The Effect of Weather on Respiratory and Cardiovascular Deaths in 12 U.S. Cities

Alfésio L. F. Braga,[1,2] Antonella Zanobetti,[1] and Joel Schwartz[1]

[1]Environmental Epidemiology Program, Harvard School of Public Health, Boston, Massachusetts, USA; [2]Environmental Pediatrics Program, University of Santo Amaro School of Medicine, and Laboratory of Experimental Air Pollution, Department of Pathology, University of São Paulo School of Medicine, São Paulo, Brazil

We carried out time-series analyses in 12 U.S. cities to estimate both the acute effects and the lagged influence of weather on respiratory and cardiovascular disease (CVD) deaths. We fit generalized additive Poisson regressions for each city using nonparametric smooth functions to control for long time trend, season, and barometric pressure. We also controlled for day of the week. We estimated the effect and the lag structure of both temperature and humidity based on a distributed lag model. In cold cities, both high and low temperatures were associated with increased CVD deaths. In general, the effect of cold temperatures persisted for days, whereas the effect of high temperatures was restricted to the day of the death or the day before. For myocardial infarctions (MI), the effect of hot days was twice as large as the cold-day effect, whereas for all CVD deaths the hot-day effect was five times smaller than the cold-day effect. The effect of hot days included some harvesting, because we observed a deficit of deaths a few days later, which we did not observe for the cold-day effect. In hot cities, neither hot nor cold temperatures had much effect on CVD or pneumonia deaths. However, for MI and chronic obstructive pulmonary disease deaths, we observed lagged effects of hot temperatures (lags 4–6 and lags 3 and 4, respectively). We saw no clear pattern for the effect of humidity. In hierarchical models, greater variance of summer and winter temperature was associated with larger effects for hot and cold days, respectively, on respiratory deaths. *Key words:* cardiovascular deaths, nonparametric smoothing, respiratory deaths, temperature, time series, weather. *Environ Health Perspect* 110:859–863 (2002). [Online 18 July 2002]
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Weather is known to modulate health. Seasonal changes of temperature promote changes in the daily number of respiratory and cardiovascular diseases (CVD) as well as in total and cause-specific mortality. These effects are more prominent among elderly people and children (*1*).

Although cold temperatures show greater effects than do hot temperatures, other factors such as respiratory epidemics, usually present during the winter, make unclear the precise role of temperature on increased morbidity and mortality. On the other hand, heat and heat waves are associated with increased morbidity and mortality (*2*). Increases of heat-related illnesses have been reported during episodes of excessive temperature, especially in mid-latitude cities (*3,4*). The effect of heat waves has gained more attention because of the expected changes in mean temperature with the increase of greenhouse gases. Because other factors contribute to the seasonal patterns in mortality, studies have begun to focus on the short-term effects of weather, controlling for season. In this regard, realization has been growing that weather changes might cause delayed effects and that some of the heat-related deaths might be very short-term displacements of the deaths of critically ill people, a phenomenon sometimes referred to as harvesting.

To address these issues, we have studied the effect of temperature on mortality, focusing on its lag structure. Rather than look at simple means of, for example, the previous week's or 3 weeks' temperature, we have allowed the effect of weather to vary with the lag time between exposure and the related death, with lags up to 3 weeks. To reduce the noise that accompanies estimating the effects of temperature on 21 different days, we applied a polynomial distributed lag model (*5,6*). In our previous study (*7*), we examined 12 U.S. cities and estimated the effect of mean daily temperature and relative humidity on each of the 21 days before the death on total deaths in each of the cities. We did meta-analyses stratifying the analyses in two groups: hot and cold cities. In cold cities, we found both high and low temperatures associated with increased deaths. Although the cold effect persisted for days, the effect of high temperatures was more immediate (day of and day before the death) and was twice as large as the cold effect. However, the hot temperature effect appears to involve primarily harvesting. In hot cities, neither hot nor cold temperatures had much effect on deaths. Moreover, the magnitude of the effect of hot temperature varied with central air conditioning use and the variance of summer temperatures. These results agree with other studies that have pointed out the impact of housing, air conditioning, and variability of mean temperature as important factors on heat-related health effects (*2*).

Total mortality encompasses deaths from a wide variety of causes. Different disease states may show different sensitivities to extremes in temperature. Understanding these differences may help in understanding both the sensitive populations and the mechanisms of action. In this study, we assessed the lag structure between weather and respiratory and CVD daily deaths in 12 U.S. cities, applying polynomial distributed lag models.

Materials and Methods

Data. We extracted daily counts of deaths caused by pneumonia [*International Classification of Diseases, 9th Revision* (ICD-9), 480–487] (*8*), deaths caused by chronic obstructive pulmonary diseases (COPD) (ICD-9: 490–496), all CVD (ICD-9: 390–429), and specifically myocardial infarction (MI) (ICD-9: 410) in the metropolitan counties containing the cities of Atlanta, Georgia; Birmingham, Alabama; Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Houston, Texas; Minneapolis-St. Paul, Minnesota; New Haven, Connecticut; Pittsburgh, Pennsylvania; and Seattle and Spokane, Washington from National Center for Health Statistics mortality tapes for the years 1986 through 1993 (*9*). We combined data from Minneapolis and St. Paul and treated them as one city. We obtained daily weather data from the nearest airport station (*10*).

Methods. We modeled counts of daily deaths in a Poisson regression. Our models included two basic components. We examined the effects of temperature and humidity allowing for nonlinear effects and for those effects that persisted for up to 3 weeks. We describe the methods for doing this below. We found 3 weeks to be more than sufficient to capture the effects on total deaths in our

Address correspondence to J. Schwartz, Environmental Epidemiology Program, Department of Environmental Health, Harvard School of Public Health, 665 Huntington Ave., Bldg. I, Room 1414, Boston, MA 02115 USA. Telephone: (617) 384-8752. Fax: (617) 384-8745. E-mail: jschwrtz@hsph.harvard.edu

previous study (7). We modeled the covariates we controlled for (season and trend, day of the week, and barometric pressure) by using nonparametric smoothing as described below.

In environmental epidemiologic studies, we expect the relationship between the outcome and some variables to be nonlinear. The generalized additive model (11) fits smooth functions for these variables. We chose Loess smoothes for our models (12).

In this 8-year study, we used a smooth function of time to capture the basic long time trend represented by the expected six rises and falls in daily deaths over the period because of seasonality (13). This approach has been adopted systematically in environmental epidemiologic studies of daily deaths (6,14–16). Seasonal patterns can vary greatly among cities and for different causes of death. We chose a separate smoothing parameter in each city and for each cause to both eliminate seasonal patterns in the residuals and reduce the residuals of the regression to "white noise" (i.e., remove serial correlation), as described previously (17). In models with remaining serial correlation from the residuals, we incorporated autoregressive terms (18).

The other covariates were barometric pressure on the same day and day of the week. To allow for city- and cause-specific differences, we chose the smoothing parameters for these covariates separately in each location and for each cause to minimize Akaike's information criterion (19).

Distributed lag models. Distributed lag models have been used extensively in the social sciences (20), and their use in epidemiology was described by Pope and Schwartz (21). Recently, this methodology has been applied to several studies estimating the distributed lag between air pollution and health effects (6,15,22). The motivation for the distributed lag model is the realization that temperature can affect deaths occurring not merely on the same day but also on several subsequent days. Therefore, the converse is also true: deaths today will depend on the "same-day" effect of today's temperature, the "one-day lag" effect of yesterday's temperature, and so forth. Therefore, suppressing covariates and just focusing on temperature for the moment, the unconstrained Poisson distributed lag model assumes

$$\text{Log}\,[E(Y_t)] = \alpha + \beta_0 X_t + \ldots + \beta_q X_{t-q} + \varepsilon_t, \quad [1]$$

where X_{t-q} is the temperature q days before the deaths. In this study, we examined the effect of temperature in the 12 cities on deaths with latencies (lags) ranging from zero to 20 days after the temperature event. Because the effects of temperature on

mortality are usually nonlinear, with J-, U-, or V-shaped relations commonly reported, we used both a linear and a quadratic term for temperature at each lag. Equation 1 can be recast as

$$\text{Log}\,[E(Y)] = \alpha + \omega_0 X_t + \ldots + \omega_q X_{t-q} + \omega_{q+1} X_t^2 + \ldots + \omega_{q+q} X_{t-q}^2 + \varepsilon_t, \quad [2]$$

where the ω_i are parameters.

Because substantial correlation exists between temperatures on days close together and between temperature and its square, the above regression will have a high degree of collinearity. This will produce unstable estimates of the individual ω_i and hence poor estimates of the shape of the distribution of the effect over lag.

To gain more efficiency and more insight into the shape of the distributed effect of temperature over time, constraining ω_i is useful. If this is done flexibly, substantial gains in reducing the noise of the unconstrained distributed lag model can be obtained, with minimal bias (6). The most common approach is to constrain the shape of the variation of the ω_i with lag number to fit some polynomial function. We used separate fourth-degree polynomial constraints for the linear and quadratic temperature terms, because that should be flexible enough to encompass any plausible pattern of delayed effect over time. The result is a 10 degree-of-freedom surface of the effect of temperature over the past 3 weeks on death from each specific cause. We simultaneously included linear and quadratic terms for relative humidity up to 20 days before the death in the model, subject to similar constraints.

The immediate effects of weather extremes may represent harvesting—that is, deaths brought forward by only a few days. To assess this, we compared the estimated immediate (lag 0 and 1) effect of hot days with the sum of the estimated effect over 7 days.

By fitting the same model in 12 different locations, for pneumonia, COPD, CVD, and

MI deaths, and combining effect size estimates, by lag over the cities, we can estimate the distribution of the effect of temperature and humidity over time. To combine results across cities, we used inverse variance weighted averages including a random variance component to incorporate heterogeneity.

We stratified analysis in two groups of cities: the hot cities (Atlanta, Houston, and Birmingham) and cold cities (Canton, Chicago, Colorado Springs, Detroit, Minneapolis, New Haven, Pittsburgh, and Spokane). As we observed in the total mortality study (7), the differences in the temperature ranges between these two groups of cities precluded a useful combination across all cities.

In this hierarchical study (i.e., a study with multiple levels of analysis), we first fitted a generalized additive Poisson regression for each city and each outcome. In the second stage of the analysis, we fitted an ecologic regression to investigate the role of the prevalence of central air conditioning and the variance of summer and winter temperature, the background mortality rate, percentage of population with a college degree, percent nonwhite, percent unemployed, percent living below the poverty level, city size, and mean age of the population on the estimated effect of hot days (24 hr mean of 30°C) and cold days (24 hr mean of –10°C) on cause-specific deaths. To do this, we regressed the estimated effect in each city at each of those temperatures against the above explanatory variables. We obtained prevalence of air conditioning from the American Housing Survey Web site and the remaining demographic data from the 1990 census. We used inverse variance weighting. Where heterogeneity remained, as assessed by a chi-square test, we fitted the regression including a random variance component, estimated using a maximum likelihood approach, following the method of Berkey et al. (23).

Results

Table 1 presents the descriptive analysis of the variables used in the study. The cities varied in

Table 1. The populations and the descriptive analysis of the variables in the study in the 12 locations.

Cities	1990 population	Deaths[a]	Temperature[b] (°C)			Humidity[c] (%)			Pressure[d] (mm Hg)
			5%	Mean	95%	5%	Mean	95%	
Atlanta	1,642,533	36.2	3.3	17.1	28.3	41.0	67.0	93.0	736
Birmingham	651,525	19.1	2.8	16.9	27.8	49.0	70.5	91.0	747
Canton	367,585	9.9	−6.1	10.0	24.4	51.0	73.7	93.0	729
Chicago	5,105,067	133.4	−7.2	10.1	25.6	50.0	70.8	92.0	744
Colorado Springs	397,014	6.0	−6.1	9.5	22.8	25.0	51.0	84.0	610
Detroit	2,111,687	59.7	−6.1	10.5	25.6	49.0	69.2	89.0	744
Houston	2,818,199	47.0	7.2	20.3	30.0	54.0	75.0	92.0	760
Minneapolis	1,518,196	32.3	−13.3	7.9	25.0	45.0	68.7	90.0	739
New Haven	804,219	20.4	−6.1	10.7	25.0	43.0	66.8	92.0	760
Pittsburgh	1,336,449	42.4	−5.0	11.2	25.0	48.0	69.3	90.0	732
Seattle	1,507,319	29.3	2.8	11.4	20.6	52.0	77.0	93.0	752
Spokane	361,364	8.7	−5.6	8.8	22.8	35.0	68.0	95.0	699

[a]Daily mean. [b]Daily mean temperature. [c]Relative humidity. [d]Barometric pressure.

size, although in 1990 seven cities of the study had more than one million inhabitants. We divided the cities in two groups according to their meteorologic characteristics: hot (Atlanta, Birmingham, and Houston) and cold (Canton, Chicago, Colorado Springs, Detroit, Minneapolis, New Haven, Pittsburgh, and Spokane). Among the hot cities, Houston was the hottest and most humid; among the cold cities, Minneapolis was the coldest and had the widest range of temperatures. Seattle, located in the extreme northwest of the United States, had the narrowest range of temperatures of the cities in this study and rarely exhibited extreme temperatures.

In the hot cities and in New Haven, temperature was positively associated with humidity. Correlations between temperature and barometric pressure were, in general, small and negative.

We estimated the covariate-adjusted (including humidity) effects of temperature on respiratory and CVD daily deaths by lag in the 12 cities, using a standard range of temperatures. We then performed a meta-analysis of temperature effect for hot and cold cities. We did not include Seattle in this stratified analysis by temperature groups because its mild temperature range did not fit in either group.

In cold cities (Figure 1), both high and low temperatures were associated with increased numbers of CVD deaths. In general, the effect of cold temperatures persisted for days, whereas the effect of high temperatures was restricted to the day of the death or immediately the day before. For MI deaths, the hot-day effect at lag zero was twice as large the cold-day effect (6% and 3% increases in

daily deaths, respectively), whereas for all CVD deaths it was five times smaller than the cold-day effect (1% and 5% increases in daily deaths, respectively). For MI deaths and hot days we observed a harvesting effect: After 2 days we found a 12% increase in deaths, which decreased to 4% when we looked at the cumulative effects up to 7 days. For CVD deaths, we found a 3% increase after 2 days that decreased to –0.6% after 7 days.

Also, only hot temperatures increased COPD deaths (25%); the cold effect was zero. Pneumonia deaths differed from the other causes of death in that the cold-day effect was larger, and the effect of hot temperatures was stronger at lags 3–5 (an average of 15% increase).

In hot cities (Figure 2), neither hot nor cold temperatures had much effect on CVD or pneumonia deaths. However, for MI and COPD deaths, we observed lagged effects of hot temperatures (lags 4–6, 4% increase, and lags 3 and 4, 6% increase, respectively).

Similar to that observed in total mortality analysis (7), when we estimated the effect of humidity on respiratory and CVD daily deaths in each of the 12 cities, we observed no consistent pattern, in terms of either lag structure or differences between high and low humidity. Stratifying the cities by weather characteristics also did not suggest any pattern for humidity.

In the meta-regressions, none of the predictors significantly modified the effects of hot or cold days on CVD deaths (Table 2). However, for both COPD and pneumonia, the variance in summer temperature was associated with substantial increases in the effect

of a hot day. The variance of winter temperature was similarly associated with substantial increase in the death rate on cold days.

None of the demographic factors (background mortality rate, percentage of population with a college degree, percent nonwhite, unemployment rate, percent below poverty level, city size, and mean age of the population) modified the effect of either cold or heat waves in our data ($p > 0.12$).

Discussion

Temperature has been recognized as a physical agent able to induce health effects (1,2,24). The rapid buildup of greenhouse gases is expected to increase both mean temperature and temperature variability around the world (25). This has added urgency to the need to better understand the direct effects of such changes on daily death rates, and to better understand the modifiers of those effects. One issue that has been extensively explored in this field is the shape of the relationship between temperature and deaths. U- and V-shapes have been reported in regions where both hot and cold temperatures have been associated with fatal events with similar magnitudes of effects, whereas J-shapes and even a linear shape have been reported in regions where the susceptibilities for extreme temperatures are not similar (22). We have focused our attention on exploring the lag structure between temperature and daily deaths using a systematic approach to look at the delayed effects of weather on mortality up to 3 weeks afterwards. In this study we looked at the temperature effect on cause-specific deaths in 12 U.S. cities. As observed in the total mortality study (7), hot and cold temperatures were associated with increased deaths, and the shape of this relationship varied according to climatic characteristics of the cities. However, we found sizable effects of temperature on daily deaths just at lag 0. We found lagged effects of hot temperatures in hot cities and specifically for MI and COPD, and in cold cities for pneumonia.

In cold cities, we found differences in terms of temperature effect on CVD. Although both hot and cold temperatures affected MI and total CVD deaths, the relative impacts of the extreme temperatures were different. Cold presented more homogeneous and persistent effects on both outcomes, with no evidence of harvesting. Heat presented a much more important effect on MI deaths than it did on CVD deaths. These effects were predominantly short-term mortality displacement. The pattern observed for temperature effects on CVD deaths in cold cities is similar to those observed for total deaths, probably because most of the total mortality is due to CVD deaths.

Cold temperatures did not have much effect on respiratory mortality in cold cities. However, heat increased respiratory deaths.
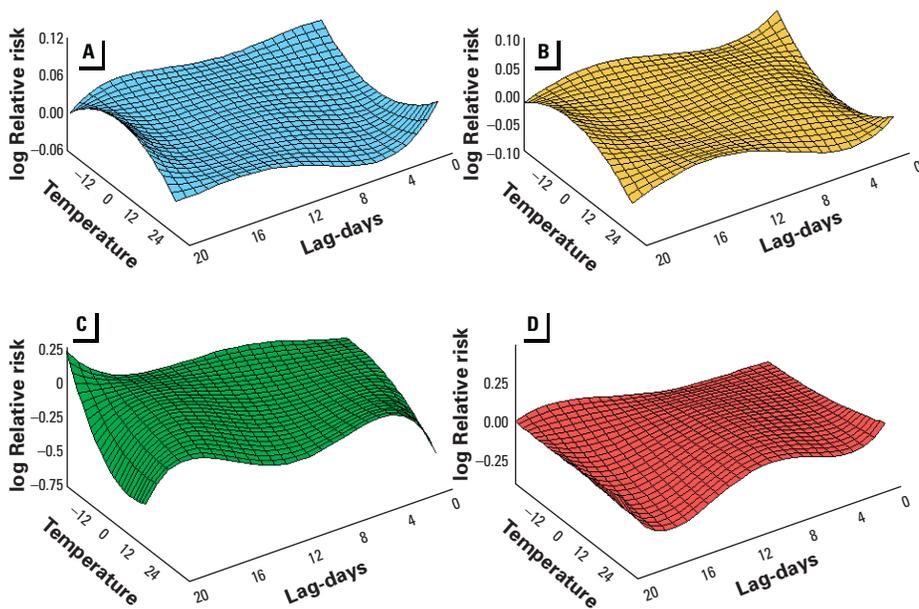

Figure 1. Overall effect of temperature (°C) on MI (*A*), CVD (*B*), pneumonia (*C*), and COPD (*D*) daily deaths in the eight cold cities: Canton, Colorado Springs, Chicago, Detroit, Minneapolis, New Haven, Pittsburgh, and Spokane. In the *z*-axis, a log relative risk of 0.1 represents a 10% increase in mortality.

For COPD, the heat effect was remarkable and acute (lag 0, 25-fold higher than the cold effect), whereas we observed a lagged effect for pneumonia.

In hot cities, we found no relevant effects of cold on both respiratory death and CVD deaths. When we analyzed pneumonia, we observed no association with temperature. The same behavior could be seen for CVD. However, for the relation between heat and both MI and COPD deaths, we saw a pattern different from the total mortality results: We observed lagged effects for these two causes of death. Hence, even in hot cities, where people are more accustomed to hot temperatures and air conditioning is common (26), the effect of heat on health, leading to increased deaths, can overcome adaptive mechanisms.

In our hierarchical model, we found that the variance of summer and winter temperature was associated with substantial changes in the effects of hot and cold days on respiratory but not CVD deaths. The substantial mortality increase in cities with more variable temperature suggests that increased temperature variability is the most relevant change in climate for the direct effects of weather on respiratory mortality.

In many ways, the results of this study and our previous study of total mortality parallel those of the Eurowinter study (27), which assessed the association between daily deaths and temperature in the winter in eight European regions. Daily deaths increased with falling temperatures in all regions. However, the effect of a cold day was greater in warmer climates than in colder climates. In our 12-city U.S. studies, the converse was true: The effect of hot days was worse in cities where they were less common. In the Eurowinter study, the effect of cold days was reduced by warmer temperatures in the living room and more hours per day of heat in the bedroom—that is, by greater use of space conditioning to reduce exposure to the cold weather. In our study, greater use of central air conditioning was associated with a reduced effect of hot days for total and for cause-specific mortality, although the results were less significant for the cause-specific mortality. Greater variability in either summer or winter temperatures, which might be expected to reduce protective behavior such as always wearing hats, was associated with increased effects of cold or heat waves. The overall message seems to be that space conditioning and behavior can substantially modify the adverse impacts of temperature extremes, but that this behavior is more frequently found in the climates where those extremes are common.

We found no association in the second-stage analysis with baseline mortality rates or social or demographic factors. However, a log-linear regression builds in interactions by design—that is, we estimated our temperature effect as a relative or percentage change in each city. In cities with a higher baseline rate, a greater absolute effect is built in. The second-stage regression therefore tests super-multiplicativity. This makes the failure to find interactions with direct or indirect markers of baseline risk understandable and the association with the temperature variances more impressive.

In the present study and in the previous one (7), we have used mean temperature. The best indicator of the temperature effect on health is still debated (2). Further analysis using different parameters (e.g., minimum temperature and dew point temperature) are needed to compare the results presented here and elsewhere, and for finding the best instrument for estimating the health effect due to extreme weather exposure.

In this cause-specific death study, we saw no consistent patterns for the relation of humidity to daily deaths by city. The combined city estimate reinforced this idea, showing no overall effect of humidity on total daily deaths. Using dew point temperature can give a more reasonable estimate of the humidity effect on daily deaths and should be pursued in the future.

Air pollution is a predictor of daily deaths. Effect modification was tested by Samet et al. (26) in a study of 20 years of data in Philadelphia. They stratified days into 20 categories based on synoptic weather conditions and found no effect modification. This does not preclude the possibility that effect
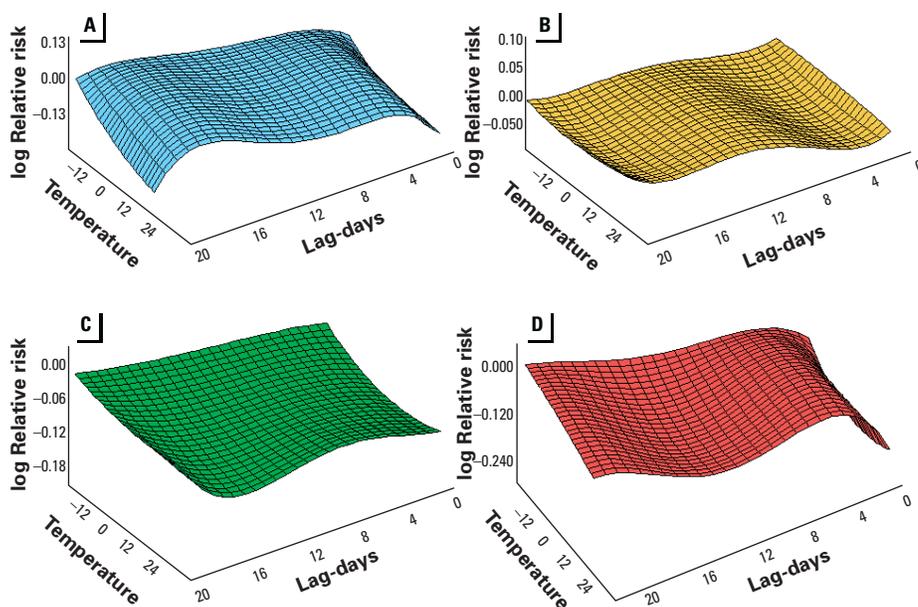

Figure 2. Overall effect of temperature (°C) on MI (*A*), CVD (*B*), pneumonia (*C*), and COPD (*D*) daily deaths in the three hottest cities: Atlanta, Birmingham, and Houston. In the z-axis, a log relative risk of 0.1 represents a 10% increase in mortality.

Table 2. Percentage increase in cause-specific deaths at 30°C and at −10°C for the difference between the 90th and 10th percentiles in air conditioning, variance of summer temperature, and variance of winter temperature.

	Summer effect		Winter effect	
	Percent	95% CI	Percent	95% CI
CVD				
Air conditioning	−1.15	⁻14.72–14.60		
Variance summertime temperature	0.93	⁻9.67–12.77		
Variance wintertime temperature			2.20	⁻1.19–5.71
MI				
Air conditioning	−16.99	⁻35.64–7.06		
Variance summertime temperature	15.67	⁻7.54–44.71		
Variance wintertime temperature			−3.63	⁻11.62–5.08
COPD				
Air conditioning	−13.44	⁻45.89–38.49		
Variance summertime temperature	42.76	4.54–94.94		
Variance wintertime temperature			25.86	⁻1.12–60.20
Pneumonia				
Air conditioning	−8.31	⁻30.79–21.47		
Variance summertime temperature	28.01	3.96–57.63		
Variance wintertime temperature			12.57	2.87–23.19

modification may be seen in other studies. However, the only air pollutant consistently associated with daily deaths in the U.S. is airborne particles (*28*). Unfortunately, airborne particles are measured only one day in six in most U.S. cities. This would prevent us from examining the effect of multiple lags of weather in our study. Hence we have chosen not to include it in our models.

In summary, we found that temperature is associated with increased daily cause-specific deaths in both cold and hot cities. In cold cities, both heat and cold contributed with daily cause-specific deaths. In hot cities, only heat presented important effect on daily deaths, and its effect was smaller than those observed in cold cities. In these cities, people seem to be more adapted to heat waves and also are not exposed to very low temperatures. Therefore, we reinforce the concept that analysis of the impact of any climatic change should take into account regional weather differences and that further analysis using different weather indicators must be done.

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