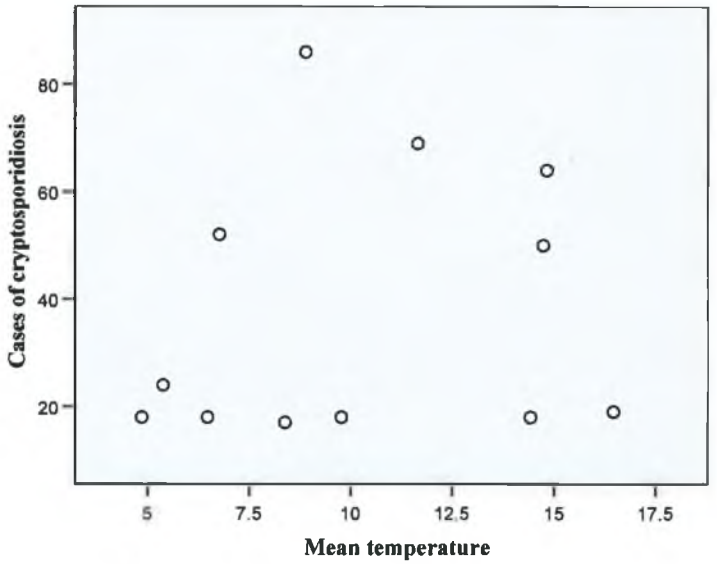


Figure 6.3 Mean monthly cases of cryptosporidiosis and mean monthly temperature 2004-5

Discussion

The incidence of cryptosporidiosis in Ireland appears is low. It is higher in children, although this may in part reflect the fact that children may have more medical investigations undertaken when they are unwell (HPSC, 2005 (a). A peak in the incidence is evident in the beginning of both years; this has been noted previously and the reason for this rise is not clear (HPSC, 2006(b)). The low incidence of cryptosporidiosis leads to difficulties when estimating the impact of rainfall and temperature on the incidence. From the data presented, there is no clear link apparent between the incidence of cryptosporidiosis and rainfall when data is examined on a national basis in the short time period (Figures 6.1 and 6.2). Although it may not be possible to date, to ascertain a link between national data and the incidence, nevertheless links between local climatic influences and the incidence of cryptosporidiosis in Ireland may be made. Heavy rainfall was thought to have contributed to an outbreak of cryptosporidiosis in Co. Westmeath, in April 2002, which was thought to have been caused by contaminated water, preceded by heavy rainfall, facilitating the entry of oocysts from the land to the unfiltered water supply (Gillooly and Green, 2003). This was the first recorded outbreak of cryptosporidiosis associated with a public water supply (Jennings and Rhatigan, 2002). In addition, during the wet summer of 2007, where rainfall totals for July were exceptionally high in the east and the south of the country (Met Eireann, 2007), cryptosporidiosis was reported in the public drinking water in Clonmel, Co. Tipperary, although no outbreak was recorded. This contamination may have arisen from droppings from sheep and wild animals which may have entered the public drinking water supply following the heavy rain (The Irish Times, 2007(a)).

There are many such examples in the literature detailing increases in the number of spores of cryptosporidium in water supplies after episodes of heavy rain, and examples of subsequent illness (Hoxie *et al*, 1997; Atherholt *et al,* 1998; Curriero *et al,* 2001, for example). An outbreak of cryptosporidiosis in Wales in 2005 was linked to heavy rainfall two weeks prior to the event, causing sewage runoff to enter a drinking water reservoir (Carnicer-Pont *et al,* 2005), and outbreaks of cryptosporidiosis have also been associated with high river flows (Lake *et al,* 2005). A severe outbreak of cryptosporidiosis occurred in Milwaukee in 1993, and resulted in 403,000 people falling

ill and 50 deaths among people suffering from immune suppression (MacKenzie *et al,* 1994). Contributory factors to this episode included heavy rainfall, human sewage and faecal contamination from cattle of two rivers that supply Lake Michigan (Mc Kenzie *et al,* 1994). Older people appeared to be more vulnerable; they were severely affected, and suffered a more rapid onset of the illness (Naumova *et al,* 2003). Similarly, a cluster of cryptosporidium among people with AIDS alerted health officials to a waterborne outbreak in Nevada (Rose et al, 2001) and it has been recommended that waterborne routes of transmission should be considered when clusters of cryptosporidiosis associated with potable water occur (Smith et al, 1989).

Although not directly linked with increased rainfall, links have also been made between outbreaks of cryptosporidiosis and the quality of public drinking water supplies locally in many areas in Ireland including Carlow and Galway (HPSC, 2005(b); (HPSC, 2007(a)). An outbreak of cryptosporidiosis in Carlow in 2005, resulted in 31 people becoming ill, and links were made to the public drinking water supply (HPSC 2006 (b)). Furthermore, in 2007, an extensive outbreak of cryptosporidiosis in the public drinking supply occurred in Galway. 125 laboratory-confirmed cases were reported in March (up to 29/3/2007) compared with total of just 6 cases in March 2006 (HPSC, 2007(a)), and in total 260 confirmed cases were reported (Mc Keown, 2007). This outbreak has been preliminarily attributed to inadequate water treatment (Pelly *et al,* 2007). The source of the drinking water in Galway was subject to pressures from urban waste water, septic tanks and agricultural runoff, all of which may contain the organism. Cryptosporidium is resistant to chlorination and water treatment plants in sub-optimal condition may be overwhelmed with heavy rainfall and unable to filter cryptosporidiosis adequately. It has been estimated that currently 135,000 people on public water supplies in Ireland at present are at risk of this disease, because they do not have filtration systems in place (EPA, 2007 (b)). This has been known for some time, as a previous risk assessment undertaken on a sample of public water supplies in Ireland found that over a fifth of public water supplies were in the high risk or very high risk category for an outbreak of cryptosporidiosis, (EPA, 2005). Furthermore, significant levels of cryptosporidiosis have been found in studies of waterways in Ireland (Chalmers *et al,* 1997; Graczyk *et al,* 2004). Following three earlier outbreaks of cryptosporidiosis in 2005, in Carlow, Ennis and Roscommon (EPA, 2006(a)), a Cryptosporidiosis Working Group was established by the Environmental Protection Agency. The aims of this group, which

includes representatives of the local authorities, health and environment agencies was to investigate issues such as risk assessments, the availability of laboratories for testing, water treatment works and the management of outbreaks; it is expected to report and to make recommendations early in 2008 (EPA, 2008 (b)).

No clear link between the incidence of cryptosporidiosis and temperature is apparent (Figure 6.3), although the presence of cryptosporidiosis oocysts in the soil has been linked to soil temperature (Jenkins *et al,* 2002). However, the association with disease is not clear cut and a link identified between the incidence of cryptosporidiosis in summer and autumn in New Zealand was attributed to exposure due to outdoor recreational behaviour (Lake *et al*, 2007).

In conclusion, while no clear picture is evident when data is examined on a national basis, it is clear that both inadequate water treatment and locally heavy rain may contribute to outbreaks of cryptosporidiosis locally.

6.5.2 Giardia

Introduction

Giardia is a gastrointestinal infection, and is often asymptomatic. However, it can also cause chronic diarrhoea, abdominal pain, bloating, fatigue and weight loss. Person to person transmission occurs by hand to mouth transfer of cysts from the faeces of an infected individual, often from asymptomatic individuals. Wild and domestic animals may also harbour the organism, and it is commonly found in waters that have been polluted by animal faeces. It is possible for giardia to pass through water treatment plants, as turbid water inhibits the access of the chlorine to giardia, and the concentration of chlorine used in routine water treatment does not kill giardia cysts.

Results

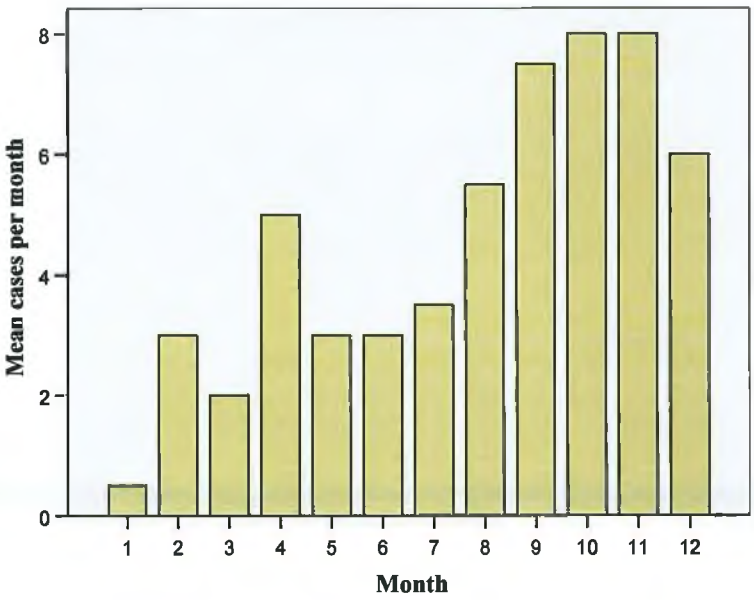
Giardia is not as common as cryptosporidiosis in Ireland; there were a total of 53 cases of giardia in Ireland in 2004, and 57 cases in 2005. Almost a third of cases occurred in the under 14 year age group (Table 6.3).

Age (years)	Number of cases
0-4	22
5-9	7
10-14	3
Less than 14	77
Not known	1
Total	110

(adapted from HPSC2006(b))

Table 6.3 Age distribution of giardia 2004

Over a half of the cases of giardia in Ireland in this time period (53%) occurred in the three months from September to November (Figure 6.4).



(adapted from HPSC 2005(a) and 2006(b))

Figure 6.4 Monthly distribution of cases of giardia 2004-5

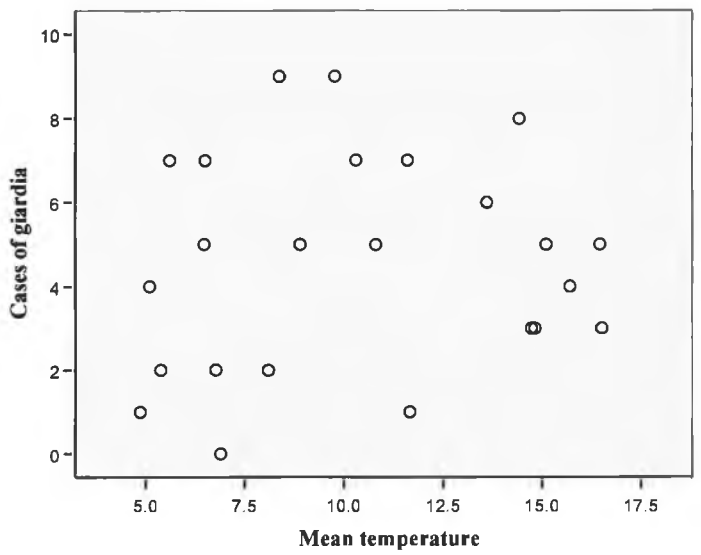


Figure 6.5 Cases of giardia and mean monthly temperature 2004-5

A link between the incidence of giardia and temperature was not identified in the literature, and no link was evident in the present study (Figure 6.5).

Discussion

An increased incidence of giardia is noted in October, the month in which rainfall was highest rainfall in the time series (Figure 6.2), and in the following month (Figure 6.4), however no increase was evident in January when heavy rain also occurred in all three locations (Figure 6.2). Similar to the situation with cryptosporidiosis, local data and data on meteorological conditions are necessary before a link between increased rainfall, leading to a deterioration in water quality and the incidence of giardia in Ireland can be identified, and no report was identified to date of giardia being associated with either climatic parameters or inadequate water treatment in Ireland. However, associations have been made between an increase in the incidence of giardia and high levels of turbidity in drinking water supplies (Atherholt *et al,* 1998). This raises concerns in relation to the increased rainfall that will occur, and resultant turbidity in drinking water that may arise resulting in contamination of the water with giardia. An outbreak of giardia, which occurred in the United States, in two peaks in June and July 1980, resulted in 780 people became ill, with giardia isolated in both symptomatic and asymptomatic people. Each peak occurred approximately three weeks after an episode of very heavy water runoff, and the water supply to the town was both unfiltered and inadequately chlorinated (Weniger *et al,* 1983). Similar to cryptosporidiosis, significant

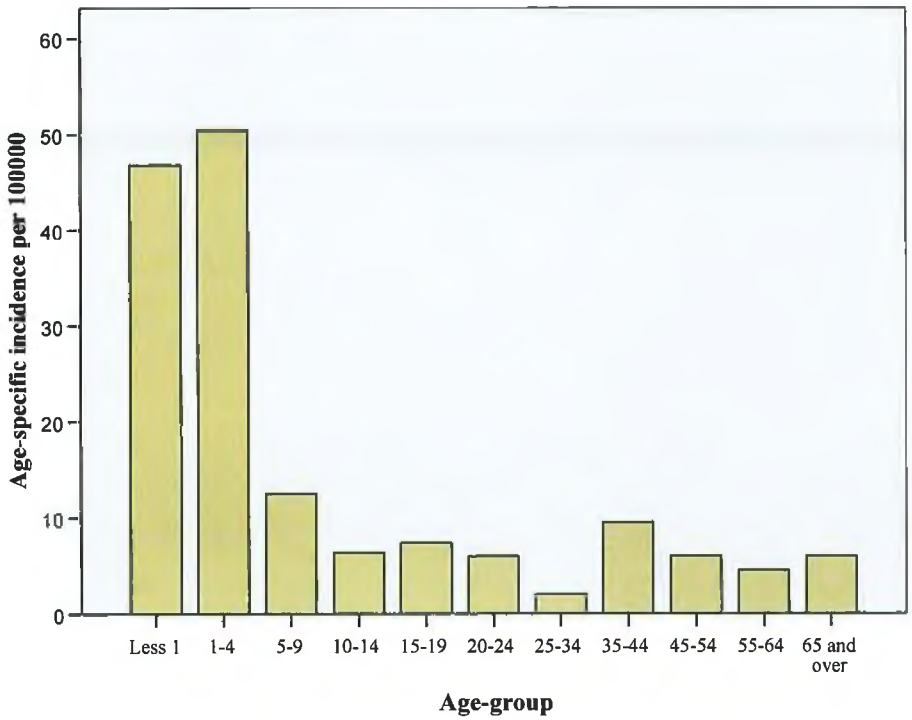
levels of the organism have also been found in studies of waterways in Ireland (Graczyk *et al,* 2004).

6.5.3 VTEC

Although E. Coli is generally a common, and advantageous bacterium, aiding digestion, a toxic form of this bacterium also exists, Verocytogenic E. Coli or VTEC. This organism produces a toxin, the most common type being O157, although other types have now been identified. The presence of E.Coli in water indicates contamination of the water by animal or human waste. Episodes of heavy precipitation, resulting from climate change may increase surface run-off, with consequent such contamination of drinking water. The epidemiology of this serious disease is discussed in Chapter 7.

Results

It is fortunately not common and there were 125 cases of all types of VTEC reported in 2005 in Ireland, a rate of 3.2 per 100,000, and an increase on the previous year of 1.6 per 100,000. Again, similar to the situation with cryptosporidiosis and giardia, the condition is commoner in young children (Figure 6.6).



(adapted from HPSC 2005(a) and 2006(b))

Figure 6.6 Age distribution of cases of VTEC 2004-5

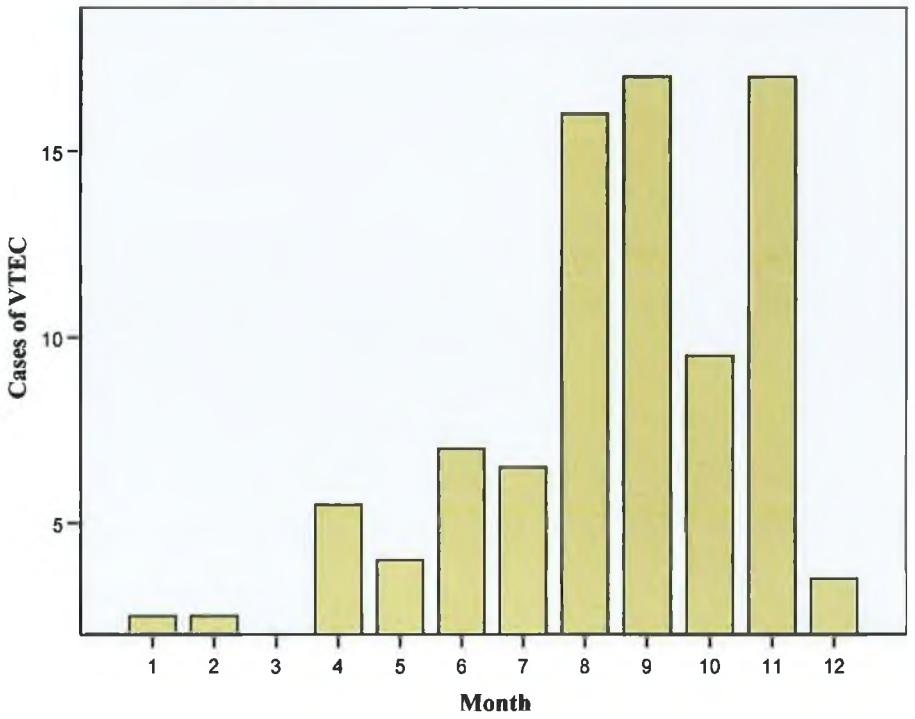


Figure 6.7 Mean monthly cases of VTEC 2004-5

The association VTEC and mean temperature is discussed in Chapter 7.

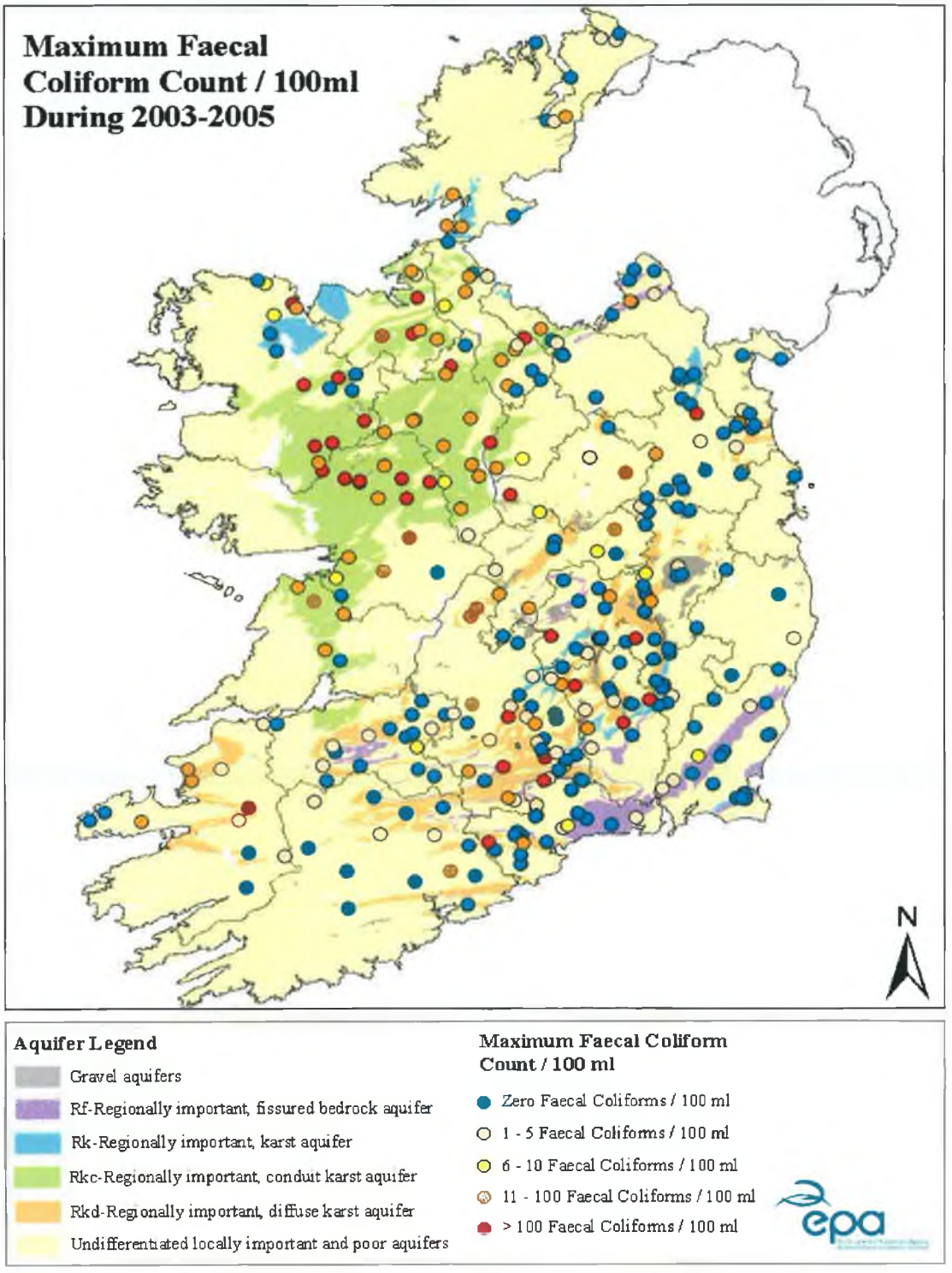
Discussion

Although an increase in the incidence of VTEC is seen to occur later in the year (Figure 6.7), no clear relationship between the incidence of this disease and the high rainfall noted in October (Figure 6.2) is evident. While no clear link between rainfall and the incidence of VTEC was identified in this study when data was analysed on a national basis, the largest outbreak of VTEC in Ireland was reported in 2005, and although the organism was not isolated from the water supply, the water supply involved was a private well, close to land close to areas where cattle grazed and slurry was spread, and a strain indistinguishable from the outbreak strain was isolated from these farm samples (HPSC, 2006 (b)). Examples of how this may occur are widespread; for example, heavy rain resulted in raw sewage running into Loch an Mhuilinn, Galway, the main source of fresh drinking water for more than 1,000 houses in 2004, although no illnesses were noted. (Irish Times 2004 (b)). Heavy rain has resulted in outbreaks of VTEC; the largest reported outbreak of this disease occurred in New York in 1999, and was linked to contaminated well water, 65 people were hospitalized, 2 of whom died (MMWR, 1999).

A further outbreak of both VTEC and campylobacter occurred in Walkerton Canada in 2000, where a total of 1,636 people were affected, 6 of whom died. Infected faecal material from animals was identified in the water and analysis of the drainage pattern suggested that water from these animals would drain towards the well. In addition, alongside the heavy rains and flooding, the water treatment system may have been overwhelmed by the increased turbidity (CCDR, 2000). In Ireland, several links have been made between the incidence of VTEC and the quality of drinking water locally, in particular water from private[1] group water schemes (NDSC, 2004; HPSC, 2005(b); HPSC, 2007(c)). People who consumed water from private supplies in 2003 were found to be over represented among sufferers of E Coli 0157, as only 64% of sufferers of E. Coli 0157 received water from public supplies, although almost three quarters (72%) of the population consumed water from public supplies (NDSC, 2004). In the following year, private wells contaminated with E. Coli 0157 were responsible for 6 confirmed cases of E. Coli 0157 in two separate incidents (HPSC 2005 (a)). It is of concern that a third of private water supplies were contaminated at least once with E.Coli (EPA, 2007(b)) with over a half (55%) failing to meet the E. Coli criteria on at least one occasion (EPA, 2007(c)). Group water schemes serve over 11,000 people, and are slightly more dependent on ground water sources (EPA 2004 (a)). This is of concern as there is widespread contamination of groundwater across the country, with approximately 30% of samples showing E. Coli contamination, and 11% being grossly contaminated, (EPA, 2006(c)). Vulnerable areas of groundwater are evident particularly in the West and midlands areas (Figure 6.8), and this is of concern in view of the increased rainfall expected in the North and North-western areas. Similarities exist between the porous hydrological setting in Walkerton, Ontario (where the large outbreak of waterborne VTEC occurred) and in Ireland (shallow subsoil over karst limestone), and in addition, intensive rainfall sufficient to generate such a hazardous flow also occur in both countries, which have similar hazards, namely grazing animals

[1] *Drinking water in Ireland is classified as either 'public' or 'private', depending on how it is supplied. Public water supplies refer to water which is both sourced and distributed by local authorities, and supplies approximately 90% of the population. Private water supplies are distributed and in some cases also sourced by local groups.

and slurry spreading (Daly, 2003). The problems outlined are of particular concern in the North and West, where future increases in rainfall in this century are estimated.



(from Lucey, 2005)

Figure 6.8 Vulnerable groundwater is evident

Concerns have also been expressed in relation to public water supplies; although E. Coli was detected in less 1% of drinking water samples in the most recent report on Irish drinking water, it was present in over 8% of actual public water supplies. Although chlorine is effective in inactivating E. Coli, over a third (40%) of the incidents where E. Coli contamination of the drinking water occurred were due to a failure of the chlorination equipment that was not detected immediately (EPA, 2006(a)). In addition, almost two thirds of Irish public drinking water supplies (61%) did not have a residual chlorine monitor to measure the effectiveness of chlorination. Furthermore, not all public water supplies are monitored; the quality of public water supplies was not assessed in 6% of supplies in 2006, and while this was an improvement on the figure of 14% for 2005, it is 'unacceptably high' (EPA, 2007(b)). In most situations where the public water quality standards were breached, the contamination was noted to have come from improperly sited septic tanks and slurry pits, direct animal access to water sources, intensification of animal rearing and inappropriate spreading of animal manures (EPA, 2005). All of these sites are possible sources of cryptosporidiosis, giardia and E.Coli.

Two other methods by which heavy rainfall may increase the risk of waterborne disease have been identified. Increased precipitation has been found to enhance the food resources of small mammals, leading to an abundance of plague hosts (Parmenter *et al,* 1999). Following the extreme rainfall from the 1991-1992 El Niño event, an increase in the mouse population is believed to have contributed to cases of hantavirus pulmonary syndrome in the United States (Engelthaler *et al,* 1999; Hjelle and Glass, 2000). In addition, the number of rats may also increase after flooding episodes (Dangendorf, 2004), further increasing the risk of disease. Secondly, increased rainfall can also carry nutrients into water sources, encouraging algal blooms and planktonic species.

Nevertheless, the outlook will improve with the implementation of the new Drinking Water Regulations. The Environmental Protection Agency now has enforcement powers to ensure that local authorities take action when there is a breach of quality in the public water supply. Significantly, since 2007, under new Drinking Water Regulations, the Environmental Protection Agency has the authority to prosecute a Local Authority who is not adopting adequate measures to preserve water quality (EPA, 2007(b)). In turn, the

local authority is now the designated the supervisory authority over private schemes. It is clear that much remains to be done. Since the new drinking water regulations came into force in March 2007, over 113,000 people utilizing 21 supplies were either asked not to use the water or to boil it first (EPA, 2007(b)).

6.5.4 Discussion of water-related diseases associated with contaminated drinking water

The results of the present study clearly indicate an association between waterborne disease and rainfall, and this is well established in the literature (Smith *et al,* 1989; Miettinen *et al,* 2001; Curriero *et al,* 2001, for example). Although it was not possible to derive quantitative data in the present study, statistically significant relationships between rainfall and waterborne disease have been calculated; over two-thirds (68%) of waterborne disease outbreaks between 1948 and 1994 in the United States were preceded by precipitation events above the 80th percentile in the month of the outbreak (Curriero *et al,* 2001). Outbreaks due to surface water contamination had the strongest association, and occurred earlier than in groundwater, where a two month lag was evident. Surface water is at a higher risk of contamination than ground water because of the absence of the protective and filtration properties of natural soil (Bridgman et al, 1995; Kiestemann et al, 2002; Hunter, 2003). This is also of concern as over 80% of drinking water in Ireland is sourced from surface water. Turbidity itself has been recommended as a proxy for the ability of filtration to remove pathogens (Kiestmann et al, 2002), and it has frequently been shown to be associated with gastroenteritis (Morris et al, 1996; Schwartz et al, 1997; Atherholt et al,1998). This association has been quantified: an interquartile range increase in turbidity has been associated with a 9% increase in hospital admissions for gastroenteritis both in people aged over 65, and in particular aged over 75 (Schwartz et al, 2000), and also in younger and older children by 13% and 31% respectively (Schwartz et al, 1997) (an inter-quartile range is the range of concentrations containing the central 50% of the data). It is of concern therefore that, although turbidity will increase following heavy rainfall, over two thirds (68%) of audited public water supplies in Ireland had no turbidity monitor, and 40% of the supplies monitored did not meet the standards for turbidity whereby the risk of cryptosporidiosis would be reduced (EPA, 2007 (b)).

6.5.5 Other waterborne diseases

Gastroenteritis from any microbial cause may result from the ingestion of shellfish which have grown in contaminated waters. Waterborne illnesses may also be caused by Norwalk-like virus (also referred to as the Winter vomiting bug), hepatitis A, microsporidia and swimmer's itch. A brief synopsis of the salient points of these other waterborne diseases and their climatic relationship is outlined (Table 6.4)

Organism	Significance	Outcome	Temperature Link	Possible change in incidence with climate change established	Incidence 2005 in Ireland
Campylobacter*	Generally spread by food, but may be spread by water. Outbreaks associated with private wells	Complete recovery usual	Yes, but not clear cut (Nylen *et al,* 2002;Patrick *et al,* 2004; Kovats *et al,* 2005)	Yes	46 per 100,000
Norwalk-like Virus	Causes up to 85% of non-bacterial gastroenteritis	Complete recovery usual	None established	Not as yet	61 outbreaks 1,891 ill
Hepatitis A	Generally spread by hand contact but may be waterborne	Complete recovery usual	None established	Not as yet	56 cases
Microsporidia	Opportunistic infection affecting the ill and elderly	Recovery usual but relapses likely	None established	Not as yet	Not known

**Discussed more fully in Chapter 7*

Table 6.4 Other waterborne illnesses

6.5.6 Conclusion of risks of waterborne disease

Although no clear relationship was apparent between the months with the heaviest rainfall in Ireland and the incidence of cryptosporidiosis, giardia and VTEC, future episodes of these waterborne diseases are likely to occur after heavy rain fall in areas where water treatment facilities are inadequate. It is possible to identify two particularly vulnerable sub-groups of the population who are at risk of waterborne disease. Firstly, people who consume water from private group supplies, unless remediation procedures are implemented, and secondly, people who consume drinking water from both private and public supplies, when the quality of these supplies are inadequately monitored. In order to more fully understand the links between these diseases and climatic factors, it will be necessary to utilize the databases that are now being developed since the advent of the new statutory reporting mechanisms for infectious diseases, introduced in 2004. Such data, combined with GIS referenced data of the location of the sufferer, and details of local climatic conditions, water treatment facilities and land-use will facilitate future work into the link between climate and water-borne illness.

6.6 Water-related diseases associated with recreational use of water

Water-related infections are infections caused by contact with contaminated water and therefore are often associated with recreational exposure to water in lakes, ponds, rivers and occasionally in swimming pools and hot tubs. Although cryptosporidiosis and E.Coli may also be contracted from ingestion of water in a recreational setting, the main specific water-based infections in Ireland are leptospirosis, Legionnaire's disease and algal blooms. These diseases and their climatic relationships are discussed below.

6.6.1 Leptospirosis

Leptospirosis is a potentially serious bacterial disease that causes a high fever, severe headache, muscle pain, abdominal pain, jaundice, vomiting, diarrhoea and a rash. Treatment is with antibiotics and a full recovery is usual; however, those with renal complications may have a fatal outcome. The causative organisms have been found in a variety of both wild and domestic animals. Human infection occurs through direct contact with the urine of infected animals or by contact with surface water, soil or plants contaminated with such urine. Infection has been associated with canoeing, kayaking,

wading, and swimming in contaminated, untreated open water (CDC, 2004). The disease is an occupational hazard for people who work outdoors or with animals, and to those who swim or wade in contaminated waters (CDC, 2004). The incidence of leptospirosis in Ireland is low, and a total of 140 cases were reported in the 23 year period from 1982 to 2003, an average of 6 cases per year. Although leptospirosis had been a notifiable disease before 2004, laboratories became statutory notifiers in that year, and this may account for the slight rise in the number of cases notified in 2004, 2005 and 2006 when 15, 15 and 20 cases per year were notified in each of these years respectively (NDSC, 2004; HPSC, 2006(b); HPSC, 2007(c)). An association with the incidence of leptospirosis and with both recreational use of water and farming has been observed in Ireland (Sayers and Boland, 2002).

Climate relationship

In Ireland, the only climatic association identified in Ireland occurred following heavy rain, when the condition was reported in three canoeists who had canoed in the river Liffey in 2004 (O'Meara and Fitzgerald, 2004). However, outbreaks of leptospirosis have been reported following floods in the Ukraine and the Czech Republic in 1997, and Portugal in 1967 (IPCC, 2001; Gubler *et al*, 2001). In the devastation caused by Hurricane Mitch, a rise in the incidence of leprospirosis was attributed to the large numbers of rats leaving flooded areas (Rosa, 1998). A large outbreak occurred in the United States, in Illinois, after a triathlon, where 375 people became ill and 28 were hospitalised. The outbreak was associated with swimming in a lake that was contaminated with runoff from both agricultural and residential land that may have been contaminated by wildlife (Craun, 2004).

6.6.2 Legionnaires disease

Legionnaire's disease is caused by the bacteria Legionella pneumophilia. It is generally acquired by the inhalation of infected steam from a shower or jacuzzi, or rarely by aspiration of contaminated water. Human-to-human transmission does not occur. The disease may result in a severe pneumonia, although a less severe flu-like illness may also occur. Vulnerability is universal. A total of 66 cases of Legionnaires disease were recorded in Ireland in the 23 year period from 1982 to 2005 (NDSC, 2004; HPSC 2006(b)). Almost half of the cases in recent years were associated with foreign travel

(NDSC 2004; HPSC 2006(b)), and this further hinders the establishment of a climatic link.

Climate relationship

Legionella can multiply to high levels in stagnant water, particularly in the range 20^0C to 45^0C, but it may remain dormant in cool water and multiply when the temperature increases. Spas are ideally suited for the growth of bacteria, providing both warm temperatures and bacteria from bathers. The organism can be found in shower fittings and air conditioners, the use of which may increase with the advent of warmer weather (Stanwell-Smith, 2001). Over a fifth of spa pools surveyed in the UK contained legionella, and the majority of these pools had passed routine microbiological tests (HPSC, 2005(c). Chlorination does not guarantee elimination of the organism (HPSC, 2007(b)), and a new training course for the spa industry has been launched, which emphasises the risks of microbial infection (Mc Greevy, 2008). More research is needed before a link with climate can be established, although a tentative possible future increase is estimated in the incidence of this disease (Weiss and Mc Michael, 2004).

6.6.3 Algal blooms

Algae are at the base of the marine food chain, and while only a small number are harmful, they can produce potent toxins which may result in a variety of acute illnesses. Contamination may occur through the consumption of contaminated shellfish, through skin exposure or through inhalation of aerosols droplets. Conventional coagulation treatments do not remove cyanobacterial toxins. However, as they are normally retained in the cells of the algae, they do not contaminate drinking water as the algae are removed in filters when they are working effectively (WHO/EEA 2002). In coastal waters, the main risk from algal blooms is from contamination of shellfish (Stanwell-Smith, 2001). There are two main types of algal poisoning associated with temperate climates: paralytic shellfish poisoning which results in neurological symptoms, including paralysis and coma, with a 15% mortality rate, or the less severe diarrheic shellfish poisoning, which results in gastro—intestinal symptoms but is generally non life threatening ((Van Dolah, 2000; IPCC, 2001). However, much remains to be known about the health impacts of algal blooms as little epidemiological research has been carried out on the impacts on health of freshwater or coastal algal blooms (Stanwell-

Smith, 2001; EEA/WHO 2002; CDC 2004), in particular, the health effects of chronic exposure to low levels of algal toxins are poorly documented (Van Dolah, 2000).
No register of diseases caused by algal blooms is maintained by the Health Protection Surveillance Centre.

Climate relationship

Algal organisms respond rapidly to changes in environmental conditions and therefore are sensitive biological indicators of the combined influences of both environmental and climate change (IPCC 2001). Toxin accumulation occurs annually, with initial increases in toxicity usually occurring in early spring (Shumway *et al*, 1994) and warmer temperatures could both increase the growth of some algal blooms and extend their range to higher latitudes (AR4, 2007). Each toxic species has its own complex ecology and at present our understanding of this is incomplete and it is therefore difficult to estimate future impacts of climate change on the incidence of harmful algal blooms (Morris, 1999). Modelling has been undertaken to simulate the impact of climate change on the phytoplankton growth rate in Japan (Hassan *et al*, 1998). The impact of a 4^0C rise in temperature by the year 2100 has been investigated on the growth rates of phytoplankton species in the Netherlands, and increases in harmful blooms are estimated to result in human and fish intoxication (Peperzak, 2005). However, the increases in freshwater algal blooms that have been observed in unusually hot summers in the UK, have been attributed to increased agricultural run-off and increased nutrient loads in rivers (Stanwell-Smith, 2001). Recent evidence indicates that climate change has radically impacted on marine trophodynamics and will continue to do so in the coming decades if the climate continues to warm at its present rate (Edwards and Richardson, 2004). Nevertheless, although further research is required, there is no proven link established at present between the incidence of harmful algal species and changes in the marine climate (Nichols and Kovats, 2008), and research in the Irish context has also been urged (Boelens *et al*, 2005).

6.6.4 Other water-related diseases associated with recreational use of contaminated water

While leptospirosis, legionella and algal blooms are the most likely infections that may be affected by future climatic changes, there are other organisms whose incidence may also be affected, and these are briefly outlined (Table 6.5). Occasionally, ear and eye

infection may be caused by contaminated water, and the source sometimes being other bathers in the water (Moe, 2004). New diseases may also emerge, for example, ostreopsis ovata, which was once restricted to tropical waters, has now been reported in the Mediterranean, causing skin irritations and dizziness in over 100 holidaymakers in the Italian Riviera in early 2006 (Hunter, 2007). In addition, in 2004, the first case of E Coli 0157 associated with bathing in the sea was reported, although the source of the infection was not found (Harrison and Kinra, 2004).

Organism	Significance	Outcome	Temperature link	Possibility of link with climate change established
Naegleria fowleri	Found in warm waters, heated swimming pools	Uniformly fatal	Yes	Case reported in Italy after swimming in polluted water on hot day (Cogo 2004)
Pseudomonas Aeruginosa	'Spa' folliculitis Related to low levels of chlorine	Complete recovery Usual	None established	Not as yet
Staphyloccal aureus	Otitis externa and media	Complete recovery Usual	None established	Not as yet
Acanthamoeba	Keratitis	May adversely affect vision	None established	Not as yet
Cercarial dermatitis	Occurs when swimming in warm, untreated surface water, after heavy rain (Hunter 2003(1)).	Complete recovery Usual	Yes	If the climate becomes more favourable for the water snail (de Gentile *et al* 1996).
Vibrio vulnificus	Severe, life threatening, Illness		Yes	May be transmitted to humans is sea temperatures rise(IPCC, 2001)

Table 6.5 Other water-based diseases associated with bathing

6.6.5 *Discussion of water-related diseases associated with recreational use of water*

Although the present incidence of water related illness in Ireland appears low, it is clearly linked to the quality of recreational water. An improvement in the quality of bathing water in Ireland has occurred throughout the 1990s which has been attributed to improved waste water treatment plants (Department of the Environment, 2007), and in general, water quality at Irish bathing areas is of a high standard, with 97% of sites monitored in 2006 complying with EU legislation (EPA, 2007(c)). Nevertheless,

problems may arise in tidal waters, as both urban sewage and industrial effluents, are discharged into such waters. There have been delays in providing the required treatment plants throughout the country, and 11% of these discharges receive no treatment, with 5% receiving only preliminary treatment. Secondary treatment plants are required in parts of Dublin, Killybegs, Sligo, Tramore and Waterford (EPA 2007(a)). Aside from the lack of these facilities, two further problems can be identified. Firstly, concern has been expressed at the efficacy of the existing smaller sewage treatment plants, and problems with inadequate collection systems for waste water and sewer overflows have been identified (EPA, 2007(a)). Secondly, monitoring is not satisfactory and the required number of samples was not taken at over one third of sites where the population was greater than 2,000; furthermore, almost a half of these samples was not taken in the correct manner and recommendations to remediate the situation have been made (EPA, 2007(a)). These issues are of concern: a half of the Irish population lives within 10 km of the sea (Nairn, 2006), and there are 131 designated bathing areas in Ireland.

While saline water is inhibitory to the growth of microrganisms, some survive by attaching to phytoplankton (Mourino-Perez, 1999), and the association between swimming in sea water contaminated with sewage and illnesses, such as gastro-intestinal and skin infections is well documented, the incidence being related to the bacterial counts in the water (Kay *et al,* 1994; Prieto *et al,* 2001). This risk could be expected to rise with runoff from heavy rains (Hunter, 2003). Warmer temperatures may compound this issue. Storm effects have been shown to have a dramatic effect on water quality; 60% of sampling sites on the shoreline from California to Mexico have been shown to fail water quality standards after a rainstorm, compared to 6% after dry weather, the contaminated water arising mainly from urban runoff (Noble *et al,* 2003). In addition, bacterial levels rise substantially across beaches during wet months, and river discharge and bacterial levels are all highest during winters with the most rainfall (Dwight *et al,* 2002). An increased risk of approximately 50% in both upper respiratory and gastrointestinal illness has been reported in people who had been swimming in sea waters which receive untreated runoff from storm drains (Haile *et al,* 1999).

6.6.6 Conclusion of risks associated with recreational use of contaminated water

In conclusion, diseases associated with bathing and recreational uses of contaminated water do not appear to present a large public health problem at present. There may be an increase in the incidence of Legionnaire's disease associated with water fittings, which is difficult to quantify, but may impact on vulnerable users such as older people and those who are ill. However, with increased runoff expected, in particular in northern and western areas, urgent attention must be given to the upgrading of waste water treatment plants, and in particular the upgrading of the wastewater treatment plants in Killybegs and Sligo, in the light of increased rainfall estimated in the future for these areas. Contamination of coastal and inland waters by increased runoff resulting from heavy rains will also increase the risk of contamination of cryptosporidiosis, giardia and VTEC, resulting in increased risk to users of bathing areas.

6.7 Water-related diseases resulting from a lack of access to water

Water-related diseases are associated with a lack of access to water, which compromises cleaning practices, and includes diseases from any infectious agent that may be transmitted by contact such as salmonella, scabies, and conjunctivitis. Such diseases are often associated with poor socio-economic conditions and are not common at present in countries with a temperate climate. A small number of studies have investigated the health impacts of discontinuing water supplies due to financial reasons (WHO/EEA, 2002). Where water metering was introduced in the UK, it was found that poorer households used less water, and an association between the incidence of the infectious diseases hepatitis A, shigella, and discontinued water supplies was noted, with the implication of reduced use of water for economic reasons (EEA/WHO, 2002). There have also been reports of increased incidence of gastroenteritis where the water supply was disconnected due to an inability to pay (Middleton *et al,* 1994). There are undoubted confounding variables in such associations, such as socio-economic circumstances, and more research has been recommended (Fewtrell *et al,* 1994; Middleton *et al,* 1994). However, no deaths have been caused directly by a shortage of water in Europe in the last 50 year (EEA/WHO 2002).

It is clear however, that as a result of the rapid expansion of cities such as Dublin, Limerick and Cork, increased strain is being placed on the water infrastructure (Charlton and Moore, 2003). It is expected that 2.5 million people will be living in the Greater Dublin Area (Dublin, Wicklow and Kildare) by 2031. Dublin City Council is investigating the possibility of either installing a desalination plant on the East coast or abstracting water from the Shannon and transporting it by pipeline to address this issue (DCC Newsletter, 2007). Already, concerns have been expressed at quantities of water available in some areas and the impact of water rationing has been addressed. In June 2004, water rationing was implemented in Fethard and surrounding area in Co. Tipperary, due to the absence of heavy rain. Supplies were cut from 8 a.m. to 8 p.m. 'If we do not get significant rain, the rationing will become normal for the foreseeable future', Mr. Aidan Fennessy, the Council's senior executive officer, was quoted as saying (Irish Times 2004(c)). Reduced water supplies could also compromise the efficiency of local sewage systems, leading to increased concentrations of pathogenic organisms in raw water supplies (Acacia, 2000; IPCC, 2001). Chemical contamination of water may also be a problem because industrial pollutants may not be diluted to a safe degree (Stanwell-Smith, 2001; Sweeney *et al,* 2003).

However, neither scabies nor impetigo, both diseases that may be associated with a lack of washing facilities is notifiable in Ireland and consequently it is not possible to ascertain the incidence. Nevertheless, the reduction in rainfall predicted for the east of the country, in the absence of the provision of alternative sources of water will have serious impacts. Interestingly, it appears that Irish people are becoming more in favour of water charges (Scott, 2007), and with sensitive design, the social concerns about compulsory water metering can be dealt with (Dresner and Eakins, 2007).

6.8 Discussion

The absence of quantitative data on the incidence of water-related diseases makes it difficult to estimate quantitative changes in the future incidence of water-related diseases. Nevertheless, it is clear that the quality of both drinking and bathing waters in Ireland may be adversely affected by the impacts of climate change, resulting in possible changes in the incidence of water-related infections, in the absence of good

management of water treatment plants. Similar conclusions have been reached in qualitative assessments of the impacts of climate change on the future incidence of water-related diseases undertaken in several countries in Europe. All of the assessments found that although the possibility of an increase water-related disease exists, the extent would be determined by the quality of the infrastructure in the country. The estimated impact of climate change on the incidence of waterborne disease were not quantified in the U.S., although it was noted that extreme precipitation events would raise the risk of contamination events, and any increase would depend on policy responses and the maintenance or improvement of the infrastructure (Patz *et al*, 2000; Burke *et al,* 2001). Similar conclusions were reached in Finland (Hassi and Rytkonen, 2005), and in Portugal, where it was noted that if current water management practices were maintained, freshwater quality and consequently drinking water quality would be unlikely to deteriorate (Santos *et al,* 2002). Climate change was also not expected to result in either a significant increase in water related diseases in the Netherlands or problems associated with drought, although an increase in algal blooms and pathogenic bacteria might occur (Bresser *et al,* 2005). The possibility of both contamination of surface and groundwater from heavy rain and flooding and also the problems associated with drought, which would increase the risk of water-washed disease were stressed in Canada, and may pose a public health problem unless 'resilient and adaptive' public health infrastructures are maintained (Charron *et al*, 2004). In the UK, although changes in the future seasonality of waterborne disease may occur, it is expected that good infrastructure would militate against serious water contamination (Stanwell-Smith, 2001). The high standard of water quality in the UK has also been acknowledged by Hunter, (2003), who found that 'provided that the UK does not suffer serious economic collapse, it is difficult to see how global warming could have a major impact on risk of waterborne disease associated with mains drinking water'. This conclusion was supported in subsequent work, when it was considered that the quality of drinking water, surface water, and ground water and also the issue of drought; should not cause a problem for well managed water treatment plants (Nichols and Kovats, 2008).

Not withstanding the pressures currently on water quality in Ireland, the greatest benefit to the future quality of all waters throughout Europe will undoubtedly arise from the implementation of both the implementation of the new Drinking Water Regulations which now places local authorities at risk of prosecution for

failure to achieve adequate water quality standards and the EU Water Framework Directive. Although, this Directive makes no specific reference to the impact of climate change on drinking water quality, its overall objective is to ensure that all inland and coastal water bodies to reach at least "good status" by 2015, and is of particular relevance as 83% drinking water derives from surface waters. The directive requires that management of water quality be based on natural river basins and not on administrative boundaries, and consideration must be given to all human activities that can affect water quality. In addition, these plans will set down a strategy for water use so that there is a sufficient quantity of good quality water in the future, and will provide guidance to city and county development plans (Reynolds, 2006). The effective implementation of this important directive will result in the maintenance of 'high status' of water bodies, where it exists, the prevention of deterioration in the quality of existing waters, and that all waters in Ireland achieve 'good status' by 2015.

6.9 Further progress

Four clear issues arise, namely:

Firstly, in light of the increases in rainfall predicted for the west and northwest, there is a substantial risk of an increase in the incidence of cryptosporidiosis, giardia and VTEC from contaminated drinking water. Although recommendations had been made previously in relation to the prevention of cryptosporidiosis (EPA, 2004; HPSC, 2004(a); Carlow County Council, 2005), the increased powers recently given to the Environmental Protection Agency under the Drinking Water Regulations will undoubtedly result in an improvement in the quality of drinking water. The enforcement of these regulations will be vital to the protection of human health from the impacts of climate change.

Secondly, water supplied from treatment plants that are not functioning adequately in all climatic conditions are clearly at risk. Planning for extreme weather conditions when installing or upgrading existing water treatment plants has been recommended, and in addition, close collaboration between the meteorological and local authorities has been

proposed in order for increased surveillance after heavy rainfall (Kistemann *et al*, 2002; Auld *et al*, 2004; Rose *et al*, 2000).

Thirdly, to reduce the risk of diseases associated with the recreational use of water, swimming-pool operators have been advised to ensure chlorination and in particular that adequate filtration measures are in place (Stanwell Smith, 2001). Training in the risks associated with the recreational use of water must be made mandatory for all workers in the industry, and consideration must be given to the fitting of monitors for legionella in nursing homes, hospitals and leisure facilities. Surveillance must be undertaken for the occurrence of algal blooms in lakes and measure put in place to prohibit bathing where appropriate. Consideration must be given to making diseases resulting from contact with algal blooms notifiable.

Finally, consideration may need to be given to the implementation of dual water supplies, with separate supplies for toilet flushing, and water of a higher quality to be used for drinking. Calls has already been made by government bodies to reduce the use of water in the house and garden via national websites, such as www.taptips.ie. Options such as water-metering and water charges over a set quota per capita must be considered, with allowances for people who may not be in a position to pay. A public awareness campaign to highlight the importance and vulnerability of water may be indicated.

6.10 Conclusion

In conclusion, although difficult to quantify, there is a substantial risk of an increase in the incidence of serious water-related diseases in Ireland including VTEC, giardia and cryptosporidiosis, and other microbial waterborne diseases resulting from the impact of climate change. These changes will most likely impact on a local or regional rather than a national basis. The risk of these diseases is particularly high among people who consume drinking water from private group water schemes, using vulnerable ground water unless remedial action is undertaken. In addition, there is a risk of outbreaks of such waterborne diseases in people who consume water from public supplies where water treatment plants are not functioning adequately, or where monitoring and surveillance is inadequate. If the Irish population continues to concentrate on the East

coast, current water distribution systems will be under pressure to cope with the demand of clean potable water, necessary for the maintenance of good health. Further research into the climate-water related illness relationship will be possible in the future, as the data that will now be collected has been significantly increased since 1st January 2004, and will enable more detailed analysis of the relationship between incidence and meteorological factors. Ireland with enhanced data sources from 2004 should be well positioned to undertake future research on the relationship between climate and waterborne diseases. The successful implementation of the Drinking Water Regulations and the European Water Framework Directives target of good water status by 2015, will ensure that the quality of Irish waters is sufficient to ensure the maintenance of good health, although major programmes will be required to in order to reach the required quality. Water is a fundamental requirement for life on earth, and has been described as 'arguably the most precious of all substances' (EPA, 2001). It is perhaps also the most unappreciated substance (Leder *et al,* 2002). In the words of Dr. P. Wall former CEO of the Food Safety Authority of Ireland

'*Safe water cannot be taken for granted*'

(Wall, 2001)

CHAPTER 7 - CLIMATE CHANGE AND FOOD-BORNE DISEASE

7.1 Introduction

The availability of safe food in an adequate supply is vital for the maintenance of good health. While climate variability may impact on agricultural productivity, the availability of food relates more to economic than agricultural issues. This results in poorer countries being more vulnerable to the impacts of climate change. Today, hunger and poverty already claim 25,000 lives a day, and 852 million people do not have enough to eat (World Food Programme, 2006), in a world with the capacity to provide sufficient food for all (FAO, 2002). The incidence of food-borne disease is also expected to increase, as a result of climate change (AR4, 2007). Although fatalities from food-poisoning are extremely rare in Ireland, the prevalence of gastrointestinal illness is high; almost 5% of respondents in a national survey reported suffering from acute gastroenteritis in a four week period (FSPB, 2003). The possibility for an increase in the incidence of food-borne disease in Ireland therefore clearly exists.

The impact of the projected changes in temperature in Ireland on the future incidence of food-borne disease is the subject of this chapter. The approach will be as follows: the impact of climate change on food supply and food-borne disease will be discussed, followed by a discussion of methodological issues relating to the present study. The present impact of temperature on the incidence of food-borne illness in Ireland will then be ascertained, and following this, future estimates of the impact of changes in temperature related to climate change in Ireland will be quantified.

7.2 Impacts of climate change on food production

The world's dominant food commodity is cereal, and at high and mid-latitudes, climate change is predicted to increase yields of cereal, while in contrast, outputs will decrease at lower latitudes (AR4, 2007). The reduction in cereal production in these predominantly poorer countries has been estimated at approximately 10% (FAO, 2003). A projected 2 – 3% reduction in African cereal production for 2020 is enough to put 10 million people at risk (FAO, 2003). In addition, subsistence farming is also more

common at lower latitudes, and increased heat stress will impact on both crop outputs and livestock.

In Ireland, while it is expected that there will be major changes in the types of crops grown, for example, maize will become a major crop, and potatoes will require irrigation schemes to remain commercially viable, no catastrophic effects are expected (Sweeney *et al,* 2003). As the overall availability of food is not expected to reduce, this chapter will therefore investigate the impact of climate change on food-borne disease in Ireland.

7.3 Impacts of climate change on food-borne disease

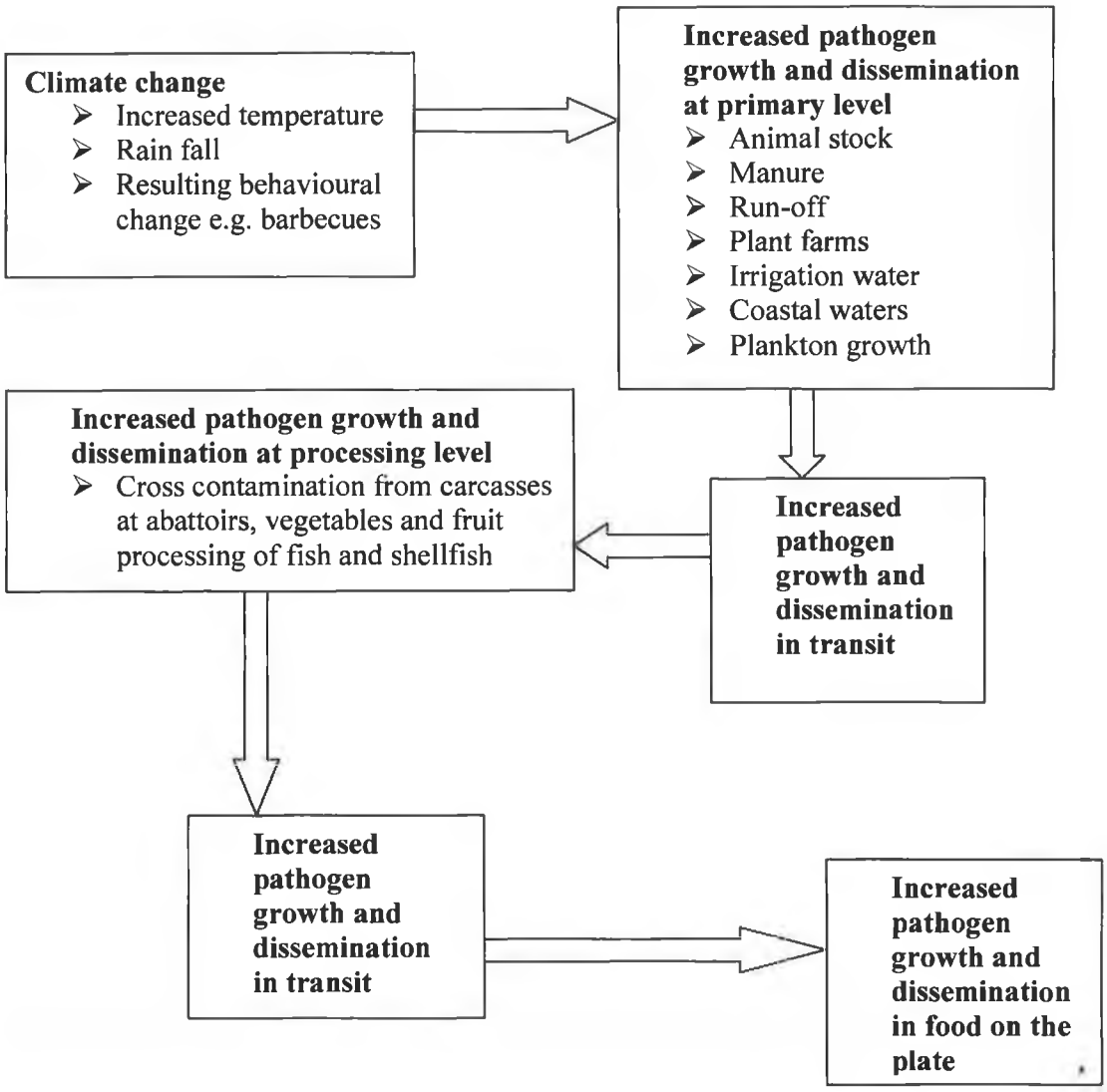
7.3.1 Food-borne disease

In this context, food-borne disease will be defined as illness caused by the consumption of food, contaminated with infectious material. Although food and water-borne diarrhoeal diseases are leading causes of death and disability in poorer countries, they are also a problem in richer countries. The symptoms of food-borne disease range from mild gastro-enteritis, not requiring medical treatment to life-threatening neurological, hepatic and renal syndromes (Mead *et al,* 1999). Aside from personal distress, food-borne disease also causes an avoidable economic burden. Gastroenteritis has been estimated to result in approximately 1.5 million working days being lost annually, with an estimated loss of 173.5 million euros in earnings (FSPB, 2003). The percentage of people affected each year in industrialized countries by food-borne disease has been reported to be up to 30% (WHO, 2002 (a)), in addition, long-term problems have been estimated to occur in between 2-3% of all cases of food-borne disease (WHO 2003 (c)). Every person is at risk of food-borne disease and it can cause fatalities in people who are vulnerable, e.g. the elderly, the very young, those who are ill and people whose immune systems are compromised.

7.3.2 Food-borne disease and climate change

Similar to most micro organisms, the major pathogens responsible for acute gastroenteritis multiply more rapidly in warmer conditions (Acacia, 2000), and higher temperatures are often associated with an increase in gastrointestinal infections (AR4, 2007; Kovats, 2004 (b)). Corresponding to an increase in ambient temperatures, a

seasonal pattern is frequently observed with a peak in cases of food poisoning during the summer months (cCASHh, 2005). In addition, warmer weather is associated with an increase in the number of barbecues which have been associated with food poisoning (de Jong *et al,* 2004; Neimann *et al,* 2003), and also with a trend towards dietary items such as lettuce and raw fruit that are associated with increased risk of disease (Bentham, 2001(b)). A warmer climate may also contribute to an increase or change in the distribution of carriers of food-borne disease, such as flies (Rogers *et al*, 2001). In addition, the number of bacteria in animal feed may also increase with higher temperatures (Bentham, 2001(b)). Heavier rainfall, resulting from the impacts of climate change, will increase surface water run-off, and the risk of contamination of potable water, and resultant contamination of food (AR4, 2007). The diversity of the determinants of food-borne disease in Figure 7.1 illustrates the complexity of the 'causal web' of disease (Section 3.2), and the many ways by which climate change may impact on the incidence of food-borne disease have been outlined (Figure 7.1).



(*after Hall et al, 2002)*

Figure 7.1 How climate change may affect the food production chain

Studies between gastro-intestinal illness and mean temperatures have confirmed a relationship in poorer countries of the world. For each 1^{0}C rise in temperature, incremental increases in cases of diarrhoea of 3% have been recorded in Fiji (Singh *et al,* 2001) and 8% in Lima (Checkley *et al,* 2000) respectively. This relationship has also been noted in western countries and quantitative analysis of the temperature-morbidity relationship has shown that mean monthly temperature explained almost three quarters of the variance in the food poisoning notifications in the UK (Bentham and

Langford, 1995). If current warming trends continue, additional cases of food-borne disease may therefore be expected (Bentham and Langford, 1995; Githeko and Woodward, 2003; WHO, 2003(b)), and in particular, an increase in the number of cases of salmonella (Kovats 2003(b)).

7.4 Future national estimates of impact of climate change on food-borne illness

Both quantitative and qualitative national assessments of the impact of climate change on food-borne disease have been identified. The national assessments of Portugal and the United States both acknowledged that higher future temperatures may increase food poisoning, but did not quantify the possible increase (Santos *et al,* 2002; Rose *et al,* 2001). Two quantitative national assessments were identified, in the UK (Bentham, 2001) and in Australia (Mc Michael *et al,* 2002), each of which estimated future increases in the incidence of food-borne disease. The approach used in both of these assessments, was an analogue approach, where the present impact of temperature on food-borne illness was estimated, and this relationship was then used with the temperature outputs from scenario driven climate change models. In this way, future estimates of the incidence of food poisoning were produced for the UK, for the years 2010, 2030 and 2050 (Bentham, 2001). With corresponding projected increases of 1^0C, 2^0C and 3^0C in mean temperatures, estimates of notifications of food poisoning increased by 4.5%, 9.5% and 14.8% respectively, giving rise to absolute increases of 4,000, 9,000 and 14,000 cases per year respectively. In the vulnerable aboriginal community, similar estimates were reported, and increases in mean temperature of between 0.5^0C to 1^0C by 2020, and 1^0C to 3.5^0C by 2050 were associated with a 3-5% increase and a 5-18% increase in diarrhoeal cases requiring hospital admission respectively (Mc Michael *et al,* 2002). No study identified undertook a quantitative assessment of specific food-borne illnesses. Although one previous study has been identified in the literature which quantified the changes in the future incidence of food-borne disease, arising from climate change, this study was confined to food-borne diseases from all microbiological causes (Langford and Bentham, 1995), and reported estimated increases of between 2-8% in the early part of this century, increasing to 5 to 24% by 2050. The increases estimated in the present study for salmonella are lower than this, but the results for both campylobacter and VTEC 0157 by mid century are

comparable. However, the temperature morbidity relationship for different food-borne pathogens needs to be studied separately (D'Souza, 2004).

7.5 Methodological considerations

Not all microbiological causes of food-borne disease are suitable for monitoring the impacts of climate change (WHO 2001(a)). To detect an impact, large numbers of people would need to be affected and therefore rare diseases such as hepatitis A are unsuitable. In addition, some diseases, for example, shigella, are primarily spread by person to person, thus leading to difficulties in assessing the impact of temperature changes on the incidence. Finally, diseases whose incidence is not reliably recorded, such as viral causes of food-borne disease, are also unsuitable. Using such criteria, three infections have been recommended as the most suitable illnesses for monitoring the impacts of climate change in Europe, namely salmonella, campylobacter and cryptosporidiosis (WHO 2001(a)). Salmonella and campylobacter are the commonest bacterial causes of gastroenteritis in Ireland (HSPC 2005(a); HPSC, 2006(b)) and will be discussed here, along with E.Coli 0157 because of the serious nature of the illness. Viral gastroenteritis is the commonest cause of gastroenteritis in Ireland (NDSC, 2003), but is generally self-limiting. The impact of climate change on the future incidence of cryptosporidiosis is discussed in chapter 6.

To ascertain the future incidence of specific food-borne disease in Ireland, it is first necessary to ascertain the present incidence. From this, a model of the present temperature- morbidity relationship may be established, and used to establish future morbidity rates. However, prior to establishing the incidence, there are two issues that need to be addressed. Firstly, there is a problem in the ascertainment of the true incidence of food-borne illness. A second issue relates to factors that may distort the temporal relationship between the morbidity data and the recorded date of onset. These issues will be discussed briefly.

7.5.1 Problems in ascertaining the true incidence of food-borne disease

It is difficult to ascertain the true incidence of food-borne disease; data from both developed and developing countries on the extent of morbidity and mortality from food-borne disease are very incomplete and underestimate the extent of the problem (WHO,

2003(a)). The problems of ascertainment arise from several factors. In general, surveillance systems are passive, and consequently many cases go unreported. In addition, symptoms of food-borne disease such as diarrhoea may be regarded as a transitory inconvenience and not a symptom of disease ((Rose *et al,* 2001; Mead *et al,* 1999; WHO 2003(a)). Furthermore, the cause of the illness may not be known to be related to food, or the causative organism may not yet have been identified – campylobacter and VTEC were not recognized as causative agents of food-borne disease 25 years ago. Overall, it is estimated that in industrialized countries, less than 10% of cases of food-borne illness are reported and that fewer are investigated (WHO, 2003(a)). As Rose notes, commenting on the situation in the United States (Rose *et al,* 2001) 'with current surveillance programmes, even an outbreak resulting in many medically attended illnesses could be unrecognised'. In France, routine mandatory reporting of food-borne salmonella outbreaks is very incomplete (Gallay *et al,* 2000) and in Australia 'probably as few as 5-10% of people with salmonella have a positive stool sample reported to surveillance' (Hall *et al,* 2002). In Europe, surveillance for viral gastroenteritis is poorly developed (Lopman *et al,* 2003). Three major investigations, undertaken in the UK, the USA and Ireland confirm the inadequacies of surveillance systems that have been used. In the UK, although infectious intestinal disease occurs in 1 in 5 people each year, only 1 in 6 of people so affected present to a general practitioner (the ratio was understandably higher for less serious diseases such as viral causes and lower for more serious diseases such as VTEC). Less than a third of salmonella cases were reported to national surveillance and overall, only 0.7% of infectious intestinal diseases were reported (Wheeler *et al,* 1999). An even higher degree of under-reporting was found in the United States, where the incidence of salmonella and campylobacter was 38 times the reported number, and for serious illnesses such as E. Coli 057, it was 20 times the reported number (Mead *et al,* 1999). In common with other countries therefore, it is likely that under reporting of infectious diseases also occurs in Ireland. Information collected on risk factors for sporadic cases of food poisoning in Ireland had been described as inadequate (FSAI, 2002(a)), and the system of surveillance of general enteric disease outbreaks in Ireland as 'in its infancy' (WHO, 2000). However, evidence of the prevalence of food-borne illness was borne out by the results of a national survey; almost 5% of respondents reported suffering from acute gastroenteritis in a four week period (FSPB, 2003). Interestingly, of those who had been ill in this time, less than a third attended their general practitioner, and in this sub-

group, 9% were asked to submit a stool sample for testing. Three-quarters of these did so. Overall, the study found that for every 100 persons in the community with acute gastroenteritis in Ireland, 2 stool samples are submitted for laboratory testing. Although the study did not examine in detail the causes of the gastroenteritis, when asked what they thought might have caused their illness, over half of the respondents did not know, while almost a quarter suspected their illness was due to consuming contaminated food or water. The problems associated with under-reporting of disease have been graphically illustrated (Figure 7.2).

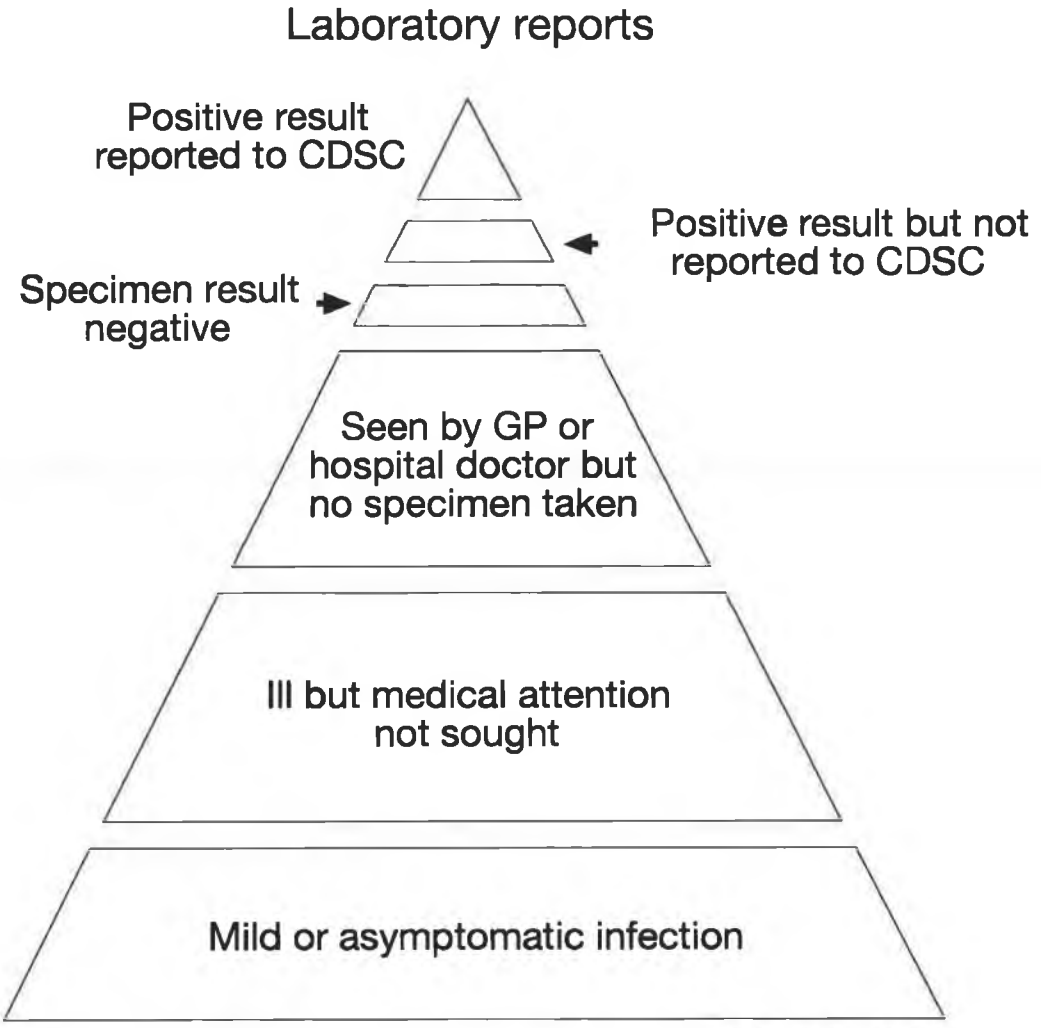


Figure 7.2 Notified cases to CDSC (Communicable Diseases Surveillance Centre) represent only a proportion of morbidity caused by food-poisoning

The surveillance of food-borne illness in Ireland has improved since the introduction of new infectious diseases legislation in 2004. Up to that year, data on food-borne illness in Ireland was limited, and such data was recorded under only three headings: salmonella, food poisoning other than salmonella, and gastroenteritis in children less than 2 years. Many more infectious diseases are now statutorily notifiable and demographic and other ancillary information is also collected. As a result, data on infectious intestinal disease may be expected to be of a very high standard in the future.

7.5.2 Problems in ascertaining the temperature-morbidity relationship

There are three specific issues relating to data on the incidence of food-borne disease that may distort the relationship between the temperature and the onset of illness. Firstly, the date of recording of the illness may not be the date of onset of the illness. Secondly, there may be a lag time between the onset of the illness and current temperature, representing the time taken between the contamination of the food and the development of symptoms. This time interval indicates the time taken for the contaminated food to reach the consumer and cause illness (Bentham and Langford, 2001). Finally, particularly in the case of salmonella, outbreaks of disease may occur, during which the incidence rate is determined primarily by person to person spread and not by temperature, thus distorting the illness/temperature relationship.

7.6 Food-borne disease related to climate change in Ireland

Notwithstanding these difficulties, it is still necessary to estimate the changes in the future incidence of specific food-borne diseases, namely salmonella, campylobacter and VTEC in Ireland that may result from climate change. These diseases will now be discussed and their link with temperature ascertained.

7.6.1 Salmonella

Salmonella is a bacterial infection, presenting with acute abdominal pain, diarrhoea, nausea, occasionally vomiting and headache. The bacteria are killed by cooking. Mortality is rare, except in the very young, the old and those who are debilitated; the hospitalization rate and case fatality rate from salmonella being estimated at 0.2 and

0.008 respectively (Mead *et al,* 1999). It is transmitted by the ingestion of food from infected animals, principally chickens and eggs, although milk, meat and meat products, including pork may also carry the disease (Leonard, 2004). In Ireland, eggs are the food most commonly implicated, followed by chicken, steak and desserts (Fitzgerald *et al,* 2001). There are two main types of Salmonella: S.Enteriditis (the more common type) and S. Typhymurium. The former is found almost exclusively in poultry and eggs and appears to be more sensitive to temperature than S.Typhimurium which is more commonly associated with meat (Kovats *et al,* 2004 (b)). In Ireland, the incidence of salmonella is highest in young children, and in 2006, over 20% of cases were in children under 5; however, this may reflect the fact that more samples are taken from this age group (HPSC, 2007(c)).

Salmonella multiplies at room temperature (Baird-Parker, 1993), and temperature has been found to influence transmission of the disease in approximately a third of cases (Kovats *et al,* 2004(b)). Country specific threshold temperatures have been identified in some European countries above which a linear association between temperature and the number of cases of salmonella has been found (Table 7.1). Interestingly, the varying threshold temperatures noted in European countries are not correlated with latitude.

Country	Threshold temperature ^{0}C	% change in incidence per degree ^{0}C rise above threshold temperature
Poland	6	8.7
Scotland	3	4.7
Denmark	15	1.1
England and Wales	5	12.4
Estonia	13	18.3
The Netherlands	7	9.3
Czech Republic	-2	9.5
Switzerland	3	8.8
Slovak Republic	6	2.5
Spain	6	4.9

(after Kovats *et al,* 2004(b))

Table 7.1 Temperature thresholds for salmonella

While no quantification of the incidence of salmonella in relation to the temperature threshold in Ireland was identified, a seasonal peak in the incidence of salmonella has been noted. It typically occurs in late August or early September (HPSC, 2005(a); HPSC, 2006(b). An increased incidence of salmonella could therefore be expected

during warmer summer weather (Bentham, 1995; WHO, 2000; Hall *et al,* 2002; Thornley and Nicol, 2002; Kovats *et al,* 2004(b); D'Souza *et al,* 2004). A lag effect in salmonella infections has also been identified, where the temperature peak precedes the incidence peak, and indicating that the infection of the food may have occurred earlier in the food production process (Kovats *et al,* 2004(b); Fleury *et al,* 2006). Other meteorological variables such as relative humidity are not regarded as having any relationship with the incidence of salmonella (Kovats *et al,* 2004 (b)).

7.6.2 Campylobacter

Campylobacter is an acute bacterial disease, causing diarrhoea, sometimes with blood, abdominal pain, nausea and vomiting, and fever, although the illness may also be asymptomatic. The illness generally lasts between 2-5 days, but may be protracted. Treatment is generally symptomatic and antibiotics are required only if the disease is very severe or if the patient has impaired immunity. The hospitalization rate and case fatality rate for campylobacter are lower than for salmonella, and have been calculated as 0.1 and 0.001 respectively (Mead *et al,* 1999). Relapse occurs in 10 to 20% of patients (FSAI, 2002) but is usually less severe than the original episode of illness. In 2 to 20% of cases the infection may lead to chronic health problems, some of which may be serious (WHO, 2002 (a)). They include Guillain-Barré Syndrome, and arthritis, although a typhoid like illness and rarely febrile convulsions and meningitis may also occur (WHO, 2003(c)).

The bacteria are widespread in the intestinal tract of farm animals, poultry, pets and wild birds. The foods and drinks most commonly associated with outbreaks of campylobacter include unpasteurized milk, untreated surface water, and poultry and meat food products (Pebody *et al,* 1997; FSAI, 2002 (a)). An outbreak of campylobacter infection has also been associated with failed pasteurization techniques in the UK (Fahey *et al,* 1995) and with bottled mineral water in Wales (Evans *et al,* 2003).

Unlike salmonella, the bacteria are fragile. They need temperatures above 30°C for growth to take place, and prefer a low oxygen environment, and are therefore well-suited to the intestinal tracts of warm-blooded animals (FSAI, 2002(a)). These requirements tend to prevent their multiplication in food. Most cases of campylobacter are therefore sporadic and person to person transmission is uncommon, although may occur in children (FSAI, 2002(a)). As a consequence,

outbreaks tend to be rare, although large amounts of food may be initially contaminated, as in the case of poultry products, where the food is infected at source (Pebody *et al*, 1997). However, outbreaks might be more common than suspected, since a high proportion of cases reported other illness in the home or in the community at the same time as their illness (Gillespie *et al,* 2003).

While a seasonal pattern in the incidence of campylobacter is evident, unlike the impact of temperature on the incidence of salmonella, the impact of ambient temperature on the incidence of campylobacter is not as clear cut. Nevertheless, campylobacter transmission has a seasonal pattern of transmission, with peaks tending to occur later in the year at more northern latitudes (Meldrum *et al*, 2004 (b)). Peaks have been noted in late June and early July in Scotland (Miller *et al,* 2004), and in June in Wales (Meldrum *et al*, 2004(a)) and Ireland (HPSC 2006(b)). Rainfall and humidity are not regarded as having any relationship with the incidence of campylobacter (Kovats *et al,* 2005).

7.6.3 Verocytogenic E Coli (VTEC)

Although E. Coli is generally a common and beneficial bacterium in the gut, playing a role in digestion, a pathogenic type of this bacterium also exists, Verocytogenic E. Coli or VTEC. This bacterium causes a serious form of food-poisoning. It produces a toxin, the most common type being O157, although other types have now been identified and they come under the general term Verocytogenic E Coli, or VTEC. The symptoms range from mild diarrhoea to a haemorrhagic colitis, with severe abdominal pain and bloody diarrhoea. While the illness is usually self limiting, it progresses in 2-7% of patients to the condition of haemolytic-uraemic syndrome (HUS), after approximately 8 days. One third of people who suffer from HUS continue to have long term problems, including possible paralysis, seizures, and ongoing renal problems. The case fatality rate for VTEC is between 3% to 5%. A higher incidence is noted in children under the age of 5, reflecting the more extensive investigations undertaken in this age group (HPSC, 2006(b)).

VTEC may be carried by cattle, sheep, horses, and birds, and may be spread by multiple routes, including through food and water, by animals and from person to person (Garvey *et al*, 2006). Higher temperatures have been found to lengthen the survival of E

Coli in contaminated food (Black, *et al,* 1995), and a seasonal component to the incidence of VTEC in Ireland, has been found, with the incidence peaking in late summer (HPSC, 2005(b)). Although the incidence of VTEC is higher in summer months, higher levels of the bacteria are found in cattle in winter. This may be related both to the increased exposure of the population to pastures in summer time and increased ambient temperature (Ogden *et al,* 2004).

7.7 Future estimation of incidence of food-borne diseases in Ireland

7.7.1. Data sources

In all instances, data on the incidence of illness were obtained from the National Disease Surveillance Centre, now the Health Protection Surveillance Centre. However, as mentioned earlier (Section 7.5.1), data on food-borne illness in Ireland is limited up to 2004, and up until that year, only one specific food-borne disease, namely salmonella was notifiable. However from that year, many more infectious diseases became statutorily notifiable, and for 2004 and 2005, weekly data on the incidence of campylobacter were also obtained. Although all serotypes of VTEC became legally notifiable in 2004, the incidence of serotype VTEC 0157 and campylobacter while not statutorily notifiable until 2004 came under a system of voluntary surveillance since 1999. Under this system, laboratories and hospital personnel voluntarily notified cases and records of the incidence may be precise (Garvey, 2006). Monthly data on the incidence of VTEC 0157 was therefore obtained for the years 1999-2005. VTEC 0157 is a rare but serious disease and it may be presumed that almost all cases are notified. On the contrary, campylobacter, is a much more common infection, but is generally not considered a serious disease, and is rarely fatal. Therefore all cases may not have been notified prior to the enactment of the new legislation. Because its relationship with temperature is not straightforward, it was deemed more appropriate to use the weekly data, available since 2004 in order to elucidate the temperature relationship with this organism.

Population figures for census years and estimates for populations for the intervening years were obtained from the Public Health Information systems of the Department of Health and rates per 100,000 population obtained for salmonella. For campylobacter and VTEC, the numbers affected were low and therefore only the numbers of cases

were assessed. Temperature data for the time periods when the food-borne diseases occurred was obtained from Met Eireann. Mean temperatures for the future time periods 2010-39, 2040-69 and 2070-99 for Ireland were obtained as discussed in Section 2.8.

7.7.2 Methodology

The approach used was similar to that used in the estimation of the changes in temperature related mortality (outlined in section 5.2.1). Initially the present temperature-food-borne illness morbidity relationship was established. Temperature outputs from the three global climate models, each driven by two scenarios A2 and B2 were obtained and these were incorporated into the temperature-morbidity relationship model established. Six temperature-related morbidity outputs were therefore established for the baseline scenario, 1961-1990 and for the three future time periods, 2010 to 2039, 2040 to 2069 and 2070 to 2099. The output from the baseline scenario was subtracted from the output of the future scenarios, to reduce systematic errors introduced by the modelling process. The differences in morbidity rates are ascribed to the impacts of climate change. The results are presented as a range and a mean of the data. Salmonella (and to a lesser extent VTEC) are associated with outbreaks. In an outbreak, the spread of disease is largely determined by contact, and not by temperature. Two methods were identified, by which the impacts of outbreaks may be removed. Firstly, the weekly incidence data may be confined to weeks where the weekly incidence rates were below the mean plus two standard deviations of the rate (D'Souza *et al,* 2004). A second method has also been used, namely weeks were excluded from the calculations, when outbreaks were known to have occurred (Kovats *et al,* 2004(b)).

7.7.3 Description of the data

Of the three food-borne diseases, salmonella, campylobacter and VTEC, campylobacter was the most common, causing an average of 34 cases a week. However the propensity of salmonella to cause outbreaks resulted in salmonella causing the maximum number of cases (214) in one week. VTEC 0157 is the least common, but the most serious resulting in an average of 5 cases a month (Table 7.2).

	Mean	Maximum	Minimum
Salmonella per week (1988-2004)	11	214	0
Campylobacter per week (2004-2005)	34	66	8
VTEC 0157 per month (1999-2005)	6	30	0

Table 7.2 Incidence of food-borne diseases

A decline in the incidence of salmonella since the late 1990s is evident (Figure 7.3(a)).

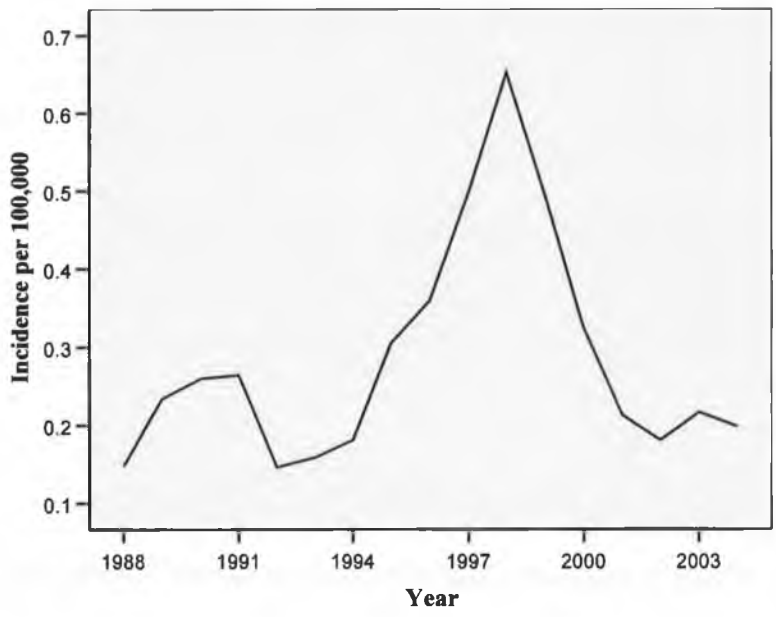


Figure 7.3 (a) Mean yearly rate of salmonella per 100,000 population 1982-2004

The majority of the rise in the incidence of salmonella since 1996 has been attributed to surveillance artefact (Garvey, 2004).

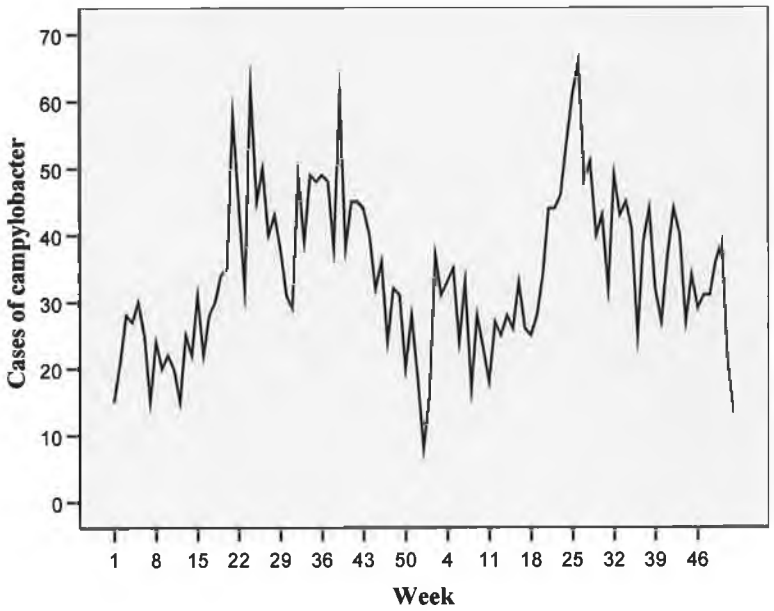


Figure 7.3 (b) Weekly cases of campylobacter 2004-2005

The rate of campylobacter infection in Ireland was 43.7 per 100,000 in 2004, and 46 per 100,000 in 2005 (HPSC, 2005(a) and 2006(b)) (Figure 7.3 (b)). No major change in trend over time is evident in the incidence of VTEC 0157 Figures 7.3 (c) although there is evidence of an outbreak of VTEC in 2005.

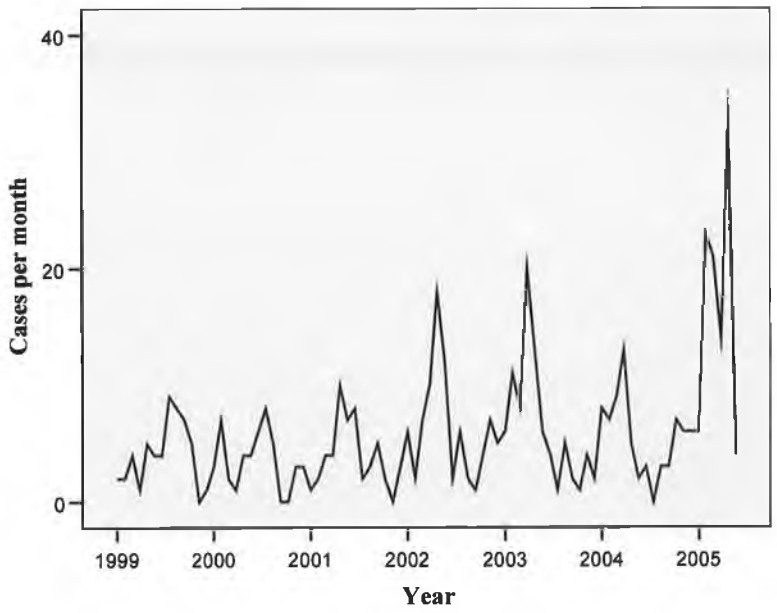


Figure 7.3 (c) Monthly cases of VTEC 0157 1999-2005

This was the largest out break of VTEC to-date, and the rate in that year was 2.8 per 100,000, a significant increase over the mean rate of 1.9 per 100,000 in the time period.

7.7.4 Analysis

Prior to an analysis of the data, it was necessary to both remove the impacts of outbreaks where possible, as these cases are not directly related to temperature, and also to ascertain the presence of a lag period.

Outbreaks

An outbreak occurs when the number of cases exceeds the expected number (HPSC, 2008). Salmonella, unlike campylobacter is particularly associated with outbreaks. Cases associated with outbreaks may not necessarily be associated with temperature, as they may be spread by direct person to person contact, and the incidence may be more closely related to social factors than to temperature. Therefore, it was necessary to allow for the impact of outbreaks on the incidence data (Section 7.7.2). As outbreaks of infectious diseases were not statutorily notifiable until 2004, the dates or locations of all outbreaks were not known. To reduce the impact of such cases on the temperature-morbidity relationship, such cases were excluded from the data when the incidence rate rose above the mean and two standard deviations of the incidence data (D'Souza *et al,* 2004). In the case of salmonella, the mean and the standard deviation of the data were 0.284 cases and 0.325 cases per 100,000 respectively. Data was therefore analysed when the number of cases was less than or equal to 0.93 cases per 100,000 per week (i.e. the mean and two standard deviations). This entailed the loss of 3% of the data (Figure 7.4(b). Reports on known outbreaks was also obtained (Fitzgerald *et al*, 2001; Foley, 2005; HPSC, 2004), and this method incurred the loss of 1% of the data (Figure 7.4 (c)).

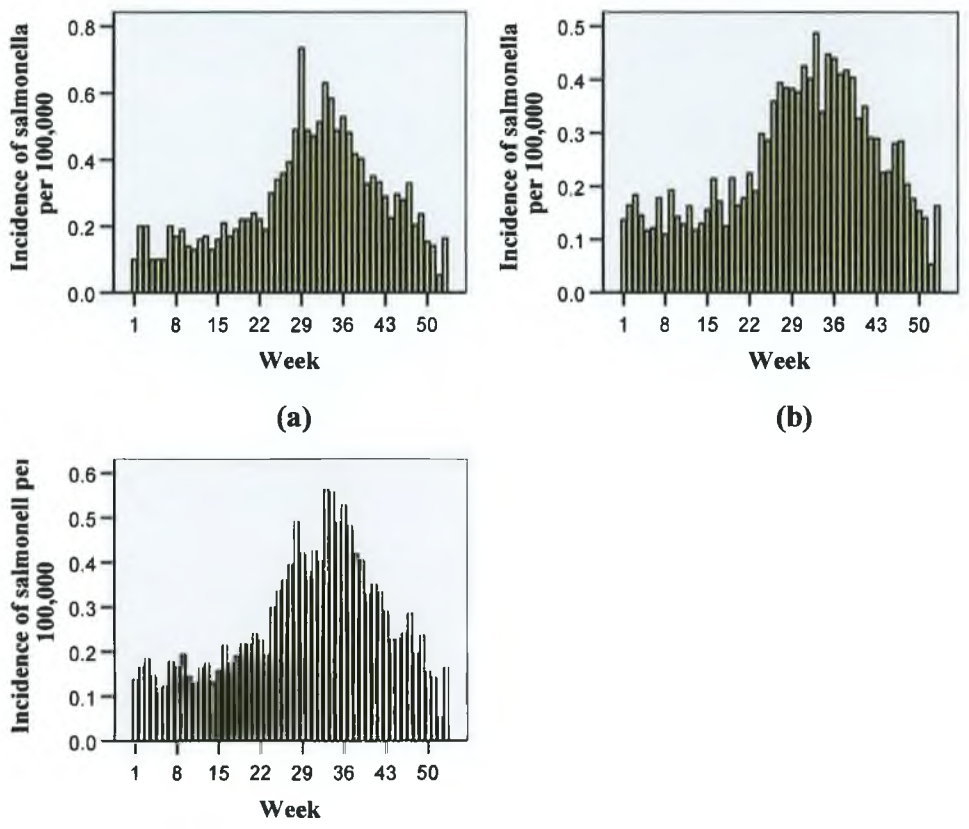
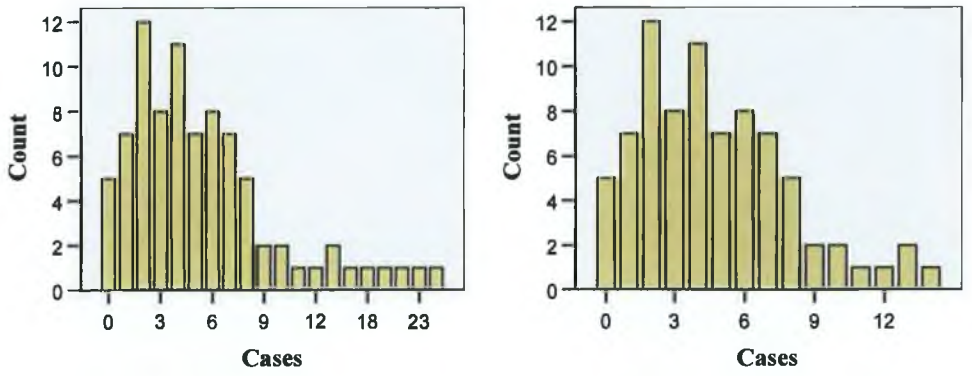


Figure 7.4 Frequency distribution of weekly incidence of salmonella cases 1988-2004 before Figure 7.4 (a) and after adjustment for outbreaks using the D'Souza method (b) (D'Souza *et al, 2004*), and the Kovats method (c) (Kovats *et al*, 2004(b))

The method used by D'Souza for accounting for outbreaks was adopted, as the extreme values obtained were less using this method (Figures 7.4(a) to (c)). In the same way, the data on VTEC 0157 was adjusted for the impact of outbreaks (Figures 7.5 (a) and (b)). The mean of the monthly cases of VTEC 0157 was 5.79 and the standard deviation was 5.48. Therefore, cases of VTEC 0157 when the incidence was greater than 16.75 cases per month were also removed from the data set. This incurred the loss of 6% of the data.



(a) (b)

Figure 7.5 Frequency distribution of monthly incidence of VTEC cases 1999-2005 before (a) and after (b) adjustment for outbreaks after D'Souza (D'Souza *et al, 2004*)

The changes to the data incurred by the adjustment are evident (Table 7.3).

	No. of cases	Mean	Maximum	Minimum
Salmonella per 100,000 per week	855 (885)	0.25 (0.28)	0.93 (5.78)	0
VTEC 0157 per month	79 (84)	5 (6)	14 (30)	0

Table 7.3 Mean monthly numbers of cases of salmonella per 100,000 and VTEC following adjustments for outbreaks (unadjusted data in brackets)

Lag periods

The possibility of a lag period was investigated, in an iterative process, by serial regression of the incidence data, lagged by successive weeks with respect to mean temperature. As the shape of the salmonella-temperature relationship is generally regarded as quadratic, such a model was used. The R-square value was highest with no lag period with respect to salmonella (Table 7.4).

Weeks lagged	R square
0	0.204
1	0.184
2	0.119
3	0.120
4	0.116
5	0.114
6	0.092

All significant p<.001

Table 7.4 Association between weeks lagged and incidence of salmonella

In contrast to salmonella, the temperature-campylobacter relationship is not straightforward, possibly as a result of its spread via multiple environmental factors, and its unusual temperature requirements. No lag period was observed with linear and quadratic models, but a 1 week lag period was noted, using a cubic model (Table 7.5).

Weeks lagged	R square Linear	R square Quadratic	R square Cubic
0	0.442	0.443	0.456
1	0.434	0.438	0.463
2	0.406	0.415	0.433
3	0.376	0.408	0.449
4	0.370	0.398	0.408
5	0.307	0.324	0.330
6	0.225	0.229	0.236

All significant p<.001

Table 7.5 Association between weeks lagged and incidence of campylobacter

In relation to VTEC 0157, the VTEC 0157 -temperature relationship retained statistical significance until the second month and no lag period was noted (Table 7.6).

Months lagged	R square Linear	R square Quadratic
0	0.349	0.221
1	0.194	0.221
2	0.265	0.287

All significant p<.001

Table 7.6 Association between months lagged and incidence of VTEC 0157

With the adjusted data, a summer-time increase in incidence is evident in all three food-borne diseases (Figures 7.6(a)-(c)).

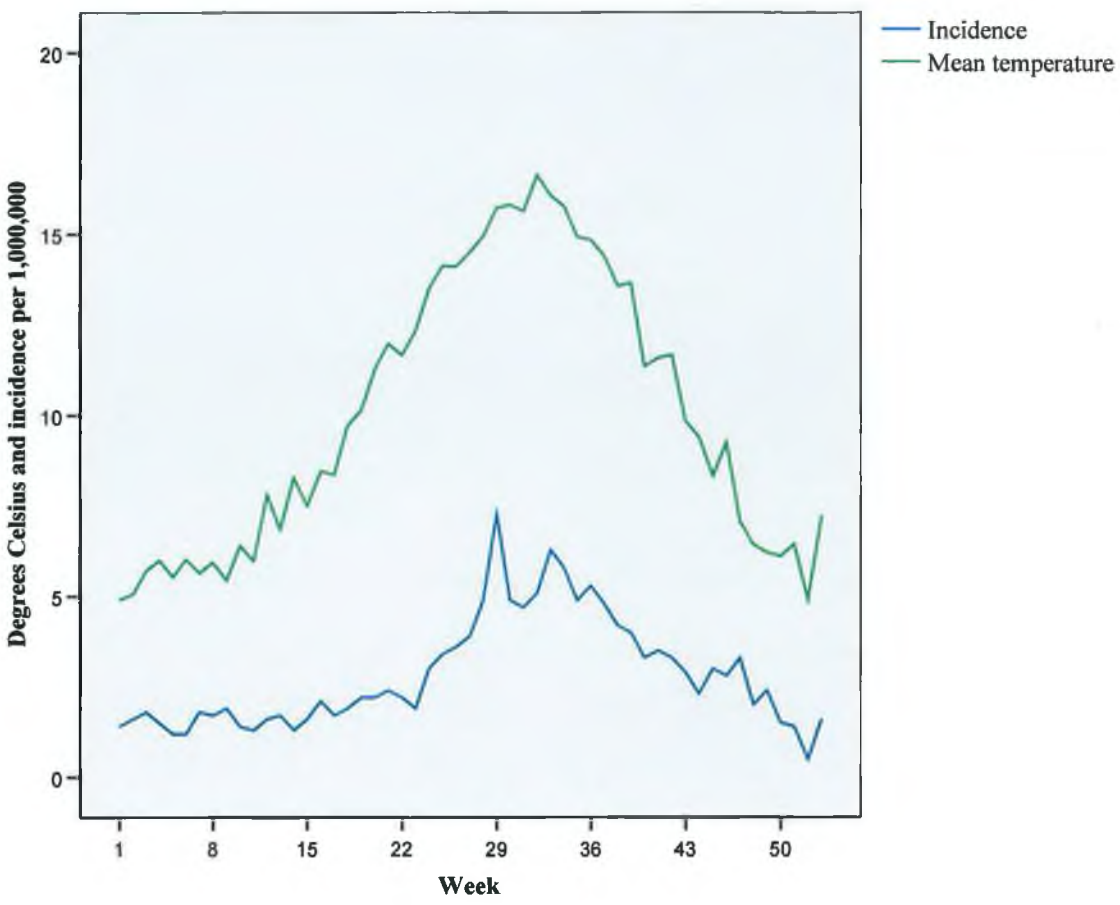


Figure 7.6 (a) Weekly incidence of salmonella per 100,000 population and mean temperature 1988-2004

Over a half (51%) of all cases of salmonella occurred in the months of June July, August and September, with the peak in July (Fig 7.6(a)).

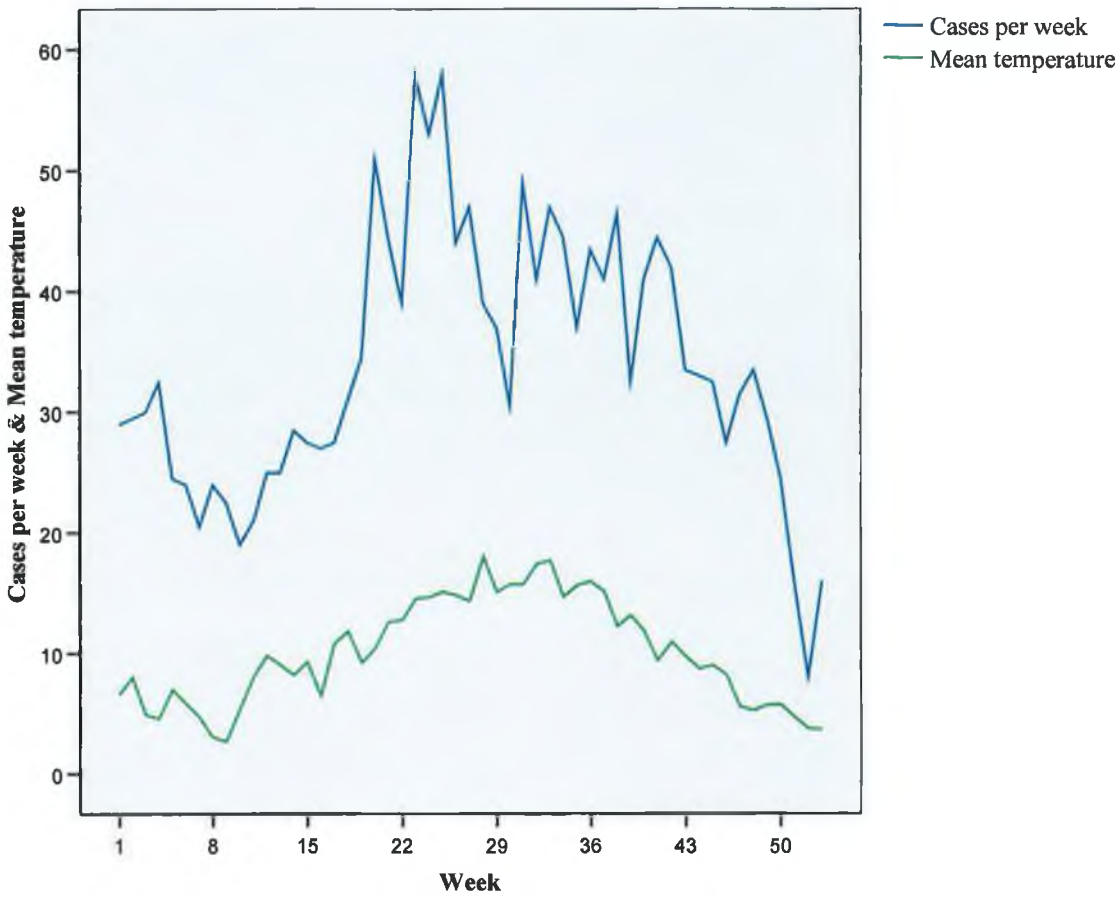


Figure 7.6(b) Weekly incidence of campylobacter and mean temperature 2004-5

A similar mid-year rise is also evident between mean temperatures and campylobacter, where 40% of the cases occur in June, July, August and September (Figure 7.6(b)).

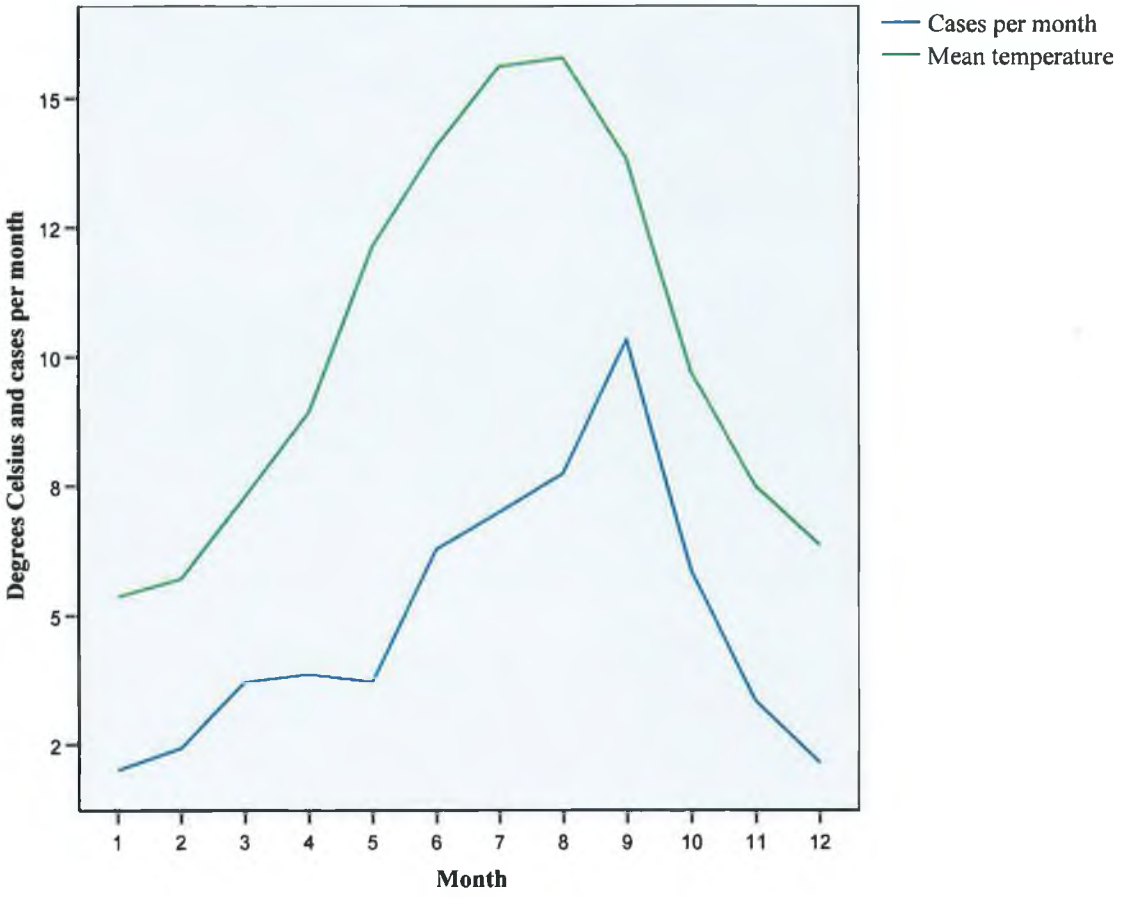


Figure 7.6(c) Monthly incidence of VTEC 0157 1999-2005 and mean temperature 1999-2005

The peak in the incidence of VTEC occurs in late summer, and 54% of cases arise in the June to September period. (Figure 7.6(c)).

7.8 Results

The incidence of salmonella, campylobacter and VTEC 0157 and mean temperature per 1 degree band of mean temperature was determined, using lagged data where a lag effect was noted.

7.8.1 Salmonella

The mean temperature-salmonella relationship is generally accepted as quadratic, and this was observed (Figure 7.7 (a)).

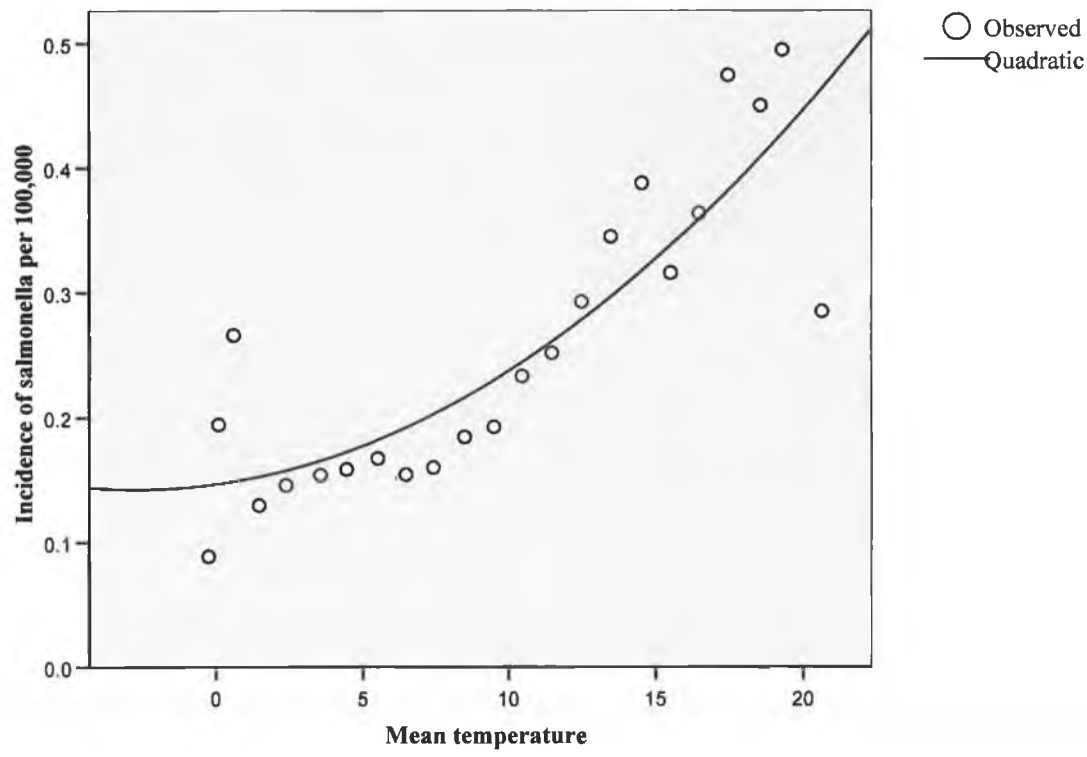


Figure 7.7 (a) Mean temperature and weekly incidence of salmonella 1988-2004

In the case of salmonella, the equation was in the format

$y = a\,(t)^0 + b(t)^1 + c(t)^2$ i.e.

$y = 0.147 + 0.0032\,(t)^1 + 0.001\,(t)^2$

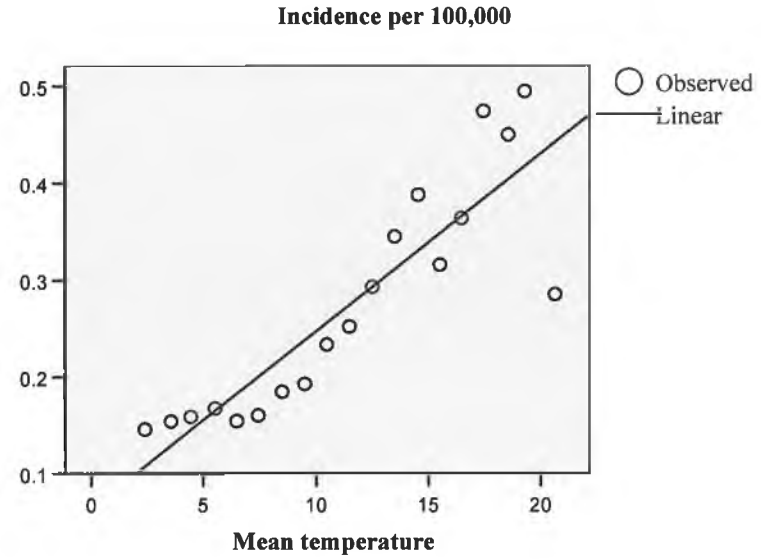
$p<0.001$, R sq = 0.732.

Differentiating the equation allows the establishment of the equation representing the line where the slope is zero, and thereby allows the calculation of the threshold temperature above which the incidence of salmonella increases.

The threshold temperature[1] was estimated to be 2.1^0C.

[1] To achieve a more accurate estimation, the incidence variable was multiplied by 100 and the formula $14.784 + 0.257\,(t)^1 + 0.062\,(t)^2$ was used

Above this temperature, a linear relationship is evident ($p<0.001$), while below this temperature, no significant relationship was evident.



R square0.777 p<0.001

Figure 7.7 (b) Mean temperature above 2.1^{0}C and weekly incidence of salmonella 1988-2004

The equation of the line being:

$y = 0.064 + .018(t)$,

This indicates an increase of 0.018 cases of salmonella per 100,000 per week, or an increase of 7.3% increase in the incidence of salmonella per degree rise in temperature over the mean baseline of 0.25 cases per 100,000 per week.

The projected rise in incidence of salmonella that might be expected from future increases in temperatures over the entire range of temperatures was assessed and calculated as a change in rate per 100,000 (Table 7.7 (a)).

Scenario	2010-39	2040-69	2070-99
Mean	0.2	0.3	0.5
Range	(0.1 to 0.2)	(0.2 to 0.4)	(0.3 to 0.6)

Table 7.7 (a) Estimated increases in cases of salmonella per 100,000 population per year

The percentage increases in cases of salmonella per year from the mean rate of 0.25 per 100,000 per week in the time series or 13 cases per 100,000 per year was also assessed

(Table 7.7 (b)). The results are outlined for all outputs from the climate models with the A2 and B2 scenarios in Table 1 and Table 2 respectively, in Appendix G.

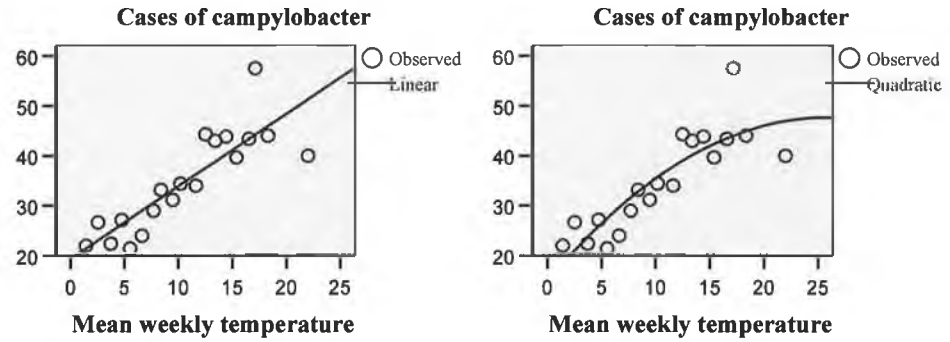
Scenario	2010-39	2040-69	2070-99
Mean	1.3	2.4	3.6
Range	(0.7 to 1.8)	(1.4 to 3.4)	(2.2 to 4.6)

Table 7.7 (b) Estimated percentage increases in cases of salmonella per year from the baseline of 13 cases per 100,000 per year

Increases in the incidence of salmonella are seen, with mean rises in the region of 1.0% occurring early in the century, rising to a mean of 2.4 % by mid-century and to over 3% above present rates towards the end of the century. With a population of 4.5 million, an extra 108 cases of salmonella per year may be expected by mid century.

7.8.2 Campylobacter

Although the incidence of campylobacter increases with mean temperature, the relationship is not straightforward. It is clear that the incidence of campylobacter rises with increasing temperature. The relationship appears to be curvilinear, and high R square values were obtained with linear, quadratic and cubic expressions (Figures 7.8(a) and (b))



R square= 0.725 p<0.001
$y = 19.432\text{-}1.448\ (t)^1$

R square=0.753 p<0.001
$y = 14.989\text{-}2.538\ (t)^1 + 0.049(t)^2$

Figure 7.8(a) Mean weekly temperature and incidence of campylobacter 2004-2005 using linear and quadratic expressions

However, a cubic relationship was chosen, as this type of associate has been noted previously (Figure 1, Appendix H) (Tam *et al,* 2006)

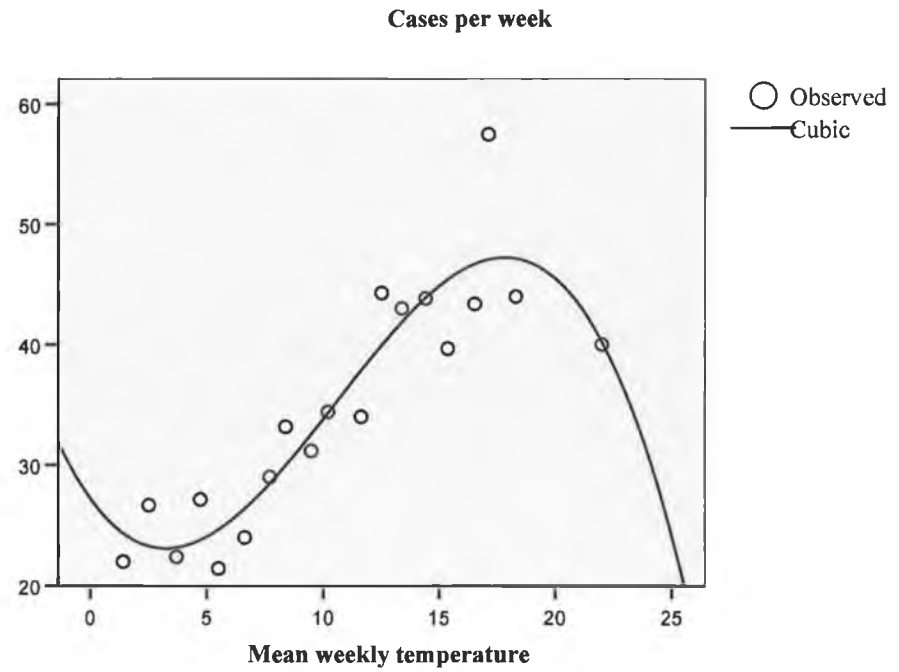


Figure 7.8(b) Mean weekly temperature and incidence of campylobacter 2004-2005 using cubic expressions

The cubic equation of the campylobacter-temperature relationship was expressed by the equation

$y = 27.211\text{-}2.700\ (t)^1 + 0.492(t)^2 - 0.016(t)^3$

Increases are seen in the number of cases of campylobacter mean increases in the range of 65 cases per year in the coming decades, over the present incidence of a mean of almost 1,800 cases a year (Table 7.9 (a)).

Scenario	2010-39	2040-69	2070-99
Mean	65	106	144
Range	(21 to 93)	(46 to 141)	(79 to 144)

Table 7.8 (a) Estimated increase in cases of campylobacter per year

The percentage increases in cases of campylobacter per year over the mean rate in the time series was also assessed (Table 7.7 (b)).

Scenario	2010-39	2040-69	2070-99
Mean	3.7	6.0	8.1
Range	(1.2 to 5.2)	(2.6 to 8.4)	(4.5 to 11.0)

Table 7.8 (b) Estimated percent increases in cases of campylobacter per year (from baseline of 34 cases per week)

The results are outlined for all outputs from the climate models with the A2 and B2 scenarios in Table 1 and Table 2 respectively, in Appendix H.

If the data is confined to days when the mean temperature is between 6 and 17^0C, in the curvilinear expression, the incremental rise in incidence can be quantified.

The equation of the line is represented by the equation y = 14.797- 1.878(t)

An increase of 2 cases per week or an increase of 5.5% above the mean rate of 34 cases per week in the incidence was seen for every degree rise in temperature in this range.

7.8.3 VTEC

The VTEC 0157 was expressed with a linear relationship (Figures 7.9).

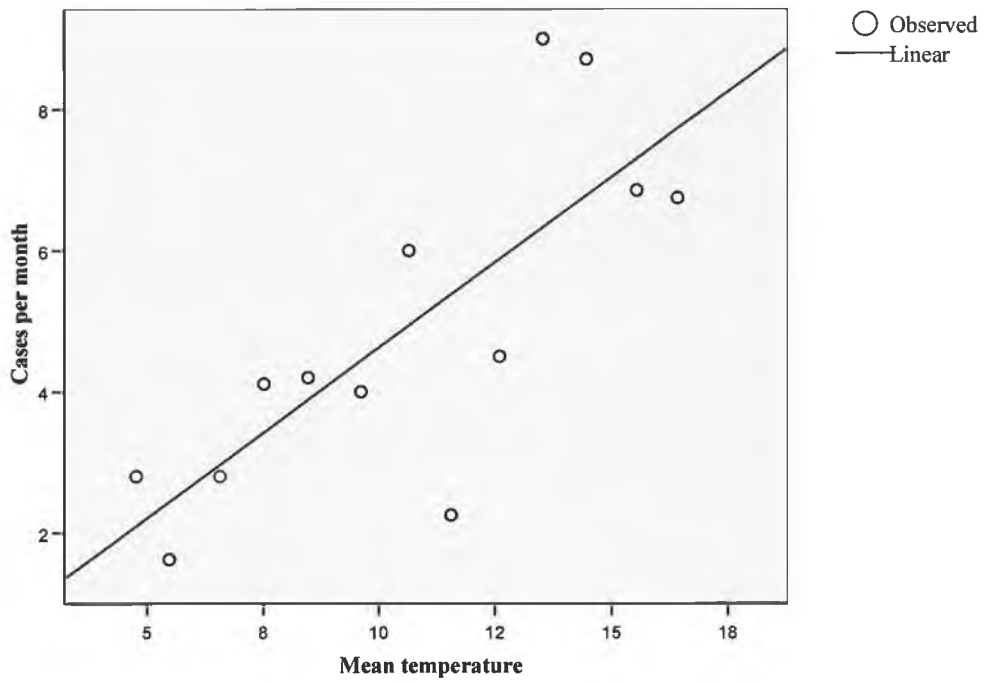


Figure 7.9 Mean temperature and incidence of VTEC 0157 1999-2005, less outbreaks

The equation of the relationship was in the format

$y = a(t)^0 + b(t)$

i.e. $y = -0.210 + 0.483(t)$

p=0.002 R sq 0.609

An increase of 0.48 approximately of a case per month is seen for every degree rise in mean temperature, or an increase of 9.6% per degree rise in mean temperature over the yearly rate of 60 cases a year.

Scenario	2010-39	2040-69	2070-99
Mean	8	11	16
Range	(2 to 13)	(6 to 15)	(9 to 19)

Table 7.9 (a) Estimated increases in cases of VTEC 0157 per year

Scenario	2010-39	2040-69	2079-2099
Mean	12.7	18.0	26.0
Range	(2.9 to 21.3)	(9.4 to 24.9)	(15.2 to 32.1)

Table 7.9 (b) Estimated percentage increases in cases of VTEC 0157 per year from mean baseline of 60 cases per year

The results are outlined for all outputs from the climate models with the A2 and B2 scenarios in Table 1 and Table 2 respectively, in Appendix I.

Substantial increases are evident in the incidence of this serious disease. Increases of approximately 13% will be evident in the coming decades, rising to over a quarter above the present incidence by the end of the century (Table 7.9 (a) and (b)).

7.9 Discussion

This is the first study identified in the literature that quantifies future estimates of specific food-borne disease associated with the impacts of climate change, and each will be discussed separately.

7.9.1. Salmonella

The temperature relationship established in this study is consistent with previous work. The seasonal pattern noted in the incidence of salmonella is in keeping with previous findings of a summer peak (Fleury *et al,* 2006, Kovats *et al,* 2005; D'Souza *et al*, 2004, Bentham and Langford, 1995). The temperature-morbidity relationship depicted in Figure 7.6(a) is also compatible with the findings of the European study (Kovats *et al,* 2004(b)), where a clear temperature-incidence relationship with salmonella is evident (Figure 1, Appendix G). Although the impact of temperature on salmonella was found to continue for at least 6 weeks, the impact was noted to be maximal in the week of onset in the present study by the method adopted (Table 7.4), and no clear evidence of a lag was evident. There is evidence that the peak in temperature precedes to a slight

degree the peak in incidence (Figure 7.6(a)). This may be accounted in part by outbreaks, which may be expected to occur more often in summer, and may not be fully accounted for by the method used. Lag periods have been seen to vary; using weekly data, the lag period for salmonella in European countries was noted peak at one week but was apparent up to 5 weeks later (Kovats *et al*, 2004 (b)), and lag periods ranging from 0 to 6 weeks have also been previously noted with salmonella (Fleury *et al,* 2006). Salmonella produces symptoms soon after ingestion and it is unlikely that longer lag times reported relate to the virulence of the organism. Longer lag times have also been attributed to infection occurring earlier in the food-production system (Hall *et al,* 2002), and it is possible that little time elapsed between the contamination, ingestion and the development of the illness in Ireland. Lags of one month have been noted in the UK, although this study included food-borne pathogens, whose temperature relationships may differ and was not solely confined to salmonella (Bentham and Langford, 1995).

The quadratic nature of the salmonella-temperature curve depicted in Figure 7.7 is similar to previous reports (Figure 2, Appendix G).

The threshold temperatures of 2.1^{0}C found in the present study is low; the lower limit for salmonella growth is generally regarded as being in the region of 7^{0}C (Fehlhaber and Kruger, 1998). Nevertheless, the threshold temperature identified in the present study is comparable to the 3^{0}C threshold temperature found in Scotland and Switzerland, but lower than two threshold values noted in the U.K. of 5^{0}C (Kovats *et al,* 2004), and 7.5^{0}C (Bentham and Langford, 1995). These findings are shown for comparison with the data from the present study included (Table 7.10)

Country	**Threshold temperature ^{0}C**	**% change per degree ^{0}C rise above threshold**
Poland	6	8.7
Scotland	3	4.7
England and Wales	5	12.4
Estonia	13	18.3
The Netherlands	7	9.3
Czech Republic	-2	9.5
Switzerland	3	8.8
Spain	6	4.9
Ireland	**2.1**	**7.3**

(after Kovats *et al*, 2004(b))

Table 7.10 Comparison of findings of present study

The range of threshold temperatures noted in the European study (from -2^{0}C to 15^{0}C) does not seem to be related to either latitude or to summer temperatures, and furthermore, no threshold temperature was noted for Denmark (Kovats *et al.* 2004(b)). The low threshold temperature obtained in the present study may in part be explained by contracting the infection overseas. Approximately one fifth of cases of salmonella reported in Ireland since 2004 were associated with foreign travel, the majority of cases having occurred in people who had travelled to Spain (HPSC, 2005(a); HPSC, 2006(b)). It is possible that this travel occurred in the winter time, however, it was not possible to account for this, as information on travel has only been available since 2004, although other studies have accounted for this, where possible (Kovats *et al.* 2004(b)). The incremental increase in incidence above the threshold depends in part on the threshold temperature identified (Kovats *et al,* 2004(b)); nevertheless, the increase in incidence of salmonella of 7.3 % per degree rise in temperature is comparable with the increases noted in Switzerland, the Netherlands, and the Czech Republic, where increases of 8.8%, 9.3% and 9.5% respectively were found per degree rise in temperature, but lower then the 12.4% increase found in the UK (Kovats *et al,* 2004(b)). The present results are also comparable with the 4-10% increase in risk of salmonella per degree rise in mean temperature in Australia (D'Souza *et al,* 2004).

The accordance of the temperature relationship established in this study with the international literature gives confidence to the temperature relationship established, and therefore to the validity of future estimates. Small increases in the incidence of salmonella may be expected in the future as a result of climate change (Tables 7.7(a) and (b)). This increase however, is not unimportant. Salmonella is an extremely significant cause of gastroenteritis in Ireland (HPSC, 2006(b)) and causes more deaths annually in Ireland and the UK than any other food-borne pathogen (Adak *et al,* 2002). Almost half of outbreaks of infectious intestinal disease in Ireland have been attributed to this bacterium (Fitzgerald *et al,* 2001). Similarly, salmonella caused the majority of outbreaks in the U.K. (Kessel *et al*, 2001), and over three quarters of outbreaks of food-borne disease in European countries (WHO, 2000). The commonest location of an outbreak of salmonella in Ireland is in public facilities; where over a third of outbreaks occurred in restaurants, followed by hospitals, residential facilities and private residences (WHO, 2000), and have been most commonly attributed to inadequate cooking and cross-contamination (WHO, 2000). It is also of concern that high levels of

antibiotic resistance have been found against S. Typhimurium; many of which have been found to be resistant to at least five anti-microbial agents (WHO, 2003(c); Helms *et al* 2005), and multidrug resistant S. Typhimurium has also been noted in Ireland (Gorman and Adley, 2004).

It is clear that temperature is one factor affecting the incidence of salmonella, however major changes have occurred in the incidence of this disease in Ireland in the absence of significant changes in mean temperature. The rise in the incidence of salmonella in Ireland peaked in the late 1990s and since then the rate had been steadily declining (Figure 7.3 (a)). This pattern is consistent with the pattern in the UK and other European countries (WHO, 2000; WHO, 2003(c)). Similar to the decline in incidence in the UK, which was attributed in part to interventions to reduce the carriage of salmonella in poultry farms (Cogan and Humphrey, 2003), the decline in Ireland has been attributed to increased consumer awareness of good food hygiene and control programmes for salmonella such as the Quality Egg Assurance Schemes, (FSAI, 2004(a)). Under this scheme, all egg farms are tested and monitored for salmonella in Ireland by the Department of Agriculture and Food. It is reassuring to note that Ireland has a low prevalence of salmonella in poultry flocks, with a prevalence of 1.4% for all species of salmonella with no recordings of the most common S. Enterica and S. Typhimurium, although a fifth of large scale egg laying holdings are positive for salmonella globally (EFSA, 2006). However, continuous surveillance, especially of imported eggs, is strongly recommended (van Pelt *et al,* 2004). However, the elucidation of the impact of a rise in temperatures on the incidence of salmonella is not a straightforward matter. The complex 'causal web' (Figure 7.1) of the aetiology of salmonella was clearly seen in the Netherlands, during the hot summer of 2003; although an increase in salmonella during this time was initially attributed to the higher temperatures, subsequent investigations found that the increase in incidence resulted from infected imported eggs and not to the high temperatures (van Pelt *et al,* 2004). Nevertheless, the results of this study indicate that, if non-temperature conditions (such as hygiene standards, foreign travel, prevalence of infection etc.) remain unchanged, increases in the incidence of salmonella may occur, from averages of 1.0%, early in the 21st century rising to over 3% above present rates by the end of the century.

7.9.2 Campylobacter

The campylobacter-temperature relationship established, namely a rise in the summer months is consistent with the literature. The seasonal aspect in the incidence of campylobacter in Ireland is clearly apparent (Figure 7.6(b)), and consistent with the results of a European study, and in particular the Irish data included (Figure 1, Appendix H). The impact of temperature on the incidence of campylobacter in the present study was seen to extend up to 6 weeks, although it was maximal after 1 week (Table 7.5). Although an impact extending up to 6 weeks has also been noted in a Canadian study, interestingly, it appeared to peak after both 1 week and also 3 weeks (Fleury *et al,* 2006). The lag period noted in the present study is shorter than the 4 weeks previously reported in Denmark (Patrick *et al*, 2004). A longer lag time still of 6 weeks has been noted in the U.K. (Tam *et al,* 2006) and lag times extending up to 10-14 weeks have been noted in a European Study (Kovats *et al,* 2005). Arising from the complex pathways in the environment whereby contamination of food by campylobacter may occur, it is not unexpected that varying lag times have been reported.

Campylobacter exhibits limited growth below 30°C (FSAI, 2002(a); Tam *et al,* 2006), nevertheless, the organism may remain viable in cooler water for over four months (Rollins and Colwell, 1986), and interestingly, a significant number of cases of campylobacter in Ireland (40%) were found to have had contact with both animals and with drinking water from a private or group water scheme (FSAI, 2002). Animals, wild birds, and surface water may be involved, and significant correlations have been found between cases of campylobacter and such variables as the number of sheep, pigs and poultry in an area (Louis *et al,* 2004). A sensitivity of the organism to mean temperatures in the 6^0C to 17^0C was found in the present study, in a cubic-type relationship (Figure 7.8). These findings are consistent with the nature of the relationship previously identified (Kovats *et al,* 2005), and a similar relationship with an upper threshold of 14^0C (Tam *et al,* 2006) (Figure 2, Appendix H).

The increase of 5.5% in the number of cases per degree rise in mean weekly temperature noted in the present study is again comparable to the 5% increase per degree rise in mean weekly temperature noted previously (Tam *et al,* 2006). Although a lower incremental increase of 2.2% for the log relative risk has been noted, the

threshold temperature in this Canadian study was -10^{0}C (Fleury *et al,* 2006), and this may explain the smaller increase. Work undertaken to clarify the cause of seasonality of cases of campylobacter, in nine European countries and New Zealand, did not reach a definitive conclusion; and recommended that 'other environmental reservoirs' be explored (Nylen *et al,* 2002). It is clear that the relationship between campylobacter and temperature is not straightforward and may relate to such issues as the number and type of animal reservoirs in the area. Nevertheless, the consistency of the campylobacter-temperature relationship established gives confidence to the results obtained of future estimates. This is the first study identified that quantified the increase in the number of cases of campylobacter that may arise in the future, resulting from climate change. Increases in campylobacter may be expected, with increases in the region of 0.5% early in the coming decades, rising to almost 1% by mid century and up 1.5% over present levels by the end of the century (Tables 7.9(a) and (b)).

This increase is significant as campylobacter is now the most commonly isolated bacterial cause of human gastrointestinal illness in Ireland (Foley and Mc Keown, 2004), as in many European countries (WHO, 2000). In Ireland, campylobacter continues to be a significant public health issue, both in terms of personal suffering and economic costs (HPSC 2006(b)). The reported incidence of this infection in most developed countries has risen substantially during the past 20 years, in particular since 1990 (WHO 2000(b); Frost *et al,* 2002), although this may partly be due to an improvement in diagnosis (WHO 2000 (b)).

The absence of a characteristic food associated with the disease (such as eggs with salmonella) may contribute to a difficulty in making inter country comparisons, and in Ireland the majority of cases of campylobacter remain unexplained by generally recognized risk factors for disease (HPSC 2006(b)). However, the prevalence of campylobacter in meat products is high in Ireland. In a study of a range of Irish meats and poultry, campylobacter was found in over half of poultry and offal samples and in a fifth of minced meat samples (Cloak *et al,* 2001). In addition, over half of broilers were found to contain the bacteria in their faeces when sampled at the farm of origin (FSAI, 2002). Similar to salmonella, there is a problem with resistance to antibiotics, and this has been attributed in part to the use of antimicrobial agents in animal husbandry (Aarestrup and Engberg, 2004) There is only limited data available to quantify the

problems of antimicrobial resistance in Campylobacter species isolated from humans or animals in Ireland (FSAI, 2004(a). Nevertheless, as with salmonella, the necessity for strict adherence to hygiene protocols remains, in order to reduce the risk of this disease, particularly in the light of the high prevalence of campylobacter in foodstuffs on sale in Ireland. This is even more relevant with higher temperatures expected, and possible increases in the incidence of this infection.

7.9.3 VTEC

The seasonal pattern in the incidence of VTEC with a summer peak (Figure 7.6 (c) is similar to previous reports (Fleury *et al,* 2006; Chapman, 1995). This the first study to estimate future incidence of VTEC 0157 that may result from climate change and reports a 9.6 % increase per degree rise in weekly mean temperature. Only one other study was identified in the literature that quantified the increase in VTEC with temperature increases, reporting a 6% increase per degree rise in weekly mean temperature (Fleury *et al,* 2006). A significant rise in incidence is therefore estimated in the future incidence of this serious infection in Ireland, with increases of 12.7% early in the century, rising to 18% by mid century and to over a quarter by the end of the century (Tables 7.10(a) and (b)). However, these large increases estimated must be accompanied by a large degree of uncertainty, and there are two reasons for this. Firstly, although an attempt was made to remove the impact of outbreaks from the dataset, there is a difficulty in completely eliminating such cases. As these cases could be expected to distort the VTEC 0157 morbidity-temperature relationship, they may contribute to a spuriously high result. Secondly, this disease is also waterborne, and outbreaks of VTEC in Ireland have been associated with a history of contact with water (HPSC, 2006(b), which again may distort the temperature relationship.

Livestock is the most important reservoir for VTEC in Ireland, with both dairy and beef cattle being the principal source (FSAI, 1999), and this serious pathogen has been noted in 7% of cattle hides in Ireland (O'Brien *et al,* 2005). In addition, minced beef found on sale in Ireland has been found to have been contaminated with both non-toxic E.Coli (Murphy *et al,* 2005) and VTEC (Cagney *et al,* 2004), and an association with the consumption of unpasteurized milk or cheese and the incidence of VTEC has also been found (NDSC, 2004). Its presence in retail outlets is of concern, and the possibility of

resultant infection from this pathogen presents 'a real risk to public health' (FSAI, 2002(b)).

7.10 Issues of concern

It is clear that significant increases may occur in the incidence of three specified food-borne disease in Ireland resulting from climate change, and five issues of concern have been identified. Firstly, the high prevalence of VTEC and campylobacter in the community, particularly in meat, is a cause for concern in the light of future temperature rises. Secondly, in the light of the foregoing, the necessity of strict adherence to hygiene protocols, and the importance of excluding staff members who report gastrointestinal symptoms from food-handling have been stressed (FSAI, 2002). Although the issue of hygiene is being addressed, the low awareness of some staff in the food industry of hygiene procedures is a cause for concern (FSAI, 2006(b), and focussed education programmes on food hygiene to relevant occupational groups have been recommended (Fleury *et al,* 2006). The importance of continued surveillance and adherence to hygiene regulations will remain an important public health issue in the coming decades. In addition, recommendations have been issued to food business operators to regularly test their water supply and if necessary install water treatment systems (FSAI, 2004 (b)). Thirdly, difficulties are also posed when some of the pathogens do not cause illness in the animal reservoir, e.g. neither E. Coli 157 in calves or salmonella in chicken cause symptoms. The sale of unpasteurized milk was prohibited in Ireland in 1997, however such milk was consumed by the farm family on 84% of 230 farms investigated, raising the possibility of outbreaks of salmonella, campylobacter and other food-borne pathogens occurring on such farms (FSAI, 2004). Finally, new infectious agents continue to be identified, whose incidence may be affected by climate change. Enterobacter sakazakii has been only recently recognized as a potential emerging food-borne pathogen (Farber, 2004). Few laboratories test for this disease which, to date, has mainly affected preterm and new born babies (Kandhai *et al*, 2003).

7.11 Sources of uncertainty

There is uncertainty in the results of this study and four sources have been identified. Firstly, the results may be an underestimate as it is well established that under reporting

of food-borne disease occurs, and a comprehensive surveillance system has only recently been put in place in Ireland. Under-reporting of incidences of food-borne disease may be particularly significant if the nature of cases which are not reported differ significantly from those illnesses which are reported, although it may be expected that reporting is complete in more severe cases of food-borne disease (such as VTEC 0157). Secondly temperature data from Kilkenny was used on national data. This may obscure important local differences, such as farming practices that may be masked by the averaging effect of national data. In addition, the aetiology of food-borne disease is complex, and temperature may be a marker for animal reproductive cycles or farming practices, which may blur the temperature-morbidity relationship (Hall, 2002). Thirdly, the present temperature-morbidity relationship established may not replicate the impacts of gradually increasing mean temperatures on the incidence of food-borne illness (D'Souza *et al,* 2004). Finally, it is clear that while temperature increases rise may contribute to a significant rise in food-borne disease in Ireland, it is only one factor in the potential for a significant rise in the incidence of salmonella, campylobacter and VTEC. The pattern of infectious intestinal disease has been described as 'complex and evolving' (Adak *et al,* 2002). Other non-climatic factors such as changes in consumer behaviour, foreign travel, changes in agricultural practice and in increasing international trade, which facilitates the rapid spread of disease play a role. These factors occurring together with a rise in temperature may make it difficult to clearly associate a changing incidence of food-borne disease with increases in temperature arising from climate change.

7.12 Future work

In order to detect early evidence of the impact of climate change (WHO 2003(b)) changes in seasonal patterns and changes in the distribution over space and time need to be assessed. From January 2004, the greater range of diseases that must be statutorily notified in Ireland, together with more detailed information on the cases will undoubtedly contribute to a greater elucidation of the impacts of climate change on food-borne disease. Significantly, details of cases of campylobacter, cryptosporidiosis, and VTEC, will be documented. Unusual clusters and changing patterns of illness are now reported. Such information will allow valuable information to be gleaned in

subsequent work on the impact of changes in temperature on the incidence of food-borne disease in Ireland.

7.13 Conclusion

It is clear that future increases in mean temperatures, in the absence of other mitigating factors, will increase the incidence of salmonella, campylobacter and VTEC 0157, and possibly other food-borne infectious diseases in Ireland. This is significant as food-borne disease is prevalent; acute gastro-enteritis is one of the commonest reasons for visiting the family doctor in Ireland (FSPB, 2003). This is the first study identified in the literature that presents a quantitative estimate of the impact of future temperature increases on the incidence of specific food-borne diseases. By the middle of this century, substantial increases in the incidence of VTEC may be expected, with smaller increases in the incidence of salmonella and campylobacter. The difficulty in gauging the extent of the problem and the multi-factorial aetiology of food-borne disease militate against an accurate estimate of the impact of climate change on the future incidence. However, recent statutory improvements in the reporting of infectious disease, augurs well for the future, and will enable further assessments of evidence of early evidence of the impact of climate change on food-borne disease in Ireland, to be readily undertaken.

CHAPTER 8 - OTHER INDIRECT IMPACTS OF CLIMATE CHANGE ON HEALTH

"It is well to remember the possibility of cases [of malaria] *returning to Cork at some future date".*

(Marshall-Cummins, 1957)

Aside from the impacts of climate change on temperature-related mortality, and food and water-borne disease, climate change will also impact on many other health-related areas (AR4, 2007). The incidence of vector-borne disease, including malaria and tick-borne disease may change as meteorological variables affect both the physical environment and the physiological variables of the organism. Changes in rainfall and temperature patterns may also affect levels of allergens, and both sea-level rise and changes in rainfall will increase the risk of flooding. Exposure to ultra-violet radiation will rise, resulting in possible increases in cases of skin cancer. A brief discussion of these diverse issues, followed by a discussion of the relationship with climate in Ireland is the subject of this chapter. Lack of data again militates against future quantitative estimates of these indirect impacts, and estimates of the future incidence will be made by a review of the data and an examination of the data that is available.

8.1 Vector-borne disease and climate change

The incidence of vector borne diseases may be affected by climate change. However, the aetiology of vector-borne disease is complex, and for such diseases to become established, three factors are required. Firstly, these diseases require the presence of the causative infectious agent, for example, in the case of malaria, the plasmodium organism, or in the case of Lyme disease and tick-borne encephalitis, the borrelia and virus organisms respectively. Secondly, a vector is required to carry the organism, such as the mosquito in the case of malaria, or a tick in the case of Lyme disease and tick-borne encephalitis. While mosquitoes are the main vectors for disease in warmer climates, ticks are the main human disease vectors at northern latitudes (Lindgren, 1998). Ticks are parasitic insects that live on mammals and birds. Finally, a host organism (infected individual) is needed who, as in the case of malaria, may also be necessary for the completion of the lifecycle of the organism. Climate may have a

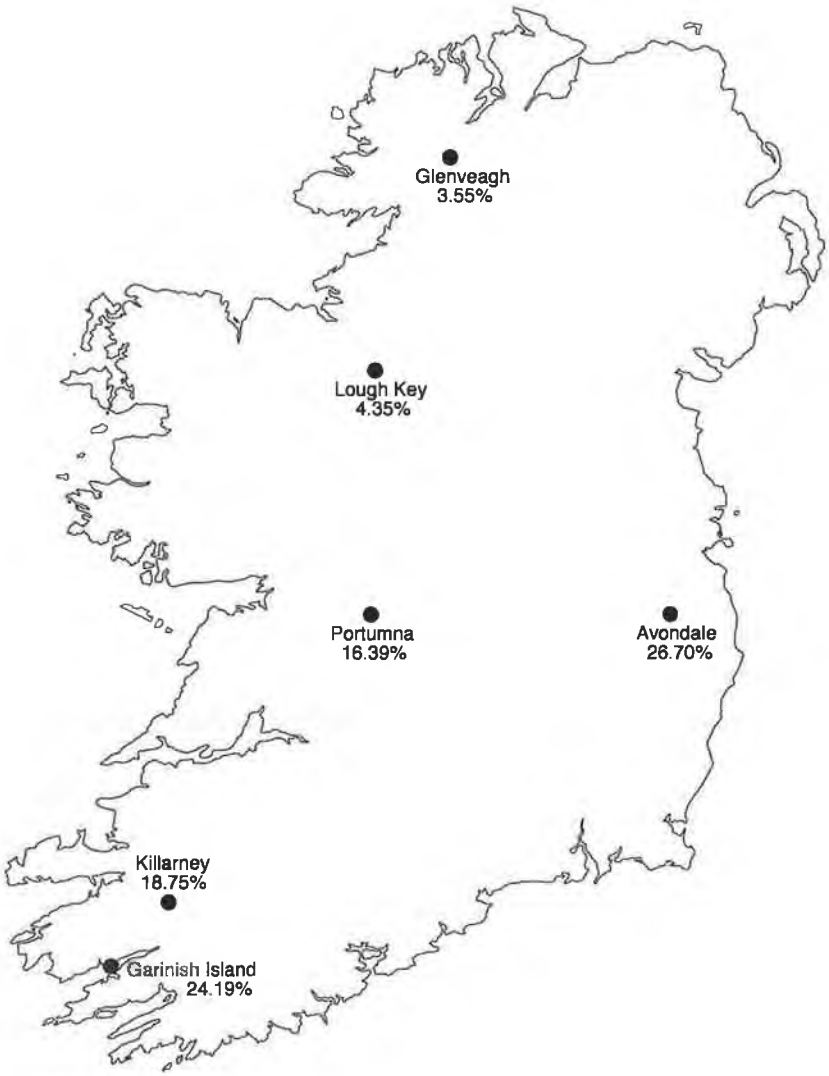
significant influence on the incidence of vector-borne diseases because the causative organisms live outside the human host for a part of their lives, and are therefore more vulnerable to environmental factors. Changes in the distribution and behaviour of insect and bird species are occurring worldwide as a result of climate change, as new areas become environmentally more favourable (AR4, 2007). As these species may be a part of the life cycle of vectors, it could be expected that climate change would also influence the extent of vector-borne infectious diseases. Nevertheless, although there is evidence of a change in the distribution of some vector borne diseases (AR4, 2007), no report unequivocally associates vector-borne diseases with increased temperature and the environmental changes expected to accompany it (Zell, 2004). In Ireland, the only vector-borne disease of significance is Lyme disease.

8.2 Lyme disease

Lyme disease is the most common tick-borne disease in the Northern hemisphere (Stanek and Strle, 2003). The vector for Lyme disease is the Ixodid tick, which is widely distributed in temperate regions. It takes its name from the town of Lyme, in Connecticut, USA, where it was first recognized in 1975. The illness typically begins in summer, and is characterized by general malaise, headache, stiff neck, muscular pains and swelling of the lymph nodes; a single or multiple red circular lesions on the skin may precede or follow the illness. In the following months, serious cardiac, neurological and joint abnormalities may develop. The disease can be difficult to diagnose because it resembles many other medical conditions, and underreporting is therefore a problem. Treatment is by antibiotics, and while complete recovery is usual, some patients continue to suffer from post-treatment chronic Lyme borreliosis, with ongoing symptoms such as persistent fatigue, muscle pain and neurological problems, including memory and mood disturbances. People who live or work in areas surrounded by tick-infested vegetation are at risk of getting Lyme disease, in particular people who camp outdoors, engage in hunting, or whose work involves outdoor occupations such as forestry, and wildlife and parks management.

8.2.1. Incidence

Although the tick is widespread in Ireland (Figure 8.1), many tick-infested cattle, sheep and enclosed red deer do not carry borrelia (Gray *et al,* 1994), and it is estimated that only 15% of the tick population is infected (Kirstein *et al,* 1997).



(after Kirstein et al, 1997)

Figure 8.1 Prevalence of Borrelia in Ixodes ricinus in Ireland

The optimal habitat for the tick vector is mature heterogeneous woodland that is inhabited by an abundant and diverse fauna, including deer (Gray *et al,* 1999). Interestingly, evidence of past infection with borrelia in blood donors correlates well with the perceived risk at the locations concerned (Smith *et al,* 1991). Ground-feeding

birds such as the robin, the blackbird and the wood-mouse may also be important hosts for the tick in Ireland (Gray *et al,* 1994; Gray *et al,* 1999; Gray *et al,* 2000). The most predominant type of Borrelia (B. valaisianna) in Ireland has not been associated to date with Lyme disease, and furthermore, many strains of the second commonest variety (B. garinii) may be of low pathogenicity (Gray *et al,* 1999). These factors may contribute to the apparent low incidence of diagnosed Lyme disease in Ireland, where a total of 8 cases were diagnosed in 2005 (Table 8.1), although under-reporting may be a problem as under detection and under reporting of Lyme disease is a problem in the UK (Rogers *et al,* 2001), and also in the U.S. (Campbell *et al,* 1998). However, Lyme disease is not a notifiable disease in Ireland and consequently the actual incidence is not known.

Year	Tests undertaken	Positive
2000	950	9
2001	1005	10
2002	1050	6
2003	1051	10
2004	1357	13
2005	1617	8

(data from VRL 2006)

Table 8.1 Cases of laboratory diagnosed Lyme disease in Ireland

8.2.2 Lyme disease and climatic influences

The tick requires temperatures in the range 15-30^{0}C to continue its development, and transmission of the disease below 7^{0}C is rare (Santos *et al,* 2002). The relationship with summer temperature seems to have become more marked in recent years, and significant correlations between the incidence of Lyme disease in early summer and the moisture index of the June two years earlier in the US have been noted (Subak, 2003). In addition, milder temperatures benefit many smaller animals, and the influence of a warmer climate cannot be ruled out as a cause of the recent increase in Borrelia infection in ticks in Germany (Kampen *et al,* 2004). However, changes in the incidence of Lyme disease in the USA have also been linked to changes in land use patterns and residential developments in wooded areas (Gubler *et al,* 2001). The complexity of the life-cycle and the environmental determinants that impact on it, make it difficult ascertain a link between climatic changes and incidence.

8.2.3 Future estimates

There are two opposing factors arising from climate change that would influence the prevalence of Borrelia. Firstly, warmer temperatures would accelerate their development, particularly during the winter leading to more opportunities in subsequent summers for ticks to infect humans (Subak, 2003). On the other hand, a dry summer would increase tick mortality. However, due to the complex influences of climate on the life cycle of the tick and organism, it is therefore 'almost impossible' to reliably predict any change in tick abundance with climate change in the UK (Rogers *et al,* 2001), and future estimates of the incidence vary. For example, modelling techniques have depicted an increase in suitable habitat of 213% by the 2080s in North Canada, with a retraction of the vector from the southern U.S. and movement into the central U.S. (Brownstein *et al,* 2005; Ogden *et al,* 2006). In contrast, it is estimated that areas of endemicity in Portugal would be unlikely to develop in the absence of large influxes of animal reservoirs and the deterioration of the social and healthcare conditions (Lopez-Velez and Molina Moreno, 2005). Communities at risk of developing Lyme disease from land use patterns have been identified by Geographic Information Systems (GIS), using spatially referenced data on land-use (Dister *et al,* 1997).

8.2.4. Future estimates for Ireland

The future incidence of the disease in Ireland will be determined by the exposure of the population to infected ticks, and this will be related to both land cover and the behaviour of the population. There are no plans to make Lyme disease a notifiable disease on European Community disease surveillance systems (Smith and Takkinen, 2006), and it is not a notifiable disease in Ireland. The use of Geographical Information Systems to ascertain land-use may be helpful in determining the risk, but a quantification of the future incidence of this disease is not readily achieved at present.

8.3 Tick-borne encephalitis

Tick borne encephalitis (TBE) is a viral infection of the central nervous system. Infection most commonly occurs during the highest period of tick activity (between April and November), when an individual is bitten by an infected tick, although the tick

that spreads tick-borne encephalitis prefers rats. Infection may also follow the consumption of raw milk from infected goats, sheep, or cows. There is an initial phase lasting 2 to 4 days when the patient has flu-like symptoms with headache and nausea. After approximately 8 days of remission, a second phase of the disease occurs in up to a third of patients, which involves the central nervous system, with symptoms of meningitis or encephalitis, including drowsiness, confusion, sensory disturbances, and possibly paralysis. Long-lasting or permanent neurological or psychiatric sequelae are observed in up to 20% of infected patients. There is no specific treatment for this disease.

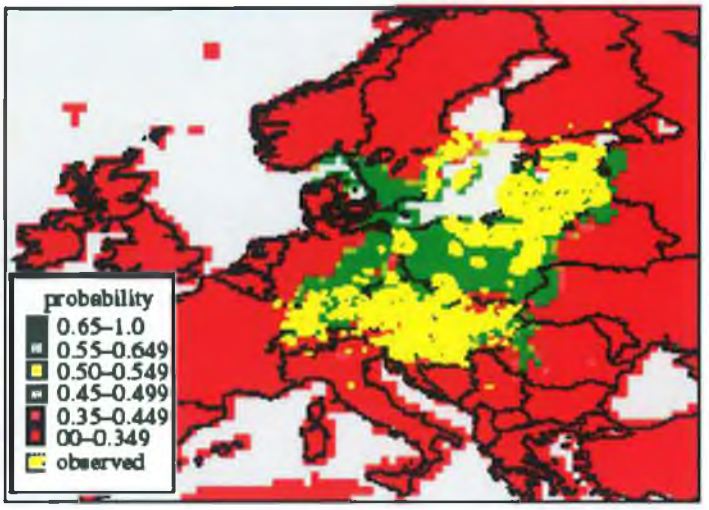
8.3.1 Tick-borne disease and climatic influences

Temporal analysis of climatic variables in the Baltic States indicates a very specific change in spring temperature conditions from 1993 onwards that could enhance the transmission of TBE virus (Sumilo *et al,* 2006). There is also evidence that a warmer climate is impacting on the incidence of TBE, and work in Sweden (Lindgren, 1998; Lindgren and Gustafson, 2001) suggests that the relatively mild climate of the 1990s is one of the primary reasons for the increase of density and geographic range of ticks. In addition, the marked increase in average annual temperature in the Czech Republic from 1989 has been proposed as a contributory factor to the incidence of TBE rising there in recent years, with ticks appearing at higher altitudes in mountains than in earlier years (Beran, 2004). Furthermore, a warm winter was thought to have been associated with an increase in the number of cases of TBE in Germany in 1994, leading both to more ticks surviving and also more people being outdoors (Kaiser, 1995). Nevertheless, underlying the difficulties in attributing an aetiological role to climate, the increases in TBE in most parts of Europe since 1993, although marked, have also been attributed to non-biological causes, such as political and sociological changes (Randolph and Rogers, 2000). In addition, the spatial changes in incidence of TBE have been found not to accord with spatial changes in meteorological variables, and the increases in TBE in Sweden pre-dated the onset of warmer springs and winters (Randolph, 2004). Other non-climatic factors may also contribute, and both a vaccination against TBE, which has been available since 1986 and increased awareness of ticks, may cause a reduction in incidence, leading to an underestimate of the impact of warmer climate (Lindgren and Gustafson, 2001). The situation is not resolved and more research is required, including

analysis of the impact of such factors as increases in deer abundance and changing habitat structures on the incidence of the disease (Randolph, 2004).

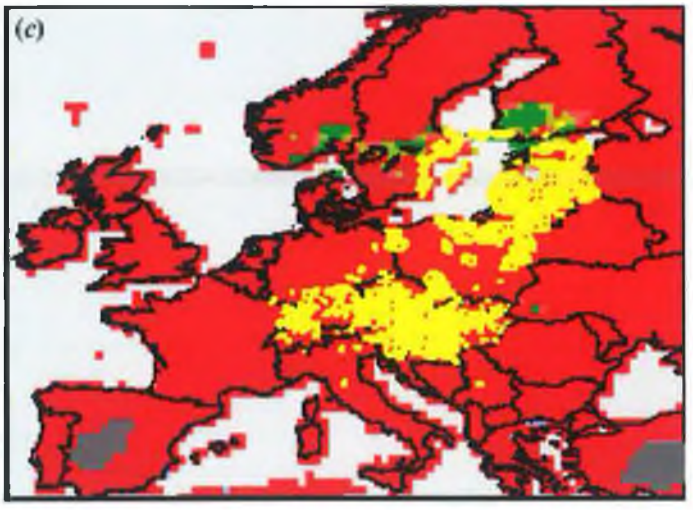
8.3.2 Future estimates

Although it is clear that many factors affect the incidence of TBE, the extent of the present distribution of this illness in Europe was found to be predicted very well by five climatic variables (Randolph and Rogers, 2000). This relationship was applied to outputs of a general circulation model, and used to predict future distributions of TBE. The expected changes in temperature and rainfall appeared to drive the distribution into higher-latitude and higher-altitude regions progressively through the 2020s, 2050s and 2080s, so that, by the 2080s the distribution may be confined to a small part of Scandinavia (Figures 8.2 and 8.3).



(after Randolph and Rogers, 2000)

Figure 8.2 Present day distribution of tick-borne encephalitis virus in Europe (yellow area), and predicted distribution based on 1961-1990 monthly temperature and rainfall data



(after Randolph and Rogers, 2000)

Figure 8.3 Future distribution of tick-borne encephalitis virus for 'medium high' scenario in the 2080s (Yellow area and probability scale as in Figure 8.2 Grey area: no predictions possible).

From this study of the dynamics of tick-borne encephalitis, the disease is not expected to become established in the UK (Rogers *et al,* 2001). To date, no instances of TBE have been recorded by the Health Protection Surveillance Centre (Cotter, 2006), and

following from the results of the modelling study of (Randolph and Rogers, 2000), the risk of TBE in Ireland with climate change would appear to be negligible.

8.4 Malaria

> *For it is clear ... that all that is necessary is a return of regularly recurring hotter summers over a definite period of years, and the malaria –carrying Anopheles will inevitably re-establish themselves and infect a certain proportion of the community, in the course of time. Given suitable breeding conditions, malaria becomes inevitable".*
>
> (Marshall-Cummins, 1957)

Malaria is considered the world's most important vector borne disease (Mc Michael *et al,* 2002). It is a serious and sometimes fatal disease, caused by the one-celled organism, plasmodium which is carried by the mosquito. Malaria occurs in over 100 countries and more than 40% of the people in the world are at risk, including Central and South America, Africa, and Asia, where 300-500 million cases of malaria occur and more than 1 million people die of malaria every year (WHO, 2006).

The transmission of malaria is complex, because a part of the reproductive cycle of the causative plasmodium organism occurs in the infected human. Female mosquitoes initially become infected with this organism from an infected person whom she bites. She ingests this blood which contains the immature male and female developmental stages of the plasmodium. Following ingestion of such blood, these immature stages of the causative organism unite in the stomach of the mosquito, and develop into the mature organism. From there, they travel to its salivary glands, where they enter the blood supply of a human when they are bitten. Four types of plasmodium can cause malaria in humans: Plasmodium falciparum (P.Falciparum), Plasmodium vivax, (P.vivax), Plasmodium ovale (P.Ovale), and Plasmodium malariae (P. malariae). The most serious form is P.falciparum; other types of malaria are generally not life threatening. In general, the symptoms of malaria comprise an indefinite malaise and the gradual onset of fever, followed by chills and a more rapidly rising fever, accompanied by nausea, headache and sweating. This cycle is repeated, a prominent feature of the

attacks being their periodicity, which is specific to the type of plasmodium. Relapses may occur at irregular intervals for up to 2 and 5 years with P.vivax and P. ovale respectively. The more serious form, P. falciparum presents a more varied clinical picture, including fever and chills, which may progress to jaundice, coagulation defects, renal and liver failure, pulmonary and cerebral oedema, coma and death. Mortality rates in children and non-immune adults from malaria exceed 10% and malaria is a significant cause of peri-natal mortality, low birth weight and maternal anaemia. Children who survive an episode of severe malaria may suffer from brain damage.

For malaria to be endemic in a country, three elements are essential. Firstly, the mosquitoes must be present in the country. Secondly, there must be a supply of hosts (sufferers) with the plasmodium organism in their blood. Finally, the climate must be suitable for the development of the parasite in the mosquito. The development of P. vivax is completely inhibited at temperatures below 15^0C and the more serious P. Falciparum, below 19^0C (Walker, 1998). While temperatures may control the seasonality of malaria (Craig *et al,* 2004(a)), it is unclear whether the northern limit of transmission of malaria is defined by the warmth of the summers, or the coldness of the winters as the boundary may be the 15^0C July isotherm (Reiter, 2000) or the 15^0C winter isotherm (Patz *et al,* 1996). The interaction between these three variables can be seen in Australia, where malaria was eradicated in the 1960s. In the north of the country, the climate is suitable for the transmission of malaria, and there are many vectors (mosquitoes), but there are too few infected individuals to act as a reservoir of infection. In the south, there are more infected individuals but there are fewer vectors and the mean temperatures are often around or below the critical temperature for the development of the parasite in the vector (Walker, 1998).

8.*4.1 Decline in incidence of malaria in Europe*

It is interesting to note that it was the geographer John Macculloch F.R.S. (1773-1835), author of the first geological map of Scotland, who introduced the word 'malaria' to the English scientific language. At this time malaria was common in most of England, cases being noted as far north as Inverness and it was also endemic in southern Norway, Sweden, and Finland (Reiter, 2000). The earliest reference to the existence of malaria in Ireland is in 1652, from Boate, who was Cromwell's 'Doctor of Physick to the State

of Ireland', who noted its existence in Ireland at the time (Bruce-Chwatt, and de Zulueta, 1980). However, the incidence rose in later years, and malaria was until relatively recently, a common cause of death in Ireland (Marshall-Cummins, 1957). Three reasons are cited for the occurrence of malaria in Cork in the mid 1800s. Firstly high temperatures were experienced. Secondly, survivors returned with the disease from the war in the Crimea (1853-1856), a very malarious area. Finally, there was an area of swamp in Cork (possibly near Ballincollig), suitable for inhabitation by mosquitoes, at least a square mile in area, when the epidemic was at its height (Marshall-Cummins, 1957).

There appears to be no single reason for the decline in malaria in Europe, which occurred before the role of the mosquito in its causation became apparent. Many factors have been implicated, including the draining of marshes, improved housing standards which rendered habitations mosquito proof, greater access to medical care, a fall in the cost of quinine and the growing of mangolds (Rogers *et al,* 2001; Reiter, 2000; Bruce-Chwatt, L.J. and de Zulueta 1980). The latter enabled animals to be kept over the winter, and diverted the mosquitoes to the animals. The role of climate in the decline of malaria is therefore difficult to ascertain. Furthermore, the complicated lifecycle of malaria, allowing people with the plasmodium to harbour infection for long periods, makes it difficult to correlate changes in climate with the decline in the incidence of malaria. However, modelling of the determining factors that contributed to the decline in the incidence of malaria in the UK from 1840 to 1910 indicated that while short term variations in precipitation models were found to explain outbreaks in 1848 and 1859, changing land use caused most of the decrease, and long term changes in climate did not appear to have an impact (Kuhn *et al,* 2003). In addition, the incidence of malaria reduced during a warming phase in Europe, when temperatures were warmer than during the preceding Little Ice Age (Reiter, 2000). Nevertheless, in a study of UK temperatures from 1659 to 1959, while factors such as socio-economic conditions and agricultural practices were seen to play the predominant part in the fall in the incidence, successive cold summers in the 1800s lowered the transmission of the parasite (Lindsay and Birley, 1996), and it appears that climate by itself played a subordinate role to other factors in the decline in the incidence of malaria in Europe. The last indigenous case of malaria in the UK was in the 1950s and in Holland as late as 1961. Italy was declared

free of malaria in 1970, and in 1975, the WHO declared that Europe was free of malaria, and by the 1980s, malaria was nearly a forgotten disease in the European region (WHO 2001(b)). In more recent times, the incidence of malaria has increased. Since the 1990s increases in Azerbaijan, Tajikistan and Turkey have been attributed to political and economic instability, large population movements and irrigation projects (WHO, 2001(b)).

8.4.2. Malaria and climate change

> *"Previous to the year 1844, malaria was almost unknown in Cork, and then it commenced, year after year, increasing its ravages, until it became a perfect plague in 1857. In my registry alone, in that year, (the Ballintemple-Blackrock dispensary), 175 cases occurred"* from the record of Dr. W. Jackson Cummins. (Marshall-Cummins, 1957)

It has been estimated that mosquitoes are likely to be amongst the first organisms to extend their range with climate change, because of the rapidity with which they can increase their numbers (Lindsay and Birley, 1996). However, it is not clear how this will affect the future incidence of malaria. The impact of temperature change has different and possibly opposite impacts; increased temperature would accelerate the development of both the vectors and their parasites, however, warmer temperatures would also increase the mortality rates and tend to decrease the breeding sites (AR4, 2007; Rogers *et al,* 2001). Nevertheless, malaria is being reported at higher altitudes in Asia, Central Africa and Latin America (Epstein *et al,* 1998), and a rise in mosquito numbers in a low lying area where they are very prevalent may not be as significant as a smaller rise in an upland area where they are rare (Pascual *et al*, 2006). In the North West Frontier Province in Pakistan, unusually high temperatures at the end of the normal malaria season were found to prolong transmission and substantially increase the numbers of cases (Lindsay and Birley, 1996). However, in a study in East Africa, changes in the incidence of malaria were not found to be directly related to climate change; neither temperature, rainfall, vapour pressure and the number of months suitable for P. falciparum transmission were reported to have changed significantly during the period of reported malaria resurgence, and no increase in malaria was noted in high altitude where increases had been reported earlier (Hay *et al,* 2001). To clarify

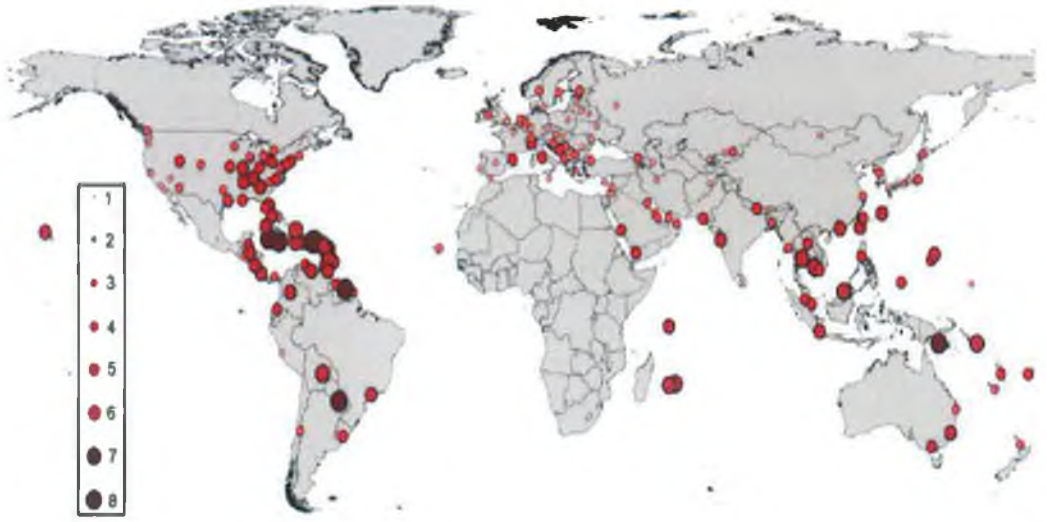
these issues, long term local monitoring of climate and disease variables has been recommended (AR4, 2007; Patz *et al,* 2002).

Aside from meteorological variables, other factors affect the survival of mosquitoes and other vectors, and caution has been urged against attributing recent outbreaks of malaria to climate change. These issues include a change of land-use, the movement of immigrant populations carrying the malarial parasite, drug resistance, improved surveillance practices and a failure of control practices (Lindsay and Birley, 1996; Craig *et al,* 2004(b); Rogers *et al*, 2001; Burke *et al*, 2001).In addition, the deterioration in health services in Eastern Europe has been cited as a specific cause for the recent resurgence in malaria in these countries. Interestingly, one particular (non-pathogenic) mosquito appears to have genetically adapted to a longer growing season, (anticipated to occur through climate change) by adopting a later dormancy period (Bradshaw and Holzapfel, 2001).

8.4.3 Malaria and international travel

Increased global travel and the inadvertent carriage of mosquitoes may contribute to the ongoing threat of malaria and the distribution of vectors by aircraft may cause local outbreaks of 'airport malaria' in areas near airports in people who may not have travelled to countries where malaria is endemic (Zucker, 1996). There are a considerable number of months in the year at present when the climate is suitable in international airports for the survival of P. Falciparum (Figure 8.4).

Random searches of airplanes at Gatwick Airport , in London found that 12 of 67 airplanes from tropical countries contained mosquitoes (Curtis and White, 1984). However, airport malaria is rare; a total of 89 cases of airport malaria have been reported in twelve countries over a thirty year time period, with the majority of cases occurring in France, Belgium and the U.K, (Gratz *et al*, 2000). However, airport malaria is dangerous as there may be a low index of suspicion, and diagnosis may be difficult in a person who has not travelled overseas.



(after Tatem et al,2006)

Fig 8.4 Number of months in a year that the climate at international airports is sufficiently similar to that of a Sub-Saharan African airport, within its primary malaria transmission season for imported *P. falciparum*-carrying *An. gambiae* survival.

Imported mosquitoes may also result in the establishment of transmission by local mosquitoes and this appears to have happened in the USA, where two cases of malaria appeared to have been caused by local mosquitoes (Layton *et al*, 1995). Interestingly, in August 1997, a case of malaria was diagnosed in a woman who was living in a remote rural part of Italy and who had not travelled to an area of the world where malaria was endemic. It appears that she had been infected by a local mosquito which had been infected with P. vivax from a carrier from India, who had arrived 7 months previously, thus indicating the possibility of malaria being reintroduced (Baldari *et al*, 1998).

8.4.4. Methodological issues

Modelling techniques have been used to estimate the impact of climate change on the future incidence of malaria, and a global mean temperature increase of 2-3^{0}C in the year 2100 has been estimated to increase the epidemic potential of the mosquito population in tropical regions twofold and more than 100-fold in temperate climates (Martens *et al*, 1995). However, the complexity of the lifecycle of plasmodium, militates against a

simple statistical approach. There are many uncertainties including the impact of human behaviour from increased biting, the impact of temperature on feeding practices, and difficulties in accurately determining the age of mosquitoes (Rogers *et al,* 2001; Lindsay and Birley, 1996; Zucker, 1996). Currently, biological approaches are not able to accurately predict the current presence of malaria and therefore cannot be used to estimate the likely impact of climate change (Rogers and Randolph, 2000). An instructive tale, illustrating the complexity of human vulnerability, and the difficulties in estimating future impacts of malaria is found in the Gambia, where an area with the lowest mosquito count had the highest incidence of malaria, because elsewhere people living in areas with a higher density of mosquitoes used protective measures (Lindsay and Birley, 1996).

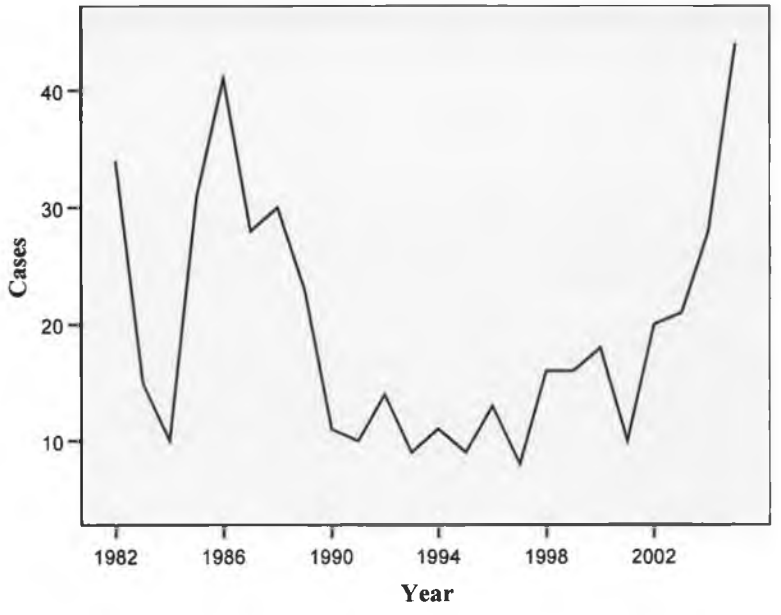
8.4.5 Future estimates

Future estimates of the incidence of malaria have been undertaken in the U.K., the U.S., and Australia. Five indigenous species of mosquito are capable of transmitting malaria in the UK, all of which can transmit P. vivax, but An. atroparvus which is most suited, prefers brackish water along river estuaries (Rogers *et al,* 2001). If coastal defences are breached slowly in the UK, salt marshes may develop, thus increasing the habitat of An. atroparvus. The risk of malaria is predicted to increase in the south of the UK, and spread northwards to Scotland (Rogers *et al,* 2001). While it was considered that the present standards of living would reduce the risk of malaria, it may not do so completely in high risk areas. Although the land may be drier in the future, areas suitable for the survival of the mosquito may also increase, from inflows of sea water arising from rises in sea-level, and which might be expected to increase salt marshes, and provide breeding sites (Rogers *et al,* 2001). The report concluded however, that it would be difficult to envisage a situation whereby public heath infrastructure would break down and indigenous malaria would return to the UK, although small outbreaks might occur near airports. A similar conclusion was reached using modelling techniques in the U.K., using data from the 18th century. It was estimated that a rise of between 1 and 2.5^{0}C in the average UK temperatures would increase the risk of local malaria transmission by 8-15%, but that this increase could easily be addressed by the health system at present (Kuhn *et al,* 2003), and an efficient public health service in addition to the draining of wetlands would militate against a rise in the incidence of malaria. The

US assessment did not model possible future impacts, stating that more research is needed on the behaviour and vector competence of the mosquito (Gubler *et al,* 2001). Modelling techniques were used in both Australia and the U.K. In Australia, it is estimated that with an increase of 10% in rainfall and an increase in temperature of 1.5^0C by 2030, the distribution of mosquitoes would extends 800 km southwards in Australia, (Bryan *et al,* 1996). Nevertheless, the possibility of re-emergence of malaria in Australia was thought to be 'limited' by the Australian national impact assessment because of the existence of an efficient public health system (Mc Michael *et al,* 2002).

8.4.6 Present incidence of malaria in Ireland

Malaria is not endemic in Ireland and cases of malaria are generally associated with people travelling to Ireland from areas where it is endemic, in people who did not avail of full prophylactic treatment while abroad or are relapses of an earlier illness (HPSC(b), 2006). Approximately three quarters of the cases reported in 2004 and 2005 were the more serious P.Falciparum (HPSC, 2005(a);HPSC (b), 2006). The incidence of malaria in Ireland has appeared to have fallen since a peak in the mid 1980s, when there was a peak of 40 cases in 1986 (Figure 8.5). The reasons for the high numbers between 1985 to1989 are unknown (Garvey, 2004).



(after; HPSC, 2005 (a), HPSC 2006))

Figure 8.5 Number of cases of malaria in Ireland 1982 to 2005

At present, there are seventeen species of mosquito in Ireland, although it is possible that more remain to be discovered, as there are thirty two species in the UK (Ashe *et al,* 1991). In Ireland, An. atroparvus, An. algeriensis, An. claviger and An. plumbeus are capable of transmitting P. vivax. However, An. atroparvus is considered the most important potential vector of malaria in the UK, as the distribution of this anthrophilic mosquito is coincident with the historical distribution of the disease in the U.K. (Nash, 2006).

8.4.7 Future estimates of malaria in Ireland

The three criteria for malaria to become endemic will be applied to ascertain the possible future incidence of malaria in Ireland. Firstly, the mean summer temperature in Ireland, in the time period 1981-2002 was 15^0C, which is the threshold for the development of P.vivax. The climate around Dublin Airport is therefore climatically suitable for the survival of the Anopheles mosquito during the summer months (Figure 8.4) (Tatem *et al,* 2006), and in addition, mean summer temperatures are expected to rise by approximately 2^0C by 2050 (Table 3.7). Secondly, the mosquito, Anopheles atroparvus is already here (Nash, 2006), and, in addition, there are 238 salt marshes in Ireland at present (Curtis and Sheehy-Skeffington, 1998). However, the third criterion may not be met, namely that there would be sufficient number of people with infected parasites in their blood, although increases may be expected in the number of cases of malaria arising from the numbers of people travelling back to Ireland from areas of the world where malaria is endemic, and immigrants where the condition is endemic. Such areas may be expected to increase as global warming increases.

Nevertheless, the possibility exists that airport malaria may occur, particularly if there are salt marshes in the vicinity of airports. However, it is unlikely that malaria would get a foothold in Ireland; existing public health resources, including disease surveillance, medical treatment and surface water management would make the re-emergence of malaria unlikely in Europe (AR4, 2007). Nevertheless, with the growth of international travel, the need for continued surveillance and prompt investigations has been stressed, alongside effective disinfection procedures on planes (Layton *et al,* 1995; Gratz *et al,* 2000; Tatem *et al,* 2006). In addition, as part of a long term strategy for the control of malaria in Europe, the WHO Regional Office has set up the European 1998-

2000 Malaria Surveillance and Control Programme, with the aims of establishing surveillance systems, and strengthening national capabilities in case detection and treatment (WHO, 2001(b)). Guidelines have also been published in order to prevent a locally acquired mosquito-transmitted malaria outbreak from becoming a source of sustained transmission (Filler *et al,* 2006).

In conclusion, the multi-factorial aetiology of malaria makes it difficult to accurately quantify the impact of climate change on the incidence of malaria, in Ireland. It is evident that a rise in mean temperatures will enable the development of the Plasmodium in the mosquito in Ireland. Nevertheless, while sporadic cases of malaria may be expected, in particular near international airports, the risk of emergence of malaria in Ireland, in the absence of a collapse in public health structures, appears to be unlikely.

8.5 West Nile Virus

West Nile virus (WNV) is a viral illness transmitted by mosquitoes, who have fed on infected birds, and was first isolated and identified in 1937 in an infected person in the West Nile district of Uganda. It is a febrile illness with a sudden onset, and may be accompanied by nausea and vomiting, muscle pain and a swelling of the lymph nodes. It is generally a self-limiting illness, lasting from 3-6 days and up to 80% of infections may be mild or go unnoticed. However, approximately 1 in 150 cases will develop severe neurological complications and the case fatality rate may rise to 7% of these severely affected (HPSC, 2006 (a). West Nile Virus was first identified in the Western Hemisphere in New York in 1999 (Nash *et al,* 2001). Since then, it has been documented in 27 states (Petersen and Marfin, 2002). Peak incidence occurs in late summer, in the USA, although onset has occurred from July through December (Petersen and Marfin, 2002).

8.5.1 Incidence

There were three cases of West Nile Virus in Western Europe in 2003 (HPSC 2005(c). The first Irish cases were reported in July 2004, when two Irish tourists who had travelled to Portugal were diagnosed upon their return to Ireland (HPSC, 2006(a)). West Nile Virus does not yet present a major hazard to humans in most parts Europe

(Higgs *et al,* 2004), and there appear to be few situations in the British Isles where humans and livestock are exposed to sustained risks of exposure to potential WNV vectors (Medlock *et al,* 2005). West Nile Virus is not a notifiable disease in Ireland. The public health risk of acquiring WNV in Ireland is considered to be minimal, and strict precautions are in place to minimize contact with the disease through blood transfusions (HPSC, 2006(a)). The link between climate and outbreaks of West Nile Virus is unclear and animal surveillance has been recommended as an early warning system (WHO, 2005).

8.6 Other viruses

Wildlife is increasingly becoming an important source of infection for humans (Cunningham, 2005), and the combination of susceptible hosts, vectors, and a suitable climate for transmission may increase during the 21st century (Gould *et al,* 2006). Relatively rare vector-borne diseases may therefore become more apparent in the future. St. Louis encephalitis and other viral encephalopathies could extend into currently unreceptive northern areas in the US (Reeves *et al,*1994), and climate-induced changes in sand fly abundance could also increase the risk of the emergence of new diseases such as leishmania, currently found in Southern Europe at present (cCASHh, 2006). However, although antibodies to other rare viral diseases carried by vectors, such as Sindbis virus, Usutu virus, Tahyna virus and Uukuniemi virus have been found in Ireland and the UK no imminent threat of an epidemic exists, as the requirements of susceptible hosts, suitable conditions for transmission and dispersal render this unlikely (Gould *et al,* 2006). Nevertheless, monitoring of mosquitoes and viral surveillance are required to guard against the introduction of more anthropophilic mosquitoes and outbreaks of diseases (Medlock *et al,* 2005). A framework for the assessment of the risk posed by vector-borne disease has been developed (Table 8.2).

Parasite	None present	Imported cases only	Low prevalence in vectors/hosts	High prevalence in vectors/hosts
Vector				
None present	No risk	No risk	No risk	No Risk
Local distribution	No risk	Very low risk	Low risk	Low Risk
Regional distribution	No risk	Very low risk	Low risk	Medium Risk
Widespread distribution	No risk	Very low risk	Medium risk	High Risk

(*after Santos et al, 2002*)

Table 8.2 Risk of vector borne disease

In conclusion, no report has clearly associated vector-borne diseases with increases in temperatures to date, and it appears that because of the low prevalence of vector-borne disease in Ireland, that the risk at present to human health is low. However the emergence of an infectious disease is a complex process, involving biological, social and environmental processes and climate-related impacts on health are only one factor in the context of other influences on disease dynamics, including such issues as land-use and the effectiveness of public health infrastructures. However, the low endemicity of vector-borne illness in Ireland, leads to the conclusion that while ongoing surveillance is necessary, the risk to human health from the impact of climate change on vector-borne disease in Ireland, is low.

8.7 Allergies

The main determinant of pollen concentrations is land use and farming practice (Emberlin, 1994). However, increased ambient temperature may have significant effects on the distribution and overgrowth of allergenic plants (AR4, 2007). There appears to be a disproportionate rise in pollen production in response to an increase in carbon dioxide concentrations in some plants, e.g. ragweed and birch (IPCC, 2001; Rogers *et al,* 2006; AR4, 2007). Furthermore, higher temperatures and lower rainfall at the time of pollen dispersal are also likely to result in increased concentrations of airborne pollen during the peak season (Emberlin, 1994), and the pollen season itself may start earlier (Rogers *et al,* 2006). In addition, stronger allergenicity has been reported in the pollen from trees grown at higher temperatures (Beggs, 2004).

Attribute	Plant	Co_2 concentration	Change
Pollen	Ragweed (2 studies)	280 to 370 p.p.m. 370 to 600 p.p.m.	132% increase 90% increase
Shoot growth	Rye grass	390 to 690 p.p.m	17% increase

(after Beggs et al, 2004)

Table 8.3 Impacts of elevated carbon dioxide on allergenic plants

The impact of rising carbon dioxide levels on pollen production of allergenic plants has been quantified and the results quantified (Table 8.3).

8.7.1 Future estimates of allergic disorders in Ireland

A rise in global asthma has been postulated as an early health effect of climate change (Beggs and Bambrick, 2005). The incidence of allergic disorders appears to be rising in Europe (Upton *et al* 2000; Ring *et al,* 2001), and an increase in hayfever in the summer of 2006 has been attributed to the hot weather experienced that year (Emberlin, 2007). The growth of ragweed will increase and be seen over a larger geographical distribution in Ireland (Finn, 2006). However, it is difficult to assess the prevalence of allergic disorders in Ireland. There is no national register of asthma, and in addition, data on the amount of medications dispensed for allergic disorders is not collated nationally. There is also no national database on allergenic pollen trends in Ireland. Nevertheless, an international survey indicated that the incidence of asthma in Ireland was third after Australia and the UK (Asthma Care Ireland, 2006), and estimates of 450,000 people in Ireland suffering from asthma have been made, over half of these being children (Manning, 2006). This indicates that a substantial proportion of the population have a propensity to allergic disorders, and this may be expected to change and possibly increase with the impact of climate change.

8.8 Flooding

Climate change will increase the risk of river and coastal flooding (IPCC, 2001; AR4, 2007). In Europe, floods are the most common natural disaster (Hajat *et al,* 2005) and the most costly in economic terms (WHO 2002(c)). The Bruun rule of erosion states that for every centimetre rise in sea-level, a retreat in land of approximately 1 metre on sandy coastlines can be expected (UNESCO, 1997), and a rise in sea-level of 0.49

metres with storm surge events and high tide frequencies will put at risk approximately 300 square kilometres of land at risk of flooding in Ireland (Sweeney *et al,* 2003).

8.8.1 Health impacts of flooding

The health impacts of flooding have been classified into those that occur at the time of flooding and those that occur after the floods have receded (IPCC, 2001; Hunter 2003). Immediate health impacts include death by drowning, although this is rare in temperate countries. Following an episodes of flooding, the incidence of gastrointestinal illness increases, particularly if there is a septic tank on the property (Wade *et al,* 2004). Flooding may also markedly decrease the quality of river water, and infectious diseases might be expected to rise in the aftermath of a flood, particularly if water purification facilities are overwhelmed. The marked deterioration in the quality of source water following a heavy flood in the USA in 2001 is depicted in Appendix J. Following an episode of severe flooding in Lewes, in Southern England, in 2000, earache, skin rashes, exacerbations of asthma and mild gastroenteritis were reported (Reacher *et al,* 2004). In the time period following a flood, moulds may develop on interior surfaces, and may lead to respiratory problems, particularly in infants ((American Academy of Paediatrics, 1998). Although microbiological pollution is the main concern arising from increased rainfall and flooding, there is also the possibility of water pollution. Following major floods in Dresden in 2002, oil tanks, old mines, water treatment plants and other facilities were flooded and initially large quantities of heavy metals, pesticides and other harmful substances were released into the flood waters. However, the levels reduced quickly due to large dilution factors (Meusel and Kirch, 2005). In addition, contamination by trihalomethanes may occur when the carbon in peat areas combines with chlorine in water purification systems, during flooding episodes. These compounds have been incriminated in the aetiology of bladder cancer (EEA/WHO 2002).

Longer term impacts on health resulting from flooding include psychological problems such as depression, anxiety and insomnia (WHO, 2002(b).The major adverse impact from the Lewes flooding appeared to have been psychological. Almost a half of respondents scored significant levels of psychological distress both at the time of the survey, and 9 months after the flood occurred. However, the authors remark that casualties could have been much higher if the floods had occurred at night, if there had been no prior warning, and also if the weather had been colder. Similarly, in a follow up

one year after floods in Brisbane, psychological symptoms, including irritability and depressed mood persisted (Abrahams *et al,* 1976). It is often only after peoples' homes have been put back in order that the full psychological impact is apparent (Hajat *et al,* 2005), and increases in suicide, alcoholism, and psychological and behavioural disorders, particularly among children, were reported following floods in Poland in 1997 (IPCC, 2001). In addition, flooding may damage infrastructure, impacting on electricity and the running of hospitals and other essential services. The importance of autonomous power generating facilities in flood prone areas has been stressed (Meusel and Kirch, 2005).

8.8.2 Flooding in Ireland

In Ireland, many areas have histories of seasonal flooding, such as the Shannon, and the first account of a weather related event in Ireland or Britain was recorded in the Annals of the Four Masters, concerning an event in Lough Conn, allegedly in 2,688 B.C. Later, in 801 A.D. the Annals of Clonmacnoise recorded:

> *"There was such horrible and great thunder the next day before St. Patrick's Day, that it put asunder one thousand and ten men between Corck Bascynn (west Co. Clare) and the land about it; the sea divided an island there in three parts, the seas and sands therefore did cover the earth near it.".* (Annals of Clonmacnoise)

At present, river flooding is a problem at present in a number of towns and cities in Ireland, such as Waterford, Clonmel, Mallow and Cork, and the vulnerability is compounded by underlying geology (Charlton and Moore, 2003). Flooding may also be exacerbated in coastal areas where there is interaction between high tides and river flows, and is likely to become more widespread, as the impacts of climate change become more pronounced, from both increased run-off and also sea-level rise (Charlton and Moore, 2003). Vulnerability to flooding is also increased by the use of tarmacadam and other non-permeable surfaces that do not allow the absorption of rain by the earth, instead conducting it to river channels, thereby increasing flood peaks (Charlton and Moore, 2003). No fatalities are on record from storms of floods suffered while at work (Slater, 2006), although fatalities have occurred in rivers during times of flooding.

8.8.3 Future estimates of flooding in Ireland

With the impact of climate change, seasonal flooding is likely to be more extensive than at present and to persist longer into the spring (Sweeney *et al,* 2003). In Ireland, the impact of sea-level rise will be most apparent in Cork, Dublin and Galway (Fealy, 2003). The prevention and management of flooding episodes is in the remit of local authorities, who prohibit the building of houses on flood plains, and who also implement river management measures, for example the Camac river in Dublin (Murray, 2000). The requirements of a national flood warning system have been outlined (Reville, 2004); and include accurate rain forecasts, procedures by which the public may be informed and protected, instantaneous updating of expected river flood levels and coordination procedures established between the many statutory agencies involved. In 2004, the Office of Public Works was confirmed as the State's lead agency on flooding, and the Report of the Flood Policy Review Group was published in that year by the Office. This report outlined the work programmes to be undertaken by the statutory authorities, in relation to such issues as maintenance of water channels and emergency responses. A guidance document for the population was issued by this Office in 2007, giving advice on strategies to cope with household flooding.

8.9 Skin cancer

The ozone layer shields the earth from harmful ultra-violet radiation, and is found in the stratosphere, 15-30 kilometres high in the atmosphere. It has been damaged by chemical pollution. However international agreements have banned the production of many ozone destroying chemicals and there is now evidence of a recovery (NASA, 2003). Exposure to ultra-violet radiation is linked to skin cancer and cataract formation in the eyes (Mc Carty and Taylor, 2002) and damage to the immune system (Duthie *et al,* 1999). The link between exposure to sun and the incidence of skin cancer is well established, however, the latency period may be over twenty years (Han *et al,* 2006). An increase in sunburn may be expected with increased solar radiation, and the risk of sunburn has already increased in the Arctic regions where ozone levels are low (GEO, 2006).

8.9.1 Skin cancer in Ireland

Skin cancer is the commonest cancer in Ireland, accounting for over almost a third (31%) of all cases of cancer in the years 1994-2001 (National Cancer Registry, 2005). The majority of these skin cancers are non-melanotic, with a good prognosis. However, 2% of the total cancers are melanotic, which are more serious, and in contrast to non-melanotic skin cancer, this form of skin cancer is increasing. The 5 year survival for men and women from this condition is approximately 80% and 90% respectively, and of those who succumb, 44% are under 65 (Campo *et al,* 2004). There is a regional disparity in the incidence of skin cancer, with a higher incidence of melanotic skin cancer in the South of the country, and a higher incidence of non-melanotic skin cancer in the East, however the reasons for this are not known (National Cancer Registry, 2005). While the majority of Irish people are aware that unprotected exposure to the sun increases the risk of skin cancer, the use of sunscreen is disproportionately low, and very few people consider the use of other measures such as protective clothing; it appears that peer pressure and fashion play a disproportionate role (Murphy. 2002).

8.9.2 Future estimates of skin cancer

It is probable that increased exposure to sunlight, in the absence of protective measures, will result in a rise in the incidence of skin cancer, and that the increases will outweigh any reduction that may arise from the restoration of the ozone layer (Diffey, 2004). The future incidence of melanotic skin cancer has been estimated by the National Cancer Registry. Increases from the 1998 to 2002 baseline period, in the region of 115% for men and 95% for women, are anticipated (National Cancer Registry, 2006 (a)). These estimates do not include allowances for climate change (National Cancer Registry, 2006 (b)). The evidence that the younger population are adopting protective behaviour practices in relation to sun exposure is disappointing (Cokkinides *et al,* 2006), and the absence therefore of a change in behaviour, leads to an expected increase in the incidence of skin cancer.

8.10 Other indirect effects

Other indirect effects of climate change include changes in levels of air-pollutants, and possible social and economic disruption. At present not enough data is available on the impacts of climate change on air quality and this area could be the subject of further research. Data collated from PHEWE (Assessment and Prevention of acute Health Effects of Weather conditions in Europe) may be useful for future work in this area. Mercury levels in fish have been predicted to rise, as the formation of the more toxic form of mercury (methyl mercury), which accumulates in the body, is enhanced by warmer waters (Booth and Zeller, 2005). Warming may also increase the concentration of radon (a carcinogenic agent), in the lower atmosphere (IPCC, 2001).

8.11 Conclusion

Climate change will significantly impact on many facets of health of Irish people in the future. Increases in the incidence of Lyme disease may occur, and while the risk of malaria and other vector borne diseases will increase, the possibility of these diseases becoming established appears remote. The incidence and prevalence of allergies will change and injuries and morbidity and mortality from flooding episodes may occur. In the absence of a change in behaviour in relation to sun screens and other protective interventions, further increases in the incidence of skin cancer may also be expected.

CHAPTER 9 - CONCLUSIONS AND RECOMMENDATIONS

> *"It will no longer be possible for us to exclaim surprise at these climatic events and their consequence. We must reinforce policies for forecast, alert and prevention".*
>
> (Brucker, 2005)

9.1 Introduction

This aim of this thesis was to identify populations at risk from adverse health impacts from projected climate change scenarios, particularly those most vulnerable and to make recommendations regarding early warning systems that would enable the detection of future health-related changes in Ireland. Quantitative assessments were developed of future changes in temperature related mortality, and this study also reports the first quantitative estimates of future changes in morbidity from specific food-borne illnesses. Qualitative changes in the incidence of water-borne illness and the impacts of other indirect impacts of climate change on health were also estimated. The findings of this study were discussed as factors in the *'complex web'* (Bernard and Ebi, 2001), of other determinants of health. In terms of climate change, vulnerability refers to the degree by which a system is able to cope with adverse changes, and is dependent on the magnitude and rate of change, alongside the sensitivity of the system (IPCC, 2001). As a result of the findings, a picture was established of the national vulnerability to the health impacts of climate change, and arising from this, several recommendations may be made.

9.2. Direct impact

9.2.1 Temperature related mortality

An analysis of Irish daily temperatures and mortality over a twenty-two year period shows a clear relationship between temperature and mortality. This relationship was consistent with the national and international literature and confirmed the benefit of warmer temperatures to health. The most significant impact of future changes in mean temperature will be reductions in mortality, specifically in respiratory and cardiovascular mortality, and particularly in those aged over 65 (Table 9.1 and 9.2).

Age group	2010-39 Change % mortality	2040-69 Change % mortality	2070-99 Change % mortality
15-64	-1.0 (-0.7 to -1.6)	-1.7 (-1.0 to -2.3)	-2.2 (-1.3 to -2.6)
65-74	-1.5 (-0.9 to -2.3)	-2.6 (-1.3 to -3.4)	-3.6 (-2.2 to -4.5)
75-84	-1.8 (-0.5 to -2.8)	-3.0 (-1.5 to -3.4)	-4.2 (-2.5 to -5.4)
Over 85	-2.0 (-0.5 to -3.1)	-3.5 -(1.8 to -4.7)	-4.9 (-2.9 to -6.3)
Total	-1.5 (-0.4 to -2.3)	-2.6 (-1.3 to -3.4)	-3.5 (-2.1 to -4.6)

Table 9.1 Mean and range of future estimates of percent change in age-specific mortality rates resulting from increases in mean temperatures from the mean mortality rate in the adjusted time series 1981-2002

Disease category	2010-39 Change % mortality	2040-69 Change % mortality	2070-99 Change % mortality
Cardiovascular	-1.5 (-0.8 to -2.2)	-2.6 (-1.2 to -3.5)	-3.7 (-2.0 to -4.7)
Ischaemic Heart disease	-1.4 (-0.8 to -2.3)	-2.5 (-1.1 to –3.4)	-3.4 (-1.9 to -4.6)
Stroke	-0.9 (0 to -2.7)	-2.6 (-0.9 to -3.7)	-1.8 (-1.8 to -4.6)
Respiratory	-3.5 (-0.8 to -4.8)	-6.0 (-3.2 to -8.1)	-8.5 (-4.8 to -10.5)

Table 9.2 Mean and range of future estimates of changes in mortality in relation to disease category, resulting from increases in mean temperatures from the mean mortality rate in the adjusted time series 1981-2002

Higher maximum temperatures will occur, and the number of days when maximum temperatures will exceed the threshold at which mortality increases has been quantified (Tables 5.14 and 5.15). This will undoubtedly result in increases in summer mortality, although such impacts are difficult to quantify. Although not a significant problem in Ireland at present, increases in the levels of ground-level ozone may contribute to increased mortality on very hot days. However, due to the dominant impact of cold on mortality rates, it may be expected that overall mortality will decrease as a result of future increases in temperatures.

Vulnerability

There are indications of vulnerability in people suffering from respiratory and cardiovascular diseases, resulting from the impacts of higher maximum temperatures. A possible vulnerability in the over 65 year age groups, and in particular in the over 85 and the under 14 year age groups has also been identified. However mortality from heat-waves is essentially avoidable (Semenza *et al,* 1996; WHO, 2003(a)), and mortality rates have reduced in the U.S. following the implementation of such plans (Weisskopf *et al,* 2002). A heat wave health alert system, established in France following the tragic outcome of the 2003 heatwave, was brought into operation in July 2005, when temperatures were less intense, but lasted longer than in the heat wave of 2003. While a 6% increase in total mortality, and specifically a 10% increase in mortality in the over 75 year age group was recorded, analysis of the meteorological data, demonstrated that over 4,000 deaths had been saved as a result of the heat alert plan (Houssin, 2006).

The impact of heat waves also raises challenges in how we care for the most vulnerable in the population, including the nature of housing and social care that is available to older people both in the community and also in nursing homes (Brucker, 2005). Air conditioning has been described as being of critical importance in the prevention of heat-related deaths (Rogot *et al,* 2002), although the number of buildings in Ireland that have air conditioning in Ireland is unknown (Forsythe, 2005). The current National Emergency Plan, launched by the Department of the Environment in 2008, does not contain plans for a heat-wave.

Early warning systems

The importance of reactive health surveillance systems in the prevention of heat-related deaths has clearly been demonstrated, and enhanced surveillance of routine health data for early detection of heat-wave effects has been recommended (Haines *et al,* 2006). Consideration must be given to the establishment of an Irish national heat wave action plan. Such plans have been developed in many European countries including the U.K.,

Portugal and Italy (WHO, 2004(a)). The essential requirements of such a plan have been outlined (Whitman *et al,* 1997; WHO, 2004 (a); Kovats and Ebi, 2006; Houssin, 2006), and criteria for their evaluation have been suggested (Bernard and Mc Geehin, 2004; Kovats and Ebi, 2006). The heat wave plan in the U.K. is initiated when temperatures exceed a regionally specific temperature threshold (averaging 30^0C in the daytime and 15^0C at night), and comprises four graduated levels of responses. Advice is issued to the general population and to health workers, particularly those working with at-risk groups, to prioritize those most vulnerable (U.K.Department of Health, 2006).

9.3. Indirect impacts

9.3.1 Water-borne disease

Although it was not possible to quantify future changes in the incidence of waterborne diseases, there is a clear risk to water quality from climate change, resulting from increased rainfall and resultant flooding, and a resultant increase in the risk of waterborne diseases such as cryptosporidiosis, VTEC and giardia where water quality is compromised. In addition, there will also be risks associated with shortages of water, particularly in the East of the country.

Vulnerability

The total population is vulnerable to water-borne disease. However, waterborne disease will impact most severely on the sub-group of the population whose immune systems are compromised. Resulting from increased rainfall, the quality of drinking water may deteriorate and two particularly vulnerable sections of the population have been identified. Firstly, in the absence of remedial action, there is a serious risk of an increased incidence of VTEC, cryptosporidiosis and giardia in the sub-group of the population whose drinking water comes from private group water schemes. Secondly, there is also a risk of such diseases in people who consume water from both public and private water supplies in the absence of adequate functioning of water treatment systems which may be under pressure from increased rainfall. It is crucial that the new Drinking Water Regulations are effectively implemented, and that the objectives of the European Water Framework are achieved.

The risk of infection contacted by the recreational use of water will increase and the need to upgrade urban waste water treatment plants must be a priority. Consideration should be given to the licensing of swimming pools to ensure water quality and to all facilities that offer jacuzzis and spa treatments. Training must be provided for mangers of swimming pools, spas and leisure centres on the correct procedures to be adopted to minimize the possibility of microbial infection with such organisms as legionella and cryptosporidium. Resulting from decreased water availability in the summer months, particularly in the east, measures to increase water conservation will become increasingly important.

Early warning systems

Increased monitoring of water quality during episodes of heavy rainfall will be necessary. It will be important to examine changes in the timing, duration and spatial distribution of cases of cryptosporidiosis and serotypes of VTEC, in conjunction with information on precipitation patterns and flooding, in order to detect the impacts of climate change on water-borne disease. Monitoring of algal blooms will be necessary and consideration also given to making diseases that arise from contact with these organisms statutorily notifiable. Better understanding of the mechanisms by which waterborne pathogens can be adequately controlled has been called for (Betancourt and Rose, 2004), and similar to the impact of climate change on food-borne disease, the advent in 2004 of statutory regulations governing the reporting of infectious diseases in Ireland augurs well for future monitoring of the health impacts of climate change on the incidence of water-borne disease.

9.3.2 Food-borne disease

This is the first study identified that quantified changes in future morbidity from food-borne disease. A clear relationship between mean temperature and food-borne disease was evident, in particular, for salmonella, campylobacter and VTEC, the incidence increasing as temperature rises. A threshold temperature of 2.1^{O}C was identified for salmonella and the optimum temperature range for the growth of campylobacter was identified between 6 ^{O}C and 17 ^{O}C.

	Salmonella per 100,000 (% increase)	Cases of Campylobacter (% increase)	Cases of VTEC 0157 (% increase)
2010-39	0.2 (1.3%)	65 (3.7%)	8 (12.7%)
2040-69	0.3 (2.4%)	106 (6.0%)	11 (18%)
2070-99	0.5 (3.6%)	144 (8.1%)	16 (26%)

Table 9. 3 Mean annual future increases in the number of temperature related cases of food-borne diseases per year (and % reduction in mortality rates) resulting from increases in mean temperatures

Although increases in the number of cases of salmonella and campylobacter will occur, the increase in VTEC infection, a serious and sometimes fatal disease with possible life long serious sequalae is of particular concern (Table 9.3).

Vulnerability

The prevalence of food-borne diseases in the community results in the entire population being vulnerable to increases in such diseases. In particular, people whose immune systems are weakened are at particular risk, as are people whose hygiene practices are compromised. The importance of kitchen hygienic practices, and refrigeration, both at home and in public facilities must be kept to the fore by the statutory authorities.

Early warning systems

It will be necessary to monitor changes in the timing, duration and spatial distribution of cases of salmonella, campylobacter and serotypes of VTEC, in conjunction with temperature records in order to detect the impacts of climate change on food-borne disease. Close liaison between the meteorological and both animal and health surveillance systems will be necessary. Similar to the situation with water-borne disease, the statutory regulations governing the reporting of infectious diseases, which came into effect in 2004, augurs well for future monitoring of the health impacts of climate change on food-borne disease.

9. 4 Vector-borne disease and climate change

The complex lifecycle of the vectors of infectious diseases results in difficulties in estimating the incidence of diseases carried by these insects.

9.4.1 Lyme disease

Vulnerability

People whose occupations or leisure interests result in time spent outdoors in areas where the tick density is high are at risk of Lyme disease; such areas may change as temperatures increase.

Early warning systems

Geographical Information Systems may be used to ascertain changes in land-use patterns associated with climate change, and in the case that high risk areas are identified, educational campaigns may be undertaken on the necessity for protective clothing. In addition, consideration should be given to making Lyme disease statutorily notifiable.

9.4.2 Malaria

Increased temperatures will increase the risk of malaria, by enabling the causative plasmodium organism to develop in the host vector, the mosquito.

Vulnerability

The complex lifecycle of the plasmodium, in particular the lack of people infected with the causative organism, renders it unlikely that malaria will become endemic in Ireland, although there will be a risk of isolated cases, near international airports. People residing near low lying salt marshes will be most at risk, and in particular, people living near salt marshes adjacent to international airports.

Early warning systems

The importance of adequate prophylaxis for people travelling to lands where malaria is endemic must be stressed, and prompt investigation of cases of malaria will be necessary. In addition, surveillance will be required to guard against the introduction of other anthrophilic mosquitoes and vectors carrying other viral diseases. Disinfection procedures on planes may be necessary.

9.4.3 Allergies

The incidence and timing of allergies may change with higher temperatures.

Vulnerability

Children and atopic adults are most prone to allergies. As higher temperatures will enable plants to flourish that may not have previously grown here, the incidence of allergies may change with the exposure of the population to the pollen of new species.

Early warning systems

Surveillance systems will be necessary that link the seasonal distribution of pollen, with data from health facilities, in order for health alerts to be issued (Haines and Mc Michael, 1997; Wilkinson *et al,* 2003). In addition, pollen forecasting and pollen avoidance strategies for sensitive individuals will be required (Rogers *et al,* 2006).

9.4.4 Flooding

Increases in flooding will also undoubtedly result in increases in infectious diseases and psychological problems for those affected. Practical advice and assistance, including shelter and access to medical services will be necessary if flood mitigation measures fail. Local authorities are the responsible authorities for the emergency response.

Vulnerability

Particularly vulnerable groups living in flood-prone may be identified, and include children, older people, people who are immobile, and those who do not speak the national language (Ebi, 2006).

Early warning systems

The ability to predict the weather conditions that result in flooding is improving, and flood warnings, combined with an appropriate emergency response has been effective in reducing fatalities (Ebi, 2006). Close collaboration between the many state agencies who have responsibility in this area will be necessary, including the health authorities, as health education on such issues as the importance of boiling water will be necessary if water treatment facilities are compromised (Haines *et al*, 2006). Information on assessing the risk of flooding, and practical advice is available from the Office of Public Works on www.flooding.ie and the current National Emergency Plan, launched by the Department of the Environment in 2008, and distributed to all householders.

9.4.5 Skin cancer

Although increased exposure to the sun will undoubtedly result in an increase in the incidence of skin cancer, the long latency between exposure to the sun and the development of skin cancer makes it difficult to estimate the possible future incidence of this disease. The future incidence will depend on the behaviour of the population in relation to both exposure to the sun and the use of sun screens and other protective measures.

Vulnerability

People whose spend much time out of doors without adequate protection will be at increased risk of skin cancer. The evidence that the younger population are adopting protective behaviour practices in relation to sun exposure is disappointing, both in the United States (Cokkinides *et al,* 2006) and Ireland (Murphy, 2006). Therefore, the absence of a change in behaviour, an increase in the incidence of skin cancer may therefore be expected.

Early warning systems

It is recommended that warnings be given with the weather forecasts in order to alert people to an increased risk when hotter temperatures are expected. In addition, it will be necessary to implement educational programmes on the importance of minimizing exposure to the sun. Close collaboration between with meteorological service, the health authorities and the broadcasting agencies will be necessary.

9.5 Vulnerability

This work is a preliminary step on the path to ascertaining the future impacts of climate change on health in Ireland, and to identifying the populations that are most vulnerable. Already, it appears that the majority of adverse health impacts from climate change are occurring in vulnerable people in poorer countries (AR4, 2007), although the heat-wave of 2003 affected many sick and older people in Europe. A similar picture is emerging in Ireland, where the most vulnerable people are those who are suffering from respiratory and cardiovascular disease and also people on lower incomes in the population, who may not be in a position to install air-conditioning. As advancing age increases vulnerability to ill-health generally, the increasing age of the Irish population will also increase susceptibility to heat-related problems. In addition, the population in cities is expected to increase, where temperatures are higher and people will be more vulnerable to the "heat island effect". While physiological adaptation may occur, and behaviours may change in order to minimize some of these impacts, the rapid and sustained rate in the increase in temperatures may militate against this, and adversely impact on the vulnerable groups identified.

Impact	Outcome
Temperature related mortality	
Warmer winters	Reduction in total mortality of between 9 to 26 per 100,000 per year (higher in older age-groups).
Hotter summers	Overall reduction in morality but possible increase in mortality from cardiovascular disease
Water-related diseases	Increase in incidence*
Food-borne diseases	
Salmonella	Increase of 0.3 cases per 100,000 per year
Campylobacter	Increase of 14 cases per year
E.Coli 0157	Increase of 11 cases per year
Vector-borne diseases	
Lyme disease	No significant change anticipated
Malaria	No significant change anticipated
Floods	Increase in incidence of flood related mortality and morbidity
Skin cancer	Increase in incidence

**quantification not possible due to lack of morbidity data*

Table 9.4 Summary of findings by mid century

9.6 Future work

This study has established statistical relations between climatic variations and health in Ireland, has identified populations at risk and has made recommendations in relation to early warning systems. The need for the establishment of surveillance systems between the meteorological service and the health service is the common theme of the recommendations of this work. Such surveillance systems would monitor changes in such variables as temperature and rainfall and the relationship with food and waterborne illnesses, temperature related mortality and morbidity, incidence of allergies and other illnesses.

The importance of monitoring and surveillance of the impacts of climate change on health has been stressed (WHO, 2008). Future work may involve the development of improved mathematical models to represent the climate-mortality and morbidity relationship, thereby enabling local and regional impact assessments to be made. Further work may also elucidate the impact of air-pollution on heat related deaths, and an assessment of the potential health

impacts of strategies that minimize greenhouse gas emissions. It will also be necessary to devise new methods of organizing our affairs that do not require such an investment of fossil fuels and our consumption of goods that require such intensive inputs. Dr. Margaret Chan, Director General of the World Health Organization noted that many of the actions to reduce our impact on the global climate system will also reduce pollution (Chan, 2007).

9.7 Final conclusion

Sustainable development has been defined as development that fulfils the needs of the present generation without endangering the needs of future generations. It is clear that, arising from the impacts of climate change, that meeting the needs of the present generation will endanger the fundamentals of good health, namely clean water, food and air for all. Climate change presents problems that are global and which will persist into the future. The uneven impact of climatic hazards raises humanitarian concerns for development and equity (IPCC, 2001). While these impacts will undoubtedly impact most severely in poor countries, who have fewer resources by which to adapt to these impacts, it will benefit the health of all people on our planet if emissions of greenhouse gases reduce. Public health has a role and a responsibility in this matter, and doctors have a particular responsibility in the fight to achieve urgent international reductions in carbon dioxide emissions (Kefford, 2006). Reduction in fossil fuel usage and strict adherence to international protocols for the reduction of carbon emissions are public health issues. It has been established in this work that older population and people who are ill and immune suppressed are the most vulnerable to adverse impacts of climate change on health. The challenge of climate change is also a global opportunity, by which the problems of both pollution and equity may be addressed, both nationally and internationally. This is the public health challenge of our time.

APPENDIX A

Trends in mortality rates 1981-2002

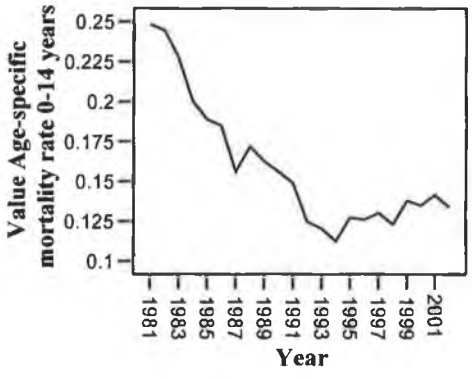


Figure 1 Trend in age-specific mortality 0-14 age group per 100,000 population per day

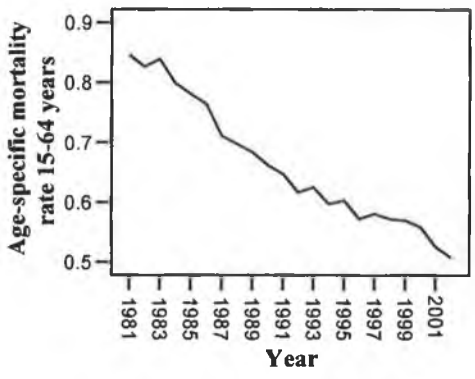


Figure 2 Trend in age specific mortality 15-64 age group per 100,000 population per day

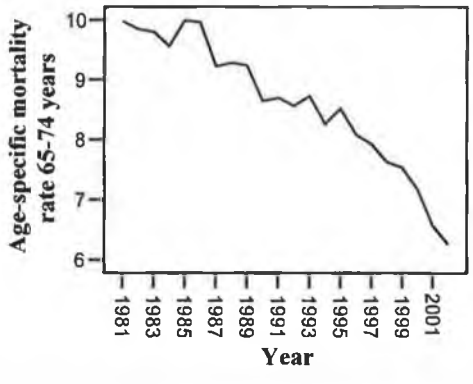


Figure 3 Trend in age-specific mortality 65-74 age group per 100,000 population per day

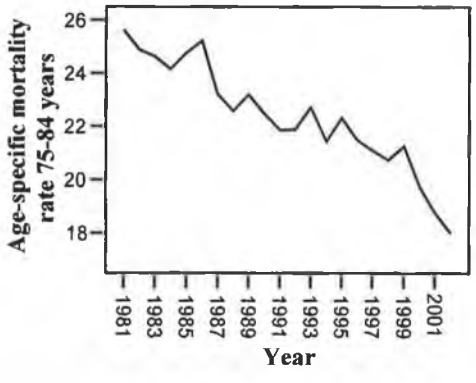


Figure 4 Trend in age specific mortality 75-84 age group per 100,000 population per day

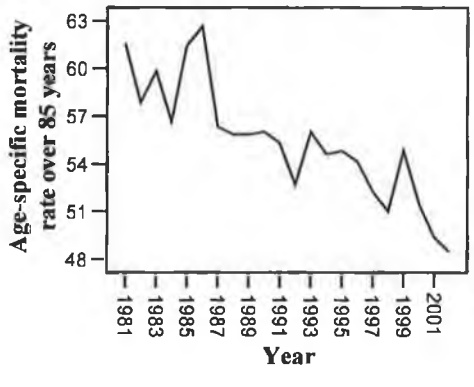


Figure 5 Trend in age-specific mortality in the age 85 and over age group per 100,000 population per day

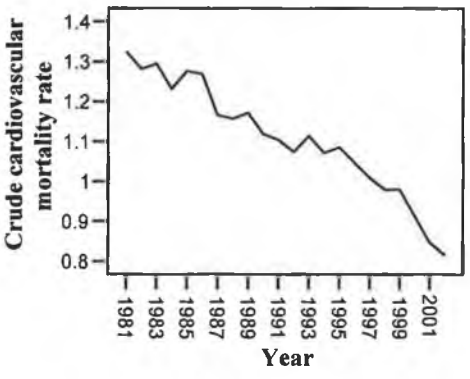


Figure 6 Trend in crude mortality rate from cardiovascular disease per 100,000 population per day

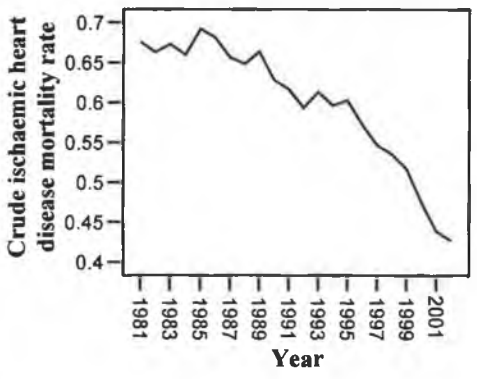


Figure 7 Trend in crude mortality from ischaemic heart per per 100,000 population per day

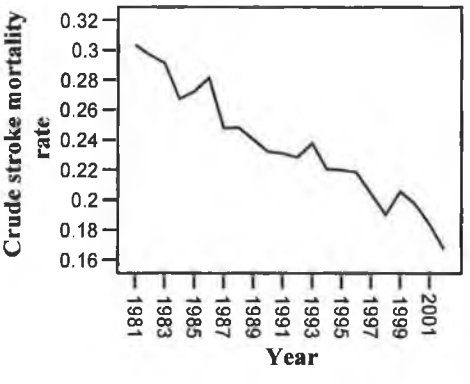


Figure 8 Trend in crude mortality rate from stroke per 100,000 population per day

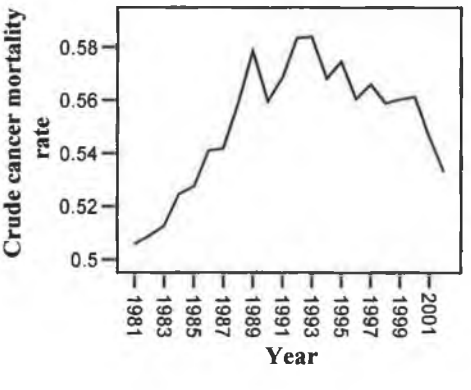


Figure 9 Trend in crude mortality from cancer per 100,000 population per day

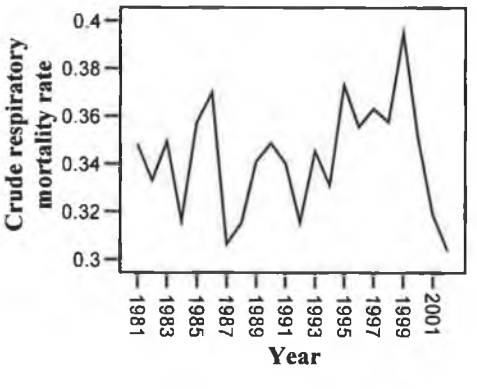


Figure 10 Trend in crude mortality from respiratory disease per 100,000 population per day

APPPENDIX B

Present impact of mean temperatures

Age 14 and under

While mortality is clearly higher at hotter temperatures in the under 14 age-group it appears constant for the majority of days in the time series, although a period of low mortality is evident in the temperature range 17^0C to 20^0C. However, mortality is low in this age-group and the typical U shaped curve in this age-group is not apparent (Figure 1).

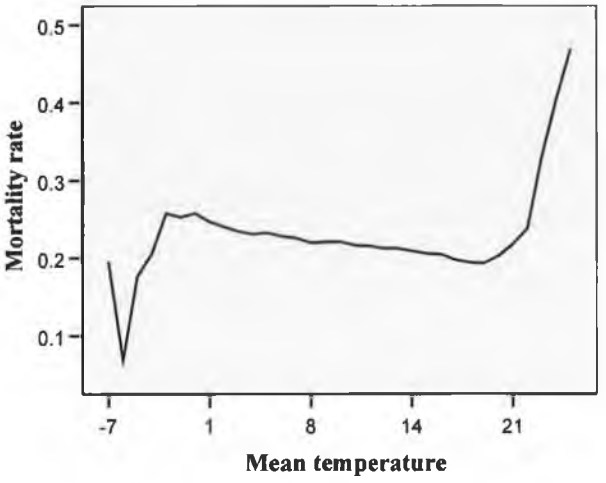
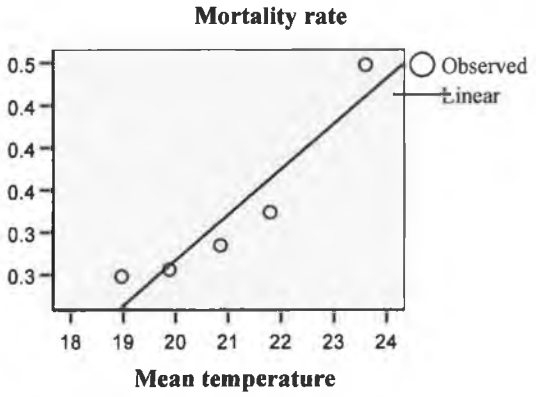


Figure 1(a) Relationship between daily mean temperature and mortality in the age-group 14 and under per 100,000 age-specific population.

At temperatures below 18.5^0C, it is not possible to derive a linear temperature-mortality relationship, however, at temperatures above this threshold, a linear relationship is apparent (Figure 1(b)).



R Square = 0.878 p<0.05

Figure 1(b) Relationship between daily mean temperature and mortality in the age-group 14 and under per 100,000 age-specific population above 18.5^{0}C

At these higher temperatures,

Mortality = -0.335+0.032 (t).

Mortality increases by 0.049 per 100,000 per day per degree rise in temperature, and mortality increases by 6.5%, over the mean mortality in this age-group by 0.22 per 100,000 per day.

15-64 year age-group

The U-shaped relationship is more apparent in the 15-64 year age group and the three degree temperature range where mortality is lowest is between 13^{0}C and 16^{0}C (Figure 2(a)).

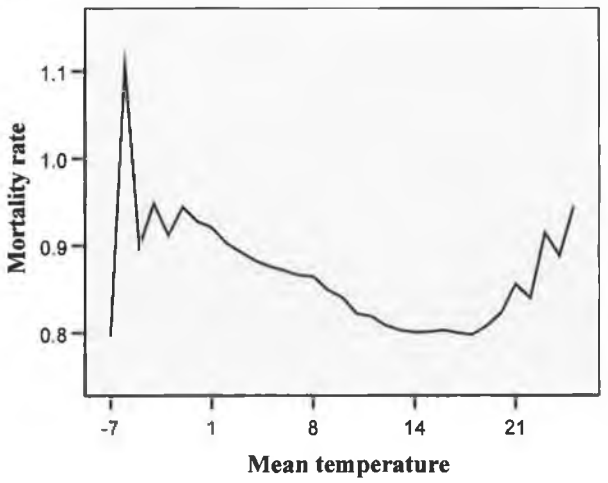
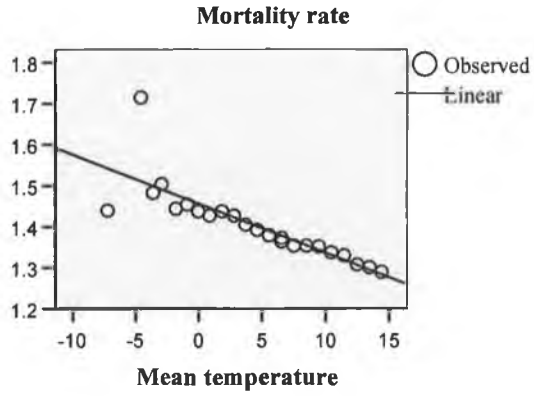


Figure 2(a) Association between all cause mortality in 15-64 age group per 100,000 population and daily mean temperature in three degree bands 1981-2002

At temperatures below the mid-point of this range, 14.5^{0}C, a linear relationship is evident between mortality and mean temperature (Figure 2(b)).

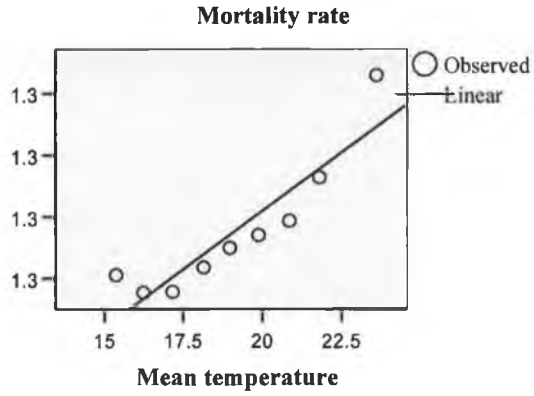


R Square = 0.684 p<0.05

Figure 2 (b) Relationship between mean temperatures below 14.5^{0}C in the 15-64 year age-group per 10,000 age-specific population

At temperatures below 14.5^{0}C

Mortality = 1.456-0.012(t). Mortality increases by 0.012 per 100,000 per day per degree fall in temperature, and mortality increases by 1.4%, over the mean mortality in this age-group of 0.84 per 100,000 per day.



R Square = 0.828 p<0.001

Figure 2(c) Relationship between mean temperatures above 14.5^{0}C in the 15-64 year age-group per 10,000 age-specific population

At temperatures above 14.5^{0}C, a linear relationship is also seen (Figure 2 (c).

Mortality = 1.150+0.008(t). Per degree rise in temperature, mortality in this age group increases by 0.015 per 100,000 per day and by 1.0%, over the mean mortality in this age-group of 0.84 per 100,000 per day.

65-74 year age-group

The quadratic nature of the relationship is clearer in the 65-74 year age group, and mortality is lowest in the temperature range 18^0C to 21^0C (Figure 3 (a))

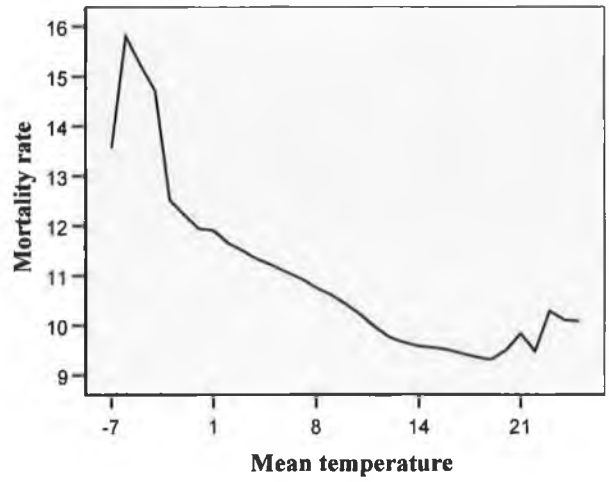
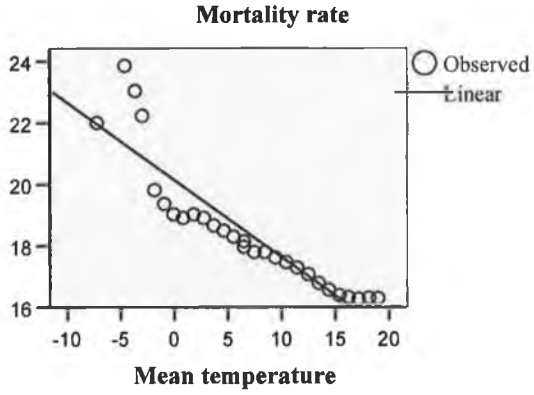


Figure 3 (a) Relationship between daily mean temperature and mortality in the 65-74 age group per 100,000 age-specific population

At temperatures below 19.5^0C, the mid-point of the range, a linear relationship is evident (Figure 3 (b)).



R Square = 0.828 p<0.001

Figure 3 (b) Relationship between mean temperatures below 19.5^0C in the 65-74 year age-group per 10,000 age-specific population

Mortality = 20.142-0.251(t). Mortality increases by 0.275 per 100,000 per day per degree fall in temperature, and mortality in this age group increases by 2.4%, over the

mean mortality in this age-group of 10.38 per 100,000 per day per degree fall in temperatures below 19.5^{0}C

However, there are insufficient days with mean temperatures above this threshold to determine the incremental change in mortality.

75-84 year age-group

The U-shaped curve is more apparent again in the 75-84 year age-group (Figure 3 (h)). and mortality is lowest in the range16^{0}C to 19^{0}C.

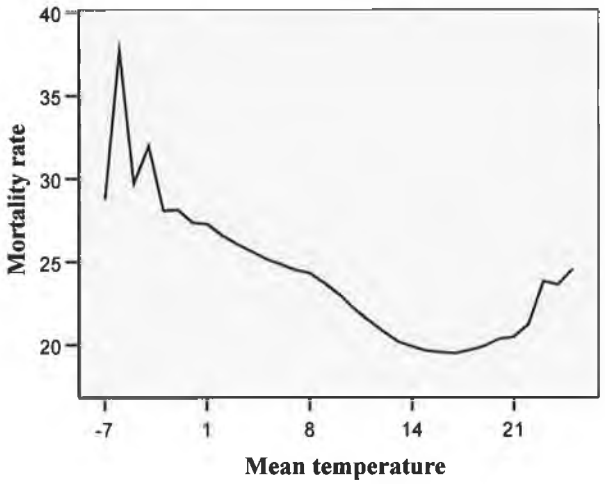
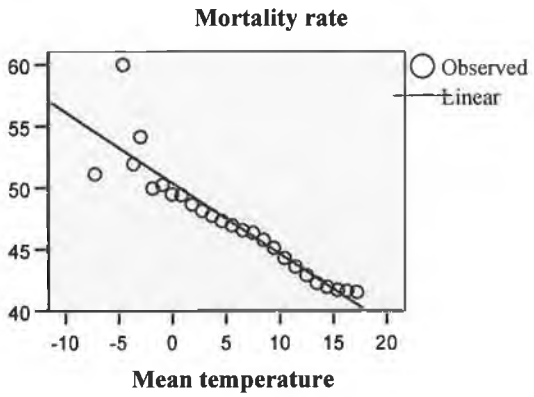


Figure 4 (a) Relationship between daily mean temperature and mortality in the 75-84 age group per 100,000 age-specific population

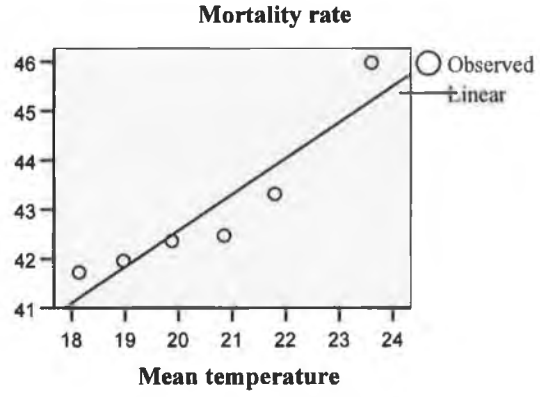
At temperatures below 17.5^{0}C, a linear relationship is seen (Figure 4(b))



R Square = 0.849 p<0.001

Figure 4 (b) Relationship between mean temperatures below 17.5^{0}C in the 75-84 year age-group per 10,000 age-specific population

Mortality = 50.371-0.573(t). Mortality increases linearly at 0.56 per 100,000 per day per degree fall in temperature, and mortality in this age group increases by 2.5%, over the mean mortality in this age-group of 22.69 per 100,000 per day per degree fall in temperatures below 17.5^{0}C . Above these temperatures, a linear relationship is also evident (Figure 5 (c))



R Square = 0.858 p<0.001

Figure 4 (c) Relationship between mean temperatures above 17.5^{0}C in the 75-84 year age-group per 10,000 age-specific population

Mortality = 27.904+0.733 (t)

Mortality increases at 0.733 per 100,000 per day, resulting in an increase of 3.2% in mortality per degree rise in mean temperature over the mean mortality rate of 60.35 per 100,000 in this age group.

Age 85 and over

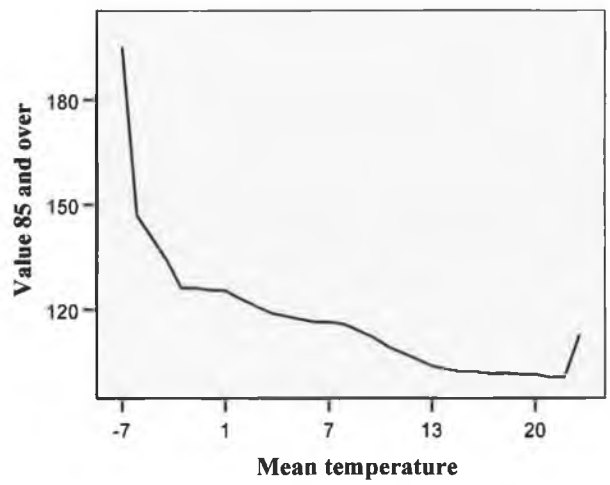
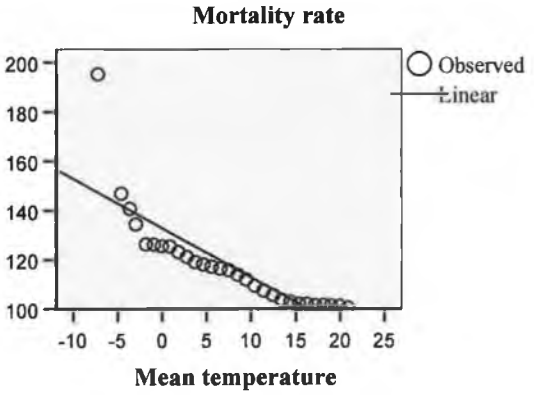


Figure 5 (a) Relationship between daily mean temperature and mortality in the over 85 age group per 100,000 age-specific population

The U-shaped temperature-mortality relationship in the over 85 year age-group is higher, between 20^0C and 23^0C and a linear relationship with increasing mortality with temperatures below 21.5^0C is evident.

Using the expression, mortality = 133.556-2.337 (t), at temperatures below 21.5^0C, mortality in this age group increases by 1.957 per 100,000 per day, or by 3.9%, over the mean mortality in this age-group of 60.35 per 100,000 per day in this age-group (Figure 6(b)).



R Square = 0.707 p<0.001

Figure 5(b) Relationship between mean temperatures below 21.5^{0}C in the 85 and over year age-group per 10,000 age-specific population

No relationship was evident above this threshold.

The temperature-mortality relationship for the specified diseases was then examined.

Cardiovascular diseases

For cardiovascular disease, the characteristic curve was again apparent (Figure 6(a)).

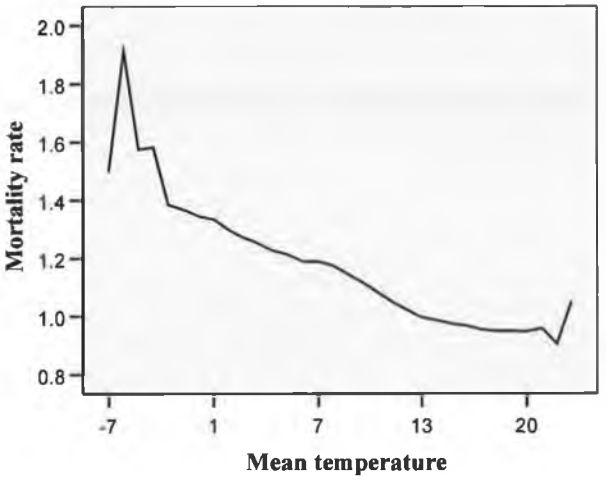
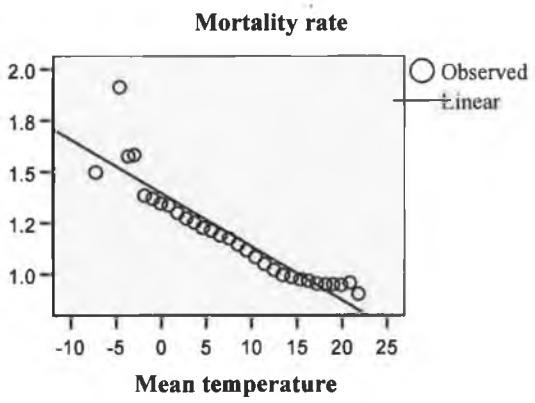


Figure 6 (a) Relationship between daily mean temperature and mortality from cardiovascular disease per 100,000 population

The temperature range where mortality was lowest was found to lie between 21^{0}C and 24^{0}C. A linear relationship with increasing mortality with decreasing temperatures was obtained at temperatures below 22.5^{0}C

Mortality = 1.396-0.026 (t). At these lower temperatures, mortality from cardiovascular disease increases by 0.024 per 100,000 per day, or by 1.9%, over the mean mortality of 1.34 per 100,000 per day (Figure 7(b)).



R Square = 0.852 p<0.001

Figure 6(b) Relationship between mean temperatures below 22.5^{0}C from cardiovascular disease per 100,000 population

No significant relationship was obtained with temperatures above this point.

Ischaemic heart disease

The shape of the temperature-mortality relationship for ischaemic heart disease (Figure 8(a)) was similar to that of cardiovascular disease (Figure 7(a)).

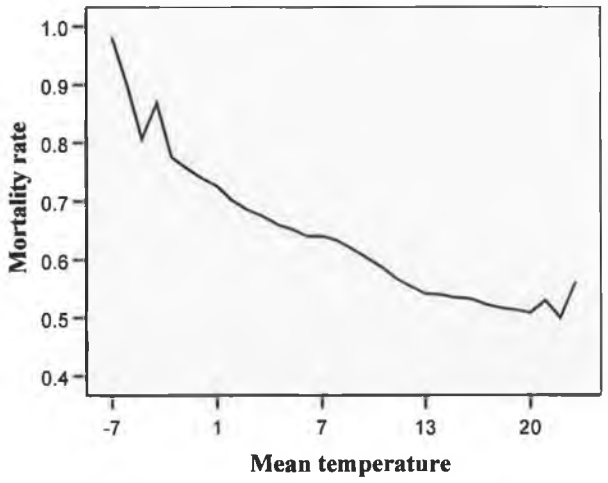
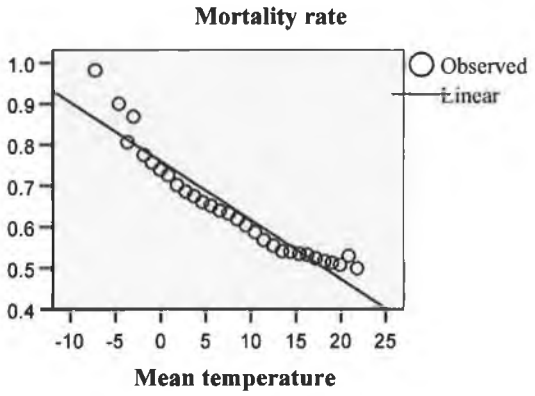


Figure 7(a) Relationship between daily mean temperature and mortality from ischaemic heart disease per 100,000 population

Similar to cardiovascular disease, the temperature range where mortality was lowest was found to lie between 21^0C and 24^0C. A clear linear relationship between cold temperatures and mortality from ischaemic heart disease was evident at was evident at temperatures below 22.5^0C (Figure 8(b)).



R Square =0.904 p<0.001

Figure 7 (b) Relationship between mean temperatures below 21.5^0C from ischaemic heart disease per 100,000 population

Mortality = 0.761-0.015 (t). At these lower temperatures, mortality from ischaemic heart disease increased by 0.015 per 100,000 per day and mortality increased by 2.1% over the mean mortality rate of 0.72 per 100,000 per day, per degree fall in temperature below 21.5^0C. It was not possible to develop a significant linear or quadratic with temperatures above this point.

Respiratory disease

The temperature range where mortality was lowest was found to lie between 18^0C and 21^0C.

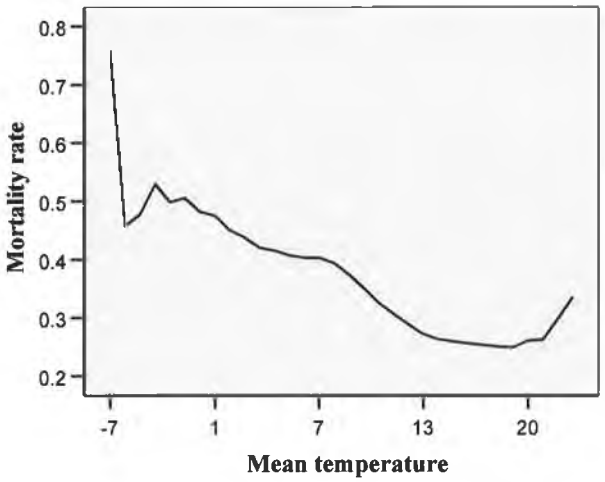
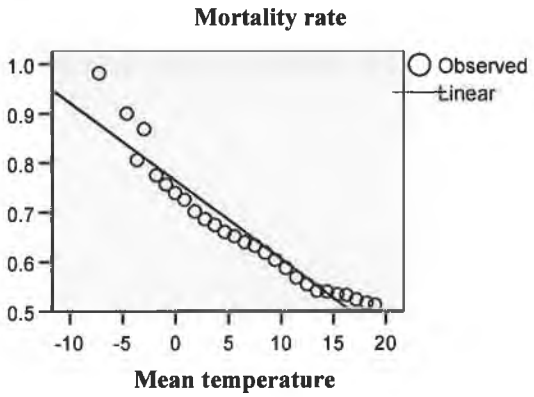


Figure 8 (a) Relationship between daily mean temperature and mortality from respiratory disease per 100,000 population

A clear linear relationship between cold temperatures and mortality from respiratory disease was evident at temperatures below 19.5^0C (Figure 9(b))

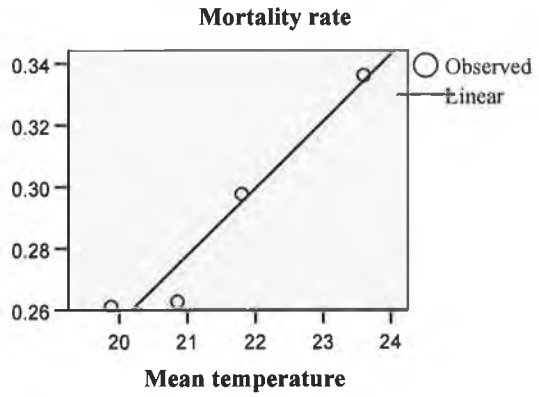


R Square =0.921 p<0.001

Figure 8 (b) Relationship between mean temperatures below 19.5^0C from respiratory disease per 100,000 population

Mortality = 0.764-0.016 (t). At these lower temperatures, mortality from respiratory disease increased by 0.014 per 100,000 per day and mortality increased by 4.1% over the mean mortality rate per day of 0.34 per 100,000 per degree fall in temperature below

19.5^{0}C. At temperatures above 19.5^{0}C, a linear relationship was also established (Figure 9(c))



R Square =0.945 p<0.05

Figure 8 (c) Relationship between mean temperatures above 19.5^{0}C and mortality from respiratory disease per 10,000 age-specific population

A

t temperatures below this threshold, a linear relationship between mortality and cold temperatures was evident.

Mortality = -0.178+0.022 (t). At these higher temperatures, mortality from respiratory disease increased by 0.022 per 100,000 per day and mortality increased by 6.5 % over the mean mortality rate per day of 0.34 per 100,000 per degree fall in temperature below 19.5^{0}C.

Stroke

While a curvi-linear relationship is evident in the temperature-mortality relationship for stroke, it is not clear as for other disease (Figure 10 (a)).

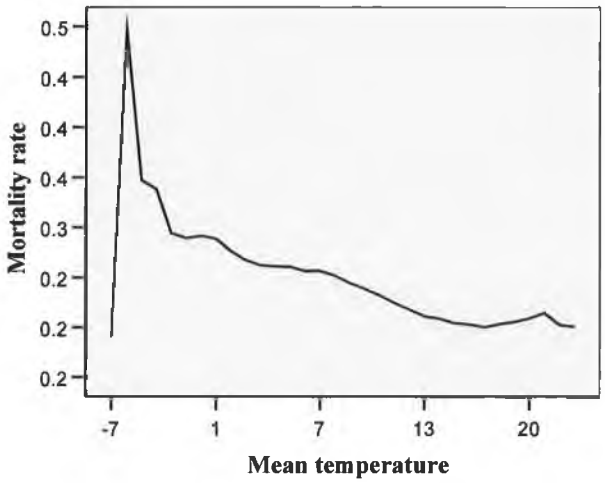
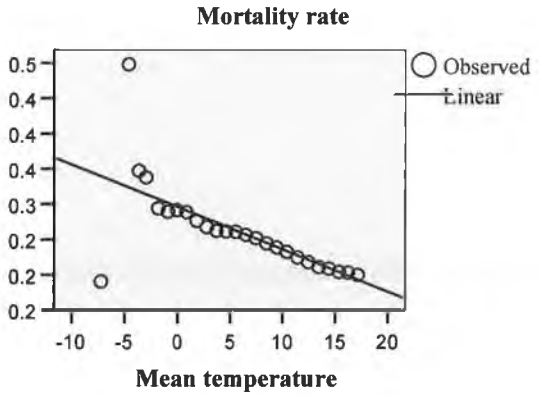


Figure 9 (a) Relationship between daily mean temperature and mortality from stroke per 100,000 population

The range where mortality is lowest lies between 16^0C and 19^0C.



R Square =0.444 p<0.001

Figure 9 (b) Relationship between mean temperatures below 17.5^0C and mortality from stroke per 100,000 population

At temperatures below 17.5^0C, the equation for the mean temperature-stroke mortality relationship is linear. Mortality = 0.296 -0.006(t)

At these lower temperatures, mortality from stroke increased by 0.015 per 100,000 per day and mortality from this disease increased by 2.0% over the mean mortality rate per day of 0.30 per 100,000 per degree fall in temperature below 17.5^0C.

It was not possible to ascertain a relationship above 17.5^0C

Cancer

No clear relationship is seen between daily mean temperature and mortality from cancer per 100,000 age-specific population (Figure 11).

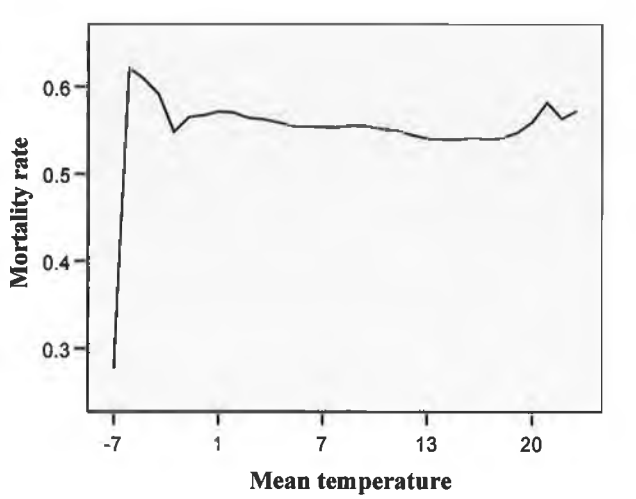
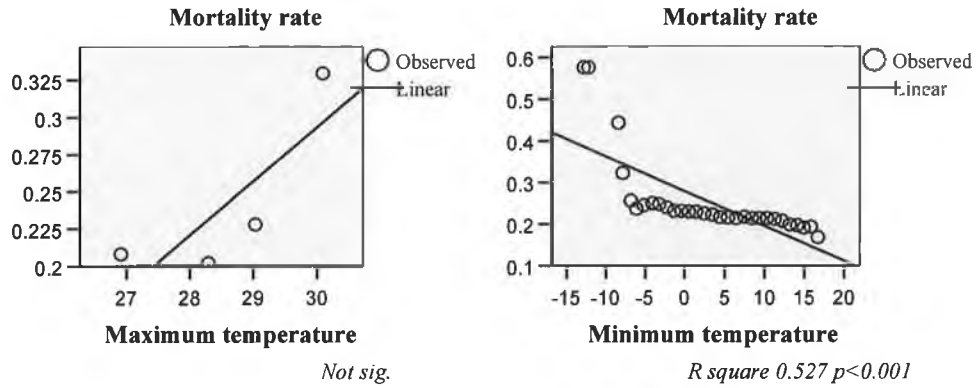


Figure 10 Relationship between daily mean temperature and mortality from cancer per 100,000 population

APPENDIX C

Present impact of extremes of temperatures



Not sig.

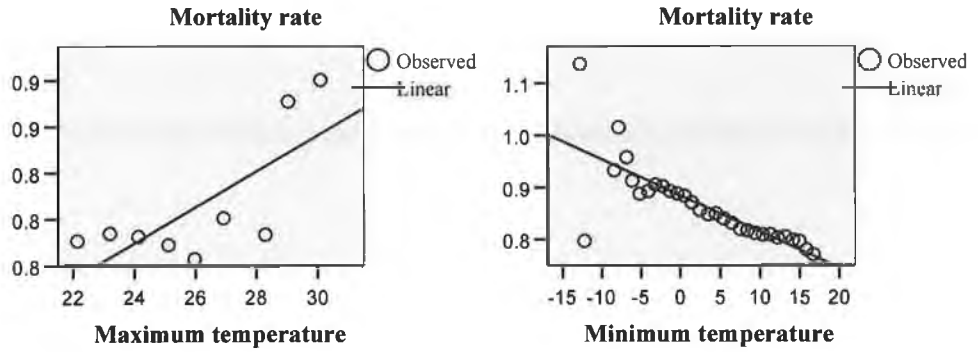
R square 0.527 p<0.001

Figure 1 Relationship between maximum and lagged minimum temperatures and mortality in the 14 and under year age group per 100,000 age-specific population

Maximum temperatures
Threshold: above 26.5^{0}C

Minimum temperatures
No threshold identified
Mortality =0.279-0.008(t)
Increase of 3.6% per degree fall

15-64 Age group



R square 0.545 p<0.05

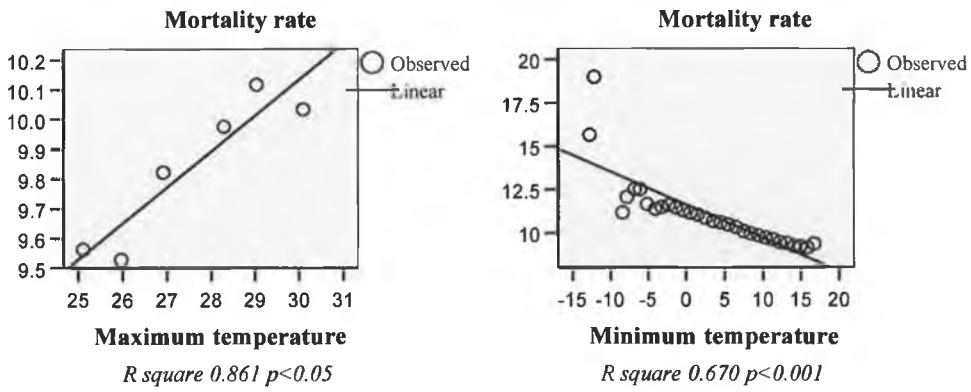
R square 0.613 p<0.001

Figure 2 Relationship between maximum and lagged minimum temperatures and mortality in the 15-64 year age group per 100,000 age-specific population

Maximum temperatures
Threshold: above 21.5^{0}C
Mortality =0.884-0.007(t)
Increase of 1.0% per degree rise above 21.5^{0}C

Minimum temperatures
No threshold identified
Mortality =0.623-0.008(t)
Increase of 0.8% per degree fall

65-74 Age group



R square 0.861 p<0.05

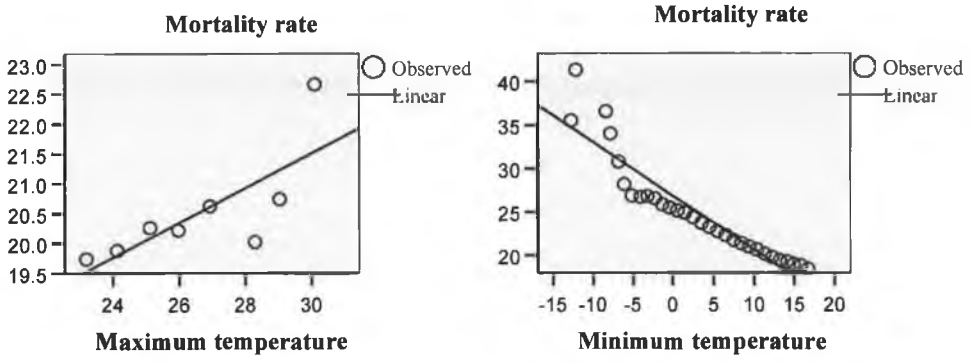
R square 0.670 p<0.001

Figure 3 Relationship between maximum and lagged minimum temperatures and mortality in the 65-74 year age group per 100,000 age-specific population

Maximum temperatures
Threshold: above 24.5^{0}C
Mortality =65.12-0.121(t)
Increase of 1.2% per ^{0}C rise above 24.5^{0}C

Minimum temperatures
Threshold: above 16.5^{0}C
Mortality =11.608-0.197(t)
Increase of 1.9% per ^{0}C fall below 16.5^{0}C

75-84 Age group



R square 0.576 p<0.05

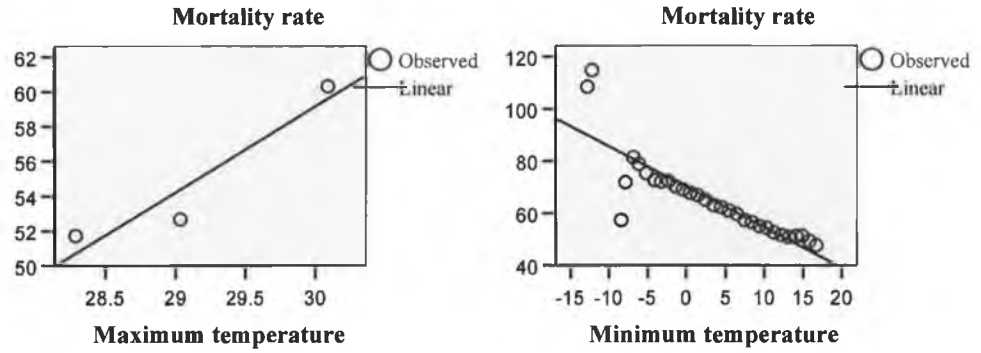
R square 0.866 p<0.001

Figure 4 Relationship between maximum and lagged minimum temperatures and mortality in the 75-84 year age group per 100,000 age-specific population

Maximum temperatures
Threshold: above 22.5^{0}C
Mortality =12.774-0.291(t)
Increase of 1.3% per ^{0}C rise above 22.5^{0}C

Minimum temperatures
No threshold identified
Mortality =26.837-0.618(t)
Increase of 2.7% per ^{0}C fall

85 and over age group



Not sig. *R square 0.734 p<0.001*

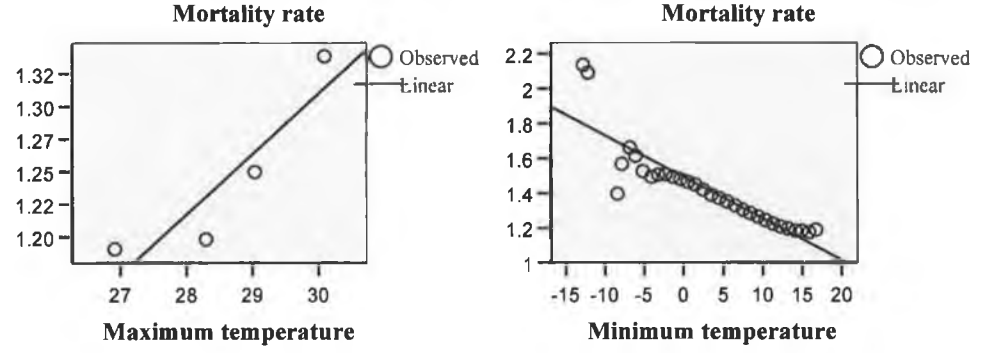
Figure 5 Relationship between maximum and lagged minimum temperatures and mortality in the over 85 year age group per 100,000 age-specific population

Maximum temperatures
Threshold: above 27.5^{0}C

Minimum temperatures
No threshold identified
Mortality =70.098-1.551(t)
Increase of 2.6% per ^{0}C fall

Mortality from specific disease categories was then examined.

Cardiovascular disease



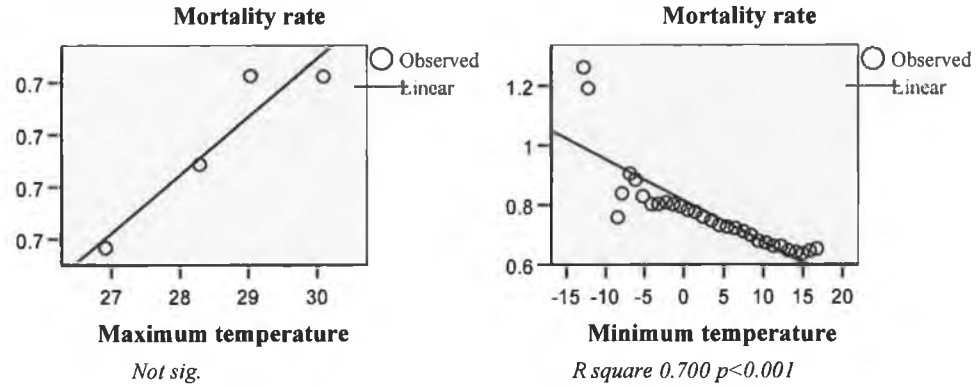
Not sig. *R square 0.778 p<0.001*

Figure 6 Relationship between maximum and lagged minimum temperatures and mortality from cardiovascular disease per 100,000 population

Maximum temperatures
Threshold: above 26.5^{0}C
No sig. relationship

Minimum temperatures
Threshold: above 16.5^{0}C
Mortality =1.491-0.025(t)
Increase of 1.9% per ^{0}C fall below 16. 5^{0}C

Ischaemic heart disease



Not sig.

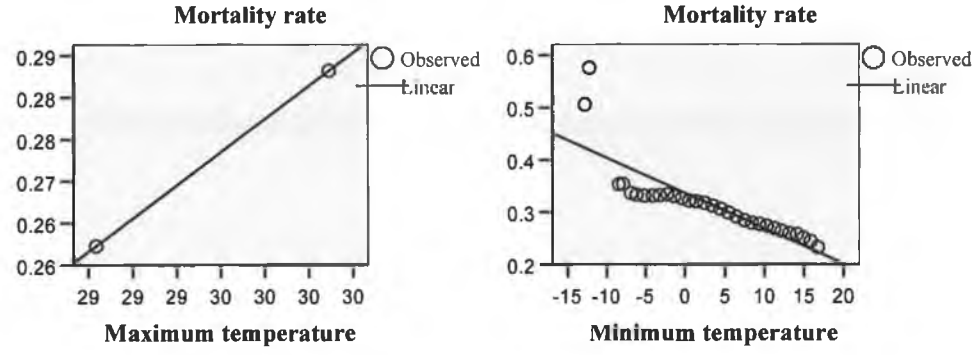
R square 0.700 p<0.001

Figure 7 Relationship between maximum and lagged minimum temperatures and mortality from ischaemic heart disease per 100,000 population

Maximum temperatures
Threshold: above 26.5^{0}C
No sig. relationship

Minimum temperatures
Threshold: No threshold identified
Mortality =0.814-0.014(t
Increase of 1.9% per ^{0}C fall

Stroke



Not sig.

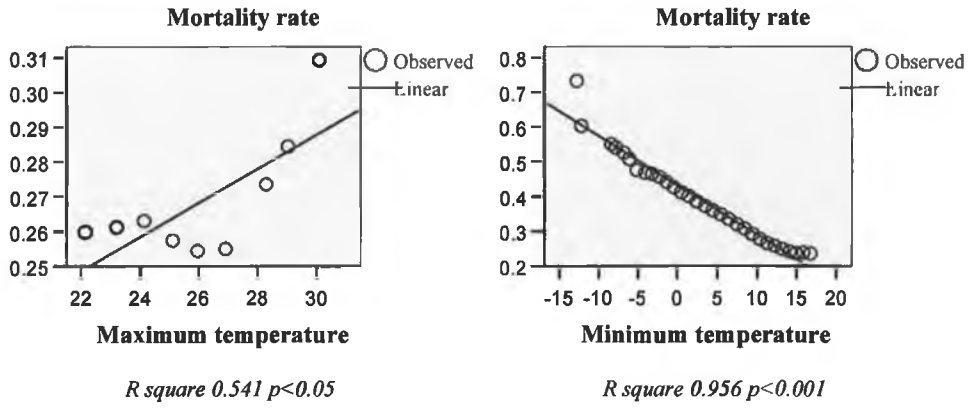
R square 0.693 p<0.001

Figure 8 Relationship between maximum and lagged minimum temperatures and mortality from stroke per 100,000 population

Maximum temperatures
Threshold: above 26.5^{0}C
No sig. relationship

Minimum temperatures
Threshold: above 14.5^{0}C
Mortality =0.352-0.004(t)
Increase of 1.3% per ^{0}C fall

Respiratory disease



R square 0.541 p<0.05

R square 0.956 p<0.001

Figure 9 Relationship between maximum and lagged minimum temperatures and mortality from respiratory disease per 100,000 population

Maximum temperatures	Minimum temperatures
Threshold: above 21.5^{0}C	No threshold identified
Mortality =0.141-0.005(t)	Mortality =26.837-0.618(t)
Increase of 1.5% per ^{0}C rise above 21.5^{0}C	Increase of 2.7% per ^{0}C fall

APPENDIX D
Temporal patterns of mortality in relation to the hottest days

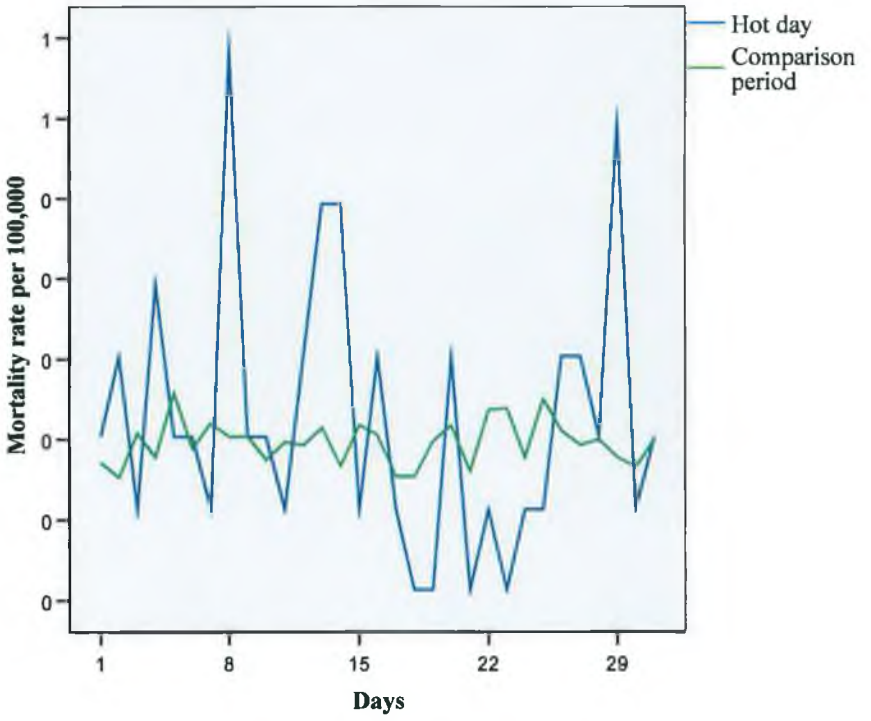


Figure 1 Temporal pattern of mortality in the 14 and under age group in relation to the hottest days in the time series

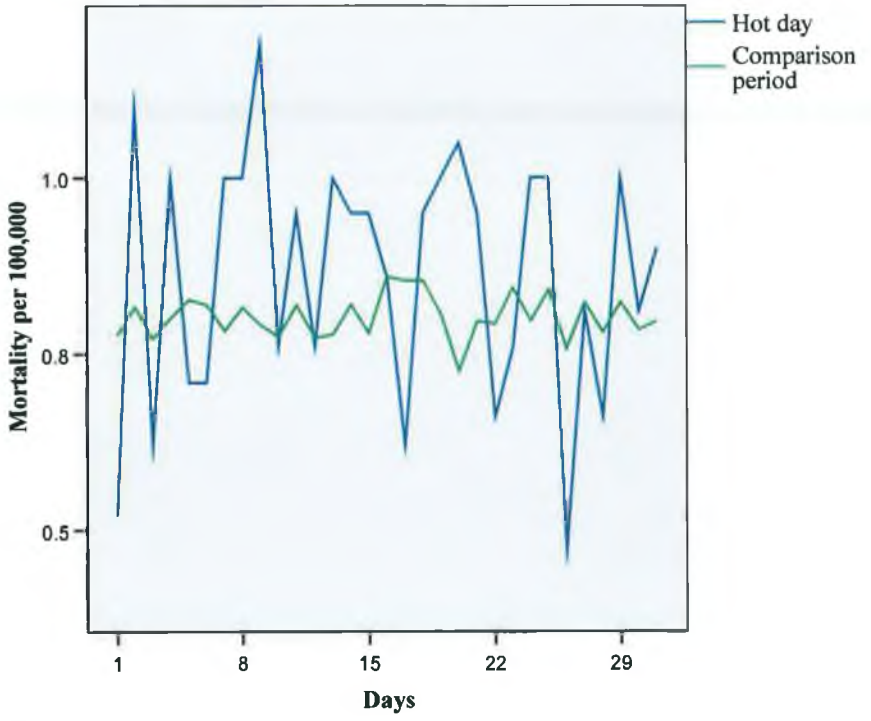


Figure 2 Temporal pattern of mortality in 15-64 year age group in relation to the hottest days in the time series

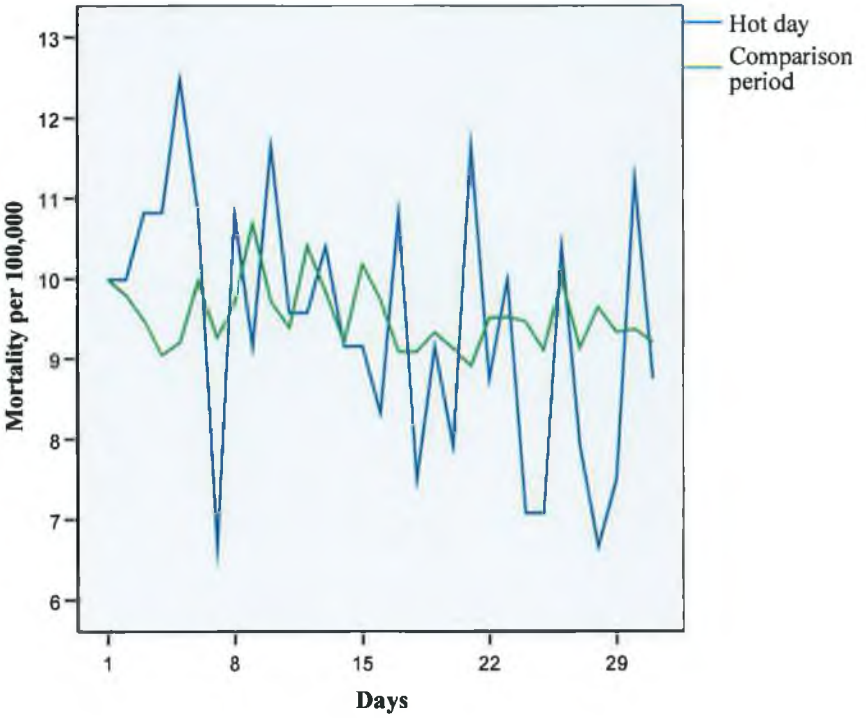


Figure 3 Temporal pattern of mortality in 65-74 year age group in relation to the hottest days in the time series

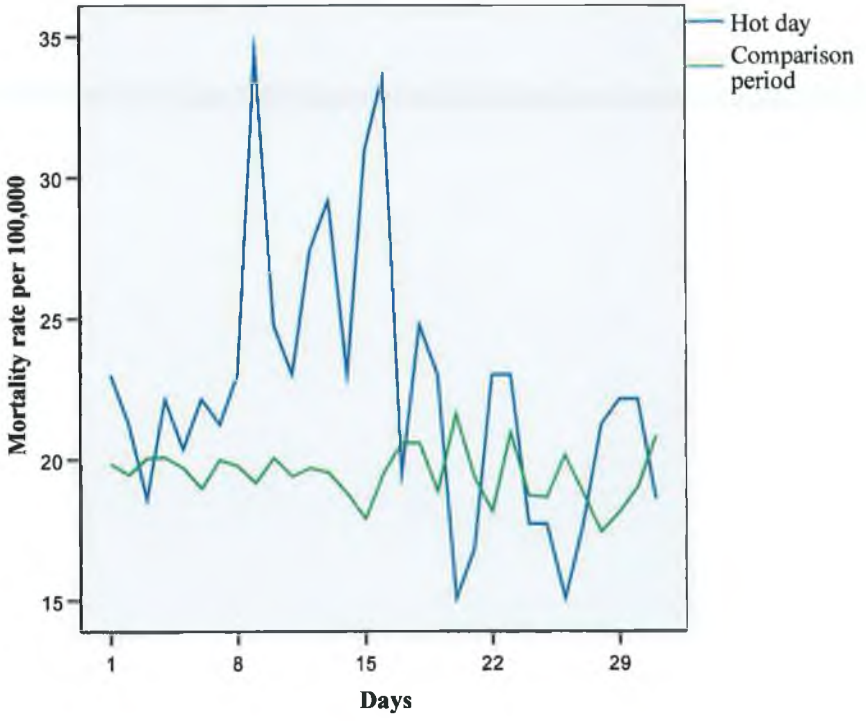


Figure 4 Temporal pattern of mortality in 75-84 year age group in relation to the hottest days in the time series

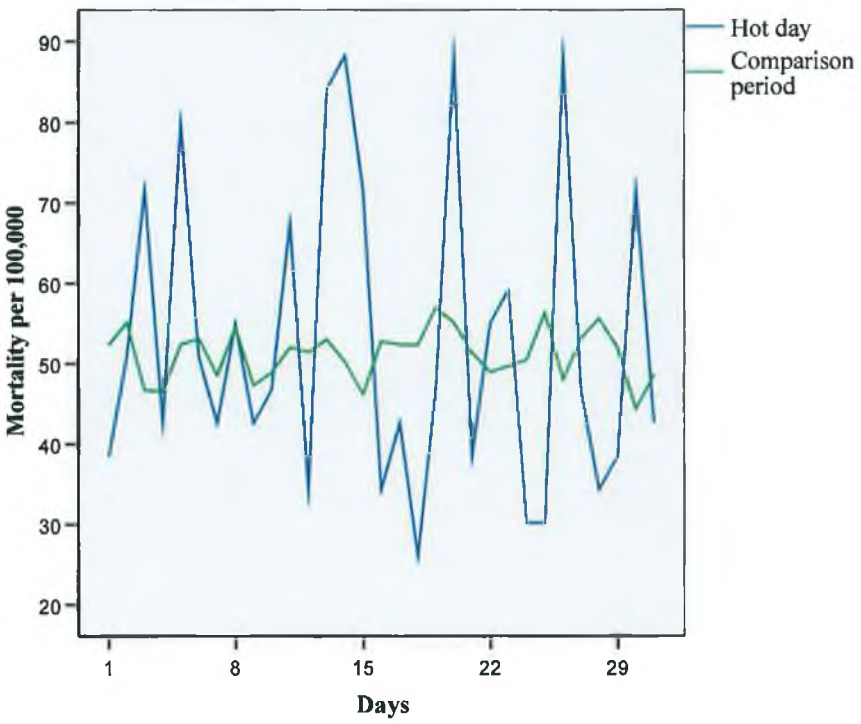


Figure 5 Temporal pattern of mortality in the 85 and over age group in relation to the hottest days in the time series

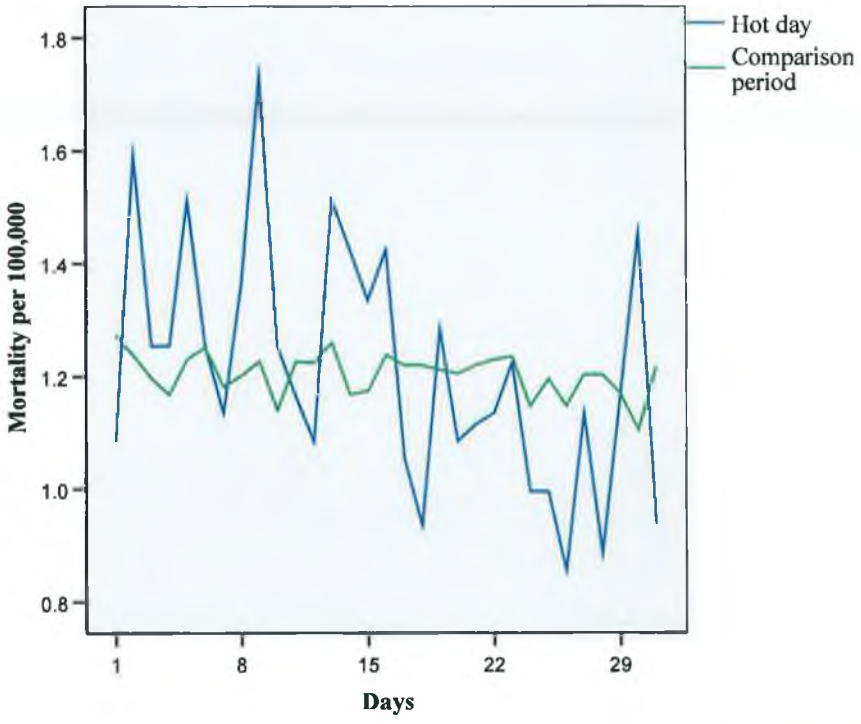


Figure 6 Temporal pattern of mortality from cardiovascular disease in relation to the hottest days in the time series

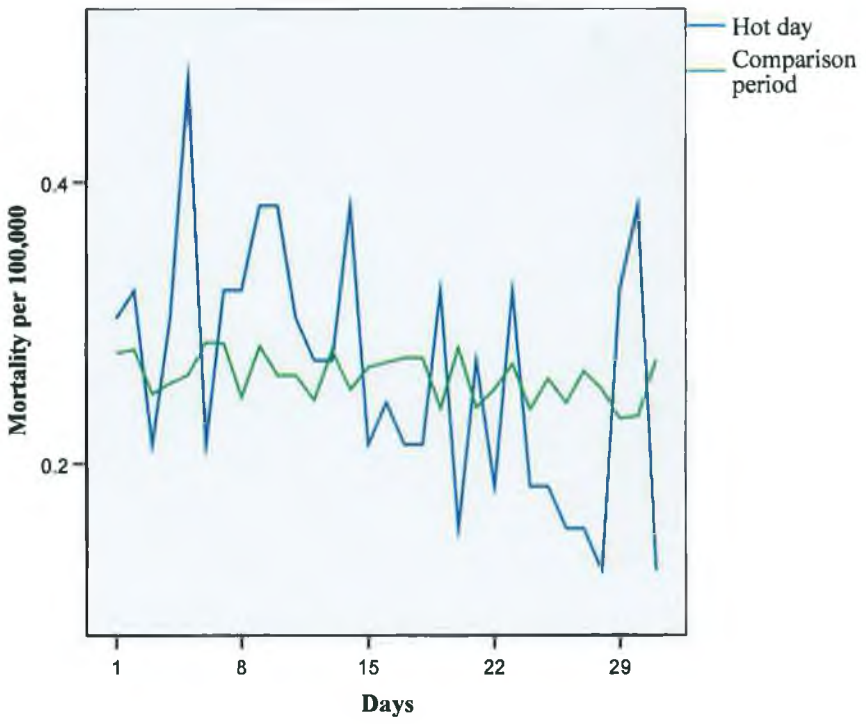


Figure 7 Temporal pattern of mortality from stroke in relation to the hottest days in the time series

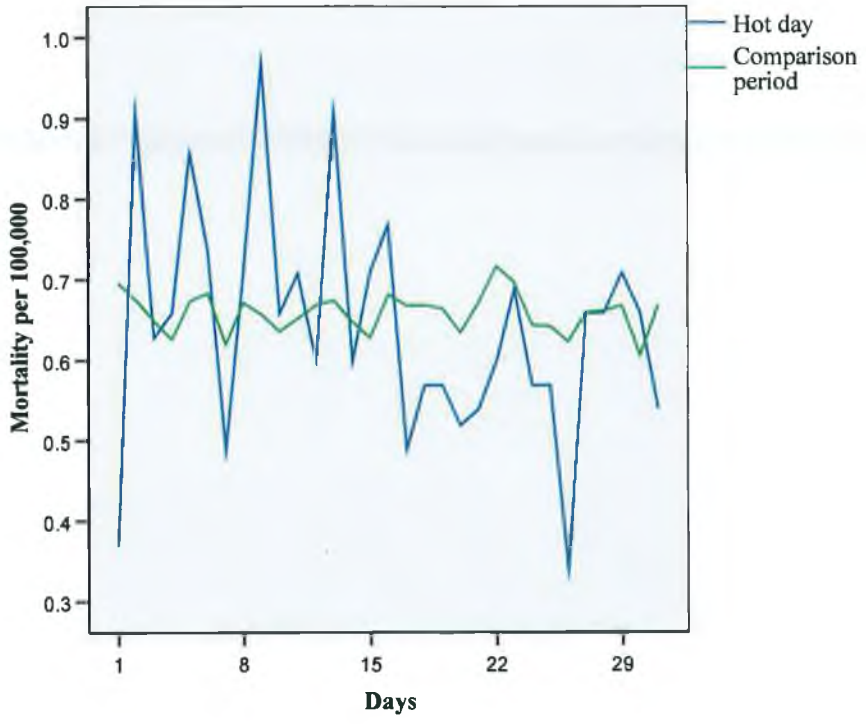


Figure 8 Temporal pattern of mortality from ischaemic heart disease in relation to the hottest days in the time series

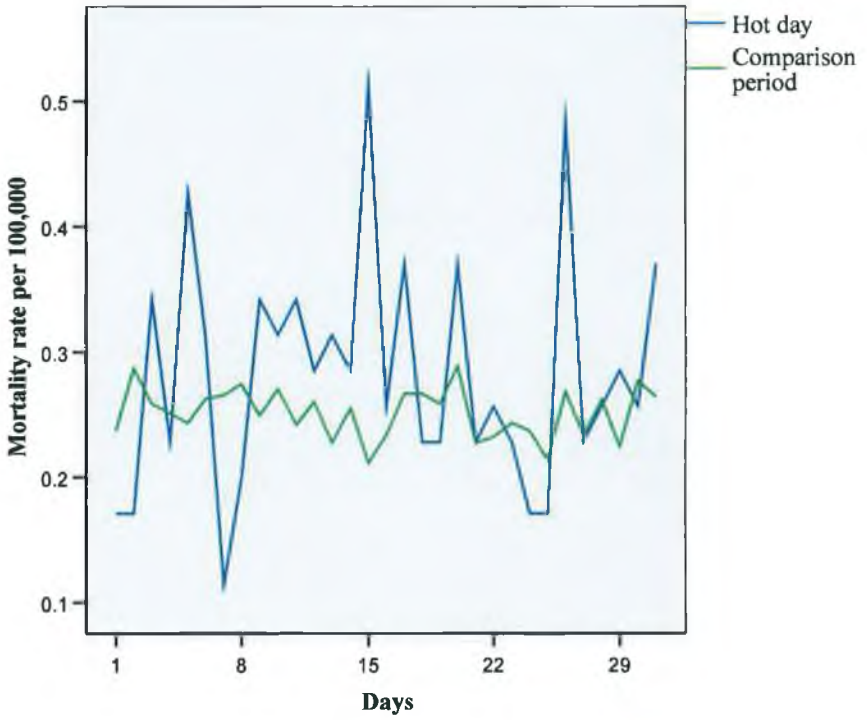


Figure 9 Temporal pattern of mortality from respiratory disease in relation to the hottest days in the time series

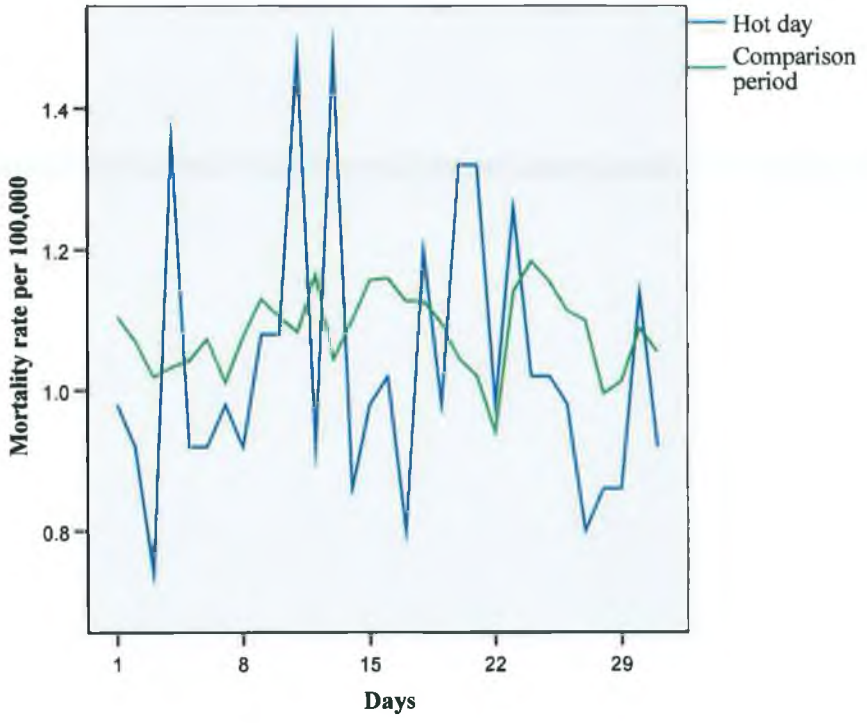


Figure 10 Temporal pattern of mortality from cancer in relation to the hottest days in the time series

APPENDIX E
Future impacts of changes in mean temperature

15-64 Age-specific population

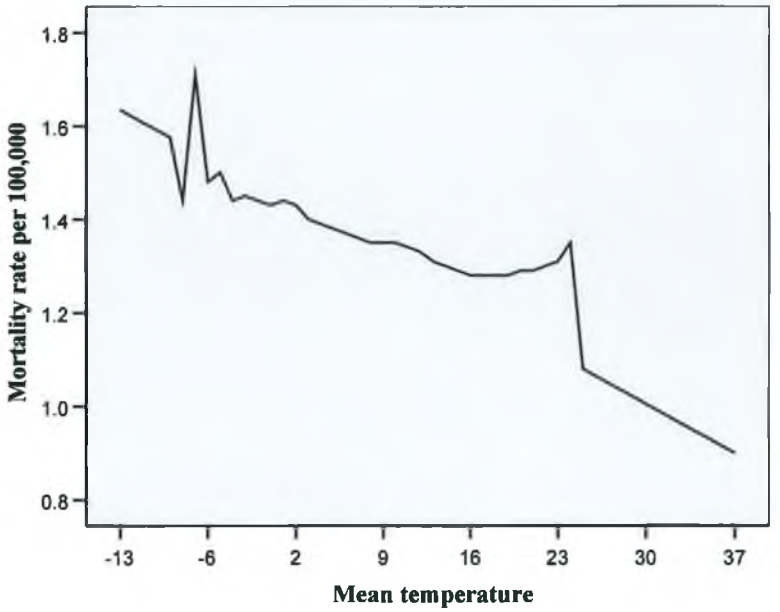


Figure 1(a) Extrapolation of mean temperature - mortality relationship in the 15-64 age specific population per 100,000
R square = 0.756 p<0.001 1.447-0.015(t)+0.000(t)²

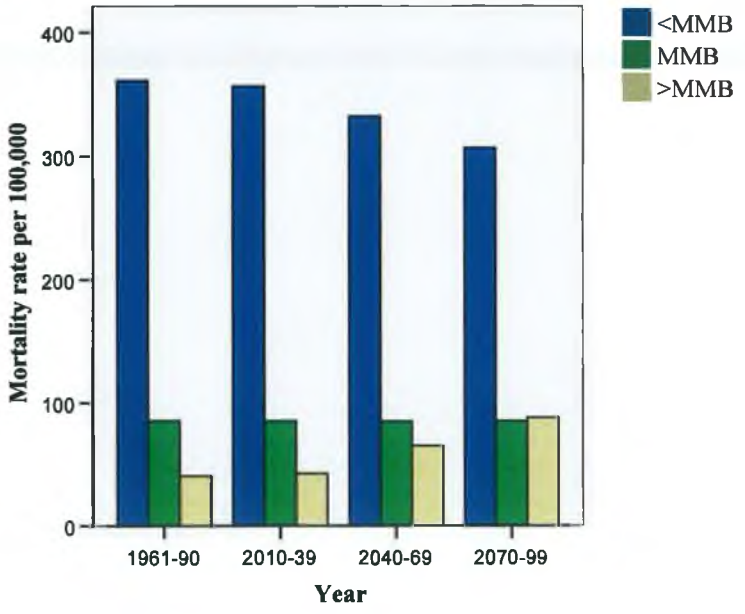


Figure 1(b) Impact of changes in mean temperature on age-specific mortality in 15-64 population per 100,000 per year in relation to minimum mortality band (MMB) of 13°C -16°C

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-3	-7	-8
CCM B2	-4	-6	-7
CSIRO A2	-3	-6	-8
CSIRO B2	-5	-6	-7
Had3CM A2	-1	-3	-7
Had3CM B2	-2	-3	-4
Mean	-3	-5	-7

Table 1 (a) Mean changes in total mortality in 15-64 age specific population per 100,000 per year resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1.0	-2.3	-2.6
CCM B2	-1.3	-2.0	-2.3
CSIRO A2	-1.0	-2.0	-2.6
CSIRO B2	-1.6	-2.0	-2.3
Had3CM A2	-0.3	-1.0	-2.3
Had3CM B2	-0.7	-1.0	-1.3
Mean	-1.0	-1.7	-2.2

Table 1 (b) Mean percent changes in total mortality in 15-64 age specific population per year from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

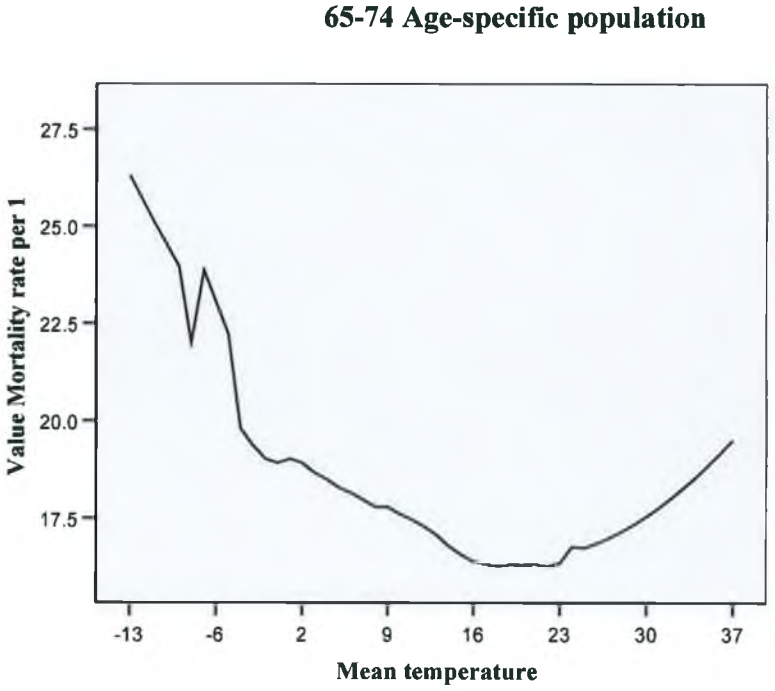


Figure 2 (a) Extrapolation of mean temperature - mortality relationship in the 65-74 age specific population per 100,000
R square = 0.903 p<0.001 20.018-0.379(t)+0.010(t)²

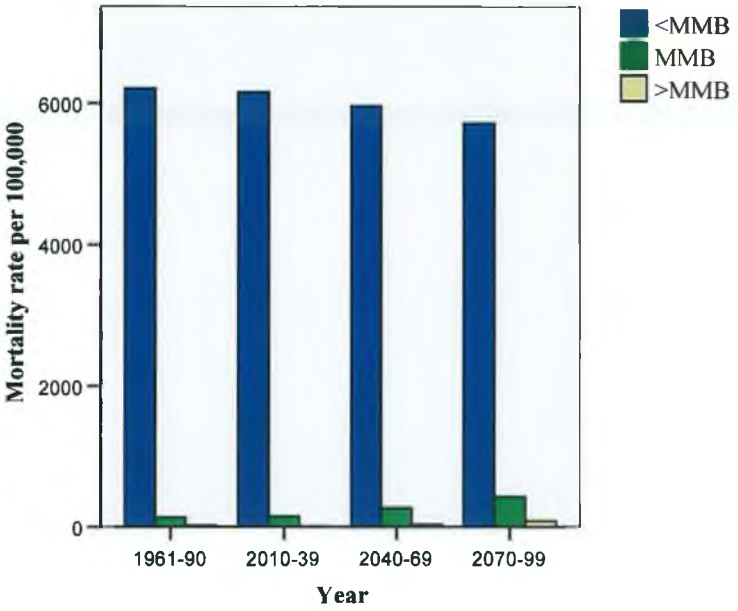


Figure 2(b) Impact of changes in mean temperature on age-specific mortality in 65-74 population per 100,000 per year in relation to minimum mortality band (MMB) of 18 °C -21 °C

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-62	-129	-157
CCM B2	-82	-112	-141
CSIRO A2	-67	-114	-169
CSIRO B2	-89	-124	-147
Had3CM A2	-17	-58	-119
Had3CM B2	-34	-51	-84
Mean	-59	-98	-136

Table 2(a) Mean changes in total mortality in 65-74 age specific population per 100,000 resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1.6	-3.4	-4.1
CCM B2	-2.2	-3.0	-3.7
CSIRO A2	-1.8	-3.0	-4.5
CSIRO B2	-2.3	-3.3	-3.9
Had3CM A2	-0.4	-1.5	-3.1
Had3CM B2	-0.9	-1.3	-2.2
Mean	-1.5	-2.6	-3.6

Table 2(b) Mean percent changes in total mortality in the 65-74 age specific population from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

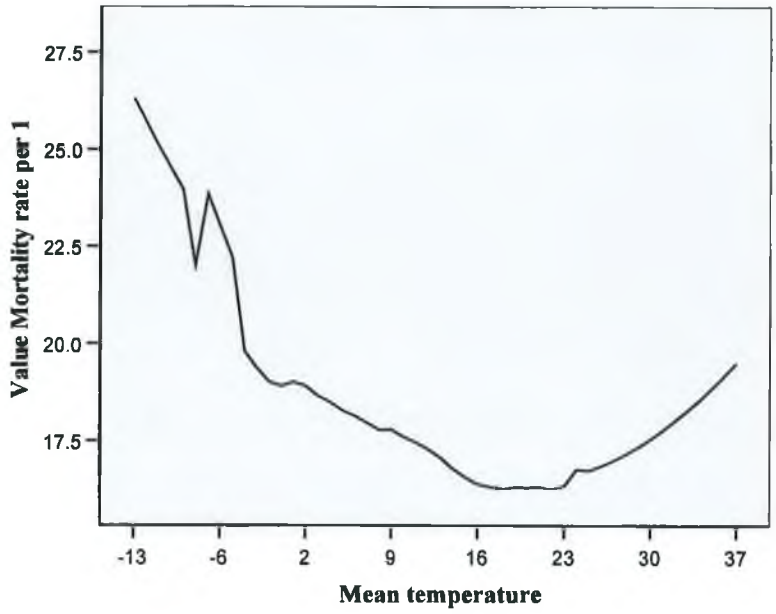


Figure 3 (a) Extrapolation of mean temperature - mortality relationship in the 75-84 age specific population per 100,000
R square = 0.834 p<0.001 49.934-0.749(t)+0.019(t)²

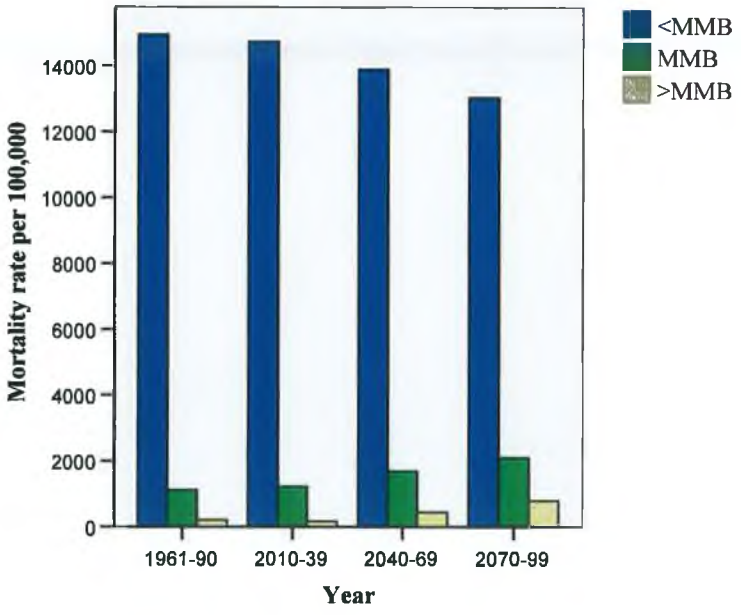


Figure 3(b) Impact of changes in mean temperature on age-specific mortality in the 75-84 population per 100,000 per year in relation to minimum mortality band (MMB) of 16 °C -19 °C

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-161	-333	-389
CCM B2	-201	-281	-355
CSIRO A2	-176	-300	-445
CSIRO B2	-228	-327	-396
Had3CM A2	-40	-140	-285
Had3CM B2	-90	-128	-204

Table 3 (a) Mean changes in total mortality in the 75-84 age specific population per 100,000 resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1.9	-4.0	-4.7
CCM B2	-2.4	-3.4	-4.3
CSIRO A2	-2.1	-3.6	-5.4
CSIRO B2	-2.8	-3.9	-4.8
Had3CM A2	-0.5	-1.7	-3.4
Had3CM B2	-1.1	-1.5	-2.5
Mean	-1.8	-3.0	-4.2

Table 3(b) Mean percent changes in total mortality in the 75-84 age specific population from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

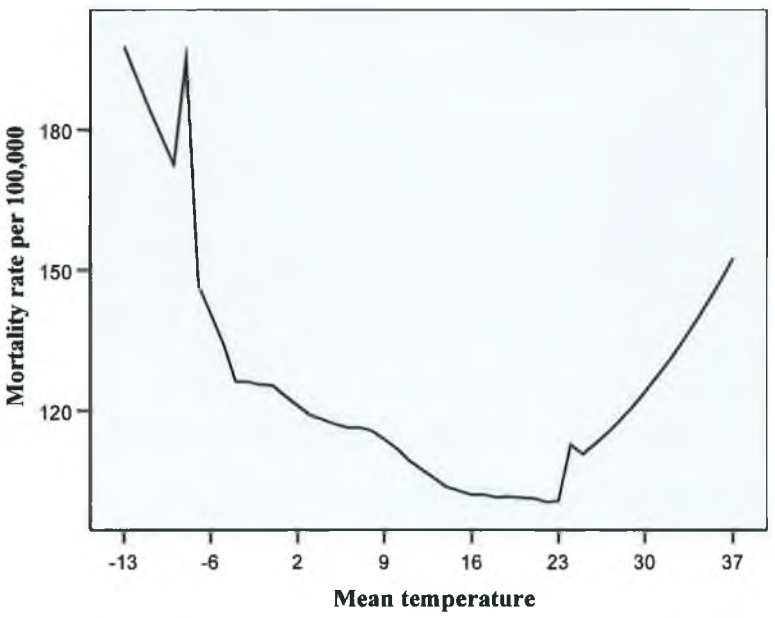


Figure 4 (a) Extrapolation of mean temperature - mortality relationship in the over 85 year age specific population per 100,000
R square = 0.854 p<0.001 131.882-0.378(t)+0.119(t)²

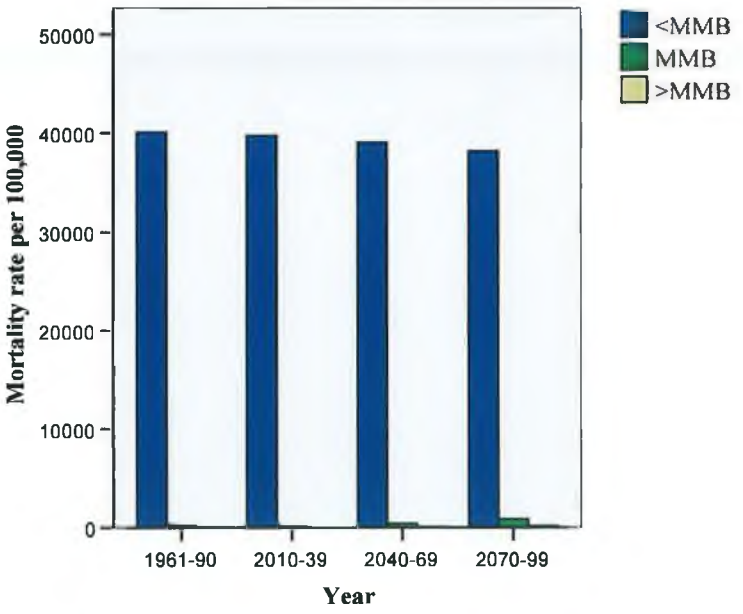


Figure 4(b) Impact of changes in mean temperature on age-specific mortality in the 85 year and over population per 100,000 per year in relation to minimum mortality band (MMB) of 20 °C -23 °C

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-487	-1043	-1258
CCM B2	-613	-875	-1126
CSIRO A2	-526	-911	-1389
CSIRO B2	-678	-988	-1201
Had3CM A2	-121	-431	-864
Had3CM B2	-269	-389	-629
Mean	-449	-773	-1078

Table 4 (a) Mean changes in total mortality in the 85 year and over age specific population per 100,000 resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-2.2	-4.7	-5.7
CCM B2	-2.8	-4.0	-5.1
CSIRO A2	-2.4	-4.1	-6.3
CSIRO B2	-3.1	-4.5	-5.5
Had3CM A2	-0.5	-2.0	-3.9
Had3CM B2	-1.2	-1.8	-2.9
Mean	-2.0	-3.5	-4.9

Table 4 (b) Mean percent changes in total mortality in the age 85 and over age specific population from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Cardiovascular mortality

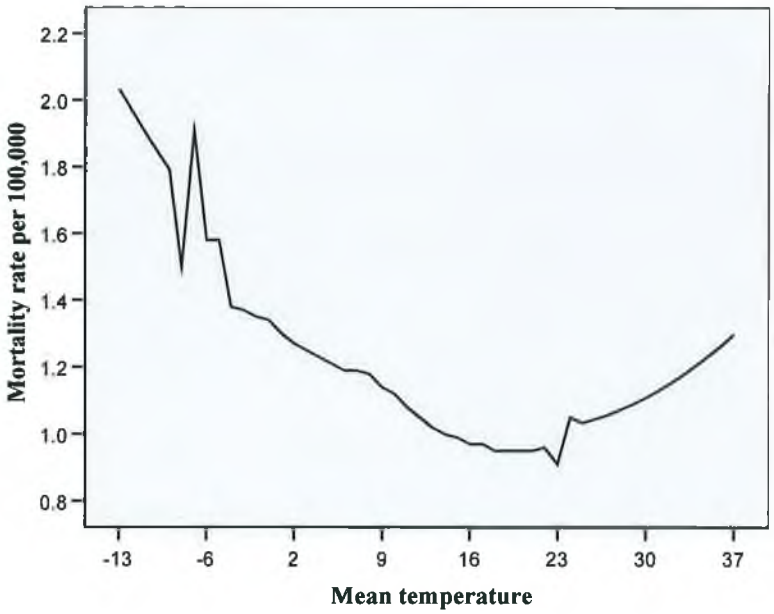


Figure 5 Extrapolation of mean temperature – cardiovascular mortality relationship per 100,000 population *R square = 0.887 p<0.001 1.389-0.039(t)+0.001(t)²*

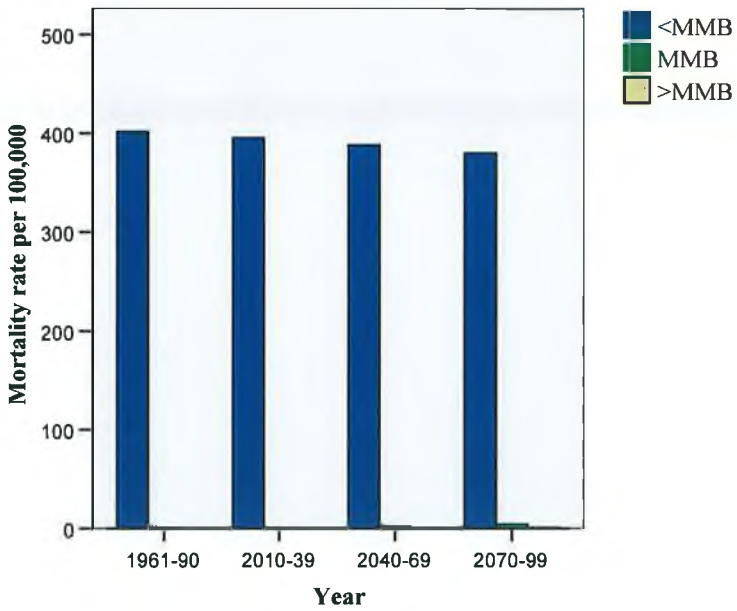


Figure 5 (b) Impact of change in mean temperature on mortality rates from cardiovascular mortality per 100,000 population per year in relation to minimum mortality band (MMB) of 21 °C -24 °C

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-8	-17	-21
CCM B2	-10	-15	-19
CSIRO A2	-9	-15	-23
CSIRO B2	-11	-16	-20
Had3CM A2	-2	-7	-15
Had3CM B2	-4	-6	-10
Mean	-7	-13	-18

Table 5 (a) Mean changes in total mortality from cardiovascular disease per 100,000 population resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1.6	-3.5	-4.3
CCM B2	-2.0	-3.1	-3.9
CSIRO A2	-1.8	-3.1	-4.7
CSIRO B2	-2.2	-3.3	-4.1
Had3CM A2	-0.4	-1.4	-3.1
Had3CM B2	-0.8	-1.2	-2.0
Mean	-1.5	-2.6	-3.7

Table 5 (b) Mean percent changes in mortality from cardiovascular disease from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Ischaemic heart disease

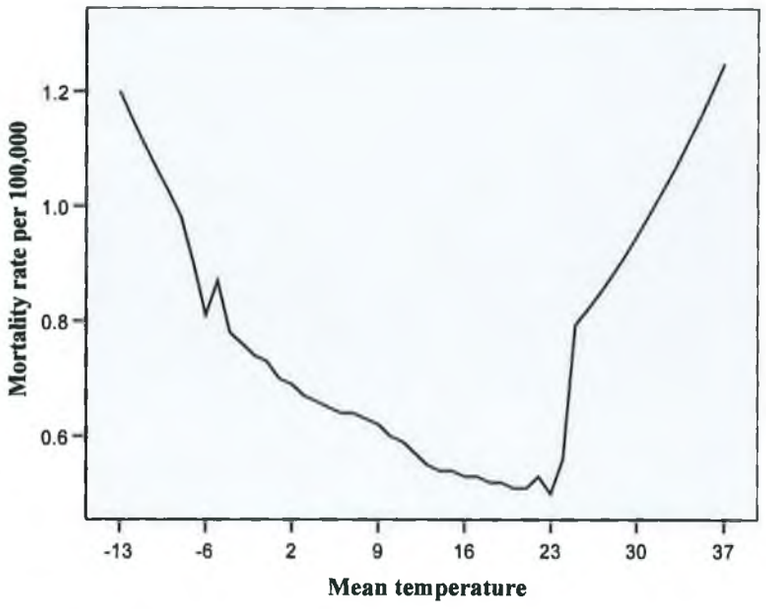


Figure 6 (a) Extrapolation of mean temperature – ischaemic heart disease mortality relationship per 100,000 population *R square = 0.980 p<0.001 0.756-0.023(t)+0.001(t)²*

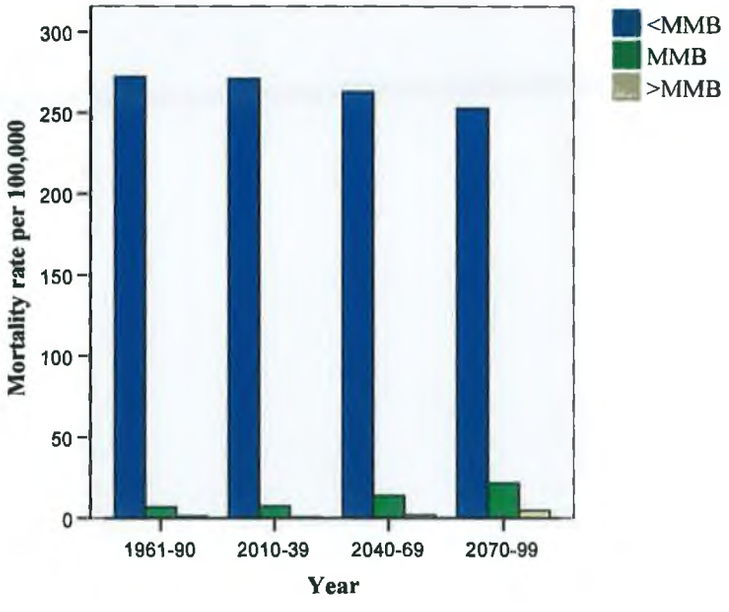


Figure 6 (b) Impact of change in mean temperature with respect to mortality from ischaemic heart disease per 100,000 population per year in relation to minimum mortality band (MMB) of 21 °C -24 °C

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-4	-9	-10
CCM B2	-5	-8	-10
CSIRO A2	-4	-8	-12
CSIRO B2	-6	-8	-10
Had3CM A2	-1	-3	-6
Had3CM B2	-2	-3	-5
Mean	-4	-7	-9

Table 6 (a) Mean changes in total mortality from ischaemic heart disease per 100,000 population resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1.5	-3.4	-3.8
CCM B2	-1.9	-3.0	-3.8
CSIRO A2	-1.5	-3.0	-4.6
CSIRO B2	-2.3	-3.0	-3.8
Had3CM A2	-0.4	-1.1	-2.3
Had3CM B2	-0.8	-1.1	-1.9
Mean	-1.4	-2.5	-3.4

Table 6 (b) Mean percent changes in mortality from ischaemic heart disease from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Stroke

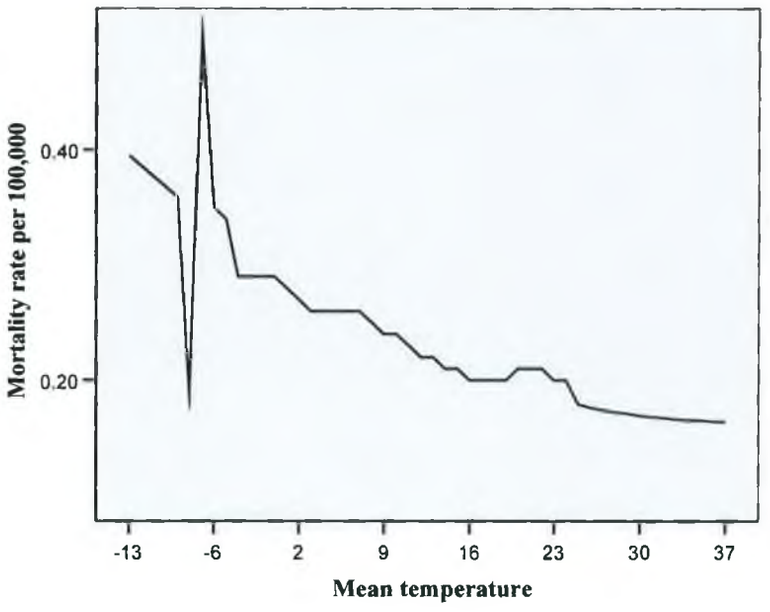


Figure 7 (a) Extrapolation of mean temperature – stroke mortality relationship per 100,000 population *R square = 0.503 p<0.001 0.293-0.007(t)+0.000095(t)²*

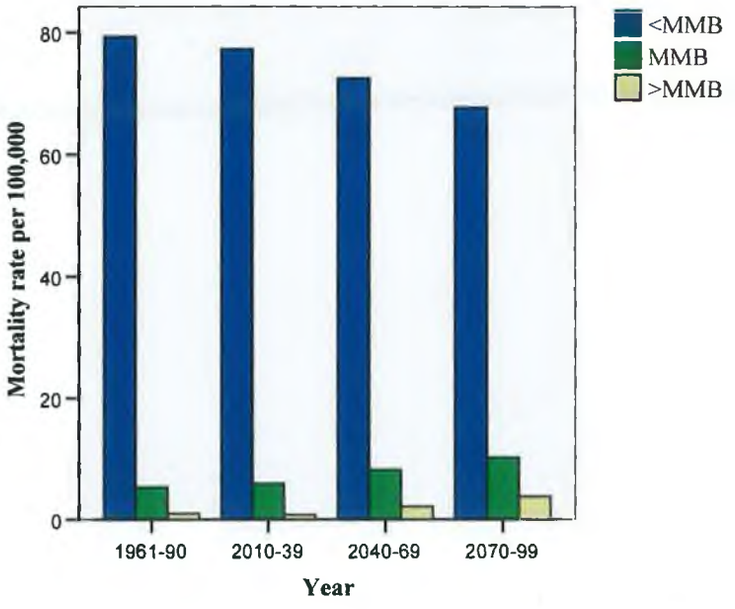


Figure 7 (b) Impact of change in mean temperature with respect to mortality from ischaemic heart disease per 100,000 population per year in relation to minimum mortality band (MMB) of 16 °C -19 °C

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-2	-4	-4
CCM B2	-2	-3	-4
CSIRO A2	-2	-3	-5
CSIRO B2	-3	-4	-4
Had3CM A2	0	-2	-3
Had3CM B2	-1	-1	-2
Mean	-2	-3	-4

Table 7 (a) Mean changes in total mortality from stroke per 100,000 population resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1.8	-3.7	-3.7
CCM B2	-1.8	-2.7	-3.7
CSIRO A2	-1.8	-2.7	-4.6
CSIRO B2	-2.7	-3.7	-3.7
Had3CM A2	0.0	-1.8	-2.7
Had3CM B2	-0.9	-0.9	-1.8
Mean	-1.5	-2.6	-3.3

Table 7 (b) Mean percent changes in mortality from stroke from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

Respiratory mortality

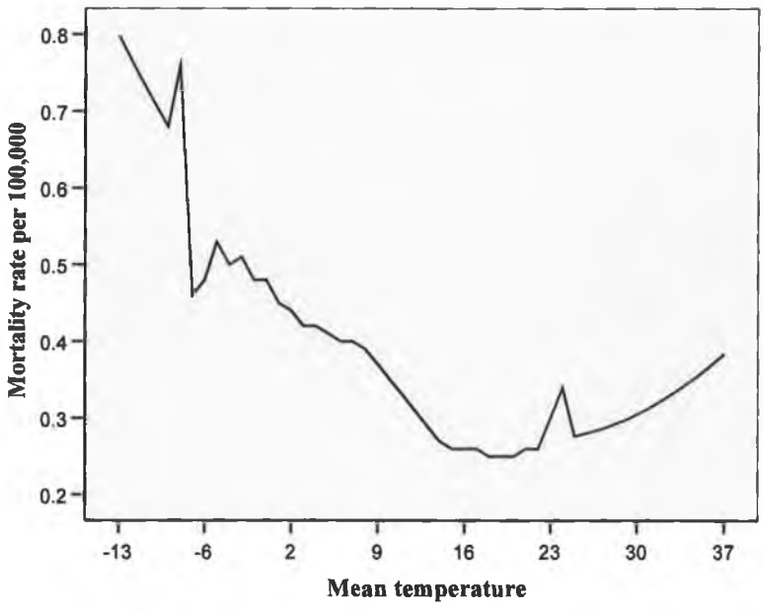


Figure 8 (a) Extrapolation of mean temperature – respiratory mortality relationship per 100,000 population *R square = 0.876 p<0.001 0.478-0.02(t)+0.00047(t)²*

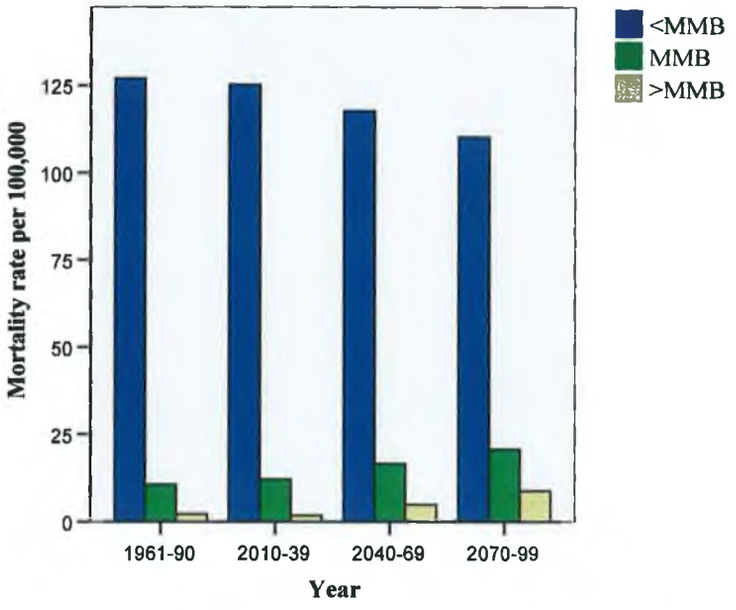


Figure 8(b) Impact of change in mean temperature with respect to mortality from respiratory disease per 100,000 population per year in relation to minimum mortality band (MMB) of 16 °C -19 °C

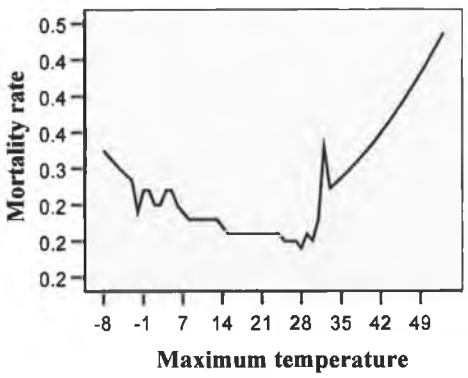
Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-5	-10	-12
CCM B2	-6	-8	-11
CSIRO A2	-5	-9	-13
CSIRO B2	-6	-10	-12
Had3CM A2	-1	-4	-9
Had3CM B2	-3	-4	-6
Mean	-4	-8	-11

Table 8(a) Mean changes in total mortality from respiratory disease per 100,000 population resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

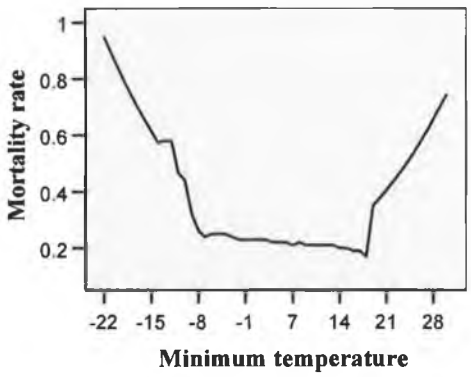
Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-4.0	-8.1	-9.7
CCM B2	-4.8	-6.4	-8.9
CSIRO A2	-4.0	-7.3	-10.5
CSIRO B2	-4.8	-8.1	-9.7
Had3CM A2	-0.8	-3.2	-7.3
Had3CM B2	-2.4	-3.2	-4.8
Mean	-3.5	-6.0	-8.5

Table 8 (b) Mean percent changes in mortality from respiratory disease from the mean mortality rate in the adjusted time series, resulting from outputs from three climate models using A2 and B2 scenarios through three future time periods

APPENDIX F
Future impacts of changes in extreme temperatures



R square quadratic = 0.456 p<0.001



R square quadratic = 0.796 p<0.001

Figure 1 Extrapolation of the maximum and lagged minimum temperature-mortality relationship in the under 15 age specific population per 100,000

Maximum temperature
Mortality = $0.2663-0.0063(t)+0.00018(t)^2$

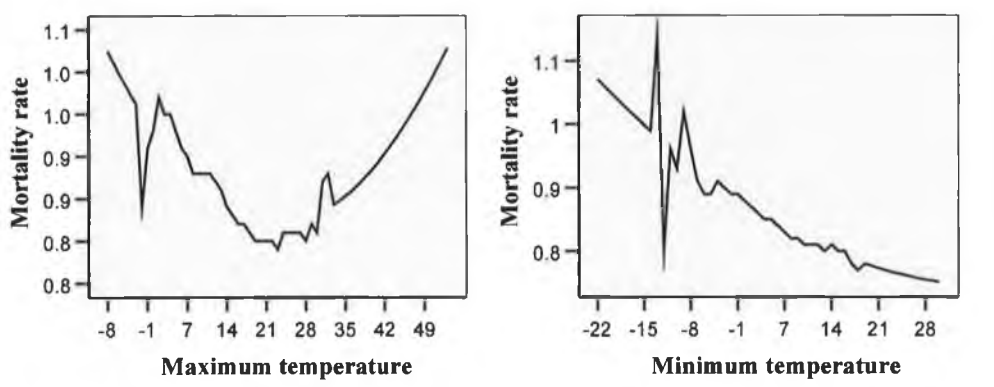
Minimum temperature
Mortality = $0.230-0.012(t)+0.001(t)^2$

Climate model and scenario	2010 – 2039	2040 - 2069	2070 - 2099
CCM A2	0	-1	-2
CCM B2	0	-1	-1
CSIRO A2	-1	-2	-3
CSIRO B2	-1	-2	-4
Had3CM A2	0	-1	-1
Had3CM B2	0	-1	-2
Mean Range	0 (-1 to 0)	-1 (-1 to -2)	-2 (-1 to -4)

Table 1(a) Range in the changes in mortality in the 14 and under age-specific population per 100,000 resulting from future maximum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1	-1	-2
CCM B2	-1	-2	-2
CSIRO A2	0	-1	-2
CSIRO B2	-1	-1	-1
Had3CM A2	0	-1	-1
Had3CM B2	0	0	0
Mean Range	-1 (-1 to 0)	-1 (-2 to 0)	-1 (-2 to 0)

Table 1(b) Range in the changes in mortality in the under 15 year age-specific population per 100,000 resulting from future minimum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios



R square quadratic = 0.653 p<0.001

R square quadratic = 0.620 p<0.001

Figure 2 Extrapolation of the maximum and lagged minimum temperature-mortality relationship in the 15-64 age specific population per 100,000

Maximum temperature
Mortality = $0.9345-0.0130(t)+0.00023(t)^2$

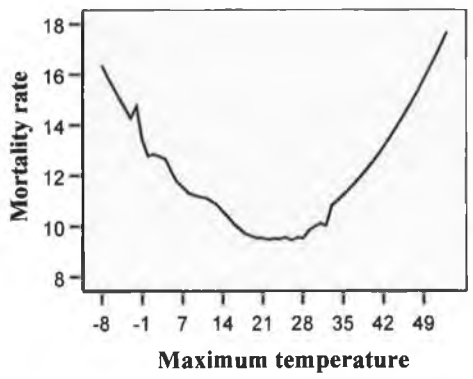
Minimum temperature
Mortality = $0.878-0.007(t)+0.000092(t)^2$

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	0	-3	-4
CCM B2	-1	-2	-3
CSIRO A2	-3	-5	-9
CSIRO B2	-4	-5	-10
Had3CM A2	-1	-2	-4
Had3CM B2	-1	-2	-7
Mean Range	-2 (0 to -3)	-3 (-2 to -5)	-6 (-3 to -10)

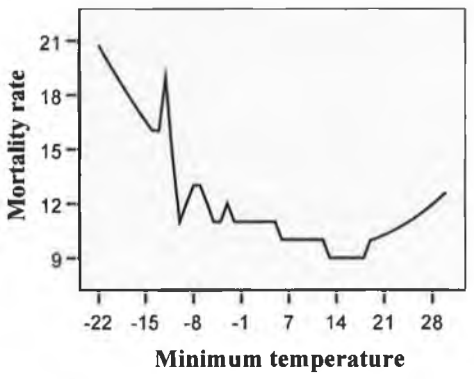
Table 2 (a) Range in the changes in mortality in the 15-64 year age-specific population per 100,000 resulting from future maximum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-2	-5	-7
CCM B2	-2	-3	-4
CSIRO A2	0	-2	-7
CSIRO B2	-3	-2	-3
Had3CM A2	0	-2	-5
Had3CM B2	1	0	-1
Mean Range	-1 (+1 to -3)	-2 (0 to -5)	-5 (-1 to -7)

Table 2 (b) Range in the changes in mortality in the 15-64 year age-specific population per 100,000 resulting from future minimum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios



R square quadratic = 0.972 p<0.001



R square quadratic = 0.782 p<0.001

Figure 3 Extrapolation of the maximum and lagged minimum temperature-mortality relationship in the 65-74 age specific population per 100,000

Maximum temperature
Mortality = $13.372-0.338(t)+0.008(t)^2$

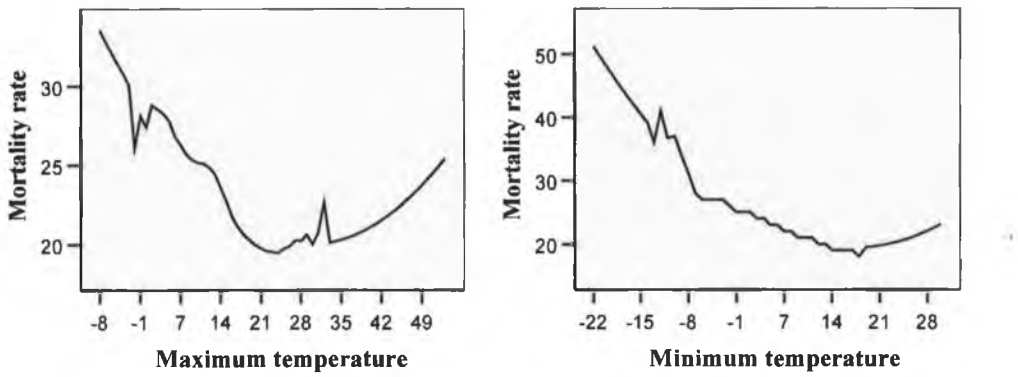
Minimum temperature
Mortality = $10.983-0.240(t)+0.010(t)^2$

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-3	-62	-84
CCM B2	-16	-37	-66
CSIRO A2	-60	-101	-194
CSIRO B2	-78	-115	-218
Had3CM A2	-13	-48	-94
Had3CM B2	-28	-43	-148
Mean Range	-33 (-3 to -78)	-68 (-37 to -101)	-134 (-66 to -218)

Table 3 (a) Range in the changes in mortality in the 65-74 year age-specific population per 100,000 resulting from future maximum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-55	-124	-159
CCM B2	-41	-76	-101
CSIRO A2	-5	-51	-154
CSIRO B2	-77	-58	-85
Had3CM A2	-12	-48	-100
Had3CM B2	19	7	-25
Mean Range	-29 (-12 to -77)	-58 (+7 to -124)	-104 (-25 to -154)

Table 3(b) Range in the changes in mortality in the 65-74 year age-specific population per 100,000 resulting from future minimum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios



R square quadratic = 0.865 p<0.001

R square quadratic = 0.934 p<0.001

Figure 4 Extrapolation of the maximum and lagged minimum temperature-mortality relationship in the 75-84 age specific population per 100,000

Maximum temperature
Mortality = $28.578-0.585(t)+0.010(t)^2$

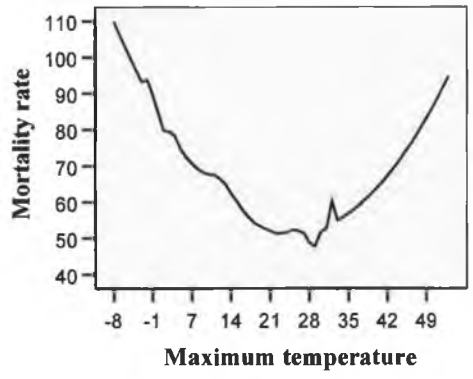
Minimum temperature
Mortality = $25.409-0.727(t)+0.022(t)^2$

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-15	-196	-264
CCM B2	-46	-120	-209
CSIRO A2	-177	-309	-607
CSIRO B2	-232	-349	-692
Had3CM A2	-46	-156	-321
Had3CM B2	-91	-145	-486
Mean Range	-101 (-15 to -232)	-213 (-120 to -349)	-430 (-209 to -692)

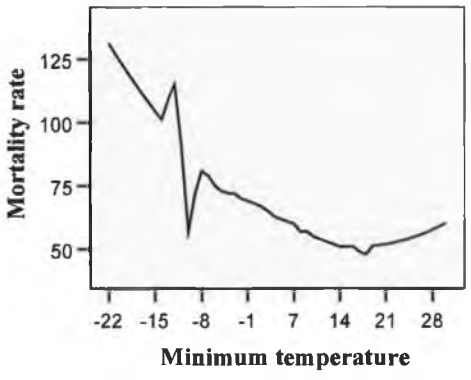
Table 4 (a) Range in the changes in mortality in the 75-84 year age-specific population per 100,000 resulting from future maximum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-167	-309	-351
CCM B2	-159	-226	-257
CSIRO A2	-30	-145	-347
CSIRO B2	-235	-189	-256
Had3CM A2	-30	-121	-238
Had3CM B2	64	49	-60
Mean Range	-93 (+64 to -235)	-157 (+49 to -309)	-252 (-60 to -351)

Table 4 (b) Range in the changes in mortality in the 75-84 year age-specific population per 100,000 resulting from future minimum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios



R square quadratic = 0.970 p<0.001



R square quadratic = 0.970 p<0.001

Figure 5 Extrapolation of the maximum and lagged minimum temperature-mortality relationship in the 85 and over age specific population per 100,000

Maximum temperature
Mortality = $85.915-2.773(t)+0.056(t)^2$

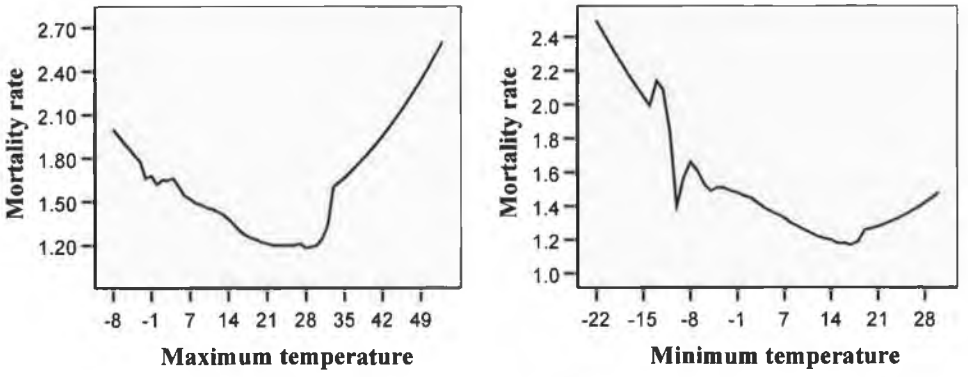
Minimum temperature
Mortality = $66.431-1.831(t)+0.055(t)^2$

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-33	-586	-815
CCM B2	-136	-353	-621
CSIRO A2	-547	-948	-1851
CSIRO B2	-714	-1057	-2082
Had3CM A2	-137	-478	-970
Had3CM B2	-268	-440	-1460
Mean Range	-306 (-33 to -714)	-644 (-353 to -1057)	-1300 (-621 to -2082)

Table 5 (a) Range in the changes in mortality in the 85 and over age-specific population per 100,000 resulting from future maximum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-519	-1128	-1417
CCM B2	-347	-663	-897
CSIRO A2	-64	-442	-1354
CSIRO B2	-648	-494	-710
Had3CM A2	-98	-428	-934
Had3CM B2	202	67	-171
Mean	-246	-515	-914
Range	(+202 to -648)	(+67 to -1128)	(-171 to -1417)

Table 5 (b) Range in the changes in mortality in the 85 and over age-specific population per 100,000 resulting from future minimum temperatures through three time periods using three climate model outputs with A2 and B2 scenarios



R square quadratic = 0.943 p<0.001

R square quadratic = 0.848 p<0.001

Figure 6 Extrapolation of the maximum and lagged minimum temperature-mortality relationship from cardiovascular disease per 100,000 population

Maximum temperature
Mortality = $1.683-0.035(t)+0.001(t)^2$

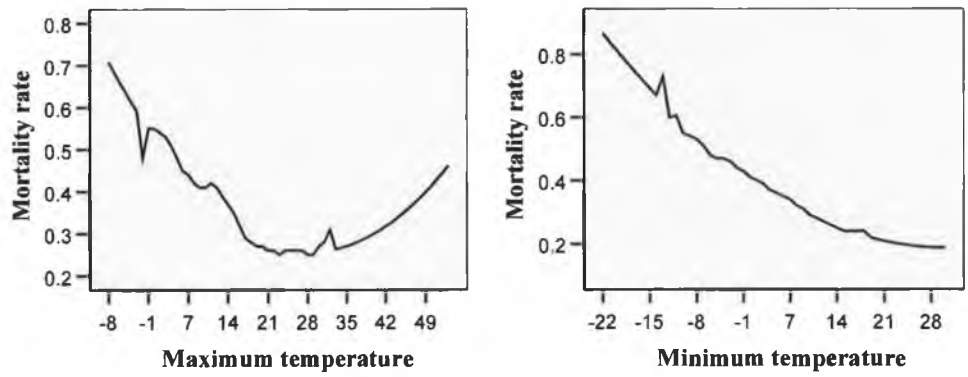
Minimum temperature
Mortality = $1.434-0.028(t)+0.001(t)^2$

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-1	-10	-13
CCM B2	-2	-6	-10
CSIRO A2	-9	-15	-30
CSIRO B2	-12	-17	-34
Had3CM A2	-2	-7	-14
Had3CM B2	-4	-7	-22
Mean	-5	-10	-21
Range	(-1 to -12)	(-6 to -17)	(-10 to -34)

Table 6 (a) Range in the changes in mortality from cardiovascular disease per 100,000 population resulting from future maximum temperatures through three time periods, using three climate model outputs with A2 and B2 scenarios

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-8	-17	-21
CCM B2	-5	-10	-13
CSIRO A2	-1	-7	-21
CSIRO B2	-10	-8	-11
Had3CM A2	-2	-7	-14
Had3CM B2	3	1	-3
Mean Range	-4 (+3 to -10)	-8 (+1 to -17)	-14 (-3 to -21)

Table 6 (b) Range in the changes in mortality from cardiovascular disease per 100,000 population resulting from future minimum temperatures through three time periods, using three climate model outputs with A2 and B2 scenarios



R square quadratic = 0.927 p<0.001 *R square quadratic = 0.927 p<0.001*

Figure 7 Extrapolation of the maximum and lagged minimum temperature-mortality relationship from respiratory disease per 100,000 population

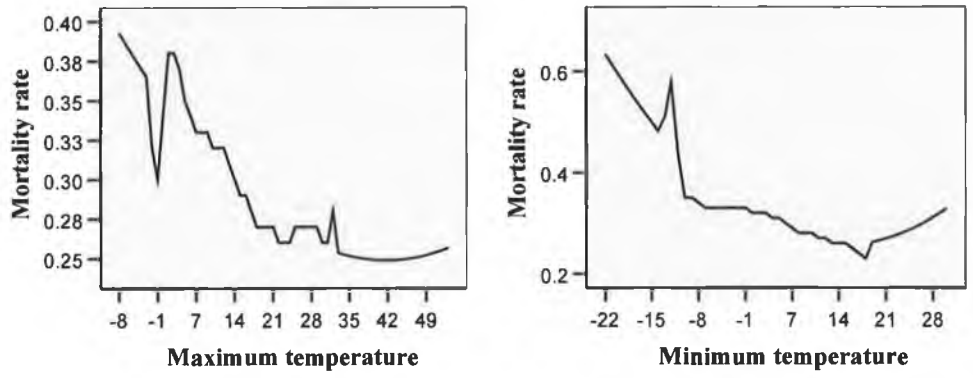
Maximum temperature Minimum temperature
Mortality = $0.5390-0.00198(t)+0.00035(t)^2$ Mortality = $0.415-0.0154(t)+0.00026(t)^2$

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	0	-5	-8
CCM B2	-1	-3	-6
CSIRO A2	-5	-9	-17
CSIRO B2	-6	-10	-20
Had3CM A2	-1	-4	-9
Had3CM B2	-2	-4	-14
Mean Range	-3 (0 to -6)	-6 (-4 to -10)	-12 (-6 to -20)

Table 7 (a) Range in the changes in mortality from respiratory disease per 100,000 population resulting from future maximum temperatures through three time periods, using three climate model outputs with A2 and B2 scenarios

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-5	-11	-14
CCM B2	-3	-6	-9
CSIRO A2	-1	-4	-14
CSIRO B2	-7	-5	-7
Had3CM A2	-1	-4	-10
Had3CM B2	2	1	-2
Mean Range	-3 (+2 to -7)	-5 (+1 to -11)	-9 (-2 to -14)

Table 7 (b) Range in the changes in mortality from respiratory disease per 100,000 population resulting from future minimum temperatures through three time periods, using three climate model outputs with A2 and B2 scenarios



R square quadratic = 0.750 p<0.001 *R square quadratic = 0.779 p<0.001*

Figure 8 Extrapolation of the maximum and lagged minimum temperature-mortality relationship from stroke per 100,000 population

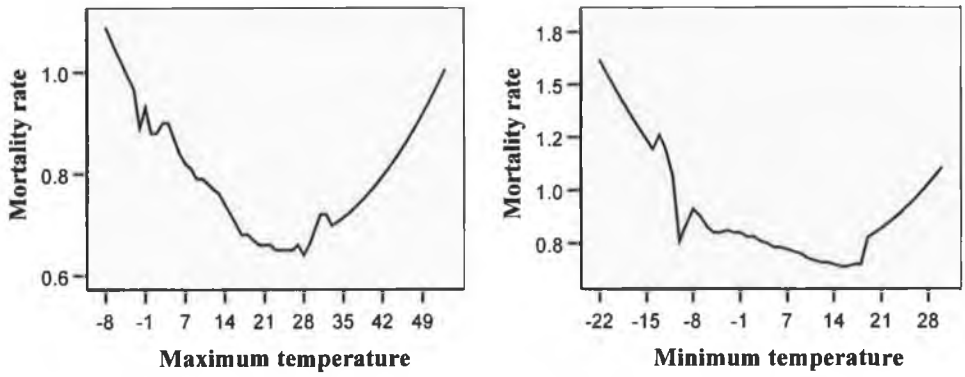
Maximum temperature Minimum temperature
Mortality = $0.352-0.005(t)+0.00006(t)^2$ Mortality = $0.315-0.0084(t)+0.0003(t)^2$

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	0	-2	-3
CCM B2	-1	-1	-2
CSIRO A2	-2	-3	-7
CSIRO B2	-3	-4	-7
Had3CM A2	0	-2	-4
Had3CM B2	-1	-2	-5
Mean Range	-1 (0 to -3)	-2 (-1 to -4)	-5 (-4 to -7)

Table 8 (a) Range in the changes in mortality from stroke per 100,000 population resulting from future maximum temperatures through three time periods, using three climate model outputs with A2 and B2 scenarios

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-2	-4	-6
CCM B2	-1	-2	-3
CSIRO A2	0	-2	-5
CSIRO B2	-2	-2	-3
Had3CM A2	0	-2	-4
Had3CM B2	1	0	-1
Mean Range	-1 (0 to -2)	-2 (0 to -4)	-4 (-1 to -6)

Table 8 (b) Range in the changes in mortality from stroke per 100,000 population resulting from future minimum temperatures through three time periods, using three climate model outputs with A2 and B2 scenarios



R square quadratic = 0.936 p<0.001

R square quadratic = 0.820 p<0.001

Figure 9 Extrapolation of the maximum and lagged minimum temperature-mortality relationship from ischaemic heart disease per 100,000 population

Maximum temperature
Mortality = $0.914-0.002(t)+0.0004(t)^2$

Minimum temperature
Mortality = $0.768-0.018(t)+0.001(t)^2$

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	0	-5	-7
CCM B2	-1	-3	-6
CSIRO A2	-5	-8	-16
CSIRO B2	-6	-9	-18
Had3CM A2	-1	-4	-8
Had3CM B2	-2	-4	-12
Mean Range	-2 (0 to -6)	-6 (-3 to -9)	-10 (-6 to -18)

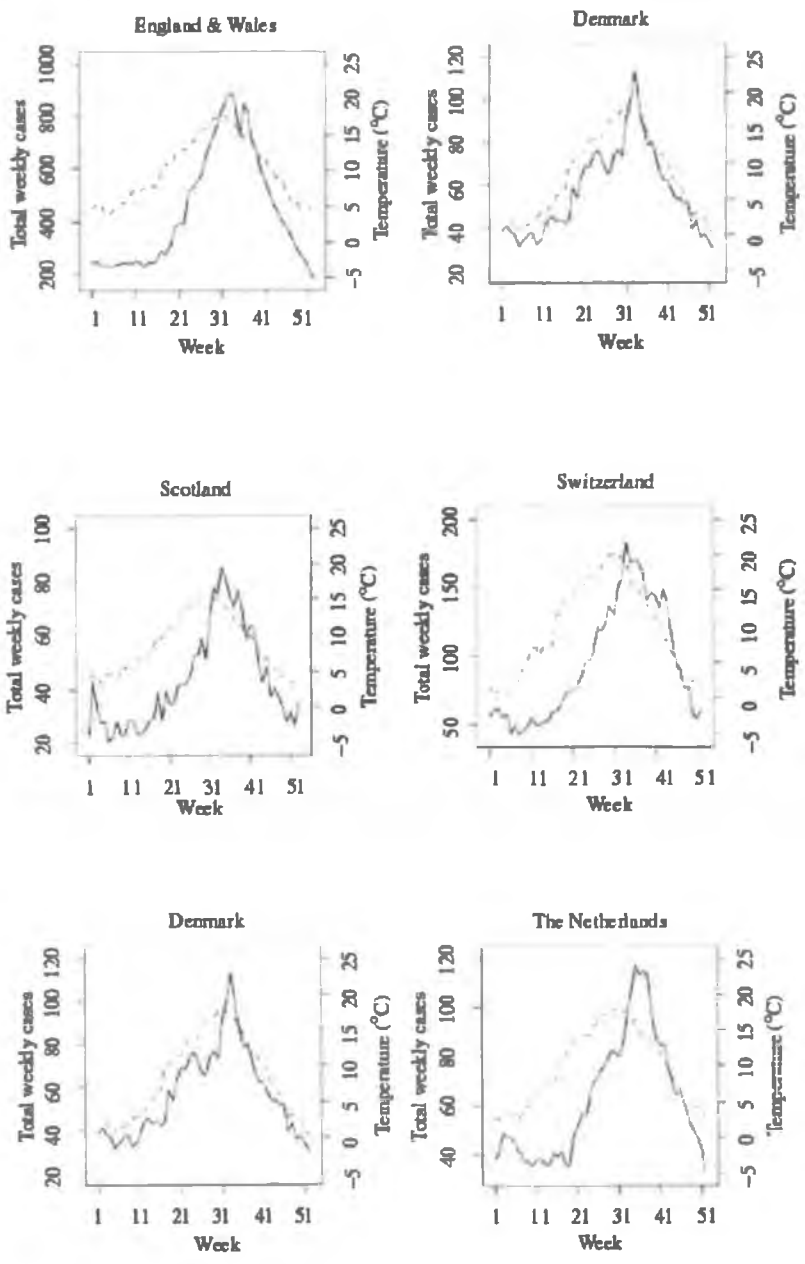
Table 9 (a) Range in the changes in mortality from ischaemic heart disease per 100,000 population resulting from future maximum temperatures through three time periods, using three climate model outputs with A2 and B2 scenarios

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	-4	-8	-10
CCM B2	-3	-5	-7
CSIRO A2	-1	-3	-10
CSIRO B2	-5	-4	-6
Had3CM A2	-1	-3	-7
Had3CM B2	1	1	-1
Mean Range	-2 (+1 to -4)	-4 (+1 to -8)	-7 (-1 to -10)

Table 9 (b) Range in the changes in mortality from ischaemic heart disease per 100,000 population resulting from future minimum temperatures through three time periods, using three climate model outputs with A2 and B2 scenarios

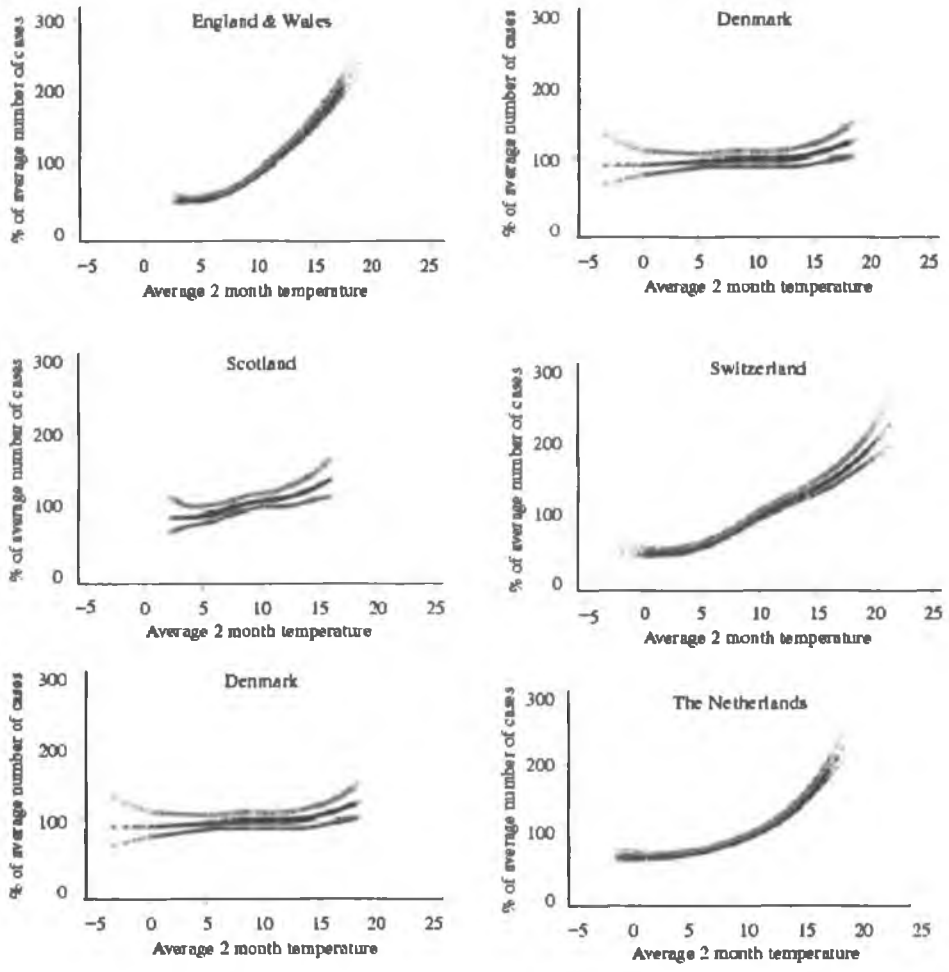
APPENDIX G

Salmonella-temperature relationship



Dashed line = average temperature; straight line =number of cases *(from Kovats et al, 2004(b))*

Figure 1 Seasonality of Salmonella and the lag effect



(from Kovats et al, 2004(b))

Figure 2 Temperature and the incidence of salmonella

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	0.2	0.4	0.6
CCM B2	0.2	0.4	0.5
CSIRO A2	0.2	0.3	0.6
CSIRO B2	0.2	0.4	0.4
Had3CM A2	0.1	0.2	0.5
Had3CM B2	0.1	0.2	0.3
Mean **Range**	0.2 (0.1 to 0.2)	0.3 (0.2 to 0.4)	0.5 (0.3 to 0.6)

Table 1 Changes in case of salmonella per year resulting from increases in mean temperatures

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	1.5	3.4	4.6
CCM B2	1.8	2.8	3.7
CSIRO A2	1.3	2.6	4.2
CSIRO B2	1.8	2.8	3.4
Had3CM A2	0.4	1.5	3.5
Had3CM B2	0.7	1.4	2.2
Mean **Range**	0.2 (0.1 to 0.2)	0.3 (0.2 to 0.4)	0.5 (0.3 to 0.6)

Table 2 Percentage changes in the incidence of salmonella resulting from increases in mean temperatures

APPENDIX H

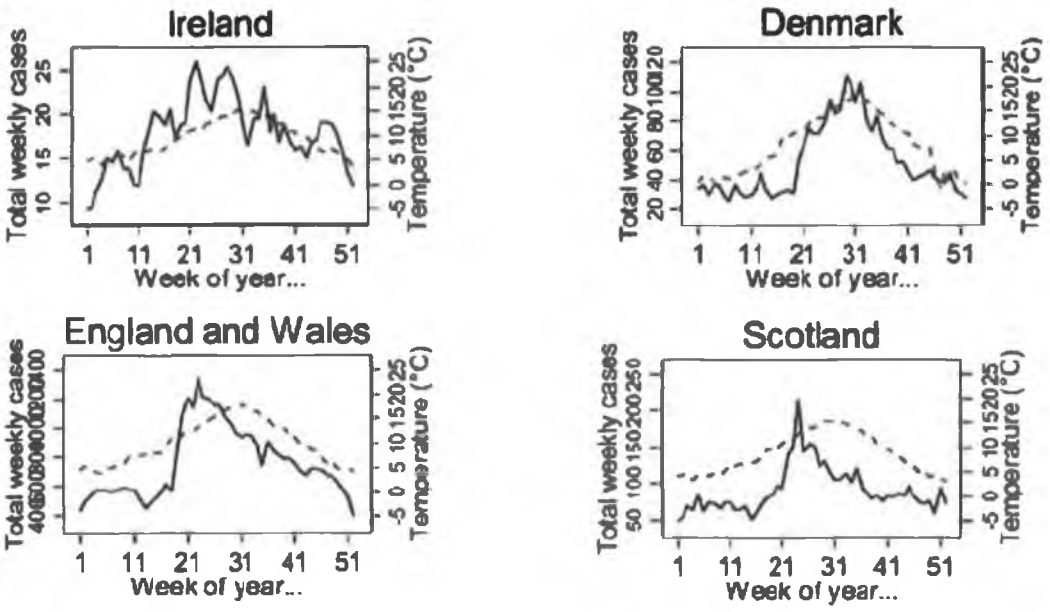
Campylobacter-temperature relationship

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	76	149	164
CCM B2	85	124	176
CSIRO A2	73	127	194
CSIRO B2	93	141	158
Had3CM A2	21	47	93
Had3CM B2	44	46	79
Mean Range	65 (21 to 93)	106 (46 to 141)	144 (79 to 144)

Table 1 Changes in case of campylobacter per year resulting from increases in mean temperatures

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	4.3	8.4	9.3
CCM B2	4.8	7.0	9.9
CSIRO A2	4.1	7.2	11.0
CSIRO B2	5.2	8.0	9.0
Had3CM A2	1.2	2.6	5.2
Had3CM B2	2.5	2.6	4.5
Mean Range	3.7 (1.2 to 5.2)	6.0 (2.6 to 8.4)	8.1 (4.5 to 11.0)

Table 2 Percentage changes in the incidence of campylobacter resulting from increases in mean temperatures



(from Kovats et al, 2005).

Figure 1 Seasonal patterns of campylobacter in selected European countries

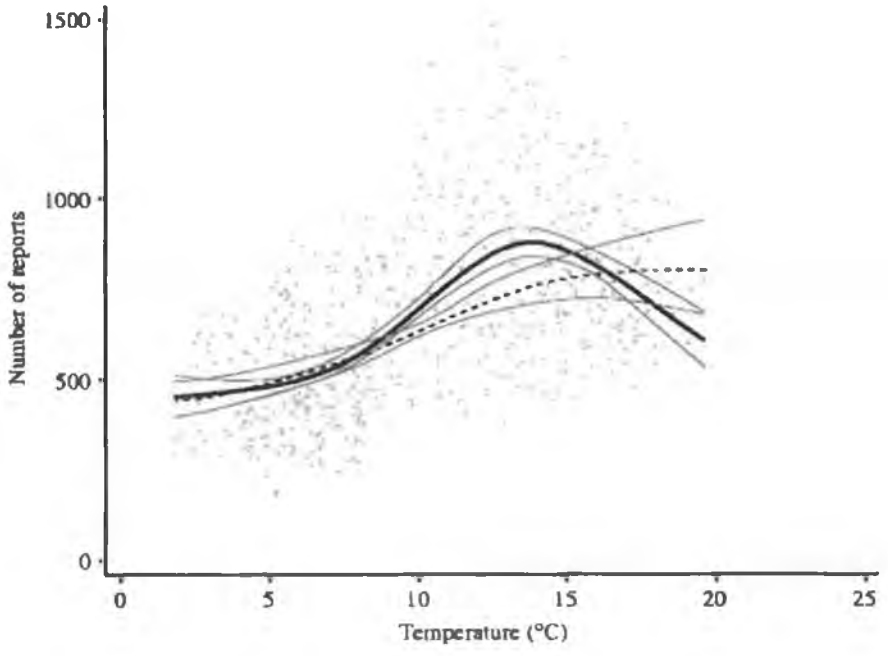


Figure 2 Campylobacter-temperature relationship (Bold line- unadjusted natural cubic spline models with two knots for the temperature series. —, Unadjusted model; - - -, the effect of temperature adjusted for trend, seasonality (up to the 8th harmonic), public holidays and relative humidity).

APPENDIX I

VTEC 0157-temperature relationship

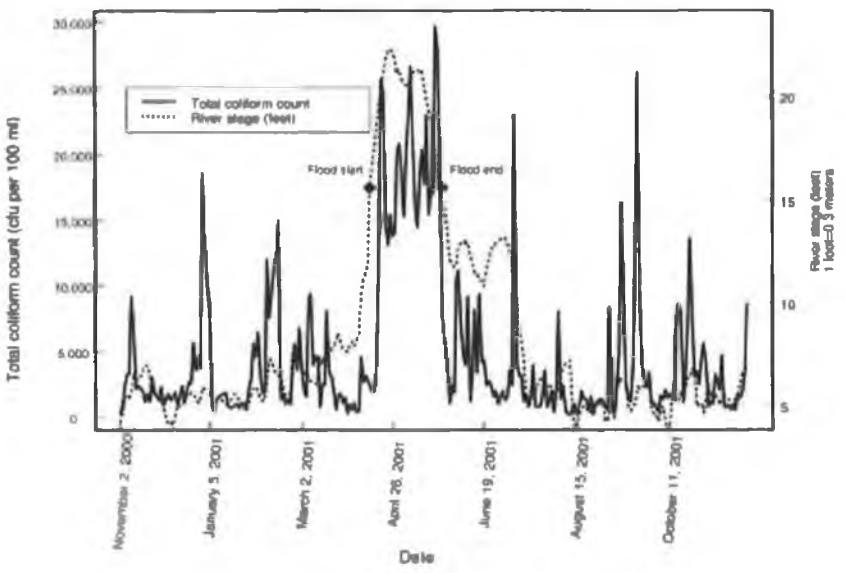
Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	7	15	19
CCM B2	13	13	16
CSIRO A2	7	12	19
CSIRO B2	9	13	16
Had3CM A2	2	6	14
Had3CM B2	9	6	9
Mean Range	8 (2 to 13)	11 (6 to 15)	16 (9 to 19)

Table 1 Changes in case of VTEC 0157 per year resulting from increases in mean temperatures

Climate model and scenario	2010 - 2039	2040 - 2069	2070 - 2099
CCM A2	11.3	24.9	32.1
CCM B2	21.3	21.3	27.4
CSIRO A2	11.1	20.3	31.6
CSIRO B2	15.0	21.8	26.2
Had3CM A2	2.9	10.6	23.8
Had3CM B2	15.0	9.4	15.2
Mean Range	12.7 (2.9 to 21.3)	18.0 (9.4 to 24.9)	26.0 (15.2 to 32.1)

Table 2 Percentage changes in the incidence of VTEC 0157 resulting from increases in mean temperatures

APPENDIX J
Water quality following an episode of flooding



(from Wade et al, 2004)

Figure 1Total coliform counts in raw water following flood in Mississippi River November 2000-2001

REFERENCES

Aarestrup, F.A. and Engberg, J. (2001) "Antimicrobial resistance of thermophilic Campylobacter", *Veterinary Research* 32 (3-4) 311-21

Abrahams, M.J., Price, J., Whitlock, F.A., and Williams, G. (1976) "The Brisbane floods, January 1974: their impact on health", *Medical Journal of Australia* 25-26 936-9

ACACIA (2000) "Health", in Parry, M. (Ed.) *Assessment of Potential Effects and Adaptations for Climate change in Europe* University of East Anglia, European Commission

Adak, G.K. Adak, G.K. Long, S.M. and O'Brien, S.J. (2002) "Trends in indigenous foodborne disease and deaths England and Wales:1992-2000", *Gut* 51 832-41

Alderson, M.R. (1985) "Season and Mortality", *Health Trends* 17 87-96
American Academy of Pediatrics (1998) "Toxic Effects of Indoor Molds Committee on Environmental Health", *Pediatrics,* 101 (4) 712-714

Anderson, C.A., Bushman, B.J. and Groom, R.W. (1997) "Hot years and serious and deadly assault: empirical tests of the heat hypothesis", *Journal of Personality and Social Psychology* 73 (6) 1213-23.

Annals of Clonmacnoise in *Sea level changes and Ireland*
http://www.enfo.ie/leaflets/bs27.htm Website accessed 7th August 2007

AR4 (2007), Fourth Assessment Report *Climate Change 2007: Impacts, Adaptation and Vulnerability* Parry, M., Canziani, O.F., Palutikof, J., van der Linden, P. and Hanson, C.(Eds.) International Panel on Climate Change Cambridge University Press, Cambridge

Ashe, P., O'Connor, J.P. and Casey, R.J. (1991) "Irish Mosquitoes, (Diptera: Culicidae) a checklist of the species and known distribution", *Proceedings of the Royal Irish Academy B* 91 21-36

Asthma Care (2006), httpww.asthmacare.ie/blog/?cat=25 Website accessed 30th August 2006

Atherholt, T., Thomas, B., LeChevallier, M.W., Norton, W.D. and Rosen, J.S. (1998) "Effect of rainfall on Giardia and Cryptosporidiosis", *Journal of the American Water Works Association* 90 (9) 66-80

Auld, H., MacIver, D. and Klaassen, J. (2004) "Heavy rainfall and waterborne disease outbreaks: the Walkerton example", *Journal of Toxicology and Environmental Health* 67 (20-22) 1879-87.

Aylin, P., Morris, S., Wakefield, J., Grossinho, A., Jarup, L. and Elliott, P. (2001) "Temperature, housing, deprivation and their relationship to excess winter mortality in Great Britain 1986", *International Journal of Epidemiology* 30 1100-1108

Azevedo, E., Ribeiro, J.A., Lopes, F., Martins, R. and Barros, H.(1995) "Cold: a risk factor for stroke?", *Journal of Neurology* 242 (4) 217-21.

Baird-Parker, AC. quoted in Kovats, R.S., Edwards, S.J., Hajat, S., Armstrong, B.G., Ebi, K.L. and Menne, B. (2004) "The effect of temperature on food poisoning: a time-series analysis of salmonellosis in ten European countries", *Epidemiology and Infection* 132 (3) 443-53

Balbus, J.M., Bouma, M., Kovats, S., LeSueur, D., Martens, P. and Patz, J. (1998) "Human Health" in: Feenstra, JF., Burton, I., Smith, JB. and Tol, RSJ. *"Handbook on methods for climate change impacts assessment and adaptation strategies*", United Nation Environment Programme (UNEP) and Institute for Environmental Studies, Nairobi-Amsterdam

Baldari, M., Tamburro, A., Sabatinelli, G., Romi, R., Severini, C., Cuccagna, G., Fiorilli, G., Allegri, M.P., Buriani, C. and Toti, M. (1998) "Malaria in Maremma Italy", *The Lancet* 351 (9111) 1246-1247

Ballester, F., Corella, D., Perez-Hoyos, S., Saez, M. and Hervas, A. (1997) "Mortality as a function of temperature. A study in Valencia, Spain, 1991-1993", *International Journal of Epidemiology* 26 (3) 551-61

Ballester, F. and Sunyer, J. (2000) "Drinking water and gastrointestinal disease: need of better understanding and an improvement in public health surveillance", *Journal of Epidemiology and Community Health* 54 3-5

Barnett, A.G., Dobson, A.J., McElduff, P., Salomaa, V., Kuulasmaa, K. and Sans, S. (2005). "Cold periods and coronary events: an analysis of populations worldwide", *Journal of Epidemiology and Community Health* 59 (7) 551-7.

Basu, R. and Samet, J. (2002) "Relation between elevated ambient temperature and mortality: a review of the epidemiologic evidence", *Epidemiologic Reviews* 24 (2) 190-202.

Beggs, P.J. (2004) "Impacts of climate change on aeroallergens: past and future", *Clinical and Experimental Allergy* 34 (10) 1507-13.

Beggs, P.J. and Bambrick, H.J. (2005) "Is the global rise of asthma an early impact of anthropogenic climate change?", *Environmental Health Perspectives* 113 (8) 915-9

Bell, P.A. (2005) "Reanalysis and perspective in the heat--aggression debate", *Journal of Personality and Social Psychology* 89 (1) 71-3.

Beniston, M. (2004) "The 2003 heat wave in Europe: A shape of things to come?", *Geophysical Research Letters*, (31) LO 02202.1-LO2202.4

Bennett, K., Kabir, Z., Unal, B., Shelley, E., Critchley, J., Perry, I., Feely, J. and Capewell, S. (2006) "Explaining the recent decrease in coronary heart disease mortality rates in Ireland, 1985-2000", *Journal of Epidemiology and Community Health* 60 (4) 322-7

Bentham, G. and Langford, I.H. (1995) "Climate change and the incidence of food poisoning in England and Wales", *International Journal of Biometeorology* 39 81-86

Bentham, G.B. (2001) "Food poisoning and climate change", in *Health effects of climate change in the UK* Department of Health London 81-84

Bentham, G. and Langford, I.H. (2001) "Environmental temperatures and the incidence of food poisoning in England and Wales", *International Journal of Biometeorology* 45 (1) 22-26

Beran, J. (2004) "Tickborne encephalitis in the Czech Republic" *Eurosurveillance* (8) 24.

Bernard, S. and Ebi, K. (2001) "Comments on the Process and Product of the Health Impacts Assessment Component of the National Assessment of the Potential Consequences of Climate Variability and Change for the United States", *Environmental Health Perspectives* 109 (2) 177-184

Bernard, S.M. and Mc Geehin, M.A. (2004) "Municipal heat wave response plans", *American Journal of Public Health* 9 (9) 1520-2

Betancourt, W.Q. and Rose, J.B. (2004) "Drinking water treatment processes for removal of Cryptosporidium and Giardia", *Veterinary Parasitology* 126 (1-2) 219-34

Black, R.E. *et al* (1995) quoted in Checkley, W., Epstein, L.D., Gilman, R.H., Figueroa, D., Cama, R.I., Patz, J.A. and Black, R.E. (2000) "Effects of El Niño and ambient temperature on hospital admissions for diarrhoeal diseases in Peruvian children", *The Lancet* 355 (9202) 442-50

Boelens, R. Minchin, D. and O'Sullivan, G. (2005) "Climate Change Implications for Ireland's Marine environment and resources", *Marine Foresight Series Number 2*

Bokenes, L., Alexanderson, T.E., Osterud, B., Tveita, T. and Mercer, J.B. (2000) "Physiological and haematological responses to cold in the elderly", *International Journal of Circumpolar Health* 59 (3-4) 216-221

Booth, S. and Zeller, D. (2005) "Mercury, food webs, and marine mammals: implications of diet and climate change for human health", *Environmental Health Perspectives* 113 (5) 521-6.

Boulay, F., Berthier, F., Sisteron, O., Gendreike and Y. Gibelin, P. (1999) "Seasonal variation in chronic heart failure hospitalisations and mortality in France", *Circulation* 100 280-286

Boyden, M. (Director) *Streamscapes Aquatic Education Programme* Personal Communication 29-9-06

Bradley, D. (2004) quoted in Moe, C.L. "What are the criteria for determining whether a disease is zoonotic and water related?", in Cotruvo, A., Dufour, A., Rees, G.,

Bartram, J., Carr, R., Cliver, D.O., Craun, G.F., Fayer, R. and Gannon, V.P.J.(Eds.) *Impacts of anthropogenic and environmental factors in Identification, causes and control of Waterborne disease* WHO London

Bradshaw, W.E. and Holzapfel, C.M. (2001) "Genetic shift in photoperiodic response correlated with global warming", *Proceedings of the National Academy of Sciences* 98 (25) 14509-14511

Braga, A.L., Zanobetti, A. and Schwartz, J. (2001) "The time course of weather-related deaths", *Epidemiology* 12 662–7

Brennan, N., McCormack, S. and O'Connor, T.M. (2008) "Ireland needs healthier airways and lungs: the evidence", *Irish Thoracic Society* INHALE Report 2nd edition

Bresser, A.H.M ., Berk, M.M., van den Born, G.J., van Bree L., van Gaalen, F.W., Ligtvoet, W., van Minnen, J.G., Witmer, M.C.H., Amelung, B., Huynen, M.M.T.E., Bolwidt, L., ten Brinke,W., Buiteveld, H., Dillingh, D., van Dorland, R., Leemans, R., van Strien, A., Vermaat. J., Veraart, J., Bresser, A.H.M. and Berk, M.M. (Eds.) (2005) "The effects of climate change in the Netherlands Netherlands Environmental Assesment Agency" Netherlands Environmental Assessment Agency, Bilthoven

Bridgman, S.A., Robertson, R.M., Syed, Q., Speed, N., Andrews, N. and Hunter, P.R. (1995) "Outbreak of cryptosporidiosis associated with a disinfected groundwater supply", *Epidemiology and Infection* 115 (3) 555-66

Brophy, V. quoted in Healy, J. and Clinch, P. (2002) "Fuel Poverty and health an empirical analysis using Ireland as a case study 02/06", *Environmental Studies Research Series Working papers* Department of Environmental Studies University College Dublin

Brownstein, J.S., Holford, T.R. and Fish, D. (2003) "A Climate-Based Model Predicts the Spatial Distribution of the Lyme Disease Vector Ixodes scapularis in the United States", *Environmental Health Perspectives* 111 (9) 1152-1157

Bruce-Chwatt, L.J. and de Zulueta, J. (1980) *The Rise and Fall of Malaria in Europe: A Historico-Epidemiological Study*. Oxford, Oxford University Press,

Bryan, J.H., Foley, D.H. and Sutherst, R.W. (1996). "Malaria transmission and climate change in Australia", *Medical Journal of Australia* 164 345-47

Bull, G.M. (1973) "Meteorological correlates with myocardial and cerebral infarction and respiratory disease", *British Journal of Social and Preventive Medicine* 27 108-113

Bull, G.M. and Morton. J.(1978) "Environment, temperature and death rates" *Age and Aging* 7 (4) 210-24

Burke, D. , Carmichael, A., Focks, D. ,Grimes, J.D., Harte, J., Lele, S., Martens, P., Mayer, J., Mearns, L., Pulwarty, R., Real, L., Rose, J. , Ropelewski, C., Shope, R., Simpson, J., Wilson, M. and Geller, L. (2001) "Under the Weather: Climate, Ecosystems, and Infectious Disease", *Centre for Disease Control* 7 (3) 606-608

Burke, D. (2007) Personal communication Inspector, Public Authority Enforcement Section, Environmental Protection Agency

Byrne, C. (2006) "*A future for our waters*" Presented at "A Future for our Waters" 29th September 2006 Water Department Cork County Council

Cagney C., Crowley, H., Duffy, G., Sheridan, J.J., O'Brien, S., Carney, E., Anderson, W., Mc Dowell, D.A., Blair. I.S. and Bishop, R.H. (2004) "Prevalence and numbers of Escherichia coli O157:H7 in minced beef and beef burgers from butcher shops and supermarkets in the Republic of Ireland", *Food Microbiology* 21 (2) 203-212

Campbell, L., Fritz, C., Fish, D., Nowakowski, J.R., Nadelman, R.B. and Wormser, G.P. (1998) "Estimation of the incidence of Lyme disease", *American Journal of Epidemiol*ogy 148 1018–1026

Campo, J., Comer, H. and Gavin, A. (2004) *All-Ireland Cancer statistics Second Report 1998-2000* Department of Health and Children, Ireland, Department of Health, Social Services ad Public Policy, Northern Ireland and United states National Cancer Institute

Carder, M., McNamee, R., Beverland, I., Elton, R., Cohen, G.R., Boyd, J. and Agius, R.M. (2005) "The lagged effect of cold temperature and wind chill on cardiorespiratory mortality in Scotland", *Occupational and Environmental Medicine* 62 (10) 702-10.

Carlow County Council (2005) http://216.239.59.104/search?q=cache:8nABsme3OtgJ:www.countycarlow.ie/services/healthwarnings/reports/CryptosporidiumReportFinal.doc+carlow+cryptosporidiosis&hl=en&ct=clnk&cd=1 Website accessed 4th June 2007

Carnicer-Pont, D., Atenstaedt, R., Walker, M., Chalmers, R., Rees, A., Rowlands, K. Evans, M. and Salmon, R. (2005) "An outbreak of cryptosporidiosis in Wales, November 2005", *Eurosurveillance* 10 (12) E051208.4

Carson, J.W. (1989) "Changing patterns in childhood gastroenteritis", *Irish Medical Journal* 82 (2) 66–67.

Carson, C., Hajat, S. and Wilkinson, P. (2006) "Declining vulnerability to temperature-related mortality in London over the 20th century", *American Journal of Epidemiology* 164 (1) 77-84

Casimiro, E., Calheiros, J., Santos, F.J. and Kovats, R.S. (2006) "National Assessment of Human Health Impacts of Climate Change in Portugal: Approach and Key Findings", *Environmental Health Perspectives* 114 (112) 1950-1956

cCASHh (2005) *Climate Change and Adaptation Strategies for Human health* WHO Regional Office for Europe Website http www.euro.who.int/ccashh Website accessed 4th November 2005

CCDR Canadian Communicable Disease Report (2000) "Waterborne outbreak of gastroenteritis associated with a contaminated municipal water supply, Walkerton Ontario, May-June 2000", Vol 16-20 http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/00vol26/dr2620eb.html Website accessed 5th July 2006

CCIRG Climate change Impacts Review Group (1996) "Review of the Potential effects of Climate change in the United Kingdom", London UK HMSO

CDC Centre for Disease Control (2004) Website accessed 23rd December 2004 http://www.cdc.gov/hab/cyanobacteria/about.htm

Chalmers, R.M., Sturdee, A.P., Mellors, P., Nicholson, V., Lawlor, F., Kenny, F. and Timpson, P. (1997) "Cryptosporidium parvum in environmental samples in the Sligo area Republic of Ireland: a preliminary report", *Letters in Applied Microbiology* 25 380-384

Chan, M. "A warning on health in a changing climate", *Irish Medical Times* 15-06-07

Chapman (1995) "Verocytotoxin-producing Escherichia. coli: an overview with emphasis on the epidemiology and prospects for control of E. coli O157" *Food Control* 1995 6 (4) 187-193

Charlton, R. and Moore, S. (2003) "The impact of climate change on water resources in Ireland" in *Climate Change: Scenarios and Impacts for Ireland* Environmental Protection Agency Ireland

Charron, D., Thomas, M., Waltner-Toews, D., Aramini, J., Edge, T., Kent, R., Maarouf, A. and Wilson, J. (2004) "Vulnerability of waterborne diseases to climate change in Canada: a review", *Journal of Toxicology and Environmental Health* 67 (20-22) 1667-77.

Checkley, W., Epstein, L.D., Gilman, R.H., Figueroa, D., Cama, R.I., Patz, J.A. and Black, R.E. (2000) "Effects of El Niño and ambient temperature on hospital admissions for diarrhoeal diseases in Peruvian children" *The Lancet* 355 (9202) 442-50

Clancy, L., Goodman, P., Sinclair, H. and Dockery, D.W. (2002) "Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study", *The Lancet* 360 (9341) 1210-4

Clinch, J. and Healy ,J.D. (2000) "Housing standards and excess winter mortality", *Journal of Epidemiology and Community Health* 54 719-720

Cloak, O.M., Duffy, G., Sheridan, J.J., Blair, I.S., and McDowell, D.A. (2001) "A survey on the incidence of campylobacter spp. and the development of a surface adhesion polymerase chain reaction (SA-PCR) assay for the detection of campylobacter jejuni in retail meat products" *Food microbiology* 18 (3) 287-298

Cogan, T.A. and Humphrey, T.J. (2003) "The rise and fall of Salmonella Enteritidis in the UK", *Journal of Applied Microbiology* 94 Supplement 114S-119S.

Cohen, J.C., Veysseire, J.M. and Bessemoulin, B. (2005) "Bioclimatological aspects of summer 2003 over France" in (Eds.) Kirch, W., Menne, B. and Bertollini, R. *Extreme Weather Events and Public health Responses* New York Springer 33-46

Cokkinides, V., Weinstock, M., Glanz, K., Albano, J., Ward, E. and Thun, M. (2006) "Trends in sunburns, sun protection practices, and attitudes toward sun exposure protection and tanning among US adolescents, 1998-2004", *Pediatrics* 118 (3) 853-64.

Conti, S., Meli, P., Minelli, G., Renata, R., Toccaceli, V., Vichi, M., Beltrano, C. and Perini, L. (2005) "Epidemiologic study of mortality during the Summer 2003 heat wave in Italy *Environmental Research",* 98 (3) 390-399

Corbett-Feeney, G. (1987) "Cryptosporidium among children with acute diarrhoea in the west of Ireland", *Journal of Infection* 14 (1) 79-84

Coris, E.E., Ramirez, A.M. and Van Durme, D.J. (2004) "Heat illness in athletes: the dangerous combination of heat, humidity and exercise", *Sports Medicine* 34 (1) 9-16

Corvalan, C.F., Gopalan, H.N.B. and Llanso, P. (2003) "Conclusions and recommendations for action" in Mc Michael, A.J., Campell-Lendrum, D.H., Corvalan, C., Ebi, K., Githeko, A., Scheraga, J.D. and Woodward A, (Eds.) *Climate change Risks and Responses* GENEVA WHO WMO UNEP Geneva WHO 267-283

Corvalan, C., Hales, S. and Mc Michael, A.J. (2005) "A Core Writing Team Eco-systems and human Well-being Health Systems" in Hassan, R. Scholes, R. Ash, N. (Eds.) A *Report of the Millennium Ecosystem Assessment* World Health Organization Geneva

Cotter, S. 2006 Health Protection Surveillance Centre Personal Communication on file

Craig, M.H., Kleinschmidt, I., Nawn, D., Le Sueur, D. and Sharp, B.L. 2004 (a) "Exploring 30 years of malaria case data in KwaZulu-Natal, South Africa: Part I. The impact of climatic factors", *Tropical Medicine and International Health.* 9 (12) 1247-57.

Craig, M.H., Kleinschmidt, I., Le Sueur, D. and Sharp, B.L. 2004 (b) "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 (12) 1258-66.

Craun, G.F., Calderon, R.L. and Craun, M.F. (2004) "Waterborne outbreaks caused by zoonotic pathogens in the USA" in Cotruvo, A., Dufour, A., Rees, G., Bartram, J., Carr, R., Cliver, D.O., Craun, G.F,, Fayer, R. and Gannon, V.P.J. (Eds.) *Identification, causes and control of Waterborne disease* World Health Organization

CSO Central statistics Office (2004) Populations and Labour Force Projections 2006-2036

CSO Central statistics Office (2005) Personal communication, data on file

Cunningham, A. (2005) "A walk on the wild side--emerging wildlife diseases", *British Medical Journal* 331 (7527) 1214-5.

Curriero, F.C., Patz, J.A., Rose, J.B. and Lele, S. (2001) "The association between extreme precipitation and waterborne disease outbreaks in the United States, 1948-1994", *American Journal of Public Health 91 (8) 1194-9.*

Curtis, C.F. and White, G.B. (1984) "Plasmodium falciparum mission in England: entomological and epidemiological data relative to cases in 1983" *Journal of Tropical Medicine and Hygiene* 87: 101-14.

Curtis, T.G.F. and Sheehy-Skeffington, M.J. (1998) "The Salt Marshes of Ireland: An inventory and an account of their geographical variation", *Proceedings of the Royal Irish Academy* 98B (2) 87-104

Curwen, M. (1990/1) "Excess winter mortality: a British phenomenon?", *Health Trends* 22 No 4 Registrar General's 2nd report

Daly, D. (2003) "The Walkerton Pollution incident A Summary" *The Geological Survey of Ireland Groundwater Newsletter* 42 13

Dangendorf, F. (2004) "Impacts of anthropogenic and environmental factors" in *Identification, causes and control of Waterborne disease* in Cotruvo, A., Dufour, A., Rees, G., Bartram, J., Carr, R., Cliver, D.O., Craun, G.F., Fayer, R., and Gannon V.P.J. World Health Organization

Davis, R.E., Knappenberger, P.C., Michaels, P,J. and Novicoff, W.M. (2003) "Changing heat related mortalities in the United States", *Environmental Health Perspectives* 111 (14) 1712-8.

DCC Newsletter (2007) "*Investigating potential new sources of water for the Greater Dublin Area*" July 1-3 Dublin City Council, RPS, Veolia

Department of the Environment, (1999) "*National Environmental Health Action Plan*" Department of the Environment Dublin

Department of the Environment (2000) "*National Climate Change Strategy* " Department of the Environment Dublin

Department of the Environment (2007) http://www.environ.ie/en/Environment/SustainableDevelopment/PublicationsDocuments/FileDownLoad,1847,en.pdf Website accessed 2nd February 2008

Dessai, S. (2002) "Heat stress and mortality in Lisbon Part I. model construction and validation", *International Journal of Biometeorology* 47 (1) 6-12.

Dessai, S (2003) "Heat stress and mortality in Lisbon Part II. An assessment of the potential impacts of climate change" *International Journal of Biometeorology* 48 (1) 37-44

Diaz, J., Garcia-Herrera, R., Trigo, R.M., Linares, C., Valente, M.A. ,De Miguel, J.M. and Hernandez, E .(2006) "The impact of the summer 2003 heat wave in Iberia: how should we measure it?", *International Journal of Biometeorology* 50 (3) 159-66.

Diffey, B. (2004) "Climate change, ozone depletion and the impact on ultraviolet exposure of human skin", *Physics in Medicine and Biology* 49 (1) R1-11

van Dolah, F.M. (2000) "Marine Algal Toxins: Origins, Health Effects, and Their Increased Occurrence", *Environmental Health Perspectives* 108 (1) 133-41

Donaldson, G.C. and Keatinge, W.R. (1997) "Early increases in ischaemic heart disease mortality dissociated from and later changes associated with respiratory mortality after cold weather in south east England", *Journal of Epidemiology and Community Health.* 51 (6) 643-8

Donaldson, G.C. and Keatinge, W.R. (2002) "Mortality related to cold weather in elderly people in southeast England 1979-94", *British Medical Journal* 315 1055-1056

Donaldson, G.C., Robinson, D. and Allaway, S.L.(1997) "An analysis of arterial disease mortality and BUPA health screening data in men in relation to outdoor temperature" *Clinical Science* 92 (3) 261-268

Donaldson, G.C., Seemungal, T., Jeffries, D.J. and Wedzicha, J.A. (1999) "Effect of temperature on lung function and symptoms in chronic obstructive pulmonary disease", *European Respiratory Journal* 13 (4) 844-9

Donaldson, G.C., Rintamaki, H. and Nayha, S. 2001 (a) "Outdoor clothing its relationship to geography, climate behaviour and cold related mortality in Europe", *International Journal of Biometeorology* 45 45-51

Donaldson, G., Kovats, R.S., Keatinge, W.R. and Mc Michael, A.J. (2001(b)) in "Heat and cold related mortality and climate change" *Health effects of climate change in the UK Health effects of climate change in the UK* Department of Health London 70-80 London

Donaldson, G.C. and Keatinge, W.R. (2002) "Excess winter mortality: influenza or cold stress? Observational study" *British Medical Journal* 324 89-90

Donaldson, G.C., Keatinge, W.R. and Nayha, S. (2003 (a)) "Changes in summer temperature and heat-related mortality since 1971 in North Carolina, South Finland, and Southeast England", *Environmental Research* 91(1) 1-7

Donaldson, G.C., Keatinge, W.R. and Saunders, R.D. (2003(b)) "Cardiovascular response to heat stress", *International Journal of hyperthermia* 19 (3) 225-235

Dresner, S. and Eakins, P. 2007 quoted by Scott S. in paper given at Water Framework Directive Implementation Conference 2nd May 2007 Dublin Water Department Dublin City Council

D'Souza, R.M., Becker, N.G., Hall, G. and Moodie, K.B. (2004) "Does ambient temperature affect foodborne disease?, *Epidemiology* 15 (1) 86-92

Dukes, J. (2003) "Burning buried sunshine: human consumption of ancient solar energy". *Climatic Change*, 61 (1-2) 31-44.

Duncan, S. (2003) http://www.hc-sc.gc.ca/ewh-semt/pubs/climat/consensus-conference-concertation/index_e.html Website accessed 29th June 06

Dunphy, P (2007) "*One million Irish homes poorly insulated*" says Renewable Energy Skills www.prlog.org http://www.prlog.org/10024486-one-million-irish-homes-poorly-insulated-says-renewable-energy-skills.html Website accessed 4th August 2007

Duthie, M.S., Kimber, I. and Norval, M. (1999) "The effects of ultraviolet radiation on the human immune system", *British Journal of Dermatology* 140 (6) 995-1009

Dwight, R.H., Semenza, J.C., Baker, D.B. and Olson, B.H. (2002) "Association of urban runoff with coastal water quality in Orange County California", *Water Environment Research* 74 (1) 82-90

Edwards, M. and Richardson, A.J. (2004) "Impact of climate change on marine pelagic phenology and trophic mismatch", *Nature* 430 881-884

EEA/WHO European Environment Agency/World Health Organization (2002) *Water and Health in Europe* World Health Organization Regional Publications No 93 Finland

EFSA European Food Safety Association (2006) "Analysis of the baseline study on the prevalence of salmonella in laying hen flocks of Gallus gallus", *European Food Safety Journal* 81 1-71 No author cited

Eisenreich, S. (2005) *Climate Change and the European Water Dimension* European Commission Directorate General Joint Research Centre Brussels

Elliot A.J. and Fleming D.M. (2006) "Surveillance of influenza-like illness in England and Wales during 1966-2006" *Eurosurveillance,* 11, (10)

Elwood, P.C., Beswick, A., O'Brien, J.R., Renaud, S., Fifield, R., Limb, E.S. and Bainton, D. (1993) "Temperature and risk factors for ischaemic heart dosease in the Caerphilly prospective study", *British Heart Journal* (70) 520-523

Emberlin, J. (1994) "The effects of patterns in climate and pollen abundance on allergy", *Allergy* 94 15-20.

Emberlin, J. (2007) "Climate change blamed for rise in asthma and hay fever misery" *The Sunday Times* May 13 2007

Eng, H. and Mercer, J. (1998) "Seasonal variation in mortality caused by cardiovascular diseases in Norway and Ireland", *Journal of Cardiovascular Risk* 5 (2) 89-95

Engelthaler, D.M., Mosley, D.G., Cheek, J.E., Levy, C.E., Komatsu, K.K., Ettestad, P., Davis, T., Tanda, D.T., Miller, L., Frampton, J.W., Porter, R. and Bryan, R.T. (1999) "Climatic and Environmental Patterns Associated with Hantavirus Pulmonary Syndrome, Four Corners Region, United States ", *Emerging Infectious Diseases* 5 (1) 87-94

Enquselassie, F., Dobson, A.J., Alexander, H.M. and Steele, P.L. (1993) "Seasons, Temperature and coronary disease", *International Journal of Epidemiology* 22 (4) 632-636

EPA Environmental Protection Agency (2001) *"Quality of Drinking water in Ireland A report for the year 2000 with a review of the period 1998-2000"* Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2004) *"Quality of Drinking water in Ireland 2003 with a review of the period 2001-200"3* Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2005) "*The quality of drinking water in Ireland A report for the year 2004"* Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2006 (a)) "*The quality of drinking water in Ireland A report for the year 2005"* Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2006 (b)) *Bathing water quality in Ireland report* 2004 Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2006 (c)) "*Water Quality in Ireland 2005 Key indicators of the Aquatic environment"* Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2006 (d)) "*Environment in Focus* 2006" Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2007 (a)) *"Urban waster water discharges in Ireland A report for the years 2002 and 2003"* Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2007 (b)) "*The Provision and Quality of Drinking Water in Ireland - A Report for the Years 2006-200"7* Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2007 (c)) *Bathing water quality in Ireland in 2006* Environmental Protection Agency, Johnstown Castle, Wexford Ireland

EPA Environmental Protection Agency (2008(a))
http://www.epa.ie/environment/air/quality/glo/_Website accessed 2nd March 2008

EPA, Environmental Protection Agency (2008 (b)) Personal communication 12-3-2008 (on file)

Epstein, P., Diaz, H., Elias, S., Grabherr, G., Graham , N.E., Martens, W., Mosley-Thompson, E. and Susskind, J . (1998) "Biological and physical signs of climate change: focus on mosquito-borne diseases", *Bulletin of the American Meteorological Society* 79 (3) 409-417

Eurostat (2006) website
http://epp.eurostat.ec.europa.eu/pls/portal/docs/PAGE/PGP_PRD_CAT_PREREL/PGE_CAT_PREREL_YEAR_2005/PGE_CAT_PREREL_YEAR_2005_MONTH_04/3-08042005-EN-AP.PDF Website accessed 11th January 2006

Eurowinter Group (1997) "Cold exposure and winter mortality from ischaemic heart disease, cerebrovascular disease, respiratory disease, and all causes in warm and cold regions of Europe", *The Lancet* 349 (9062) 1341-1346

Evans, M.R., Ribeiro, C.D., and Salmon, RL. (2003) "Hazards of healthy living: bottled water and salad vegetables as risk factors for Campylobacter infection", *Emerging Infectious Diseases* (10) 1219-25.

Ezzati, M., Lopez, A.D., Rodgers, A., Vander Hoorn, and S. Murray, C.J. Comparative Risk Assessment Collaborating Group (2002) "Selected major risk factors and global and regional burden of disease", *The Lancet,* 360 (9343) 1347-1360

Fahey, T., Morgan, D., Gunneburg, C., Adak, G.K., Majid, F. and Kaczmarski, E.(1995) "An outbreak of Campylobacter jejuni enteritis associated with failed milk pasteurisation, *Journal of Infection"* 31 (2) 137-43.

FAO Food and Agricultural Organization (2002) "*Reducing Poverty and Hunger: The critical role of financing for food, agriculture and rural development"* Paper Prepared for the International Conference on Financing for Development Monterrey, Mexico, 18-22 March 2002 Food and Agricultural Organization; International Fund for Agricultural Development; World Food Programme Rome

FAO Food and Agricultural Organization (2003) Food and Agriculture Organization Corporate Document Repository
www.fao.org/DOCREP/MEETING/006/Y9151e.HTM Website accessed 23rd December 2006

FAO Food and Agricultural Organization (2007) "*State of the World's Forests* 2007" Food and Agricultural Organization Rome 2007

Farber, J.M. (2004) "Enterobacter sakazakii—new foods for thought?", *The Lancet* 363 (9402) 5-6

Farrar, J.F. and Vaze, P.(2000) "*Wales changing climate challenging choices A Scoping study of climate change impacts in Wales"* National Assembly for Wales Bangor

Farrell, J.P. (1886) " The Great Cholera Epidemic" http://www.longfordancestry.com/longford-articles.php?ver=20&op=5 Website accessed 26th May 2007

Fealy, R "The impacts of Climate change on seal level and the Irish coast" in Sweeney, J., Brereton, T., Byrne, C.,Charlton, R., Emblow, C., Fealy, R., Holden, N., Jones, M., Donnelly, A.,Moore, S., Purser, P., Byrne, K., Farrell, E. and Mayes, E., (2003) *Climate change Scenarios and Impacts for Ireland* Environmental Protection Agency Ireland Johnstown Castle, Wexford Ireland

Fehlhaber, K,. and Krüger G. (1998) "The study of Salmonella enteritidis growth kinetics using Rapid Automated Bacterial Impedance Technique". *Applied Microbiology* 84(6) 945-9

Fewtrell, L. ,Kay, D., Dunlop, J., O'Neill, G.and Wyer, M. (1994). "Infectious diseases and water supply disconnections ", *The Lancet* 343 1370

Filler, S., MacArthur, J., Parise, M., Wirtz, R., Eliades, R., Dasilva, A. and Steketee,R. (2006) "Locally Acquired Mosquito-Transmitted Malaria: A Guide for Investigations in the United States", *Mortality and Morbidity Weekly Reports* 55 (RR13) 1-9

Finn J Personal communication 25-9-06

Fischer, P.H., Brunekreef, B. and Lebret, E. (2004) "Air pollution related deaths during the 2003 heat wave in the Netherlands" *Atmospheric Environment* 38, (8) 1083-1085

Fitzgerald, M., Bonner, C., Foley, B. and Wall, P. (2001) "Analysis of outbreaks of infectious intestinal disease in Ireland:1998 and 1999" *Irish Medical Journal* (94) 5 142-4

Fleming, J.R. (1998) *Historical Perspectives on climate change* New York and Oxford Oxford University Press
http://www.tyndall.ac.uk/general/history/JTyndall_biog_doc.pdf Website accessed 2nd April 2008

Fleury, M., Charron, D.F., Holt, J.D., Allen, O.B. and Maarouf, A.R. (2006) "A time series analysis of the relationship of ambient temperature and common bacterial enteric infections in two Canadian provinces", *International Journal of Biometeorology* 50 (6) 385-91

Foley, B. and Mc Keown, P. (2004) *Annual Report of Campylobacteriosis in Ireland* Health Protection Surveillance Centre Dublin

Foley, B. (2005) Personal communication E-mail on file

Ford, T. (1999) "Microbial safety of drinking water: United States and global perspectives", *Environmental Health Perspectives* 107 (1) 191-206

Forsythe, E. (2005) Department of the Environment, Personal Communication

Frost, J.A., Gillespie, I.A. and O'Brien, S.J. (2002) "Public health implications of campylobacter outbreaks in Engand and Wales, 1995-9: epidemiological and microbiological investigations", *Epidemiology and Infection* 128 111-118

FSAI Food Safety Authority of Ireland (1999) "The Prevention of *E.coli* O157:H7 infection: A shared responsibility" Food Safety Authority of Ireland Dublin

FSAI Food Safety Authority of Ireland (2002 (a)) "Control of Campylobacter Species in the Food chain" Food Safety Authority of Ireland Dublin

FSAI Food Safety Authority of Ireland 2002 (b) "A surveillance study of E. coli O157:H7 and Enterobacteriaceae in Irish retail minced beef and beef burgers" wwwfsai.ie http://www.fsai.ie/publications/reports/VTEC_survey_report.pdf Website accessed 2nd August 2006

FSAI (2004 (a)) "Report on Zoonoses in Ireland 2000 and 2001" Food Safety Authority of Ireland Dublin

FSAI (2004(b)) "EPA Report on water supplies in Ireland: Implications for food businesses" Food Safety Authority of Ireland Alert Notification: 2004.04 16 Feb 2004

FSAI (2006 (a)) "*War over water*" FSAI News (8) 2 Food Safety Authority of Ireland Dublin

FSAI (2006 (b)) "FSAI report indicates doubling of HACCP compliance at meat outlets following nationwide information campaign". Press release 31-07-2006 Food Safety Authority of Ireland Dublin

FSPB (2003) "Acute Gastroenteritis in Ireland North and South A Telephone Survey" Food Safety Promotions Board Dublin and Belfast

Gallay, A., Vaillant, V., Bouvet, P., Grimont, P. and Desenclos, J.C. (2000) "How many foodborne outbreaks of Salmonella infection occurred in France in 1995? Application of the capture-recapture method to three surveillance systems", *American Journal of Epidemology* 152 (2) 171-7

Garssen, J., Harmsen, C. , and de Beer, J. (2005) "The effect of the summer 2003 heat wave on mortality in the Netherlands", *Eurosurveillance* 10 (7-8) 165-168

Garvey, P. (2004) Personal Communication National Disease Surveillance Centre (now Health Protection Surveillance Centre) Dublin

Garvey, P. and Mc Keon, P. (2004) *Epidemiology of Cryptosporidiosis in Ireland* 2004 Health Protection Surveillance Centre Dublin

Garvey, P. (2006) Personal Communication e-mail on file

Garvey, P., Mc Keon, P., Carroll, A. and Mc Namara, P. (2006)" Epidemiology of Verocytogenic E, Coli 2005" *Epi-insight* 7 (9)

Gemmell, I., McLoone, P., Boddy, F.A., Dickinson, G.J. and Watt, G.C.(2000) "Seasonal variation in mortality in Scotland", *International Journal of Epidemiology.* 29 (2) 274-9

de Gentile, L., Picot, H., Bourdeau, P., Bardet, R,. Kerjan, A., Piriou, M., Le Guennic, A., Bayssade-Dufour, C., Chabasse, D. and Mott, K.E. (1996). "Cercarial dermatitis in Europe: a new public health problem?", *Bulletin of the World Health Organization.*74 (2) 159-63.

GEO 3 website http://www.unep.org/geo/geo3/english/018.htm Website accessed 21st September 2006

Gillespie, I.A., O'Brien, S.J., Adak, G.K., Tam, C.C., Frost, J.A., Bolton, F.J. and Tompkins, D.S. Campylobacter Sentinel Surveillance Scheme Collaborators (2003) "Point source outbreaks of Campylobacter jejuni infection--are they more common than we think and what might cause them?", *Epidemiology and Infection* 130 (3) 367-75.

Gillooly, M. and Green, M. (2003) "Cryptosporidium in a public water supply: The Westmeath experience", *GSI Groundwater newsletter* (42) 4-9

Githeko, A. and Woodward, A.(2003) "International consensus on the science of climate and health: the IPCC Third Assessment Report" in *Climate change: Risks and responses* Mc Michael, A.J., Campell-Lendrum, D.H., Corvalan, C., Ebi, K., Githeko, A., Scheraga, J.D. and Woodward, A. (Eds.) *Climate change Risks and Responses* GENEVA WHO WMO UNEP Geneva WHO 43-60

Gomez Angelats, E., Sierra, C., Coca, A, Pare, J.C. and de la Sierra, A. (2004) "Blood pressure variability and silent cerebral damage in essential hypertension", *American Journal of Hypertension* 17 (8) 696-700

Goodale C., Aber, A.D., and Ollinger S.V. (1998) "Mapping monthly precipitation, temperature and solar radiation for Ireland, with polynomial regression and a digital elevation model". *Climate Research 10 45-49*

Goodman, P.G., Dockery, D.W. and Clancy, L. (2004) "Cause-specific mortality and the extended effects of particulate pollution and temperature exposure", *Environmental Health Perspectives* 112 (2) 179-85.

Goodwin, J., Pearce, V.R. , Taylor, R.S., Read, K.L. and Powers, S.J. (2001) "Seasonal cold and circadian changes in blood pressure and physical activity in young and elderly people", *Age and Aging* 30 (4) 311-7.

Gorman, R. and Adley, C.C. (2004) "Characterization of Salmonella enterica serotype Typhimurium isolates from human, food, and animal sources in the Republic of Ireland", *Journal of Clinical Microbiology* 42 (5) 2314-6.

Gould, E.A., Higgs, S., Buckley, A. and Gritsun, T.S. (2006) "Potential arbovirus emergence and implications for the United Kingdom, "*Emerging Infectious Diseases* 12 (4) 549-55

Graczyk, T.K., Conn, D.B., Lucy, F,. Minchin, D., Tamang, L., Moura, L.N. and Da Silva, A.J. (2004) "Human waterborne parasites in zebra mussels (*Dreissena polymorpha*) from the Shannon River drainage area, Ireland", *Parasitology Research* 93 (5) 385-91.

Gratz, N.G., Steffen, R. and Cocksedge, W. (2000) "Why aircraft disinfection", *Bulletin of the World Health Organization* 78 (8) 995-1004

Gray, J.S., Kahl, O., Janetzki, C., Stein, J. and Guy, E.(1994) "The spatial distribution of Borrelia borgdorferi infected ixodes ricinus in the Connemara region of County Galway Ireland", *Experimental and Applied Acarology* 18 (80) 485-91

Gray, J.S., Kirstein, F., Kahl, O. and Robertson, J.N. (1999) "Lyme disease in Ireland", wwwucd.ie/agri/html/homepage/research_96_99/research_1998_99/erm/erm05.html Website accessed 12th January 2004

Gray J.S., Robertson J.N. and Key S. (2000) "Limited role of rodents as reservoirs of Borrelia burgdorferi sensu lato in Ireland", *European Journal of Epidemiology* 16 (2) 101-3

Grize, L., Huss, A., Thommen, O., Schindler, C. and Braun-Fahrlander, C. (2005) "Heat wave 2003 and mortality in Switzerland", *Swiss Medical Weekly* 135 (13-14) 200-5.

Gryparis, A., Forsberg, B., Katsouyanni, K., Analitis, A., Touloumi, G., Schwartz, J., Samoli, E., Medina, S., Anderson, H.R., Niciu, E.M., Wichmann, H.E., Kriz, B., Kosnik, M., Skorkovsky J., Vonk, J.M. and Dörtbudak, Z. (2004) "Acute effects of ozone on mortality from the 'air pollution and health: a European approach' project. *American Journal of Respiratory and Critical Care Medicine* 170 (10) 1080-7

Gubler D. J., Reiter P., Ebi K.L., Yap, W. and Patz J.A. (2001)"Climate variability and change in the United states: Potential impacts on vector and rodent borne diseases, *Environmental Health Perspectives* 109 (2) 223-233

Guest, C.S., Wilson, K., Woodward, A.J., Hennessy, K., Kalkstein, L.S., Skinner, C. and Mc Michael, A.J. (1999) "Climate and mortality in Australia: retrospective study, 1970-1000, and predicted impacts in five major cities in 2030", *Climate Research* 13 1-15

Haile, R.W., Witte, J.S., Gold, M., Cressey, R., McGee, C., Millikan, R.C., Glasser, A., Harawa, N., Ervin, C., Harmon, P., Harper, J., Dermand, J., Alamillo, J., Barrett, K., Nides, M, and Wang, G. (1999) "The health effects of swimming in ocean water contaminated by storm drain runoff" *Epidemiology* 10 (4) 355-63

Haines, A., Epstein, P.R. and Mc Michael, A.J. (1993) "Global health watch: monitoring health impacts of environmental change" *The Lancet* 342 1464-1469

Haines, A, Kovats, R.S., Campbell-Lendrum. D. and Corvalan. C. (2006) Climate change and human health: impacts, vulnerability, and mitigation. *The Lancet* 367 (9528) 2101-9.

Hajat, S, Haines, A. (2002) "Associations of cold temperatures with GP consultations for respiratory and cardiovascular disease amongst the elderly in London", *International Journal of Epidemiology* 31 (4) 825-30.

Hajat, S., Kovats, R.S., Atkinson, R.W. and Haines, A.(2002) "Impact of hot temperatures on death in London: a time series approach", *Journal of Epidemiology and Community Health* 56 367-372

Hajat, S., Ebi, K., Kovats, S., Menne, B., Edwards, S. and Haines, A. (2005) The Human health consequences of flooding in Europe: a review in (Eds.) Kirch, W., Menne, B. and Bertollini, R. *Extreme Weather Events and Public health Responses* New York Springer 185-96

Hajat, S., Hajat, S., Kovats, R.S. and Lachowycz, K.(2007) Heat-related and cold-related deaths in England and Wales: who is at risk? *Occupational and Enviromental Medicine* . 64 (2) 93-100

Hales, S., Salmond, C., Town, G.I., Kjellstrom T. and Woodward, A. (2000) "Daily mortality in relation to weather and air pollution in Christchurch, New Zealand" *Australian and New Zealand Journal of Public Health.* 24 (1) 89-91.

Hall, G.V., D'Souza, R.M., and Kirk, M.D. (2002) "Foodborne disease in the new millennium:out of the frying pan into the fire?" *Medical Journal of Australia* 177 (11/12) 614-618

Han, J., Colditz, G.A. and Hunter, D.J. (2006) "Risk factors for skin cancers: a nested case-control study within the Nurses' Health Study",.*International Journal of Epidemiology* 35 (6) 1514-21.

Harrison, S. and Kinra, S. (2004) "Outbreak of Escherichia coli *O157* associated with a busy bathing beach" *Communicable Disease and Public Health* 7 (1) 47-50

Hassan, H., Hanaki, K. and Matsuo, T. (1998) "A modelling approach to simulate impact of climate change in lake water quality: phytoplankton growth rate assessment", *Water Science and Technology* 37 (2) 177-185

Hassi J. and Rytkonen, M. (2005) "Climate warming and health adaptation in Finland" Finadapt Working Paper 7 Finnish Environment Institute Mimeograph 337 Helsinki

Hay, S., Cox, J., Rogers, D., Randolph, S., Stern, D., Shanks, G., Myers, M.and Snow R. (2002) "Climate change and the resurgence of malaria in the East African highlands", *Nature* 415 (6874) 905,

Healy, J. (2003) "Excess winter mortality in Europe: a cross country analysis identifying key risk factors", *Journal of Epidemiology and Community Health* 57 (10) 784-9

Healy, J. (2005) "Energy efficient homes could end fuel poverty", *Action on Poverty Today* Combat Poverty Agency Dublin 11 6-7

Helms, M., Ethelberg, S., Vastrup, P. and Mølbak, K. (2005) "International Salmonella Typhimurium DT104 infections, 1992-2001", *Emerging Infectious Diseases* 11 (6) 859-67.

Hippocrates (a) "On Airs, Waters, and Places" http://classics.mit.edu/Hippocrates/airwatpl.mb.txt Website accessed 3rd October 2006

Hippopcrates (b) in Met Office httpwww.metoffice.com/health/index.html Website accessed 24th June 2006

Hjelle, B. and Glass, G.E. (2000) "Outbreak of Hantavirus Infection in the Four Corners Region of the United States in the Wake of the 1997–1998 El Niño–Southern Oscillation", *The Journal of Infectious Diseases*, 181 1569–1573

Houghton, J. (1997) "*Global Warming The Complete Briefing*" Cambridge University Press Cambridge

Hoxie, N.J., Davis, J.P., Vergeront, J.M., Nashold, R.D. and Blair, K.A. (1997) "Cryptosporidiosis-associated mortality following a massive waterborne outbreak in Milwaukee, Wisconsin", *American Journal of Public Health* 12 2032-5

HPSC (2004) *Report of the Waterborne Cryptosporidiosis Committee* Health Protection Surveillance Centre, Dublin

HPSC (2005(a)) *Annual Report 2004* Health Protection Surveillance Centre, Dublin

HPSC (2005(b)) "Report of the HPSC Sub-Committee on Verotoxigenic E. Coli 0157" Health Protection Surveillance Centre, Dublin

HPSC (2005 (c)) Epi-insight 6 (1) 4 Health Protection Surveillance Centre, Dublin

HPSC (2006 (a)) http://www.ndsc.ie/hpsc/A-Z/Vectorborne/WestNileVirus/Factsheet/MainBody,602,en.htmlWebsite Health Protection Surveillance Centre, Dublin Website accessed 30th August 2006

HPSC (2006 (b)) *Annual Report 2005* Health Protection Surveillance Centre, Dublin

HPSC (2007 (a)) http://www.ndsc.ie/hpsc/News/Title,2280,en.html Health Protection Surveillance Centre, Dublin Website accessed 8th May 2007

HPSC (2007 (b)) *Epidemiology of Legionnaires disease in Ireland* http www.ndsc.ie/hpsc/A-Z/Respiratory/Legionellosis/Epidemiology/File,2079,en.pdf Health Protection Surveillance Centre, Dublin Website accessed 3rd June 2007

HPSC (2007 (c)) *Annual Report 2006* Health Protection Surveillance Centre, Dublin

HPSC (2008) http
www.ndsc.ie/hpsc/NotifiableDiseases/WeeklyOutbreakReport/File,2186,en.pdf Health Protection Surveillance Centre, Dublin

Hulme, M. and T.R. Carter (2000), "2000: The changing climate of Europe" in *Assessment of Potential Effects and Adaptations for Climate Change in Europe: The Europe ACACIA Project* Parry, M.L (Ed). Jackson Environment Institute, University of East Anglia, Norwich, United Kingdom,

Hunter, P.R. (2003) "Climate change and waterborne and vector-borne disease", *Journal of Applied Microbiology* 94 37S-46S

Huynen, M.M., Martens, P., Schram, D., Weijenberg, M.P. and Kunst A.E. (2001) "The impact of heat waves and cold spells on mortality rates in the Dutch population" *Environmental Health Perspectives* 109 (5) 463-70.

IPCC Intergovernmental Panel on Climate Change (1990) "First Assessment Overview and Policymaker Summaries and 1992 IPCC Supplement", Intergovernmental Panel on Climate Change, Geneva, Switzerland.

IPCC Intergovernmental Panel on Climate Change (1995) "Contribution of Working Group II to the Second Assessment of the Intergovernmental Panel on Climate Change" Watson, R.T., Zinyowera, M.C., Moss, R.H. (Eds.) Cambridge University Press

IPCC Intergovernmental Panel on Climate Change (2001) *Climate Change 2001: Impacts, Adaptation and Vulnerability* McCarthy J, Canziani O.F, Neil A. Leary N.A, Dokken D.J, White K.S, (Eds.) Intergovernmental Panel on Climate Change Cambridge University Press, Cambridge

Ishigami, A., Hajat S., Kovats, R.S., Bisanti, L., Rognoni, M., Russo, A. and Paldy, A. (2008)"An ecological time-series study of heat-related mortality in three European cities" *Environmental Health* (7) 5

Jehn, M., Appel, L.J., Sacks, F.M. and Miller E.R. 3rd; DASH Collaborative Research Group. (2002) "The effect of ambient temperature and barometric pressure on ambulatory blood pressure variability". *American Journal of Hypertension,* 15 (11) 941-5

Jackson, M.C. (1983) "The hottest July for over 300 years" *Weather* 38 (9) 295

Jennings, P. and Rhatigan, A. (2002) "Cryptosporidiosis outbreak in Ireland liked to public water supply", *Eurosurveillance* 6 (22)

Johnson, H., Kovats, R.S., McGregor, G., Stedman, J., Gibbs, M., Walton, H., Cook, L., and Black, E. (2005(a)) "The impact of the 2003 heat wave on mortality and admissions in England", *Health Statistics Quarterly* 25 6-11

Johnson, H., Kovats, R.S., McGregor, G., Stedman, J., Gibbs, M. and Walton, H. (2005 (b))"The impact of the 2003 heat wave on daily mortality in England and Wales and the use of rapid weekly mortality estimates", *Eurosurveillance* 10 (7) 168-171

de Jong, J.T., ten Brinke, J.M., van Ouwerkerk, I.M., Siebbeles, M.F., Fitz-James, I.A., and in 't Veld, P.H. (2004)"Large-scale, acute, bacterial gastroenteritis caused by the enterotoxin of Staphylococcus aureus after a barbecue" *Nederlands Tijdschrift voor Geneeskunde* 148 (43) 2136-40

Kalkstein, L.S. and Davis, R.E (1989) "Weather and human mortality", *Annals of the Association of American Geographers* 79 44-64

Kalkstein, L. (1993) "Direct impacts in cities" *The Lancet* 34 (8884) 1397-9.

Kalkstein, L. and Smoyer, K. (1993) "The impact of global climate change on health", *Experentia* , 49 1-11

Kaiser, R. (1995) "Tick-borne encephalitis in southern Germany" *The Lancet* 345 8947

Kampen, H., Rotzel, D.C., Kurtenbach, K., Maier, W.A. and Seitz, H.M.(2004) "Substantial rise in the prevalence of Lyme borreliosis spirochetes in a region of western Germany over a 10-year period" *Applied Environmental Microbiology* 70 (3) 1576-82.

Kandhai, M.C., Reij, M.W., Grognou, C., van Schothorst, M., Gorris, L.G. and Zwietering, M.H. (2004) "Occurrence of *Enterobacter sakazakii* in food production environments and households" *The Lancet* 363 (9402) 39-40

Kay, D., Fleisher, J.M., Salmon, R.L., Jones, F., Wyer, M.D., Godfree, A.F., Zelenauch-Jacquotte Z. and Shore, R. (1994) "Predicting likelihood of gastroenteritis from sea bathing: results from randomised exposure", *The Lancet* 344 905–909.

Keating (2005) Statistician Central Statistics Office Dublin Personal Communication 5-11-05

Keatinge, W.R., Coleshaw, S.R., Cotter, F., Mattock, M., Murphy, M. and Chelliah, R. (1984) "Increases in platelet and red cell counts, blood viscosity, and arterial pressure during mild surface cooling: factors in mortality from coronary and cerebral thrombosis in winter" *British Medical Journal* (Clinical Research Edition) 289 (6456) 1405-8.

Keatinge, W.R., Coleshaw, S.R., Easton, J.C., Cotter, F., Mattock, M.B. and Chelliah, R. (1986) "Increased platelet and red cell counts, blood viscosity, and plasma cholesterol levels during heat stress, and mortality from coronary and cerebral thrombosis", *American Journal of Medicine* 81 (5) 795-800.

Keatinge, W.R. and Donaldson, G.C. (1995) "Cardiovascular mortality in winter", *Arctic Medical Research* 54 (2)16-8.

Keatinge, W.R., Donaldson, G.C., Cordioli, E., Martinelli, M., Kunst, A.E., Mackenbach, J.P., Nayha, S. and Vuori I. (2000) "Heat related mortality in warm and cold regions of Europe: observational study", *British Medical Journal* 321 670-673

Kefford, R. (2006) "Medical heat for climate change", *Medical Journal of Australia* 184 (11) 582

Kessel, A.S., Gillespie, I.A., O'Brien, S.J., Adak ,G.K., Humphrey, T.J. and Ward, L.R.(2001) "General outbreaks of infectious intestinal disease linked with poultry, England and Wales, 1992-1999", *Communicable disease and Public Health* 4 (3) 171-7

Kirstein, F., Rijpkema, S., Molkenboer, M. and Gray, J.S. (1997) "The distribution and prevalence of B. burgdorferi genomospecies in Ixodes ricinus ticks in Ireland" *European Journal of Epidemiology*13 (1) 67-72

Kistemann, T., Classen, T., Koch, C., Dangendorf, F., Fischeder, R., Gebel, J., Vacata, V. and Exner, M. (2002) "Microbial load of drinking water reservoir tributaries during extreme rainfall and runoff" *Applied and Environmental Microbiology* 68 (5) 2188-97

Klinenberg, E. (2002) "*Heat Wave: A Social Autopsy of Disaster in Chicago*" University of Chicago Press, Chicago ISBN 0-226-44321-3.

Kosatsky, T. (2005)"The 2003 European heat waves" *Eurosurveillance* 10 (7-8)

Kovats, R.S., Haines, A., Stanwell-Smith, R., Martens P., Menne, B. and Bertollini, R. (1999) "Climate change and human health in Europe", *British Medical Journal* 318 (7199) 1682-5.

Kovats, R.S., Campbell-Lendrum, D.H., McMichael, A.J., Woodward A. and Cox J.S. (2001) "Early effects of climate change: do they include changes in vector-borne disease?", Philosophical *Transactions of the Royal Society of London Series B Biological Sciences* 356 (1411) 1057-68.

Kovats, R. and Bouma, M. (2002) "Retrospective studies:analogue approaches to describing climate variability and health", in *Environmental Change, Climate and Health Issues and Research Methods* Martens, P. and McMichael A.J.(Eds.) ,Cambridge University Press 144-171

Kovats, S., Ebi, K. and Menne, B. (2003 (a)) "*Methods of assessing human health vulnerability and public health adaptation to climate change*" Series No. 1 WHO UNEP Health Canada

Kovats, R.S., Menne, B., Ahern, M.J. and Patz, J.A.,(2003 (b)) "National Assessments of health impacts of climate change: a review" in *Climate change: Risks and responses* Mc Michael AJ, Campell-Lendrum DH, Corvalan C, Ebi K, Githeko A, Scheraga, JD, Woodward A, (Eds.) GENEVA WHO WMO UNEP Geneva WHO 181-203

Kovats R.S., Hajat, S. and Wilkinson, P. (2004 (a)) "Contrasting patterns of mortality and hospital admissions during hot weather and heat waves in Greater London, UK", *Occupational and Environmental Medicine* 61 (11) 893-8

Kovats, R.S., Edwards, S.J., Hajat, S., Armstrong, B.G., Ebi, K.L. and Menne, B. (2004 (b)) "The effect of temperature on food poisoning: a time-series analysis of salmonellosis in ten European countries", *Epidemiology and Infection* 132 (3) 443-53

Kovats, R.S., Edwards, S.J., Charron, D., Cowden, J., D'Souza, R.M., Ebi, K.L., Gauci, C., Gerner-Smidt, P., Heat, S., Hales, S., Hernandez Pezzi, G., Kriz, B., Kutsa, K., McKeown, P., Mellou, K., Menne, B., O'Brien, S., van Pelt, W. and Schmid, H. (2005) "Climate variability and campylobacter infection: an international study", *International Journal of Biometeorology* 49 (4) 207-14.

Kovats, R.S., and Ebi, K.L. (2006) "Heatwaves and public health in Europe" *European Journal of Public Health.* Apr 27 E-published ahead of printing http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=16644927&query_hl=17&itool=pubmed_docsum Website accessed 3rd October 2006

Kuhn, K.G., Campbell-Lendrum, D.H., Armstrong, B. and Davies, C.R. (2003) "Malaria in Britain: Past, present and future", *Proceedings of the National Academy of Sciences* 100 (17) 9997-10001

Kunst, A.E., Looman, C.W. and Mackenbach, J.P. (1991) "The decline in winter excess mortality in The Netherlands", *International Journal of Epidemiology* 20 (4) 971-7

Kunst, A.E., Looman, C.W. and Mackenbach, J.P. (1993) "Outdoor air temperature and mortality in The Netherlands: a time-series analysis", *American Journal of Epidemiology* 137 (3) 331-41

Lake, I.R., Bentham, G., Kovats, R.S. and Nichols, G.L. (2005) "Effects of weather and river flow on cryptosporidiosis", *Journal of Water and Health* 3 (4) 469-74

Langford, I.H. and Bentham, G. (1995)"The potential effects of climate change on winter mortality in England and Wales", *International Journal of Biometeorology* 38 (3) 141-7

Layton, M. Parise, M.E., Campbell, C.C., Advani, R., Sexton, J.D., Bosler, E.M. and Zucker, J.R.(1995) "Mosquito transmitted malaria in New York City 1993", *The Lancet* 346 729-231

Leder, K., Sinclair, M.I. and McNeil, J.J. (2002) "Water and the environment: a natural resource or a limited luxury?", Medical *Journal of Australia* 177 (11/12) 609-613

Ledrans, M., Pirard, P., Tillaut, H., Pascal, M., Vandentorren, S., Suzan, F., Salines, G., Le Tertre, A., Medina, S., Maulpoix, A., Berat, B., Carre, N., Ermanel, C., Isnard, H. Ravault, C. and Delmas, M.C.(2004) "The heat wave of August 2003: what happened?", *La Revue du Praticien* 54 (12) 1289-97

Lemmen, D.S. and Warren, F.J. (2004) (Eds.) "*Climate Change Impacts and Adaptation: A Canadian Perspective*" Climate Change Impacts and Adaptation Directorate, Natural Resources Canada Ottawa, Ontario

Leonard, N. (2004) "Epidemiology and control of salmonellosis in pigs from a food safety perspective" UCD Faculty of Veterinary Medicine Website accessed 23rd February 2004

Lerchyl, A. (1998) "Changes in the seasonality of mortality in Germany from 1946 to 1995: the role of temperature", *International Journal of Biometeorology* 42 (2) 84-8

Le Tertre, A., Lefranc, A., Eilstein, D., Declercq, C., Medina, S., Blanchard, M., Chardon, B., Fabre, P., Filleul, L., Jusot, J.F., Pascal, L., Prouvost, H., Cassadou, S., and Ledrans, M.(2006) "Impact of the 2003 heatwave on all-cause mortality in 9 French cities".*Epidemiology.* (1) 75-9.

Levy, J.I. Chemerynski, S.M. and Sarnat, J.A. (2005) "Ozone exposure and mortality: an empiric bayes metaregression analysis", *Epidemiology.* 16 (4) 458-68.

Lindgren, E. (1998) "Climate and tick borne encephalitis", *Conservation Ecology* 2 (1) 5

Lindgren, E., Tälleklin, L. and Polfeldt, T., (2000) "Impact of climate change on the Northgern latitude limit and population density of the disease-transmitting European tick ixodes ricinus", *Environmental Health Perspectives* 108. (2) 119-23

Lindgren, E. and Gustafson, R (2001) "Tick-borne encephalitis in Sweden and climate change", *The Lancet* 358 16-18

Lindsay, S.W. and Birley, M.H. (1996) "Climate change and malaria transmission", *,Annals of Tropical medicine and parasitology* 90 (6) 573-588

Lopez-Velez, R. and Molina Moreno, R. (2005) "Climate change in Spain and risk of infectious and parasitic diseases transmitted by arthropods and rodents", *Revista Española de Salud Pública,* 79 (2) 177-190

Lopman, B.A., Reacher, M.H., Van Duijnhoven, Y., Hanon, F.X., Brown, D. and Koopmans M. "Viral gastroenteritis outbreaks in Europe, 1995-2000", *Emerging Infectious Diseases* 9 (1) 90-6.

Louis V.R., Gillespie I.A. O'Brien, S.J., Russek-Cohen E., Pearson, A.D., and Colwell R.R. (2005) "Temperature-Driven Campylobacter Seasonality in England and Wales", *Applied and Environmental Microbiology* 71 (1) 85–92

Lucey, J. (2005) *"Key Indicators of the Aquatic Environment"* Presentation at Conference "Working together on Water Protection" Galway Environmental Protection Agency

Mackenbach , J.P., Looman ,C.W. and Kunst, A.E. (1993) "Air pollution, lagged effect of temperature and mortality: the Netherlands 1979-87", *Journal of Epidemiology and Community Health* 47 (2) 121-6

Manning, P. (2006) "Asthma and the art of planning" *Irish Medical News* Sept 4

Martens, P. (1998) in "Health and Climate change" in *Modelling the impacts of global warming and ozone depletion* Martens P. (ed.) Earthscan London

Marshall Cummins, N. (1957) *Some chapters of Cork Medical History* Cork University Press

McCarty, C.A.and Taylor, H.R. (2002) "A review of the epidemiologic evidence linking ultraviolet radiation and cataracts", *Developments in Ophthalmology* 35 21-31

Mc Elwain, L. and Sweeney, J. (2007) "*Key Meteorological Indicators of Climate Change in Ireland"* Environmental Research Centre - ERC Report 6 Environmental Protection Agency

Mc Geehin, M.A. and Mirabelli, M. (2001) "The potential impacts of climate variability and change on temperature-related morbidity and mortality in the United States", *Environmental Health Perspectives* 109 (2) 185-189

Mc Greevy R.(2008) "Spa pool workers offered training" The Irish Times 22 April

Mc Kee, C.M. (1989) "Can Britain learn from Europe?", *European Journal of Epidemiology* 5 (2) 178-82

Mc Kee, C.M. (1990) "Deaths in winter in Northern Ireland: the role of low temperature", *Ulster Medical Journal.* 59 (1)17-22.

Mac Kenzie, W.R., Hoxie, N.J., Proctor, M.E., Gradus, M.S., Blair, K.A., Peterson, D.E., Kazmierczak, J.J., Addiss, D.G., Fox, K.R., Rose, J.B. and Davis, J.P. (1994) "A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply", *New England Journal of Medicine* 331 (3)161-7.

Mc Keown, D (2007). (Personal communication) Department of Public Health, Health Services Executive, Galway

Mc Michael, A.J. and Haines, A. (1997) "A Global climate change: the potential effects on health", *British Medical Journal* 315 805-809

Mc Michael, A.J. and Kovats, R.S. (2000) "Strategies for Assessing the Health Impacts of Global Environmental Change", in (Eds.) Crabbe, P., Holland, A., Ryszkowski, L. and Westra, L. in *Implementing Ecological Integrity* NATO Science series Kluwer Academic Publishers Dordrecht Netherlands

Mc Michael, A.J., Haines A., and Kovats, S. (2001) "Methods to assess the impact of climate change on health", in *Health effects of climate change in the UK* UK Department of Health p.55-69

Mc Michael, A.J., Woodruff, R., Whetton, P., Hennessy, K., Nicholls ,N., Hales, S., Woodward, A. and Kjellstrom, T. (2002) "*Human Health and Climate Change in Oceania: A Risk Assessment"* Australian government Department of Health and Ageing Canberra

Mead, P.S., Slutsker, L., Dietz, V., McCaig, L.F., Bresee, J.S., Shapiro, C. Griffin, P.M. and Tauxe, R.V. (1999) "Food-related illness and death in the United States", *Emerging Infectious Diseases* 5 (5) 607-25.

Medina-Ramón, M. and Schwartz, J. (2007) "Temperature, Temperature Extremes, and Mortality: A Study of Acclimatization and Effect Modification in 50 United States Cities", *Occupational and Environmental Medicine* 0 1-7

Medlock, J.M., Snow, K.R. and Leach, S. (2005)" Potential transmission of West Nile virus in the British Isles: an ecological review of candidate mosquito bridge vectors", *Medical and Veterinary Entomology* 19 (1) 2-21

Meldrum, R.J., Tucker, D. and Edwards, C. (2004(a)) "Baseline rates of Campylobacter and Salmonella in raw chicken in Wales, United Kingdom, in 2002", *Journal of Food Protection* 67 (6) 1226-8

Meldrum, R.J. (2004(b)) "Seasonality of human campylobacter infection and campylobacter isolates from retail chicken", National Public Health service for Wales http://www2.nphs.wales.nhs.uk/icds/docmetadata.cfm?id=122 Website accessed 31st December 2004

Mercer, J.B. (2003) "Cold--an underrated risk factor for health", *Environmental Research* 92 (1) 8-13

Met Eireann 2007 http://www.met.ie/climate/monthly-summary.asp Website accessed 8th August 2007

Met Eireann, 2008 http://www.met.ie/climate/temperature.asp?LW=Kilkenny Website accessed 14th February 2008

Meusel, D. and Kirch, W. (2005), "Lessons to be learned from the 2002 floods in Dresden Germany", in Kirch, W., Menne, B. and Bertollini, R. (Eds.,) *Extreme Weather Events and Public health Responses* New York Springer 175-183

Michelozzi, P., de' Donato, F., Accetta, G., Forastiere, F., D'Ovidio, M., Perucci, C. and Kalkstein, L.(2004) "Impact of Heat Waves on Mortality, Rome, Italy, June-August 2003", *Mortality and Morbidity Weekly Reports* 53(17) 369-371

Middleton, J.D., Saunders, P. and Corson, J. (1994) "Water disconnection and disease", *The Lancet* 344 (8914) 62

Miettinen, I.T., Zacheus, O., von Bonsdorff, C.H. and Vartiainen, T.(2000) "Waterborne epidemics in Finland in 1998-1999", *Water Science and Technology* 43 (12) 67-71

Miller, G., Dunn, G.M., Smith-Palmer, A., Ogden, I.D. and Strachan, N.J. (2004) "Human campylobacteriosis in Scotland: seasonality, regional trends and bursts of infection", *Epidemiology and Infection* 132 (4) 585-93.

MMWR 1999 "Outbreak of Escherichia Coli 0157 and Campylobacter among attendees of the Washington County Fair", *Mortality and Morbidity Weekly Reports* 48 (36) 803

Moe, C.L. (2004) "What are the criteria for determining whether a disease is zoonotic and water related?", in Cotruvo, A., Dufour, A., Rees, G., Bartram, J., Carr, R., Cliver, D.O., Craun, G.F., Fayer, R. and Gannon V.P.J (Eds.). *Identification, causes and control of Waterborne disease* 27-45 World Health Organization IWA Publishing

Moran, C., Johnson, H. and Johnson, Z. (2000) "Seasonal patterns of morbidity and mortality in the elderly in Ireland", *International Journal of Circumpolar Health* 59 (3-4) 170-5

Morris, R.D., Naumova, E.N., Levin, R. and Munasinghe, R.L. (1996) "Temporal variation in drinking water turbidity and diagnosed gastroenteritis in Milwaukee", *American Journal of Public Health* 86 (2) 237-9

Morris, J.G. (1999) "Harmful Algal Blooms: An emerging public health problem with possible links to human stress on the environment", *Annual Review of Energy and the Environment* 24 367-90

Mourino-Perez, R.R. (1999) "Studying the risks of ocean swimming", *Epidemiology* 10 (4) 355-63

Murphy G. (2002)"Photoprotection: public campaigns in Ireland and the U.K", *British Journal of Dermatology* 146 (61) 31-33

Murphy, M., Carroll, A., Whyte, P., O'Mahony, M., Anderson, W., McNamara, E. and Fanning, S. (2005) "Prevalence and characterization of Escherichia coli O26 and O111 in retail minced beef in Ireland", *Foodborne Pathogens and Disease* 2 (4) 357-60

Murray, S. (2000) Dublin City Council National Hydrology Seminar 2000 http://www.opw.ie/hydrology/data/speeches/National%20Hydrology%20Seminar%202000%20No%203%20-%20URBAN%20RIVER%20MANAGEMENT.pdf

Nafstad, P., Skrondal, A. and Bjertness, E. (2001) "Mortality and temperature in Oslo, Norway, 1990-1995", *European Journal of Epidemiology* 17 (7) 621-7

Nairn R. (2006) "Ireland's Coastline - Exploring its Nature and Heritage" Collins Press Dublin

NASA National Aeronautics and Space Administration (2003) "*Nasa observations that confirm expected ozone layer recovery* " NASA News release Langley Research Centre , Hampton, Virginia July 29 2003

Nash, D., Mostashari, F, Fine A., Miller,J., O'Leary D., Murray, K., Huang A., Rosenberg, A., Greenberg, A., Sherman, M., Wong, S., and Layton M. West Nile Outbreak Response Working Group (2001) "The outbreak of West Nile virus infection in the New York City area in 1999", *New England Journal of Medicine* 344 1807-14.

Nash, R. (2006) National Museums Northern Ireland Personal Communication 3rd October

National Statistics Online (2005) http://www.statistics.gov.uk/ Website accessed 4th November 2005

National Cancer Registry (2005) www.ncri.ie Website accessed 21st September 2006

National Cancer Registry (2006 (a)) Trends in Irish Cancer incidence 1994-2002 with predictions to 2020 http://www.ncri.ie/pubs/pubfiles/proj_2020.pdf Website accessed 4 Jan 2007

National Cancer Registry (2006 (b)) Personal communication 27th September 2006

National Council on Aging and Older People (2001) http://www.ncaop.ie/publications/research/factfiles/FF14Accidents.pdf Website accessed 4th January 2007

Naumova, E.N., Egorov, A.I., Morris, R.D. and Griffiths, J.K. (2003) "The elderly and waterborne Cryptosporidium infection: gastroenteritis hospitalizations before and during the 1993 Milwaukee outbreak", *Emerging Infectious Diseases* 9 (4) 418-25

NDSC National Disease Surveillance Centre (2003) "*National Guidelines on the management of outbreaks of Norovirus in the health care setting*" National Disease Surveillance Centre (now the Health Protection Surveillance Centre) Dublin

NDSC National Disease Surveillance Centre (2004) *Annual Report 2003* National Disease Surveillance Centre Dublin (now the Health Protection Surveillance Centre) Dublin

Neimann, J., Engberg, J., Molbak, K. and Wegener, H.C. (2003) "A case-control study of risk factors for sporadic campylobacter infections in Denmark", *Epidemiology and Infection* 130 (3) 353-66

Nichols, G. and Kovats, S. (2008) "Health effects of climate change in the UK 2008: an update of the Department of Health report 2001/2002" Department of Health and Health Protection Agency London 76-79

Noble, R.T., Weisberg, S.B., Leecaster, M,K., McGee, C.D., Dorsey, J.H., Vanik, P. and Orozco-Borbon, V. (2003). "Storm effects on regional beach water quality along the Southern California coastline", *Journal of Water and Health* 1 (2) 23-31

Nylen, G., Dunstan, F., Palmer, S.R., Andersson, Y., Bager, F., Cowden, J., Feierl, G., Galloway, Y., Kapperud, G., Megraud, F., Molbak, K., Petersen, L.R. and Ruutu, P. (2002)"The seasonal distribution of campylobacter infection in nine European countries and New Zealand", *Epidemiology and Infection* 128 (3) 383-90

O'Brien, S.B., Duffy, G., Carney, E., Sheridan, J.J., McDowell, D.A. and Blair, I.S. (2005) "Prevalence and numbers of Escherichia coli O157 on bovine hides at a beef slaughter plant", *Journal of Food Protection* 68 (4) 660-5.

Oceana (2008) http www.oceana.org/climate/cause/greenhouse-gases/ Website accessed 30th January 2008

Office of Public Works, (2004) "Report of the Flood Policy Review Group" Office of Public Works, Dublin

Ogden, I.D., MacRae, M. and Strachan, N.J. (2004) "Is the prevalence and shedding concentrations of E. coli O157 in beef cattle in Scotland seasonal?", *FEMS Microbiology Letters* 233 (2) 297-300

Ogden, N.H., Maarouf, A., Barker, I.K., Bigras-Poulin, M., Lindsay, L.R., Morshed, M.G., O'Callaghan, C.J., Ramay, F., Waltner-Toews, D. and Charron, D.F. (2006) "Climate change and the potential for range expansion of the Lyme disease vector Ixodes scapularis in Canada", *International Journal of Parasitology* 36 (1) 63-70.

O'Meara, M. and Fitzgerald, M. (2004) "Leptospirosis in canoeists on a Dublin river" *Eurosurveillance* 8 (48)

O'Neill, M.S., Hajatt, S., Zanobetti, A., Ramirez-Afguilar, M. and Schwartz, J. (2005) "Impact of control for air pollution and respiratory epidemics on the estimated associations of temperature and daily mortality", *International Journal of Biometeorology* 50 (2) 121-9.

Paldy, A., Bobvos, J., Vamos, A., Kovats, R.S. and Heat, S. (2005) "The effect of temperature and heat waves on daily mortality in Budapest Hungary 1970-2000", in Kirch, W., Menne, B. and Bertollini, R. (Eds.) *Extreme Weather Events and Public health Responses* New York Springer 99-108

Parmenter, R.R., Yadav, E.P., Parmenter, C.A., Ettestad, P. and Gage, K.L.(1999) "Incidence of plague associated with increased winter-spring precipitation in New Mexico", *American Journal of Tropical Medical Hygiene* 61 (5) 814-21.

Parry, M. and Carter, T. (1998) "The first Step: Defining the Problem" in *Climate Impact and Adaptation Assessment* Earthscan Publications London 1998

Pascual, M., Ahumada, J.A., Chaves, L.F., Rodó, X. and Bouma, M. (2006) "Malaria resurgence in the East African highlands: Temperature trends revisited", *Proceedings of the National Academy of Sciences* 103 (15) 5829-5834

Patrick, M.E., Christiansen, L.E., Waino, M., Ethelberg, S., Madsen, H. and Wegener, H.C. (2004) "Effects of Climate on Incidence of Campylobacter spp. in Humans and Prevalence in Broiler Flocks in Denmark" *Applied and Environmental Microbiology* 70 (12) 7474-80.

Pattenden, S., Nikiforov, B. and Armstrong, B.G.(2003) "Mortality and temperature in Sofia and London", *Journal of Epidemiology and Community Health* 57 (8) 628-33.

Patz, J.A., Epstein, P.R., Burke, T.A., and Balbus, J.M. (1996) "Global climate change and emerging infectious diseases" *Journal of the American Medical Association* 275 (3) 217-23.

Patz, J.A., McGeehin, M.A., Bernard, S.S., Ebi, K.L., Epstein, P.R., Grambsch, A., Gubler, D.J., Reiter, P., Romieu, I., Rose, J.B., Samet, J.M. and Trtanj, T, (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 (4)367-376

Patz, J.A. (2002) " Climate change (Communication arising): Regional warming and malaria resurgence", *Nature* 420 627-628

Patz J.A. and Kovats R.S. (2002) "Hotspots in climate change and human health" *British Medical Journal* 325 1094-1098

Patz, J.A. and Olsen, S.H.(2006) "Malaria risk and temperature: influences from global climate change and local land use practices", *Proceedings of the National Academy of Sciences.* 103 (15) 5635-6.

Pebody, R.G., Ryan, M.J., and Wall, P.G. (1997) "Outbreak of campylobacter infection: rare event for a common pathogen" ,*Communicable Disease Review* 7 (3)

Pelly, H., Cormican, M., O'Donovan, D., Chalmers, R.M., Hanahoe, B.R., Cloughley R., McKeown, P. and Corbett-Feeney G. (2007) "A large outbreak of cryptosporidiosis in western Ireland linked to public water supply: a preliminary report" *Eurosurveillance weekly releases (12) 5*

van Pelt, W., Mevius, D., Stoelhorst, H.G., Kovats, S., van de Giessen, A.W., Wannet, W. Duynhoven, Y.T.H.P. (2004) "A large increase of Salmonella infections in 2003 in the Netherlands: hot summer or side effect of the avian influenza outbreak?" , *Eurosurveillance* 9(7):17-9

Peperzak, L. (2005) "Future increase in harmful algal blooms in the North Sea due to climate change", *Water Science and Technology* 51 (5) 31-6

Petersen, L.R. and Marfin, A.A. (2002) "West Nile virus:a primer for the physician", *Annals of Internal Medicine* 137 (3) 173-9

PHIS Public Health Information System (2004) Department of Health Dublin

Pirard, P., Vandentorren, S., Pascal, M., Laaidi, K., Le Tertre, A., Cassadou, S. and Ledrans, M. (2005) "Summary of the mortality impact assessment of the 2003 heat wave in France", *Eurosurveillance* 10 (7) 153-156

Prieto, M.D., Lopez, B., Juanes, J.A., Revilla, J.A., Llorca, J. and Delgado-Rodriguez, M. (2001) "Recreation in coastal waters: health risks associated with bathing in sea water", *Journal of Epidemiology and Community Health* 55 442-447

Ramlow, J. and Kuller, L .(1990) "Effects of the summer heat wave of 1988 on daily mortality in Allegheny County, PA", *Public Health Reports* 105 (3) 283-289

Randolph, S.E. and Rogers, D.J. (2000) "Fragile transmission cycles of tick-borne encephalitis virus may be disrupted by predicted climate change", *Proceedings of the Royal Society of Lond Series B Biological Sciences* 267 (1454) 1741-4

Randolph, S.E. (2004) "Evidence that climate change has caused 'emergence' of tick-borne diseases in Europe?", *International Journal of Medical Microbiology* 293 (37) 5-15

Ratcliffe, R.A.S. (1983) "Review of Summer 1983 over the Northern Hemisphere" Weather 38 (10) 319-20

Reacher, M., McKenzie, K., Lane, C., Nichols, T., Kedge, I., Iversen, A., Hepple, P., Walter, T., Laxton, C. and Simpson, J. Lewes Flood Action Recovery Team (2004) "Health impacts of flooding in Lewes: a comparison of reported gastrointestinal and other illness and mental health in flooded and non-flooded households", *Communicable diseases and Public health* 7 (1) 39-46

Reeves, W.C., Hardy, J.L., Reisen, W.K. and Milby, M.M. (1994) "Potential effect of global warming on mosquito-borne arboviruses", *Journal of Medical Entomology* 31 (3) 323-32

Reiter, P.(2000) "From Shakespeare to Defoe: Malaria in England in the Little Ice Age", *Emerging Infectious Diseases* 6 (1) 1-11

Ren, C., Williams, G.M., Mengersen, K., Morawska, L. and Tong, S. (2007) "Does temperature modify short-term effects of ozone on total mortality in 60 large eastern US communities? - An assessment using the NMMAPS data". *Environment International.* Nov 9 doi:10.1016/j.physletb.2003.10.071

Reville, W.(2004) "*No marsh as Mallow blocks the floodwater*" The Irish Times 9th December

Reynolds, J. 2006 "*The implication of the Water Framwork Directive for Local Authority Policies*" Presentation at Conference A Future for our Waters 29th September 2006 South Western River Basin District, Voice, NDP, The Heritage Council

Riberon, J., Vandentorren, S., Bretin, P., Zeghnoun, A., Salines, G., Cochet, C., Thibault, C., Henin, M. and Ledrans, M. *Building and Urban factors in heat related deaths during the 2003 heat wave in France* On file

Ring, J,. Kramer, U,. Schafer, T. and Behrendt, H. (2001) "Why are allergies increasing?", *Current Opinion in Immunology* 13 (6) 701-8

Robinson, P. J. (2001) "On the definition of a heat wave", *Journal of Applied Meteorology*, 40 (4) 762-755

Rogers, D.J. and Randolph, S. (2000) "The Global Spread of Malaria in a Future, Warmer World", *Science* 289 1763-1766

Rogers, D.J., Randolph, S., Lindsay S. and Thomas C. (2001) "Vectorborne disease and climate change," in *Health effects of climate change in the UK* Department of Health 85-98

Rogers, C.A., Wayne, P.M., Macklin, E.A., Muilenberg, M.L., Wagner, C.J., Epstein, P.R. and Bazzaz, F.A. (2006) "Interaction of the onset of spring and elevated atmospheric CO2 on ragweed (Ambrosia artemisiifolia L.) pollen production", *Environmental Health Perspectives* 114 (6) 865-9

Rollins, D.M. and Colwell, R.R. (1986) "Viable but nonculturable stage of Campylobacter jejuni and its role in survival in the natural aquatic environment". *Applied Environmental Microbiology* 52 (3) 531-8.

Rooney, C., McMichael, A.J., Kovats, R.S. and Coleman M.P.(1998) "Excess mortality in England and Wales, and in Greater London, during the 1995 heat wave", *Journal of Epidemiology and Community Health* 52 (8) 482-6.

Rosa (1998) BBC World: Americas Honduras calls national alert http//news.bbc.co.uk/1/hi/world/americas/228337.stm BBC Website accessed 5th December 1998

Rose, J.B., Epstein, P.R., Lipp, E.K., Sherman, B.H., Bernard, S.M. and Patz, J.A. (2001) "Climate variability and change in the United States: Potential impacts on water and food borne diseases caused by microbiological agents", *Environmental Health Perspectives* 109 (2) 211-221

van Rossum, C.T., Shipley, M.J., Hemingway, H., Grobbee, D.E., Mackenbach, J.P., and Marmot, M.G. (2001) "Seasonal variation in cause specific mortality: Are there high-risk groups? 25 year follow-up of civil servants from the first Whitehall study", *International Journal of Epidemiology* 30 (5) 1109-1116

Rowntree Foundation (2001) "Cold comfort The social and environmental determinants of excess winter deaths in England, 1986-1996 "A report by the Joseph Rowntree Foundation 2001 London

Saez, M., Sunyer, J., Castellsague, J., Murillo, C. and Anto, J.M. (1995)"Relationship between weather temperature and mortality: a time series approach in Barcelona", *International Journal of Epidemiology* 24 (3) 576-82

Santos, F.D., Forbes, K. and Moita, R. (2002) *Climate change in Portugal scenarios, impacts and adaptation measures* SIAM Project, Gravida Lisbon Portugal

Sartor, F., Snacken, R., Demuth, C. and Walckiers, D. (1995) "Temperature, Ambient Ozone Levels and Mortality during Summer 1994, in Belgium", *Environmental Research* 70, 105-113

Sayers, G. and Boland, M. (2002) "Leptospirosis Outbreak", *Epi-insight* 3 (3) 1

Schwartz, J., Levin, R. and Hodge, K. (1997) "Drinking water turbidity and paediatric hospital use from gastrointestinal illness in Philadelphia", *Epidemiology* 8 (6) 615-620

Schwartz, J., Levin, R. and Goldstein, R. (2000) "Drinking water turbidity and gastrointestinal illness in the elderly of Philadelphia", *Journal of Epidemiology and Community Health.* 54 (1) 45-51.

Scott, S. (2007) "*Application of the Polluter pays principle in Ireland"* Paper presented at the Water Framework Directive Implementation Conference 2nd May 2007 Carlton Hotel Dublin

Scragg, R.(1981) "Seasonality of cardiovascular disease mortality and the possible protective effect of ultra violet radiation", *International Journal of Epidemiology* 10 337-341 1981

Semenza, J.C., Rubin, C.H., Falter, K.H., Selanikio, J.D., Flanders, W.D., Howe, H.L. and Wilhelm, J.L. (1996) "Heat related deaths during the July 1995 heat wave in Chicago", *New England Journal of Medicine* 335 (2) 84-90

Shelley, E., O'Reilly, O., Mulcahy, R. and Graham, I.(1991) "Trends in mortality from cardiovascular diseases in Ireland", *Irish Journal of Medical Science* 160 (9) 5-9

Shumway, S.E., Serman, S.A., Cembella, A.D. and Selvin, R.(1994) "Accumulation of paralytic shellfish toxins by surfclams, Spisula solidissima (Dillwyn, 1897) in the Gulf of Maine: seasonal changes, distribution between tissues, and notes on feeding habits", *Natural Toxins* 2 (4) 236-51.

Shumway, R.H., Azari, A.S. and Pawitan, Y. (1988) "Modeling mortality fluctuations in Los Angeles as functions of pollution and weather effects", *Environmental Research* 45 (2) 224-41.

Singh, R.B., Hales ,S., de Wet, N., Raj, R., Hearnden, M. and Weinstein, P. (2001) "The influence of climate variation and change on diarrheal disease in the Pacific islands", *Environmental Health Perspectives* 109 (2) 155-9

Slater, A.(2006) Health and Safety Authority Personal Communication 30-8-06

Smith, H.V., Smith, H.V., Patterson, W.J., Hardie, R., Greene, L.A., Benton, C., Tulloch, W., Gilmour, R.A., Girdwood, R.W., Sharp, J.C. and Forbes, G.I. (1989) "An outbreak of waterborne cryptosporidiosis caused by post-treatment contamination", *Epidemiology and Infection* 103 (3) 703-15

Smith, H.V., Gray, J.S. and Mckenzie, G.(1991) "A Lyme Borreliosis human serosurvey of asymptomatic adults in Ireland", *Zentralblatt fur Bakteriologie.* 275 (3) 382-9

Smith, R. and Takkinen, J. (2006) "Lyme borreliosis: Europe-wide coordinated surveillance and action needed?", *Eurosurveillance* 11 (6)

Stanek, G. and Strle, F. (2003) "Lyme Borellosis" *The Lancet* 362 (9396) 1639-47

Stanwell-Smith, R. (1997) "UK reports first outbreak of cryptosporidiosis associated with filtered borehole water", *Eurosurveillance* 1 (16)

Stanwell-Smith, R. (2001) "Waterborne disease and climate change", UK in *Health effects of climate change in the UK* Department of Health 2001 120-132

Stedman, J. (2004) "The predicted number of air pollution related deaths in the UK during the August 2003 heatwave", *Atmospheric Environment* 38 (8) 1087-1090

Stout, R.W. and Crawford, V.L. (1991) "Seasonal variations in fibrinogen concentrations among elderly people", *The Lancet* 338 (8758) 9-13.

Subak, S. (2003) "Effects of climate variability in Lyme disease incidence in the Northeastern United States", *American Journal of Epidemiology* 157 531-538

Sumilo, D., Bormane, A., Asokliene, L., Lucenko, I., Vasilenko, V,. and Randolph, S. (2006) "Tick-borne encephalitis in the Baltic States: identifying risk factors in space and time", *International Journal of Medical Microbiology*.296 Suppl 40 76-9.

Sweeney, J., Donnelly, A., Mc Elwain, L. and Jones, M. (2002) *Climate Change: Indicators for Ireland* Environmental Protection Agency Ireland

Sweeney, J., Brereton, T., Byrne, C., Charlton, R., Emblow, C., Fealy, R., Holden, N., Jones, M., Donnelly, A., Moore, S., Purser, P., Byrne, K., Farrell, E. and Mayes, E. (2003) *Climate change Scenarios and Impacts for Ireland* Environmental Protection Agency Ireland

Tam, C.C., Rodrigues, L.C., O'Brien , S.J. and Hajat, S. (2006) "Temperature dependence of reported Campylobacter infection in England, 1989-1999", *Epidemiology and Infection* 134 (1) 119-25.

Tatem, A.J., Rogers, D.J. and Hay, S.I. (2006) "Estimating the malaria risk of African mosquito movement by air travel", *Malaria Journal* 14 (5) 5-57

The Irish Times 2007 (a) on file

The Irish Times 2007 (b) on file

The Irish Times 2007 (c) on file

Thornley, D. and Nicol, C. (2002) "The rising incidence of salmonella in New Zealand", *New Zealand Public Health Report* 9 (4) 25-8

UK Department of Health (2001) *Health effects of climate change in the UK* Department of Health London

Upton, M.N., McConnachie, A., McSharry, C., Hart, C.L., Smith, G.D., Gillis, C.R and Watt, G.C. (2000) "Intergenerational 20 year trends in the prevalence of asthma and hay fever in adults: the Midspan family study surveys of parents and offspring", *British*

Medical Journal. 321 (7253) 88-92.

Vandentorren, S., Suzan, F., Medina, S., Pascal, M., Maulpoix, A., Cohen, J.C. and Ledrans, M. (2004) "Mortality in 13 French cities during the August 2003 heat wave", *American Journal of Public Health* 94 (9) 1518-20

Vandentorren, S. and Empereur-Bissonnet P. (2005) in Kirch, W. Menne, B. and Bertollini, R.(Eds.) "Health Impact of the 2003 Heat Wave in France", *Extreme Weather Events and Public health Responses* New York Springer 89-98

Wade, T.J., Sandhu, S.K., Levy, D., Lee, S., LeChevallier, M.W., Katz, L., Colford, J.M. Jr. (2004) "Did a severe flood in the Midwest cause an increase in the incidence of gastrointestinal symptoms?" *American Journal of Epidemiology* 159 (4) 398-405.

Walker, J. (1998) "Malaria in a changing world: an Australian perspective", *International Journal of Parasitology* (6) 947-53.

Wall, P.G., de Louvois, J., Gilbert, R.J., Rowe, B. (1996) "Food poisoning: notifications, laboratory reports, and outbreaks – where do the statistics come from and what do they mean?", *Comminucable Disease Review* 6 (7) 93-100

Wall, P. (2001) *FSAI News* 6 (3) 1 Food Safety Authority of Ireland

Weiss, R.A. and McMichael, A.J (2004) "Social and environmental risk factors in the emergence of infectious diseases", *Nature Medicine* 10 (12) S70-6

Weisskopf, M.G., Anderson, H.A., Foldy, S., Hanrahan, L.P., Blair, K., Torok, T.J. and Rumm, P.D. (2002) "Heat wave morbidity and mortality, Milwaukee, Wis, 1999 vs 1995: an improved response?", *American Journal of Public Health* 92 (5) 830-3

Weniger, B.G., Blaser, M.J., Gedrose, J., Lippy, E.C. and Juranek, D.D. (1983) "An outbreak of waterborne giardia associated with heavy water runoff due to warm weather and volcanic ashfall", *American Journal of Public Health* 73 (8) 868-72

Wheeler, J.G., Sethi, D., Cowden, J.M., Wall, P.G., Rodrigues, L.C., Tompkins, D.S., Hudson, M.J. and Roderick, P.J. (1999) "Study of infectious disease in England: rates in the community, presenting to general practice and reported to national surveillance", *British Medical Journal* 318 1046-1050

White, M.R. (1985). in White, M. R., and Hertz-Picciotto, (Eds.) "Human health: Analysis of climate related to health", in *Characterization of information requirements for studies of CO2 effects: Water resources, agriculture, fisheries, forests and human health,* 72-206. Washington, D.C.: Department of Energy.

Whitman, S., Good, G., Donoghue, E.R., Benbow, N., Shou, W. and Mou, S. (1997) "Mortality in Chicago attributed to the July 1995 heat wave", *American Journal of Public Health* 87 (9) 1515-8

WHO (1996) "*Climate change and World Health",* World Health Organization WHO, WMO and UNEP Geneva 1996

WHO (1999) "*Declaration Third ministerial conference on environment and health*" London 1999 Commission of European Communities, Action in Partnership, World Health Organization London

WHO (2000) "*WHO Surveillance Programme for Control of Foodborne Diseases in Europe*" 7th report World Health Organization Geneva

WHO (2001 (a)) "*Monitoring health impacts of climate change in Europe* Meeting Report" World Health Organization London

WHO World Health Organization (2001 (b)) "*Progress with Roll Back Malaria in the WHO European region*" April 2001 World Health Organization Geneva

WHO (2002 (a)) "*Food Safety and Foodborne illness*" Fact sheet No 237 World Health Organization Geneva

WHO (2002 (b)) "*Flooding: health effects and preventive measures*" WHO Europe Fact sheet 05/02 World Health Organization Bonn

WHO (2003 (a)) "*The present state of foodborne disease in OECD countries*" World Health Organization Geneva

WHO (2003(b)) "*Methods of assessing human health vulnerability and public health adaptation to climate change*" World Health Organization Series No. 1 2003 WHO UNEP Health Canada 2003

WHO (2003 (c)) "*The World Health Organisation Surveillance 8th report on the programme for the control of foodborne infections and intoxications in Europe 1999-2000*" World Health Organization Geneva

WHO (2004 (a)) "*Heat waves: risks and responses Health and Global Environmental change*" Series No 2 World Health Organization Geneva

WHO (2004(b)) "*Climate change Risks and Responses*" World Health Organization Geneva 2004

WHO (2004(c)) World Health Organization fact sheet Website accessed 3rd June http://www.rbm.who.int/cmc_upload/0/000/015/372/RBMInfosheet_1.htm

WHO (2004(d)) "*Fifth Futures Forum on rapid response decision making tools*" World Health Organization Regional Office for Europe Madrid

WHO (2005) "*Using climate to predict infectious disease epidemics*" World Health Organization Geneva

WHO (2006) World Health Organization Geneva http://www.who.int/genomics/public/geneticdiseases/en/index4.html#Malaria Website accessed 2nd September 2006

WHO (2007 (a)) "*New country-by-country data show in detail the impact of environmental factors on health*" World Health Organization Press Office Geneva 13 June 2007 Information available on http//www.who.int/quantifying_ehimpacts/countryprofilesebd.xls

WHO (2007(b)) "*The Millennium Development Goals Report*" World Health Organization NewYork

WHO (2007(c)) International Classification of Diseases (ICD) World Health Organization http://www.who.int/classifications/icd/en/ Website accessed 9th September 2007

WHO (2007(d)) "Climate change Quantifying the health impacts at national and local levels" Campbell-Lendrum D. and Woodruff R. Geneva 2007

WHO (2008) EB122/SR/5 122nd Session Agenda item 4.1 24th January 2008 WHO Geneva

Woodhouse, S. (1880) "*Report made to the Local Government Board in Ireland by Dr. S. Woodhouse ... on an outbreak of fever, from the 1st day of November 1879...*" HMSO http//www.bopcris.ac.uk/browse/eppiLCSH/720_1.html Website accessed 26th June 2007

Woodhouse, P.R., Khaw, K.T., Plummer, M., Foley, A. and Meade, T.W. (1994) "Seasonal variations of plasma fibrinogen and factor VII activity in the elderly: winter infections and death from cardiovascular disease", *The Lancet*. 343 (8895) 435-9.

Woodward, A. and Hales, S.(2001) "*Climate change and potential effects on human health in New Zealand A Report prepared for the Ministry of the Environment 2001*" New Zealand Climate Change Programme Wellington New Zealand

World Food Programme, (2006) wwwwfp.org/aboutwfp/facts/hunger_facts.asp Website accessed 28th July 2006

Zebisch, M., Grothmann, T., Schröter, D., Hasse, C., Fritsch, U., & Cramer, W. (2005) "Climate Change in Germany – Vulnerability and Adaption of Climate sensitive Sectors" Potsdam Institute for Climate Impact Research Report 201 (41) 253

Zell, R. (2004) "Global Climate change and the emergence/reemergence of infectious diseases", *International Journal of Medical Microbiology* 293 (37)16-26

Zhang, J.N., Wood, J., Bergeron, A.L., McBride, L., Ball, C., Yu, Q., Pusiteri, A.E., Holcomb, J.B.and Dong, J.F. (2004) "Effects of low temperature on shear-induced platelet aggregation and activation", *The Journal of Trauma* 57 (2) 216-23.

Zucker, J. (1996) "Changing patterns of Autochthonous malaria transmission in the United states: A review of recent outbreaks", *Emerging Infectious Diseases* 2 (1) 37-43