Environmental Research 111 (2011) 853–860

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

The short-term influence of temperature on daily mortality in the temperate climate of Montreal, Canada ☆

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ARTICLE INFO

Article history:
Received 8 January 2011
Received in revised form
18 May 2011
Accepted 31 May 2011

Keywords:
Temperature
Mortality
Time series study
Distributed lag non-linear models
Air pollution

ABSTRACT

The purpose of this study was to determine whether short-term changes in ambient temperature were associated with daily mortality among persons who lived in Montreal, Canada, and who died in the urban area between 1984 and 2007. We made use of newly developed distributed lag non-linear Poisson models, constrained to a 30 day lag period, and we adjusted for temporal trends and nitrogen dioxide and ozone. We found a strong non-linear association with high daily maximum temperatures showing an apparent threshold at about 27 °C; this association persisted until about lag 5 days. For example, we found across all lag periods that daily non-accidental mortality increased by 28.4% (95% confidence interval: 13.8–44.9%) when temperatures increased from 22.5 to 31.8 °C (75–99th percentiles). This association was essentially invariant to different smoothers for time. Cold temperatures were not found to be associated with daily mortality over 30 days, although there was some evidence of a modest increased risk from 2 to 5 days. The adverse association with colder temperatures was sensitive to the smoother for time. For cardio-respiratory mortality we found increased risks for higher temperatures of a similar magnitude to that of non-accidental mortality but no effects at cold temperatures.

1. Introduction

Extreme weather conditions have been shown to increase daily mortality (Basu and Samet, 2002b; Gasparrini and Armstrong, 2010; Gosling et al., 2009). For example, the heat wave in Europe during the summer of 2003 may have caused an additional 22,000 deaths in France, Italy, Great Britain, and Spain (Conti et al., 2005; Kosatsky, 2005; le Tertre et al., 2006). These figures may be underestimates as not all heat-related deaths are recognized as such (Donoghue et al., 1997). The response to increasing temperature does not occur just at the upper range; there is a steep gradient in daily mortality and daily hospitalizations, usually above a location-specific "threshold" (Basu and Samet, 2002a; Gosling et al., 2009; Gouveia et al., 2003; Kovats et al., 2005, 1998; Martens, 1998; McMichael et al., 2006). The effects of increased temperatures are primarily found within a few days of the hot day, although longer lag effects are sometimes found, and the increased risks attenuate with increasing lag time (Braga et al., 2002; Conti et al., 2005; Curriero et al., 2002; Davis et al., 2003a, 2003b; Dessai, 2002; Gosling et al., 2009; Hajat et al., 2006; Hajat et al., 2005).

In addition, there are data suggesting that colder than normal temperatures can increase risk (Anderson and Bell, 2009; Curriero et al., 2002). These effects may be delayed for as many as two weeks into the future (Pattenden et al., 2003).

The heat events in Europe and elsewhere suggested that the elderly may be at higher risk, and this may have been due to a lack of support structures to ensure sufficient hydration and other measures to alleviate effects from extreme heat. As well, certain other sub-groups may be at higher risk; for example, a recent study from our group suggested that persons with congestive heart failure may be susceptible to increasing temperatures (Kolb et al., 2007). Other investigations of the effects of weather in this subpopulation have shown that the risk of hospitalization for congestive heart failure among elderly persons living in Denver, Colorado, during the summer increased monotonically with increasing maximum temperature (13% increase for a 5.3 °C increase) (Koken et al., 2003).

Abbreviations: df, degrees of freedom; dlnm, distributed lag non-linear models; ICD, International Classification of Diseases; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter having an aerodynamic diameter of 2.5 μm or less

☆ **Funding sources**: None.

Ethics approval: Ethics approval was obtained from the Institutional Review Board of McGill University and from the Commission de l'accès à l'information du Québec. This research was completed in accordance with the Helsinki Declaration.

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doi:10.1016/j.envres.2011.05.022

An analysis of the acute effects of weather needs to account for the fact that the response pattern for temperature is non-linear, the effects can be delayed in time, and different response functions may apply at different lag periods. In most of the previous studies the methods did not allow estimates of the complexities of these joint effects. Recently, Armstrong and colleagues (Armstrong, 2006; Gasparrini et al., 2010) have developed a formal theory and software for distributed lag non-linear models that is a generalization of the usual (linear) distributed lag linear model (Almon, 1965; Moshammer et al., 2006; Schwartz, 2000; Wyzga, 1978; Zanobetti et al., 2000). Using these new methods, our primary objective was to assess in Montreal, Canada, a city with a large range in daily temperatures, the duration and response function of the effect of higher temperature and to determine whether there were effects from colder temperatures.

2. Material and methods

2.1. The study population

The study population comprised residents of Montreal who died in the city between 1984 and 2007 of any non-accidental cause. Montreal has about two million inhabitants (in 2001) and they live in an area of about 500 km^2. The city is in a temperate zone and experiences both very cold and quite hot temperatures. A feature of the city and the province is that there is little air conditioning in homes (25.6% in the Province of Quebec in 2008 (Statistics Canada, 2010)) but that the buildings are well-heated during cold periods (usually mid-October until mid-April, with December to February being the coldest periods of the year).

Deceased subjects were identified from the computerized provincial database of death certificates and they were provided to us without personal identifiers. Approval to have access to the mortality data was granted by the provincial agency responsible for allowing access (Commission de l'accès à l'information du Québec) and ethical approval was granted by the Institutional Review Board of the Faculty of Medicine, McGill University.

2.2. Weather and air pollution data

Previous papers described in detail the environmental data (Goldberg et al., 2003, 2009). Daily weather data, comprising hourly measurements of temperature and other parameters, were provided by Environment Canada from their monitoring station located at the Pierre-Elliott-Trudeau International Airport (latitude: 45°28′05″N; longitude: 73°44′29″W) situated approximately 30 km west of downtown Montreal. We computed daily averages of temperature, humidex, humidity, and maximum temperature.

The air pollution data comprised bi-hourly or hourly measurements in Montreal of a number of criteria gaseous pollutants (sulfur dioxide, carbon monoxide, nitrogen dioxide (NO_2), and ozone (O_3)) at 12 fixed-site monitoring stations. We chose to include two of these as covariates in the substantive analysis: NO_2 was measured at eight stations and O_3 was measured at nine stations, both pollutants were measured using chemiluminescence (Thermo electron 14 V). Mean daily concentrations of NO_2 and O_3 were derived by taking a simple daily average for each monitor and then averaging these across monitors to obtain a final daily mean value. Respirable and fine particles were measured using high-volume samplers approximately every six days during 1984–2004 period and in 1996 these were replaced by tapered element oscillating microbalances. Because of the large number of missing days in the early part of the study period and the difficulty of combining high-volume samples with the measurements from the tapered element oscillating microbalances, we excluded fine particles from all analyses.

2.3. Statistical methods

We selected maximum temperature as the exposure metric and we assessed the association with non-accidental mortality using a time series approach (Goldberg et al., 2004) that has been generalized to handle the distributed lag non-linear models (Armstrong, 2006; Gasparrini et al., 2010). Specifically, we used quasi-likelihood Poisson regression in a generalized linear model to model the natural logarithm of daily counts of cause-specific deaths as functions of predictor variables. We accounted for the over-dispersed Poisson data by assuming that the total variance was proportional to the number of counts, with the over-dispersion constant estimated through quasi-likelihood. To remove seasonal and sub-seasonal cycles in the mortality time series, we included a natural cubic spline function on day of study and we included a factor for day-of-the-week. Following the analyses of the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) (Dominici et al., 2004; Samet et al., 2000), we specified a "primary" model using a smoother for time of 7 degrees of freedom (df) per annum and we investigated the sensitivity of the results using temporal smoothers having 5, 9, and 13 df.

2.3.1. Other potential confounding variables

We accounted for the effects of air pollution by including mean daily concentrations of nitrogen dioxide and ozone. Our previous work showed that the effects of these two air pollutants were linear and that their effects persisted over the concurrent day (lag 0 days) and the two previous days (lags 1 and 2 days) (Brook et al., 2007; Goldberg et al., 2001d) We could not account for influenza epidemics, as monitoring and recording of these epidemics is not carried out routinely.

2.3.2. Distributed lag non-linear models of the effects of temperature on mortality

We made use of the distributed lag non-linear models developed by two of us (B.A. and A.G.; referred to as dlnm) (Armstrong, 2006; Gasparrini et al., 2010) to describe simultaneously non-linear and delayed dependencies in the association between mortality and temperature. Briefly, these models are a generalization of the traditional distributed lag models (Almon, 1965; Moshammer et al., 2006; Schwartz, 2000; Wyzga, 1978; Zanobetti et al., 2000) to allow the model to contain a flexible representation of the time-course of the exposure–response relationship, which also provides an estimate of the overall effect in the presence of delayed contributions or "harvesting". The dlnm allow for the simultaneous estimation of different non-linear functions of the associations with temperature at each lag period and also allows for the estimation of non-linear effects across lags. The methodology is based on the definition of a "cross-basis" function, a bi-dimensional space of functions specifying the possible non-linear association between temperature and mortality across lag periods. The cross-basis functions are combined from the basis functions for the two dimensions (temperature and lag), chosen among a set of possible bases. We used the dlnm package in the R project for statistical computing (version 2.10.1; http://www.r-project.org/) that was written by one of us (A.G.).

The amount of smoothing chosen for the temperature and lag spaces is independent, as they are modeled by two different functions. Having equally spaced knots over the temperature space does indeed imply similar degree of flexibility across the range, and one could use alternative positioning of the knots, a priori or data-based, although there are issues associated with selecting the knots. Our approach to investigating curvature assumptions has been through sensitivity analyses. The knots in the lag space were, however, placed unequally across the lag space, following the default in the dlnm package (equally on a logarithmic scale), to reflect greater expected smoothness as lags increase (e.g., smoother over lags 29–30 days versus lags 0–1 days).

Among the possible non-linear functions, including linear thresholds, polynomials, and spline transformations, we selected cubic b-splines to model the temperature effect, as they are flexible at the endpoints where some degree of non-linearity is expected. Using a dlnm model that was constrained to assess effects for a lag period of 30 days, we placed knots evenly across the range of maximum temperature and selected a priori, following the work of Gasparrini et al. (2010), the "primary" model having a natural cubic spline with 5 degrees of freedom (df) in the lag space (knots placed at logarithmically equal intervals) and a cubic b-spline with 6 df (three equally spaced knots) in the temperature space.

As sensitivity analyses, we also investigated b-splines having three knots chosen from the quantiles of the temperature distribution (6 df total) and we investigated threshold models. Analyses were also conducted in the smaller group of individuals who were under age 65 years at time of death. In addition, we conducted analyses that were not adjusted for air pollution to determine the extent of confounding on temperature.

3. Results

Table 1 shows that during the study period, 1984–2007 (comprising 8766 days), the average maximum daily temperature was 11.5 °C (average mean daily temperature was 6.8 °C), varying from −23.9 to 36.2 °C (interquartile range of 20.6 °C). Air pollution was relatively low in Montreal as compared to most North American cities, with mean daily concentrations of NO_2 and O_3 of 38 and 33 μg/m^3, respectively. Table 2 shows that the different metrics for temperature were highly correlated (Pearson correlation coefficients of 0.99) and that, as expected, NO_2 and O_3 were negatively and positively correlated with temperature, respectively. In what follows, we will only show results for maximum temperature.

Table 3 shows the distributions of mortality from non-accidental causes and from cardiovascular diseases (International Classification of Diseases (ICD), revision-9 390–459; ICD-10 I00–I99) and

Table 1
Distribution of selected weather and air pollution variables, Montreal, 1984–2007.

	Units	Number of days of measurements	Mean	Standard deviation	Minimum	Percentiles				Interquartile range
						25th	50th	75th	100th	
Maximum temperature	°C	8703	11.55	12.36	−23.9	1.9	12.4	22.5	36.2	20.6
Mean temperature	°C	8700	6.78	11.86	−27.3	−2.1	7.7	17.1	29.3	19.2
Minimum temperature	°C	8720	1.99	11.61	−31.8	−6.1	2.9	11.7	24.6	17.8
Maximum humidex[a]	°C	8766	11.14	15.55	−29.4	−1.1	11.0	24.6	46.1	25.7
Mean relative humidity	%	8766	69.57	12.41	28	61	70	78	100	17
Change in pressure in 24 h ending at 08:00	kPa	8756	0.00	0.92	−4.22	−0.54	0.00	0.54	5.03	1.08
NO_2	μg/m^3	8764	37.99	14.95	7.34	27.36	35.88	45.91	165.67	18.55
O_3	μg/m^3	8764	32.77	18.00	1.86	19.55	30.28	42.96	163.93	23.41

[a] Humidex is calculated as mean temperature (°C)+0.5555 (6.11E−10) where E=exp(5417.753 × (1/273.16))−(1/Dew Point Temperature (°K)) (see for the definition http://www.weatheroffice.gc.ca/mainmenu/faq_e.html; accessed June 2011).

Table 2
Pearson correlation coefficients between selected weather and air pollution variables, Montreal, 1984–2007.

	Daily maximum temperature (°C)	Daily mean temperature (°C)	Daily minimum temperature (°C)	Daily humidex (°C)	NO_2 (μg/m^3)	O_3 (μg/m^3)
Daily maximum temperature	1	0.99	0.95	0.99	−0.21	0.41
Daily mean temperature		1	0.99	0.99	−0.25	0.39
Daily minimum temperature			1	0.96	−0.26	0.35
Daily humidex				1	−0.23	0.40
NO_2					1	−0.20

Table 3
Distribution of mortality from non-accidental causes, cardiovascular diseases, and respiratory diseases, by age and sex, Montreal, 1984–2007.

	Number of days of measurements	Mean	Standard deviation	Minimum	Percentiles				Interquartile range
					25th	50th	75th	100th	
Non-accidental mortality									
All	8766	38.08	7.60	10	33	38	43	95	10
< 65 years of age	8766	7.80	3.01	0	6	8	10	23	4
≥ 65 years	8766	30.29	6.92	6	26	30	35	79	9
Men	8766	18.59	4.75	4	15	18	22	43	7
Women	8766	19.50	5.17	2	16	19	23	52	7
Respiratorymortality									
All	8766	3.38	2.12	0	2	3	5	15	3
< 65 years of age	8766	0.34	0.59	0	0	0	1	4	1
≥ 65 years	8766	3.04	2.00	0	2	3	4	15	2
Men	8766	1.74	1.40	0	1	2	3	9	2
Women	8766	1.64	1.41	0	1	1	2	11	1
Cardiovascular mortality									
All	8766	14.34	4.58	1	11	14	17	48	6
< 65 years of age	8766	2.11	1.57	0	1	2	3	11	2
≥ 65 years	8766	12.23	4.08	1	9	12	15	40	6
Men	8766	6.83	2.89	0	5	7	9	22	4
Women	8766	7.51	3.06	0	5	7	9	33	4
Cardio-respiratory mortality									
All	8766	17.73	5.34	2	14	17	21	52	7
< 65 years of age	8766	2.45	1.70	0	1	2	3	13	2
≥ 65 years	8766	15.27	4.82	2	12	15	18	50	6
Men	8766	8.57	3.34	0	6	8	11	24	5
Women	8766	9.15	3.47	1	7	9	11	34	4

respiratory diseases (ICD-9 460–519; ICD-10 J00–J99). The mean number of daily non-accidental deaths was 38.1 and the variance was 57.8. The daily mean number of deaths from respiratory diseases was 3.4 and from cardiovascular diseases it was 14.3. (Time series plots for the endpoints and for temperature are shown in Supplementary Annex Figs. 1 and 2.)

The model that accounted only for seasonal and secular trends (a time smoother of 7 df per annum and a term for day-of-the-week) had an over-dispersion parameter of 1.11 and a serial autocorrelation coefficient that was close to zero by lag 5 days. (See Supplementary Annex Table 1 for these parameters across all of the time smoothers used: 5, 7, 9, and 13 df.)

Fig. 1 shows a three-dimensional plot of non-accidental mortality and temperature that was modeled as a cubic b-spline having three equally spaced knots (total of 6 df), constrained to a lag period of 30 days. This model included natural cubic splines for the temporal

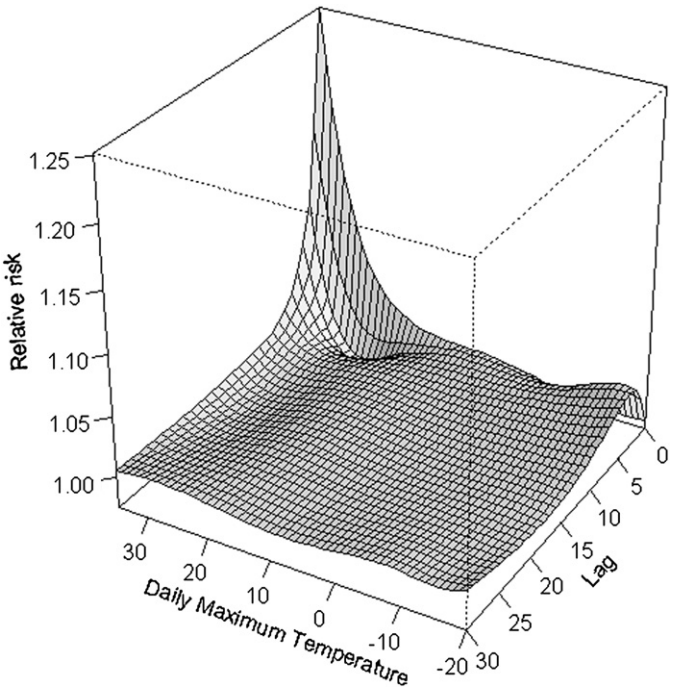

Fig. 1. The relative risk of daily non-accidental mortality and maximum temperature by lag period, from a distributed lag non-linear model, adjusted for nitrogen dioxide and ozone, Montreal, 1984–2007. The model comprised a smoother for time of 7 df per annum, three equally spaced knots for the effect of temperature (6 df total) and 5 df for the lag space. The *z*-axis represents the relative increase in daily counts of mortality with respect to the mean value (11.55 °C) and the other axes represent maximum temperature and lag period.

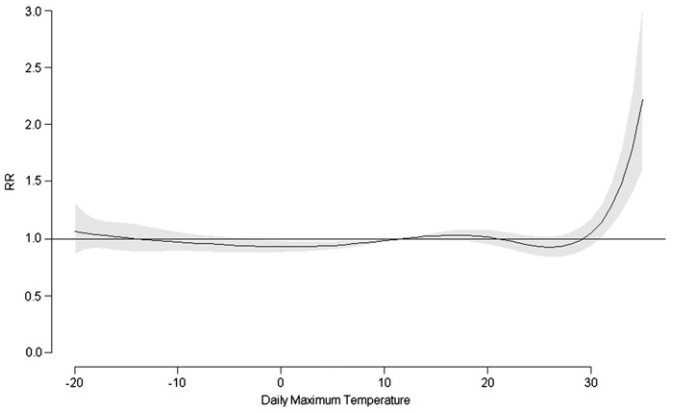

Fig. 2. Cumulative effects between daily non-accidental mortality and maximum temperature, from a distributed lag non-linear model, adjusted for nitrogen dioxide and ozone, Montreal, 1984–2007. The model comprised a smoother for time of 7 df per annum, three equally spaced knots for the effect of temperature (6 df total) and 5 df for the lag space. The *y*-axis represents the relative increase in daily counts of mortality with respect to the mean value (11.55 °C). The maximum likelihood estimate is shown as a smooth line and the pointwise 95% confidence intervals are shown in the shaded area.

smoother (7 df) and for the lag space (5 df), a term for day-of-the-week, as well as terms for the two air pollutants. The figure shows the strong effect at high temperatures that persisted up to lag 5 days and a cold effect starting at about −15 °C between lags 2 and 5 days. This graph must be interpreted cautiously as it is not possible to provide estimates of variability, but it does show the general pattern of risk by lag and by temperature. The following results help explain this overall pattern.

Based on the same model, Fig. 2 shows the fitted cumulative distributed non-linear lag function. This function is interpreted as the total effect on mortality on the concurrent day from the effects of temperature accumulated over the concurrent day and out to lag 30 days, inclusive, in the hypothetical case in which temperature is constant over that period. The relative increase in the number of daily deaths (referred to as "relative risk") for temperature is compared to the average maximum temperature of 11.55 °C. At high maximum temperatures, there is a strong monotonic increase in the number of deaths starting at about 27 °C. (Similar effects were observed for other temporal smoothers and other smooth functions for temperature; Supplementary Annex Fig. 4.) We also found a small non-significant cold effect at about −18 °C. Most of the other models for which we used different temporal smoothers showed no or protective overall effects at colder temperatures. The protective effects for colder temperatures were more pronounced as the number of df on the smoother for time increased (Supplementary Annex Fig. 4).

To illustrate the delayed effects of maximum temperature on mortality, we show the response function at lag 4 days (Fig. 3).

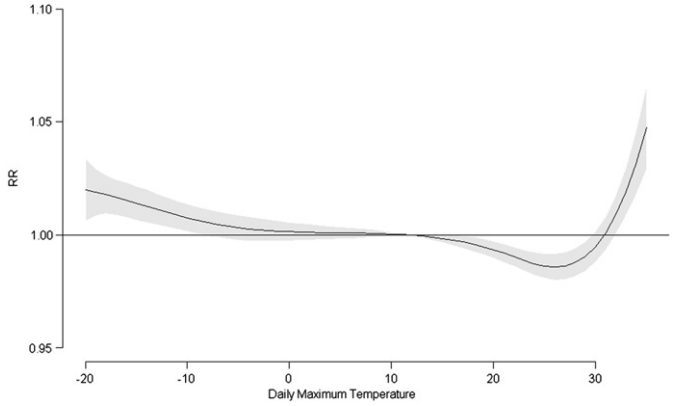

Fig. 3. Effects on daily non-accidental mortality evaluated at lag 4 days and maximum temperature, from a distributed lag non-linear model, adjusted for nitrogen dioxide and ozone, Montreal, 1984–2007. The model comprised a smoother for time of 7 df per annum and three equally spaced knots for the effect of temperature (6 df total) and 5 df for the lag space. The *y*-axis represents the excess mortality with respect to the mean value (11.55 °C). The maximum likelihood estimate is shown as a smooth line and the pointwise 95% confidence intervals are shown in the shaded area.

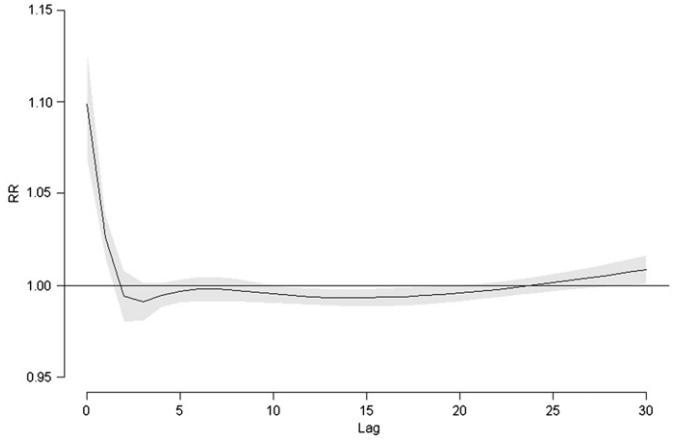

Fig. 4. Effects on daily non-accidental mortality evaluated at warm maximum temperatures (30 °C), from a distributed lag non-linear model, adjusted for nitrogen dioxide and ozone, Montreal, 1984–2007. The model comprised a smoother for time of 7 df per annum and three equally spaced knots for the effect of temperature (6 df total) and 5 df for the lag space. The *y*-axis represents the relative increase in daily counts of mortality with respect to the mean value (11.55 °C). The maximum likelihood estimate is shown as a smooth line and the pointwise 95% confidence intervals are shown in the shaded area.

A pronounced effect at high temperatures was found (and in all models) as well as a cold effect starting about −10 °C. The effect at cold temperatures vanished when 13 df per year was used as the temporal smoother (Supplementary Annex Fig. 5).

Fig. 4 shows the increased risk of high maximum daily temperatures (30 °C) relative to the average maximum temperature (11.55 °C). Effects were found for lags 0 and 1 days independent of the model (Supplementary Annex Table 4 and Fig. 6), with protective effects seen from lags 2–23 days, and a suggestion of an increase starting at lag 25 days.

Fig. 5 shows the results comparing a temperature of −15 °C to the average maximum. The maximum cold effect occurred at lag 3 days, with the models having smoothers for time of 5 and 7 df per year showing significant effects (Supplementary Annex Fig. 7 and Table 3). The figure also shows a slight apparent protective effect from lags 11–27 days, and those models with 9 and 13 df showed much stronger protective effects (in the Supplementary Annex).

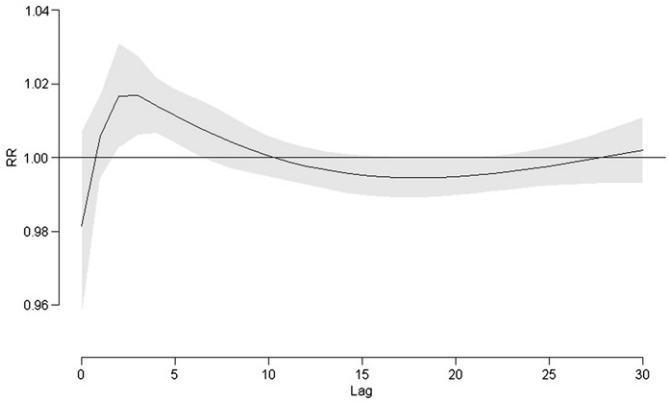

Fig. 5. Effects on daily non-accidental mortality evaluated at cold temperatures (−15 °C), from a distributed lag non-linear model, adjusted for nitrogen dioxide and ozone, Montreal, 1984–2007. The model comprised using a smoother for time of 7 df per annum and three equally spaced knots for the effect of temperature (6 df total) and 5 df for the lag space. The *y*-axis represents the relative increase in daily counts of mortality with respect to the mean value (11.55 °C). The maximum likelihood estimate is shown as a smooth line and the pointwise 95% confidence intervals are shown in the shaded area.

Table 4 summarizes the results of these figures by tabulating the percentage change in daily non-accidental mortality for the cumulative distributive lag model as well as for lagged effects from 0 days to 14 days. The first column compares the 1st percentile to the tenth percentile (risks at −16.3 °C relative to −5.3 °C) and it shows essentially the cold effects, with small increases in risk found at lags 2–5 days. (The positive sign on the percentage change is interpreted as an increase in risk as temperatures decrease.) The cumulative effect for cold temperatures was large but had considerable statistical variability; significant effects were found, however, from around lag 2 days until lag 5 days.

The second and third columns show the effects of heat on daily mortality, comparing the 99th percentile (31.8 °C) to the 75th (22.5 °C) and to the 90th (26.9 °C) percentiles, respectively. For hot temperatures, we found strong positive increases in daily mortality for the cumulative model (28.4% and 34.3% increases, respectively) and the effects declined in magnitude but persisted until lag 6 days. (These results are slightly different than shown in the figures as we are now comparing different temperature ranges, but they are derived from the same statistical model.)

3.1. Sensitivity analyses for non-accidental mortality

We found that the effects of heat were fairly insensitive to the smoother for time used but that there was some variation in the cold effects (Supplementary Annex Tables 3–7). In particular, use of smoothers for time having more than 7 df removed the deleterious cold effect and showed protective effects at higher lags. Although it is difficult to assess which models are preferable, it is possible that the smoothers for time using 9 and 13 df are over-fitting the data as the serial autocorrelation coefficients within seven day lags are mostly negative (Supplementary Annex Table 1).

We also made use of other smoothers for temperature (cubic b-splines with three knots based on quantiles of the distribution of temperature with 5 or 6 df) and threshold models, but we did not find any important differences in the response functions

Table 4
Percentage change in daily non-accidental mortality, and associated 95% confidence intervals (CI), for changes in maximum temperature between selected cut-points in the distribution, adjusted for nitrogen dioxide and ozone[a], Montreal, 1984–2007.

Lagged effect (days)	1st percentile relative to the 10th percentile[b]		99th percentile relative to the 75th percentile[c]		99th percentile relative to the 90th percentile[d]	
	% Change	95% CI	% Change	95% CI	% Change	95% CI
Cumulative	7.80	−3.87–20.90	28.40	13.76–44.93	34.34	19.18–51.43
0 days	−0.61	−2.96–1.81	11.93	8.95–15.00	8.48	6.08–10.93
1	1.13	−0.02–2.30	6.88	5.67–8.10	6.14	5.10–7.19
2	1.79	0.40–3.20	3.90	2.48–5.35	4.34	3.11–5.57
3	1.60	0.56–2.65	2.54	1.48–3.60	3.05	2.13–3.97
4	1.24	0.50–1.99	1.76	1.06–2.48	2.11	1.45–2.78
5	0.96	0.22–1.70	1.18	0.51–1.85	1.44	0.78–2.09
6	0.74	−0.03–1.53	0.75	0.04–1.46	0.97	0.28–1.66
7	0.59	−0.18–1.36	0.44	−0.27–1.16	0.67	−0.02–1.36
8	0.47	−0.23–1.18	0.23	−0.43–0.90	0.49	−0.14–1.13
9	0.39	−0.23–1.01	0.09	−0.50–0.68	0.40	−0.17–0.97
10	0.32	−0.22–0.87	−0.01	−0.54–0.52	0.35	−0.16–0.87
11	0.27	−0.25–0.78	−0.09	−0.59–0.41	0.31	−0.18–0.81
12	0.21	−0.29–0.72	−0.16	−0.66–0.33	0.28	−0.21–0.77
13	0.16	−0.35–0.68	−0.22	−0.72–0.28	0.25	−0.26–0.75
14	0.12	−0.41–0.65	−0.26	−0.77–0.25	0.21	−0.30–0.73

[a] Model included a cubic b-spline using three equally spaced knots (total of 6 df) for maximum temperature, a natural cubic spline with 5 df for the lag space, a natural cubic spline with 7 df per year for the time filter, day of the week, and NO_2 and O_3.
[b] 10th percentile = −5.3 °C, 1st percentile = −16.3 °C.
[c] 75th percentile = 22.5 °C, 99th percentile = 31.8 °C.
[d] 90th percentile = 26.9 °C, 99th percentile = 31.8 °C.

(selected results shown in Supplementary Annex Tables 3–7, sensitivity model 4, and under additional sensitivity analyses). Results of analyses amongst persons who died under the age of 65 years of age had large variability and were not informative and we did not find major differences between men and women (data not shown). The findings did not change when we did not adjust for the two air pollution variables, NO_2 and O_3 (Supplementary Annex Table 8). We also found similar response functions for different metrics of temperature (see additional sensitivity analyses).

3.2. Analyses of cardio-respiratory mortality

Because of uncertainties regarding the actual underlying cause of death, we combined deaths from cardiovascular and respiratory diseases. Fig. 6 shows the cumulative effects for cardio-respiratory mortality for a model using the same parameters as used in the analyses of non-accidental mortality. We did not find any effects at colder temperatures but we observed effects from hot weather that persisted to lag 5 days (Table 5).

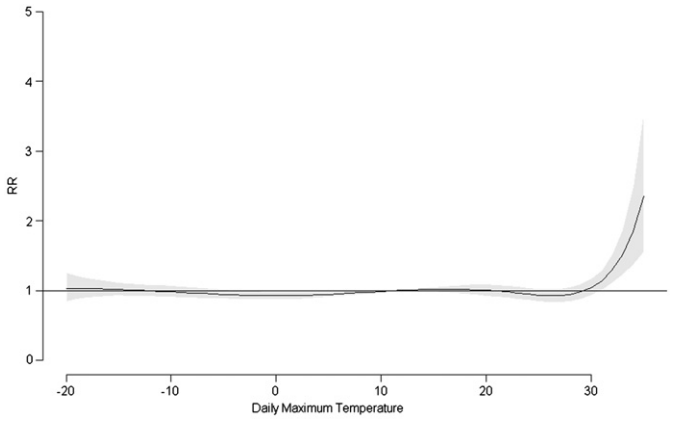

Fig. 6. Cumulative effects between daily mortality from cardio-respiratory diseases and maximum temperature, from a distributed lag non-linear model, adjusted for nitrogen dioxide and ozone, Montreal, 1984–2007. The model comprised a smoother for time of 7 df per annum, three equally spaced knots for the effect of temperature (6 df total) and 5 df for the lag space. The y-axis represents the relative increase in daily counts of mortality with respect to the mean value (11.55 °C). The maximum likelihood estimate is shown as a smooth line and the pointwise 95% confidence intervals are shown in the shaded area.

4. Discussion

As in most other studies, we have found that higher temperatures conferred excess risks of daily deaths from non-accidental causes and from cardio-respiratory disease, with the models being fairly robust to various specifications. The approximate "threshold" of maximum temperature for cumulative effects for non-accidental deaths over a 30 day period was about 27 °C (the 91st percentile of the temperature distribution). We did not find any cumulative effects at the colder end of the spectrum, although increased risks were apparent between lags 2 and 5 days. These effects were not, however, robust to the type of smoother for time used, with smoothers of more than 7 df per annum causing these effects to disappear. The analyses of cardio-respiratory mortality showed similar effects to that of non-accidental mortality, but there were no apparent effects at colder temperatures.

In the analyses of the cold effects, the occurrence of lagged effects with no cumulative effect is consistent with a "harvesting" effect, whereby the dates of death are moved up just a few days among a subpopulation at higher risk.

Association of elevated mortality with cold temperatures has been reported widely (e.g., Curriero et al., 2002; Keatinge et al., 1997; Analitis et al., 2008; Anderson and Bell, 2009; Barnett et al., 2005; Curriero et al., 2002; Keatinge et al., 1997). Studies from Europe (Pattenden et al., 2003; Analitis et al., 2008) have often found effects delayed by two weeks or more. In North America there seems to be less evidence of such a long delay, though Anderson and Bell (2009) did find effects up to two weeks. The absence of a strong association of cold temperatures with elevated mortality in Montreal is thus unusual. However, in Montreal, in contrast to many cities in milder climates, all homes are well-heated, so that this adaptation to continuously uncomfortable climatic conditions may explain the lack of a pronounced cold effect. The very small effects of

Table 5
Comparison of the estimated percentage change in daily mortality by cause of death, and associated 95% confidence intervals (CI), for changes in maximum temperature between the 75th and 99th percentiles and between the 10th and 1st percentiles, adjusted for nitrogen dioxide and ozone,[a] Montreal, 1984–2007.

Lagged effect (days)[a]	99th percentile relative to the 75th percentile				1st percentile relative to the 10th percentile			
	Non-accidental[b]		Cardio-respiratory[b]		Non-accidental[b]		Cardio-respiratory[b]	
	% Change	95% CI	% Change	95% CI	% Change	95% CI	% Change	95% CI
Cumulative	28.40	13.76–44.93	24.01	3.40–48.74	7.80	−3.87–20.90	6.39	−9.55–25.14
0	11.93	8.95–15.00	10.22	5.91–14.70	−0.61	−2.96–1.81	−0.08	−3.38–3.34
1	6.88	5.67–8.10	9.22	7.42–11.05	1.13	−0.02–2.30	0.86	−0.75–2.51
2	3.90	2.48–5.35	7.32	5.17–9.50	1.79	0.40–3.20	1.24	−0.70–3.22
3	2.54	1.48–3.60	4.96	3.38–6.56	1.60	0.56–2.65	1.16	−0.29–2.63
4	1.76	1.06–2.48	2.96	1.91–4.02	1.24	0.50–1.99	0.95	−0.09–2.01
5	1.18	0.51–1.85	1.55	0.55–2.55	0.96	0.22–1.70	0.75	−0.29–1.79
6	0.75	0.04–1.46	0.61	−0.45–1.67	0.74	−0.03–1.53	0.55	−0.54–1.65
7	0.44	−0.27–1.16	0.04	−1.01–1.10	0.59	−0.18–1.36	0.36	−0.72–1.46
8	0.23	−0.43–0.90	−0.26	−1.24–0.73	0.47	−0.23–1.18	0.19	−0.80–1.20
9	0.09	−0.50–0.68	−0.38	−1.25–0.51	0.39	−0.23–1.01	0.04	−0.83–0.92
10	−0.01	−0.54–0.52	−0.41	−1.19–0.39	0.32	−0.22–0.87	−0.09	−0.86–0.69
11	−0.09	−0.59–0.41	−0.42	−1.17–0.33	0.27	−0.25–0.78	−0.19	−0.91–0.53
12	−0.16	−0.66–0.33	−0.44	−1.18–0.30	0.21	−0.29–0.72	−0.27	−0.99–0.44
13	−0.22	−0.72–0.28	−0.46	−1.21–0.30	0.16	−0.35–0.68	−0.33	−1.06–0.40
14	−0.26	−0.77–0.25	−0.48	−1.25–0.30	0.12	−0.41–0.65	−0.37	−1.12–0.38

[a] 1st percentile = −16.3 °C, 10th percentile = −5.3 °C, 75th percentile = 22.5 °C, and 99th percentile = 31.8 °C.
[b] Main model using a cubic spline (BS) three equally spaced knots with a total of 6 df for maximum temperature, a cubic spline (NS) with 5 df for the lag space, cubic spline (NS) with 7 df per year for the time filter and is also adjusted for day of the week, NO_2 and O_3.

cold on mortality has also been observed in some other very cold climates, for example in Finland (Keatinge et al., 1997), but the only publication we are aware of reporting no association is in Yakutsk, Siberia (Donaldson et al., 1998). In an analysis of the MONICA project, Barnett et al. (2005) found that rates of morbidity and mortality from coronary problems, mostly myocardial infarctions, were higher amongst persons living in warmer climates as compared to those living in colder climates.

The "adaptation" to cold is mirrored by a lack of adaptation to heat. As we noted in Section 1, air conditioning of homes is rather limited in Quebec, and thus many individuals will be exposed to higher temperatures and thus the strong effect at high temperatures is indeed plausible physiologically and is certainly consistent with the literature.

We note that the selection of the smoothing functions is critical regarding the shape of the curve near the tails. Natural cubic splines are constrained to define a linear relationship beyond the boundaries and this often affects the shape near the ends. Although it is frequently reported that natural cubic splines have an "optimal behavior" in the tails, meaning that they are less prone to the effect of outliers and more able to capture the true curve, there is actually very little written on this. One of us (Gasparrini) has conducted some simulations (unpublished) and the natural cubic splines performed worse (by an Aikaike Information Criterion) than unconstrained cubic b-splines of the same df. In particular, it is true that the linearity constraint on the natural cubic splines could produce some underestimate of the width of the confidence intervals near the tails. This is why we have preferred simple cubic b-splines for modeling the relationship in the space of the predictor within dlnms.

There are some limitations that need to be considered in interpreting these results. We discussed the issue of misclassification of causes of death previously (Goldberg et al., 2001b), where we indicated that respiratory and cardiovascular diseases are often confused because the conditions can occur concurrently and both can contribute to death, so that there may be some uncertainty about which cause should be selected as the primary underlying cause. As well, we suggested that there may be errors in selecting one underlying cause in a complex chain of health events (e.g., cancer leading to pneumonia and then to respiratory failure).

In our analysis of air pollution in Montreal (Goldberg et al., 2000, 2001a, 2001c, 2003), we have found much higher risks in some sub-populations, such as those with diabetes and cardiovascular disease and those with congestive heart failure. Indeed, in a case-crossover analysis of the sub-group of persons who died between 1984 and 1993 from non-accidental causes but who had congestive heart failure one year before death, the adjusted odds ratios comparing temperatures between 30 and 35 °C were 1.08, 1.22, and 1.13 for the concurrent day and lags 1 and 2 days, respectively (Kolb et al., 2007). We also found in these analyses a delayed cold effect in the colder seasons of the year.

In the present analyses, we made use of distributed lag regression models to identify possible associations. Although these analyses are complex, and have many tunable parameters, our extensive sensitivity analyses indicate that the findings are robust. Although the cold effect did disappear with the use of temporal smoother that explain more of the short-term variation (i.e., 9 df or more per annum), it is possible that these smoothers are obscuring important signals from the data and, thus, may lead to biased findings.

The effects of air pollution were included in our analyses as possible confounding variables. We could not account for daily variations of concentrations of fine particles during the study period because these were measured every six days, so there was a considerable amount of missing data. We adjusted for NO_2 and O_3 because NO_2 is somewhat higher in the colder months, as are particles, and ozone is high in the warmer months. The Pearson correlation coefficients between the high-volume sampling of $PM_{2.5}$ and NO_2 was 0.61 and for O_3 it was -0.01; the correlation between $PM_{2.5}$ measured by the tapered element oscillating microbalances and NO_2 was 0.54, for ozone it was 0.13, and for high-volume samples for $PM_{2.5}$ it was 0.89. It is also possible that the effect of pollution is on a causal pathway between weather, indexed by temperature, and mortality (weather causes fluctuations in concentrations), in which case controlling for these variables may not be warranted. However, we found that adjustments for them did not greatly change the unadjusted estimates of effect, although we cannot exclude the possibility of sensitivity to control for fine particles. Our findings that air pollution did not confound the association are consistent with that of other studies, notably of the 107 American cities included in the analysis of Anderson and Bell (2009).

We could not control explicitly for the effects of infectious disease epidemics (e.g., influenza, which occurs mostly in the fall and winter) because there are no databases that could be used for this purpose. However, the smooth function of time should have eliminated most such residual secular effects, and there is no reason to expect an association of influenza with cold temperatures (after accounting for season).

We have shown that there are indeed heat islands in the city and that slightly stronger response functions for mortality were found in areas where temperatures were generally higher (Smargiassi et al., 2009). The analysis presented herein ignored these local effects and, given that the datasets used in the two papers overlapped, we may be underestimating effects in heat islands.

In summary, we have found that in Montreal hot weather was clearly associated with increases in short-term risk of mortality, but cold weather was associated with at most a small association with increased risk and at an intermediate lag with subsequent compensating decreased risk.

Acknowledgments

We thank the Montreal Urban Community, and Environment Canada for providing the air pollution data and the meteorological data and we are grateful to the Ministère de la santé et des services sociaux de Québec for providing the health data. We thank Chia-Yi Tseng for assistance with the statistical analyses.

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.envres.2011.05.022.

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