

Cause-Specific Mortality and the Extended Effects of Particulate Pollution and Temperature Exposure

Patrick G. Goodman,^{1,2} Douglas W. Dockery,³ and Luke Clancy^{2,4}

¹School of Physics, Dublin Institute of Technology, Dublin, Ireland; ²St. James Hospital, Dublin, Ireland; ³Environmental Epidemiology, Harvard School of Public Health, Boston, Massachusetts, USA; ⁴Trinity College, Dublin, Ireland

Air pollution exposure studies in the past decade have focused on acute (days) or long-term (years) effects. We present an analysis of medium-term (weeks to months) exposure effects of particulate pollution and temperature. We assessed the associations of particulate pollution (black smoke) and temperature with age-standardized daily mortality rates over 17 years in Dublin, Ireland, using a polynomial distributed lag model of both temperature and particulate air pollution simultaneously through 40 days after exposure. When only acute effects (3-day mean) were considered, we found total mortality increased by 0.4% for each 10- $\mu\text{g}/\text{m}^3$ increase in black smoke concentration. When deaths in the 40 days after exposure were considered, we found a 1.1% increase. For respiratory mortality, the estimated effect was 0.9% for acute exposures, but 3.6% for the extended follow-up. We found each increase in current-day temperature by 1°C was associated with a 0.4% increase in total mortality, whereas each decrease of 1°C was associated with a 2.6% increase in mortality in the following 40 days. For both temperature and pollution, the largest effects on cardiovascular mortality were observed immediately, whereas respiratory mortality was delayed and distributed over several weeks. These effects were two to three times greater than the acute effects reported in other studies, and approach the effects reported in longer-term survival studies. This analysis suggests that studies on the acute effects of air pollution have underestimated the total effects of temperature and particulate air pollution on mortality. **Key words:** black smoke, lagged, mortality, pollution, temperature. *Environ Health Perspect* 112:179–185 (2004). doi:10.1289/ehp.6451 available via <http://dx.doi.org/> [Online 12 November 2003]

Over the past decade, more than 100 published studies have reported statistically significant increased risk of mortality associated with acute particulate air pollution exposures. Most of these studies have limited their analyses to deaths on the day of and the 1–2 days after exposure. This observed increase in mortality may represent the advancement of death by only a few days of people who were about to die (mortality displacement or harvesting). In complementary analyses of data from Philadelphia, Pennsylvania, and Boston, Massachusetts, Zeger et al. (1999) and Schwartz (2000a) estimated the effects of particle pollution on mortality independent of harvesting effects; they each found that particulate air pollution association with mortality increased in magnitude as the follow-up period increased. Applying distributed lag models to daily particulate pollution and mortality in 10 U.S. cities, Schwartz (2000b) found that the net estimated effect of particles on mortality over 6 days of follow-up was more than twice as large as the estimated effect for 1 day of follow-up. In a pooled analysis of 10 European cities, Zanobetti et al. (2002) found statistically significant associations of particulate pollution with mortality over the 5 weeks after exposure, and a net effect on mortality over the subsequent 40 days that was twice as large as the single-day estimate.

The evidence from historical acute episodes also suggests extended air pollution effects on mortality. In the London smog of

1952 [Her Majesty's Stationery Office (HMSO) 1954], mortality increased by 4,000 deaths over the 7-day period of the smog, and was twice as high in the week after the air cleared than during the week before the smog episode. Bell and Davis (2001) have recently argued that mortality rates remained elevated for 3 months after the smog episode, with an additional 8,000 excess deaths.

In the early 1940s, Leonard et al. (1941) reported an average lag of 5 weeks between air pollution exposure episodes and increased mortality in Dublin, Ireland. More recently, Goodman (1999) and Goodman et al. (1999) reported increased mortality in Dublin associated with particulate air pollution lasting for several weeks after exposure.

In response to concerns regarding frequent winter air pollution episodes, the sale, marketing, and distribution of coal were banned within Dublin city starting 1 September 1990. Particulate air pollution levels fell immediately by 70%. Clancy et al. (2002) reported that mortality also fell by an amount larger than expected based on acute time-series studies. This suggested that the net effects of particulate air pollution exposures extended beyond the few days after exposure.

Other environmental stresses also may have extended influences on mortality that have not been adequately considered in air pollution time-series studies. Hennessy (2002) suggested that the observed air pollution associations in mortality time-series studies might

be the result of uncontrolled confounding by extended effects of exposure to cold temperatures. To assess the cumulative effects of particulate air pollution on daily mortality over an extended postexposure period, we applied distributed lag models to black smoke and daily mortality in Dublin in the 40 days after exposure. To address possible confounding by extended effects of cold temperatures, we simultaneously assessed the effects of temperature over the same period.

Methods and Data

Daily black smoke (BS) air pollution concentrations were measured at six residential monitoring stations in the city of Dublin (Dublin County Borough) starting on 1 April 1980 and continuing through 31 December 1996. In a previous analysis, Goodman (1999) showed no significant sulfur dioxide association with daily mortality detectable in the Dublin data independent of the BS association. Daily mean BS concentrations were calculated as the average of the site-specific measurements. For 43 days with no BS measurements, concentrations were estimated as the average of the prior and subsequent days. Daily minimum temperatures (degrees centigrade) and daily mean relative humidity (percent) were measured at Dublin airport.

Daily counts of death of Dublin residents, who died within the city, were calculated for total nontrauma deaths [*International Classification of Diseases* (ICD-9; 1978) for ICD-9 codes < 800], for cardiovascular causes (ICD-9 codes 390–448; 46% of all deaths), respiratory causes (ICD-9 codes 460–496, 507; 14% of all deaths), and “other” causes (total nontrauma deaths minus cardiovascular and respiratory deaths). Daily deaths were also

Address correspondence to P.G. Goodman, School of Physics, Dublin Institute of Technology, Kevin St., Dublin 8, Dublin, Ireland. Telephone: + 353-1-4024782. Fax: + 353-1-4024988. E-mail: pat.goodman@dit.ie

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categorized by age: 0–64 years (23%), 65–74 years (27%), and ≥ 75 years (50%). There were 80,307 deaths over the study period, averaging 13.1 deaths/day. To adjust for changes in the age distribution of the Dublin population, we calculated the directly age-standardized death rate for each day of the study (Clancy et al. 2002).

Respiratory disease (influenza) epidemics produce excess deaths that vary between years. There was no surveillance system for influenza epidemics in Ireland, so we constructed an indicator for respiratory disease epidemics from mortality data from the entire Republic of Ireland following methods used in the United States (Braga et al. 2000). Daily pneumonia and influenza deaths for all of Ireland were tabulated and the 95th percentile of the distribution calculated. Days on which the 14-day running average of pneumonia and influenza deaths was above the 95th percentile were designated as “epidemic” days. Six respiratory disease epidemics were identified over the seventeen years considered (December 1980/January 1981, March 1985, February/March 1986, December 1989/January 1990, November 1993, and December 1995/January 1996). These epidemic periods were concordant with influenza epidemic identified by a surveillance system in England, Wales, Scotland, and Northern Ireland (Fleming et al. 1999).

Analytic methods. We assessed the acute particulate pollution effects using traditional time-series analysis of the 3-day moving average of BS concentrations. We separately assessed the cumulative net effect of daily minimum temperature and BS particulate air pollution exposures on cause- and age-specific mortality over the subsequent 40 days using distributed lag models. The log of age-standardized Dublin mortality rates was regressed assuming a Poisson distribution in a generalized additive model (GAM; Hastie and Tibshirani 1990). The GAM allows the use of smooth functions to control for covariates such as weather, which may not have a linear relationship with the outcome.

Dominici et al. (2002) reported the estimated standard errors of the GAM regression coefficients could be underestimated when more than one nonparametric smoother was included. We used only one Loess smoother to account for seasonal patterns avoiding this potential problem. Dominici et al. (2002) also reported that the default convergence criteria used for GAMs in S-plus and other statistical software packages were inadequate and recommended stricter convergence criteria, which were used in this analysis.

Acute particulate pollution model. In the analysis of the acute effects of particulate pollution exposure (3-day mean BS), Poisson regression models for the log counts of daily

age-standardized mortality rates were adjusted for minimum temperatures on the same day (day of death) and the mean of the minimum temperature on the previous 2 days, mean relative humidity on the same and previous day, plus indicators for day of week and for respiratory epidemics. In sensitivity analyses we adjusted for the ban on coal sales in Dublin and separately assessed modification of the associations by the ban. Models were constructed and evaluated separately for total (nontrauma), cardiovascular, respiratory, and other causes of death and for age-specific total mortality. We expressed the effect as a percent change in mortality associated with each $10\text{-}\mu\text{g}/\text{m}^3$ increase in BS pollution over the exposure period (3 days), calculated as the exponential of 10 times the Poisson regression coefficient minus 1×100 .

Distributed temperature and particulate pollution model. The cumulative effects of temperature and particulate air pollution were analyzed simultaneously in a GAM including distributed lag functions for minimum temperature and BS. The approach is based on the concept that environmental exposures may produce increased risk of death on the day of exposure, the succeeding day, or possibly many days after exposure. Thus mortality on any day may depend on the same-day environmental exposures, plus a contribution from environmental exposures on each of the preceding days.

For Gaussian data, the distributed lag model can take the form

$$Y_t = \alpha + \beta_0 X_t + \dots + \beta_q X_{q-t} + \varepsilon_t \quad [1]$$

where X_{q-t} is the environmental exposure (e.g., temperature or particulate pollution) q days before day of death t (Schwartz 2000b). The total net effect of an environmental exposure (e.g., particulate air pollution or temperature) is the sum of the estimated effects of the q subsequent days and can be written as $\beta_0 + \dots + \beta_q$. Because estimated individual β_q s are unstable due to highly correlated data in consecutive days, we constrain the β_q s to vary smoothly as a polynomial function of days of lag. This polynomial distributed lag model with q lags and d degrees of freedom has the restriction that

$$\beta_j = \sum_{k=0}^d \eta_k j^k, j \in [0, q]. \quad [2]$$

Almon (1965) introduced polynomial distributed lag models for Gaussian data, and Schwartz (2000b) showed that this approach could be adapted for use in a generalized additive model. The polynomial model is one of many models that could be used to constrain the distributed lags. Schwartz (2000b) and Zanobetti et al. (2002) have described the

application of these methods to air pollution time series analyses.

We estimated the extended effects of minimum temperature and BS particulate air pollution simultaneously through 40 days with polynomials of order six. While previous studies have examined long distributed lag models for air pollution (Schwartz 2000b; Zanobetti et al. 2000) or temperature (Braga et al. 2002), in this article we assess both simultaneously. A sixth-order polynomial was chosen to allow adequate degrees of freedom to define the shape of the distributed lag response. Too many degrees would induce noise into the response, too few degrees would constrain the response curve, and the underlying relationship may not be observable. A sensitivity analysis was conducted using both fourth- and eighth-order polynomials. The beta values from both models were almost identical with the sixth order, giving the slightly more conservative estimate. A period of 40 days was chosen based on previous analysis that suggested the effect was substantially diminished at 40 days after exposure. (Goodman 1999; Goodman et al. 1999).

The estimate of β at each lag can be determined from Equation 2. The sum of the β s is the estimated increase in mortality on the day of and the 40 days after exposure. The standard errors for each β and the sum of the β s are calculated from the covariance of the η s as described by Zanobetti et al. (2002).

A Loess smooth of date was fitted to control for seasonal and long-term trends in the data. (Cleveland and Devlin 1988; Schwartz 1994). The span for date was adjusted to minimize seasonality in the deviance residuals and to minimize the expected value of the sum of the partial autocorrelations of the deviance residuals (Schwartz 1994, 2000c; Zanobetti and Schwartz 2000). Indicators for the day of the week and respiratory epidemics were included in the model for both the acute and distributed lag models.

Initial analysis of the effect of temperature showed an immediate positive association—excess deaths associated with hot temperatures on the same—and negative effects over succeeding days—increased mortality associated with cold temperatures distributed over the subsequent days to weeks. To parsimoniously model these two temperature phenomena, we included current-day minimum temperature plus a polynomial distributed lag for minimum temperature, similar to recent analysis of mortality versus temperature effects in 11 U.S. cities (Curriero et al. 2002).

Relative humidity was significantly associated with mortality on the same and previous day and therefore was modeled by a 2-day moving average. Separate models were developed for each cause- and age-specific mortality stratum. For each model, the estimated effects (betas) for BS (lags 0–40) and temperature

(lags 1–41) were computed. Day-specific log odds ratios and 95% confidence intervals (CIs) were calculated and plotted.

We expressed the net effect of temperature as the percent change in mortality associated with each increase of 1°C in daily minimum temperature on the same day, plus the effect of each 1°C decrease in daily minimum temperature over the subsequent 40 days, calculated as the exponential of the sum of the day-specific distributed lag Poisson regression coefficient -1×100 . We expressed the net air pollution effect as a percent change in mortality associated with an increase of 10 $\mu\text{g}/\text{m}^3$ in daily mean BS particulate pollution, calculated as the exponential of 10 times the sum of the day-specific distributed lag Poisson regression coefficient minus 1×100 .

All analyses were run in S-Plus (S-Plus 4.5; MathSoft, Inc., Cambridge, MA, USA) using the convergence criteria recommended by Dominici et al. (2002).

Results

Table 1 presents the mean and percentile distributions of the daily weather, air pollution measurements, and mortality. Daily mean BS concentrations averaged 46 $\mu\text{g}/\text{m}^3$ (maximum 901 $\mu\text{g}/\text{m}^3$) before the ban on coal sales, and 14 $\mu\text{g}/\text{m}^3$ (maximum 198 $\mu\text{g}/\text{m}^3$) after the ban.

Temperature associations. Each 1°C increase in temperature was associated with a 0.4% (95% CI, 0.1–0.6%) increase in total nontrauma mortality on the same day (Table 2).

Each decrease of 1°C was associated with an increase of 2.6% (95% CI, 2.3–2.9%) over the next 40 days (Table 2). Most of this excess mortality associated with cold temperatures was observed in the first 3 weeks after exposure (Figure 1). The immediate effects of heat and the delayed effects of cold were stronger for the older populations compared to those < 65 years of age (Table 2, Figure 2). In the younger group (< 65 years) the effects of lagged cold temperatures were not as pronounced and were of a shorter duration.

There was no association of cardiovascular mortality with same-day warm temperatures but there was a cumulative association with cold temperature. There was an immediate effect of cold that diminished over the next three weeks (Figure 3). The net estimated increase in cardiovascular mortality was 2.5% (95% CI, 2.0–3.0%) over the next 40 days for each 1°C decrease in minimum temperature (Table 2).

There was an estimated 0.8% (95% CI, 0.1–1.5%) increase in respiratory deaths for each 1°C increase in daily minimum temperature (Table 2). The associations of cold temperatures with respiratory mortality were strongest 3 days to 3 weeks after exposure and fell to effectively zero after four weeks (Figure 3). Each 1°C decrease was associated with a 6.7% (95% CI, 5.8–7.6%) increase in respiratory mortality over the next 40 days (Table 2).

For other causes of death, a 1°C increase in minimum temperature was associated with

Table 1. Mean and percentile distribution of daily mean air pollution, weather, and deaths per day in Dublin County Borough for period 1 April 1980–31 December 1996 ($n = 6,191$ days).

	Mean	Min	25th	50th	75th	Max
BS air pollution ($\mu\text{g}/\text{m}^3$)	40	1	10	19	39	901
Weather						
Temperature ($^{\circ}\text{C}$)	6.5	-7.9	3.1	6.6	9.7	18.4
Relative humidity (%)	82	53	76	81	87	100
Deaths per day						
Total nontrauma	13	1	10	13	16	44
Respiratory	2.4	0	1	2	3	13
Cardiovascular	5.9	0	4	6	8	27
Standardized death rate/10,000						
Total nontrauma	9.22	0.66	7.25	8.93	10.96	28.56
Respiratory	1.33	0.0	0.66	1.32	2.02	9.58
Cardiovascular	4.23	0.0	2.79	4.05	5.04	13.54

Abbreviations: Max, maximum; Min, minimum.

Table 2. Estimated percent increase in mortality (95% CI) associated with each 1°C increase in same-day mean temperature and cumulative 40-day increase in mortality associated with each 1°C decrease in mean temperature.

Category	Same-day heat effect	40-day cumulative cold
All nontrauma deaths	0.4 (0.1–0.6)	2.6 (2.3–2.9)
Age category (years)		
0–64	-0.1 (-0.7 to 0.5)	1.4 (0.7–2.2)
65–74	0.7 (0.2–1.3)	2.8 (2.2–3.5)
≥ 75	0.3 (-0.1 to 0.7)	3.0 (2.6–3.5)
Cause-specific		
Cardiovascular	0.0 (-0.4 to 0.4)	2.5 (2.0–3.0)
Respiratory	0.8 (0.1–1.5)	6.7 (5.8–7.6)
Other	0.5 (0.5–0.6)	1.5 (0.90–2.0)

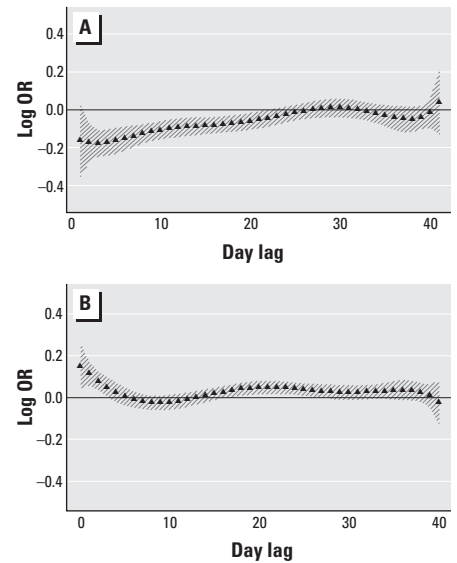


Figure 1. Polynomial distributed lag analysis of total nontrauma mortality versus minimum temperature adjusted for same-day minimum temperature. (A) Percent increase in total mortality for each 1°C increase in minimum daily temperature and (B) 10- $\mu\text{g}/\text{m}^3$ increase in daily mean BS for lags 0–40 days fitted with a sixth-degree polynomial.

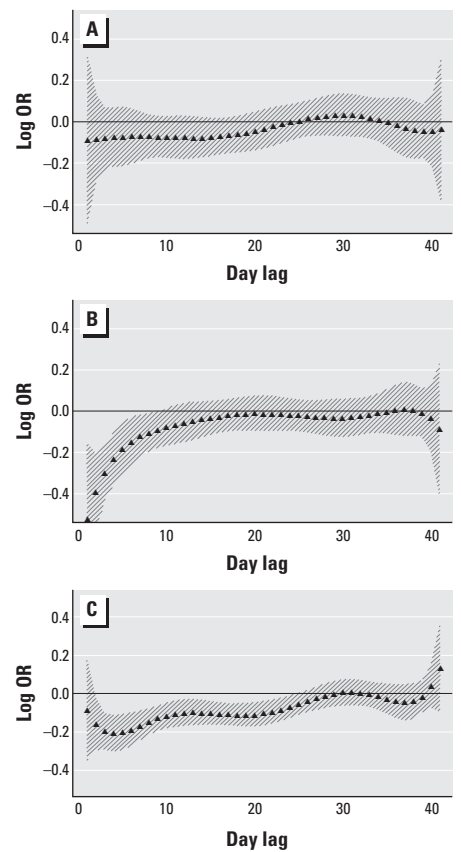


Figure 2. Polynomial distributed lag analysis of total nontrauma mortality versus minimum temperature adjusted for (A) same-day minimum temperature for ages 0–64, (B) 65–74, and (C) ≥ 75 years. Percent increase in total mortality for each 1°C decrease in minimum daily temperature for lags 1–41 days fitted with a sixth-degree polynomial.

an increase of 0.54% (95% CI, 0.50–0.58%) on the same day. The lagged effect of cold temperature persisted for about 10 days (Figure 3) with each 1°C decrease in temperature associated with a 1.5% (95% CI, 0.9–2.0%) increase in mortality over the subsequent 40 days.

Black smoke associations. Black smoke was associated with significantly increased numbers of total nontrauma deaths on the same and subsequent days (Figure 1). We estimated a 0.4% (95% CI, 0.3–0.6%, $p < 0.0001$) increase in total nontrauma mortality associated with each 10- $\mu\text{g}/\text{m}^3$ increase in 3-day mean BS (Table 3). In the distributed lag model we found significantly increased risk for total nontrauma mortality for 3 days after exposure (Figure 1), followed by a suggestion of decreased risk between 1 and 2 weeks after exposure, and then significantly increased risk between 2 and 4 weeks after exposure. The net cumulative effect of each 10- $\mu\text{g}/\text{m}^3$ increase in daily mean BS over the succeeding 40 days was a 1.1% (95% CI, 0.8–1.3%; $p < 0.0001$) increase in the risk of total nontrauma mortality (Table 3).

We found little effect of BS on total nontrauma mortality among deaths in the < 65-year-old population (Figure 4). The estimated net effect of each 10- $\mu\text{g}/\text{m}^3$ increase in BS over the subsequent 40 days was -0.1 (95% CI, -0.6 to 0.5%; $p = 0.83$) (Table 3). Among those 65–74 years of age, positive associations were found in the first 2 days after exposure, and elevated risk out to 5 weeks after exposure (Figure 4) with a net effect of 1.6% (95% CI, 1.1–2.1%; $p < 0.0001$) (Table 3). This is more than three times larger than the estimated 0.5% (95% CI, 0.3–0.8%; $p = 0.0003$) associated with a 10- $\mu\text{g}/\text{m}^3$ increase in 3-day running mean BS (Table 3).

For those ≥ 75 years of age, we found significantly positive risk in the 3 days after exposure, but a negative association between 7 and 11 days after exposure (Figure 4), suggesting harvesting in this oldest age group. However, there was then significantly increased risk from 2 to 3 weeks after exposure, suggesting a delayed elevated risk. We found the net effect was a 1.4% (95% CI, 1.1–1.8%) increase in risk of total nontrauma mortality (Table 3), again substantially larger than the estimated 0.5% (95% CI, 0.3–0.7%; $p < 0.0001$) associated with the 3-day running mean BS (Table 3).

For cardiovascular deaths (Figure 5), most of the effect of BS exposures was observed on the day of and the 2 days after exposure. The net effect of BS on cardiovascular deaths over the succeeding 40 days was 1.1% (95% CI, 0.7–1.5%; $p < 0.0001$), more than twice the estimated 0.4% (95% CI, 0.2–0.7%; $p = 0.0002$) with 3-day mean BS (Table 3).

For respiratory deaths (Figure 5), we found that BS exposure was associated with

significantly increased risk over the first week, with a suggestion of harvesting (negative associations) in the second week, but significantly elevated risk in the third and fourth weeks after exposure. The net effect of BS on respiratory deaths over the succeeding 40 days was 3.6% (95% CI, 3.0–4.3%; $p < 0.0001$), more than four times the estimated 0.9% (95% CI, 0.2–0.7%; $p < 0.0001$) increased respiratory mortality risk associated with 3-day mean BS.

For other causes of death (Figure 5) we found BS exposure was associated with increased risk in the 3 days after exposure, significantly negative risk between 1 and 2

weeks after exposure, and little association after 2 weeks. The net effect of BS on other causes of death during the succeeding 40 days was -0.2% (95% CI, -0.7 to 0.2%; $p = 0.29$). We found a similarly small, statistically nonsignificant association of a 10- $\mu\text{g}/\text{m}^3$ increase in 3-day mean BS with an estimated 0.2% (95% CI, 0.0–0.5%; $p = 0.085$) increase respiratory in death from other causes.

Sensitivity to ban on coal sales. Including an indicator for the ban in the distributed lag model produced no change in the estimated associations with temperature or BS. For example, for total nontrauma deaths the

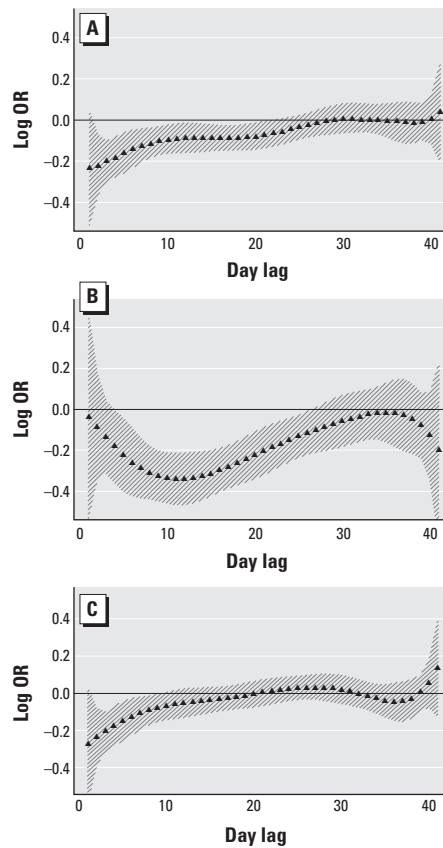


Figure 3. Polynomial distributed lag analysis of (A) cardiovascular, (B) respiratory, and (C) other mortality versus minimum temperature adjusted for same-day minimum temperature. Percent increase in cause-specific mortality for each 1°C decrease in daily minimum temperature for lags 1–41 days fitted with a sixth-degree polynomial.

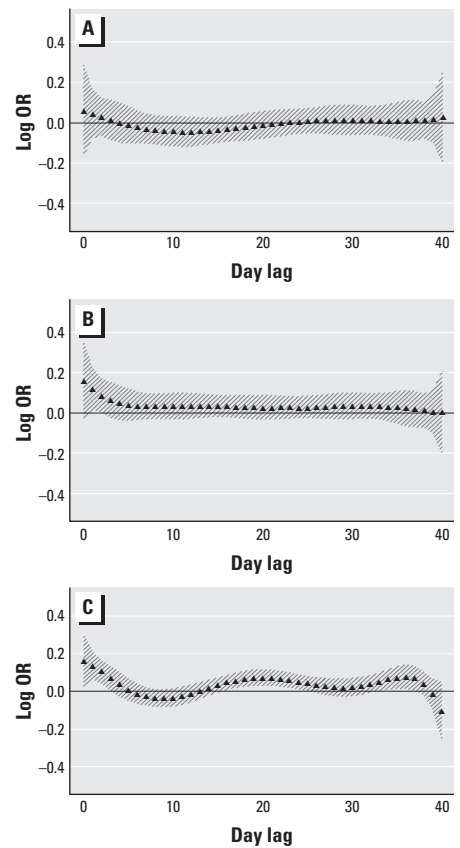


Figure 4. Polynomial distributed lag analysis of total nontrauma mortality versus BS adjusted for minimum temperature for ages (A) 0–64, (B) 65–74, and (C) ≥ 75 years. Percent increase in total mortality for each 10- $\mu\text{g}/\text{m}^3$ increase in mean BS for lags 0–40 days fitted with a sixth-degree polynomial.

Table 3. Estimated percent increase in mortality (95% CI) associated with each 10- $\mu\text{g}/\text{m}^3$ increase in 3-day running mean BS and cumulative 40-day association with each 10- $\mu\text{g}/\text{m}^3$ increase in BS.

Category	Same-day heat effect	40-day cumulative cold
All nontrauma deaths	0.4 (0.3–0.6)	1.1 (0.8–1.3)
Age category (years)		
0–64	0.1 (–0.3 to 0.4)	–0.1 (–0.6 to 0.5)
65–74	0.5 (0.3–0.8)	1.6 (1.1–2.1)
≥ 75	0.5 (0.3–0.7)	1.4 (1.1–1.8)
Cause-specific		
Cardiovascular	0.4 (0.2–0.7)	1.1 (0.7–1.5)
Respiratory	0.9 (0.5–1.2)	3.6 (3.0–4.3)
Other	0.2 (0.0–0.5)	–0.2 (–0.7 to 0.2)

effects of each $10\text{-}\mu\text{g}/\text{m}^3$ increase in BS daily mean concentrations after adjustment for the coal sale ban was 1.1% (95% CI, 0.8–1.4%; $p < 0.0001$), equivalent to the results without adjustment for the ban (Table 3).

We also assessed differences in the temperature and/or BS effects in the periods before versus after the ban on coal sales. We found that each 1°C decrease in minimum temperature was associated with an increase of 2.6% (95% CI, 2.1–3.0%; $p < 0.001$) in total nontrauma deaths over the subsequent 40 days before the ban on coal sales and 2.5% (95% CI, 1.4–3.5%; $p < 0.001$) after the ban. Similarly, no differences in the temperature effect by period were found for any of the analyses stratified by age or cause of death. On the other hand, for each $10\text{-}\mu\text{g}/\text{m}^3$ increase in daily mean BS we found an increase of 1.0% (95% CI, 0.7–1.4%; $p < 0.001$) in total nontrauma deaths over the subsequent 40 days before the ban on coal sales, but 5.4% (95% CI, 3.3–7.5%; $p < 0.0001$) after the ban. Similarly, in analyses stratified by age or by cause of death, we found the estimated association after the ban on coal sales was approximately five times larger than that before the ban, suggesting a nonlinear response function.

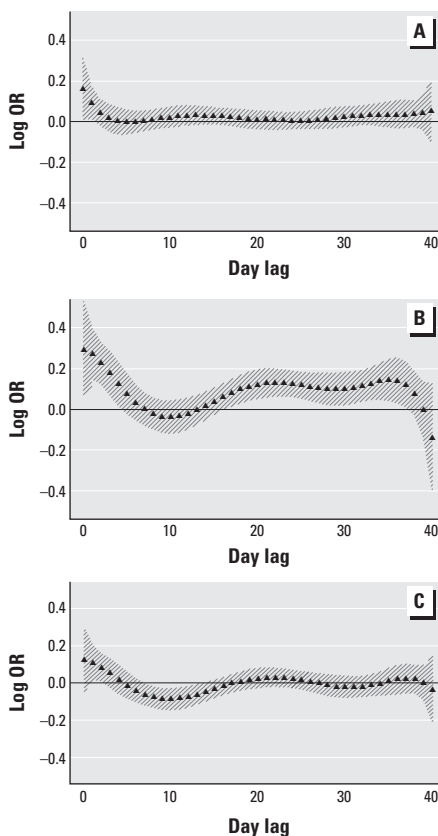


Figure 5. Polynomial distributed lag analysis of (A) cardiovascular, (B) respiratory, and (C) other mortality versus BS adjusted for minimum temperature. Percent increase in total mortality for each $10\text{-}\mu\text{g}/\text{m}^3$ increase in mean BS for lags 0–40 days fitted with a sixth-degree polynomial.

Discussion

Temperature effects. We found an immediate effect of warm temperatures on all-cause mortality in Dublin. Each 1°C increase in temperature was associated with an increase of 0.4% in total mortality on the same day (Table 2). These heat effects were restricted to the older population (≥ 65 years). We also found that cold temperatures were associated with increased all-cause mortality, which extended for 2 to 3 weeks in all age groups. Each 1°C decrease in temperature was associated with 2.6% increased total mortality over the subsequent 40 days.

These effects of temperature on mortality in Dublin were similar to reported associations from other northern European cities. In an analysis of mortality in Scotland, Gemmell et al. (2000) reported that each 1°C reduction in weekly average temperature was associated with an approximately 1% increase in total mortality, which persisted for several weeks. Donaldson and Keatinge (1997) reported that lower mean temperatures in southeast England (including London) was associated with excess deaths from all causes. The maximum effects were observed 3 days after the cold peak and effects lasted out to 40 days. More recently Keatinge and Donaldson (2001) reported that a 1°C decrease in temperature was associated with 3.0% increase in total deaths over the next 24 days in greater London. Others have also reported strong associations between increased mortality and prolonged periods of cold weather in London (Eurowinter Group, 1997; Wilkinson et al. 1999).

In the Netherlands Huynen et al. (2001) found that total mortality increased in the 30 days after heat events by 2.72% for each increase of 1°C , and increased over the 30 days after cold events by 1.37% for each 1°C decrease in mean temperature. The largest effect of heat was observed on the same day as the warm temperature event, whereas mortality effects of cold extended out to 30 days. As in our analysis, they found little effect in the Netherlands of temperature among those < 65 years of age.

These temporal associations with mortality vary with cause of death. In Dublin, we found no effect of heat events on cardiovascular deaths, but an immediate effect of cold on cardiovascular mortality that decreased over the subsequent 3 weeks. The net effect of each 1°C decrease in temperature was a 2.5% increase in cardiovascular mortality over the succeeding 40 days. In the Netherlands Huynen et al. (2001) reported a 1.7% increase in cardiovascular mortality in the 30 days after each 1°C temperature decrease. In Scotland, ischemic heart disease mortality increased by around 1% in the week of and the week after a 1°C decrease in temperature, with little association thereafter (Gemmell et al. 2000). Winter deaths in

London due to coronary thrombosis peak about 2 days after a cold spell (Keatinge 2002). In an analysis of cardiovascular mortality in eight cold-climate U.S. cities, Braga et al. (2002) found that both high and low temperatures were associated with increased cardiovascular mortality. The effect of high temperatures on cardiovascular deaths was restricted to the day of and the day after the heat events, whereas the effects of cold temperatures persisted for several days. Cardiovascular general practitioner visits by the elderly in London were not associated with cold temperature (Hajat and Haines 2002).

Respiratory deaths in Dublin increased on the day of a heat event by 0.8% for each 1°C increase in temperature. In the 40 days after each decrease of 1°C in temperature, respiratory deaths increased by 6.7%. In the Netherlands a 12.8% increase in respiratory deaths was reported after each 1°C increase in monthly temperature (Huynen et al. 2001). These respiratory mortality effects were strongest in the days immediately after the heat event. For cold events they found a 5.2% increase in respiratory deaths after each 1°C decrease in average monthly temperature. The cold effects on respiratory mortality were strongest 2 to 4 weeks after the cold event. Winter respiratory deaths were reported to peak about 12 days after a cold spell (Keatinge 2002). In Scotland respiratory deaths increased by about 1% 1 to 2 weeks after a decrease of 1°C in weekly mean temperature (Gemmell et al. 2000). In eight cold-climate U.S. cities, little effect of cold temperatures on deaths from chronic obstructive pulmonary disease or pneumonia was reported (Braga et al. 2002). General practitioner visits by the elderly for respiratory disease in London were found to rise by 10.5% for each 1°C drop in temperature (Hajat and Haines 2002).

In summary, warm temperatures have an immediate effect on mortality, specifically cardiovascular mortality, and cold temperature have strong effects on both cardiovascular and respiratory mortality that can persist from days to weeks. The mechanism by which temperature contributes to increased risk of death varies by cause of death. These links must be considered when assessing the cumulative effects of air pollution on death over extended periods of time after exposures.

Particulate pollution effects. After adjusting for temperature effects on the same day through 40 days lag, we found a net increase of 1.1% in all nontrauma deaths associated with each $10\text{-}\mu\text{g}/\text{m}^3$ increase in daily mean BS in the 40 days after exposure (Table 3). Previous studies have focused on the effects of particulate air pollution over the few days immediately after exposure. We compared the cumulative 40-day effects to the estimated effect for a 3-day moving average. All-cause mortality (Table 3) in the subsequent 40 days (1.1%) was almost

three times that for the 3-day moving average (0.4%).

Other studies have compared the estimated associations with acute particle exposures (1- to 3-day mean) with the estimated cumulative associations over extended follow-up periods (Table 4). For follow-up periods of 5 days, it was found the estimated effect of particulate matter < 10 μm aerodynamic diameter (PM_{10}) was approximately 20% higher than the estimated effects based on the 2-day mean (Schwartz 2000b). However, in analyses that considered cumulative effects over 40- to 60-day follow-up periods, the estimated associations were two to three times larger than the acute effects of 1- to 2-day mean exposures. Thus studies focusing on mortality in short follow-up periods after particle exposure (APHEA Project 1996; Dockery and Pope 1994; Katsouyanni et al. 1990) consistently and substantially report smaller associations than comparable analyses of longer follow-up (Braga et al. 2001; Rossi et al. 1999; Schwartz 2000a; Zanobetti et al. 2002).

In this analysis we considered extended effects of temperature and particulate air pollution simultaneously. Previous studies have considered temperature effects only in the 1–3 days before deaths. The consistency of the Dublin results with these other studies, despite the differences in adjustment for temperature, suggest that although there are substantial extended effects of temperature, the observed particle associations do not appear to be the result of uncontrolled confounding by temperature as suggested by Hennessy (2002).

We found the strongest associations within the few days after particle exposure. We also found decreasing associations over the first week with negative associations 1–2 weeks after exposure (possible harvesting), and positive associations 3–5 weeks after exposure (Figure 1). This pattern is consistent with the distributed lag model results for total mortality from Milan (Rossi et al. 1999) and the analysis of 10 European cities (Zanobetti et al. 2002).

For the younger age group (< 65 years), we found no evidence of an extended air pollution effect (Table 2; Figure 4). However, for the population ≥ 65 years of age there was evidence of immediate increased mortality effects of particulate air pollution in the 4–5 days after exposure, with a small residual increased risk

that extended for approximately 5 weeks (Figure 4). Moreover, for the population ≥ 65 years of age there is clear evidence that the total cumulative effect of particulate pollution exposure over 40 days of follow-up is much larger than the 3-day mean exposure estimates.

We found the largest relative increase in mortality associated with particles was from respiratory causes in the 5 days after BS exposure. There was some suggestion of mortality displacement (harvesting) shown by the negative associations 8–13 days after exposure. However, delayed respiratory mortality risk was significantly elevated 3–5 weeks after exposure. The net effect of each 10- $\mu\text{g}/\text{m}^3$ increase in BS concentrations was a 3.6% increase in respiratory mortality over the subsequent 40 days. This is four times the estimated effect (0.9%) of each 10- $\mu\text{g}/\text{m}^3$ increase in the 3-day mean BS concentration.

In a meta-analysis of the distributed lag effects in the 5 days after PM_{10} exposure in 10 U.S. cities, Braga et al. (2001) found the largest increase in deaths from pneumonia and from chronic obstructive pulmonary disease in the 2–3 days after exposure. In Boston, Schwartz (2000a) found the estimated cumulative effect of particulate matter < 2.5 μm aerodynamic diameter ($\text{PM}_{2.5}$) on pneumonia deaths initially decreased, as the follow-up window was increased from 0 to 15 days but then increased substantially as the window was increased to 45 and 60 days follow-up (consistent with the Dublin results). On the other hand, deaths from chronic obstructive pulmonary disease associated with $\text{PM}_{2.5}$ increased as the window increased from 5 to 15 days follow-up, but then decreased to zero association at the 60-day follow-up window. These Boston results suggest that most of the cumulative respiratory deaths attributable to particles in the Dublin analyses may be specific to pneumonia.

Cardiovascular mortality increased immediately after particle exposure but declined within 3 days after exposure to zero (Figure 5). There was a very small increased risk extending out to 40 days after exposure. It is possible that this slight increase at long follow-up times may represent cross-coding or miscoding of some respiratory deaths as cardiovascular. The cumulative effect of each 10- $\mu\text{g}/\text{m}^3$ increase in BS over the 40 days after exposure was a 1.1% increase in cardiovascular mortality (Table 3).

This is almost three times the estimated effect of the same increase in 3-day average BS (0.4%, Table 3).

Braga et al. (2001) reported that cardiovascular mortality associated with PM_{10} exposures was associated most strongly on the day of and the day after exposures, although they considered follow-up periods only through 5 days. In Boston, the association of $\text{PM}_{2.5}$ with ischemic heart disease deaths increased monotonically as the follow-up window increased from 0 to 60 days (Schwartz 2000a).

Issues relating to the black smoke method.

The BS method was developed to monitor pollution from coal burning (British Standards Institute 1969) and was thus well suited to Dublin, where coal burning was the major source of particulate pollution. BS is measured by reflectance, where the “blackness” rather than mass of the filter is determined. The BS sampler has a upper cut-off at about 4.5 μm (McFarland et al. 1982). Dockery and Pope (1994) suggested BS is well correlated with PM_{10} for the purpose of health studies. In Dublin the effect of the ban on coal sales was clearly seen with the BS measurements (Clancy et al. 2002), whereas in other parts of Ireland where there was no ban there was no change in BS levels. Le Tertre et al. (2002) showed that the BS measurement showed the most robust results for cardiovascular diseases.

Conclusions

We found the effects of both particulate air pollution and temperature on mortality persisted for 3–4 weeks after exposure. We found that the temperature effects are more prolonged for respiratory deaths compared to cardiovascular deaths. We also found the effects of particulate air pollution on mortality are strongest on the day of and the few days after exposure but extend out through about 40 days after exposure. This extended air pollution association is most marked for the elderly population groups and for respiratory causes of death. These extended follow-up effects were two to three times greater than the acute effects reported in other studies, and approach the effects reported in longer-term survival studies. This analysis suggests that the studies on the acute effects of air pollution have underestimated the total effects of temperature and particulate air pollution on mortality.

Table 4. Estimated percent increase in mortality (95% CI) associated with each 10- $\mu\text{g}/\text{m}^3$ increase in particulate air pollution in studies comparing acute effects (1–3 days) with extended follow-up (up to 60 days).

Study	Particle measure	Period	Acute effects		Extended effects	
			Period	Percent increase (95% CI)	Period	Percent increase (95% CI)
Boston (Schwartz 2000a)	$\text{PM}_{2.5}$	2 day		2.2 (1.5–2.9)	60-day window	3.8 (3.2–4.3)
Milan (Rossi et al. 1999)	TSP	1 day		0.3 (0.2–0.4)	45-day follow-up	0.8 (0.4–1.1)
Dublin	BS	3 day		0.4 (0.3–0.6)	40-day follow-up	1.1 (0.8–1.3)
10 U.S. cities (Braga et al. 2001)	PM_{10}	2 day		1.1 (0.9–1.2)	5-day follow-up	1.3 (1.0–1.6)
10 European cities (Zanobetti et al. 2002)	PM_{10}	2 day		0.7 (0.4–1.0)	40-day follow-up	1.6 (0.8–2.4)

TSP, total suspended particulates.

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