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An analysis of the mortality risks associated with heat and heat waves in Ireland, to assist in planning for climate change

Mathilde Pascal

Submitted in Fulfillment of the Requirements of the Award of PhD

Dublin Institute of Technology

School of Physics

Supervisor Professor Patrick Goodman

Advisory Supervisor: Professor Joel Schwartz

Submitted May 2011

Declaration

I certify that this thesis which I now submit for examination for the award of PhD is entirely my own work and has not been taken from the work of others, save and to the extent that such work has been cited and acknowledged within the text of my work.

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31/05/2011

Abstract

Although extreme temperatures have not been identified as a major cause of mortality in Ireland, climate change calls for an evaluation of the past, present and future health risks associated with heat and heat waves.

The health impacts of heat were investigated using mortality and temperature data for the period 1981-2003. Data were aggregated in urban areas (Dublin, Cork, Drogheda, Arklow, Dundalk, Galway, Limerick, Waterford and Wexford) and rural areas. Seven heat waves were identified between 1981 and 2003, corresponding to 254 excess deaths (197 in rural areas, and 57 in urban areas). A major episode was observed in rural areas in 1983: +115 [confidence interval CI 95% 96:137] extra deaths between the 5th and the 23rd July 1983.

During summer, a 1°C increase above 15°C in the mean temperature was associated with a 1.5% [CI 95% 0.9:2.1] increase in total mortality in rural areas, and a 1.6% [0.6:2.5] increase in total mortality in urban areas. Risks were modified by the mortality observed in the preceding winter. There are indications that the heat-related risks have been decreasing between the 80s and the 90s.

A better geographical resolution of the mortality data is an asset to refine this analysis and to study any relationship between a health topic and an environmental exposure.

Despite limits on the data, an increase in temperature was associated with an increase in mortality during summer in Ireland, and past heat waves were associated with a small but observable excess mortality. With the perspective of climate change, and with the ageing of the population, it may be that more severe heat episodes results in a larger mortality burden, as was observed during the July 1983 heat wave. Steps to reduce vulnerability to heat during extreme episodes should be considered.

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For any errors or inadequacies that may remain in this work the responsibility is entirely my own.

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EXECUTIVE SUMMARY

1. Objectives of the PhD

The Fourth Assessment Report (AR4) of the Intergovernmental Panel on Climate Change (Solomon *et al.* 2007) concludes that warming of the climate system is unequivocal, and that temperatures will continue to rise in the next decades. The estimates of the global temperature increases vary between $\pm 1.8^{\circ}$ C and $\pm 6.4^{\circ}$ C by 2100, compared to the 1961-2000 mean temperature (Solomon *et al.* 2007). Between 1961 and 2005, the climatic trends identified in Ireland were largely consistent with global trends, and projected temperature increases range between $\pm 1^{\circ}$ C and $\pm 3^{\circ}$ C by 2100, compared to the 1961-2000 mean temperature (Sweeney *et al.* 2003).

Climate change may have several health impacts, one of them related to the increase in temperature and the increase in the frequency and severity of extreme events such as extremes of cold, drought, floods, and heat waves. Heat waves have been associated with significant mortality and morbidity impacts in many countries. Heat-related risks have been known for long. In Europe, adaptation to heat and heat waves is now considered a priority. Although extreme temperatures have not been identified as a major cause of mortality in Ireland, climate change calls for an evaluation of the past, present and future health risks associated with heat and heat waves. It is the purpose of this PhD to investigate if past heat waves have been characterized by an observable excess mortality in Ireland, and to quantify

the temperature-mortality relationship during summer, in order to identify relevant adaptation strategies.

2. The health impacts of heat waves

Several studies showed that warm temperatures are associated with high mortality levels (Basu, 2009b). The negative impact is particularly visible during periods of sustained heat, qualified as heat waves. A lag period of one to three days is usually observed between the peak in temperatures and the peak in mortality. In most cases, the net mortality effects are significant. For instance, in England and Wales, a heat wave between the 30th July and the 3rd August 1995 resulted in 768 excess deaths (Rooney *et al.* 1998). In Europe, there is little indication of an impact on morbidity, e.g. hospital admissions or emergency calls (Kovats *et al.* 2004).

At risk populations include young children, the elderly, people with underlying diseases, and workers. The mortality burden is concentrated on urban areas, where the population is over-exposed because of the additional heat created by the urban heat-island (Laaidi *et al.* 2011).

3. The mortality impacts of heat waves in Ireland

The work focused on mortality, as it required at least 10 years of data, which are not available for other health indicators such as hospital admissions. It used the individual mortality data collected by the Central Statistics Office for the period 1981-2003. The work

focused on the main causes of death that had been associated with heat exposure in the literature; total, cardiovascular and respiratory mortality.

Meteorological data (temperature, humidity, pressure and wind speed) were obtained from Met Éireann for eleven meteorological stations covering the whole country.

As the location of death was only available for the main population centres, aggregated indicators had to been created to match the mortality and the temperature data. Since we may expect differences in the health impacts of heat waves in urban and rural areas, the choice was to create a urban and a rural indicator. Urban areas included Dublin, Cork, Drogheda, Arklow, Dundalk, Galway, Limerick, and Waterford, while the remaining areas were classified as rural. Temperature indicators were aggregated at a similar spatial resolution, to obtain a proxy of urban and rural heat exposure.

The first part of the PhD identified heat waves and estimated the mortality impacts of past heat waves. Heat waves are periods of sustained heat during several consecutive days, with intensity and duration that depend on the local context. This work tested several definitions of heat waves, and retained periods when both the minimum and the maximum temperature were above the 90th percentile of the temperatures summer distributions for at least two days. Between 1981 and 2003, a limited number of periods were characterized as heat waves observed in most of the meteorological stations: July 1983, August 1984, July 1989, June 1995, August 1995, June 2003 and August 2003. July 1983 was characterised by a classical heat wave pattern, i.e. a rapid increase in temperatures, followed by a rapid decrease. The pattern was less consistent during the other periods, especially August 1995 when temperatures were unusually warm throughout the summer, but without marked peaks.

The mortality impact of heat waves was estimated by comparing the observed mortality during heat waves to reference mortality. The reference mortality was predicted from two methods; 1) an average over the previous year, and 2) a statistical model taking into account the usual temperatures observed during the same period, controlling for long-term trend, seasons, day of the weeks and influenza epidemics.

Overall, a total of 254 excess deaths attributable to heat waves was estimated, 197 in rural areas, and 57 in urban areas. The 1983 and to a lesser extent the 1984 heat waves were characterised by a significant excess mortality, especially in rural areas (+115 [CI 95% 96:137] extra deaths between the 5th and the 23rd July 1983, +49 [29:68] deaths between the 18th and the 31st August 1984). The July 1983 episode was the only one presenting a characteristic heat wave mortality response, although with moderate intensity. A maximum relative risk of mortality due to the heat wave effect was observed on the 14th of July 1983 (1.23 [CI 95% 1.13:1.34]). A large part of the mortality burden was observed for the oldest age group (>74 years old). In the most recent episodes, especially in 1995 and 2003 deficit mortality was observed in rural areas, while the mortality slightly increased in urban areas. In urban areas, the estimates associated with the 1995 heat wave were highly variable depending on the method, which indicates a confounding factor, possibly air pollution.

4. The summer temperature-mortality relationship in Ireland

A further aspect to this study was the investigation of the temperature-mortality relationship. This was investigated using time-series models with the same datasets. In both rural and urban areas, and for all causes of deaths, the mortality was found to increase when the mean temperature of the same day increased. The relationship was roughly linear above 15° C. A 1°C increase above 15° C in the mean temperature was associated with a 1.5% [CI 95% 0.9:2.1] increase in total mortality in rural areas, and a 1.6% [0.6:2.5] increase in total mortality in rural areas, and a 1.6% [0.6:2.5] increase in total mortality in rural areas, and a 1.6% [0.6:2.5] increase in total mortality in urban areas. The impact was slightly lower for the total mortality over 75 years old (respectively 1.4% [0.6:2.2] and 1.5% [0.3:2.7]). In rural areas, a significant impact was observed on cardiovascular mortality (1.1% [0.3:1.9]) but not on respiratory mortality (+0.0 [-1.1:1.0]). The opposite was observed in urban areas, with no impact on cardiovascular mortality (+0.2 [-0.8:1.3]) but a large impact on respiratory mortality (+2.8% [0.5:5.1]). This may indicate that respiratory mortality is driven by a confounding factor, which may be air pollution.

It was also found that the risks were significantly larger during summers preceded by lowmortality winter, than during summers preceded by high-mortality winter. For instance, for the total mortality in rural areas, the risks changed from 2.2% [1.3:3.1] to 0.9% [0.1:1.7] depending on the mortality observed during the preceding winters.

The same models were run on four year periods (1981-1984, 1985-1988, 1989-1992, 1993-1996, 1997-2000 and 2001-2003) to investigate possible changes in the temperaturemortality relationship over time. These periods were relatively homogenous in terms of temperatures and mortality. In rural areas, the risks associated with a 1°C increase above 15°C were around 2% for the periods 1981-1984, 1985-1988 and 1989-1992. They started to decrease between 1993 and 1996, and became negative after 1997. A similar evolution was observed for the mortality above 74 years old in rural areas and for the cardiovascular mortality. As for the respiratory mortality, low and non-significant risks were observed throughout all the periods. In urban areas, a large risk was observed during the first period (+5.1% [2.9:7.4]), while the response was lower and non-significant in the other periods. This may be partly due to changes in the population characteristics (e.g. ages), improvements in the health care system, but also an improvement in air quality.

5. Opportunities for adaptation

The results should be interpreted with care, due to the limits introduced by the rough geographical aggregations that were used. This PhD found that an increase in temperature was associated with an increase in mortality during summer in Ireland, and that past heat waves were associated with a small but observable excess mortality. There are indications that the heat-related risks have been decreasing between the 80s and the 90s. However, with the perspective of climate change, and with the ageing of the population, it may be that more severe heat episodes results in a larger mortality burden, as was observed during the July 1983 heat wave.

The perspective of facing more and more intense heat waves, together with potential increasing vulnerabilities calls for dedicating some efforts to the prevention of heat waves.

These efforts should however be proportionate to the risks expected in Ireland. Communication to promote appropriate behaviours in the population and in the health professionals may be a first and essential step to limit the adverse impacts of heat waves. The relevance of setting a heat warning system to anticipate heat waves episodes that could result in increased mortality should be discussed. Such system would alert stakeholders who in turn can promote preventive actions and disseminate appropriated information. As night temperatures are usually low, most of the heat-exposure is a day-time exposure, and the warning system may rely on the maximum temperature only. For instance, a threshold of 25°C may prevent an excess mortality above 2% in both urban and rural areas, and occurs on a moderate number of days, most of them corresponding to the 1983, 1995 and 2003 heat waves. This may be integrated in a simple system where the health authorities would be informed of a potentially dangerous heat wave, and would be able to reinforced communication on appropriate behaviours.

6. Ways forward

In addition to heat prevention, a first and essential step to adapt to climate change would be to improve the surveillance of health data, and especially of the mortality data. A better geographical resolution is an asset to study any relationship between a health topic and an environmental exposure.

Such data would be essential to contribute to adaptation in at least three different ways; 1) to contribute to the scientific evidence on the health impacts of climate change, 2) to warn for unexpected impacts, and 3) to prioritize adaptation.

Finally, adaptation plans to climate change are being developed worldwide, involving various stakeholders who have different understanding of the "climate change and health issues". The framework proposed in this PhD is a simple way to organize available epidemiological information in order to identify where the climate may interfere and what are the main sources of vulnerabilities. Although we detailed an example based on heat waves, as it is one of the most studied impacts of climate change, the framework can be applied to any type of risks, including infectious diseases.

7. Added value of the PhD

This PhD was able to document the mortality impacts of heat and heat waves in Ireland, using classical epidemiological methods. Despite the limits introduced by the data availability and quality, an excess mortality attributable to heat waves was observed in Ireland, especially during the July 1983 heat wave. Results also showed that temperatures above 15°C are associated with an increase in mortality. Heat should therefore be considered as a risk in Ireland, and steps to reduce vulnerability to heat during extreme episodes should be appropriate.

The PhD also underlined the need for good surveillance data at a low geographical scale, which are an asset to perform any epidemiological studies in relation to climate change.

CHAPTER I. INTRODUCTION

1.1. Heat waves and mortality: a potential threat in Ireland

Since the first report issued by the Intergovernmental Panel on Climate Change, observations and models improved our understanding of the observed and forecasted changes in climate, and of their impacts on the environment and on society. It is acknowledged today that climate change will affect human health, via direct and indirect mechanisms. Therefore, adaptation, i.e. the "adjustment in natural or human systems in response to actual or expected climatic stimuli or their effects, which moderates harm or exploits beneficial opportunities" (McCarthy *et al.* 2001) is considered a public health priority (World Health Assembly, 2008).

Whilst the most urgent need concerns adaptation in the developing world (Costello *et al.* 2009b), European countries are required to adapt to prevent the health effects of climate change. Risks consistently reported for Europe include the emergence or re-emergence of infectious diseases, the increase in frequency and intensity of extreme climatic events and profound changes in the environment (Haines *et al.* 2006b; McMichael *et al.* 2006a; Parry *et al.* 2007a). Heat waves are one of the main concerns, as they had already resulted in significant excess mortality in several countries (Basu, 2009b; Basu and Samet, 2002; Kovats and Kristie, 2006; Parry *et al.* 2007a). For instance, the European project EuroHeat including Athens, Barcelona, Budapest, London, Milan, Munich, Paris, Rome and Valencia estimated that heat waves resulted in 1,280 excess deaths between 1990 and 2004 (D'Ippoliti *et al.* 2010).

Climate change should result in a change in the variance and the mean of the temperatures distribution. The change in the variance would result in an increase frequency and duration of episodes which are today considered as extremes. In the last 50 years, a significant increase in the number of warm nights has been observed, while the increase in the occurrence of hot days is less marked (Meehl and Tebaldi, 2004; Solomon *et al.* 2007). (Della-Marta *et al.* 2007) showed that the length of episodes defined as the maximum number of consecutive days where the daily maximum temperature exceeds the long-term daily 95th percentile of daily maximum temperature over Western Europe has doubled between 1880 and 2005, and that the frequency of hot days (i.e. when the daily maximum temperature) has tripled during the same period. In Ireland, the number of episodes when for at least 6 consecutive days, the maximum temperature is at least 5°C greater than the 1961-1990 climatologically mean value, has already increased at a number of stations (McElwain and Sweeney, 2007).

In addition to the increase in temperature, the concentration of people in urban areas, and the ageing of the population may result in an increase of the number of people sensitive to heat. In Ireland, the number of people in the over 74 age group is expected to increase to between 273 289 and 284 413 in 2021, compared with 190 398 recorded in the 2002 census. It is also expected that more than 20% of people older than 65 years old will be living in Dublin city and county (National Council on Ageing and older people, 2009).

Therefore adaptation to heat is considered a priority in Europe. Although extreme temperatures have not been identified as a main cause of mortality in Ireland, climate change calls for an evaluation of the present and future health risks associated with heat and heat waves. Heat stress and increased mortality in vulnerable groups is considered a potential threat in Ireland (Environmental Protection Agency, 2009).

It is the purpose of this PhD to investigate if past heat waves have been characterized by a significant excess mortality in Ireland, and to quantify the temperature-mortality relationship during summer, in order to identify relevant adaptation strategies.

1.2. How to study the health impacts of climate change and the adaptation needs?

The first step of this PhD was to propose a general framework to assess the health impacts of climate change and the adaptation needs (Chapter II. A framework to study the health impacts of climate change). The framework was then applied to the issue of heat waves to select the determinants influencing the health impacts of heat exposure, and to determine how climate change could interact with those determinants. This step allowed identifying areas of vulnerabilities and knowledge gaps (Chapter III. Determinants of the health impacts of heat waves).

1.3. How to define heat waves in Ireland?

Despite a growing concern, there is till no consensus on how to define a heat wave (Meehl and Tebaldi, 2004).

From a climatologic point of view, a heat wave is an anomaly of temperature observed during several consecutive days. It is the impacts of one event on the population or the environment that gives it its importance, and transform a statistical anomaly of temperature into a "heat wave". It is likely that several factors such as humidity, demography or urban densities can explain that similar anomalies of temperature result in different health outcomes. How to define a heat wave is therefore a key question, as temperature is only one of the relevant parameters. Yet, since it is the most important one, and easily assessed, heat waves are often defined based on temperature, or on temperature and humidity.

(Meehl and Tebaldi, 2004) proposed two methods to define a heat wave. The first is based on the concept of an "annual worst heat event", using the night-temperatures, based on the assumption that they are the most important when considering a health impact. Each year, heat waves are identified as the three consecutive warmest nights. In that definition, the duration is fixed, but the intensity varies each year.

The second is based on the concept of exceeding specific temperature thresholds, which fixes the intensity but allows variations in the duration. These approach is the most largely used in the literature on the health impacts of heat waves, each studies using thresholds tailored to each event (D'Ippoliti *et al.* 2010; Garssen *et al.* 2005; Huang *et al.* 2010; Nitschke *et al.* 2007; Tong *et al.* 2010b).

A specific definition for Ireland is proposed in this PhD (Chapter V. Health impacts of heat waves).

1.4. What was the impact of past heat waves in Ireland?

Based on the definition specifically designed for Ireland, the main heat waves occurring between 1981 and 2003 were identified. Their impact on mortality was analyzed comparing different statistical methods proposed in the literature (Chapter V. Health impacts of heat waves). The comparison of the estimates obtained by the different methods gives a range of the real impact of the heat waves.

1.5. The quantification of the temperature-mortality relationship during summer

On a distinct step, the impacts of warm temperature on mortality were investigated, without focusing on heat waves. The use of classical methods for the analysis of time-series allows quantifying the relative risk of mortality for each degree increase above a pre-defined threshold (Chapter VI. Characterization of temperature-mortality relationship during summer). The relative risks were then applied to future climate, in order to obtain an estimation of the health impacts of climate change (Chapter VII. Estimation of the mortality impacts associated with climate change).

1.6. Opportunities for adaptation

The above results were used to evaluate the risks associated with heat waves in Ireland, and to propose possible preventive actions (Chapter VIII. Opportunities for adaptation). This chapter proposes a review of the possible adaptation; physiological, social (e.g. heat prevention plan) and technological (e.g. air conditioning, urbanisation plan...), with a focus

on heat prevention plans that are developed to anticipate the effects of adverse heat waves. It discusses the main components of such plans, and proposes possible warning thresholds for Ireland.

1.7. A perspective on other risks sensitive to climate change, and of research needs

Since heat stress is only one of the many impacts associated with climate change, the thesis proposes a rapid review of some other risks that could be modified by climate change (Chapter IX. A review of other risks that could be impacted by climate change). It concludes with research needs to improve the understanding of the health impacts of warming temperatures in Ireland (Chapter X. Conclusions).

CHAPTER II. A FRAMEWORK TO STUDY THE HEALTH IMPACTS OF CLIMATE CHANGE

Climate change, its impacts, and the associated adaptation are relatively new concepts, surrounded by many uncertainties. The lists of the potential impacts of climate change can be overwhelming, resulting in a picture so complex that it becomes difficult to act. Epidemiologists are not familiar with the methods of climatologists, and vice versa. For instance, time scales are different; years when working on health, hundreds of years when working on climate change. Yet, a good understanding of the health impacts of climate change is essential to promote efficient adaptation.

This chapter proposes a framework to organize the available information on any health issue (heat waves, air pollution, infectious diseases...), in order to help identifying where climate change may interfere, what are the main sources of vulnerabilities, and what are the opportunities for interventions. This framework was initially developed to identified the health impacts of climate change and surveillance needs in France (Beaudeau *et al.* 2011; Pascal, 2010b).

2.1. Definitions

2.1.1. Climate change

In this framework, climate is defined as the statistical description, in terms of the mean and the variability, of meteorological parameters (temperature, pluviometry, wind, etc.) in a given region, over timescales ranging between a few months to several thousands of years. Traditionally, a timescale of thirty years is used for the calculation of climatic norms (Hulme et al. 2009a). Changes in the climate indicate a significant statistical variation in the average state of the climate or its persistent variability over long periods (generally over decades or longer). Although temperature is the symbolic variable of climate changes, the entire range of climatic parameters (humidity, cloud cover, rainfall, the CO₂ content of the atmosphere, etc.) are all likely to change. In the past, the Earth has witnessed cycles of changes, to varying degrees and of varying durations. However, observations currently show that the average annual temperature is increasing at an unprecedented rate (Solomon et al. 2007). Eleven out of the twelve years from 1995 to 2006 were among the twelve warmest years since 1850. Global warming has become more pronounced over the last 50 years, with an increase in temperature of 0.13 degrees per decade between 1956 and 2005. It is also more marked in the northern hemisphere and over continental zones (Solomon et al. 2007). Rainfall levels have increased significantly between 1900 and 2005 in northern Europe and decreased within the Mediterranean boundary. Observations also show that ice caps and glaciers have diminished in size. On average, sea levels have risen at an annual rate of 1.8mm [1.3 - 2.3] between 1961 and 2003 and by 3.1mm [2.4 - 3.8] between 1993 and 2003 (Solomon *et al.* 2007). The increase in levels of CO_2 in the atmosphere has made a significant contribution to the acidification of the oceans. The average pH of the oceans (8.1) has decreased by 0.1 since the beginning of the industrial era (Solomon *et al.* 2007).

Sudden changes in climate are defined as major changes occurring over a few decades or less and lasting for several decades. The changes that are currently most frequently mentioned are a sudden rise in sea levels, a sudden change in the water cycle, a change in the North Atlantic circulation, the melting of the ice caps in Greenland, the disappearance of the Amazon rainforest, changes in the amplitude and frequency of El Niño events and a change in the methane levels in the atmosphere (Allison *et al.* 2009). Such changes would have social, environmental and health consequences that it is impossible to evaluate today (Hulme, 2003). Therefore, this PhD focuses on climate change, but does not take into account sudden changes in climate.

2.1.2. Global change

Global changes cover climate changes and all other changes of anthropic origin having general consequences, such as the thinning of the ozone layer, reduction in biodiversity, changes to the water systems and the supply of fresh water, degradation of the soil and pressures exercised on food production systems. All of these environmental, social and economic changes lead to the weakening of human and natural systems and could potentially increase vulnerability to climate change. Therefore, they must be considered when studying the impacts of climate change.

2.2. A general overview of the health impacts of climate change

The health impacts of climate change were first mentioned since the early works on climate change. A 1985 report by the US department of Energy provided a good synthesis of various health risks that could be modified by climate change, including heat waves (White, 1985). The conceptualization of the links between climate change and health has been

proposed by (McMichael *et al.* 2006a) as schematized in Figure 2.1. reports and national reports have used this scheme to compile epidemiological studies and to identify the main risks, which corresponds to three principal types (World Health Organization Regional Office for Europe *et al.* 2003):

- an increase in the frequency and intensity of extreme climatic events,

- the emergence or re-emergence of infectious diseases,

- gradual changes to the environment and to ways of life that will modify existing exposures or even bring about new exposures.

Some of these risks are detailed in Chapter IX.



Figure 2.1. Schematization of the links between health and climate change

(McMichael et al. 2006a)

2.3. A framework for integrating climate change into existing health questions

The scheme presented in Figure 2.1 has the advantage of listing a large number of risks that could be impacted by climate change, and helps in identifying climate-sensitive health outcomes. It is also useful when trying to motivate mitigation, since it shows that climate change can affect human health in many different ways. It can be useful to put climate change on top of the political agenda, as reframing climate change as a public health problem was found to make the issue more significant to the public (Maibach *et al.* 2008b). However, when the objective is to identify needs and opportunities for adaptation, an approach risks by risks is needed to achieve the desirable level of complexity.

The World Health Organization proposed a method to estimate the burden of climatesensitive diseases (Campbell-Lendrum and Woodruff, 2006; Campbell-Lendrum and Woodruff, 2007). It consists of quantifying the links between present health outcomes and climate, and in predicting future impacts based on climate models. In addition, the modified DPSEEA model, which incorporates the influences of the social, economical and cultural background on the exposure (Morris *et al.* 2006a) may be used. A focus on the exposure<->health part of the DPSEEA model allows distinguishing three main categories of drivers; the environment, the individual and social behaviours, and the demography and background health (Figure 2.2).

For a defined health issue, each category can be expanded based on the available knowledge, the objective being to consider a large panel of the determinants of health at

stake. Indeed, in some cases, acting on one determinant, even not directly linked with climate, could be more efficient, practical and easy to implement than focusing on climate-sensitive determinants (Medeiros *et al.* 2010a).

This method is applied to the topic of heat and heat waves in Chapter 3.



Figure 2.2. Impacts of climate and climate change on the different determinants for exposure

CHAPTER III. DETERMINANTS OF THE HEALTH IMPACTS OF HEAT WAVES AND VULNERABILITIES TO CLIMATE CHANGE

The framework developed in Chapter II was used to organize the available evidence on the health impacts of heat and heat waves. The general scheme for heat waves was first developed based on a literature review detailed below. On a second step, health determinants that could be investigated in order to quantify the present and future heat-related risk for Ireland were identified.

3.1. The health impacts heat exposure

3.1.1. Thermoregulation and heat exposure

A healthy human body has an internal temperature stabilized around 36.7°C. It can vary (< 1°C) with physical activity, emotional state, time of the day, or the ovulatory cycle in women. In a healthy individual, changes in body temperature exceeding 1°C are caused by an illness. The temperature is maintained through a complex thermoregulation system, which allows the balance between heat production and heat loss. When the ambient temperature gets too warm, the thermoregulation system does not succeed in evacuating the excess heat, and the body temperature rises. This stress can result in various health outcomes, including heat cramp (a mild disorder caused by sodium depletion in the body), heat exhaustion (caused by inadequate fluid or sodium intake and characterised by thirst, fatigue, headache, nausea, elevated body temperature), and heat stroke (Batscha, 1997).

Heat stroke is diagnosed when an environmental exposure to heat results in a body temperature above 40°C. Healthy adults are more likely to develop a heat stroke after having an intense physical activity (exertional heat stroke), or by drinking too little water, or drinking alcohol for instance. The effects of heat exposure can also be aggravated by drugs impairing the thermoregulation system (e.g. diuretics, vasodilatation...), resulting in heat stroke or associated disorders (Stollberger *et al.* 2009). Children, elderly people and vulnerable adults are more susceptible to develop non-exertional heat stroke. Heat stroke is a life-threatening emergency (Bouchama and Knochel, 2002), and recovery is low. An investigation of the cases cured at the Lyon hospital during the August 2003 heat wave revealed that 71% of the patients died in the 2 years following the heat stroke (58% died 28 days after the heat stroke). Serious sequels were found in all surviving patients (Argaud *et al.* 2007).

Yet, heat strokes only represent a fraction of the heat-related mortality. Heat exposure can lead to cardiovascular exhaustion (Crandall and Gonzalez-Alonso, 2010), and aggravation of pre-existing health conditions (Kenny *et al.* 2010). In the Netherlands, 26% of the heat-related mortality (occurring on days with T>20°C) from cardiovascular diseases was explained by an increase in stress on the respiratory and circulatory systems(Kunst *et al.* 1993). Older adults, young children, and persons with chronic medical conditions are particularly susceptible to this stress and are at high risk for non-heatstroke, heat-related mortality.

3.1.2. Heat and heat wave-related mortality

The literature shows that daily increases in temperature are consistently associated with increases in mortality. For instance, in England, an analysis showed that short spells of hot weather were associated with an increase in respiratory diseases, cardiovascular diseases and cerebrovascular diseases in people aged 60 and over in Greater London, between 1965 and 1972 (Macfarlane, 1978). A later study showed that temperatures above 20°C were associated with an increase in cardiovascular mortality varying between 0.5 and 15.1% depending on age and diseases, with a stronger effect observed in the elderly (Keatinge *et al.* 1989).

A negative impact of warm temperature is also observed in warm countries. For instance in Madrid, the daily mortality (total, respiratory, and circulatory) among persons aged 65 years and older increased up to 28.4% for every degree above 37°C (Diaz *et al.* 2002).

When the elevated temperatures last several days, the mortality signal is even clearer. During a heat wave, a lag period of one to three days between the maximum temperature and the maximum mortality is usually reported (Greenberg *et al.* 1983; Kunst *et al.* 1993). A typical example is illustrated by Figure 3.1. It shows a peak of mortality in Chicago two days after the warmest temperatures of the 13th July 1995. This rapid effect is visible for all causes of mortality. In most cases, the net mortality effects are significant. The European project EuroHeat including Athens, Barcelona, Budapest, London, Milan, Munich, Paris, Rome and Valencia estimated that heat waves resulted in 1,280 excess deaths between 1990 and 2004 (D'Ippoliti *et al.* 2010). In England and Wales, a heatwave between the 30th July and the 3rd August 1995 resulted in 768 excess deaths (Rooney *et al.* 1998). In the United States (Ostro *et al.* 2009) estimated that a heat wave between the 15th and the 26th July 2006 resulted in 188 to 243 excess deaths depending on the model and on the reference period. An excess mortality of 692 excess deaths were estimated during the 1995 heat wave in Chicago (Kaiser *et al.* 2007). In Shanghai (China), (Huang *et al.* 2010) observed 258 excess deaths between the 19 July and the 6th August 2003, when the maximum temperatures were above 35°C during several consecutive days.

A most tragic illustration of the impacts of heat wave is the 2003 heat wave in France. Summer 2003 was the warmest experienced in France since the 50s, with minimum temperatures being 3.5°C above the mean for the period 1950-1980. The heat period started around the 15th July, and a heat wave of exceptional intensity occurred between the 1st and the 15th August 2003. The increase in temperatures was steep during the first days of August, and the warmest temperatures were recorded in Paris between the 11th and the 12th August. The main impact of the 2003 heat wave occurred on a brief period, with a significant increase of the mortality on the 4th August, a rapid and regular increase until the 12th, followed by a progressive diminution. Mortality reached its usual level on the 19th August. In Paris, 80% of the heat-wave related deaths occurred after the 11th of August. An additional heat wave effect was also added to the classical temperature effect, with a daily relative risk¹ of mortality increasing at the beginning of August 2003, reaching a peak around the 12th August, and then going back to usual levels around 20th August 2003 (Le Tertre *et al.* 2006).



Figure 3.1. Daily number of deaths and maximum temperature during the Chicago heat wave: June 22, 1995–August 10, 1995 (Kaiser *et al.* 2007).

Geographical differences were observed, with clear heat waves observed in Ile de France, Auvergne, Centre, Bourgogne and Franche-Comté. Coastal regions in the South of France experienced sustained warm temperatures but with less of a heat wave. Coastal regions in the West experienced warm temperatures alternating with decreasing temperatures. The impact of 2003 was found to be extremely variable between cities, with the highest burden

¹ A relative risk is the risk of developing a disease relative to the exposure

paid in Dijon, Le Mans, Lyon and especially Paris (+142% excess mortality for summer 2003) (Vandentorren *et al.* 2004).

In Europe, the mortality response during heat waves showed a great geographical heterogeneity, and the impacts were larger for long heat waves, or heat waves lasting several days and characterized by extreme temperatures (D'Ippoliti *et al.* 2010). This is illustrated in Figure 3.2, where the increase in daily mortality is presented for 9 European cities, and for different types of heat waves.



Figure 3.2. City-specific and pooled estimates of the effect of heat-waves on daily mortality (% increase and 90% CI) by duration and intensity within the EuroHeat project (D'Ippoliti *et al.* 2010)
An interesting exception of the health impacts of heat waves concerns Australia, where reported mortality impacts are less intense. In Brisbane (Tong *et al.* 2010a)estimated that a heat wave between the 7th April 2004 and the 26th February 2004 resulted in 75 excess deaths, with no later harvesting effect observed, while (Nitschke *et al.* 2007) concluded that 31 heat waves in Adelaide between 1993 and 2003 resulted in no observable mortality impact. By contrast, in Brisbane, an increase in mortality was associated with a brutal drop of temperature between two days (Guo *et al.* 2011).

One can make the assumption that the health effects of single warm days and of heat waves are different. In the first case, heat waves can be considered and analysed as single episodes. The second case is typical of studies using time-series techniques to analysis the relationship between warm temperature and mortality, without differentiating heat wave and non heat wave periods. The literature does not provide clear evidence on whether the approach using heat waves or single warm days is correct or not. An additional "heat wave effect", i.e. that the sustained heat during several days generates an additional burden to the day-by-day temperatures, has been investigated and identified in some studies. An illustration of this is given in Figure 3.3., which represents the daily relative risk of dying because of the heat wave.

For instance, in Paris, a significant additional heat wave effect was observed (Heat wave relative risk>1), while in Marseille, the effect of the heat wave did not differ from the effect of warm days (Heat wave relative risk±1) (Le Tertre *et al.* 2006). Similar effects were observed in 1995 in Chicago (Kaiser *et al.* 2007), 2003 in Germany (Hertel *et al.* 2009).

When (Gasparrini and Armstrong, 2011) investigated the combined effect of temperatures and heat wave effects study 108 US communities, they concluded that most of the excess risk observed during heat waves was comparable to the independent effects of individual days with the same temperatures. A small specific heat wave effect was observed only when the heat waves lasted more than 4 days (Figure 3.4).



Figure 3.3. Daily relative risks of death for each city due to an additional heat wave effect during the 2003 heat wave (22nd July to 2nd September 2003) in nine French cities, with dotted vertical line located on day with the highest risk (Le Tertre *et al.* 2006).



Figure 3.4. Average wave effect of consecutive heat-wave days (greater than or equal to 97th percentile), as estimated by quadratic spline (continuous line) with 95% CI (gray area), and by a step function (dashed line) (Gasparrini and Armstrong, 2011).

3.2.3. Heat wave impacts on morbidity

Although there are many studies evaluating the mortality during heat waves, or the relationship between temperature and mortality without focusing on heat waves, few studies evaluate the impacts of warm days on morbidity.

As a general pattern, in Europe, it seems that a high impact on mortality is associated with a low impact of morbidity, and vice-versa. For instance, in the United Kingdom the study of the 2003 heat wave has shown that many people died before getting to the hospitals (Kovats and Hajat, 2008). Analysis of former heat waves in England confirms the same

pattern (Kovats *et al.* 2004). However, in Italy, a study of five heat waves between 2002 and 2003 in the Veneto region found that heat wave duration increased the risk of hospital admissions for heat diseases and respiratory diseases by 16% and 5% each additional day of heat (people >74). At least four consecutive days were needed to observe an impact (Mastrangelo *et al.* 2007).

On the opposite, in Adelaide, during heat waves total hospital admissions increased by 7% [- 1%:16%]. Total mental health admissions increased by 7% [1%-13%], and total renal admissions by 13% [3%-25%], in all age groups. However, total mortality, disease- and age-specific mortality did not increase (apart from mental health-related mortality in people aged 65-74 years) and significant decreases were even observed in cardiovascular-related mortality (Nitschke *et al.* 2007). Similar results were observed during the 1995 heat wave in Chicago. The excess mortality was relatively low, while there was a significant increase in hospital admissions (11%), mainly (59%) for dehydration, heat stroke, and heat exhaustion. Acute renal failure was also significantly elevated. Diabetes (30%) and renal diseases (52%) were frequently encountered as co-morbid conditions (Semenza *et al.* 1999).

A possible explanation is that populations aware of the negative impacts of heat are able to identify adverse situations early enough to search for medical help, resulting in an increase of hospital admissions and a decrease of the global mortality burden. However, this hypothesis has not been investigated yet.

3.3. Factors influencing the mortality and morbidity response to heat exposure

3.3.1. Air pollution

Several studies have investigated the interactions between acute effects of air pollution (ozone and particular matter) and temperature, as both have been associated with changes in daily mortality. For instance, a study of the July 1987 heat wave in Greece showed that the number of excess deaths in Athens (+ 97%) was superior to the number of death recorded in other urban areas (+ 33%) and in rural areas (+27%). This was partly explained by an interaction between high levels of air pollution and temperature (Katsouyanni *et al.* 1993). During the 1995 heat wave in UK, it was estimated that air pollution accounted for about 38% of the summer excess mortality in England and Wales, and up to 62% in London (Rooney *et al.* 1998). However, another study claims that the pollutants played little part in excess mortality associated with hot weather in UK (Keatinge and Donaldson, 2006).

Ozone was found to significantly modify the temperature – cardiovascular mortality relationship across US regions. For a mean increase of 10°C, the lowest and the highest ozone levels were associated with an increase mortality of 1.17% and 8.31% the same day (Ren *et al.* 2008). During summer 2003 in France, the excess risk of death was significant (1.01%; 95% confidence interval, 0.58-1.44) for an increase of 10 μ g/m³ in O₃ level. Between the 3rd and the 17th August 2003, the excess risk of deaths linked to O₃ and temperatures together ranged from 10.6% in Le Havre to 174.7% in Paris. When they

compared the relative contributions of O_3 and temperature to this joint excess risk, the contribution of O_3 varied according to the city, ranging from 2.5% in Bordeaux to 85.3% in Toulouse. Heterogeneity was observed among the nine cities not only for the joint effect of O_3 and temperatures, but also for the relative contribution of each factor. (Filleul *et al.* 2006).

The possibility of an interaction between daily particulate air pollution and daily mean temperature should also be considered (Roberts, 2004). A few epidemiological studies have examined whether there was an interactive effect between temperature and ambient particulate matter on cardio respiratory morbidity and mortality, but the results were inconsistent. Results in Brisbane show that there existed a statistically significant interaction between PM₁₀ and temperature on most health outcomes at various lags. PM₁₀ exhibited more adverse health effects on warm days than cold days. (Ren and Tong, 2006). In Australia, it was showed that PM₁₀ statistically significantly modified the effects of temperature on respiratory and cardiovascular hospital admissions, non-external mortality, and cardiovascular mortality at different lags. The enhanced adverse temperature effects were found at higher levels of PM₁₀, but no clear evidence emerged for interactive effects on respiratory and cardiovascular emergency visits (Ren *et al.* 2006).

3.3.2. Influence of the underlying climate

As discussed above, heat-related mortality has been observed everywhere in the world. Published estimates of the impact of mean temperature on mortality ranged from no evident heat effect in Dublin (Ireland), Dallas and Charlotte (USA), and Busan (South Korea), to a 12.3% (95% CI 5.7 to 19.4) increase in mortality per 1°C increase in high temperature in Beirut (Lebanon) and 18.8% (13.0 to 25.0) in Monterrey (Mexico), with both cities having correspondingly high heat thresholds (Hajat and Kosatky, 2010).

All studies comparing the heat mortality across different cities within a country found that, as a general rule, the threshold for heat-related mortality is higher in warmer cities, as illustrated by Figure 3.5. Cities with milder climate are likely to experience larger health impacts during heat waves (Medina-Ramon and Schwartz, 2007). This influence of the underlying climate is most probably driven by the local social and cultural adaptation to heat, residents of warm cities adopting more adapted housing and behaviours.



Figure 3.5. Relationship between heat threshold and latitude for studies measuring the relationship between mortality and mean temperature, presented in (Hajat and Kosatky, 2010) (latitudes <0 represent the Southern Hemisphere)

3.3.3. Individual and social risk factors

Dependency is a major risk factor during heat waves, as it reduces the capacity to protect oneself by adopting simple and appropriate behaviours (Belmin *et al.* 2007). In Chicago, during the 1995 heat wave, being confined to bed, living alone or lacking of social contacts were risk factors for mortality (Semenza *et al.* 1996) (Naughton *et al.* 2002). In France, during the 2003 heat waves, appropriate behaviours such as dressing lightly and use of cooling techniques and devices were protective factors (Vandentorren *et al.* 2006).

Some studies found no difference by gender (Basu and Ostro, 2008; Ellis and Nelson, 1978), or higher for women depending on the location (Bell *et al.* 2008; Hajat *et al.* 2007; Ishigami *et al.* 2008; Stafoggia *et al.* 2006; Vaneckova *et al.* 2008).

Housing characteristics associated with death were lack of thermal insulation and sleeping on the top floor, right under the roof. The temperature around the building was a major risk factor (Vandentorren *et al.* 2006).

The urban heat island (UHI) has been proven to be a main risk factor during heat waves. UHI characterised the fact that ambient and surface temperatures are usually several degrees higher in urban area than in the surrounding rural areas (Oke, 1982). The main causes of UHI are the replacement of soil and vegetation by impervious surfaces such as concrete and asphalt, urban structure such as tall buildings and streets that change the radiative fluxes, and anthropogenic heat release (Arnfield, 2003). In Paris a temperature indice derived from the Landsat data showed that an increase in 1°C in the near environment of residence was associated with an odd ratio² of mortality of 1.8 [CI 95% 1.27:2.60] (Vandentorren *et al.* 2006). NOAA/AVHRR data were used to refined the temperature indice, using the average minimum temperature observed on the day of death and the six preceeding day. An increase of 0.41°C in temperature was then associated with an odd-ratio of 2.24 [IC95% 1,03-4,87] (Dousset *et al.* 2011). These results were confirmed by other studies: surface temperatures were also associated with an increase in mortality in Philadelphia during the 1993 heat wave (Johnson *et al.* 2009). In Phoenix, the urban heat island was associated with an increased number of heat-related emergency calls (Silva *et al.* 2010). In Berlin, the UHI was assessed though the density of built areas, and a high correlation was found during heat waves between mortality rates and the proportion of land covered by sealed surfaces (Gabriel and Endlicher, 2011). Similar results were found in Shangai (Tan *et al.* 2010).

Finally, deprivation may increase the mortality risk during a heat wave. During the 2003 heat wave in Paris, a significant relationship was found between a deprivation index, derived from the median household income, the percentage high school graduates in the population aged 15 years and older, the percentage bluecollar workers in the active population, and the unemployment rate, a heat exposure indicator, and the excess mortality. Excess mortality rates were two-fold higher in the most deprived areas (deprivation index above the 80th percentile of the deprivation index distribution), compared to the least deprived areas (deprivation index below the 20th percentile of the deprivation index

 $^{^{2}}$ An odd ratio describes the strength of the relationship between two variables. The odd ratio is considered a good estimate of the relative risk when the disease is rare.

distribution) (Rey *et al.* 2009). A large part of the younger victims were homeless or socially and economically isolated people (Collet, 2005). Deprivation was also associated with a higher mortality risk in Memphis (Applegate *et al.* 1981), St Louis and Kansas City (Jones *et al.* 1982) and in Texas (Greenberg *et al.* 1983). However, some studies found no difference by high school graduation (Basu and Ostro, 2008) and social deprivation (Hajat *et al.* 2007) (Gouveia *et al.* 2003).

3.3.4. Demography

The impact of a heat wave depends on the number of vulnerable available (risk pool). Several factors can modify this risk pool; mortality observed in preceding events, exposure to air pollution, influenza epidemics... In the literature, it has been documented that the mortality during winter modified the temperature effect, i.e. that a high winter mortality has been associated with a low summer mortality (Rocklov *et al.* 2009). The hypothesis, as schematized in Figure 3.6, is that the pool of people at risk is modified during winter, either because more people are dying within this pool, or because people of the general population are frailed and therefore enter the pool of people at risk.



Figure 3.6. Factors influencing the pool of vulnerable people

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3.4. The potential impacts of climate change on the heat wave – mortality response

3.4.1. A framework on heat waves, climate change and human health

Based on the literature review, Figure 3.8 proposes a schematisation of the different aspects that intervene in the health impacts of heat waves. Areas where climate change may interact were selected qualitatively. Among the selected risk factors, some are influencing the exposure (e.g. urban planning, housing), and some are increasing the risk for a similar exposure (e.g. the underlying health conditions). These risks factors are likely to interact both on exposure and on the underlying risk, for instance impaired health conditions may modify the exposing behaviours, and increase the risks for a given exposure. The picture is also highly simplified, since it does not show the links between demography and individual and social behaviours, or how air pollution effects are also modified by these parameters for instance.

3.4.2. Heat waves in a changing climate

The most documented impact of climate change on heat waves is the increase in the frequency and intensity of heat waves (Solomon *et al.* 2007). Between 1961 and 2005, the climatic trends identified in Ireland were largely consistent with global trends (Figure 3.7.): six of the ten warmest years in Ireland, since records began, have occurred since 1990. Mean annual temperature records showed a warming evident in two steps, 1910 to the mid-1940s, and 1980 to 2004. The warming in the latter period occurred at a much greater rate

than the global temperature increase. Increases in annual and seasonal mean, maximum and minimum temperatures are observed in nearly all stations. For the 1961 – 2005 periods, minimum temperatures were increasing at a faster rate than maximum temperatures except in winter (Sweeney *et al.* 2002). The number of episodes defined as days when, for at least 6 consecutive days, the maximum temperature is at least 5°C greater than the 1961-1990 climatological mean value, has increased at a number of stations (McElwain and Sweeney, 2007).



Figure 3.7. Temperature anomaly observed at the global level and for Ireland (Sweeney *et al.* 2002)



Figure 3.8. Impacts of climate and climate change on the different determinants for heat exposure

3.4.3. Determinants investigated in this PhD

Data availability and existing epidemiological tools limits our abilities to document or test all the blocks presented in Figure 3.7. In the frame of this PhD, only the blocks detailed in Figure 3.9 may be investigated for Ireland, in order to orientate adaptation and prevention.



Figure 3.9. Impacts of climate and climate change on the different determinants for heat exposure investigated in this work

CHAPTER IV. HEALTH AND ENVIRONMENTAL DATA

4.1. Health data

4.1.2. Sources

Individual mortality data were obtained from the Central Statistics Office of Ireland for the period 1981-2003. They included date of death, sex, age, primary cause of death, location of death, marital status, occupation, and reporting in living or not in institution.

The main causes of deaths identified in the literature were considered, and mortality data were sorted by causes of death: total, cardiovascular (ICD 9: 390-459) and respiratory causes (ICD9: 460-519).

Although marital status, occupation and living or not in institution have been found to be risk factors during heat waves, this information was not used in this work, due to concerns about the quality of the recording.

4.1.3. Influenza Episodes

Influenza episodes were defined as at least two consecutive weeks in which the percent deaths in Ireland from influenza and pneumonia were above the 95th percentile of expected. This approach has successfully been used before (Dockery *et al.* 2011).

4.2. Meteorological data

4.2.1. Possible indicators

To study the temperature mortality relationship, the temperature indicator must be available on a long time series, be a good proxy of exposure, and if possible have a documented physiological impact. Minimum, maximum and mean temperatures can be used as such. Daily temperatures can be used to define secondary indicators, such as the "heat load" i.e. the difference between the temperature and a threshold summed over several days. (Diaz *et al.* 2006). Day to day differences can also be investigated: $DT=T_j-T_{j-1}$ (Guo *et al.* 2011). In addition to temperatures, several methods have investigated "mixed" indicators, derived by combining various parameters such as temperature, humidity or wind speed.

The wind chill equivalent temperature (Court, 1948; Dixon and Prior, 2010) is the air temperature of a reference environment, in which the same cooling rate is acting on a plastic cylinder as in the actual environment. The new Wind Chill Factor assesses the cooling effect on the basis of a cylinder with the dimensions of a head, where the inner temperature is kept at 37 °C (Bluestein and Zecher, 1999). The initial formula was written as;

$$WCI = (10\sqrt{V} - V + 10.5) * (33 - T) (Equation 4. 1)$$

Where:

Another option is to model the heat-exchanges of the human body. The apparent temperature (AT) is an approximation of the value provided by a mathematical model of heat balance in the human body. The model estimates the heat exchange of outdoor air and a standardised person walking at 5 km per hours, with a metabolic rate of 2.7 Met. It can include the effects of temperature, humidity, wind-speed and radiation. Simplified formulas are proposed, as an approximation of the mathematical model (Steadman, 1984):

Version including the effects of temperature, humidity, and wind:

$$AT = Ta + 0.33 \times e - 0.70 \times ws - 4.00$$
 (Equation 4. 2)

Version including the effects of temperature, humidity, wind, and radiation:

$$AT = Ta + 0.348 \times e - 0.70 \times ws + 0.70 \times Q/(ws + 10) - 4.25 (Equation 4.3)$$

where: Ta = dry bulb temperature (°C)

e= water pressure (hPa), can be calculated from the temperature and relative humidity (%)

ws=wind speed (m/s) at an elevation of 10 meters

Q= net radiation absorded per unit are of body surface (W/m^2)

The Humidex is derived from a similar approach, but uses the dew point rather than the relative humidity. At equal temperature and equal relative humidity, the humidex is higher than the AT (Masterton and Richardson, 1979).

humidex = (air temperature) + h (Equation 4. 4)

$$h = (0.5555)*(e - 10.0)$$

 $e = 6.11 * \exp [5417.7530 * ((1/273.16) - (1/dewpoint in kelvins))]$

The perceived temperature was developed by the German meteorological office to take into account all mechanisms of heat exchange. The objective is to have an indicator that can adapt to any climate. It is a model of the heat exchange of a reference male human body aged 35 years, 1,75 m tall, weighing 75 kg. His work performance is 172,5 W which corresponds to a metabolic rate of 2,3 Met, and to walking ca. 4 km/h on flat ground in an environment characterised by the wind speed, the air temperature, the humidity. Summer clothes (introduced in the model has a 0,50 clothes) correspond to a pair of light long trousers, a shortsleeved shirt and sandals. Winter clothes (1,75 clothes) correspond to a suit of woollen material, a tie, a winter coat and warm, solid shoes. Physiological Equivalent Temperature (PET) is based on a complete heat budget model of the human being, adapted to an office indoor environment. The assessment is made for a standard male, aged 35 years, 1,75 m tall, weighing 75 kg. He wears unchangeable clothes with an insulation value of clo = 0,9, clothes normally worn at the office and he has a work performance of 80 W. Being developed for a healthy young male, the PT and PET may not be appropriate to represent the elderly or physically-disable people (Jendritzky *et al.* 2010).

These indicators call for several comments. The first one is that they were initially developed to quantify a feeling. It is indeed true that we feel warmer when a high humidity rate is associated with a high temperature. With a high humidity, the evaporation rate of water is reduced, and the sweating (evacuation of heat) is reduced. However, it is doubtful that the use of these indicators brings more information than the analysis of the temperature and humidity as separate variables. It was not possible to find in the literature detailed

document explaining how the simplified equations were derived from the mathematical heat exchange models.

Work done by (Barnett *et al.* 2010) comparing the predictive abilities of different temperature measures related to mortality found large differences in the best temperature measures between age groups, seasons and cities. However, there was not one temperature measure that was superior to the others, and the strong correlation between different measures of temperature resulted in similar predictive abilities on average. Therefore, in this work, temperatures were used without developing specific indicators.

4.2.2. Sources

Daily meteorological data from 1980 to 2006 were obtained from Met Éireann, the Irish National Meteorological Service. Eleven stations were selected to cover the whole country (Figure 4.1). These were chosen as they gave a good coverage with respect to the main population centres. Daily mean minimum and maximum temperatures, humidity, pressure and wind speed, were collected.

4.3. Air pollution

Air pollution data were not available for the whole periods and locations included in the analysis.

4.4. Geographical level of analysis

The lack of postal codes in the mortality records limited the ability to perform analyses of the mortality data with a good geographical resolution. Often county level was the best available spatial resolution, and the location of death was detailed for the main population centres only. That limited the possibility of describing the exposure to temperature at the location of deaths, except in the main population centres. However, there was not enough statistical power to perform the analysis at the city level, except in Dublin, due to the small numbers of daily deaths. Therefore, larger geographical aggregations were considered. The first option was to compare inland vs seaside areas, which would made sense from a meteorological point of view. However, considering the availability of the mortality data, the choice was made to create a urban and a rural indicator. Urban areas which were easily identifiable (Dublin, Cork, Drogheda, Arklow, Dundalk, Galway, Limerick, Waterford and Wexford) were used to represent the urban population, while the remaining areas were classified as rural. Temperature indicators were aggregated at a similar spatial resolution, to obtain a proxy of urban and rural heat exposure. Correspondences are described in Table 4.

A comparison of the distributions of the temperatures per seasons for different geographical aggregations is presented in Appendix 1.

Humidity, Wind speed and barometric pressure have a more local meaning, and a higher geographical heterogeneity. Thus, it was decided not to aggregate these indicators, to limit the uncertainties introduced by these indicators.

Indicator	Mortality data	Meteorological		
		stations		
Rural	Location of death $= 0$	Kilkenny, Mullingar,		
		Belmullet, Birr and		
		Valentia		
Urban	Location of death= Dublin, Cork, Drogheda, Arklow,	Casement, Rosslare,		
	Dundalk, Galway, Limerick, and Waterford	Cork, Dublin and		
		Shannon		

Table 4. 1. Data used to study the urban and rural locations



Figure 4.1. Location of the main population centres and of the meteorological stations

4.5. Descriptive analysis

4.5.1. Mortality

Between 1981 and 2003, 691 394 deaths were recorded. People above 74 years old represented more than 50% of the total mortality (0-64: 19.5%, 64-74: 25%, >74: 56%). The main causes of mortality were classified as cardiovascular (47.5%). Respiratory causes represented 15% of the total mortality.

The rural population represented more than 75% of the total mortality, while Dublin represented 15% of the urban mortality (Table 4.2). The distribution of age groups by location is similar, people older than >74 representing 51% of the urban mortality and 55% of the rural mortality.

The mean daily mortality by causes and ages are presented in Table 4.3.

Ireland exhibits an extremely strong seasonal pattern in mortality, with significantly higher wintertime mortality compared to summer, with some of the most pronounced winter peaks corresponding with the influenza epidemics period (Figure 4.2). In fact the excess winter mortality is one of the biggest in the developed world (Healy, 2003). Addressing the drivers of winter-related mortality is therefore a public health priority.

Γ	Number of deaths between	
Location	1981 and 2003	%
Arklow	443	0.06
Waterford	2 135	0.31
Drogheda	3 454	0.50
Galway	4 555	0.66
Dundalk	4 718	0.68
Limerick	6 381	0.92
Cork	25 317	3.66
Dublin	106 796	15.45
Total urban	153 799	22.24
Total rural		
(no location specified)	537 595	77.76

Table 4.2. Total number of deaths between 1981 and 2003 by location

Overall there has been a decline in mortality over the study period, with the most pronounced decreases in mortality from cardiovascular causes. This may be attributed to improvements in life style and medical treatments. There were also substantial shifts in the age distribution of the Irish population over the last 25 years. The number of children (0-14 years of age) has declined, while the number of middle-aged adults (30-59 years) has increased. The number of people 75 years and older remains a small fraction of the total population in all census years between 1981 and 2006, but there was a substantial increase (59%) in the oldest age group from 1981 to 2003 (Dockery *et al.* 2011).



Figure 4.2. Daily total, cardiovascular, respiratory mortality and influenza epidemics in Ireland between 1981 and 2003.

Table 4.3. Mean [[min:max]	daily nu	mber of	deaths	between	1981	and 2	2003	by
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Rural areas						
	Whole year	Summer				
Total mortality	63.9 [31:143]	56.8 [33:92]				
Total mortality >75	36.4 [12:90]	31.3 [12:51]				
Cardiovascular mortality	30.9 [8:75]	27.1 [8:50]				
Respiratory mortality	9.6 [0:45]	7.3 [0:18]				
Urban areas						
	Whole year	Summer				
Total mortality	18.8 [4:46]	16.6 [4:32]				
Total mortality >75	9.8 [0:29]	8.5 [1:20]				
Cardiovascular mortality	8.4 [0:24]	7.4 [0:20]				
Respiratory mortality	2.7 [0:17]	1.9 [0:8]				
	Dublin					
	Whole year	Summer				
Total mortality	12.5 [1:38]	11 [2:24]				
Total mortality >75	6.5 [0:21]	5.6 [0:16]				
Cardiovascular mortality	5.5 [0:18]	4.8 [0:14]				
Respiratory mortality	1.8 [0:13]	1.3 [0:7]				

location, during the whole year and during summer

4.5.2. Temperatures

The dominant influence on Ireland's climate is the Atlantic Ocean, resulting in a moderate climate, with air temperatures inland normally reaching 18 to 20°C during summer days, and about 8°C during wintertime. The highest air temperature recorded in Ireland was + 33.3°C at Kilkenny Castle 26thJune 1887. The record maximum during the 20th century was +32.5°C at Boora, Co. Offaly on 29th June 1976. The lowest air temperature was -19.1°C measured at Markree Castle, Co. Sligo on 16th January 1881 while the lowest observed 20th century air temperature was -18.8°C recorded at Lullymore, Co. Kildare on 2nd January 1979 (source Éireann).

There was a strong homogeneity of temperature across the meteorological stations (Table 4.4), with mean minimum temperature varying between 5.5 and 8.1°C and mean maximum temperatures varying between 12.9 and 14.0°C. A comparison between inland and coastal stations, eastern and western stations, northern and southern stations, or rural and urban stations confirms that there are no strong differences across the country (see Appendix 1).

The increasing trend of temperatures, minimum and maximum, and of relative humidity, was confirmed by the data from the study period. This general trend was observed in all stations, with varying intensity depending on the parameter and the season. Globally, the increase was more important for maximum temperature during winter and minimum temperature during summer. As a result, the thermal amplitude between day and night was decreasing.

In Dublin, the there was no increasing trends in minimum temperature during summer and winter. Figure 4.3 shows the temperature anomaly each year between the monthly mean temperature and the mean average over the period 1980-2006; 1983, 1995, 2003, 2005 and 2006 were the five warmest years.

The trends per stations are presented in Appendix 2.

	Belmullet	Birr	Casement	Clones	Cork	Dublin	Kilkenny	Mullingar	Rosslare	Shannon	Valentia
Belmullet		0,93	0,90	0,93	0,89	0,89	0,91	0,93	0,86	0,94	0,93
Birr	0,89		0,97	0,97	0,95	0,96	0,98	0,99	0,91	0,98	0,94
Casement	0,87	0,95		0,96	0,93	0,99	0,97	0,98	0,93	0,95	0,91
Clones	0,91	0,95	0,94		0,92	0,95	0,96	0,98	0,89	0,95	0,91
Cork	0,88	0,93	0,90	0,91		0,93	0,96	0,94	0,93	0,95	0,93
Dublin	0,86	0,93	0,96	0,94	0,90		0,96	0,97	0,94	0,94	0,89
Kilkenny	0,83	0,94	0,92	0,91	0,91	0,92		0,97	0,93	0,97	0,93
Mullingar	0,88	0,96	0,96	0,96	0,91	0,95	0,94		0,91	0,97	0,92
Roslare	0,85	0,89	0,90	0,90	0,94	0,91	0,90	0,90		0,89	0,87
Shannon	0,91	0,96	0,92	0,94	0,95	0,92	0,92	0,94	0,91		0,95
Valentia	0,88	0,90	0,86	0,86	0,92	0,85	0,85	0,87	0,86	0,92	

 Table 4.4. Correlation coefficient between stations for minimum (grey) and maximum temperatures





Deviation to the monthly mean temperature average between 1980 and 2006 (°C)

Figure 4.3. Difference (°C) between the monthly mean temperature per year and a reference temperature define as the monthly mean temperatures average between 1980 and 2006.

Relative humidity was high during all seasons and for all stations with averages above 80%. However, high humidity was barely associated with high temperatures during summertime, not creating the condition of humid heat in Ireland.

4.5.4. Wind

The prevailing wind direction varied between South and West (Figure 4. 4).



Figure 4. 4. Wind direction (red=% of calm days) (source: Met Éireann

http://www.met.ie)

Temperature, humidity and wind speed characteristics per station are detailed in Table 4.5.

	Minimum temperature	Maximum temperature	Relative Humidity (%)	Wind speed
	(°C)	(°C)		(m/s)
Belmullet	7.3 [-8.1:17.3]	13.0 [-1.1:29.9]	84.0 [54.3:99.3]	6.7 [0.0:23.0]
Birr	5.9 [-14.6:18.9]	13.5 [-3.5:30.8]	83.6 [50.0:100.0]	3.4 [0.0:13.3]
Casement	5.9 [-12.1:18.3]	13.4 [-2.9:31.0]	82.8 [47.0:100.0]	5.6 [0.1:20.6]
Clones	5.8 [-12.4:18.0]	12.9 [-6.4:30.5]	83.9 [49.6:99.9]	4.0 [0.0:15.6]
Cork	6.8 [-8.0:19.0]	12.9 [-4.3:28.7]	85.4 [50.7:100.0]	5.5 [0.8:18.1]
Dublin	6.3 [-7.9:18.4]	13.3 [-2.5:28.7]	82.2 [53.3:100.0]	5.2 [0.2:18.3]
Kilkenny	5.7 [-13.4:18.0]	13.9 [-3.6:31.4]	82.3 [47.9:100.0]	3.6 [0.0:14.5]
Mullingar	5.5 [-12.4:17.7]	12.9 [-3.4:29.7]	85.1 [53.8:99.6]	3.9 [0.0:14.4]
Rosslare	8.1 [-4.4:17.6]	13.1[-1.5:26.2]	84.0 [52.7:99.6]	5.7 [0.3:19.3]
Shannon	7.4 [-8.2:19.0]	14.0 [-1.8:30.6]	81.6 [49.7:100.0]	4.7 [0.0:17.2]
Valentia	8.0 [-6.8:18.4]	13.7 [-1.5:28.4]	83.1 [37.6:100.0]	5.2 [0.2:16.8]

 Table 4. 5. Temperature, Humidity and Wind speed per station mean [min:max]

CHAPTER V. HEALTH IMPACTS OF HEAT WAVES IN IRELAND BETWEEN 1981 AND 2003

5.1. Selection of heat waves

5.1.1. Definition of heat waves in Ireland

As stated in the introduction, there is no official definition of a heat wave. Therefore, the first step of this work was to propose a definition for Ireland. The approach chosen was the threshold approach presented by (Meehl and Tebaldi, 2004) was used, and several thresholds were tested:

- At least two consecutive days with minimum and maximum temperatures >90th percentile of the monthly distribution
- At least one day with minimum and maximum temperatures above the 95th percentile of the monthly distribution
- At least one day with minimum and maximum temperatures 5°C above the monthly mean of the minimum and maximum temperatures

The thresholds corresponding to these three definitions are reported in Table 5.1. Differences of 1 or 3°C were observed, especially for maximum temperatures. The thresholds may seem low, especially for minimum temperatures. However, as they are based on high percentiles of the temperatures distribution, they represent unusual events for the Irish population, and therefore may be associated with a health impact.

Considering the low thresholds on minimum temperatures, one could make the assumption that this parameter is not relevant in Ireland, and that heat waves should be defined on maximum temperatures only. However, considering that night-time temperatures are believed to be a major risk factor during a heat wave, it was decided to keep minimum temperatures in the definition. Indeed, the simultaneous use of minimum and maximum temperatures dramatically reduced the number of heat waves compared to the use of maximum temperature only. For instance, in July 1983, using the maximum temperatures only would double the number of heat waves days, as illustrated in Figure 5.1. In that case, some heat wave days with maximum temperatures exceeding 25°C could be associated with minimum temperatures below 10°C, which cannot be considered as a heat burden.



Figure 5.1. Number of heat wave days detected using "two consecutive days of maximum and minimum temperature >90th percentile (black)" or "two consecutive days of maximum temperature >90th percentile" (grey).

		Jun	e	July		August		
Station	Threshold	Tmax	Tmin	Tmax	Tmin	Tmax	Tmin	
Belmulett	Mean +5°C	21	15	23	17	23	17	
	P90	19	12	21	14	21	15	
	P95	21	13	23	15	23	15	
Kilkenny	Mean +5°C	23	14	25	16	25	16	
	P90	22	12	25	14	24	15	
	P95	24	13	26	15	25	15	
Mullingar	Mean +5°C	22	14	24	16	24	15	
	P90	21	12	23	14	22	14	
	P95	23	13	25	14	24	15	
Birr	Mean +5°C	23	14	25	16	24	16	
	P90	22	12	24	14	23	15	
	P95	23	13	26	15	25	15	
Valentia	Mean +5°C	21	15	23	17	23	17	
	P90	19	13	21	15	21	15	
	P95	21	13	23	15	22	16	
Casement	Mean +5°C	23	14	25	16	25	16	
	P90	22	12	24	14	23	14	
	P95	23	13	25	15	24	15	
Clones	Mean +5°C	22	14	24	16	24	16	
	P90	21	12	24	14	23	14	
	P95	23	13	25	15	24	15	
Cork	Mean +5°C	22	15	24	17	24	17	
	P90	20	12	22	14	22	15	
	P95	21	13	23	15	23	16	
Dublin	Mean +5°C	22	14	24	16	24	16	
	P90	21	12	23	14	22	14	
	P95	22	13	24	15	24	15	
Rosslare	Mean +5°C	21	16	23	18	24	18	
	P90	19	13	21	15	21	15	
	P95	20	14	22	15	22	16	
Shannon	Mean +5°C	23	16	25	18	25	17	
	P90	22	13	24	15	23	15	
	P95	23	14	26	16	25	16	

 Table 5.1. Possible thresholds (°C) for the selection of heat waves

5.1.2. Description of heat waves in Ireland between 1980 and 2006.

Figure 5.2, Figure 5.3 and Figure 5.4 illustrate for each station and each year the number of consecutive days complying with at least two consecutive days with minimum and maximum temperatures >90th percentile of the monthly distribution. Between 1980 and 2006, a limited number of periods are characterized by heat waves observed in almost all stations: July 1983, August 1984, July 1989, June 1995, August 1995, June 2003, August 2003 and July 2006. Only these episodes covering the whole country can be analyzed in this PhD, due to the low geographical resolution of the mortality data.





Number of days exceeding the monthly 90th percentile of the temperature distribution

Figure 5.2. Number of consecutive days with maximum and minimum temperature exceeding the monthly 90th percentile – June.


Number of days exceeding the monthly 90th percentile of the temperature distribution

Figure 5.3. Number of consecutive days with maximum and minimum temperature exceeding the monthly 90th percentile – July.



Number of days exceeding the monthly 90th percentile of the temperature distribution

Figure 5.4. Number of consecutive days with maximum and minimum temperature exceeding the monthly 90th percentile – August.

July 1983 was characterised by a progressive increase of temperatures, with the highest temperatures observed between the 10th and the 15th July. During that period, mean maximum temperatures were of 25.2°C [min-max: 24.3:28.1] in rural areas, and 27.3 [26.4:28.5] in urban areas, while mean minimum temperatures were of 15.2°C [14.1:16.1] in rural areas, and 16.6°C [15.9:17.5] in urban areas. The decrease of temperature was rapid after the 15th, especially in urban areas (Figure 5.5, Figure 5.6).



Figure 5.5. Mean temperatures observed during July 1983 in rural stations



Figure 5.6. Mean temperatures observed during July 1983 in urban stations

The 1984 heat wave in rural areas was characterised by a steep increase in temperatures between the 18th and the 19th of August, and by a moderate intensity. Between the 19th and the 21st August, maximum temperatures were of 24.8 °C [min-max: 24.7: 26.2] and minimum temperatures of 14.8°C [14.1:15.4] (Figure 5.7). In urban areas, a moderate increase in temperature was observed during the same period, mainly driven by the temperatures observed in Shannon (Figure 5.8).



Figure 5.7. Mean temperatures observed during August 1984 in rural stations



Figure 5.8. Mean temperatures observed during August 1984 in urban stations

No clear heat wave shape was observed during July 1989, when temperatures were slightly above usual values for the whole month, with a moderate peak between the 19th and the 21st (Figure 5.9, Figure 5.10).



Figure 5.9. Mean temperatures observed during July 1989 in rural stations



Figure 5.10. Mean temperatures observed during July 1989 in urban stations

Summer 1995 presented an interesting pattern, comparable in rural and urban areas. A first period of heat was observed between the 22nd and the 30th June, with a rapid increase followed by a rapid decrease of temperatures. Temperatures were consistently above the usual values during July and August, with daily peaks (Figure 5.11, Figure 5. 12). The highest values were observed between the 01st and the 04th August: maximum temperatures were of 25.8 °C [min-max: 25.4:26.0] in rural areas and of 25.9°C [24.7:26.6] in urban areas. Minimum temperatures were of 15.6 °C [14.8:16.2] in rural areas and of 16.2°C [15.3:16.8] in urban areas.



Figure 5.11. Mean temperatures observed during summer 1995 in rural stations



Figure 5. 12. Mean temperatures observed during summer 1995 in urban stations

August 2003 was characterised by two short peaks between the 05th and the 07th August and between the 24th and the 26th August. In all cases, the increase and the drop in temperature were steep (Figure 5.13, Figure 5.14). During the first episodes, maximum temperatures were of 25.4°C [22.0:27.2] in rural areas and of 25.6°C [24.4:26.4] in urban areas. Minimum temperatures were of 14.4°C [13.9:14.8] in rural areas and of 15.0°C [14.6:15.4] in urban areas.



Figure 5.13. Mean temperatures observed during summer 2003 in rural stations



Figure 5.14. Mean temperatures observed during summer 2003 in urban stations

Temperatures were above thresholds values in July 2006, but a clear heat wave was not visible in rural areas, and of very moderate intensity in urban areas (Figure 5.15, Figure 5.16).



Figure 5.15. Mean temperatures observed during July 2006 in rural stations



Figure 5.16. Mean temperatures observed during July 2006 in urban stations

In conclusion, Ireland has experienced moderate heat waves between 1980 and 2006, concentrated on four years: 1983, 1984, 1995 and 2003. Temperatures higher than usual were also recorded in 1989 and 2006, but without a clear heat wave shapes. Therefore, the analysis will focus on July 1983, August 1984, summer 1995 and August 2003 in both rural and urban areas. Since the heat wave periods are overlapping across stations, extended heat wave periods were defined, in order to include the first and the last heat waves observed in the stations. This corresponds to the following periods:

- 05-18/07/1983
- 18-31/08/1984
- 22-30/06/1995
- 01-22/08/1995
- 04-12/08/2003
- 22-27/08/2003

In Dublin, the periods were selected based on the temperatures measured at the Dublin station only. They correspond to:

- 10-20/07/1983
- 18-28/08/1984
- 20-30/07/1989
- 23/07 01/08/1990
- 11-21/08/1995
- 05-14/08/2003

5.2. Methods to estimate the mortality impact

5.2.1. Estimation of the excess mortality attributable to heat

The excess mortality was defined as:

Where Yhw is the mortality observed during the heat wave and Yref is the reference mortality. On a first step, Yhw was estimated as the mortality predicted by a model fitting on observed meteorological data, and Yref as the mortality predicted by the same model, fitting on reference meteorological data. The reference meteorological data were computed as the mean of the meteorological data between 1980 and 2003.

The excess mortality was computed for the heat wave periods + 5 days, to account for a possible lagged effect.

The model to estimate the excess mortality was built as a predictive model, i.e. that its objective is to obtain the best prediction of the mortality, and not to interpret the relative effect of each variable introduced in the model. In that case, several models can be tested, and the best one is selected relying on the Akaike criteria (AIC) (Akaike, 1973). AIC is a measure of the goodness of fit of an estimated statistical model, and a low AIC means that less information is lost when using the model (Akaike, 1973). To do so, generalized additive procedure was used (see appendix 3 and 4 for statistical details), where Y is the daily mortality, and X are the explaining variables including time, day of the weeks

influenza epidemics, and relevant meteorological parameters. A Poisson distribution was used for the daily mortality count.

In a generalized additive model, the mean of the mortality Y is related to the explanatory X variable via the smooth functions and a link function (Hastie and Tibshirani, 1990; Wood, 2006). The general model is written as:

$$E(Y) = \alpha + g^{-1} (\sum_{j=1}^{p} f_{j}(X_{j})) (Equation 5.2)$$

The smooth functions fj() can be fitted using parametric or non-parametric means. When using a non-parametric approach, no assumption is made on the shape of the relationship, and the model fit on the observed data. For that fitting, splines functions were used. They divide X into different sections and adjust a polynomial function on each section.

The final equation of the model is therefore:

$$Y_{t} \approx Poisson(\mu_{t})$$
$$\ln(\mu_{t}) = \alpha_{0} + \sum_{j=1}^{q} f_{j}(X_{ij}, \lambda_{j}) + \sum_{i=1}^{p} \beta_{i} X_{i}$$
(Equation 5.3)

Where Y_t is the number of deaths on day t

 X_{tj} are the variables we want to fit with splines, i.e. time, temperatures, humidity

X_i are the constant variables, i.e. influenza, day of the week, holidays

Several models were built, testing the different indicators listed in Table 5.2. All the models were tested for the total mortality, total mortality over 74, cardiovascular and respiratory mortality, in rural areas, urban areas and in Dublin. For each indicator, the model with the lower AIC is considered the best one. However, the AIC cannot be used to select the best indicators (AIC is suitable to compare nested models only)(see the AIC in appendix 5).

For all locations, lag 0 and a cumulated effect for lag 1-7 gave better results when using minimum temperatures only. When using other temperature indicators lag 0 to 7 gives better results, expect for the model using the night-day amplitude. Therefore the models that were selected are presented in Table 5.3. Since they all give very similar results. Results corresponding to the Tmax, Tmin model will be detailed in the following.

Name	Geographical	Parameters	Lag
	coverage		
Tmin	All	Tmin	0
Tmax	All	Tmax	0,1
Tmean	All	Tmean	0,1,2
Tmean24	Dublin	Tmean24	0,1,2,3
Tmax-Tmin	All	Tmax-Tmin	0,1,2,3,4
Tmax, Tmin	All	Tmax and Tmin	0,1,2,3,4,5*
Tmin, Tmax	All	Tmin and Tmax	0,1,2,3,4,5,6*
Tmin and H	Dublin	Tmin and H	0,1,2,3,4,5,6,7
Tmax and H	Dublin	Tmax and H	0,1-7**

Table 5.2. Description of the models tested for the different meteorological parameters

* Tmin and Tmax were combined with the following rules, for n varying between 0 and 3;

- $Tmin_{2n}$, $Tmax_{2*n+1}$

- Tmax_{2n}, Tmin_{2*n+1}

** Humidity was introduced at lag 0 and 1 only.

Name	Parameters	
Tmin	Tmin lag 0 and Tmin lag	
	1-7	
Tmax	Tmax lag0, 1,2,3,4,5,6,7	
Tmean	Tmean lag0, 1,2,3,4,5,6,7	
Tmax-Tmin	Tmax-Tmin lag 0 and lag 1	
Tmax, Tmin	Tmax lag 0,2,4,6 and Tmin	
	lag 1,3,5,7	
Tmin, Tmax	Tmin lag 0,2,4,6 and Tmax	
	lag 1,3,5,7	

Table 5.3. Models selected to compute the excess mortality

5.2.2. Estimation of an additional heat wave effect

The method described by (Le Tertre *et al.* 2006) was used to explore a possible additional heat wave effect during the main heat waves. It consists in adding a penalized cubic regression spline of time which covers a period centred on the heat wave. This period was chosen to well capture the increase of mortality during the heat wave and to allow the analysis of a potential short term mortality displacement. The degree of smoothing of this spline function was chosen to minimize autocorrelation in the residuals. The daily relative risk of the specific effect of the heat wave was computed as the number of expected deaths estimated by the heat wave spline divided by the number of expected deaths estimated in

the absence of a heat wave. The number of deaths in the absence (modelled baseline deaths) of the heat wave was predicted using references values for temperature.

5.2.3. Comparison with previous years

Although the best way to estimate the excess mortality attributed to a heat wave is to model the mortality attributable to heat, it is also interesting to develop a simple indicator that gives a rough estimation of the excess mortality during the heat wave period. This was be done by comparing the mortality observed during the period with the mortality observed for the same period in the preceding years.

To do so, the expected number of deaths was estimated as the number of deaths during the same calendar period as the heat wave, averaged over the 1, 2, 3, 4 or 5 previous years. If another heat wave was observed over one of these periods, it was excluded from the set of reference periods.

A major limitation of this approach is that it cannot determine how much of the excess mortality observed is attributable to heat and not to other confounding factors.

All computations were done using the R software, and the mgcv package of the R software (R Development Core Team, 2004; Wood, 2006), which was specifically developed to perform GAM models.

5.3. Heat wave impacts on mortality

5.3.1. The 1983 heat wave (05-18/07/1983)

5.3.1.1. Comparison with the preceding years

In rural areas, between the 5th and the 23^{rd} July 1983, 138 extra deaths were observed, 56 for people below 75 and 82 for people older than 75 (compared to 1981-1982). This corresponded to a +12% increase in mortality, +10% for people below 75 and +15% for people older than 75. 69 extra deaths (+12%) for cardiovascular causes, and 36 (+30%) extra deaths for respiratory causes were recorded for the same period (Figure 5.17). During the same period, 9 excess deaths were observed in urban areas (+3%) (Figure 5.18). A deficit of mortality was observed in the people below 75 (-10, corresponding to -5%), and an excess of mortality for people above 75 was observed (+20, corresponding to +15%).



Figure 5.17. Daily mortality and temperatures observed in rural areas during the 1983 heat wave



Figure 5.18. Daily mortality and temperatures observed in urban areas during the 1983 heat wave

5.3.1.2. Estimation of the mortality attributable to heat

In rural areas, an excess mortality of +115 [CI 95% 96:137] deaths was estimated between the 5th and the 18th July 1983 (+12%) (Figure 5.19). Most of them occurred in people older than 75: + 65 deaths (+14%). In urban areas, the estimated excess mortality was of 21 [9:33] deaths (Figure 5.20), a majority (76%) occurring in elderly people. During the same period, cardiovascular and respiratory mortality increased in rural areas respectively by +14% and +16%. In urban areas, no increase was estimated for cardiovascular mortality, and a 21% increase was estimated for respiratory mortality (Table 5.4).

These estimates were very consistent with the excess mortality estimated by a simple comparison with the previous year. This indicated that the heat was the main cause of unusual deaths during that period.

	Excess mortality [IC95%]	
	Rural areas	Urban areas
Total mortality	115 [96:137]	21 [9:33]
Total mortality >75	65 [51:79]	16 [7:24]
Cardiovascular mortality	64 [50:79]	0 [0:17]
Respiratory mortality	19 [12:26]	7 [3:11]

 Table 5.4. Excess mortality estimated during the July 1983 heat wave (5-18/07/1983)



Figure 5.19. Excess mortality predicted by the model and temperatures in rural areas during the 1983 heat wave



Figure 5.20. Excess mortality predicted by the model and temperatures in urban areas during the 1983 heat wave

5.3.1.3. Investigation of a heat wave effect during the 1983 heat wave in rural areas

In addition to the usual effect of temperature on mortality, a heat wave effect was observed with significant relative risks (RR) of dying because of an additional heat wave effect observed between the 2nd and the 23th of July (Figure 5.21). The effect was slightly more pronounced for the elderly people. A maximum RR of 1.23 [1.13;1.34] was observed on the 14th of July 1983 for the total mortality. The impact was similar for elderly people. In total 42 excess deaths can be attributed to this specific heat wave effect between the 12th and the 18th July 1983.



Figure 5.21. Daily relative risk for total mortality associated with a heat wave effect

during the 1983 heat wave in rural areas

5.3.2. The 1984 heat wave (18-31/08/1984)

5.3.2.1. Comparison with the preceding years

In rural areas, between the 18^{th} and the 31^{st} August 1984, 39 extra deaths were observed (Figure 5.22), 13 for people below 75 and 26 for people older than 75 (compared to 1983-1981). This corresponded to a +5% increase in mortality, +3% for people below 75 and +7% for people older than 75. 21 extra deaths (+5%) for cardiovascular causes, and 9 (+9%) extra deaths for respiratory causes were recorded for the same period. During the same period, 5 excess deaths were observed in urban areas (+2%) (Figure 5.23). Again, a deficit of mortality was observed in the people below 75 (-17, corresponding to -13%), and a excess of mortality for people above 75 is observed (+22, corresponding to +23%). The number of deaths for cardiovascular and respiratory diseases did not increase during the period.



Figure 5.22. Daily mortality and temperatures in rural areas during the 1984 heat wave



Figure 5.23. Daily mortality and temperatures in urban areas during the 1984 heat wave

5.3.2.2. Estimation of the mortality attributable to heat

An excess mortality of 49 [29:68] deaths between the 18^{th} and the 31^{st} August 1984 was observed for rural areas (+6%) (Figure 5.24). A majority concerned people older than 75 (37 extra deaths +9%). In urban areas, an excess mortality is observed in elderly people only (+7 deaths, +14%). Cardiovascular and respiratory mortality increased in rural areas respectively by +5 and +19%. In urban areas, no increase was observed for cardiovascular and respiratory mortality. In urban areas, there was small increase mortality between the 18^{th} and the 25^{th} , followed by a deficit in mortality associated with a decline in minimum temperatures (Figure 5.25)(Table 5.5).

Again, these estimates were very consistent with the excess mortality estimated above by a simple comparison with the previous year.

Table 5.5. Excess mortality estimated during the August 1984 heat wave (18-31/08/1984)

	Excess mortality [IC95%]	
	Rural areas	Urban areas
Total mortality	49 [29:68]	9 [-2:21]
Total mortality >75	37 [24:51]	17 [9:25]
Cardiovascular mortality	22 [7:36]	0 [-6:10]
Respiratory mortality	20 [14:26]	3 [-1:6]



Figure 5.24. Excess mortality and temperatures in rural areas during the 1984 heat

wave



Figure 5.25. Excess mortality and temperatures in urban areas during the 1984 heat wave

5.3.2.3. Investigation of a heat wave effect during the 1984 heat wave in rural areas

No heat wave effect was observed during the August 1984 heat wave, the relative risk of the heat wave variable being positive and significant for the 25th August only (1.11 [1.00;1.23]).

5.3.3. The first 1995 heat wave (22-30/06/1995)

5.3.3.1. Comparison with the preceding years

In rural areas, between the 22^{nd} and the 30^{th} June 1995, between 44 and 47 extra deaths were observed (+8%), depending on the reference period (Figure 5.26). The majority concerned people older than 75 (between 34 and 41 (+14%)). No deficit mortality was observed in younger people. Between 10 and 13 extra deaths for cardiovascular causes (+5%), and between 14 and 18 extra deaths for respiratory causes (+25%) were recorded for the same period.

During the same period, deficit mortality was observed in urban areas (between -3 and -16 depending on the reference period) (Figure 5.27). The highest deficit was observed for people below 65, while the mortality slightly increased for people aged 65-74 (between -1 and +7). Yet, for all age groups there is a large increase in mortality for respiratory causes (+7, corresponding to +45%).









Figure 5.27. Daily mortality and temperatures in urban areas during the June 1995 heat wave

5.3.3.2. Estimation of the mortality attributable to heat

An excess mortality of 54 [40:68] deaths between the 22^{nd} and the 30^{th} June 1995 was observed for rural areas (+10%) (Figure 5.28). A majority concerned people older than 75 (36 extra deaths +11%). In urban areas, a deficit in mortality is observed in the total and the elderly people only (-10 deaths, -13%) (Figure 5.29). Cardiovascular and respiratory mortality increased in rural areas respectively by +9 and +16%. In urban areas, no increase is observed for respiratory mortality, and a deficit of cardiovascular mortality is observed (Table 5.6).

Again, these estimates were very consistent with the excess mortality estimated above by a simple comparison with the previous year.

	Excess mortality	
	[IC95%]	
	Rural areas	Urban areas
Total mortality	54 [40:68]	-13 [-21:-5]
Total mortality >75	36 [26:47]	-10 [-16:-4]
Cardiovascular mortality	22 [13:31]	-5 [-15:-1]
Respiratory mortality	13 [8:18]	2 [-1:5]

Table 5.6. Excess mortality during June 1995 heat wave



Figure 5.28. Daily mortality and temperatures in rural areas during the June 1995 heat wave



Figure 5.29. Daily mortality and temperatures in urban areas during the June 1995 heat wave

5.3.4. The second 1995 heat wave (01-22/08/1995)

5.3.4.1. Comparison with the preceding years

In rural areas, between -70 and +40 deaths were observed in 1995 depending on the reference period (Figure 5.30). This high variability of results was found for all age groups and all causes of mortality. A similar variability of results was found in the urban areas, with no indication that an excess mortality occurred during consecutive days (Figure 5.31).



Figure 5.30. Daily mortality and temperatures in rural areas during the August 1995 heat wave



Figure 5.31. Daily mortality and temperatures in urban areas during the August 1995 heat wave

5.3.4.2. Estimation of the mortality attributable to heat

No excess mortality was observed in rural or urban areas between the 1st and the 22nd August 1995. A slight increase was observed for respiratory mortality in urban areas only (+17%) (Table 5. 7). In rural areas, a dynamics seems to occurred with a succession of increase in mortality in the first days of August, followed by a mortality deficit, and again a small increase in mortality (Figure 5.32). Such pattern was not observed in urban areas (Figure 5.33).

	Excess mortality [IC95%]	
	Rural areas	Urban areas
Total mortality	9 [-25:43]	-10 [-29:9]
Total mortality >75	-18 [-43:8]	-8 [-21:6]
Cardiovascular mortality	9 [-14:32]	1 [-20:5]
Respiratory mortality	6 [-5:17]	9 [2:15]

Table 5. 7. Excess mortality during August 1995 heat wave



Figure 5.32. Excess mortality and temperatures in rural areas during the August 1995 heat wave


Figure 5.33. Excess mortality and temperatures in urban areas during the August 1995 heat wave

5.3.5. The first 2003 heat wave (04-12/08/2003)

5.3.5.1. Comparison with the preceding years

A deficit mortality was observed during the period in rural areas, for all age groups and all causes. For total mortality, it varied between -48 (-12%) and -28 (-7%) deaths, depending on the reference period. In urban areas, a slight increase was observed in total mortality, between +2 (+1.6%) and +5 (+4%) deaths depending on the reference period. This excess was larger for the youngest age group (0-64): between +4 (+13%) and +7 (+21%) deaths depending on the reference period.

5.3.5.2. Estimation of the mortality attributable to heat

A deficit mortality was observed in rural areas for all age groups and all causes. It corresponded to about 11% of the usual mortality. In urban areas, a small increase was observed in the total mortality (+8%) (Table 5.8).

	Excess	mortality		
	[IC95%]			
	Rural areas	Urban areas		
Total mortality	-45 [-56:-33]	13 [6:19]		
Total mortality >75	-31 [-40:-21]	3 [-2:8]		
Cardiovascular mortality	-27 [-34:-20]	2 [0:10]		
Respiratory mortality	-10 [-15:-6]	-1 [-3:2]		

Table 5.8. Excess mortality during August 2003 first heat wave

5.3.6. The second 2003 heat wave (22-27/08/2003)

5.3.6.1. Comparison with the preceding years

A deficit in mortality was observed during the period in rural areas, for all age groups and all causes (Figure 5.34). For total mortality, it varied between -18 (-6%) and -5 (-2%) deaths, depending on the reference period. In urban areas, a slight increase was observed in total mortality, between +7 (+6%) and +12 (+11%) deaths depending on the reference

period (Figure 5.35). This excess was larger for the youngest age group (0-64): between +4.5 (+17%) and +5 (+19%) deaths depending on the reference period, and (64-75), between +3 (+13%) and +7 (+29%).



Figure 5.34. Daily mortality and temperatures in rural areas during the August 2003 heat wave



Figure 5.35. Daily mortality and temperatures in urban areas during the August 2003 heat wave

5.3.6.2. Estimation of the mortality attributable to heat

A small deficit mortality was observed in mortality in rural areas (Figure 5.36), total mortality and respiratory mortality. An impact of the heat was observed only for cardiovascular mortality. On the opposite, in urban areas, a small increase was observed in total mortality (+9%) (Figure 5.37) and in respiratory mortality, but not in cardiovascular mortality (Table 5.9).

	Excess mortality		
	[IC9	95%]	
	Rural areas	Urban areas	
Total mortality	-7 [-15:1]	10 [5:14]	
Total mortality >75	-5 [-18:14]	-2 [-5:2]	
Cardiovascular mortality	14 [9:19]	0 [-4:2]	
Respiratory mortality	-22 [-25:-19]	5 [3:7]	

Table 5.9. Excess mortality during August 2003 second heat wave



Figure 5.36. Excess mortality and temperatures in rural areas during the August 2003 heat wave



Figure 5.37. Excess mortality and temperatures in urban areas during the August 2003 heat wave

5.3.7. Heat waves impacts in Dublin

In Dublin, the analysis could not be performed on the respiratory mortality, due to the low number of daily deaths. For the other causes, heat waves were never associated with a mortality increase. On the opposite, a deficit in mortality was observed in 1995 (Table 5.9). The comparison with the previous years confirmed the estimates from the model except in 1984 and 2003. During the 1984 heat wave, the mortality was above the mortality observed the previous years (respectively +26, +30, and +5 for the total, total >74 and cardiovascular mortality). In 2003, the mortality was very close to the mortality observed the preceding years.

	Total	Total	Cardiovascular
	mortality	mortality >74	mortality
10-20/07/1983	-9 [-2:-17]	-6 [-9:-3]	-4 [-7:-2]
18-28/08/1984	-2 [-10:6]	6 [0:11]	0 [-5:5]
20-30/07/1989	-9 [-16:-2]	-13 [-18:-8]	2 [-2:7]
23/07-01/08/1990	-4 [-11:3]	-2 [-7:3]	0 [-4:5]
11-21/08/1995	2 [-6:10]	5 [-1:11]	10 [5:15]
05-14/08/2003	-17 [-23:-11]	-12 [-17:-8]	-9 [-13:-6]

 Table 5.10. Excess mortality during heat waves in Dublin [CI 95%]

5.4. Discussion

5.4.1. Limits

This study experiences several limits, the first one being the large geographical scale used to perform the analysis. Although temperature can be considered as relatively homogeneous over the country, daily variations or local peaks may be observed, leading to significant differences in the heat exposure of the population. This is a main source of uncertainty in the project, and the excess mortality may be over or under-estimated depending on the location. A second limitation is the impossibility of controlling for air pollution, especially in urban areas. Air pollution is a factor that may explain the results observed for the 1995 heat waves.

Finally, the results obtained in Dublin and in urban areas should be interpreted with care, as Dublin represents a large part of this indicator. The larger daily number of deaths for the urban indicators gives it a better statistical power than Dublin alone, but on the opposite the geographical aggregation diluted the relevance of the exposure variable. However, in urban areas, the fact that estimates are consistent with the rough estimate of comparing the mortality with the previous year is an indication of robustness.

This study quantified the mortality impacts of the heat waves, without investigating a possible morbidity impact. However, the literature suggests that mortality is the main indicator in most heat waves, especially when the heat-related risks are unknown, so that people cannot identify the symptoms that should lead to search for medical assistance.

Overall, this study provides indication that past heat waves have resulted in a moderate excess mortality, especially in 1983 and 1984. The differences observed between rural and urban areas, more pronounced in the most recent years, may be due to biases and limits of the design, or may be explained by real differences in the vulnerability to heat.

5.4.2. Synthesis

Overall, a total of 250 excess deaths attributable to heat waves was estimated, 197 in rural areas, and 57 in urban areas. The 2003 heat wave was also associated with a deficit in mortality of 52 deaths in rural areas, and the 1995 heat wave was associated with a deficit in heat-related mortality of 23 deaths.

The impacts of heat waves in rural and urban areas are synthesised in Figure 5.38 and Figure 5.39. The 1983 and to a lesser extend the 1984 heat waves were characterised by a significant excess mortality, especially in rural areas. The July 1983 episode is the only one presenting a characteristic heat wave patterns, although with moderate intensity. More recent episodes in 1995 and 2003 are associated with a decreased mortality in rural areas, and a slight increase in urban areas. Reasons for that may be due to the nature of the heat waves (long and intense in 1983, vs short spells in 1995 and 2003), and to an improvement of the health care system.



Figure 5.38. Excess mortality attributed to heat in rural areas



Figure 5.39. Excess mortality attributed to heat in urban areas

5.4.3. Influence of the modelling strategy

The choice of different temperature indicators to model the excess mortality makes little differences in rural or urban areas. The only exception is observed for the 1995 heat waves in urban areas, where using the maximum or the minimum temperature only largely over-estimates the number of heat-related deaths (Appendix 6).

5.4.3. Vulnerabilities of rural and urban areas

This work made the assumption that urban and rural areas may react differently to heat. Indeed, differences in the impacts were observed for the different heat waves, which may due to biases in the characterization of the heat waves, or to real differences in vulnerability. The larger impacts in cardiovascular mortality in rural areas may be linked with farming and outdoor work.

In urban areas, the larger impacts in respiratory mortality are to be interpreted with care due to the low number of cases that limited the statistical power of the analysis. However, especially in 1995, the large differences observed between the models and the comparison with the previous years may indicate the intervention of an additional risk factor, which could be air pollution. Urban areas are also affected by the urban heat island, which increases the heat stress within the city centres, and may exacerbate the burden of the heat wave. Urban areas may also concentrate the most vulnerable people at risk during a heat wave, e.g. the elderly, people with chronic illnesses, or physically impaired, and infants. A low socio-economical status is also a risk factor (homeless people, people with low income, or people socially isolated).

CHAPTER VI. CHARACTERIZATION OF THE TEMPERATURE-MORTALITY RELATIONSHIP DURING SUMMER

The objective of this chapter is to characterize the mortality risks associated with temperatures, considering the whole temperature range, without focusing on heat waves. The first step was to investigate the shape of the relationship between temperature and mortality, and to quantify the risk associated with a temperature increase, for different causes and age groups. On a second step, the lag structure of the temperature response was investigated. Among the various factors that can modify the temperature-mortality relationship, few can be analyzed without access to detailed individual data. Considering the additional uncertainty introduced by the large geographical unit used in this work, the only factor that can be investigated is the impact of the preceding winter mortality.

Finally, possible changes of the relationship over time were investigated, considering 5years period between 1981 and 2003.

6.1. Methods

6.1.1. General model

A generalized additive model was used to link the temperature and the mortality (Equation 5.3.). The objective of a quantitative model is to quantify the effect of the variables we are interested in. In that case, it is not possible to test several models, and variables should be chosen a priori.

There are no rules on how to choose the temperature indicators. In this work, the choice was to use the mean temperature. Indeed, warm days can frequently be associated with cold nights, and the mean temperatures have the advantage of describing an average heat stress for a given day, but also smoothes the daily variations. A sensitivity analysis was also performed using the minimum and the maximum temperatures.

Regarding the choice of the lags, the assumption was that the impacts of heat are rather immediate, while the impacts of cold are delayed in time. Therefore, it was interesting to distinguish a term at lag 0, and a term at longer lag. The term at lag 0 was assumed to capture most of the short-term heat effects. In the main models, a term averaged over lag 1 to 7 was added to capture the impacts of cold, which is likely to be reduced during summer. A more detailed investigation of the lag structure was performed in a distinct model.

6.1.2. Lagged effect

A distributed lag model up to lag 16 was used to study the temporal structure of the temperature – mortality relationship. The distributed lag models are used to investigate the impact of lagged temperatures, allowing the data to determine the shape of the lag structure. They were initially developed to specifying the lag association between air quality and mortality (Schwartz, 2000). These models are also useful to investigate a possible harvesting effect, or mortality-displacement effect (Zanobetti *et al.* 2000). The general equation is then written as;

$$Log(E(Y_{t})) = \alpha + \sum_{s=0}^{16} \beta_{s} X_{t-s} + \sum_{k=1}^{K} f_{k}(X_{k})$$
 (Equation 6. 1)

Where Y_t is the number of deaths on day t

 X_{t-s} are the temperatures at the different lag, from 0 to 16.

 X_k are the variables we want to fit with splines, i.e. time, humidity...

To allow a stable estimation of the coefficients, the estimation was constrained using a polynomial of degree 3, using the method described by (Almon, 1965). The Almon procedure assumes that for a finite number of lag L, the lag coefficients can be represented by a polynomial degree of degree d, where d < L.

$$\beta_s = \sum_{k=0}^{a} \eta_k s^k$$
(Equation 6.2)

6.1.3. Influence of the winter mortality

(Rocklov *et al.* 2009) has discussed that the same people were vulnerable to heat and to winter. Therefore, low winter mortality may increase the proportion of vulnerable people during summer, and thus be associated with larger summer mortality. To test this hypothesis, the sensitivity of heat-related mortality in summer to the mortality of the previous winter (previous winter was defined as December – March) was tested.

In the initial model, an additional term qualifying the mortality of the previous winter is introduced, following the method described by (Ha *et al.* 2011), and used during summer only.

$$Y_{t} \approx Poisson(\mu_{t})$$
$$\ln(\mu_{t}) = \alpha_{0} + \beta T_{meanlag0} + \beta_{1}T_{meanlag0} * WL + \sum_{j=1}^{q} f_{j}(X_{ij}, \lambda_{j}) + \sum_{i=1}^{p} \beta_{i}X_{i}$$

(Equation 6. 3)

Where Y_t is the number of deaths on day t

 X_k are the variables we want to fit with splines, i.e. tmean lag 1-7, time, humidity...

WL is a binary variable describing the winter mortality

WL was computed by comparing the observed winter mortality to a reference winter mortality. The reference winter mortality for each year was predicted from a linear regression of the average winter mortality against calendar year, in order to take into account time trends and mortality changes over time. Winters with observed mortality below the model predicted values were classified as low winter mortality (i.e. WL=1).

Figure 6. 1 illustrates the difference between the mean winter mortality and the predicted values. In rural areas, winters preceding summers 1981, 1983, 1984, 1995, 1988, 1989, 1998, 2001, 2002 and 2003 were characterized by mortality lower than usual. In urban areas, winters preceding summers 1984, 1986, 1987, 1988, 1992, 1993, 2001, 2002 and 2003 were characterized by mortality lower than usual.



Figure 6. 1. Differences between the mean winter mortality, and the mean winter mortality predicted based on year

6.1.4. Changes over time

In order to identify possible trends in the response, the models were developed for different time period between 1981 and 2003: 1981-1985, 1986-1990, 1991-1995, 1996-2000, 2001-2003.

6.1.5. Sensitivity analysis

As a sensitivity analysis, all the above models were alternatively developed using the minimum temperature at lag0 and the maximum temperature at lag 1-7 as a sensitivity analysis.

An alternative modelling strategy was also tested, using a time-stratified case-cross over design. The case crossover design was introduced to study the transient effect of brief exposure on the occurrence of a rare acute-onset disease (Maclure, 1991). Individuals are used both as case and controls. The information of the individual exposure to a certain activity or agent during the hazard period (i.e. just before the disease) and control period are compared. This design is appropriate to study mortality and extreme heat exposure, as heat can be considered as a brief exposure that causes a transient change in risk of an acute event. Confounding factors are including either during the design phase (characteristics of each individual, day of the week, seasons...), or as co-variables in the model (e.g. influenza, air pollution). The effect period, i.e. the period after exposure where there is a change in risk, due to exposure, is of a few days, the peaks of mortality classically occurring 48 hours after the temperature peak.

The choice of the control period is the main difficulty of a case-cross over design. It has to be close enough to the hazard period, not to lose information, but not too close to avoid autocorrelation and biases. A stratified approached was chosen. Control days are defined as the same days of the week of the same months as the cases. This approach allows controlling for short and long-term variations (Janes *et al.* 2005). Data are then analysed using a conditional logistic regression. The likelihood of this model can be defined in the same way as the likelihood of a stratified Cox model (Janes *et al.* 2005).

$$L = \prod_{i=1}^{n} \frac{e^{Z_i \beta}}{\sum_{j=0}^{J} e^{Z_j \beta}}$$

(Equation 6. 4)

- i=1...n are the individuals
 j=0 : case days
 j=1: control days
 Z= exposure variable
 β = parameter
- β estimates the odds ratio.

In this case, the case cross over model is controlled for influenza. Time trends and day of the week are controlled by design in a time-stratified case cross over model.

All computations were done using the R software, and the mgcv package of the R software (R Development Core Team, 2004; Wood, 2006), which was specifically developed to perform GAM models.

6.2. Excess mortality associated with a mean temperature increase during summer

6.2.1. Shape of the relationship

The shape of the relation was similar for total mortality (Figure 6. 2) and total mortality over 74 years old (Figure 6. 3) in urban and rural areas. Below 15°C, there was no significant impact of the temperature, and an almost linear trend is observed above 15°C. A similar shape was observed for cardiovascular mortality in rural, but not in urban areas where the mean temperature did not influence the cardiovascular mortality (Figure 6. 4). On the opposite, temperature did not influence the respiratory mortality in rural areas, but a linear relationship was observed in urban areas (Figure 6. 5). Similar shapes were observed in Dublin (Figure 6. 6, Figure 6. 7).

Considering the shapes of the temperature-mortality relationship observed in rural and urban areas for the different causes of mortality, a linear relationship was assumed above 15°C. Risk estimates were then computed for a 1°C increase above 15°C.



Figure 6. 2. Relative risk (RR) of total mortality associated with mean temperature lag 0 in rural (left) and urban areas (right) (solid line, dashed lines represent the confidence interval)



Figure 6. 3. Relative risk (RR) of total mortality>74 years old associated with mean temperature lag 0 in rural (left) and urban areas (right) (solid line, dashed lines represent the confidence interval)



Figure 6. 4. Relative risk (RR) of cardiovascular mortality associated with mean temperature lag 0 in rural (left) and urban areas (right) (solid line, dashed lines represent the confidence interval)



Figure 6. 5. Relative risk (RR) of respiratory mortality associated with mean temperature lag 0 in rural (left) and urban areas (right) (solid line, dashed lines represent the confidence interval)



Figure 6. 6. Relative risk (RR) of total mortality (left) and total mortality >74 (right) associated with mean temperature lag 0 in Dublin



Figure 6. 7. Relative risk (RR) of cardiovascular mortality associated with mean temperature lag 0 in Dublin

6.2.2. Risk estimates

A 1°C increase above 15°C in the mean temperature was associated with a 1.5% [CI 95% 0.9:2.1] increase in the total mortality in rural areas, and a 1.6% [0.6:2.5] increase in total mortality in urban areas. The estimate was lower in Dublin, +0.6% [-0.1:1.3]. The impact was slightly lower for the total mortality over 74 years old (resp. 1.4%[0.6:2.2] and 1.5% [0.3:2.7]). A significant result was obtained in Dublin(+1.4% [0.1:2.7]).

In rural areas, a significant impact was observed on cardiovascular mortality (1.1% [0.3:1.9]) but not on respiratory mortality (+0.0 [-1.1:1.0]). The opposite pattern was observed in urban areas, with no impact on cardiovascular mortality (+0.2 [-0.8:1.3], in Dublin, -0.1 [-1.2:0.9]) but a large impact on respiratory mortality (+2.8% [0.5:5.1]) (Figure 6.8).



Figure 6. 8. % increase in mortality associated with a 1°C increase >15°C in mean temperature at lag 0 – summer

6.3. Investigation of the lagged structure

The distributed lagged model in rural areas showed that the largest effect was observed on the first two days after heat exposure (Figure 6.9). A small harvesting effect was observed between lags 6 and 10. A similar pattern was found for the total mortality above >74, although results were non-significant at lag 1 and 2 (Figure 6.10). A similar trend was observed for cardiovascular mortality (Figure 6.11). In urban areas, the risk decreased with the lags, but all the estimates were non-significant (Figure 6.12). A similar shape was observed for the mortality in the elderly, and by causes.



Figure 6.9. % increase in total mortality associated with a 1°C increase in mean temperature at lag 1 to 16 during summers in rural areas



Figure 6.10. % increase in total mortality >74 associated with a 1°C increase in mean temperature at lag 1 to 16 during summers in rural areas



Figure 6.11. % increase in cardiovascular mortality associated with a 1°C increase in mean temperature at lag 1 to 16 during summers in rural areas



Figure 6.12. % increase in total mortality associated with a 1°C increase in mean temperature at lag 1 to 16 during summers in urban areas

6.4. Influence of the preceding winter mortality

In rural areas, the relative risks were consistently higher in summers preceded by winters with a low mortality, both for the total mortality, the total mortality above 74 years old and the cardiovascular mortality. A similar difference was observed in urban areas for the total mortality, but not for the mortality above 74 years old. The largest difference was associated with the respiratory mortality in urban areas, even when considering the large confidence intervals (that reflect a lack of statistical power for these analyses) (Table 6. 1).

The analysis was not performed on Dublin due to a lack of statistical power when differentiating the winters.

Table 6. 1. % increase in mortality for a 1°C increase in temperature above 15°C, depending on the preceding winter mortality

	Rural	areas	Urban areas		
	Low-mortality	High-mortality	Low-mortality	High-mortality	
	winters	winters	winters	winters	
Total mortality	2.2 [1.3:3.1]	0.9 [0.1:1.7]	2.2 [0.5:3.9]	1.2 [0.3:2.0]	
Total mortality	2.7 [1.4:4.0]	0.3 [-0.5:1.1]	1.3 [-0.3:2.9]	1.6 [0.1:3.1]	
>74					
Cardiovascular	2.4 [1.2:3.7]	0.2 [-0.5:0.9]	0.4 [-1.4:2.1]	0.3 [-0.9:1.6]	
mortality					
Respiratory	0.1 [-1.6:1.7]	0.1 [-1.3:1.5]	9.8 [4.6:15.2]	2.3 [-0.2:4.8]	
mortality					

6.5. Changes of the temperature-mortality relationship over time

The periods were relatively homogenous in terms of temperatures (Table 6. 2) and of mortality (Table 6.3). In rural areas, the risk associated with a 1°C increase above 15°C was around 2% for the periods 1981-1984, 1985-1988 and 1989-1992. It started to decrease during the 1993-1996 period. Warm temperature were even associated with a reduced mortality during the period 1997-2000 (+1°C increase above 15°C is associated with a 1.1% [-2.0:0.6] decrease in the total mortality (Figure 6.13). A similar evolution was observed for the mortality above 74 years old in rural areas (Figure 6.14) and for the cardiovascular mortality (Figure 6.15). As for the respiratory mortality, low and non-significant risks were observed throughout all the periods (Figure 6.16).

In urban areas, a large risk was observed during the first period ($\pm 5.1\%$ [2.9:7.4]), while the response was lower and non-significant in the over periods (Figure 6.17). For the total mortality >74 years old, the risks were non-significant across all the periods, expect between 1993 and 1996. However, large positive risks were observed between 1981 and 1996, while the risks had been decreasing since 1997, and were negative between 2001 and 2003 (Figure 6.18). The risks were non-significant for the cardiovascular mortality (Figure 6.19). As for respiratory mortality, large risks were observed before 1998 ($\pm 13.7\%$ [7.2:20.7] between 1981 and 1984), and dropped brutally to become non-significant since that period (Figure 6.20).

Table 6. 2. Temperature characteristics of the summers over the different time periods

	Urban areas			Rural areas			
	Mean	Min	Max	Mean	Min	Max	
1981-1984	15.3	9.9	23.0	14.8	8.8	22.0	
1985-1988	14.0	8.3	19.9	13.6	8.6	18.9	
1989-1992	15.0	8.1	20.7	14.7	8.4	20.4	
1993-1996	14.8	9.7	21.0	14.6	9.7	21.1	
1997-2000	14.8	9.8	19.9	14.7	10.0	19.5	
2001-2003	14.8	9.1	20.8	14.5	9.2	20.7	
2001-2003	14.8	9.1	20.8	14.5	9.2	20.7	

	Urban			Rural				
	Total	Total>75	Cardiovascular	Respiratory	Total	Total>75	Cardiovascular	Respiratory
1981-1984	16.5	7.3	7.8	1.9	57.5	27.9	30.4	6.8
1985-1988	16.7	7.9	7.9	1.7	59.6	30.4	30.5	7.2
1989-1992	16.5	8.4	7.4	1.8	56.4	31.1	27.3	6.8
1993-1996	16.8	8.7	7.5	2.0	57.3	32.8	26.9	7.6
1997-2000	17.0	9.4	7.3	2.0	55.5	33.1	24.2	8.1
2001-2003	15.9	9.1	5.9	2.4	53.4	32.8	22.0	7.4

Table 6.3. Mortality characteristics of the summers over the different time periods



Figure 6.13. Evolution over the time of the % increase in total mortality associated with a 1°C increase>15°C in mean temperature at lag 0 in rural areas



Figure 6.14. Evolution over the time of the % increase in total mortality>74 associated with a 1°C increase>15°C in mean temperature at lag 0 in rural areas


Figure 6.15. Evolution over the time of the % increase in cardiovascular mortality associated with a 1°C increase>15°C in mean temperature at lag 0 in rural areas



Figure 6.16. Evolution over the time of the % increase in respiratory mortality associated with a 1°C increase>15°C in mean temperature at lag 0 in rural areas



Figure 6.17. Evolution over the time of the % increase in total mortality associated with a 1°C increase>15°C in mean temperature at lag 0 in urban areas



Figure 6.18. Evolution over the time of the % increase in total mortality>74 associated with a 1°C increase>15°C in mean temperature at lag 0 in urban areas



Figure 6.19. Evolution over the time of the % increase in cardiovascular mortality associated with a 1°C increase>15°C in mean temperature at lag 0 in urban areas



Figure 6.20. Evolution over the time of the % increase in respiratory mortality associated with a 1°C increase>15°C in mean temperature at lag 0 in urban areas

6.6. Discussion

6.6.1. Limits

This study experienced the same limits as those discussed in chapter V, the main one being the availability of the mortality data at a low geographical scale. Therefore, the health outcomes and heat exposure cannot be perfectly matched. The error in the exposure assessment may lead to an over or an underestimation of the risks. The analysis is also limited by the quality of the data. Changes in reporting practices may explain part of the differences observed over time, or between rural and urban areas, especially when considering the cardiovascular and the respiratory mortality. However, as the results are consistent with the literature and the current knowledge on heatrelated mortality, they may be considered as reasonable.

6.6.2. Comparison with the literature

6.6.2.1. Temperature mortality relationship

In the literature, published estimates of the impact of mean temperature on mortality ranged from no evident heat effect in Dublin (Ireland), Dallas and Charlotte (USA), and Busan (South Korea), to a 12.3% (95% CI 5.7 to 19.4) increase in mortality per 1°C increase in high temperature in Beirut (Lebanon) and 18.8% (13.0 to 25.0) in Monterrey (Mexico), with both cities having correspondingly high heat thresholds (Hajat and Kosatky, 2010).

An increase in mortality at the highest temperatures is observed in countries with moderate climate and relatively cold summers. In Sweden, an increase in 1°C in the summer temperature above the 90th percentile of temperature distribution was associated with a 5.1% increase in mortality [0.3:10.1] (Rocklov and Forsberg, 2010). In England and Wales, during summer, a 1°C increase in maximum temperature above a heat-thresholds set as the 93rd percentile of the temperature distribution was associated with a 2.1% increase in all causes mortality [95% CI 1.6:2.6). A larger increase was found for respiratory mortality

(+4.1% [3.5:4.8]), while cardiovascular mortality was associated with a smaller risk (+1.8% [1.2:2.5]) (Armstrong *et al.* 2011; Gasparrini *et al.* 2011).

The estimates observed in this work are consistent with such results, with a 1°C increase above 15°C in the mean temperature associated with a 1.5% [CI 95% 0.9:2.1] increase in the total mortality in rural areas, and a 1.6% [0.6:2.5] increase in total mortality in urban areas. The study also found a significant increase in mortality associated with an increase in temperatures above 15°C in Dublin for the total mortality above 75 years old (1.4% [0.6:2.2]).

In urban areas, no risk was observed for cardiovascular mortality, but a large impact was observed on the respiratory mortality, similarly to what has been observed in the US. Results on respiratory mortality might be explained by an interaction with air pollution.

6.6.2.2. Effect of previous winter

The study found that the risks associated with an increase in temperature were significantly larger when the summer had been preceded by a low-mortality winter, especially in rural areas. The assumption to explain these results is that the people vulnerable to heat are also vulnerable to cold. Thus, if the mortality is low during winter, the pool of vulnerable people who would suffer from the warm temperatures is larger, resulting in a larger excess mortality (Rocklov *et al.* 2009). So far, such equilibrium has been observed in urban areas. For instance, in Seoul, a 1°C increase in the summer temperature above 27.9°C was associated with a increase in total mortality of 7.97% [5.5-10.5]. The risk estimate was of 10.57% [7.30:13.963] for summers preceded by low-mortality winter, and of 4.85 [1.54:8.26] for summers preceded by high-mortality winters (Ha *et al.* 2011). In Italy, the

relative risk of mortality comparing days with a temperature of 30°C to days with a temperature of 20°C was 1.73 [1.50:2.01] during summers preceded by a low-mortality winter, and 1.34 [1.17:1.55] during summer preceded by a high-mortality winter (Stafoggia *et al.* 2009).

Further analyses based on data with a better spatial resolution would be needed to investigate why this was observed only in rural areas in Ireland.

6.6.2.3. Changes over time

Although there is no general clear trend, the analysis over the different periods of time tends to show a decrease of the risks, with the larger risks observed before 1987. Similar decreases in the risks have been observed in the UK and the US. A comparison of the weekly mortality in UK between 1990 and 1996 showed that the temperature mortality gradient for cold deaths and heat deaths diminished over time. Overall, there was a progressive reduction in temperature-related deaths (Carson *et al.* 2006). Similar results were observed in the US, where daily cardiovascular mortality counts from 107 cities in the US National Morbidity and Mortality Air Pollution Study were regressed against daily temperature using the case-crossover method between 1987 and 2000. In summer 1987 the average increase in cardiovascular deaths due to a 10 degrees F increase in temperature was 4.7%. By summer 2000, the risk with higher temperature had disappeared (-0.4%). In contrast, an increase in temperature in fall, winter and spring was associated with a decrease in deaths, and this decrease remained constant over time (Barnett, 2007).

However, it may be that the risks observed before 1987 are driven by the 1983 and 1984 heat waves. Changes in the reporting of the mortality, and in the demographics between urban and rural areas, may also partly explain the observed differences.

6.6.3. Influence of the modeling strategy

In rural areas, the GAM and the case cross-over (CXO) models give the same estimates for all causes of mortality considering the whole year. The same pattern is found when focusing on summer, although CXO results are less significant, probably due to a problem of statistical power. Striking differences are observed however for the respiratory mortality, where the GAM founds no results and the CXO a significant estimate. Similar results are found in urban areas, with consistent estimates except for the respiratory mortality. However, during summer, the CXO design tends to give higher estimates than the GAM design in urban areas (Appendix 6 to 9).

CHAPTER VII. ESTIMATION OF THE MORTALITY IMPACTS ASSOCIATED WITH CLIMATE CHANGE

7.1. Projections of mortality increase associated with temperature rises due to climate change

7.1.1. Method for estimating future impacts

Projections of future mortality impact in a context of climate change were obtained by applying the relative risk to the temperature increase predicted by the climate models.

$$\Delta y=y_0(e^{\beta\Delta T}-1)$$
 (Equation 7.1.)

where Δy is the excess mortality associated with the change in temperature ΔT . y_0 is the mortality baseline and β is the exposure response function.

Since the risk was estimated for the temperature increase above 15°C, ΔT is estimated as additional days above 15°C compared to the current climate.

$$\Delta T = \frac{\sum_{i=1}^{N_2} T_i + \partial - 15}{N_2} - \frac{\sum_{i=1}^{N_1} T_i - 15}{N_1} (Equation \ 7.2.)$$

Where δ is the mean daily increase of temperature according to the climate scenarios. δ of 1, 2 and 3°C were tested.

 N_2 is the number of days when T+ δ exceeds 15°C.

N1 is the number of days when T exceeds 15°C.

7.1.2. Projections of mortality increase associated with temperature rises due to climate change

Results are presented in Table 7. 1. Considering the worst scenario, +3°C, the excess mortality associated with the temperatures would be of 26 extra deaths per summer in rural areas and 32 deaths per summer in urban areas.

7.2. Discussion

Similar methods were applied in Canada (Gosling *et al.* 2007), New York (Knowlton *et al.* 2007) or Europe (Baccini *et al.* 2009), always concluding that future climates are likely to increase the heat and heat wave related mortality.

These projections rely on strong hypothesis, including no demographic changes and no ageing of the population, no modification of the characteristics of the urban and rural areas, no adaptation of the population, and no evolution of the pool in vulnerable people, which can be influenced by the winter mortality. This is conditioned by the choice of a constant baseline and a constant relative risk. Therefore, they ignore all the non-climatic factors at stake in the heat-mortality relationship. These estimates also do not take into account potential heat wave impacts.

A major source of uncertainty is also the crude estimate of the temperature changes. The use of relevant climate models to estimate the number of future heat waves would have been more appropriate, and could have led to large differences in the results. For instance, in Chicago (Peng *et al.* 2010) computed the mortality risk associated with heat waves

based on the 1987 – 2005 data, and applied this risk to a climate for 2081-2100. They found that in the absence of adaptation, the city of Chicago could experience between 166 and 2 217 excess deaths per year attributable to heat waves. A significant source of uncertainty in their study resulted from the variability of the temperature estimates provided by different climate models: between 0.6 to 5.4 heat waves per year depending on the model.

Rural areas			
		Mean number of	
	Mean number of	additional degrees	Number of
Scenarios	additional days above	above 15°C per	associated excess
	15°C per summer	summer	deaths per summer
+1°C	18	47	17 [10:24]
+2°C	37	111	20 [12:28]
+3°C	49	191	26 [15:37]
Urban areas			
Mean number of			
	Mean number of	additional degrees	Number of
Scenarios	additional days above	above 15°C per	associated excess
	15°C per summer	summer	deaths per summer
+1°C	19	51	19 [8:30]
+2°C	35	119	24 [10:38]
+3°C	45	207	32 [13:51]

Table 7. 1. Projections of mortality increase associated with temperature rises due to climate change

CHAPTER VIII. OPPORTUNITIES FOR ADAPTATION

This work found that although the impacts of heat are limited in Ireland, severe heat waves may result in a significant excess mortality. With the perspective of climate change, associated with the ageing of the population, it may be that more severe heat episodes results in a larger mortality burden. The Irish EPA has a high confidence that the frequency of heat wave should increase in the future years (Environmental Protection Agency, 2009). Moreover, the number of people in the over 74 age group is expected to increase to between 273 289 and 284 413 in 2021, compared with 190 398 recorded in the 2002 census. It is also expected that more than 20% of people older than 65 years old will be living in Dublin city and county (National Council on Ageing and older people, 2009). The ageing of the population and the changing climate may increase Ireland vulnerability to extreme heat.

Adaptation to heat and heat wave can cover different aspects. It could be physiological, social (e.g. heat prevention plan) and technological (e.g. air conditioning, urbanisation plan...). The rapidity of climate change will be a major factor in the intensity of the effects and the efficiency of adaptive passive responses and ameliorative actions. For example, physiological or behavioural changes may begin within minutes of an abrupt change in temperature, in order to cope with an immediate heat stress. However, reducing the overall vulnerability to heat, through, for instance, architectural changes in housing, take long periods of time.

Based on the existing literature, two main axis of adaptation can be identified, as will be detailed in this chapter. One is a long-term adaptation, focusing on housing and the

reduction of the urban heat island, which are main risks factors of mortality and morbidity during heat wave. The second is a short-term adaptation, based on heat prevention plans that are being developed in an increasing number of countries and cities, to anticipate the effects of adverse heat waves.

8.1. Physiological adaptation

Physiological adaptation is likely to be limited. Thermo physiological studies focus on healthy population, and show that a short-term acclimatization is possible, through training for example (Robinson, 1967). Here, acclimatization is defined as an adaptive change in physiological system induced by prolonged exposure to environmental stress or a new environment, in this case a new climate. If the heat stress is not too great, healthy individuals adjust and acclimate to the new temperature fairly rapidly; after 5-10 days of continued heat stress, the body temperature and pulse rate are near normal. By about 14 days, blood volume and venous tone are approximately normal. By about 3 weeks, a new equilibrium seems to be established. If the heat stress is removed, most of the acclimation appears to be lost within a few days (White and Hertz-Picciotto, 1985). These techniques are used to train sportsmen or soldiers before travelling to warmer climates. However, these trainings do not work for elderly patients(Takamata *et al.* 1999). Therefore, a spontaneous physiological adaptation may not be observed in the most vulnerable people.

8.2. Technological, long-term adaptation

Several studies showing the decrease of the impact of heat wave on mortality over time, between the 50s and the 90s, are in favour of a technological adaptation (Carson *et al.* 2006) (Lerchl, 1998). For instance, in 19 United States cities, heat-related mortality has significantly decreased between 1964 and 1988. During the 60s and the 70s, almost all cities showed a large excess mortality during the warmest days. In the 80s, the excess mortality during warm days disappeared in several Southern cities. This effect extended to Northern and Central cities in the 90s. The authors explained these modifications by technological adaptation such as air conditioning (Davis *et al.* 2003).

8.2.1. Air conditioning

Although these results suggest a decrease in heat-related vulnerability associated with an improvement in the conditions of life, deadly heat waves are still occurring (Baccini *et al.* 2008; Medina-Ramon and Schwartz, 2007). It seems that the adaptation gained between the 50s and the 90s is not optimum in cities with moderate mean temperatures, and with large temperature variations. Air conditioning seems to be the main mean of adaptation in the United States. However, since the 70s, warning against the danger of power failure during heat waves have been raised (Ellis *et al.* 1975). They were confirmed by the mortality observed during the 2006 heat wave in California, (Knowlton *et al.* 2009), with examples of places where air conditioning was available but not used for economical reasons.

8.2.2. Housing

Housing was found to be a main risk factor during the 2003 heat wave in Paris. Based on a case-control study investigating several risk factors, it was found that improving the isolation of old buildings, especially for those apartments located directly below roof, and improving the ventilation, may reduce the heat stress during heat waves (Vandentorren *et al.* 2006).

Individual building management are now considered as part of the adaptation to climate change in many cities. For instance in London, measures to incorporate green roofs and green walls, to avoid high glare facades, or to plant street and garden streets (Greater London Authority, 2010).

8.2.3. Urbanisation, and the urban heat island

In Europe, where air conditioning is less common, adaptation may be rather linked to way of life and urbanisation.

In the 40s, the UHI was considered as a positive impact of urbanisation. Later, it was reevaluated as a problem of well-being, but without real health consequences. Recently, epidemiological studies have been able to document the main role of UHI in mortality during heat wave. As a result, efforts are made by urban planners to reduce the UHI whenever possible (Institut d'aménagement et d'urbanisme d'Ile de France, 2010).

A case-control study following the 2003 heat wave in the Paris region investigated the contribution of the UHI to the mortality burden, testing different thermal indicators to characterise the UHI. Remote sensing data were used to obtain the surface temperature measured. Landsat data allowed a good spatial resolution (50 m), but with only one image available over the period (09/08/2003). The NOAA/AVHRR data allowed a poor spatial resolution (1km), but 61 images were available over the period. Within the city of Paris, differences in surface temperature could reach 4°C. It was also noticeable that the UHI was concentrated on industrial areas, with few residential units during the day, and on the city centre where most people are living during the night (Dousset *et al.* 2011).

Similarly, the urban heat island effect in London is more important at night, with several °C differences compared to the outlying rural areas (Figure 8.1). During the heat wave of 2003, the temperatures in the center of London were up to 10°C higher than the temperatures in rural areas (Greater London Authority, 2010). In Dublin, during summer, at night, a gradient of up to 8°C may be observed due to the urban heat island (Graham, 1993).

It is also worth emphasising a study in Baltimore, where areas experiencing the highest UHI were also those areas with lower income, less education and more elderly people, leading to an additional vulnerability to heat (Huang *et al.* 2011b).



Figure 8.1. Urban heat island in London, at 21.43 pm on the 12/07/2006 (Greater London Authority, 2010)

Initiatives can be taken to reduce UHI in cities. Urban planners distinguish four types of interventions: the management of the activities and transport within the city, buildings (shape, envelope, management), public spaces (shape, composition, green spaces) and urban organisation (density, shape) (Colombert, 2010; Guigère, 2009). Many of these axes may be associated with health gains, wider than the reduction of the vulnerability to heat, by reducing greenhouse gazes emissions, air pollution (Institut d'aménagement et d'urbanisme d'Ile de France, 2010), and improving the well-being (European Environment Agency, 2009a; Rosenthal *et al.* 2007). Examples of concrete actions are developed for instance in the draft adaptation plan of London (Greater London Authority, 2010), with

commitments to develop green spaces, to provide guidelines for new buildings, to promote well-isolated roofs...

8.3. Heat prevention plans

An example of short-term societal adaptation to extreme heat is the implementation of heat prevention plans, based on an historical analysis of past heat waves, a good understanding of risk factors and systematic prevention measures can decrease the burden of heat. The literature suggests that parts of the health consequences of a heat wave are preventable through the implementation of appropriate measures and prevention. Indeed, most of the prevention measures are simple; to refresh, to protect oneself from the sun and the heat, to drink and eat regularly. However, in the absence of a pre-existing organization they were found to be not easily implemented, especially for the most vulnerable people.

8.3.1. Basics of a standard plan

An extensive work has been done by the EuroHeat project, to review existing plans in Europe and provide the basics and essential components of a standard plan (Matthies *et al.* 2009). They identify that heat prevention plans should be designed according to the following principles:

- use existing systems and link to general emergency response arrangements.
- adopt a long-term approach, as responding to an emergency is not enough. For heat waves, long-term actions aim to reduce the scale of climate change and to reduce the impact of climate change by adapting the built environment. Both of these are covered in this plan.

- adopt a multi-agency and intersectoral approach. While many of the actions fall to the health sector, active involvement of other sectors is essential.
- communicate effectively, delivering useful, timely, accessible, consistent and trustworthy information to their target audience and especially to high risk populations.
- ensure that responses to heat waves do not exacerbate the problem of climate change. In particular, although air conditioning can be an efficient protection, it is energy intensive and adds to greenhouse gas emissions. Air conditioned public spaces where vulnerable people can obtain respite from the heat during heat waves have been shown to be useful in the U.S. setting.
- evaluate afterwards the efficiency of the plans

They also identified that common heat prevention plan were composed by eight core elements:

- an agreement on a lead body who will be in charge of coordinating a multipurpose collaborative mechanism between bodies and institutions and to direct the response if an emergency occurs. This can be the Department of Health and Children, the HSE, the meteorological office or the public health agency.

- an accurate and timely heat warning systems. It relies on meteorological forecast to identify periods at risk and promote active prevention.

- a heat-related health information plan, detailing what kind of information is communicated, to whom and when.

- a reduction in indoor heat exposure (medium- and short-term strategies), including advices on how to keep indoor temperatures low during the heat wave.

- a particular care for vulnerable population groups;

- a good preparedness of the health and social care system (staff training and planning, appropriate health care and the physical environment);

- a long-term urban planning (to address building design and energy and transport policies that will ultimately reduce heat exposure, planting trees to provide more shade, etc);

- a real-time surveillance and evaluation.

An example of how this can be practically done will be illustrated later based on the French example.

It is worth noting that most of the systems in place are focusing on urban areas, and that some actions would not be fully transposable to rural areas.

8.3.2. Basics of heat wave warning systems

The heat prevention and action plans are usually relying on warning systems, commonly labelled as "heat-health warning systems" (HHWS). These systems have been designed to anticipate heat wave that present a risk to the population and to provide timeliness information to ensure an efficient and coordinated response. Timely warning is needed to ensure rapid responses from the population and the authorities. A lag period of one to three days between the maximum temperature and a peak of mortality has been reported in several heat waves, for instance 2003 in France (Le Tertre et al. 2006), in 1995 in Chicago

(Kaiser et al. 2007). This short lag requires prompt actions from all stakeholders. The warning system must thus be simple, easy to understand and to communicate, and avoid as much as possible false warnings.

Different procedures have been used in different places across North America, Europe and Asia. These differences may come from different vision of the heat and health relationship and the mechanisms underlying heat impact (e.g. epidemiological analysis of the mortality –temperature data vs thermo physiological model of a standard human body submitted to heat fluxes, individual meteorological indicators vs air masses indicators). However, the rationale behind these choices, and the practical organisation of the system, are rarely developed in the literature.

(Hajat *et al.* 2009) proposed a classification of the main types of warning systems into four categories:

- Synoptic system, which takes into account that health may be affected by a number of weather factors acting in combination. Adverse meteorological conditions are assessed using air-mass categories. Epidemiological analysis of historical mortality data is used to model the mortality relationship within each category (e.g. US, Shanghai (Tan *et al.* 2004)).
- Temperature systems are based on models of the direct relationship between temperature and mortality. Thresholds are then chosen based on temperature values which have an acceptable combination of sensitivity and specificity in terms of identifying high mortality days (e.g. France (Pascal *et al.* 2006)).
- Indices system encompasses the spectrum of indices that exist which are composite

measures of temperature and humidity, including apparent temperature or humidex (e.g. Canada).

 System based on physiologic principles of heat budget models of the human body, for instance the Environmental Stress Index (ESI) which is based on commonly used and easily measured weather variables, and was developed and tested under hot-humid and hot-dry climates. It has found to be highly correlated with the Wet Bulb and Globe Temperature (WGBT) which is a physiologically-based heat metric widely used in occupational health settings.

8.3.3. Efficiency of heat prevention plans

Although it is difficult to quantify the efficiency of such systems, by estimating the number of avoided deaths, several examples show a reduction of the heat-related mortality (McGeehin and Mirabelli, 2001). Heat wave prevention plans were developed in several cities to anticipate the risk of heat wave and promote prevention. Three elements constitute these plans: 1) a warning system, which is responsible for identifying the period at risk. Several methods have been used to develop such systems. 2) action plans and 3) a real-time health monitoring system, which can be used to estimate the impact after the event, and to provide information during the event, relevant to maintain an health alert even after the end of the heat wave, if data were increasing (Matthies *et al.* 2009).

Few studies have investigated the impacts of these prevention plans. In France, where the plan has been in place since 2004, the only available study was done by the Institute for Health Education, trying to understand if behaviours have changed between the 2003 and

the 2006 heat wave. Seventy four per cent of the people over 15 had heard or read prevention messages about the heat wave. 63% declared to have taken protective measures for themselves, and 73% for their relatives (Léon *et al.* 2007). The Health Protection Agency in the United Kingdom has questioned stakeholders of the heat prevention plan to evaluate its efficiency and acceptability. 51% of the inspectors of the Commission for Social Care Inspection reported an improvement in the majority of the care homes they visited. 12% observed an improvement in half of the care homes, and 4% observed no improvement at all. In the health care centres, 67% of the workers considered that the lists of vulnerable people were useful, but only 47% of them really used these lists during a warning.

8.4. A concrete example: the French heat prevention plan

8.4.1. The genesis of the plan

In 2003, no heat prevention plan existed in France. The meteorological office Météo-France first communicated about the heat wave on the 1st August 2003. They initially pointed the risk of drought. The first mentions of a health risk for elderly people appeared in a press release dated from the 7th August 2003. The first health warning was transmitted on the 06/08 2003, the Ddass of the Morbihan informing of 3 heat-related deaths at work. Similar information came from other places between the 06 and the 08/08. A rapid investigation of the data available collected by the hospital of Paris Assistance publique-Hôpitaux de Paris (AP-HP) and the Pompes funèbres générales (PFG) indicated that a large outbreak of mortality was going on (Institut de Veille Sanitaire, 2003). However, prevention was

mostly limited to communication, by Météo-France, by the city of Paris (preventive messages on "panneaux lumineux" from the 06/08/03, communication by the Ministry of Health giving some advices). Emergency measures were taken by the Assistance publique-Hôpitaux de Paris (AP-HP) on the 11/08/2003 only.

After summer 2003, the French congress mandated a commission to investigate the primary response during the heat wave, and identify the causes of the inappropriate responses (Sénat, 2004). They concluded that although knowledge existed on the potential impacts of a heat wave, they were underestimated due to the "exotism" of the situation. The mortality response was also so rapid that it did not allow for the implementation of extensive actions.

Following this event, several actions were taken to have a better understanding of the impacts of heat and heat wave on mortality and morbidity in France. Collaboration was initiated between Météo-France and the public health authorities to develop a heat wave prevention plan. In 2004, the French Ministry of Health developed a national prevention strategy, relying on five pillars: 1) protective measures, 2) identification of vulnerable populations, 3) warning using meteorological and health data, 4) community-support and 5) communication (Ministère de la santé et des sports, 2009). This plan includes a variety of actions which are targeted to several actors. These actions can be activated all the summer, or focused on the heat wave period. Some of the actions are enforced by law (Le Premier Ministre, 2005), for instance :

- All institutions hosting elderly people or disabled people must define the organisation, the role and responsibilities of the institution during a heat wave.

- All institutions hosting elderly people or disabled people must have access at least to a cool room. A survey conducted in October 2003 shows that 42% of the answering institutions had no cooling installation available at that time (Agence française de sécurité sanitaire de l'environnement et du travail, 2004).

- Each city must create a database of vulnerable people who should be contacted by social services during a heat wave. However, being on the list is a voluntary action.

8.5.2. The organisation of the response plan during a heatwave

Based on meteorological forecast, heat wave warnings are identified when minimum and maximum temperatures for the next three days are above pre-defined thresholds (Pascal *et al.* 2006). The warning is proposed by the French Institute for Public Health Surveillance (InVS) to the Ministry of Health, who transmits it to the prefects in each department. The final decision of following the warning and implementing special measures is under the responsibility of the prefects, who are directly under the authority of the Interior Minister, and are in charge of the local emergency preparedness and response.

The warning proposal must thus contain enough information to motivate the prefects to follow the alert proposal, and to select the measures that should be implemented. It must also clearly states when an alert should be ended. The process is schematized on Figure 8.2.



Figure 8.2. Organisation of the French warning system

8.5.3. Measures implemented at the city level; the example of Paris

The French heat prevention plan provides tools and guidelines to support the organizations of various stakeholders during a heat warning.

Based on this, the city of Paris has developed several actions that are implemented during summer and in case of a heat warning.

During summer, brochures are disseminated though the city magazine, through the city hall, information desks, social desks, shops.... They give advices on how to react during a

heat wave (Figure 8.3) and promote the registration on the Chalex database. In 2010, 19 000 vulnerable people, elderly people or disabled people, were registered in the database. Registration is voluntary, and can be done by phone or by regular mail. 320 shops have taken a commitment to the city hall, to disseminate information about heat waves, and to orientate people who required help. The city also animates a network of young citizens who identified vulnerable people and visit them, for instance to deliver books, in cooperation with the city libraries. Cool spaces were also opened to welcome elderly people during summer (67 spaces opened in 2010).

During a heat wave warning, prevention messages are disseminated through the city boards, giving advices and emergency calls. The people registred in the Chalex registries are called regularly by the city workers. In 2006, each person was called every 48 hours. If a health problem is suspected, the person is signalled to the health regulation emergency cell. Home-visits are also organised by the city social workers, NGOs and civil servants. Additional cool spaces can be opened, located in city official buildings, hostels, NGOs buildings... Transportation to these spaces is ensured by a dedicated public transportation services, which can involved up to 100 vehicles per day.



Figure 8.3. Brochure edited by the institute for prevention and health education for

English-speaking residents

8.5.4. Evaluation of the system: the 2006 heat wave in France

The impact of this plan on the reduction of the risks, and on the excess mortality and morbidity during heat waves is still to be determined. A key limitation to this evaluation is the lack of heat waves since 2004. The main one occurred in July 2006. Minimum and maximum temperatures were below those observed during the August 2003 heat wave, but July 2006 was the warmest month of July in France since 1950. Using a nation-wide model, it was estimated that if the conditions have been those prevailing before 2003, 6 452 excess deaths should have been recorded during the 2006 heat wave. About 2 100 excess deaths were observed (Fouillet *et al.* 2008).

Compared to the results obtained in 2003, no specific effect of the heat wave was observed in 2006. The maximum daily relative risk varied from 1.45 in Strasbourg ([0.97-2.16]) to 1.04 in Lille [0.92-1.18]. The only cities with a significant relative risk were Rouen and Le Havre. However, in Le Havre, this result is likely to be due to another factor than heat, given the low temperatures in the preceding days. In Rouen, the maximum relative risk is observed one day after the warmest minimum (18.6°C) and maximum (34.6°C) temperatures.

In the absence of a specific heat wave effect captured by the spline functions, which the variations of the mortality observed in the cities during summer 2006 are explained by the usual daily variations of the ozone and temperature. The high temperatures and ozone concentrations resulted in 411 excess deaths between the 27th July and the 11th August 2006 compared to the 2000-2005 average (2003 excluded).

8.6. Opportunities to develop a heat prevention plan in Ireland

8.6.1. Relevance of a prevention plan

Although few heat waves have been identified in the historical analysis, with a moderate impacts on mortality compared to what have been observed in other places, one cannot exclude the possibility of a more severe impact of a sustained and intense heat wave. The perspective of facing more and more intense heat waves, together with potential increasing vulnerabilities (urban densities, ageing, and poverty) calls for dedicating some efforts to the prevention of adverse heat waves.

Measures involved in the heat prevention should be proportionate to the risks expected in Ireland. Communication to promote appropriate behaviours in the population and in the health professionals may be a first and essential step to limit the adverse impacts of heat waves. Good practices of communication during extreme heat have been reviewed by Health Canada (Health Canada, 2011), to guide the development of targeted heat-health communication campaigns. They included special consideration for the residents of rural and small town communities, which would form a good ground for a communication in Ireland.

Additional actions, including identification of the most vulnerable populations, at-home visits... may be implemented in cooperation with NGOs. Such actions could be targeted on the most vulnerable populations (O'Neill *et al.* 2009), similarly to the initiatives taken in California (California Department of Public Health, 2011b), or in Canada (Vescovi *et al.*

2005). There, a geographical information system is used to map the number of people older than 65, number of people below a poverty threshold, number of people living alone, and number of people with low education.

8.6.2. Opportunities for a heat warning system in Ireland

The purpose of a heat warning system would be to identify heat waves episodes that could result in increased mortality and morbidity. It would alert relevant stakeholders who in turn can promote preventive actions and disseminate appropriated information.

The data available for this thesis allows working on temperature or indices systems. Indeed, synoptic system would require an identification of the main synoptic air masses in Ireland, which is beyond the scope of this thesis. System based on physiological principles would require the use of a dedicated heat-exchange model.

As a key to the success of a warning system is a limited number of false warnings, it is relevant to use indicators that can be forecasted with the highest possible confidence. Therefore, a system based on temperature seems preferable to a system based on indices involving humidity, since meteorological services have a higher confidence in temperature forecast up to several days in advance.

The basics of the temperature systems is to find a meteorological indicator associated with a meteorological threshold, able to separate the days when the mortality is above an acceptable level and the days when it is below that level. The cut-offs have to be in accordance with the objectives of the organizations in charge of the plan. Different temperatures can be used, minimum, mean, maximum temperatures, or a combination of these. For instance, in Melbourne (Nicholls *et al.* 2008), the system relies on the mean daily temperature for a single day, while in France the system relies on the minimum and maximum temperature averaged over three days. For the day j (Pascal *et al.* 2006).

In Ireland, as the heat-exposure is mostly a day-time exposure (temperatures being moderate at night, even during past heat waves), the maximum temperature seems to be a good indicator. It is routinely forecasted by the meteorological services, and easily understood by the general population.

One way to determine the thresholds is to regress the daily count of deaths on the temperature indicator, controlling on long-term trends, seasonality, and day of the week, using a generalized additive model. This model is then used to compute the excess mortality predicted for each value of the temperature indicators.

Figure 8. 4 presents the excess mortality associated with the maximum temperature for urban and rural mortality, obtained from the following model:

Total mortality=s(time)+ +day of the week+s(Tmax lag 0) (Equation 8.1.)

Excess mortality is predicted for temperatures corresponding to the percentiles 50 to 100 of The mean temperature distribution. Table 8. 1 reports the excess mortality predicted for different values of maximum temperatures above 25°C, and the corresponding numbers of days between 1981 and 2003. For instance, a threshold of 25°C may prevent an excess mortality above 2% in both urban and rural areas, and occurs on a moderate number of warning days, most of them corresponding to the 1983, 1995 and 2003 heat waves. The indicator Tmax averaged over 3 days was also tested, but did not provided any added value compared to the indicator Tmax alone.



Figure 8. 4– Excess mortality associated with the maximum temperature
Tmax (°C)	Excess mortality (% [IC95%])		Number of days ≥ Tmax	
	Rural areas	Urban areas	Rural areas	Urban areas
25	3.8 [1.3:6.3]	1.4 [-2.9:6.0]	38	25
26	5.5 [2.3:8.8]	2.7 [-2.9:8.6]	13	14
27	7.3 [3.3:11.4]	4.0 [-2.9:11.5]	5	2
28	9.2 [4.4:14.3]	5.5 [-2.9:14.6]	1	1
29	11.2 [5.5:17.2]	7.0 [-2.9:17.9]	0	0
30	13.2 [6.7:20.2]	8.5 [-2.9:21.3]	0	0

Table 8. 1. Excess mortality associated with extreme maximum temperatures

CHAPTER IX. A REVIEW OF OTHER RISKS THAT COULD BE IMPACTED BY CLIMATE CHANGE

This chapter is based on a work initially focusing on the health impacts of climate change in France, and the associated needs for adapting surveillance (Pascal, 2010b).

9.1. Observed climate change

The Fourth Assessment Report (AR4) of the Intergovernmental Panel on Climate Change concluded that warming of the climate system is unequivocal. The Earth has warmed by + 0.76° C on average during the last 100 years, with eleven of the last twelve years (1995-2006) being the warmest on record. All climate models agree that temperatures will continue to rise in the next decades. The estimates of increases vary between +1.8°C and +6.4°C by 2100, compared to the 1961-2000 average (Solomon *et al.* 2007).

On the basis of world-wide observations, the Intergovernmental Panel on Climate Change (Solomon *et al.* 2007) concluded in 2007 that:

- the average temperatures in the northern hemisphere over the second half of the 20th century were very probably (the probability being undoubtedly more than 90%) higher than those of any other 50-year period over the last 500 years, and probably the highest of the last 1,300 years.
- it is very likely that cold days and nights, as well as frosts, have become less frequent in most continents, while warmer days and nights have become more frequent.

- it is probable (with a probability of more than 66%) that heat waves have become more frequent in most continents.
- more than 8 experts out of 10 agree that the natural systems linked to snow, ice and permafrost as well as the water systems have already been impacted by climate change.
- more than 9 experts out of 10 agree that many terrestrial and marine ecosystems have already been impacted by climate change.

A number of environmental indicators (temperatures, sea levels, rainfall, flowering seasons, nest-building seasons, etc.) by the European Environment Agency show evolutions that are consistent with the overall changes (European Environment Agency, 2009b).

Between 1961 and 2005, the climatic trends identified in Ireland were largely consistent with global trends, with projected temperature increases ranging between $+1^{\circ}$ C and $+3^{\circ}$ C by 2100, compared to the 1961-2000 average (Sweeney *et al.* 2003). Climate change may have several health impacts, one of them related to the changes in temperature and the increase in the frequency and severity of heat waves.

9.2. Climate projections

Only when greenhouse gases are taken into account can models be produced that explain the increases in temperatures that have been observed over the last 50 years. In 2007 the concluded that it is more than 90% probable that current levels of global warming are due to human activity (Solomon *et al.* 2007). The radiative forcing of the atmosphere, generated by persistent greenhouse gases, increased by 26.2% between 1990 and 2008. Between 2003 and 2008, CO₂ was responsible for 85% of this increase.

Climate models enable future climates to be predicted by using scenarios of socioeconomic evolution that serve to simulate changes in greenhouse gas emissions. For example, scenario A2 corresponds to an economic development with a high level of regional focus and significant population growth. The average increase in temperature between now and 2100 simulated by the different climate models could be between 1.1 and 6.4°C (Solomon *et al.* 2007) (Figure 9.1.).



Figure 9. 1. Spread of global warming modelled according to the emissions scenarios (the numbers correspond to the numbers of the different models used for each simulation) (Solomon *et al.* 2007).

Based on similar scenarios, the projected temperature increases for Ireland range between +1°C and +3°C by 2100, compared to the 1961-2000 average. Figure 9.2. presents the estimated increase in mean July temperatures over Ireland, with a general increases of approximately 2°C apparent, and the highest values to be found inland away from north and west facing coasts (Sweeney *et al.* 2003).



Figure 9. 2. Downscaled mean temperature scenarios for Ireland for the period 2061–2090 at a resolution of 10 km². This approximates to the period around 2075 (Sweeney *et al.* 2003).

9.2. Impacts on localised phenomena (cyclones, storms, floods, forest fires)

Climate change is likely to bring about an increase in the frequency and intensity of geographically localised extreme climatic events such as floods, cyclones and forest fires. These phenomena are grouped into a single subject area, because even though they require different approaches in terms of forecasting, prevention and management, they use similar tools on the epidemiological front.

In the short term, the deaths and injuries occurring as a direct result of the event and their fundamental prognosis are the principal concern. But very quickly, the consequences of the damage provoked by the event become predominant in the occurrence of the health impact. One can therefore expect to observe:

- injuries due to actions for rehabilitation, clean-up operations or further collapses of buildings that are already weakened;
- hypothermia (due to a stay in water), carbon monoxide poisoning (due to the improper use of generating sets and improvised heating appliances), water-borne infections, dehydration due to insufficient supply of drinking water;
- an impact on the mental health of the affected population (depression, anxiety, post-traumatic stress, increase in dependence on psychoactive substances, etc.) (Figure 9.
 3).

If the event completely disrupts society and attempts at management are undermined, one can also expect to observe acts of violence. In the medium and long term the alteration in quality of life, the disruption of the social tissue, situations of psychological stress and the degradation of the environment and the habitat will have an effect on mental health and somatic health.

9.3.1. Storms

Climate change could lead to changes in the intensity of storms. The immediate dangers inherent in storms are the deaths directly resulting from accidents and injuries. Indirectly the damage could lead to the impossibility of gaining access to the emergency services through breaches in lines of communication, their destruction or quite simply because of the high number of injured people requiring the mobilisation of all the emergency services and their medical staff. The destruction of life-supporting infrastructures may also have a significant impact on health. For example, the improper use of generating sets or improvised heating appliances could lead to carbon monoxide poisoning as was the case in the immediate aftermath of the Klaus storm which hit the south-west of France in January 2009. The clean-up operation could lead to a number of injuries (e.g. chainsaw accidents, falls from roofs, etc.). Extreme decompensation of underlying chronic pathologies (treated or not) linked directly to the stress. Finally the material consequences of the disaster (loss of working tools, loss of housing, significant financial loss) and the difficulties encountered in remedying these can themselves have a significant impact on health, particularly mental health.

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9.3.2. Floods and the effects of rising sea levels

Climate change is identified as a factor that leads to a probable increase in the risk of flooding, both in terms of the seriousness of the flood and the probability of flooding occurring (European Environment Agency, 2007). There may also be an increase in the frequency of tidal waves (Higher-than-normal increase in the sea level compared to tidal forecasts. It can be caused by onshore winds, low atmospheric pressure and the tides), bringing with them an increased risk of flooding.

The health risks identified are essentially as follows:

- infection by contamination of drinking water or contact with dirty water or deterioration of the life-related habitats (mildew, fungus arising from chronic damp, etc.);
- effects on mental and somatic health due to the psychosocial impact of material losses such as housing and working tools and to the resulting deterioration of living conditions;
- effects on the environment, for example due to the spread of toxic products triggered by the flood.

The impact on mental health is important and must not be neglected, particularly in the case of large-scale, sustained or repeated flooding, which involves particularly sensitive populations or requires evacuations and rehousing operations (Institut de Veille Sanitaire, 2004; Six *et al.* 2008).

9.3.3. Forest fires

Climate change will increase the duration of the summer droughts which provide the perfect conditions for forest fires. The health impacts of forest fires in the medium and long term are little known. The smoke is formed of a complex mix of combustion products: carbon monoxide, formaldehyde, acrolein, benzene, carbon dioxide (CO₂), nitrogen oxides, polycyclic aromatic hydrocarbons (PAHs), ammoniac, 2-furaldehyde, inhalable particles and nanoparticles (Carfatan *et al.* 2004; Wegesser *et al.* 2009) Organic compounds emitted during combustion can also contribute to the formation of ozone. In Asia and North America, a link has been documented between exposure to high concentrations of particles following fires and an increase in hospital admissions for respiratory and cardiovascular problems, principally amongst asthmatics, those suffering from schronic obstructive bronchopneumopathy, children and the elderly (Moore *et al.* 2006; Mott *et al.* 2005; Naeher *et al.* 2007).

The material consequences of the waste resulting from the fire also have a significant psychosocial impact.

9.3.4. Droughts

There are few studies on the impacts of extreme climatic events of long duration such as droughts.

In Australia, the social and economic impacts and the impacts on mental health have been documented (Berry *et al.* 2008).



Figure 9. 3. Impacts of extreme climatic events on mental health, adapted from (Berry *et al.* 2009)

These events challenge the vulnerability, resilience and ability to adapt of the emergency services to repeated occurrences of such phenomena very close together, as well as the influence of the actions taken and the way in which the consequences of these events are managed in terms of the recorded effects.

9.4. Air quality

The short-term effects of atmospheric pollution on health have been quantified in a number of international studies carried out since the beginning of the 1990s. They have shown an increase in mortality rates, hospitalisations and admissions to casualty departments for respiratory and cardiovascular problems in line with an increase in atmospheric pollution. Studies into the chronic effects linked to long-term exposure to atmospheric pollution tend to show an increase in the risk of developing lung cancer or a cardio-pulmonary disease (myocardial infarction, chronic obstructive bronchopneumopathy, asthma, etc.). These effects are more significant than the short-term effects.

The interactions between climate change and air quality are complex. On the one hand, climate change will have an effect on the concentrations of pollutants. On the other hand, atmospheric pollution contributes to climate change. It is also directly impacted by policies that aim to reduce greenhouse gas emissions.

9.4.1. Impacts of climate change on atmospheric pollution

High temperatures encourage the production of ozone. Increasing temperatures are likely in particular to provoke an increase in emissions of the precursors of ozone (biogenic organic compounds of plant origin such as isoprene) and stimulate the photochemical reactions that lead to the production of ozone. The very high levels of ozone observed during the summer 2003 heat wave in Europe and their associated effects on health (Filleul *et al.* 2006; Stedman, 2004), even though they were relatively marginal in comparison to those linked with high temperatures, can certainly be considered as the model for what could be produced in future (Schär *et al.* 2004; Vautard *et al.* 2007) (Figure 9. 4). The effect on the base levels of ozone has been the subject of more discussion because the results of the models are somewhat contradictory, due to the opposing effects of the increase in temperature on the increase in ozone production on the one hand and, on the other, the increase in rainfall and the water content of the atmosphere on the increase in the elimination of ozone (Devolder, 2009). The effects of climate change on the concentrations of particles are less well-established: the impact of more frequent forest fires, higher demand for electricity and increasing recourse to thermal power stations suggest, however, a tendency for the concentration of fine particles to increase.

9.4.2. Impacts of atmospheric pollution on climate change

Air pollutants, particularly ozone and particles, have an overall impact on climate change. Ozone is classified in the third category of greenhouse gases (Solomon *et al.* 2007). Methane is one of the principal greenhouse gases and is also a precursor of ozone. The effect of particles is more complex: although carbon soot aerosols produced by the combustion of fossil fuels have a direct positive radiative forcing effect on the atmosphere, sulphates produced by the oxidation of SO₂ have a direct negative effect (reflection effect). Likewise, aerosols contribute to cloud formation, having a reflective power and therefore a negative forcing effect.

9.4.3. Impacts of policies aimed at reducing greenhouse gases on atmospheric pollution

The combustion of fossil fuels is a major source of greenhouse gases and pollutants. If their usage were reduced this would produce a twofold benefit and the net impact on health could be very significant. Given the part that transport plays in greenhouse gas emissions, this is also the case for policies that aim to reduce the usage of road, air and sea transport (for example, a modal shift away from air transport to the train). Dealing with the two

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issues of climate change and air pollution together also enables us to take advantage of the fact that the formation processes of pollutants in the troposphere will respond very quickly, while the effects of measures to reduce greenhouse gas emissions will only be discernable after several decades. An economic analysis carried out in 2009 shows that the benefits of policies that aim to reduce CO₂ and PM10 were higher than their costs, that the policies that aim to reduce greenhouse gases (GHG) would lead to a significant reduction in CO₂ emissions and in PM10 emissions to a lesser extent, while local policies that aim to fight against air pollution would reduce PM10 emissions but not CO₂ emissions. Introducing synergy between these two policies could lead to additional reductions in CO₂ estimated at 15% for Western Europe (Bollen *et al.* 2009).

Certain strategies could also have beneficial effects on one issue and damaging effects on the other: thus, the use of wood as a fuel could be encouraged due to its attractive carbon balance, but under non-optimal conditions of combustion and processing of waste, it would lead to an increase in emissions of pollutants (fine particles, NOx, PAHs). AirParif, the French organisation responsible for monitoring air quality in the Ile de France, estimated that a 150% increase in the consumption of wood for heating compared to 2005, under the same conditions of use, would leade to a 40% increase in emissions of particles (PM10 and PM2.5), a 45% increase in emissions of CO and a 50% increase in emissions of volatile organic compounds. GHG would be reduced by 2% (AirParif, 2009).

The assessment of measures for dealing with automobile emissions is also often very complex. For example, catalytic converters and particle filters enable the emission of

pollutants to be reduced (PM, NOx) but tend to reduce the energy efficiency of the vehicles and therefore increase greenhouse gas emissions per kilometre driven. Furthermore, the use of diesel has been encouraged for reasons of improved energy efficiency but its use leads to an increase in emissions of PM, especially carbon soot, and NOx, while the use of technologies to limit these emissions of PM and NOx reduces the benefit in terms of CO_2 emissions.

These few examples demonstrate the value of integrating the health element into the development of public policies, aiming both to improve local air quality and to mitigate climate change.



Figure 9. 4. Evolution of the distribution of ozone in summer (June-August) in the south of Germany between the years 1991-2000 and 2031-2039 (Forkel and Knoche, 2006)

9.5. Respiratory allergens

The principal pathologies associated with respiratory allergens are asthma and allergic rhino conjunctivitis. Pollen allergies affect 10-20% of the population in Europe. Some authors hypothesise about the role played by climate change in the increase in cases of asthma observed since the 1950s (Beggs and Bambrick, 2005). However, it is worth remembering that respiratory allergens are not the only risk factors in the occurrence of asthma, or in the aggravation of an existing case of asthma.

Meteorological conditions encourage the production and dispersal of pollen, and the climate influences the existing species in a given geographical zone. Climate change is likely to bring about changes to the vegetation zones (movement further north of some Mediterranean species, for example (Kienast *et al.* 2000b)), prolonged periods of pollination, which has already been observed for certain species (Figure 9. 5), and even an increase in the quantities of pollen produced (Damialis *et al.* 2010a). The end of the pollination period is often very late, with an average delay of 5 days over the whole continent of Europe and for all species (Menzel *et al.* 2006), even though there are exceptions for some plants and some localities. This is already leading to more prolonged periods of exposure to allergenic pollens for sufferers, and these could extend even further in the years to come. In addition, even though few studies have been carried out so far, the increase in temperatures is likely to render the pollen even more allergenic. This has been demonstrated in the case of the birch; the higher the temperature, the more of the allergen Bet v1 is contained in the pollen (Ahlholm *et al.* 1998b; Levetin and Van de, 2008a) and for

the common ragweed *Ambrosia artemisiifolia*, for which an increase in temperature of 3.5° C would lead to an increase of 30-50% in the allergen Amb a1 in its pollen grains (Singer *et al.* 2005a; Ziska *et al.* 2003b). The increase in levels of CO₂, which goes hand-in-hand with global warming, is likely to increase the quantities of pollen produced (Kimball *et al.* 1993b) and their allergenicity (Singer *et al.* 2005a; Wayne *et al.* 2002b). Models produced for the ragweed pollen have demonstrated that the production of this pollen is likely to increase by 32-55% between 2070 and 2100, due to the increase in the CO₂ content of the atmosphere (Burr, 2010a).

The damaging effect of pollens on respiratory health (risk of sensitisation and worsening of symptoms among sensitised subjects) is strengthened by atmospheric pollution (particles, ozone), which is itself interacting with the climate. Pollution can act on the allergenicity of pollen by increasing the number of allergens inside the grains and facilitating their exit (Kopferschmitt-Kubler and Pauli, 1999a); nitrogen oxides, ozone and diesel particles are the pollutants that are most often blamed as the role of sulphur dioxide and carbon monoxide appears to be more random. But pollutants are also respiratory irritants, which play a role in the adjuvant factors in allergic reactions (Svartengren *et al.* 2000b), with photochemical pollution this time being ahead of acido-particulate pollution. Thus, when associated with pollen, ozone and nitrogen dioxide can accentuate the bronchial response, as well as manifestations of rhinitis or conjunctivitis among allergy sufferers and can lower their response threshold to allergens to which they are sensitive. As for diesel particles, by stimulating the synthesis of IgE and cytokines, they facilitate the sensitisation of subjects that are predisposed to allergies. A history of pollen allergies demonstrates their increase

during the industrial era (Laaidi *et al.* 2002), and the relationships between this increase and pollution. In France, the prevalence of pollen allergies has tripled over 25 years (Annesi-Maesano and Oryszczyn, 1998). In Japan, pollen allergies to *Cryptomeria japonica* have increased from practically none to around 10% in the 1960s, among subjects living in towns or along motorways and high-speed roads (Ishizaki *et al.* 1987). This example is a good illustration of the interactions that may be expected between climate change and the other factors in the potential aggravation of a health risk situation.

Finally, the development of green spaces in towns is an important element in the improvement of the quality of life (European Environment Agency, 2009b) and the reduction of urban heat islands. However, it is important to take into account potential allergic reactions when selecting species.



Figure 9. 5. Evolution of flowering dates of the birch (*Betula*) in London between 1970 and 2006 (Emberlin *et al.* 2002)

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9.6. Habitat

Habitat is a central element in extreme climatic events, heat waves (Anderson and Bell, 2009; Vandentorren *et al.* 2006) and cold waves. The increase in the number of extreme climatic events could be associated with an increase in cases of carbon monoxide poisoning, as was seen at the time of the Klaus storm. An increase in mould or mildew contamination in the air inside buildings could also be envisaged; this is likely to develop more easily in warmer climate, or to arise more frequently following extreme climatic events such as floods. These moulds and mildews can lead to significant health problems, including respiratory symptoms, allergies and asthma (World Health Organization, 2009). The LARES study, carried out in eight cities by the WHO's regional office in Europe, found that a bad state of health was significantly associated with high levels of discomfort caused by heat, with problems linked to waterproofing and with the presence of humidity and/or mildews and mould (Ezratty *et al.* 2009).

The improvement of energy efficiency in residential properties enables mortality and morbidity rates to be reduced in summer and in winter, and is reflected in a general improvement in the quality of life (World Health Organization Regional Office for Europe, 2007). A Bristish evaluation of the impact of improvements in energy efficiency by different methods (improvement in materials, better ventilation, changes in heating systems and behaviour patterns) demonstrated an overall positive impact on health (Wilkinson *et al.* 2009). The consequences for quality of air inside buildings of these measures that aim to improve energy efficiency must of course be subject to precautions (e.g. insulation and the

inherent consequences of confinement of air) (Salagnac and Sacré, 2003), but they will be limited and will favour the implementation of adapted technologies.

9.7. Ultraviolet (UV) rays

While the risks linked to exposure to UVB rays have been well-known for a long time, the mutagenic activity of UVA rays has been known about for less than ten years (Agence française de sécurité sanitaire de l'environnement et du travail, 2005). The principal risks relate to the skin (basal cell carcinomas, spinocellular carcinomas, malignant melanomas, sunburn, allergies, and aging of the skin. It should be noted that the increase in UV rays observed in Europe does not suffice to explain the increase in cases of skin cancer recorded over recent decades. The major role played by behavioural changes linked in particular to holidays and sun seeking should be highlighted.

The evolution of UV rays as related to climate change is at the present time still uncertain. Some models predict a marked decrease in rainfall and cloud cover across parts of Europe during the summer, which would lead to an increase in ultraviolet rays. Initial measurements of the quantity of UV light per 25km² showed an increase in UV rays in June during the last decade compared to the previous decade. In addition, longer summers and an increasing number of sunny days could lead to behavioural changes that would increase the exposure of the population to ultraviolet light.

9.8. Water-related risks

Waterborne health risks include risks linked to the water supply and risks linked to water used for leisure purposes, as well as industrial water (Legionnaires' disease). This type of risk is associated with all three methods of exposure (ingestion, inhalation and contact). The dangers associated with these exposures are many and varied. The risk of infection includes around a hundred micro-organisms carried in faeces (Salmonella typhi, pliovirus, Helicobacter pylori, Cryptosporidium sp. and the other agents of gastroenteritis) and opportunistic agents (Legionella pneumophila, Naegleria fowleri, Pseudomonas aeruginosa...). The WHO consider that the risk of infection is the most widespread and the most worrying for all countries, including developed countries (World Health Organization, 1996), and advocates epidemiological surveillance of the risk in addition to assessment of the water quality and evaluation of the risk. The toxic risk is primarily a long-term one and often very localised (Davison et al. 2005). The risks that present the most concern in France are those linked to the by-products of disinfection (Hrudey, 2009) and to a lesser extent to arsenic, vinyl chloride, lead and cyanobacteria. Some very well-known toxins do not currently fall within the scope of health surveillance, either because they do not present a risk at the present time (nitrates) or because exposure to them through water is negligible (pesticides), or because the risk to human health has not yet been established and requires more research (medicines in water).

9.8.1. Water for consumption

Rather than causing new risks to emerge, climate change is likely to bring about an increase in the frequency and intensity of well-known adverse phenomena such as severe low water levels and mudslides immediately following episodes of heavy rain. It will have a direct affect on ecosystems via a rise in temperatures. The exposure of the populations will therefore be modified both by changes arising from the contamination of environments and by the modification of "determinants of exposure".

Ecology and the pathogenicity of toxic or "opportunistic" micro-organisms could be modified by changes in certain climatic parameters (De Toni *et al.* 2009). For example, that the frequency of the blooming of cyanobacteria is estimated to increase with climate change. The increase in water temperatures during the summer months will, on the one hand, encourage the growth of cyanobacteria (the optimum temperature is around 25°C) and, on the other hand, promote the stratification of water in the lakes or reservoirs which also satisfies blooming pre-requisites.

The production of toxins, which is inconstant, also seems to be determined by climatic factors but the link is insufficiently understood to be able to support any forecasts. This blooming principally affects water in reservoirs, which represents 1/3 of the surface water used for the production of drinking water, and particularly dystrophic water, through the excessive inflow of fertiliser into bodies of water. The impact of the blooming of cyanobacteria on the quality of water in the water supply may be reduced by adequate treatment of the water (for example, filtration on the banks, ultrafiltration). Assuming that

neither agricultural fertilisation practices nor methods of treatment of water to make it drinkable change, the exposure to cyanobacteria of the population supplied by reservoir water will increase in the context of climate change.

Data from countries with warm climate does not suggest that for pathogens originating in faecal matter there will be a significant rise in risk levels in line with an increase in temperature, but rather that there will be a reduction, linked to the direct stimulation of their disappearance by predation. Non-*cholera Vibrio* is the exception, due to the presence of intermediate hosts (copepods), the populations of which would increase in line with a rise in temperature, but this concerns the food-related risk via the consumption of shellfish.

The essential drivers that will increase the risk of contamination of water by faecal matter are mudslides and severe low water levels rather than variations in temperature. These two extreme water-related situations are likely to bring about severe water pollution, either by concentration of pollutants (in the case of low water levels) or by the introduction into the water of faecal matter from the ground or from overflows from sewerage systems (in the case of mudslides). The pollution by faecal matter of raw water concerns vulnerable surface water and groundwater (alluvial deposits, karsts) but the risk of infection principally affects rural populations that are supplied by karst water systems, because treatments used in small water treatment installations are often insufficient for inactivating or eliminating micro-organisims.

Mudslides and low water levels also lead to an increased concentration of soluble organic matter or particles in the surface water. The organic matter reacts with disinfecting

products (chlorine, ozone) to create by-products of disinfection such as trihalomethanes. Water in the water supply produced from surface water is particularly sensitive to the risk of appearance of by-products of disinfection.

Changes in the determinants of exposure may also be envisaged. A trend towards watersaving is already appearing via the use of private wells or rainwater harvesting, which is in theory reserved for uses other than consumption. It is difficult to forecast quantitatively the effect of this trend on exposures to toxins and pathogens. The drop in consumption leads to the water being held in the distribution network for longer, which is likely to affect the water quality: production of certain by-products of chlorination, the development of bio film sheltering opportunistic pathogens. Also of concern is a possible increase in the number of accidents due to the inflow of water into the urban network attributable to the installation of unprotected connections between the urban network and the domestic network, dedicated to water produced on the spot.

9.8.2. Water used for bathing or swimming

Water used for bathing or swimming is also likely to see an intensification of the risks linked to the presence of cyanobacteria. The free-living amoeba *Naegleria fowleri*, which is periodically found in low concentrations in fresh water, could become a cause of meningitis with the warming of fresh water bodies. The emergence of tropical algae could also be a concern, as in the growth of Mediterranean *Ostreopsis ovata*. This dinoflagellata is the cause of skin irritations in swimmers and can provoke epidemics of asthma attacks among riverside dwellers by aerosolisation of cellular debris. Exposure is therefore

dependent on particular meteorological conditions which could occur more frequently in the future. Exposure could also increase along with an increase in the practice of swimming in natural water. The food-related risk linked to the bioaccumulation of dinoflagellate toxins in the food chain, present in the tropics but limited in Europe to diarrhoea syndrome (*Dinophysis ovate*), could also increase and bring about more serious effects, such as neurological ones, for example.

The definition of the organic matter in water in order to treat it requires more research as a priority, given the influence of this parameter on the toxic and microbiological risk and the probable adverse evolution of the problem. Furthermore, it would appear necessary that research also returns to some subject areas which have been neglected, such as the risk of infection in the water supply originating from faecal matter or the risk of cancer attributable to the by-products of disinfection. Here also the priority must be to define the nature of the exposure, as the nature of the carcinogenic molecules remains controversial.

9.9. Potentially dangerous plant and animal species excluding infectious agents

The geographical distribution of a species depends on the physicochemical conditions of its environment, interactions with other species, restrictions on its spread or factors that encourage it. The interaction between these factors is complex, such that it is difficult to forecast the geographical evolution of a species. By default, it is primarily the physicochemical conditions that are used to determine a "climatic envelope" in order to predict the response of a species to global warming. For the moment, apart from some specific cases, this indicator on its own is not sufficient to be able to predict the evolution of a species, since there are many other factors, such as land use (de Chazal and Rounsevell, 2009), which come into play.

9.10. Soils

The evolution of the soils under the influence of climatic, environmental and anthropic factors is a long process that is difficult to observe. Climate change could cause changes to the quality of the soils and particularly their agricultural properties, with consequences for food production that are not discussed within the scope of this report (Climsoil, 2008). Changes to stock water may also be expected, along with changes to the physical stability of the soils and erosion.

At the present time, there are only a very few articles which mention possible changes to the transfer of chemical pollutants in the different environmental compartments (Noyes *et al.* 2009), or changes to the determinants of exposure (for example, time passed outside, agricultural practices, vector control, etc.).

Changes in land use could also generate new exposures, for example by building on contaminated land. They also pose increasing problems linked to erosion, causing dust to play a more influential role in exposure, and risk mobilising contaminants contained in sediments, during floods, for example.

Finally, soils represent a reservoir of organic carbon that could be disturbed by climate change and by changes in the usage of the soils. (Climsoil, 2008).

9.11. Infectious diseases

It is important to remember that the epidemiology of infectious diseases is multifactorial and that the role of climate change in the emergence or re-emergence of infections is considered by many authors as less important than the other determinants. These last particularly concern the factors linked to the host (susceptibility to infections, weak immune system, infections linked to medical care, risky practices, etc.), to the agent (mutations, reclassifications), and to the environment (sociodemographic changes, migrations, urbanisation, food and drink, circulation of people and goods, globalisation of trade, imbalance in ecosystems, increasing density of inter-species contact, economic decline, etc.).

The incidence of the infections transmitted by vectors and rodents could increase due to climate change, due to the spread of the habitats of the arthropods or the increase in the population of the animal populations (rodents). Infections carried by mosquitoes are rare in Europe whilst tick-borne infections are more frequent (Lyme disease, Q fever). These are for the most part considered as serious diseases.

The incidence of infections transmitted by the faecal-oral route could also increase. Breaks in the cold chain could provoke a multiplication of infections that originate in food (salmonellosis, for example). Likewise, an increase in the populations of pathogenic vibrio species in the environment due to rising temperatures of salt water in estuaries represents a plausible hypothesis.

The incidence of Legionnaires' disease could also increase with global warming, by means of two possible phenomena: dissemination of the bacteria via cooling circuits in airconditioning systems, the use of which would increase with global warming; or an increase in the average water temperature in the black waste water networks that could lead to the multiplication of bacteria.

Pathogenic fungi represent a group of very diverse germs in terms of epidemiology, particularly with regard to populations, modes of transmission and interaction between the agent and the host. Most of these fungi are ubiquitous environmental saprophytes. Their pathogenicity is most often linked to the presence of a temporary or prolonged immunosuppression in the host, which can be of variable etiology: HIV infection (candida, cryptococcosis), haematopoietic pathologies and/or bone marrow transplants (pulmonary aspergillosis, zygomycosis), etc. The elderly also seem to represent a population group at risk due to specific immune deficiencies.

Among these at-risk populations, some could be exposed more than others (dose-response effect) due to climate change; increasing periods of warm and humid weather could contribute to an increase in moulds in these people's homes. Floods also contribute to an increase in risk, via changes in the ecology of the moulds (Etzel, 2007; Health Canada, 2007; Terr, 2009).

However, the role of climate change in the expansion of the vectors, or the introduction or not of new viruses, has yet to be determined. Virological studies as well as studies on the agent-host-environment interactions could, for example, lead to a greater understanding of the causes of sustained implantation, or not, of the West Nile virus in different geographical territories. Operational research projects that aim to estimate the relative size of the role played by the determinants of occurrence or of increased incidence of these infectious diseases, by putting them into perspective as required against the role played by climate change in relation to the other factors contributing to this emergence, would be useful. Thus, the increased incidence of Lyme disease in Europe is often mentioned as being caused by climate change (as it leads to an increase in the population densities of small rodents, which are the intermediate hosts of the ticks). Various others are, however, raised as contradictory hypotheses: in Belgium, the increasing tendency amongst better-off families to seek houses close to the forests could explain this phenomenon (Linard *et al.* 2007); in the Baltic countries, the elderly could be exposed for longer to forests infested with ticks as they go picking mushrooms in order to sell them, due to the economic decline (Lindquist and Vapalahti, 2008; Sumilo *et al.* 2007). The concepts of exposure to risks and changes to human behaviour patterns are increasingly cited as being the principal factor in some emergences attributed to climate change (Randolph, 2008; Reiter *et al.* 2003).

9.12. Migration

At a global level, estimates of the number of people forced to migrate due in part to climate change give figures that vary between 25 million to one billion between now and 2050, according to sources (Black *et al.* 2011; Piguet *et al.* 2011; Warner, 2009). These population shifts could be temporary, such as the one occurring after Hurricane Katrina in August 2005 which led to more than a million people migrating. However, these migrations are more often likely to be permanent. There are no projections for the impact of climate change on the number of immigrants into Europe.

CHAPTER X. CONCLUSION

10.1. Heat waves and mortality: a threat in Ireland

Although the results should be interpreted with care, due to the limits introduced by the rough geographical aggregations that were used, this PhD found that an increase in temperature was associated with an increase in mortality during summer in Ireland, and that past heat waves were associated with a small but observable excess mortality. However, with the perspective of climate change, and with the ageing of the population, it may be that more severe heat episodes results in a larger mortality burden, as was observed during the July 1983 heat wave.

10.2. Adaptation needs

10.2.1. Heat prevention plan

The perspective of facing more and more intense heat waves, together with potential increasing vulnerabilities calls for dedicating some efforts to the prevention of heat waves. These efforts should however be proportionate to the risks expected in Ireland. Communication to promote appropriate behaviours in the population and in the health professionals may be a first and essential step to limit the adverse impacts of heat waves. The relevance of setting a heat warning system to anticipate heat waves episodes that could result in increased mortality should be discussed. Such system would alert stakeholders who in turn can promote preventive actions and disseminate appropriated information. As night temperatures are usually low, most of the heat-exposure is a day-time exposure, and

the warning system may rely on the maximum temperature only. For instance, a threshold of 25°C may prevent an excess mortality above 2% in both urban and rural areas, and occurs on a moderate number of days, most of them corresponding to the 1983, 1995 and 2003 heat waves. This may be integrated in a simple system where the health authorities would be informed of a potentially dangerous heat wave, and would be able to reinforced communication.

10.2.2. Surveillance of health data

In addition to heat prevention, a first and essential step to adapt to climate change would be to improve the surveillance of health data, and especially of the mortality data. A better geographical resolution is an asset to study any relationship between a health topic and an environmental exposure. Such data would be essential to contribute to adaptation in at least three different ways; 1) to contribute to the scientific evidence on the health impacts of climate change, 2) to warm for unexpected impacts, and 3) to prioritize adaptation.

The framework developed in Chapter 2 may be a good basis to identify the opportunities for these objectives. For instance, in the case of heat waves, Figure 10.1. illustrates that even for a direct effect of climate change many determinants interfere with the possible evolutions of the heat-related risk under a changing climate. Even for this straightforward impact of climate change, the figure allows identifying gaps in knowledge, and calls for coupling epidemiological studies with other disciplines to help formulating and testing hypotheses. For instance, the influence of the underlying climate on the exposure-response function, and its evolution over time, can be investigated in collaboration with

climatologists, using time-series designs. In collaboration with sociologists, the analysis of individual and social risk factors through classical cohort or case-control studies, may give indication to better orientate prevention and to design appropriate intervention strategies. Monitoring trends of perceptions and expectations, as well as emerging behaviours, can also be helpful to formulate prevention policies and to promote individual adaptation using for example validated KABP - Knowledge, attitude, belief and practice surveys.

The framework we suggest can also be used to identify opportunities for routine surveillance to better target key determinants of health impacts of climate change. In the example of the heat waves, routine surveillance is limited to the monitoring of temperatures via the Meteorological offices, and to the monitoring of some specific heat-related impacts via syndromic surveillance (Josseran *et al.* 2008). These systems monitor generic health data or symptoms, such as emergency admissions for different causes, to orientate public health interventions. The latest is used for warning purposes, as it has proved to be efficient in identifying unusual morbidity during heat waves, which allows for the reorientation of preventive actions. A complementary surveillance of parameters modifying the individual vulnerability to heat, such as housing conditions, deprivation, urban heat island... should be considered to enrich the surveillance system.

Besides the heat wave example, accurate data and knowledge are still needed to identify which health determinants are likely to be sensitive to climate on the long run. Long-term series of data with a quality control program over time are needed to monitor trends of health outcomes in relation with climatic trends. Yet, maintaining sustainable databases, although essential, is a difficult task, especially in a period when financial cutbacks in existing surveillance systems may endanger their maintenance and long term coverage. In this context, the scarce resources available for surveillance should be appropriately allocated to ensure a better use of the already existing knowledge and data. For example, syndromic surveillance systems mentioned earlier have been developed recently in many countries, giving access to large datasets on mortality and morbidity. These systems were conceived for warning, and work is needed on the data of these systems to adapt their use for long-term analytical surveillance purposes on the long term. The opportunity of doing case-finding surveillance, i.e. focusing on regions where most impacts are expected should also be explored. For some topics, it would make sense to select regions of the world where specific climate changes are expected, and to implement coordinated regional, specific surveillance systems. The creation of a network of pilot regions in Europe on sensitive areas would be a way to move forward.

Health data can also be used to develop early warning systems. Differentiating the drivers of the health outcomes could allow identifying better opportunities for early warnings. As mentioned earlier, syndromic surveillance is a powerful tool to detect and/or monitor in real time the impact of unexpected events. However, for the purpose of early warnings for human health, waiting for the health outcome leads to a significant loss of time, and it would be more appropriate to root the warning system into environmental monitoring systems, in order to anticipate, and whenever possible to prevent, the health impacts. Heat warning systems using meteorological forecasts are an illustration of such systems. Monitoring factors that contribute to the individual vulnerability (i.e. individual and social

risk factors, demography) could also be useful for early warnings, when an increase in vulnerability is observed or expected. For heat waves, an example would be monitoring the number of elderly people living alone.

Finally, the same figure can be used to point out areas where interventions would be the most effective to reduce current and future risks (Campbell-Lendrum *et al.* 2009; Frumkin and McMichael, 2008). For example, targeting interventions on determinants that have already contributed to a given risk is a no-cost adaptation strategy, as there will be an overall gain for public health, even if the observed evolution differs from the foreseen climate change. Also surveillance data would be useful to evaluate the health and monetary benefits of these interventions. In our example, an evaluation of the changes observed in individual behaviours during a heat wave, or a monitoring of housing conditions would give indications on the efficiency of a prevention plan.

Finally, adaptation plans to climate change are being developed worldwide, involving various stakeholders who have different understanding of the "climate change and health issues". The framework proposed in this PhD is a simple way to organize available epidemiological information in order to identify where the climate may interfere and what are the main sources of vulnerabilities. Although we detailed an example based on heat waves, as it is one of the most studied impacts of climate change, the framework can be applied to any type of risks, including infectious diseases.



Figure 10.1. Impacts of climate and climate change on the different determinants for Heat exposure

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APPENDIX

Appendix 1. Comparison of the temperature distributions according to different geographical aggregations



Figure A.1. Minimum and maximum temperature distribution per season for inland and coastal stations Inland; Clones, Mullingar, Birr, Kilkenny, Coastal; Dublin, Casement, Rosslare, Cork, Valentia, Shannon, Belmullet







Figure A.3. Minimum and maximum temperature distribution per season for southern and northern stations, Northern; Belmullet, Clones, Mullingar, Casement, Dublin, Birr, Southern; Kilkenny, Rosslare, Cork, Valentia, Shannon



Figure A.4. Minimum and maximum temperature distribution per season for rural and urban stations, Rural; Kilkenny, Mulligar, Belmullet, Birr, Valentia, Urban;

Casement, Rosslare, Cork, Dublin, Shannon

Appendix 2. Trends of the minimum and maximum temperatures per stations between

1981 and 2006



Figure A.5. Maximum temperature 1981-2006 – per stations – summer



Figure A.6. Minimum temperature 1981-2006 – per stations – summer



Figure A.7. Maximum temperature 1981-2006 – per stations – winter



Figure A.8. Minimum temperature 1981-2006 – per stations – winter

Appendix 3. Overview of the generalised additive models

A general linear model is a statistical linear model written as:

$$Y = \alpha + \beta X$$

Where Y and X are matrices containing series of observations.

 β is the matrix containing the parameters to be estimated

 α is the matrix containing the remaining errors, which are assumed to follow a normal distribution.

Generalized linear models are a generalisation of the general linear model, where the variables can have non-normal distribution, and do not have to be continuous. When studying mortality, a usual assumption is to work with a Poisson distribution. The mean of the distribution (E(Y)) is then related to the X variables via a link function g()

$$E(Y) = \alpha + g^{-1}(\beta X)$$

In additive models, Y is linked to X using smooth functions (fj) fitted from the data. The smooth functions fj() can be fit using parametric or non-parametric means. In that case, it can potentially better fit to data than other methods, relying on the data with no assumptions on the actual shape of the relationship.

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$$Y = \alpha + \sum_{j=1}^{p} f_j(X_j)$$

Finally, generalized additive models have been developed to combine the properties of generalized linear models with additive models. The mean of Y is related to the X variable via the smooth functions and a link function.

$$E(Y) = \alpha + g^{-1}(\sum_{j=1}^{p} f_j(X_j))$$

Generalized additive models are very flexible, and can provide an excellent fit in the presence of nonlinear relationships and significant noise in the predictor variables. However, over fitting is a common error, i.e. the model describes the noise instead of the real relationship.

Appendix 4. Development of the models to estimate the mortality during heat waves.

The first step is to the long-term time trend and seasonality. Figure A.9 represents the observed total mortality in rural areas, and the fitted models including time trend, seasons and day of the weeks. Figure A.10 shows that introducing influenza epidemics as an additional term improve the prediction of some of the winter peaks.



Figure A.9. Rural areas, total mortality, model including time trend, season and day of the weeks



Figure A.10. Rural areas, total mortality, model including time trend, season, day of the weeks and influenza

On a second step, temperature terms are introduced at different lags. For a given temperature indicator, the best model is then chosen relying on the AIC criteria. Not all the models will be detailed here, but AIC values are reported for each model in Appendix 3.

As an illustration, the outputs of the different models using the maximum and the minimum temperatures are reported below. Classical diagnoses are used to check for the reliability of the model (Figure A.11).

Maximum temperature lag 0 total ~ s(time, fx = TRUE, k = 88, bs = "cr") + as.factor(dow) +as.factor(influenza) + s(tx, k = 3, bs = "cr")Approximate significance of smooth terms: edf Ref.df Chi.sq p-value s(time) 87.00 87.000 3634.93 < 2e-16 *** 1.98 2.000 57.35 3.51e-13 *** s(tx) ___ Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 R-sq.(adj) = 0.502Deviance explained = 50% UBRE score = 0.20842 Scale est. = 1 n = 8375 Maximum temperature lag 0, Minimum temperature lag 1 total ~ s(time, fx = TRUE, k = 88, bs = "cr") + as.factor(dow) + as.factor(influenza) + s(tx, k = 3, bs = "cr") + s(tn1, k = 3, bs = "cr")Approximate significance of smooth terms: edf Ref.df Chi.sq p-value s(time) 87.000 87.000 3433.94 < 2e-16 *** 1.938 1.994 34.05 3.99e-08 *** s(tx) 1.808 1.961 27.27 1.12e-06 *** s(tn1) Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 R-sq.(adj) = 0.504 Deviance explained = 50.1% UBRE score = 0.20594 Scale est. = 1 n = 8375 Maximum temperature lag 0,2, Minimum temperature lag 1 total \sim s(time, fx = TRUE, k = 88, bs = "cr") + as.factor(dow) + as.factor(influenza) + s(tx, k = 3, bs = "cr") + s(tn1, k = 3,bs = "cr") + s(tx2, k = 3, bs = "cr")Approximate significance of smooth terms: edf Ref.df Chi.sq p-value s(time) 87.000 87.000 2969.788 < 2e-16 *** s(tx) 1.880 1.983 26.791 1.48e-06 *** 1.002 1.004 s(tn1) 0.002 0.969 s(tx2) 1.924 1.992 113.105 < 2e-16 *** Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 Deviance explained = 50.6% R-sq.(adj) = 0.509UBRE score = 0.19427 Scale est. = 1 n = 8375Maximum temperature lag 0,2, Minimum temperature lag 1,3 total ~ s(time, fx = TRUE, k = 88, bs = "cr") + as.factor(dow) +as.factor(influenza) + s(tx, k = 3, bs = "cr") + s(tn1, k = 3,bs = "cr") + s(tx2, k = 3, bs = "cr") + s(tn3, k = 3, bs = "cr")Approximate significance of smooth terms:

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edf Ref.df Chi.sq p-value s(time) 87.000 87.000 2836.925 < 2e-16 *** 28.407 6.61e-07 *** s(tx) 1.888 1.984 1.001 1.001 s(tn1) 0.326 0.569 1.908 1.989 71.321 3.18e-16 *** s(tx2) s(tn3) 1.001 1.001 22.783 1.82e-06 *** _ _ _ Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 R-sq.(adj) = 0.51Deviance explained = 50.7% UBRE score = 0.19182 Scale est. = 1 n = 8375Maximum temperature lag 0,2,4, Minimum temperature lag 1,3 total ~ s(time, fx = TRUE, k = 88, bs = "cr") + as.factor(dow) + as.factor(influenza) + s(tx, k = 3, bs = "cr") + s(tn1, k = 3, bs ="cr") + s(tx2, k = 3, bs = "cr") + s(tn3, k = 3, bs = "cr") + s(tx4, k = 3, bs = "cr") Approximate significance of smooth terms: edf Ref.df Chi.sq p-value s(time) 87.000 87.000 2606.280 < 2e-16 *** 27.059 1.28e-06 *** 1.855 1.975 1.000 1.000 s(tx) s(tn1) 0.010 0.922 1.831 1.963 1.291 1.492 41.208 1.05e-09 *** s(tx2) 2.678 0.175 s(tn3) 1.872 1.976 50.909 8.41e-12 *** s(tx4) ___ Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 R-sq.(adj) = 0.513Deviance explained = 50.9% UBRE score = 0.18677 Scale est. = 1 n = 8375 Maximum temperature lag 0,2,4, Minimum temperature lag 1,3, 5 total ~ s(time, fx = TRUE, k = 88, bs = "cr") + as.factor(dow) + as.factor(influenza) + s(tx, k = 3, bs = "cr") + s(tn1, k = 3, bs ="cr") + s(tx2, k = 3, bs = "cr") + s(tn3, k = 3, bs = "cr") + s(tx4, k = 3)3, bs = "cr") + s(tn5, k = 3, bs = "cr")

Approximate significance of smooth terms: edf Ref.df Chi.sq p-value s(time) 87.000 87.000 2511.441 < 2e-16 *** 1.848 1.971 27.950 8.13e-07 *** s(tx) 0.638 1.000 1.001 0.221 s(tn1) 1.813 1.953 46.506 7.29e-11 *** s(tx2) s(tn3) 1.001 1.003 1.206 0.273 s(tx4) 1.764 1.937 23.933 5.75e-06 *** s(tn5) 1.001 1.002 28.402 9.89e-08 *** Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 R-sq.(adj) = 0.514Deviance explained = 51.1% UBRE score = 0.18367 Scale est. = 1 n = 8375

Maximum temperature lag 0,2,4,6, Minimum temperature lag 1,3, 5

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total ~ s(time, fx = TRUE, k = 88, bs = "cr") + as.factor(dow) +
as.factor(influenza) + s(tx, k = 3, bs = "cr") + s(tn1, k = 3, bs =
"cr") + s(tx2, k = 3, bs = "cr") + s(tn3, k = 3, bs = "cr") + s(tx4,
k = 3, bs = "cr") + s(tn5, k = 3, bs = "cr") + s(tx6, k = 3, bs =
"cr")

Approximate significance of smooth terms:

edf Ref.df Chi.sq p-value s(time) 87.000 87.000 2348.259 < 2e-16 *** 1.824 1.962 26.843 1.39e-06 *** s(tx) 1.003 1.006 0.016 0.89993 s(tn1) 1.774 1.934 40.043 1.79e-09 *** s(tx2) s(tn3) 1.002 1.003 2.129 0.14516 1.622 1.837 10.187 0.00503 ** s(tx4) 4.820 0.02816 * s(tn5) 1.000 1.001 s(tx6) 1.722 1.910 48.695 2.24e-11 *** _ _ _ Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 R-sq.(adj) = 0.516Deviance explained = 51.3%

UBRE score = 0.179 Scale est. = 1 n = 8375

Maximum temperature lag 0,2,4,6, Minimum temperature lag 1,3, 5,7
total ~ s(time, fx = TRUE, k = 88, bs = "cr") + as.factor(dow) +
as.factor(influenza) + s(tx, k = 3, bs = "cr") + s(tn1, k = 3, bs =
"cr") + s(tx2, k = 3, bs = "cr") + s(tn3, k = 3, bs = "cr") + s(tx4,
k = 3, bs = "cr") + s(tn5, k = 3, bs = "cr") + s(tx6, k = 3, bs = "cr") +
s(tn7, k = 3, bs = "cr")

Approximate significance of smooth terms: edf Ref.df Chi.sq p-value s(time) 87.000 87.000 2290.990 < 2e-16 *** 1.854 1.974 27.678 9.36e-07 *** s(tx) 1.001 1.003 0.056 0.81341 s(tn1) 1.823 1.959 40.462 1.52e-09 *** s(tx2)1.379 1.606 s(tn3) 1.404 0.39664 s(tx4) 1.801 1.948 12.899 0.00148 ** s(tn5) 1.001 1.001 3.928 0.04758 * 1.010 1.019 18.326 1.94e-05 *** s(tx6) s(tn7) 1.881 1.985 27.666 9.58e-07 *** ___ Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 R-sq.(adj) = 0.517 Deviance explained = 51.4% UBRE score = 0.17674 Scale est. = 1 n = 8375

Maximum temperature lag 0, Minimum temperature lag 1-7

total ~ s(time, fx = TRUE, k = 88, bs = "cr") + as.factor(dow) + as.factor(influenza) + s(tx, k = 3, bs = "cr") + s(tn17, k = 3, bs = "cr")

Approximate significance of smooth terms: edf Ref.df Chi.sq p-value

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Figure A.11. Diagnostics of the model including time trend, season, day of the weeks, influenza, and maximum temperature at lag 0,2,4,6 and minimum temperature at lag 1,3,5,7

Once a model is selected, it can be fitted on the observed temperature or on reference temperatures. Reference temperatures are the daily mean of the temperature over 22 years (Figure A.12).

Heat wave impacts are estimated as the differences between the observed mortality, and the predicted mortality fitted on the reference temperature values (Figure A.13).






Figure A.13. Rural areas, total mortality, model including time trend, season, day of the weeks, influenza and minimum and maximum temperature terms up to lag 7 fitted on observed temperature (red) and on reference temperature (blue)

Appendix 5. AIC of the models to estimate the excess mortality during heat waves

				Tmax-	Tmax,	Tmin,
	Tmin	Tmax	Tmean	Tmin	Tmin	Tmax
lag0	60271	60235	60244	60285	60235	60271
lag0,1	60227	60163	60177	60279	60215	60155
lag0,1,2	60177	60095	60106	60279	60116	60102
lag0,1,2,3	60156	60033	60068	60279	60095	60020
lag0,1,2,3,4	60135	60017	60048	60279	60053	60010
lag0,1,2,3,4,5	60102	59982	60007	60281	60028	59969
lag0,1,2,3,4,5,6	60080	59954	59982	60282	59989	59950
lag0,1,2,3,4,5,6,7	60055	59940	59958	60279	59970	59928
lag0,1-7	60051	59961	59967	60285	60042	59974

Table A. 1. AIC of the models – rural areas – total mortality

				Tmax-	Tmax,	Tmin,
	Tmin	Tmax	Tmean	Tmin	Tmin	Tmax
lag0	55091	55080	55079	55100	55080	55091
lag0,1	55071	55035	55040	55097	55069	55032
lag0,1,2	55049	54986	54999	55099	55003	55005
lag0,1,2,3	55032	54954	54975	55101	54989	54954
lag0,1,2,3,4	55018	54940	54959	55102	54963	54943
lag0,1,2,3,4,5	54996	54918	54933	55103	54946	54913
lag0,1,2,3,4,5,6	54974	54896	54910	55105	54917	54894
lag0,1,2,3,4,5,6,7	54955	54882	54889	55106	54899	54876
lag0,1-7	54954	54901	54901	55100	54954	54911

Table A. 2. AIC of the models – rural areas – total mortality >74

				Tmax-	Tmax,	Tmin,
	Tmin	Tmax	Tmean	Tmin	Tmin	Tmax
lag0	53239	53213	53220	53258	53213	53239
lag0,1	53214	53168	53181	53257	53199	53166
lag0,1,2	53180	53137	53142	53254	53148	53135
lag0,1,2,3	53169	53113	53126	53256	53135	53106
lag0,1,2,3,4	53159	53096	53111	53258	53112	53099
lag0,1,2,3,4,5	53143	53083	53093	53259	53098	53077
lag0,1,2,3,4,5,6	53135	53070	53081	53261	53083	53066
lag0,1,2,3,4,5,6,7	53116	53065	53067	53260	53070	53058
lag0,1-7	53116	53075	53076	53257	53103	53083

Table A. 3. AIC of the models – rural areas – cardiovascular mortality

				Tmax-	Tmax,	Tmin,
	Tmin	Tmax	Tmean	Tmin	Tmin	Tmax
lag0	43230	43225	43225	43232	43225	43230
lag0,1	43227	43209	43215	43228	43226	43199
lag0,1,2	43219	43197	43204	43229	43199	43195
lag0,1,2,3	43211	43164	43183	43230	43193	43154
lag0,1,2,3,4	43204	43158	43176	43231	43169	43154
lag0,1,2,3,4,5	43179	43144	43151	43231	43157	43133
lag0,1,2,3,4,5,6	43174	43123	43141	43230	43134	43127
lag0,1,2,3,4,5,6,7	43169	43116	43133	43232	43133	43110
lag0,1-7	43167	43122	43134	43232	43174	43118

Table A. 4. AIC of the models – rural areas – respiratory mortality

				Tmax-	Tmax,	Tmin,
	Tmin	Tmax	Tmean	Tmin	Tmin	Tmax
lag0	46489	46487	46487	46489	46487	46489
lag0,1	46483	46474	46474	46491	46488	46464
lag0,1,2	46477	46456	46461	46492	46453	46462
lag0,1,2,3	46471	46451	46456	46492	46453	46449
lag0,1,2,3,4	46466	46443	46448	46492	46441	46448
lag0,1,2,3,4,5	46437	46423	46418	46492	46424	46419
lag0,1,2,3,4,5,6	46435	46411	46412	46494	46410	46414
lag0,1,2,3,4,5,6,7	46433	46397	46406	46495	46410	46395
lag0,1-7	46430	46398	46407	46491	46439	46391

Table A. 5. AIC of the models – urban areas – total mortality

				Tmax-	Tmax,	Tmin,
	Tmin	Tmax	Tmean	Tmin	Tmin	Tmax
lag0	40914	40916	40913	40918	40916	40914
lag0,1	40914	40910	40910	40918	40918	40904
lag0,1,2	40906	40901	40900	40920	40896	40903
lag0,1,2,3	40902	40896	40895	40922	40895	40892
lag0,1,2,3,4	40900	40891	40892	40923	40888	40890
lag0,1,2,3,4,5	40888	40883	40879	40924	40880	40878
lag0,1,2,3,4,5,6	40883	40877	40874	40926	40873	40870
lag0,1,2,3,4,5,6,7	40880	40863	40865	40928	40870	40859
lag0,1-7	40871	40861	40861	40918	40878	40857

Table A. 6. AIC of the models – urban areas – total mortality >74

				Tmax-	Tmax,	Tmin,
	Tmin	Tmax	Tmean	Tmin	Tmin	Tmax
lag0	39955	39945	39951	39951	39945	39955
lag0,1	39949	39939	39943	39953	39943	39938
lag0,1,2	39948	39928	39939	39951	39923	39940
lag0,1,2,3	39944	39916	39931	39951	39924	39916
lag0,1,2,3,4	39946	39917	39932	39953	39918	39918
lag0,1,2,3,4,5	39937	39905	39917	39955	39916	39906
lag0,1,2,3,4,5,6	39936	39902	39917	39956	39907	39906
lag0,1,2,3,4,5,6,7	39936	39892	39913	39958	39909	39896
lag0,1-7	39931	39899	39914	39951	39928	39899

Table A. 7. AIC of the models – urban areas – cardiovascular mortality

				Tmax-	Tmax,	Tmin,
	Tmin	Tmax	Tmean	Tmin	Tmin	Tmax
lag0	30291	30295	30289	30308	30295	30291
lag0,1	30292	30292	30287	30310	30294	30290
lag0,1,2	30287	30285	30282	30311	30285	30287
lag0,1,2,3	30285	30286	30281	30311	30283	30287
lag0,1,2,3,4	30273	30273	30267	30313	30274	30273
lag0,1,2,3,4,5	30268	30272	30264	30312	30265	30271
lag0,1,2,3,4,5,6	30270	30271	30264	30314	30267	30271
lag0,1,2,3,4,5,6,7	30267	30265	30258	30316	30264	30263
lag0,1-7	30262	30266	30258	30309	30264	30265

Table A. 8. AIC of the models – urban areas – respiratory mortality

Appendix 6. Central estimates of the excess mortality during heat waves depending on the model

 Table A. 9. Central estimates of the excess mortality in rural areas depending on the model

5-Tmax-Tmin, Tmax, 18/07/1983 Tmax Tmin Tmin Tmean Tmax Tmin $11\overline{2}$ 109 Total 114 110 107 115 64 Total >75 62 64 62 60 65 CV 60 63 61 61 59 63 19 18 19 18 17 19 Resp. Tmax-Tmin, Tmax, 18-31/08/1984 Tmax Tmin Tmin Tmax Tmin Tmean 44 Total 53 57 46 42 51 35 39 43 33 39 Total >75 36 23 25 23 CV 18 20 18 19 20 21 22 19 21 Resp. Tmin, Tmax, 22-Tmax-30/06/1995 Tmax Tmin Tmax Tmin Tmin Tmean 49 47 51 Total 47 46 48 Total >75 32 31 30 32 31 33 CV 19 18 17 18 18 20 11 10 10 11 11 11 Resp. 01-Tmax-Tmin. Tmax, 22/08/1995 Tmin Tmin Tmin Tmax Tmean Tmax 5 2 -2 14 Total 11 4 -22 Total >75 -21 -23 -17 -24 -16 CV 6 6 11 6 4 11 6 7 6 8 6 7 Resp. Tmax, Tmax-04-Tmin, Tmin Tmin 12/08/2003 Tmax Tmin Tmean Tmax -45 -44 -42 -45 -46 -45 Total -32 -31 -30 -32 -33 -32 Total >75 -27 -26 -27 -27 -27 CV -27 -9 -10 -10 -10 -10 -10 Resp. 22-Tmax-Tmin, Tmax, 27/08/2003 Tmax Tmin Tmax Tmin Tmin Tmean -7 -7 -7 Total -6 -7 -8 Total >75 -12 -11 -11 -12 -12 -11 14 14 14 15 CV 15 15 Resp. -22 -22 -22 -22 -22 -22

5-			Tmax-		Tmin,	Tmax,
18/07/1983	Tmax	Tmin	Tmin	Tmean	Tmax	Tmin
Total	17	18	20	17	18	16
Total >75	13	14	15	13	14	13
CV	8	8	8	8	7	8
Resp.	5	5	7	5	5	5
18-			Tmax-		Tmin,	Tmax,
31/08/1984	Tmax	Tmin	Tmin	Tmean	Tmax	Tmin
Total	7	7	10	6	7	6
Total >75	16	15	17	15	16	14
CV	2	3	2	2	2	2
Resp.	1	1	2	1	1	1
22-			Tmax-		Tmin,	Tmax,
30/06/1995	Tmax	Tmin	Tmin	Tmean	Tmax	Tmin
Total	-16	-13	-13	-15	-14	-16
Total >75	-12	-10	-10	-11	-10	-11
CV	-10	-10	-10	-10	-10	-10
Resn	0	1	2	Ο	1	0
Kesp.	0	1	2	0	1	0
01-	0	1	Tmax-	0	Tmin,	Tmax,
01- 22/08/1995	Tmax	Tmin	Tmax- Tmin	Tmean	Tmin, Tmax	Tmax, Tmin
01- 22/08/1995 Total	Tmax -160	Tmin 70	Tmax- Tmin -15	Tmean -9	Tmin, Tmax -6	Tmax, Tmin -13
01- 22/08/1995 Total Total >75	Tmax -160 -11	Tmin 70 -8	Tmax- Tmin -15 -5	Tmean -9 -11	Tmin, Tmax -6 -9	Tmax, Tmin -13 -11
01- 22/08/1995 Total Total >75 CV	Tmax -160 -11 -9	Tmin 70 -8 -5	Tmax- Tmin -15 -5 -6	Tmean -9 -11 -7	Tmin, -6 -9 -9	Tmax, Tmin -13 -11 -6
01- 22/08/1995 Total Total >75 CV Resp.	Tmax -160 -11 -9 5	Tmin 70 -8 -5 7	Tmax- Tmin -15 -5 -6 9	Tmean -9 -11 -7 5	Tmin, Tmax -6 -9 -9 6	Tmax, Tmin -13 -11 -6 5
01- 22/08/1995 Total Total >75 CV Resp. 04-	Tmax -160 -11 -9 5	Tmin 70 -8 -5 7	Tmax- Tmin -15 -5 -6 9 Tmax-	Tmean -9 -11 -7 5	Tmin, Tmax -6 -9 -9 6 Tmin,	Tmax, Tmin -13 -11 -6 5 Tmax,
Rcsp. 01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003	Tmax -160 -11 -9 5 Tmax	Tmin 70 -8 -5 7 Tmin	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin	Tmean -9 -11 -7 5 Tmean	Tmin, Tmax -6 -9 -9 6 100	Tmax, Tmin -13 -11 -6 5 Tmax, Tmin
Ncsp. 01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003 Total	Tmax -160 -11 -9 5 Tmax 11	Tmin 70 -8 -5 7 7 Tmin 12	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin 14	Tmean -9 -11 -7 5 Tmean 11	Tmin, Tmax -6 -9 -9 6 12	Tmax, Tmin -13 -11 -6 5 Tmax, Tmax, Tmin 10
01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003 Total Total Solution	Tmax -160 -11 -9 5 Tmax 11 2	Tmin 70 -8 -5 -7 7 Tmin 12 2 2	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin 14 4	Tmean -9 -11 -7 5 Tmean 11 1 1	Tmin, Tmax -6 -9 -9 6 Tmin, Tmax 12 3	Tmax, Tmin -13 -11 -6 5 Tmax, Tmin 10 1
No. 01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003 Total Total CV CV Resp. 04- 12/08/2003 Total Total >75 CV	Tmax -160 -11 -9 5 Tmax 11 2 6	Tmin 70 -8 -5 7 Tmin 12 2 7	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin 14 4 6	Tmean -9 -11 -7 5 5 Tmean 11 1 6 6	Tmin, Tmax -6 -9 -9 6 79 6 12 3 6	Tmax, Tmin -13 -11 -6 5 Tmax, Tmin 10 1 6
Ncsp. 01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003 Total Total CV Resp. 04- 12/08/2003 CV Resp.	Tmax -160 -11 -9 5 Tmax 11 2 6 -2	Tmin 70 -8 -5 -5 7 Tmin 12 2 7 -7 -2	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin 14 4 6 0	Tmean -9 -11 -7 5 Tmean 11 6 -2	Tmin, Tmax -6 -9 -9 6 Tmin, Tmax 12 3 6 -1	Tmax, Tmin -13 -11 -6 5 Tmax, Tmin 10 1 6 -2
Nesp. 01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003 Total Total CV Resp. 04- 12/08/2003 Total CV Resp. 22-	Tmax -160 -11 -9 5 Tmax 11 2 6 -2	Tmin 70 -8 -5 -5 7 Tmin 12 2 7 -2 7	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin 14 4 6 0 Tmax- Tmax-	Tmean -9 -11 -7 5 Tmean 11 1 1 6 -2	Tmin, Tmax -6 -9 -9 6 Tmin, Tmax 12 3 6 -1	Tmax, Tmin -13 -11 -6 5 Tmax, Tmin 10 1 6 -2 Tmax,
Nesp. 01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003 Total Total CV Resp. 04- 12/08/2003 Total Total >75 CV Resp. 22- 27/08/2003	Tmax -160 -11 -9 5 Tmax 11 2 6 -2 Tmax	Tmin 70 -8 -5 7 Tmin 12 2 7 -2 Tmin	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin 14 4 6 0 Tmax- Tmax- Tmin	Tmean -9 -11 -7 5 Tmean 11 1 6 -2 Tmean	Tmin, Tmax -6 -9 -9 6 Tmin, Tmax 12 3 6 -1 Tmin, Tmin, 12 3 6 -1 Tmin, Tmin, Tmin, Tmin, Tmax	Tmax, Tmin -13 -11 -6 5 Tmax, Tmin 10 1 6 -2 Tmax, Tmin
Ncsp. 01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003 Total Total CV Resp. 04- 12/08/2003 Total Potal >75 CV Resp. 22- 27/08/2003 Total	Tmax -160 -11 -9 5 Tmax 11 2 6 -2 Tmax 9 9	Tmin 70 -8 -5 7 Tmin 12 2 7 -2 Tmin 8 8	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin 14 4 6 0 Tmax- Tmin 12 14 4 6 0 Tmax- Tmin 10	Tmean -9 -11 -7 5 Tmean 11 1 6 -2 Tmean 8	Tmin, 6 Tmax 6 -9 -9 6 -9 Tmin, 12 Tmax -1 Tmin, -1 Tmin, 8	Tmax, Tmin -13 -11 -6 5 Tmax, Tmin 10 1 6 -2 Tmax, Tmax, 10 1 6 -2 Tmax, Tmin 9
Nesp. 01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003 Total Total CV Resp. 22- 27/08/2003 Total Total Total	Tmax -160 -11 -9 5 Tmax 11 2 6 -2 Tmax 9 -2	Tmin 70 -8 -5 -5 7 Tmin 12 2 7 -2 7 Tmin 8 -3 8	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin 14 4 6 0 Tmax- Tmin 10 -2	Tmean -9 -11 -7 -7 5 Tmean 11 1 6 -2 Tmean 8 -3	Tmin, -6 -9 -9 -9 6 Tmin, -9 Tmin, 12 3 6 -1 -1 Tmin, 8 -3 8 -3 8 -3 -3	Tmax, Tmin -13 -11 -6 5 Tmax, Tmin 10 1 6 -2 Tmax, Tmax, 10 1 6 -2 Tmax, Tmin 9 -3
Nesp. 01- 22/08/1995 Total Total >75 CV Resp. 04- 12/08/2003 Total Total Total 2003 Total 22- 27/08/2003 Total Total Total CV Resp. 22- 27/08/2003 Total Total Total CV	Tmax -160 -11 -9 5 Tmax 11 2 6 -2 Tmax 9 -2 -11	Tmin 70 -8 -5 7 Tmin 12 2 7 -2 Tmin 8 -3 -1	Tmax- Tmin -15 -5 -6 9 Tmax- Tmin 14 4 6 0 Tmax- Tmin 10 -2 -15	Tmean -9 -11 -7 5 Tmean 11 1 6 -2 7 5 7 7 5 7 7 5 7 7 5 7 7 5 7 7 5 7 7 5 7 7 5 7 7 5 7 7 5 7 7 5 7 7 7 5 7 7 6 -2 7 6 -2 7 7 1 <th1< th=""> <th1< th=""> <th1< th=""></th1<></th1<></th1<>	Tmin, Tmax -6 -9 -9 6 Tmin, Tmax 12 3 6 -1 Tmin, Tmin, Tmin, 3 6 -1 Tmin, Tmax	Tmax, Tmin -13 -11 -6 5 Tmax, Tmin 10 1 6 -2 Tmax, Tmin 9 -3 -1

 Table A. 10. Central estimates of the excess mortality in urban areas depending on the

 model

Appendix7. Outputs of the models to estimate the risk associated with an increase in

temperature during summer

Rural areas – Total mortality

Rural areas – Total mortality>74

Rural areas – Cardiovascular mortality

cardio ~ s(tm, k = 3, bs = "cr") + s(tm17, k = 3, bs = "cr") + s(time, fx= TRUE, k = 20, bs = "cr") + as.factor(dow)Approximate significance of smooth terms: edf Ref.df Chi.sq p-value 1.603 1.834 7.159 0.0233 * s(tm) s(tm17) 1.753 1.931 38.936 3.10e-09 *** s(time) 19.000 19.000 714.229 < 2e-16 *** Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 R-sq.(adj) = 0.266Deviance explained = 27.5% UBRE score = 0.08676 Scale est. = 1 n = 2116

Rural areas – Respiratory mortality

Urban areas – Total mortality

total ~ s(tm, k = 3, bs = "cr") + s(tm17, k = 3, bs = "cr") + s(time, fx = TRUE, k = 20, bs = "cr") + as.factor(dow)

Urban areas – Total mortality>74

total75 ~ s(tm, k = 3, bs = "cr") + s(tm17, k = 3, bs = "cr") + s(time, fx = TRUE, k = 20, bs = "cr") + as.factor(dow)

Urban areas - Cardiovascular mortality

cardio ~ s(tm, k = 3, bs = "cr") + s(tm17, k = 3, bs = "cr") + s(time, fx = TRUE, k = 20, bs = "cr") + as.factor(dow) Approximate significance of smooth terms: edf Ref.df Chi.sq p-value s(tm) 1 1.001 0.182 0.67 s(tm17) 1 1.001 15.594 7.86e-05 *** s(time) 19 19.000 118.837 < 2e-16 *** ---Signif. codes: 0 `***' 0.001 `**' 0.01 `*' 0.05 `.' 0.1 ` ' 1 R-sq.(adj) = 0.0588 Deviance explained = 7.12% UBRE score = 0.043449 Scale est. = 1 n = 2024

Urban areas – Respiratory mortality

Appendix 8. Relative risks associated with a 1°C increase in the mean temperature above

15°C, rural areas

P	All summers			
	RR	Inf	Sup	р
Total mortality	1.014922	1.0088	1.021082	3.77E-05
Total mortality> 75	1.014051	1.006049	1.022116	0.0054
Cardiovascular				
mortality	1.010883	1.002858	1.018972	0.0233
Respiratory mortality	0.9997284	0.9892633	1.010304	0.96

Summers - Low-mortality winter

	RR	Inf	Sup	р
Total mortality	1.021923	1.012864	1.031063	4.52E-06
Total mortality> 75	1.027222	1.01485	1.039745	7.37E-05
Cardiovascular				
mortality	1.024415	1.011511	1.037483	0.00121
Respiratory mortality	1.0005094	0.9840734	1.0172199	0.95196

Summers -	High-mort	ality winter
-----------	------------------	--------------

	RR	Inf	Sup	р
Total mortality	1.008911	1.001354	1.016525	0.214
Total mortality> 75	1.002728	0.994712	1.010809	0.860709
Cardiovascular				
mortality	1.0017807	0.9945452	1.0090689	0.63
Respiratory mortality	1.0010327	0.9873775	1.0148767	0.883

	Total mortality				
	RR	Inf	Sup	р	
1981-1984	1.02583	1.013085	1.038734	0.001509	
1985-1988	1.024323	1.006853	1.042097	0.0542	
1989-1992	1.021497	1.010064	1.03306	0.0145	
1993-1996	1.010823	1.001086	1.020654	0.029346	
1997-2000	0.9888691	0.9794553	0.9983733	0.0218	
2001-2003	0.9991376	0.9886264	1.0097605	0.873	

Total mortality>74

	RR	Inf	Sup	р
1981-1984	1.02369	1.006906	1.040753	0.02166
1985-1988	1.021056	1.000275	1.042269	0.2559
1989-1992	1.029468	1.013685	1.045496	0.01912
1993-1996	1.0039011	0.9914273	1.0165319	0.5419
1997-2000	0.977752	0.9593317	0.9965259	0.176
2001-2003	0.9964386	0.9828483	1.0102167	0.6105

Cardiovascular mortality

	RR	Inf	Sup	р
1981-1984	1.035966	1.018374	1.053862	0.00166
1985-1988	1.0157155	0.9993584	1.0323403	0.120515
1989-1992	1.012538	0.99723	1.028082	0.51161
1993-1996	1.0037289	0.9896697	1.0179878	0.60517
1997-2000	0.9730955	0.9507238	0.9959937	0.19271
2001-2003	0.9917247	0.9750317	1.0087036	0.3373

Respiratory mortality

	RR	Inf	Sup	р
1981-1984	0.9956366	0.9704424	1.021485	0.73815
1985-1988	1.0163002	0.9718434	1.0627908	0.335749
1989-1992	1.0161567	0.9919704	1.0409328	0.19228
1993-1996	1.0019947	0.9760865	1.0285906	0.881527
1997-2000	0.9958975	0.9713621	1.0210526	0.747
2001-2003	0.9871389	0.9593029	1.0157827	0.375

Appendix 9. Relative risks associated with a $1^{\circ}C$ increase in the mean temperature above

15°C, urban areas

	RR	Inf	Sup	р
Total mortality	1.015533	1.006326	1.024824	0.003145
Total mortality> 75	1.015182	1.003081	1.027428	0.0442
Cardiovascular				
mortality	1.002435	0.992405	1.012566	0.67
Respiratory mortality	1.027533	1.005037	1.050533	0.02606

Summers - Low-mortality winter

RR	Inf	Sup	р
1.021657	1.004807	1.03879	0.1167
1.0128667	0.9969335	1.0290546	0.17557
1.00364	0.986236	1.021352	0.615
1.097757	1.046322	1.151721	0.0134
	RR 1.021657 1.0128667 1.00364 1.097757	RRInf1.0216571.0048071.01286670.99693351.003640.9862361.0977571.046322	RRInfSup1.0216571.0048071.038791.01286670.99693351.02905461.003640.9862361.0213521.0977571.0463221.151721

Summers - High-mortality winter

	RR	Inf	Sup	р
Total mortality	1.011609	1.003024	1.020267	0.00796
Total mortality> 75	1.015995	1.000871	1.031348	0.10638
Cardiovascular				
mortality	1.0031363	0.9906225	1.0158082	0.625207
Respiratory mortality	1.0225338	0.9975168	1.0481782	0.07792

Total mortality

	RR	Inf	Sup	р
1981-1984	1.05099407	1.02876329	1.07370524	0.000368
1985-1988	1.01680506	0.99839743	1.03555207	0.2942
1989-1992	1.00861473	0.99322572	1.02424219	0.27477
1993-1996	1.01559991	0.99932524	1.03213963	0.06037
1997-2000	0.99776326	0.9773762	1.01857558	0.42229
2001-2003	1.02079296	0.99536113	1.04687457	0.0364
	1.05099407	1.02876329	1.07370524	0.000368

Total mortality>74

	RR	Inf	Sup	р
1981-1984	1.01927834	0.99478566	1.04437406	0.2043
1985-1988	1.02491759	0.99819705	1.05235341	0.2815
1989-1992	1.01818506	0.99231254	1.04473215	0.495
1993-1996	1.02628304	1.00351097	1.04957185	0.023444
1997-2000	1.0053364	0.9836118	1.02754082	0.633
2001-2003	0.98868074	0.9630853	1.01495641	0.347

Cardiovascular mortality

	RR	Inf	Sup	р
1981-1984	1.01424801	0.99233759	1.03664219	0.23846
1985-1988	1.00067709	0.97464079	1.02740891	0.74598
1989-1992	1.01094158	0.98649777	1.03599106	0.807
1993-1996	1.01121592	0.98700196	1.03602393	0.36709
1997-2000	0.9864455	0.95998275	1.01363773	0.598
2001-2003	1.02852459	0.98763004	1.07111246	0.138

Respiratory mortality

	RR	Inf	Sup	р
1981-1984	1.13737777	1.07162962	1.20715978	0.00167
1985-1988	1.09558787	1.00908402	1.18950728	0.0483
1989-1992	1.01183705	0.96575978	1.0601127	0.621
1993-1996	1.0180961	0.97184404	1.06654939	0.449671
1997-2000	0.94266824	0.87739406	1.01279853	0.214
2001-2003	1.01791762	0.96434132	1.07447049	0.739

Appendix 10. Comparison of the GAM and the case cross-over modelling strategies

In the comparison, the risks were computed based on the assumption of a linear relationship between temperature and mortality during summer, even for temperatures below 15°C. This is why the risk estimates are lower than those computed for the temperatures above 15°C only.

In rural areas, the GAM and the case cross-over (CXO) models give the same estimates for all causes of mortality (Figure A.14), although CXO results are less significant, probably due to a problem of statistical power. Striking differences are observed however for the respiratory mortality, where the GAM founds no results and the CXO a significant estimate. Similar results are found in urban areas, with consistent estimates except for the respiratory mortality (Figure A.15), but CXO design tends to give higher estimates than the GAM design.



Figure A.14. % increase in mortality to a 1°C increase in mean temperature at lag 0 during summers in rural areas – comparison of modeling strategies



Figure A.15. % increase in mortality to a 1°C increase in mean temperature at lag 0 during summers in urban areas – comparison of modeling strategies

Appendix 11. How to integrate a climate change perspective into public health surveillance? – paper submitted to Public Health

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Abstract

Objectives: Climate change may be considered as a factor for change in the state of environment, exposures and pathogens, impairing the state of health among populations. Efficient health surveillance systems are required to support adaptation to climate change. Yet, despite a growing awareness, the public health surveillance sector as been little involved into adaptation plans. We proposed a method to assess the adaptation needs of health surveillance systems and to raise awareness about climate change in the public health community.

Methods: We animated a working group of surveillance experts of environmental health, chronic, and infectious diseases, those tasks were to define common objectives, to propose a framework and to apply it to relevant health risks in France.

Results: The framework helps organizing the available information on climate-sensitive health risks, making a distinction between three main determinants as follows 1) the environment, 2) the individual and social behaviors, and 3) the demography and health status. We illustrate the process using two examples; heat waves and airborne allergens.

Conclusion: Health surveillance systems allow triggering early warnings, creating databases useful to improve the scientific evidence of the health impacts of climate change, identifying and prioritizing needs for intervention and adaptation measures, and evaluating these measures. Adaptation required that public health professionals consider climate change as a concrete input in their daily practice, and that they create partnership with other disciplines.

Key words: Climate Change, Public Health, Surveillance

Introduction

A conceptualization of the links between climate change and health has been proposed by McMichael¹, and has been widely used to compile epidemiological evidence and to identify the main health risks associated with climate change. Several synthesis papers are available in the literature and provide good overview of the expected impacts ²⁻⁶. They also have been very useful in raising awareness and promoting climate change adaptation. Indeed, reframing climate change as a public health problem makes the issue more significant to the public, and may help putting it on the top of the political agenda ⁷.

Important conclusions of these papers are that climate change may be considered as a factor for change in the state of environments and pathogens, exposures to health risks, determinants of exposure, the state of health among populations and migration of populations. It could aggravate territorial imbalances and inequalities in healthcare, on a regional, national and a global scale. At the same time, there will be other major changes, such as population growth, urbanization... that increase the vulnerabilities of our societies.

Several voices already pointed the adaptation needs of the public health sector ⁸. Efficient health surveillance systems are required to support any adaptation to climate change. Surveillance in public health is "the continuous and systematic collection and the analysis and interpretation of data that is essential for the planning, implementation and evaluation of public health practices, closely allied with the diffusion, at the opportune moment, of such data to those that require it" ⁹. The development of a health surveillance system must

answer well-defined objectives in order to bring relevant information to support the planning and implementation of health-relevant actions, and evaluation of public health interventions ⁹. Surveillance of chronic and infectious diseases traditionally focuses on the occurrence of pathologies. Surveillance in the field of environmental health has a wider scope: it includes, for defined geographical areas, the surveillance of the occurrence of pathologies, but also of environmental data, of exposure, and of the exposure-impacts relationship. In a context of climate change, these health surveillance systems may allow 1) triggering early warnings, 2) creating databases useful to improve the scientific evidence of the health impacts of climate change, 3) identifying and prioritizing needs for intervention and adaptation measures, and 4) evaluating these measures.

Yet, despite a growing awareness, the public health sector as been little involved into adaptation plans, for many reasons among which are the complexity of the relationships between climate change and health, the perception that climate change is an environmental issue and not a health issue, the lack of awareness of the possible health impacts of climate change, and the lack of vision about possible needs for changes of practices in relation to climate change (surveillance, communication, health care, prevention). Public health professional also face several levels of practical, institutional and financial constraints ¹⁰, making climate change a low priority.

Methods

To increase the interest and involvement of the health sector, it is deemed needed to translate the challenges of the climate change into issues that health professionals are able to integrate in their daily practices. In terms of surveillance, this means that we need to needed to look carefully at the existing surveillance systems, to assess how they can contribute to climate change adaptation (i.e which information should be channeled and delivered to relevant stakeholders), and to assess the needs for new developments.

To do so, we animated a working group of surveillance experts of environmental health, chronic, and infectious diseases, those tasks were to define common objectives, to propose a framework and to apply it to relevant health risks in France. We described these three steps in this paper. For the sake of clarity, we illustrate the process using two examples; heat waves and airborne allergens.

Results

The need for a common culture on climate change

In our experience the concept of climate change was initially not well understood by the members of the working group. Consultations of several scientists working on climate change, including meteorologists, climate modelers, biologists, social scientists or historians, were used to develop a common vocabulary and background about climate change and climate-related risks.

Agreeing on vocabulary was an important preliminary step, with an extensive discussion on the terminology to be used, "global warming" or "global change", "climate change". "Global warming" was considered as misleading, as it gave the sense that the matter relates to increasing temperatures only. However, although temperature is the symbolic parameter of climate change, much more need be taken into consideration (humidity, cloud cover, rainfall, storms, sea level raising, etc...). "Global change" was considered to be a more appropriate to take into account the diversity of climatic changes and of their interactions with demographic and social changes. Yet, the term "climate change" was finally used as it was so deeply root in all discussion about national and local adaptation plans. However, we stated clearly that our approach encompassed climatic, environmental and societal changes.

On a second step, we clarified that climate is a statistical description in terms of the mean and the variability of meteorological parameters (temperature, precipitation, wind, etc.) in a given region, over time periods ranging between a few months to several thousands of years ¹¹. As far as climate is concerned, a change is defined by a significant statistical variation in the average state of the climate or by its variability over long periods (generally over decades or longer). Referring to a statistical definition was very useful to promote a concrete integration of climate change into epidemiological thinking. For instance, questions could be reframed from the "what would happen if the temperature increases by 2°C?" to "what would happen if the mean and variance of temperature were to change?"

A framework to organize the available information

A third step was to suggest a way to introduce climate change to public health professionals in charge of a surveillance system, through a workable framework. The choice of the framework was an iterative process. Starting from the modified DPSEEA (driving forces, pressures, state, exposures, health effects and actions), model, which takes into account the influence of social, economical and cultural backgrounds on exposure ¹², we decided to focus on the determinants of exposure, rather than on a meteorological parameter-health relationship. Indeed, working on determinants of exposure distinguished as 1) environment, 2) the individual and social behaviors, and 3) the demography and health status (Figure 1) allowed a more integrative approach, and was more intuitive to epidemiologists. It was also assumed that the surveillance of one determinant, even indirectly linked with climate, could be more efficient, practical and easy to implement than the monitoring of a weathersensitive indicator ¹³. It also allowed taking into consideration environmental and demographic evolutions that are likely to negatively affect the exposure, as well as individual and social behaviors that may either exacerbate or reduce the exposure.

In this framework, for a specific health topic, each determinant can be expanded based on the available knowledge and on selected hypothesis, as will be illustrated in our examples.

The last step was to introduce this framework to experts of health risks. Health risks were selected on the following criteria; existence of credible / reliable documentation about the role of the climate on the health outcomes, and plausibility of observing the risk in France. Criteria were filled based on a literature review (including grey literature) and from the expert knowledge of the working group with regards to environmental health, infectious

diseases and chronic diseases. The final selection was consistent with the literature review for Europe, i.e. extreme meteorological events, air pollution, airborne allergens, water pollution, UV radiation, and infectious diseases.

The example of heat waves

Although heat-related risks have been documented for long in the literature, the 2003 summer in France was a sharp reminder that extreme temperatures remain a considerable danger for developed countries. Nearly 15 000 extra deaths were recorded between the 1st and the 20th August 2003 ¹⁴. The most vulnerable groups were the elderly, people suffering from chronic diseases, those living alone or in social isolation ¹⁵. The urban population was most severely affected, and the urban heat island phenomenon has been found to be a major risk factor of mortality. Since 2004, a heat prevention plan has been developed. This plan is a good example of a reactive adaptation in response to a disaster. In terms of surveillance, it includes a surveillance of a meteorological indicator derived from temperature forecast, and a near real-time surveillance of selected heat-related health indicators. Both systems are dedicated to provide early warnings and to support decision-making during a heat wave ¹⁶.

Climate change is likely to result in an increase in the intensity and frequency of heat waves ¹⁷, and projections for France document a large increase in the number of heat wave days in 2030 and later, whatever the climate scenario. France will face a significant ageing of its population, and an increase in urbanization rates; the number of people aged 75 years

and older would increase from 4.2 to 8.3 millions between 2000 and 2030 and those aged 85 and older would increase from 1.2 to 2.4 millions 18 .

All this information was summarized in Figure 2. Given the available knowledge, it is reasonable to expect an increase in the vulnerable population exposed to heat-related health risks. Individual and social behaviors, housing and urban planning would play a key role in reducing the impact of heat waves. Therefore, the working group identified three axes of surveillance that could be useful in that context.

A first option would be to undertake the surveillance of the temperature-health exposureresponse function: can we quantify a change in this relationship that would support that the vulnerability of the population is changing? Can we identify new health endpoints, or new vulnerable populations that would require specific prevention? Can we use this information to update the warning system and ensure its efficiency over time? Despite extensive literatures on the temperature – mortality relationship ¹⁹, these questions remain unanswered.

Another option would be a surveillance of vulnerabilities, which can be divided into three types ²⁰: social vulnerabilities, such as isolation, environmental vulnerabilities associated with housing, and underlying health conditions. A surveillance of vulnerable populations would help in documenting if this population is really increasing and changing, and how it is geographically distributed. Examples of how to monitor vulnerable populations can be found in Italy where susceptible people are identified based on age, health conditions and

living conditions criteria and notified by GPs and social workers ²¹. Interesting initiatives based on simpler vulnerabilities' indicators, such as population density, population of people above 65 years old, population below poverty level, or urban heat island have been implemented in California ²² and Québec ²³. In France, data are available from several epidemiological studies performed after 2003, but which have not been repeated on recent heat waves. There working group identified a need for in-depth analysis of future episode, and opportunities to monitor vulnerabilities. For instance, each city must register people who wish to be contacted by social services during a heat wave (Law n°2004-626 30 June 2004, decree n° 2004-926 1st September 2004), but this information has not been used for epidemiological purposes. Regarding housing, the National Housing Survey includes detailed information on the characteristics of residential dwellings and the social and economic characteristics of the occupants. A proposition of the working group was to assess how a health related module could be added to such survey.

In addition, the working group concluded that dissemination of the knowledge deriving from the current surveillance system was sufficient toward health professionals. However, communication to professional of housing and urban planning was poor, although they can plan a significant role in reducing the long-term risk. Therefore, a dissemination plan dedicated to those professionals was developed.

The example of airborne allergens

Our second example refers to airborne allergens. The working group started from the position statement proposed by the European respiratory society on mould and pollens ²⁴.

They concluded that if climate change is likely to have an influence on respiratory diseases, the extent of the effect remains unclear.

Indeed, meteorological conditions encourage the production and dispersal of pollen, and the climate influences the existing species in a given geographical zone. Climate change is likely to bring about changes to the vegetation zones²⁵, prolonged periods of pollination, which has already been observed for certain species and even an increase in the quantities of pollen produced ²⁶. The end of the pollination period is often very late, with an average delay of 5 days over the whole continent of Europe and for all species ²⁷, even though there are exceptions for some plants and some localities. This is already leading to more prolonged periods of exposure to allergenic pollens. In addition, even though few studies have been carried out so far, the increase in temperatures is likely to render the pollen even more allergenic. This has been demonstrated in the case of the birch ^{28,29} and for the common ragweed Ambrosia artemisiifolia, ^{30,31}. The increase in levels of CO2, which goes hand-in-hand with global warming, is likely to increase the quantities of pollen produced ³² and their allergenicity ^{31,33}. Models produced for the ragweed pollen have demonstrated that the production of this pollen is likely to increase by 32-55% between 2070 and 2100, due to the increase in the CO_2 content of the atmosphere ³⁴. In addition, pollution can act on the allergenicity of pollen by increasing the number of allergens inside the grains and facilitating their exit ³⁵ or by accentuating the response to pollen ³⁶.

Climate change could also be associated with an increase of some indoor allergens, for instance, an increase in mould contamination in indoor air. For instance, mould is likely to

develop more easily in warmer climate, or to arise more frequently following extreme climatic events such as floods. However, as time spent indoor may also change, the overall impact on health is conjectural. Several meteorological parameters could enhance, or on the opposite limit mites.

In summary, as described in Figure 3, climate change should result in changes in exposure to airborne allergens, but without a clear direction. In that case, it is relevant to focus the surveillance efforts on the observation of the environment and of the main health outcomes.

Regarding the environment, surveillance of pollen counts is carried out by the French National Aerobiological Surveillance Network. Ragweed is the subject of a specific strategy for surveillance, prevention and early eradication in the areas where it still has a small presence, as well as monitoring of its growth.

In the indoor environment, surveillance data on the presence of mould and humidity levels as well as their origin would also be of value. The development of qualitative or semiquantitative methods to measure the presence of mould and humidity levels would enable this monitoring to be carried out.

Epidemiological surveillance of asthma is carried out in France through regular monitoring of prevalence of asthma, mortality, hospitalizations and emergency visits ^{37,38}. To enhance the current surveillance of asthma, regular measurements of the prevalence of sensitization

to various allergens within the general population could be envisaged. Such surveys should allow the spread of allergic diseases within the community to be monitored.

Discussion

This framework was applied to several other risks with the objectives of disseminating knowledge about climate change to health professionals, and to help them assessing the needs for adaptation of surveillance systems ^{39 40}.

Given the surveillance systems available in France, a majority of the identified risks are already covered by existing environmental or health surveillance. In order to be able to monitor climate change it is essential to maintain and strengthen these systems and to ensure their sustainability. A general conclusion was the need for a better integration of the environmental and health databases, and if possible to promote interdisciplinary. This interdisciplinarity is needed, for example, to improve the collection of environmental data representative of exposure (e.g. with environmental disciplines), or to understand the role of behavior patterns in exposure (e.g. with social scientists). Collaboration with and between environmental specialists and veterinarians is an asset to look at the evolution of parasites and vectors of diseases, considering changes in temperature as well as in ecological conditions due to changes in human activity (e.g. deforestation). Yet, efficient interdisciplinarity for action is a long term process, as will be adaptation of the public health sector to climate change. Therefore, we consider that the most valuable outcome of our approach was to raise awareness of the public health and surveillance experts about

climate change, so that they consider it as a concrete input in their daily practice.

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Appendix 12. Publications and communications

Pascal M., Schwarts J, Clancy C, Goodman P, Heat waves and mortality in Dublin Ireland: 1981 - 2003 International Conference of Environmental Epidemiology, 12-16 october 2008

Beaudeau, P., Pascal, M., Mouly, A., Galey, C., & Thomas, O. 2011, "Health risk associated with drinking water in a context of climate change in France: a review of surveillance needs", *Journal of Water and Climate Change*, vol. 2, no. 4, pp. 230-245

Pascal M., Viso A-C., Medina S., Delmas M-C, Beaudeau P., **How to integrate a climate** change perspective into public health surveillance?, submitted to Public Health

Antics A., Pascal M., Laaidi K., Wagner V., Corso M., Declercq C., Beaudeau P., A simple indicator to monitor the short-term impact of heat waves on mortality in France, submitted to International Journal of Biometeorology

Pascal M., Wagner V., Le Tertre A., Laaidi K., Honoré C., Bénichou F., Beaudeau P.,Definition of temperature thresholds: the example of the French heat wave warning system, accepted by International Journal of Biometeorology.