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Causes for the recent changes in cold- and heat-related mortality in England and Wales

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Abstract Cold related mortality among people aged over 50 in England and Wales has decreased at a rate of 85 deaths per million population per year over the period 1976–2005. This trend is two orders of magnitude higher than the increase in heat-related mortality observed after 1976. Long term changes in temperature-related mortality may be linked to human activity, natural climatic forcings, or to adaptation of the population to a wider range of temperatures. Here we employ optimal detection, a formal statistical methodology, to carry out an end to end attribution analysis. We find that adaptation is a major influence on changing mortality rates. We also find that adaptation has prevented a significant increase in heat-related mortality and considerably enhanced a significant decrease in cold-related mortality. Our analysis suggests that in the absence of adaptation, the human influence on climate would have been the main contributor to increases in heat-related mortality and decreases in cold-related mortality.

1 Introduction

Human health is affected in an array of ways by climatic variability and change. Changes in mortality from cardiovascular and respiratory illnesses in hot or cold weather are among the direct physiological impacts (Basu and Samet 2002; Diaz et al.

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2002; Donaldson and Keatinge 1997; Hajat et al. 2004; Huynen et al. 2001; Keatinge et al. 2000a). The transmission of infectious diseases is also known to accelerate with warming and to be sensitive to rainfall changes (Curriero et al. 2001; Rose et al. 2001; Singh et al. 2001). This relationship is manifest during El Niño events (Hales et al. 1996; Malezer et al. 1999). In addition to natural variability, climate change due to human activity poses a major concern (McMichael et al. 2006; Patz et al. 2005). The Department of Health in the UK recently published a report on the health effects of climate change in the country (Kovats 2008). Apart from rising temperatures, various other aspects of climate change affect human health. Changes in droughts, floods, storms and land use affect regional food yields which could be a precursor of malnutrition or hunger (Hari Kumar et al. 2005; McMichael 2001). Together with sea level rise these factors are also linked to exposure to storm surges (Guha-Sapir et al. 2004), degradation of water availability and quality (Schwartz and Levin 1999), population displacement (Nicholls and Tol 2006) and poor sanitation (Sur et al. 2000). Air quality, as a final example, is degraded by emissions of air pollutants like ozone associated with a range of respiratory diseases (Ito et al. 2005). A comprehensive overview of the impacts of climate change on health is given in Confalonieri et al. (2007) in the fourth assessment report (AR4) of the Intergovernmental Panel on Climate Change (IPCC). The interplay between health and climate change, including both its direct effects (i.e. rising temperatures), as well as the resulting socio-economic, political and demographic disruptions, is multi-faceted and complex. In order to be better understood, a multidisciplinary approach is required that facilitates collaboration between experts in a range of fields. This paper provides such an example that reports results from a joint medicine and climate science project.

There is a wealth of evidence for changes in cold- and heat-related mortality during the past few decades for cities or small sub-continental regions mainly in Europe, Australia and the United States (Davis et al. 2004; Donaldson et al. 2003; Guest et al. 1999; Keatinge et al. 2000b; Medina-Ramón and Schwartz 2007). In response to anthropogenic climate change, one would expect that warmer summers lead to increases in mortality, while milder winters to decreases. The overall effect, however, is also influenced by better adaptation of the population to extreme temperatures. In the UK, for example, heat-related mortality showed no such trend during 1971–2003, which could be indicative of adaptation, while cold-related mortality decreased by more than 33% (Kovats 2008). Although the overall effect appears to be beneficial, the numerous other impacts of climate change on human health, like those already mentioned earlier, are almost invariably detrimental. Moreover, even if the synergy between adaptation and milder winters decreases the total mortality related to cold and heat, extreme events like heatwaves may still exert a stress beyond the adaptation limits on the population. Such events are accompanied by sharp increases in daily mortality which cause public concern and attract ample media attention. A well studied example is the 2003 European heatwave which cost the lives of more than 30,000 people (Conti et al. 2005) with almost half the deaths in France (Vandentorren and Empereur-Bissonnet 2005). Even in the less affected area of England and Wales, there was a 16% increase in mortality during August 2003 (Health Statistics 2006). Stott et al. (2004) estimated that climate change has at least doubled the risk of an event like the 2003 European heatwave. Climate model projections show that in the coming decades heatwaves will increase both in

frequency (Stott et al. 2004; Jones et al. 2008) and in intensity (Christidis et al. 2005). Local effects like the urban heat island give rise to additional warming and therefore exacerbate the impact of high temperatures on health (Frumkin 2002). Adaptation to extreme temperatures becomes a challenge, especially to poorer countries. These are more vulnerable because of limited access to air-conditioning, less developed health care and perhaps insufficient public awareness.

The IPCC AR4 states with very high confidence that climate change contributes to the global burden of disease and to increased mortality (Confalonieri et al. 2007). Links between health-related changes and possible causes, however, have so far been derived in a more empirical than formal way. Formal attribution is made difficult because of (a) the effect of natural variability which is superimposed on the anthropogenic signal, (b) the lack of long term health data with a good coverage, (c) the effect of adaptation which can vary considerably geographically depending on economic, social and political factors. The World Health Organisation (WHO) carried out the most comprehensive assessment exercise that aimed to estimate the global burden of disease attributable to climate change (World Health Organisation 2002; McMichael et al. 2004). The assessment employed climate-health models for a range of health outcomes (cardiovascular diseases, malaria, diarrhoea, malnutrition etc) in conjunction with climate data from a General Circulation Model (GCM) to calculate the burden of disease due to climate change by the year 2000 and also in the future by 2030. The study was limited to quantifiable health outcomes only and was constrained by the efficiency of the models and the GCM employed. The results were reported relative to the baseline period of 1961–1990. It should be noted that human activity had already had an impact on the climate during this period. A formal attribution study would combine both observations and model estimates of health outcomes to statistically separate the components of the response to external climatic forcings. This paper provides a first example of such an approach based on optimal detection, a method that has served as the basis of the IPCC attribution statements for changes in climatic parameters like temperature (Hegerl et al. 2007). We concentrate on cold and heat-related mortality changes in England and Wales and consider only people aged over 50, as mortality is less sensitive to temperature in younger age groups. Unlike most of the published applications of optimal detection, mortality is not part of the standard GCM output, so a transfer function is required to convert GCM data, in this case temperature, to mortality. This extra step enables an "end-to-end" attribution investigation that combines information from observed mortality data and GCM derived estimates in a rigorous statistical context. It is envisaged that similar end-to-end applications will gain popularity in studies of climate change impacts, and will thus provide a consistent research framework with climate change science.

In this work we investigate the role of three factors in recent changes in mortality, namely anthropogenic and natural influences on the climate and adaptation. The effect of different degrees of adaptation to a wider range of temperatures is also examined. Adaptation to hot and cold weather can be either physiological or behavioural. Examples of physiological adaptation to cold include early and more vigorous shivering and more pronounced constriction of superficial blood vessels that will reduce conductive and convective heat loss. Behavioural adaptation results from actions taken against cold or heat stress and, unlike physiological adaptation, can operate in a preventive manner, i.e. before the stress is actually experienced.

Examples of behavioural adaptation to cold include constructing better heated and/or insulated houses, shopping in heated malls rather than an unheated high street during cold weather, vaccination against winter influenza epidemics etc. Behavioural adaptation resulting from increasing individual affluence leading to better health, housing and diet and, at population level, from better housing and healthcare would make people more resistant to extreme weather, even in absence of climate change. It is the effect of such factors that we consider in this study when we investigate the impact of adaptation.

The remainder of the paper is structured as follows. In Section 2 details on the mortality and temperature data used in the analysis are given. Section 3 discusses the methodology. First, we describe the mortality-temperature model, which provides the transfer function. This also takes adaptation into account. We then briefly introduce optimal detection which measures the anthropogenic and natural components of the mortality change. Results from the analysis are shown in Section 4. A summary of the results and a discussion on the findings are given in Section 5.

2 Data

Daily deaths from all causes were extracted from death registration data supplied by the Office of National Statistics for men and women aged over 50 in England and Wales for the period 1976–2005. These were divided by daily estimates of population obtained by fitting a fifth order polynomial to mid-year population estimates, to give mortality as deaths per million people, which is used in this paper. The daily temperature observations used in the analysis are from the record of Central England Temperature (CET), the longest available instrumental record that spans more than 300 years (Manley 1974; Parker and Horton 2005). CET for the reference period is computed using surface air temperature observations taken at four stations (three stations after 2004). The data show a warming trend during the same period of 0.47 degrees per decade. Karoly and Stott (2006) reported a very similar value of 0.42 degrees per decade and also showed that the observed warming since the 1950s cannot be explained by natural climate variations, but is consistent with the response to anthropogenic forcings.

Daily temperature model data are taken from experiments with the Met Office Hadley Centre coupled Atmosphere-Ocean GCM, HadGEM1 (Johns et al. 2006; Stott et al. 2006). Two experiments are considered here which will be referred to as: a) ALL, forced with historical changes in well-mixed greenhouse gases, sulphate aerosols (indirect effect included), tropospheric and stratospheric ozone, industrial black carbon, biomass burning aerosols, as well as land use changes and natural forcings (volcanic aerosol emissions and changes in the solar output) and b) ANTHRO, which includes the same forcings as ALL, except for the natural ones. Each experiment is an ensemble of simulations that start from well separated points of a long control simulation without external forcings. In total, there are three runs for the ALL and 4 runs for the ANTHRO experiment. Averaging across the ensemble members reduces the effect of internal climate variability and thus the ensemble mean is used to represent the characteristic fingerprint of the forcings considered in each experiment. The control simulation is also used to give an estimate of the internal climate variability. Eight hundred twenty years of the HadGEM1

control are used for this purpose. As the length of the simulation may not be sufficient to represent variability on long timescales, we also used 1760 years of a control simulation with the predecessor of HadGEM1, the HadCM3 model (Tett et al. 2002). Power spectra analyses (Stott et al. 2000) show that the variability estimated from both models is consistent with the observed variability. Estimates of the CET with HadGEM1 come from the average of three grid box values in the area where the observations are taken. The grid boxes lie mainly over land. For HADCM3, which has a coarser resolution, CET is estimated using a single grid-box, as in Karoly and Stott (2006).

3 Methods

As temperature-related mortality is not part of our GCM output, the relationship between temperature and mortality is used as a transfer function to provide the model estimates. This relationship displays a characteristic U-shape (Kalkstein 1993; Donaldson et al. 2003) with the vicinity around its minimum representing the thermo-comfort zone (Fig. 1). This relationship varies greatly with location and also among different population groups, with elderly people being more vulnerable to temperatures outside the comfort zone (McGeehin and Mirabelli 2001). Keatinge et al. (2000b) showed that the slope of the line on the right of the comfort zone is less steep in warmer places, while the slope of the line on the left is less steep in colder places. The comfort zone itself is shifted to higher (lower) temperatures at warmer (colder) places. These changes over time illustrate the effect of acclimatisation and/or adaptation. The comfort zone is represented here by a 3°C band around

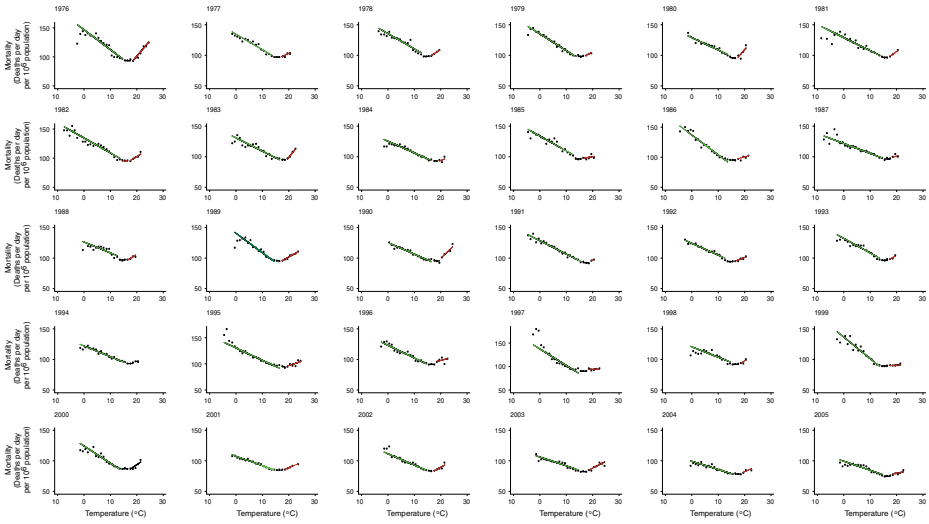

Fig. 1 Changes in daily mortality per 10^6 population for people aged over 50 in England and Wales with daily temperature for each year in period 1976–2005. The *purple line* is the 3°C minimum mortality band representing the comfort zone. Linear fits to the data are shown for the cold (*green*) and the hot (*red*) line, for temperatures below and above the comfort zone respectively

the minimum mortality and corresponds to the baseline mortality (Keatinge et al. 2000b). For temperatures on the left (right) of the 3°C band the cold- (heat-) related mortality is measured as the difference between the actual mortality and the baseline value and is often referred to as excess mortality. The two arms of the line on either side of the comfort zone are often modelled using simple linear relationships (Guest et al. 1999; Keatinge et al. 2000b), while non linear models have also been employed (Dessai 2002; Gosling et al. 2007). The 3°C band was identified by calculating the mortality in successive 3°C bands, incremented by 0.1°C, from a starting temperature of 15°C for the upper end of the band. One important issue to consider in mortality-temperature models is the effect of adaptation to a wider range of temperatures. Stationary models, which assume no adaptation, cannot adequately represent a warming world (Davis et al. 2003) and would therefore lead to misleading future predictions. Although there are simple techniques to remove the effect of adaptation (Davis et al. 2004), we will here attempt to explicitly investigate its effect rather than remove it.

We use simple linear models to represent the cold and hot line of the mortality-temperature relationship in England and Wales for people aged over 50. Although this representation does not provide a smooth transition from cold- to heat-related mortality in the vicinity of the comfort zone, this part of the curve makes the least contribution to the estimated excess mortality. As the relationship is expected to change with time because of adaptation, a different model is considered for each year in period 1976–2005. Hence, adaptation is represented here explicitly without relying on statistical estimates of its effect as in previous studies (e.g. Davis et al. 2004). A caveat of this approach is that the modelled mortality estimates cannot be extrapolated into the future, but this is beyond the scope of our attribution analysis which aims to trace the origins of changes that have already taken place. Figure 1 shows the daily mortality data plotted against observed CET values for each year together with the regression lines. The 3°C minimum mortality band is also marked on each plot. Details of the models are given in Table 1. Even though the position of the comfort zone has not changed much over the period considered here, the slopes of the hot and cold lines display a considerable decrease in steepness with time, implying that the population is able to cope better with heat and cold in the more recent years. Timeseries of the annual temperature-related mortality constructed by applying the models in Table 1 to daily CET data are well correlated with timeseries from actual mortality observations (correlation coefficient higher than 0.9). Our results show that linear regression, despite its simplicity, is able to provide a reasonable representation of the mortality dependence on temperature.

In our attribution analysis we use timeseries of three indices, which will be referred to hereafter as cold-related mortality (CRM), heat-related mortality (HRM) and total mortality (TM). CRM is computed by adding together the excess daily mortality for all the days in a year with temperatures below the comfort zone. HRM is calculated in a similar manner for temperatures above the comfort zone. TM is the total annual mortality related to heat and cold and is simply the sum of HRM and CRM. The annual index values are reported in this paper as anomalies relative to the mean of the whole period. Model estimates of daily mortality are computed by applying the linear relationships summarised in Table 1 to GCM daily temperature data. These are then used to obtain the annual model values of the three indices for the ALL, ANTHRO and the control experiments.

Table 1 Regression coefficients (with their standard error in parenthesis) that model the relationship between daily mortality per million population and daily temperature for each year in 1976–2005. The cold and hot lines correspond to temperatures above and below the 3°C mortality band respectively. The top end of the band is also given

Year	Slope of the cold line (SE)	Intercept of the cold line (SE)	Slope of the hot line (SE)	Intercept of the hot line (SE)	Top end of the 3°C band
1976	−3.437 (0.243)	146.830 (2.127)	5.380 (0.574)	−6.294 (11.760)	18.2
1977	−2.547 (0.147)	134.566 (1.268)	2.146 (1.200)	60.353 (21.738)	16.9
1978	−2.476 (0.185)	135.447 (1.488)	2.755 (0.796)	54.472 (13.543)	15.7
1979	−2.721 (0.103)	134.956 (0.870)	2.347 (1.017)	58.605 (18.513)	17.4
1980	−2.041 (0.116)	128.921 (1.026)	5.129 (1.479)	6.794 (27.618)	17.4
1981	−2.133 (0.139)	128.961 (1.153)	2.968 (1.087)	46.861 (19.203)	16.6
1982	−2.568 (0.145)	134.667 (1.272)	2.768 (0.780)	47.476 (14.713)	17.4
1983	−2.158 (0.141)	130.560 (1.335)	5.394 (0.878)	−9.020 (18.304)	19.5
1984	−1.911 (0.093)	125.344 (0.875)	1.669 (2.467)	60.403 (50.106)	19.5
1985	−2.435 (0.159)	133.742 (1.206)	0.798 (0.613)	84.188 (10.503)	16.0
1986	−3.303 (0.142)	136.853 (1.191)	1.304 (0.667)	74.546 (12.395)	16.6
1987	−1.624 (0.090)	121.536 (0.813)	1.186 (1.202)	77.235 (22.481)	17.7
1988	−1.785 (0.164)	126.323 (1.354)	2.655 (0.687)	53.032 (11.911)	16.0
1989	−3.185 (0.371)	140.572 (3.227)	2.426 (0.477)	53.291 (9.108)	17.5
1990	−1.898 (0.130)	125.357 (1.327)	4.155 (1.393)	16.440 (30.025)	19.6
1991	−2.184 (0.129)	128.329 (1.150)	1.233 (1.820)	72.185 (36.000)	18.8
1992	−1.931 (0.171)	124.132 (1.306)	1.641 (0.495)	68.151 (8.416)	15.7
1993	−2.375 (0.174)	132.143 (1.476)	2.449 (1.214)	55.459 (21.807)	16.9
1994	−1.745 (0.114)	122.028 (1.131)	0.972 (1.836)	77.485 (35.991)	18.6
1995	−2.349 (0.110)	130.309 (1.086)	1.689 (0.629)	62.952 (13.451)	19.5
1996	−2.085 (0.158)	123.117 (1.395)	1.118 (1.202)	76.032 (22.982)	18.0
1997	−3.510 (0.213)	137.347 (2.027)	0.070 (0.672)	92.929 (13.757)	18.4
1998	−1.695 (0.177)	120.580 (1.674)	2.616 (1.145)	45.290 (21.688)	17.9
1999	−3.512 (0.328)	136.507 (2.922)	0.327 (0.445)	84.177 (8.317)	16.8
2000	−2.803 (0.312)	124.134 (2.742)	2.900 (0.634)	35.678 (11.539)	17.0
2001	−1.552 (0.087)	107.589 (0.792)	1.829 (0.627)	52.075 (12.633)	18.1
2002	−1.851 (0.129)	111.507 (1.232)	2.332 (0.603)	42.832 (11.242)	17.5
2003	−1.434 (0.101)	106.206 (0.887)	2.207 (0.518)	44.034 (10.256)	17.6
2004	−1.359 (0.104)	99.376 (1.039)	1.984 (0.863)	42.512 (16.895)	18.6
2005	−1.489 (0.112)	99.383 (1.051)	0.866 (0.970)	62.352 (19.232)	18.2

Our attribution analysis is based on optimal detection (Allen and Stott 2003), a powerful statistical tool that underpins the IPCC attribution statements linking climate change to human activity (Hegerl et al. 2007). Optimal detection is a multiple regression model that represents the observations vector, y, as the linear sum of m model fingerprints, x_i, taking into account the noise of the internally generated variability, u_0, and the noise in the model vectors, u_i:

$$y = \sum_{i=1}^{m} (x_i - u_i)\beta_i + u_0. \tag{1}$$

In our case two experiments are considered ($m = 2$) and so Eq. 1 becomes:

$$y = (x_{\mathrm{ALL}} - u_1)\beta_1 + (x_{\mathrm{ANTHRO}} - u_2)\beta_2 + u_0. \tag{2}$$

The control simulations provide the covariance matrix of u_0 and u_i and the observations and fingerprints are projected onto the leading eigenvectors of the control variability. Detection is optimised by giving more weight to modes with high signal to noise ratios. A standard consistency test (Allen and Tett 1999) confirms that residual variability is consistent with the model derived internal variability. Details on the method and examples of applications are available in numerous papers (see Hegerl et al. 2007 and references therein).

The scaling factor for each model experiment, βi, measures the level of consistency between the model fingerprint and the observations. The amount of scaling required for an individual fingerprint does not necessarily depend on the amount of scaling required for the other fingerprints. If the 5–95% uncertainty range of the scaling factor is inconsistent with zero then the signal is considered detectable. Values consistent with unity and with a small uncertainty range imply good agreement between the model and the observations. Here we attempt to partition the response of the climate to external forcings between its anthropogenic and natural components. To do this, we assume that the fingerprint from the ALL experiment, x_{ALL}, is the sum of the anthropogenic and natural fingerprints $x_{\mathrm{ANTHRO}} + x_{\mathrm{NAT}}$ and Eq. 2 becomes:

$$y = (x_{\mathrm{ANTHRO}} + x_{\mathrm{NAT}} - u_1)\beta_1 + (x_{\mathrm{ANTHRO}} - u_2)\beta_2 + u_0. \quad (3)$$

Hence the anthropogenic and natural scaling factors are calculated from Eq. 3 as $\beta_{\mathrm{ANTHRO}} = \beta_1 + \beta_2$ and $\beta_{\mathrm{NAT}} = \beta_1$. The scaled, noise-reduced fingerprints, represented by the terms in parenthesis in Eq. 2, define the anthropogenic and natural components of the observed changes in temperature-related mortality.

4 Results

The observations and model signals (y and x_i in Eq. 1) used in this analysis are timeseries of annual values of the three indices (CRM, HRM and TM) expressed as anomalies relative to the 1976–2005 mean. Adaptation to a wider range of temperatures is represented by the change in the mortality-temperature relationship from year to year. Two hypothetical scenarios are also examined here. These are the "no adaptation" case, which assumes that no adaptation has taken place since 1976, and the "early adaptation" case, which assumes the same level of adaptation as in 2005 throughout the reference period. The signal in these two cases is constructed by applying the 1976 and 2005 transfer functions to the model CET data, or to the observed CET data (reconstructed mortality observations). We use these two cases to separate the effect of adaptation and external climatic forcings on mortality and examine how the latter might have contributed to mortality changes independent of adaptation. The timeseries of the three indices for all the cases are shown in Fig. 2. The hatched areas represent the 5–95% uncertainty range estimated using 30 non-overlapping segments of the control simulations and t-statistics. CRM and TM decrease with time and the decrease is markedly steeper in the actual adaptation case (left column of panels in Fig. 2). Both adaptation and the increasingly milder winters can explain such a reduction in mortality. These two effects, however, play a different role in the cases considered here. In the actual adaptation case (left column) both effects are at work, except of course in the control climate, which has no expected warming trend. Inspection of the hatched areas in the CRM and TM panels suggests

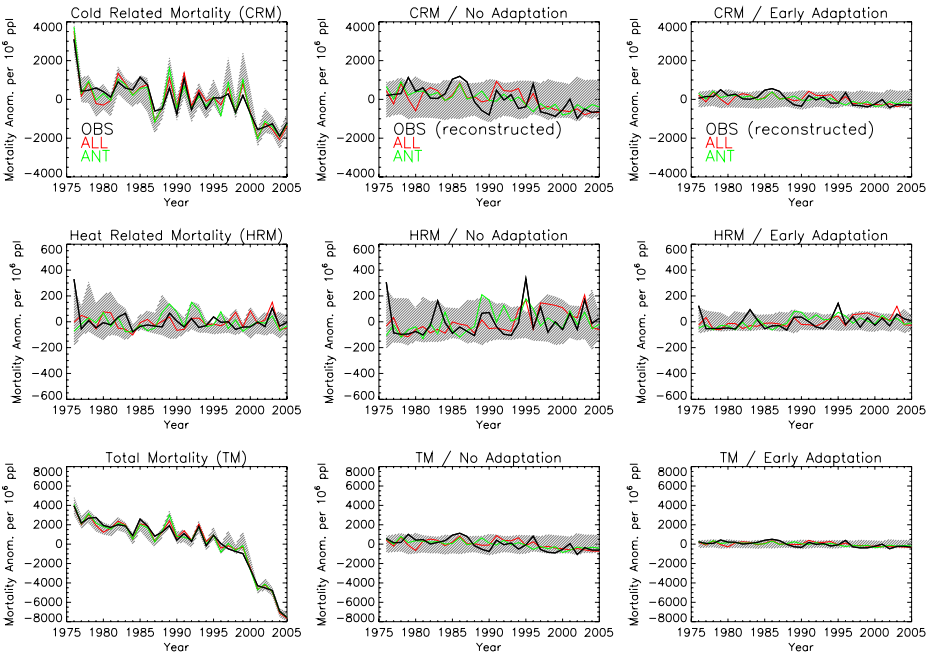

Fig. 2 Annual values of the CRM (*top row*), HRM (*middle row*) and TM (*bottom row*) indices expressed as anomalies relative to the 1976–2005 mean for the observations (*black lines*), the ALL experiment (*red lines*, ensemble mean) and the ANTHRO experiment (*green lines*, ensemble mean). The hatched area on each plot marks the 5–95% uncertainty range. The timeseries are shown for three cases, i.e. actual adaptation (*left column*), no adaptation (*middle column*) and early adaptation (*right column*). The observations in the last two cases are reconstructed from the observed CET using the transfer functions for 1976 and 2005

that there is a large reduction in mortality even in this control climate that can only be explained by a better adjustment of the population to cold with time. On the other hand, in the cases with adaptation fixed at the levels of a specific year (middle and right columns), it is only the effect of warmer winters that would account for the negative trend in mortality. The considerably smaller decreases in CRM and TM in these cases imply that warming is less effective than adaptation in reducing mortality. As expected, the decrease is smaller in the early compared to the no adaptation case, as better adjustment to cold throughout the period masks to some degree the benefit of the warming. HRM starts off with a high value in 1976, the year with the largest heatwave in the reference period, followed by a small positive trend in the remaining period. Nevertheless, the decrease in CRM far outweighs the changes in HRM. This finding is consistent with previous work (Kovats 2008). Mortality observations show a decrease in CRM of 85 deaths per million population per year over the period 1976–2005 and an increase in HRM of only 0.7 deaths per million population per year over the period 1977–2005. If no adaptation had taken place, the decrease in CRM would have been more moderate (47 deaths per million population per year over the period 1976–2005) and the increase in HRM more prominent (1.6 deaths per million population per year over the period 1977–2005).

Although the timeseries of the three indices shown in Fig. 2 lie within the range of internal variability, the cases with adaptation fixed at a certain level display a trend that is absent in a climate without external forcings. This is more clearly illustrated in Fig. 3, which shows the trends for the observations and the two forcing experiments together with internal variability, represented by a probability density function (PDF). The PDF is estimated using again control segments and *t*-statistics. All the ANTHRO trends for no adaptation and early adaptation are significant at the 10% level and so are most cases for ALL and the observations. The effect of human activity under these adaptation scenarios would therefore be expected to be detectable. This will be more rigorously tested using optimal detection. When the actual adaptation to more extreme temperatures is considered (left column panels in Fig. 3), the index trends lie within the range of internal variability and the control PDFs are not centred around zero. Even in this case, however, the trends tend to shift near the 10% level and one might expect that they would become significant, given either a small amount of extra warming, and/or a somewhat smaller degree of adaptation.

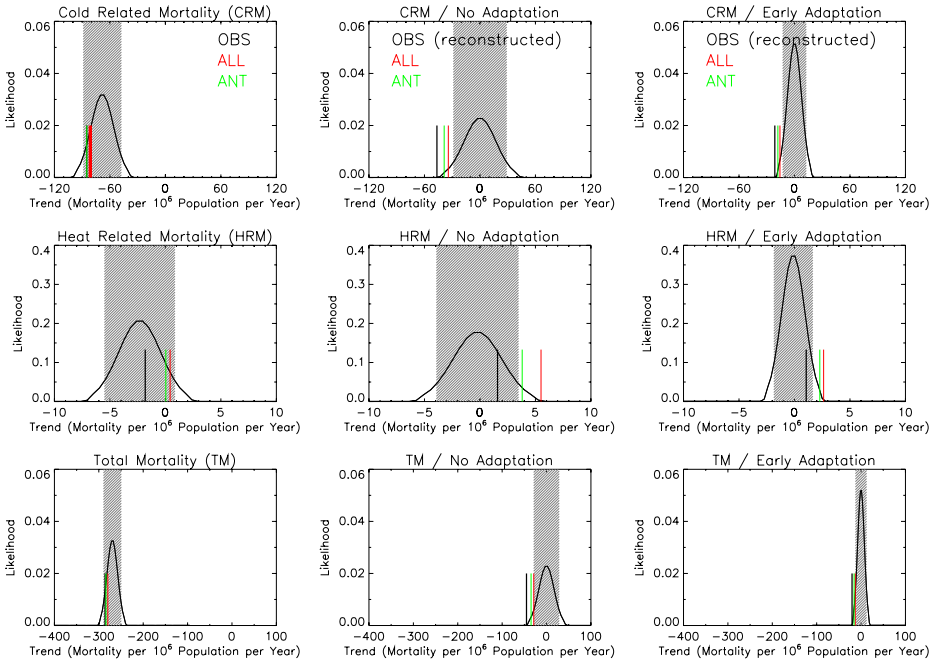

Fig. 3 Trends in the annual values of the CRM (*top row*), HRM (*middle row*) and TM (*bottom row*) indices for the observations (*black lines*), the ALL experiment (*red lines*, ensemble mean) and the ANTHRO experiment (*green lines*, ensemble mean). The PDFs representing internal variability are also illustrated (*bell-shaped lines*) and the hatched area on each plot marks the 5–95% uncertainty range. The trends are shown for three cases, i.e. actual adaptation (*left column*), no adaptation (*middle column*) and early adaptation (*right column*). The observations in the last two cases are reconstructed from the observed CET using the transfer functions for 1976 and 2005

4.1 Optimal detection analysis

Optimal detection compares model results with observations, taking into account the trend in the timeseries as well as their shape and optimises the signal to noise ratio given the uncertainty due to internal climate variability. Here we apply the method to separate the anthropogenic and natural components of the response, as explained in Section 3, and obtain the scaling factors plotted in Fig. 4 for the no adaptation and early adaptation cases. The results show that in the two hypothetical cases with fixed adaptation the anthropogenic signal might have been detectable for all indices and the natural signal might have been detectable for HRM. The uncertainties in the natural scaling factors are higher, except for HRM and the early adaptation case, and the anthropogenic scaling factors are always consistent with unity. Given the small trend in the HRM index and considering that the natural forcings are weaker than the anthropogenic ones during the reference period, the potential detection of the natural signal is an interesting and perhaps unexpected finding. Much of the natural signal stems from a cooling following the eruption of Mt Pinatubo in 1991, evident in the summer CET timeseries, which could lead to a detectable reduction of HRM.

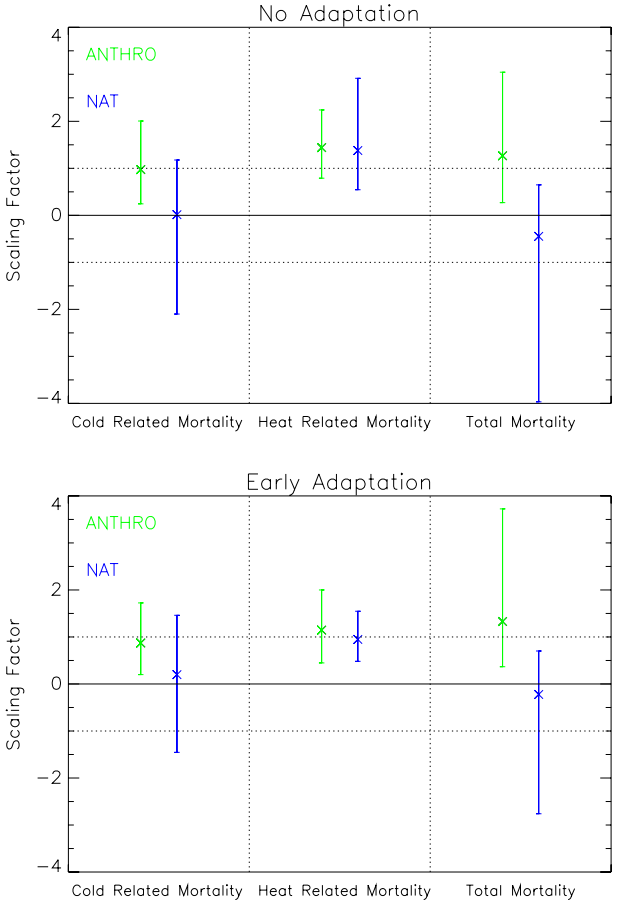

Fig. 4 Scaling factors and their 5–95% uncertainty range from an optimal detection analysis. The response is decomposed between its anthropogenic and natural components (scaling factors plotted in *green* and *blue* respectively). The top plot corresponds to the no adaptation case and the bottom plot to the early adaptation case. Results are shown for changes in CRM (*left section of each plot*), HRM (*middle section*) and TM (*right section*)

There is an indication of this in Fig. 2, which shows that HRM in the ALL experiment is lower than in the ANTHRO and more consistent with the observations. It is important to examine whether the results shown in Fig. 4 are sensitive to the year at which the adaptation levels are fixed. We repeated the analysis using the transfer functions for each of the available years in the 70s for the no adaptation case and for each of the years after 2000 for the early adaptation case. This test showed that the scaling factors are not sensitive to the change of the adaptation level and hence the attribution results in Fig. 4 are typical of the two adaptation scenarios considered here. In the real adaptation case none of the signals was found to be detectable, as one might expect from Fig. 3, even though the decrease in CRM and TM in this case is much larger (Figs. 2 and 3). We can therefore conclude that the dominant driver for these changes is the adaptation of the population to a wider range of temperatures rather than the warming itself.

5 Conclusions

We have carried out an attribution study to investigate recent changes in mortality of people over 50 in England and Wales. The need for a formal statistical tool when one attempts to make attribution statements that link impacts of climate change to possible causes is clear. A less stringent approach could be very misleading. For example, it would be easy to compare the recent decrease in cold-related mortality with the increase in temperature and make the seemingly logical assumption that fewer people have died because of milder winters. Our work, however, shows that this is not the case. We find that adaptation of the population to colder temperatures can explain much of the observed change. We also show that if adaptation to cold weather had remained unchanged, then the anthropogenic warming would have produced a detectable decrease in winter mortality. Nevertheless, in the real world the effect of adaptation appears to be more important than the impact of the anthropogenic warming. The fact that people in England and Wales have taken measures to protect themselves better from the effects of cold weather may seem to be at odds with the fact that winters have in fact become milder. Improved fuel efficient and house insulation, better diet, more affordable heating and government cold weather payments that have helped the elderly afford central heating, could be some possible reasons for this adaptation to cold weather.

Unlike cold-related mortality, deaths related to heat show only a small trend after 1976, which is positive. In the hypothetical cases where adaptation remains fixed at a certain level, the human contribution to the modest increase in mortality might have been detectable as the analysis suggests. What is more interesting is that in these cases the effect of natural climatic forcings is also suggested to be potentially detectable, with the cooling from volcanic aerosols in the early 1990s being a main feature of the natural signal. The potential detectability of this weaker signal could be explained in view of the smaller internal variability in summer that would give a higher signal to noise ratio.

Our attribution analysis is based on optimal detection. Though the method is well established in the science of climate change, it has been very little utilised in impact studies. The study of Gillett et al. (2004), probably the only other example of an end-to-end analysis, applied the method to detect the effect of climate change on

Canadian forest fires. There is a wide scope for similar applications of the method, given the plethora of climate change impacts. This would contribute towards a more consistent framework between different working groups of the IPCC. A prerequisite is of course better communication and collaboration between experts in different areas.

This analysis is specific to England and Wales and one could expect attribution results to vary in parts of the world with different adaptive capacity to heat and cold and different effects of climate change. It would therefore be interesting to extend this work and investigate changes in other locations, a task that would very much depend on the availability of data, especially in poorer countries. Benefits from rising winter temperatures would be more pronounced in countries with milder winters, where cold-related mortality is higher. However, even if climate change significantly decreases cold-related mortality, this benefit should be considered alongside the other, predominantly detrimental, health impacts discussed in Section 1, which could be severe in other regions. With regard to heat-related mortality, future changes in the frequency and intensity of heatwaves also pose a concern and raise the question whether adaptation will manage to keep pace with such changes. In the UK there has been little adaptation to warmer temperatures. This could be explained by the lack of measures to relieve heat stress, like more air conditioning in buildings or in the London Underground.

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