Associations between Outdoor Air Pollution and Daily Mortality in Brisbane, Australia

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ABSTRACT. The results of several studies have indicated significant associations between daily mortality and air pollution, with little evidence of a threshold. In the current study, the authors examined daily mortality during the period 1987–1993 for the Brisbane region, which is the fastest-growing urban region in Australia (annual average concentration of particulate matter less than 10 µm in diameter = 27 µg/m^3, maximum hourly sulfur dioxide level = 60 ppb, and maximum daily ozone hourly level = 118 ppb). The authors conducted a general estimating equation analysis, and they used autoregressive Poisson models for daily mortality to examine associations with air pollution variables. The authors used research methods developed in the Air Pollution on Health, European Approach (APHEA), project to control confounding effects of weather and temporal trends. The air pollutants examined included particulate pollution (measured by nephelometry [bsp data]), sulfur dioxide, ozone, and nitrogen dioxide. The results indicated that the associations between total daily mortality and particulate levels found in studies in the United States and other countries may be applicable in Brisbane, Australia. Ozone levels were also associated significantly with total daily mortality. There was little evidence of interaction between the ozone effects (mainly in summer) and particulates or with sulfur dioxide and nitrogen dioxide. The associations between pollutants (ozone, bsp) and daily mortality were significant only for individuals who were older than 65 y of age; positive associations were also found with cardiovascular disease categories, and the regression coefficients—when significant—were higher than those for total mortality. The results indicated a possible threshold for ozone levels, but a similar result for particulate levels was not apparent.

CONSISTENT ASSOCIATIONS between air pollution and daily mortality, and which occur across a range of climatic conditions and cities, have been described in recent comprehensive reviews.[1–3] One of the most significant findings noted in these reviews is that adverse health effects are occurring at levels below current guidelines, and there is no evidence of a threshold for particulate pollution. With respect to the effects of particulate pollution, Dockery and Pope[2] noted a good consistency across a range of studies, with estimates ranging from an increase in mortality of 0.6–1.6% for each 10-µg/m^3 increase in concentrations of particulate matter less than 10 µm in diameter (PM_{10}) (a weighted mean of 1.0%). They noted stronger associations for cardiovascular mortality and even stronger ones for respiratory mortality. The clinical explanation for such a result is not clear; perhaps the true association is with some other pollutant correlated with PM_{10} or with a specific component of the mixture of particles that makes up PM_{10} (e.g., acidic aerosols arising from emis-

sions of sulfur dioxide [SO_2]). However, investigators found that the dose-response relationship remained in areas that had both low and high levels of SO_2. The results of other studies in the United Kingdom,[4] as well as preliminary ones in Australia[5] (where the SO_2 levels are very low) have indicated that both particulates and ozone (O_3) are associated with daily mortality.

Regardless of whether the associations are valid for PM_{10}, SO_2, or O_3 levels, the question remains: do they persist to low concentrations, or in different regions apart from North America? This question is of some importance to countries, such as Australia, and, in particular, to an urban region (e.g., around the city of Brisbane—the fastest-growing urban region in Australia). In Europe, investigators have made a concerted attempt to address this question via the short-term effects of Air Pollution on Health: European Approach project (APHEA).[6,7] In this project, European researchers have made a collaborative effort to estimate and compare the associations found between air pollution and population health measures (e.g., daily mortality and hospital admissions) for 15 European cities. In several of the studies in this project, researchers concluded that particulates are associated with daily mortality, but these associations may be linked to the effects of sulfur dioxide[8-11]; however, the results of other studies have not indicated any significant associations.[12-14]

In the present study, we used the APHEA protocol[6,7,15] to examine associations between daily mortality and air pollution levels monitored in Brisbane, Australia.

Material and Method: The APHEA Protocol

We followed the research protocol[6,7,15] developed for the APHEA project so that we could make comparisons between this study and overseas studies. In the first stage of this project, we developed and standardized the methods to be used to derive estimates for the short-term health effects of air pollution. The existing APHEA protocol involves the modeling of all potential confounding factors (e.g., season, meteorological effects, day of week, holidays, influenza epidemics); selection of the "best" model (using a variety of diagnostic tools); and, finally, use of autoregressive Poisson models that allow for overdispersion to identify associations between air pollution and population health outcomes.[6,7,15]

Mortality data. We obtained mortality data from the Queensland State Government Statistician's Office for the Brisbane Statistical Sub-Division (BSSD), which is within the Brisbane Statistical Division (BSD) (Fig. 1). We did not include deaths from accidental causes or deaths outside the BSSD in the analysis. We used data in the analysis for the period January 1, 1987, to October 31, 1993 (i.e., 2 496 d of observations). In 1991, the population for the BSSD was 0.75 million (the total BSD population was 1.36 million).[16] Of the 15 European cities in the APHEA project, the BSSD population is less than that for 7 cities[6] (the BSD population is less than that for 4 cities).

In this study, we used the International Classification of Diseases (ICD) code numbers 460.0-519.9 to classify mortality from respiratory diseases; and ICD code numbers 393.0-398.9, 402.0-402.9, 404.0-404.9, 410.0-416.9, 420.0-420.9, 429.0-429.9 to classify mortality from cardiovascular diseases (restricted to heart disease categories). We excluded mortality for ICD codes > 800.0 (injury and poisons). The statistics for the period under study are shown in Table 1. The BSSD daily mortality was greater than that for 3 of 15 European cities studied in the APHEA project, and it was comparable with 2 other cities[6] (the BSD rate, which was 40% higher on average than in other cities, was greater than that for 7 cities).

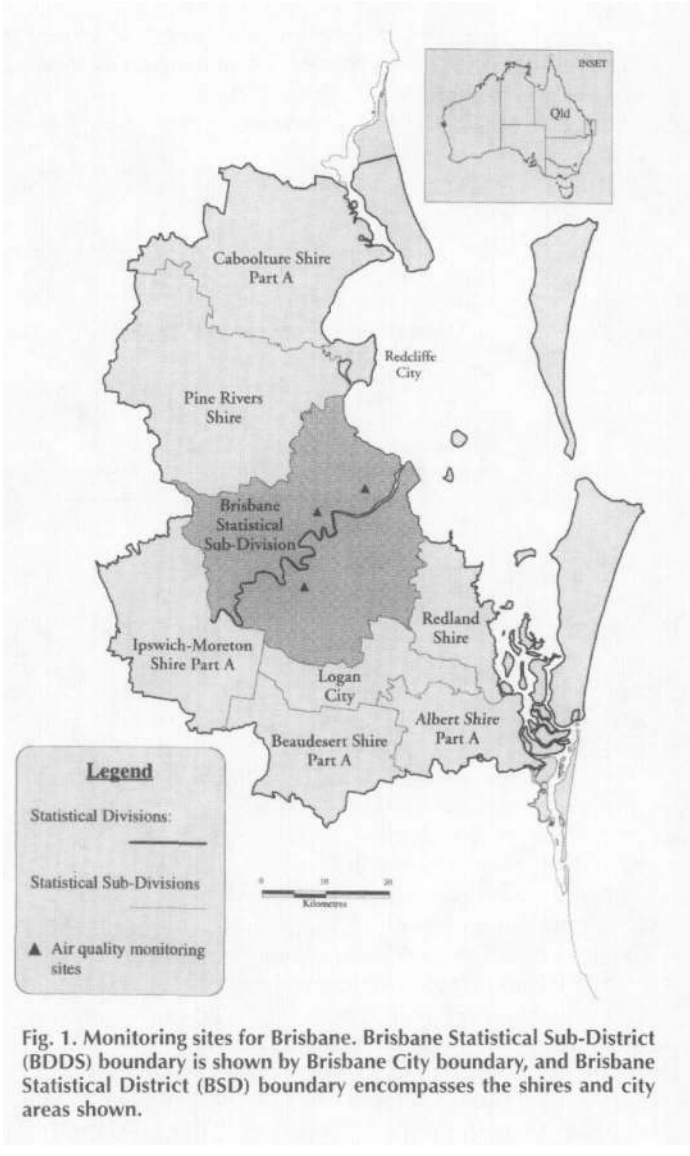

Fig. 1. Monitoring sites for Brisbane. Brisbane Statistical Sub-District (BDDS) boundary is shown by Brisbane City boundary, and Brisbane Statistical District (BSD) boundary encompasses the shires and city areas shown.

Air monitoring and weather data. Air pollution data were collected at three monitoring sites (i.e., Fortitude Valley, Rocklea, and Eagle Farm) run by the Queensland State Government Department of Environment (DoE) in Brisbane (Fig. 1). The data for nephelometry (bsp), oxides of nitrogen (nitrogen oxide and nitrogen dioxide [NO_2]), ozone (O_3), and SO_2 were collected and reported as half-hour averages. Investigators derived average air pollution values in Brisbane by averaging NO_2, bsp, and O_3 levels recorded at the three sites and

Table 1.—Daily Mortality (Nonaccidental) for the Brisbane Statistical Sub-District (BSSD) for January 1, 1987 to October 31, 1993 (N = 2 496)

Category	Daily mortality (deaths/d)									
	Summer		Autumn		Winter		Spring		All year	
	$\bar{x}$	*SD*	$\bar{x}$	*SD*	$\bar{x}$	*SD*	$\bar{x}$	*SD*	$\bar{x}$	*SD*
Total mortality										
All years	14	4	15	4	19	5	16	4	16	5
> 65 age group	11	3	12	4	15	4	13	4	13	4
Cardiovascular mortality										
All years	4	2	5	2	6	3	5	2	5	2
> 65 age group	4	2	4	2	5	2	4	2	4	2
Respiratory mortality										
All years	1	1	1	1	2	1	1	1	1	1
> 65 age group	1	1	1	1	2	1	1	1	1	1

by averaging SO_2 data recorded at the Fortitude Valley and Eagle Farm sites (data were not available at the other site). We used these data to construct 24-h averages, and we constructed 8-hour O_3 averages for the period 10:00 A.M. to 6.00 P.M. We derived daily maximum hourly NO_2 and SO_2 levels from the average of the maximum values for these sites; we derived daily maximum hourly O_3 levels from the average of the maximum values for these sites for the period 10:00 A.M. to 6:00 P.M. We collected PM_{10} data every 6 d at 5–6 sites; therefore, only the data for light scattering by nephelometry (bsp) constituted the daily data set for particulate pollution used here. We obtained meteorological data (i.e., minimum and maximum temperature, and relative humidity [A.M./P.M.]) from the Federal Government Bureau of Meteorology for Brisbane Airport, which is near the Eagle Farm air-quality monitoring site (i.e., site nearest the coast in Fig. 1).

The APHEA protocol suggests that three or more monitoring sites should be used and that at least one should be near an urban traffic road and at least one should be near a "background" station. Of the three sites used in our study, one was an inner-city site and the other two were in commercial/residential areas; all were urban sites, and the three sites were more representative of the more concentrated urban BSSD area than the BSD region (Fig. 1). Previous work in Brisbane[17-20] indicated that the pollutant data for all the sites were correlated significantly, especially PM_{10} data. With respect to the period under study, all correlations between data collected at the stations were significant ($p < .0001$ [O_3 data—correlations ranged from 0.4 to 0.8; NO_2—0.4 to 0.5, bsp—0.5 to 0.6, and SO_2—0.2 {only the two stations located nearest the coast shown in Figure 1}]). These correlations generally reflected results from previous work[17] in Brisbane and indicated the strong correlations between pollutant (e.g., O_3, NO_2, bsp) readings at these sites that arose mainly from diffuse sources (e.g., motor vehicles) but not for those that arose predominantly from point sources (e.g., SO_2).

The mean pollution levels for the 1987–1993 period are shown in Table 2 for all year, summer, and winter periods. With respect to the major air pollutants, the maximum hourly O_3 levels in Brisbane appeared to be higher than those in most of the cities used in the APHEA project[6]; however, the highest 1-h O_3 level recorded for the 1987–1993 period in Brisbane was 118 ppb, with approximately 80% of the data records reflecting levels below 30 ppb. The particulate monitoring stations in the European cities used Black Smoke, which, for comparative purposes,[2] can be assumed to be roughly equivalent to the PM_{10} measure as an upper limit; in that case, the Brisbane PM_{10} levels were lower than those for 5 of the 10 cities for which there was particulate monitoring.[6] The annual reported average PM_{10} concentrations in the United States (as reported in the studies considered in one review[2]) ranged from 28 $\mu g/m^3$ to 61 $\mu g/m^3$, whereas the recorded mean levels at sites in Brisbane during the 1987–1993 period was 27 $\mu g/m^3$. The mean SO_2 level in Brisbane was low (i.e., 4.2 ppb) for the 1987–1993 period, and this level was lower than any found in 7 of the 15 cities monitored in the APHEA project.[6]

Data for the mean minimum and maximum temperatures and for relative humidity are shown in Table 2. Brisbane has a subtropical climate,[18] and it is much warmer than any of the cities included in the APHEA project.[6]

Statistical models. We used category of death (i.e., cardiovascular, respiratory, and total) to analyze the mortality date, by age (i.e., < 65 y, ≥ 65 y, and total years). Given that daily mortality is a count of a rare event, we used Poisson regression to examine the associations. The APHEA protocol suggests that autoregressive Poisson regression models should be used for daily mortality data, thus allowing for overdispersion. We used generalized estimating equation (GEE) techniques.[21,22] Temperature, together with seasonality, is likely the most important environmental predictor of mortality patterns.[6] The APHEA protocol suggests that temperature and humidity measurements be used in all analyses to control for any weather effects. Seasonal and chronological variables (i.e., day of week, cycles) may also need to be included in the analysis. Other variables used should include influenza events and holidays.

Modeling for Control of Confounders

It is clear that the impact of air pollution on daily mortality, if significant, is very small,[7-15] and several other fac-

Table 2.—Environmental Measurements for Brisbane, 1987–1993

Variable	Mean	Median	Minimum	Maximum
PM_{10} ($\mu g/m^3$) 24-h average*				
All year	26.9	24.8	4.3	76.2
Summer†	25.1	22.7	10.7	59.6
Winter	28.6	27.5	4.3	76.2
bsp (10^5/m) 24-h average				
All year	2.59	2.16	0.30	15.16
Summer	2.27	1.90	0.42	14.17
Winter	2.93	2.53	0.30	15.16
bsp (10^5/m) daily 1-h max				
All year	6.68	5.17	0.78	77.73
Summer	4.89	4.30	0.78	50.88
Winter	7.70	6.27	0.95	77.73
SO_2 (ppb) 24-h average‡				
All year	4.2	3.6	0	35.5
Summer	3.9	3.4	0	20.6
Winter	4.5	3.8	0	35.5
SO_2 (ppb) daily 1-h max‡				
All year	9.6	8.3	0	59.5
Summer	8.5	7.5	0.1	55.0
Winter	10.7	9.0	0	59.5
NO_2 (ppb) 24-h average				
All year	14.0	13.2	1.2	41.7
Summer	11.5	10.7	2.0	36.7
Winter	16.4	15.8	1.2	41.7
NO_2 (ppb) daily 1-h max				
All year	28.4	27	3.5	82
Summer	24.1	22.7	3.5	82
Winter	32.6	31	3.5	80.5
O_3 (ppb) 8-h average§				
All year	18.1	16.7	1.7	63.4
Summer	20.2	18.7	2.7	63.4
Winter	16.1	15.3	1.7	56.9
O_3 (ppb) daily 1-h max				
All year	24.2	21.3	2.5	101.5
Summer	26.9	23.7	4.5	97.5
Winter	21.7	20	2.5	101.5
Minimum temperature				
All year	15.7	16.5	1.3	25.9
Summer	19.1	19.4	7.8	25.9
Winter	12.4	12.6	1.3	22.0
Relative humidity (A.M.)// (%)				
All year	67.4	68	17	99
Summer	66.4	66	17	99
Winter	68.4	69	20	99
Maximum temperature				
All year	25.2	25.4	13.8	38.5
Summer	27.9	28	18.5	38.5
Winter	22.7	22.6	13.8	32.6

Notes: PM_{10} = particulate matter < 10 μm in diameter, bsp = data for nephelometry, SO_2 = sulfur dioxide, NO_2 = nitrogen dioxide, and O_3 = ozone.
*Twenty-four-hour average every 6 d.
†Summer = October–March.
‡Valley and Eagle Farm data were used.
§Average: 10:00 A.M. to 6:00 P.M.
//Brisbane Airport data (daily); temperature was in °C.

tors must be controlled carefully. However, it is also clear that such "corrections" can yield misleading results[10] unless much care is taken. We followed the APHEA protocol, and the steps we used are set out in the following subsections.

Seasonality and other cyclical patterns. Time-series plots of daily mortality data revealed distinct seasonal cycles (for example, see the data for total mortality in Fig. 2). The approach adopted by investigators in the APHEA protocol was to use harmonic wave terms to smooth these patterns. In other studies, researchers have relied on dummy variables for each month, or season, of the analysis, and this can lead to, respectively, overspecification or underspecification of the model.[15] The frequency or period of the harmonic wave (cosine or sine) is determined by term ωt ($\omega = 2\pi/365$), where t

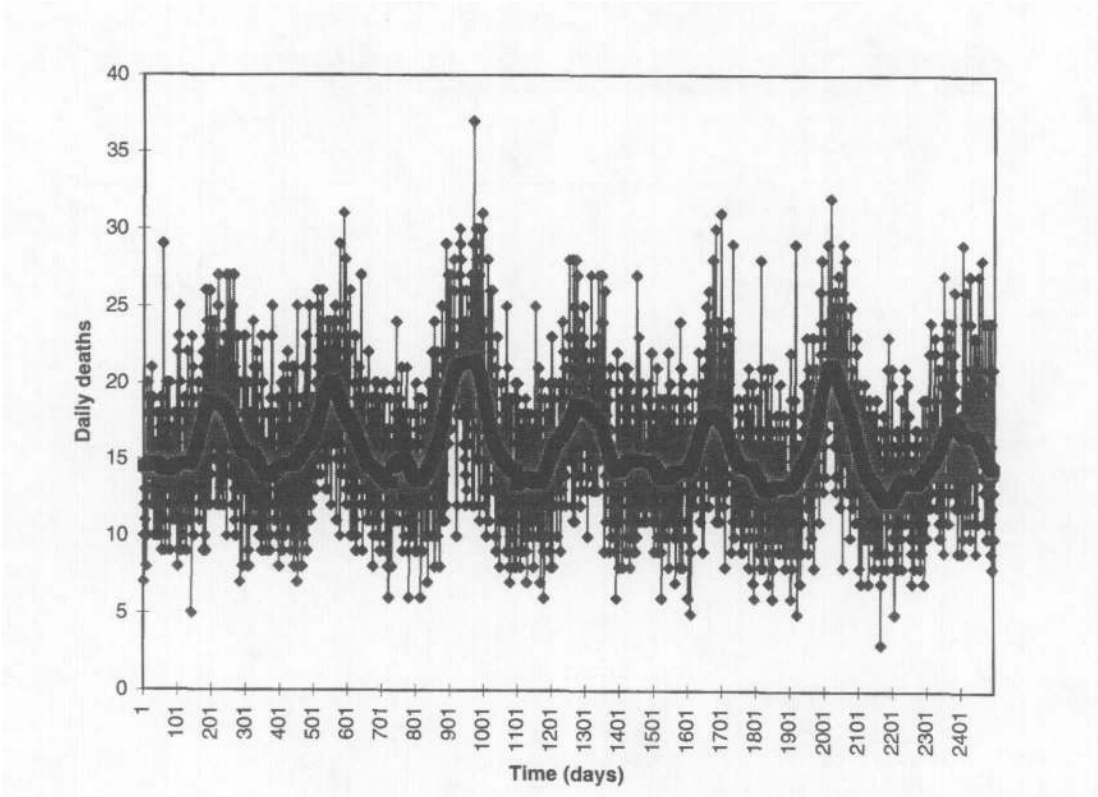

Fig. 2. Daily mortality for Brisbane Statistical Sub-District (BSSD) versus time, with "deseasonalized" model fit.

is the time variable (herein it was daily) for the 1-y period, and, for shorter periods, by terms $k\omega t$ ($\omega = 2\pi k/365.25$); in the APHEA studies, investigators considered cosine and sine terms with $k \leq 6$ (both cosine and sine terms of a given period have to be included).

We used the SAS 6.11 package to analyze the mortality data for spectral density to identify cycles for k with integer (and noninteger) values.[15] The annual cycle ($k = 1$) was by far the most dominant (Fig. 2). Corrections were needed for $k < 1$ if there were differences between years (seasons in different years) or differences in the data (the APHEA protocol indicates that, at times, a value of k, such as 0.5, may need to be included). In addition, corrections were needed for $k > 1$, up to $k = 6$, thus causing removal of significant cycles down to periods of 2 mo, which were related to steep increases or flat "dips" in the data (in part from short summers and long winters), and the removal of additional peaks and dips beyond simple seasonal patterns.

Clearly, controlling for each summer/winter period was important. We chose the following months: summer—October to March, winter—April to September (in Brisbane the seasons divide into a wet spring/summer period and a dry autumn/winter period, with the rainy season usually ending at the end of March). We then examined the use of dummy variables. However, as noted elsewhere,[15] this approach is equivalent to fitting a histogram to the data and can easily lead to either overspecification or underspecification of the model; we therefore preferred fitting the data with smooth harmonic waves. Nevertheless, analysis of the total mortality data indicated that a dummy variable for 1989 was necessary (but not for any of the other years after harmonic analysis was carried out). In Sydney there was an increase in mortality from an influenza epidemic in 1989 (G. Morgan, private communication, 1997).

Making the final decision on this "deseasonalized" model was the most critical part of the analysis. In several of the APHEA papers (e.g., Spix and Wichmann[10]) the sensitivity of the analysis at this point was discussed. The decision rules we used were as follows: (a) First, fit the "best" harmonic wave model so that the residuals are independent of the harmonic waves ($k \leq 6$) and such that spectral density analysis shows the long waves have been filtered out; (b) then use year and/or season dummy variables and choose the model with the fewest terms that reduces the residuals as close as possible to white noise. We tested the latter by examining the serial correlation in the residuals (up to lag 7). In this analysis, we found that significant serial correlation in lags 1–7 could arise by either overspecification or underspecification of the model.

In most of the APHEA papers, investigators used integer values of k (as well as $k = 0.5$), although in the example cited in the introductory paper by Schwartz et al.[16] on methodology, results of spectral density analysis on the data were used to reveal the need to include a noninteger value of k. In our study, we used spectral density analysis to identify significant integer and noninteger values of k according to the decision rule chosen for part (b) described earlier. We also found that if we used only integer values of k in fitting harmonic waves to the data, spectral density analysis on the resulting residuals revealed that significant long-term cycles remained in the data. We also found that the

inclusion of noninteger values of k in the harmonic analysis removed the need in the latter part of the analysis to control for other temporal effects (e.g., needing dummy variables for year, season). For example, for total daily mortality data, integer values of k = 1, 2, and 5 (approximately corresponding to effects at annual, 6-mo, and 73-d cycles, respectively) were identified as significant terms, as well as noninteger k values of 0.44, 0.73, 1.32, 1.46, 1.61, and 3.23 (approximately corresponding to 27-mo, 17-mo, 10-mo, 9-mo, 8-mo, and 4-mo cycles, respectively); the fit of these harmonic waves is shown in Figure 2. The resulting residuals in this "deseasonalized" model are shown in Figure 3. We compared the results of spectral density analysis on the residuals for the "deseasonalized" model from Figure 3 with those for the raw data for total daily mortality (minus the annual cycle) (Fig. 4) to show how the long-wave terms were filtered out and the short-wave terms were retained.

Short-term effects of meteorological factors. Several temperature and humidity models are suggested in the APHEA protocol, including polynomial terms and segments for different parts of the data (in some studies, up to 7 different categories of weather variables were used[15]). In our study, we used a variety of models, including those with linear and polynomial terms, and investigated lags of 0, 1, and 2 (following the APHEA protocol). We found that, in general, a combination of minimum temperature (T_{min}), maximum temperature (T_{max}), and relative humidity (A.M. only), all at a lag of 1 d, led to good results.

The "best" model we identified at lag 1 included linear terms in minimum temperature and relative humidity, as well as a linear term in the variable TT = (T_{max} – 28.0) when T_{max} > 28 °C, and in TT = 0 when T_{max} < 28 °C. The results for the analysis of total daily mortality are shown in Figures 5 and 6. In Figure 5, we plotted the "deseasonalized" residuals from Figure 3 (as decile averages) against maximum temperature (at lag 1), and we "corrected" the residuals for weather effects by fitting the weather-correction model (Fig. 6). The associations between weather variables and the residuals in the "deseasonalized" models were nonlinear. For example, in Figure 5 it is clear that the "deseasonalized" residuals were higher for the coldest and hottest weather. Therefore, we used the model that (a) had a linear term in minimum temperature (to account for the inverse temperature relationship); and (b) had the line element, TT, for high maximum temperatures (i.e., 28 °C [Fig. 5]), to account for the hot temperature effect. After we fitted regression models of weather variables to the "deseasonalized" residuals, we chose the "best models," after which we tested the "corrected" residuals for any remaining correlation with weather variables. After we made corrections to temperature (and even before), the effects of relative humidity were not significant, but we included relative humidity (A.M.) and temperature for completeness, as is suggested in the APHEA procedures.

Long-term trends and calendar effects. In the APHEA protocol, researchers suggested testing for day of week, using all six dummy variables in the analysis. With respect to the data used here, these terms were not significant and, therefore, we did not include them. Long-term temporal trends in mortality data that

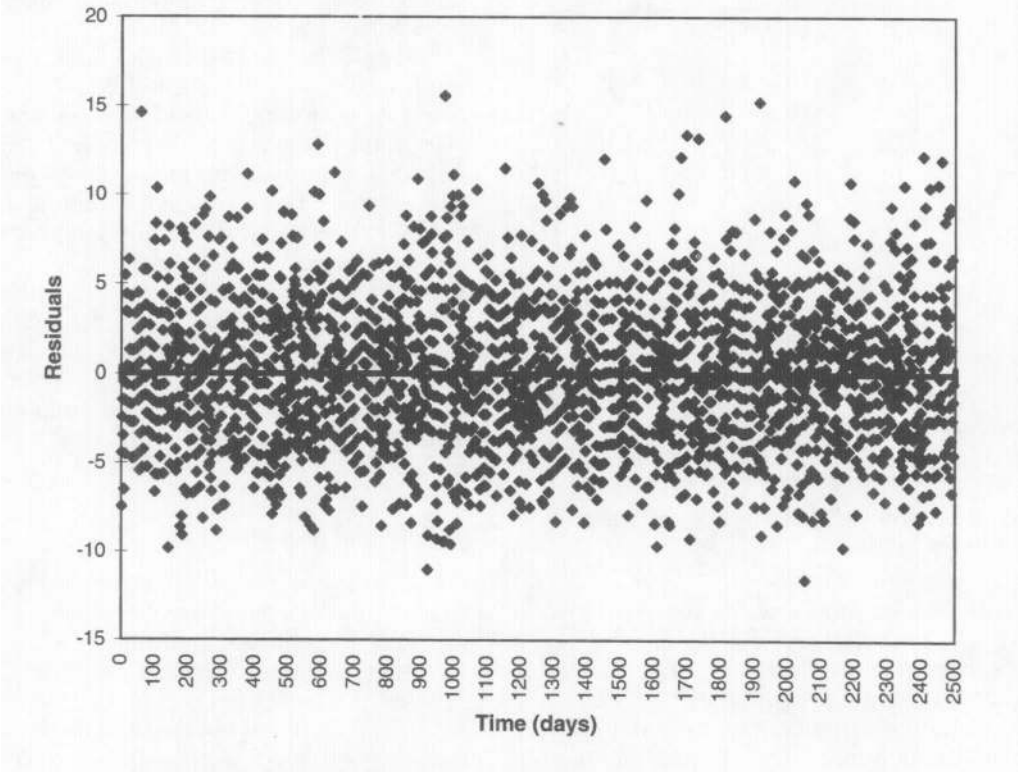

Fig. 3. Residuals of "deseasonalized" model shown in Figure 2.

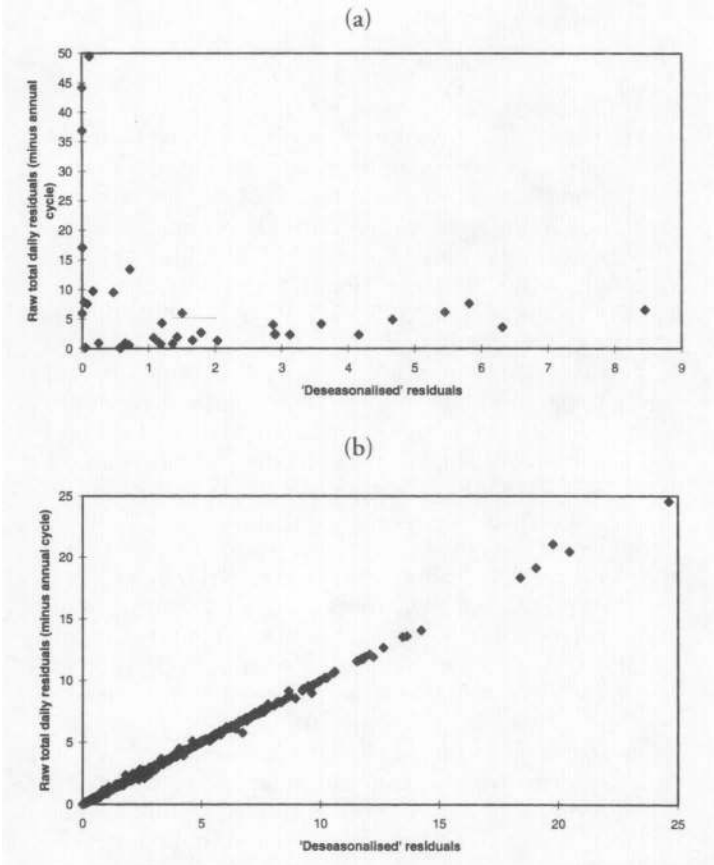

Fig. 4. Comparison of spectral densities (a) between raw total daily (minus annual cycle) and "deseasonalized" residuals from Figure 3 for periods greater than 60 d, and (b) same as (a) but for periods less than 60 d.

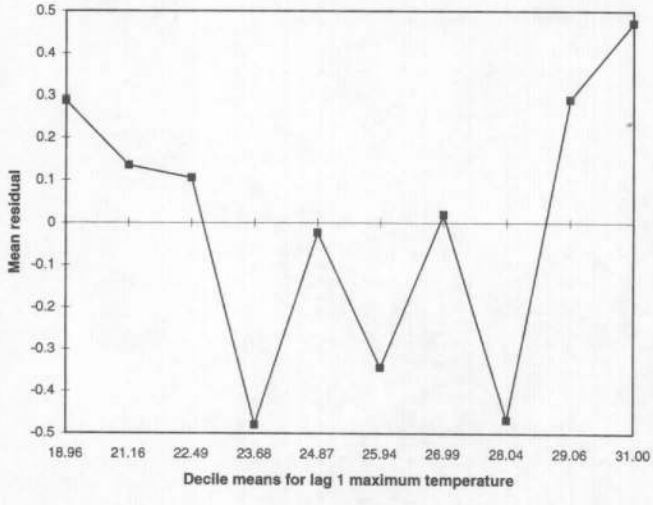

Fig. 5. Residuals of "deseasonalized" model from Figure 3 plotted against decile means for lag 1 of T_{max}.

are not removed by the harmonic wave analysis ($k <$ 1) can generally be controlled for if a linear term is included, or if both a linear and a quadratic term are included, in the time variable (measured by day). Such corrections were sometimes significant for our data, so we included linear and quadratic terms throughout the analysis. Corrections for the Christmas–New Year holiday period in Australia did not yield significant results.

Influenza and other events. The Virology Section of the Center for Public Health Sciences, Queensland Health (B. Harrower, Laboratory of Microbiology and Pathology [personal communication, 1997]) supplied data regarding monthly totals of influenza isolations for the state of Queensland. The data indicated that influenza outbreaks occurred during the period of June to October for most of the years considered. We determined that the results of analysis for total daily mortality necessitated inclusion of a dummy variable for the year 1989—a year during which it was acknowledged that an influenza outbreak occurred in Sydney. The isolation data did not indicate that 1989 was the most severe year, but data were representative of the state, not the region. For respiratory mortality data, we found that a dummy month variable for September 1989 was significant, and we included it, rather than a dummy variable, for that year; the data supplied indicated that during 1989 the most severe influenza isolations occurred in September. With respect to cardiovascular data, no correction was significant. Influenza effects, therefore, appeared to be controlled by harmonic wave analysis or by use of dummy year or month variables.

Autocorrelation. We used autoregressive error models to control for remaining autocorrelation. We found that for all of the models, all serial correlation had effectively ceased after all previous filtering (allowing for weather effects). Nevertheless, we assumed an autocorrelation at lag 1 and used GEE[21,22] algorithms for SAS (supplied by Joel Schwartz, personal communication) in autoregressive Poisson models that allowed for overdispersion.

Final models. After making all these "corrections," we found in all cases that the residuals were white noise (identified by testing for serial correlation up to lag 7). An example of the Poisson regression coefficients for the model fitted to total mortality data is given in Table 3 (autoregressive Poisson model assuming autocorrelation at lag 1). We tested alternative approaches and used only dummy variables for year, season, and/or month; other approaches attempted a combination of harmonic waves with only integer values of k (but including k = 0.5) with these dummy season variables. We used harmonic analysis with integer and noninteger k values, thus yielding results with the smallest number of variables and the smallest number of "adjustments" by other temporal dummy variables.

Air Pollutant Models

In accordance with APHEA protocol, we used single-pollutant models, with lags 0, 1, and 2 for bsp, NO_2 and SO_2 data, respectively, and lags 0–5 for O_3 data. We also tested cumulative effects and used 2-d (lags 0, 1) averages, 3-d averages (lags 0, 1, 2), and 4-d averages (lags 0, 1, 2, 3) for all but the O_3 data, for which we also examined 5-d averages (lags 0–4 d), 6-d averages (lags 0–5), and 7-d averages (lags 0–6). The different effects of

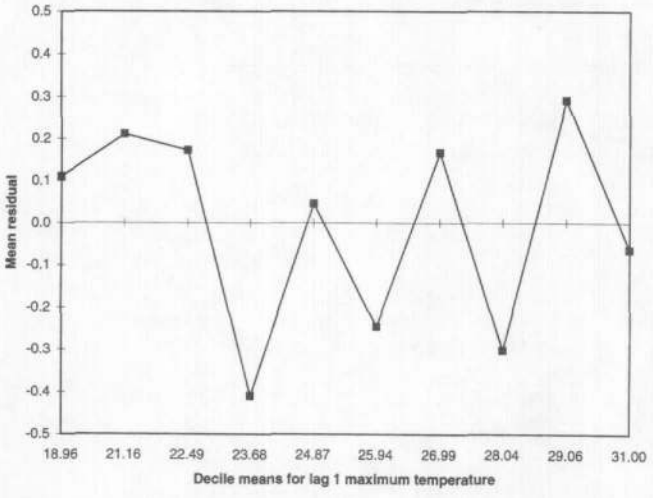

Fig. 6. Mean residuals of "deseasonalized" model from Figure 3, "corrected" by weather model (see Table 4), plotted decile means against lag 1 of T_{max}. Note that the extremes in Figure 4 are absent here.

air pollutants during different seasons are likely important (especially for O_3 in summer during which levels are highest); therefore, we included a dummy variable for summer/winter, as well as an interaction term of the air pollutant and the season variable. Also, the interactions between pollutants may be important; in the foreign studies, investigators have reported a clear relationship between SO_2 and particulates.[8–11] The APHEA protocol does not recommend the use of multi-pollutant models because the collinearity problem exists, so single-pollutant models are used in the first instance. For those pollutants that yielded significant results, we accounted for interaction effects by introduction of a dummy day variable for "low" and "high" values of any possible confounding pollutants. With respect to the pollutants considered in our study, we examined the "high" values for the highest 10% of the data. For example, in the model for bsp, we introduced a dummy variable for "high/low" in a number of cases; we assumed "high" days to be days when the maximum hourly O_3 value exceeded 39 ppb. There was also an interaction term of this dummy variable and the bsp variable. The "high" value we used for bsp data was 4.5×10^{-5}/m. For 24-h average SO_2 data, we used the "high" value of 8 ppb—well below the "high" value of 40 ppb addressed in the APHEA studies.[7] For 24-h average NO_2, we used the "high" value of 22 ppb, which was also well below the "high" APHEA value of 43 ppb.[7]

Correlation analysis revealed many significant relationships between the pollutants, and between the pollutants and meteorological variables (Table 4). There were strong positive relationships between O_3, NO_2, and bsp; however, such associations were strongest for summer, especially between O_3 and NO_2. It was clear that high levels of O_3 associated with warm dry conditions, as well as the positive associations with bsp and NO_2, were strongest in summer. However there was an inverse relationship between both bsp and NO_2 with maximum temperature—unlike the results for O_3. The positive association between bsp and NO_2 was strongest in winter. Also, there were small, but significant, positive associations between SO_2 and NO_2 throughout the year and between bsp and SO_2 in winter.

Results

Same-day pollutant averages were associated most significantly with total daily mortality. We used the GEE models[21,22] introduced earlier to derive the associations between total daily mortality, daily mortality from cardiovascular causes, daily mortality from respiratory causes, and each pollutant (Table 5) for all year, summer, and winter data sets.

Maximum 1-h and 24-h bsp, as well as maximum 1-h and 8-h average O_3 levels were associated significantly with total daily mortality, especially in summer. These associations did not remain significant in the respiratory mortality category, but the daily total number of deaths from respiratory illness was very low (Table 1). The positive associations persisted for the cardiovascular mortality category, but they were significant only for maximum 1-h bsp data (i.e., all year and in winter and 2–3 times higher than for the total mortality coefficients). The results for SO_2 and NO_2 were not significant.

Table 3.—Parameter Estimates for the "Core" Model Variables from Poisson Model, Controlling for Autocorrelation for Total Daily Mortality for Brisbane Statistical Subdivision

Variable	Regression coefficient	*SE*	*t*
Intercept	2.9061	0.038	77.00
Year 1989*	0.0727	0.019	3.88
Cos *y*/0.44	–0.0230	0.008	–2.87
Sin *y*/0.44	0.0040	0.008	0.53
Cos *y*/0.73	0.0306	0.007	4.33
Sin *y*/0.73	–0.0112	0.007	–1.51
Cos 1 *y*	–0.1135	0.014	–7.83
Sin 1 *y*	–0.0521	0.009	–5.94
Cos *y*/1.32	–0.0116	0.007	–1.67
Sin *y*/1.32	0.0232	0.007	3.30
Cos *y*/1.46	0.0327	0.007	4.65
Sin *y*/1.46	0.0014	0.007	0.20
Cos *y*/1.61	0.0017	0.007	0.24
Sin *y*/1.61	0.0140	0.007	2.01
Cos *y*/2	0.0266	0.007	3.61
Sin *y*/2	0.0272	0.007	3.78
Cos *y*/3.23	0.0074	0.007	1.07
Sin *y*/3.23	–0.0111	0.007	–2.03
Cos *y*/5	–0.0153	0.007	–2.22
Sin *y*/5	–0.0007	0.007	–0.11
Lag 1 T_{min}	–0.0082	0.002	–3.48
Lag 1 TT†	0.0219	0.006	3.66
Lag1 $RH_{A.M.}$	–0.0003	0	–0.81
Linear trend	–1.2E – 5	0	–0.40
Quadratic trend‡	–4.7E – 9	0	–0.38

*Dummy variable = 1 for 1989; 0 otherwise.
†TT = (T_{max} – 28) when T_{max} > 28 °C, and TT = 0 when $T_{max} \leq$ 28 °C.
‡Trend is (time/1 000).

Table 4.—Spearman Correlation Coefficients for Variables in Analysis

	T_{max}	RH	O_3 (8 h average)	O_3 (max 1 h)	NO_2 (24 h)	NO_2 (max 1 h)	bsp (24 h)	SO_2 (24 h)
T_{min}								
All year	.728*	.271*	.059†	.014	−.605*	−.587*	−.296*	−.081*
Summer	.523*	.421*	−.210*	−.169*	−.444*	−.461*	−.157*	.058‡
Winter	.554*	.519*	−.105†	−.142*	−.437*	−.475*	−.203*	−.133*
T_{max}								
All year		−.026	.375*	.348*	−.424*	−.362*	−.119	−.091*
Summer		.026	.281*	.341*	−.120*	−.087§	.100†	.095†
Winter		.128*	.333*	.327*	−.160*	−.120	.086§	−.049
RH								
All year			−.336*	−.249*	−.085*	−.133*	.021	−.003
Summer			−.304*	−.230*	−.186*	−.233*	−.106†	.020
Winter			−.338*	−.239*	−.093†	−.153*	.073§	−.038
O_3 (8 h)								
All year				.949*	.042‡	.115*	.224*	−.106*
Summer				.961*	.333*	.384*	.424*	.005
Winter				.932*	.036	.124*	.192*	−.182*
O_3 (max 1 h)								
All year					.146*	.213*	.325*	−.048‡
Summer					.362*	.405*	.451*	.045
Winter					.176*	.257*	.336*	−.110*
NO_2 (24 h)								
All year						.900*	.515*	.184*
Summer						.874*	.397*	.160*
Winter						.879*	.538*	.166*
NO_2 (max 1 h)								
All year							.508*	.217*
Summer							.413*	.226*
Winter							.517*	.174*
bsp (24 h)								
All year								.125*
Summer								.092§
Winter								.134*

Notes: T_{min} = minimum temperature, RH = relative humidity, and T_{max} = maximum temperature. bsp and SO_2—24-h average values; other pollutants—24-h average values and 1-h maximums.
*$p < .0001$.
†$p < .001$.
‡$p < .05$.
§$p < .01$.

The relative risk ratios that arose from these models for bsp and O_3 for the three mortality categories considered are shown in Table 6 for the three different age groups: (1) all years, (2) > 65 y, and (3) < 65 y. Clearly the positive and significant associations arose in the > 65-y category, not in the < 65-y category, although the counts in the latter category were very low (Table 1).

Discussion

Measures of particulate pollution. To compare the results for the surrogate for particulate pollution used here (bsp) with foreign results derived with PM_{10} data, we examined the relationship between bsp and PM_{10} data. We derived a regression model of $|bsp| = (.094)*|PM_{10}|$ ($R^2 = 0.76$) from the data for the 1987–1993 period (and from Rocklea PM_{10} data). In one Brisbane study, Chan[23] found that the concentration of fine particulate matter (|FPM|), or $PM_{2.5}$ (i.e., particulate matter less than 2.5 μm in aerodynamic diameter), was approximately $0.4*|PM_{10}|$, and that |bsp| was approximately $0.3*|FPM|$; therefore, |bsp| was approximately $0.12*|PM_{10}|$, which approximates the regression results in our study. In the foreign studies, investigators expressed the results as the percentage increase in mortality for each increase of 10 μg/m³ in PM_{10} levels. From this simple linear regression model we can conclude that a 10-μg/m³ increase in PM_{10} levels corresponds to an increase in total daily mortality of approximately 0.9% (Table 5). In Perth, an Australian city the same size as Brisbane, Gras derived the relationship between FPM, or $PM_{2.5}$, and nephelometry as $|FPM| = 2.0*|bsp|$.[24] The Brisbane relationship between |FPM| and $|PM_{10}|$ implies, therefore, that the linear relationship expected between bsp and PM_{10} data in Brisbane would be $|bsp| = 0.2*|PM_{10}|$ (i.e., an increase in PM_{10} levels of 10 μg/m³ corresponds to an increase in daily mortality of 1.8%). Both results are comparable with the 0.7–1.6% range (weighted average = 1.0%) found in other studies.[2]

Of course, bsp data, which are measures of light scattered by particles, are much more closely associated with $PM_{2.5}$ levels,[2,23,24] given that only particles with

Table 5.—Autoregressive Poisson Coefficients for Pollutants at Lag 0, after Controlling for Confounding Effects: Coefficient

Pollutant	Total			Cardiovascular			Respiratory		
	Coef.	*SE*	*t*	Coef.	*SE*	*t*	Coef.	*SE*	*t*
Ozone									
8-h average									
All year	.0235	.008	2.95	.0197	.014	1.36	.0380	.029	1.31
Summer	.0294	.010	3.01	.0237	.018	1.33	.0226	.039	0.59
Winter	.0129	.014	0.89	.0145	.026	0.56	.0101	.030	0.33
Maximum 1 h									
All year	.0157	.005	3.15	.0122	.009	1.35	.0127	.018	0.70
Summer	.0158	.006	2.64	.0116	.011	1.06	.0032	.024	0.13
Winter	.0157	.009	1.75	.0166	.016	1.02	.0101	.030	0.33
Nitrogen dioxide									
24-h average									
All year	−.0051	.011	−0.45	−.0154	.020	−0.78	−.0418	.039	−1.08
Summer	−.0184	.021	−0.90	−.0710	.037	−1.90	−.0733	.081	−0.91
Winter	.0059	.014	0.41	.0129	.025	0.52	−.0372	.046	−0.80
Maximum 1h									
All year	.0029	.006	0.52	−.0004	.010	0.04	.0100	.019	0.52
Summer	−.0978	.010	−0.78	−.0299	.018	−1.65	−.0071	.039	−0.18
Winter	.0107	.007	1.51	.0155	.012	1.27	.0149	.023	0.65
Sulfur dioxide									
24-h average									
All year	.0276	.028	0.99	.0101	.050	0.20	−.0166	.099	−0.17
Summer	.0275	.058	0.48	−.0461	.109	−0.42	−.0596	.232	−0.26
Winter	.0272	.034	0.79	.0279	.060	0.46	.0452	.113	0.40
bsp									
24-h average									
All year	.0088	.003	2.76	.0102	.006	1.75	.0009	.011	0.08
Summer	.0114	.006	1.97	.0102	.011	0.96	.0141	.022	0.65
Winter	.0073	.004	1.84	.0097	.007	1.39	−.0060	.013	−0.45
Maximum 1h									
All year	.0022	.001	2.42	.0042	.002	2.51	−.0004	.003	−0.10
Summer	.0031	.002	1.96	.0022	.003	0.76	.0033	.006	0.56

Table 6.—Relative Risk (RR) Ratios and 95% Confidence Intervals (95% CIs) for Autoregressive Models for Different Age Categories*

Category	Ozone				bsp			
	Max 1 h		8-h average		Max 1 h		24-h average	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Total mortality								
All years	1.016	1.006, 1.026	1.024	1.008, 1.040	1.002	1.000, 1.004	1.009	1.003, 1.015
≥ 65	1.016	1.004, 1.028	1.024	1.005, 1.042	1.002	1.000, 1.004	1.010	1.002, 1.018
< 65	1.014	0.992, 1.036	1.024	0.988, 1.061	1.001	0.997, 1.005	1.001	0.986, 1.018
Cardiovascular mortality								
All years	1.012	0.994, 1.031	1.020	0.992, 1.049	1.004	1.001, 1.008	1.010	0.998, 1.002
≥ 65	1.013	0.993, 1.034	1.019	0.987, 1.052	1.005	1.001, 1.009	1.011	0.999, 1.023
< 65	1.008	0.958, 1.059	1.027	0.950, 1.111	1.001	0.991, 1.011	1.004	0.971, 1.039
Respiratory mortality								
All years	1.013	0.977, 1.050	1.039	0.980, 1.101	0.999	0.993, 1.005	1.001	0.979, 1.023
≥ 65	1.019	0.982, 1.059	1.045	0.982, 1.112	1.001	0.993, 1.009	1.007	0.983, 1.094
< 65	0.974	0.880, 1.079	1.002	0.856, 1.174	0.987	0.967, 1.007	0.962	0.902, 1.026

*The ratios for ozone are per 10 ppb increase in ozone level and for bsp are per 1×10^{-5}/m.

aerodynamic diameters less than 2 μm scatter light efficiently. Our results show that such fine particles, which penetrate deep into the lungs, are associated closely with daily mortality; this would be expected if particulate matter were a factor associated with mortality. We conducted the analysis in which PM_{10} levels were derived from bsp data only to compare our results with foreign reviews,[2] in which investigators used PM_{10} measures as surrogates for all particulate levels.

Interaction effects. A key question when one considers associations such as those found in Tables 5 and 6 is whether the associations arising between mortality and pollutants (such as bsp or O_3) result from some other pollutant. For example, in the APHEA studies in Europe, close relationships between particulate pollution effects and SO_2 were reported. The correlation analysis in Table 3 presents little support for this hypothesis; nevertheless, we examined such interactions. The results in Table 3 also suggested that we examine possible interactions between O_3 and bsp data (i.e., separate results for these pollutants in Tables 5 and 6 may have only been one result for one pollutant, the other acting as a surrogate). The results of interaction effects for total daily mortality are shown in Table 7 (the APHEA protocol for the examination of such effects, discussed earlier, was followed); the "high" terms we considered were for 90th percentiles of the pollution data. The results in Table 7 indicate an absence of significant interaction effects. With respect to both O_3 and bsp impacts, we found no apparent interactions for either SO_2 or NO_2 data. The SO_2 levels were very low (Table 2); therefore, we would not have expected a confounding effect. Also, the interaction effects between O_3 and bsp data were not significant.

Cumulative effects. The most significant effects existed for same-day averages. Associations between bsp and O_3 for the 3-d averages (average of lags 0, 1, and 2 d) were generally significant ($p = .1$) with respect to total daily mortality. Longer time averages were less significant, except for the 7-d average of O_3 data, in which case associations were significant ($p = .05$).

Thresholds. To examine the existence of thresholds, we also examined the increase in relative risk with percentiles of pollutant data. The relative risk for bsp quintile data is shown in Table 8, and relative risk increased concomitantly with percentile value. This compares with results reported by Schwartz[25] for PM_{10} and by Schwartz and Dockery[26] for TSP data; in our analysis, a threshold effect was not indicated, even at low levels. The results of an analysis for which we used quintiles for daily maximum 1-h O_3 levels are shown in Table 9, and there was a threshold effect for the top quintile (i.e., only 20% of the data affected mortality). However, in our study, the range of values for the upper quintile for maximum O_3 was 31–102 ppb (mean = 42 ppb), indicating a threshold much lower than that traditionally used for O_3 standards (i.e., usually 60–120 ppb).

Confounders. It is also possible that a few high pollutant values may have distorted the results. This is a matter of particular interest for Brisbane because dust storms and bush fires are often associated with high bsp particulate pollution days.[27] We conducted classical Poisson regression analysis for daily BSSD mortality and 24-h average bsp data, and we deleted all days on which the bsp values exceeded 7×10^{-5}/m (i.e., approximately 3% of the data—these values would correspond to levels of 70–140 μg/m³); the results indicated the existence of even stronger associations than were obtained previously. We conducted a similar analysis for maximum 1-h O_3, and we deleted all days on which maximum O_3 levels exceeded 60 ppb (i.e., approximately 1.5% of the data); the results differed little from the previous analysis.

It is also possible that the O_3 effect is linked to maximum temperature effects, although we controlled for

Table 7.—Autoregressive Poisson Coefficients for Pollutants at Lag 0, after Controlling for Interaction Effects

Category	All year Coef.	*SE*	*t*
Total mortality			
O_3 max 1 h	.0157	.005	3.15
O_3 max 1 h + bsp (24 h)*	.0142	.006	2.49
bsp (24 h)	.0088	.003	2.76
bsp (24 h) + O_3 max 1 h†	.0078	.004	2.15
bsp (24 h) + SO_2 24 h‡	.0098	.003	2.95
bsp (24 h) + NO_2 24 h§	.0098	.004	2.50
bsp max 1 h	.0022	.001	2.42
bsp max 1 h + O_3 max 1 h†	.0019	.001	1.85
Cardiovascular mortality			
O_3 max 1 h	.0122	.009	1.35
O_3 max 1 h + bsp (24 h)*	.0117	.010	1.14
bsp (24 h)	.0102	.006	1.75
bsp (24 h) + O_3 max 1 h†	.0156	.007	2.40
bsp (24 h) + SO_2 24 h‡	.0096	.006	1.56
bsp (24 h) + NO_2 24 h§	.0131	.007	1.83
bsp max 1 h	.0041	.002	2.50
bsp max 1 h + O_3 max 1 h†	.0048	.002	2.66

*Assuming a "high" bsp value > 4.54×10^{-5}/m.
†Assuming a "high" ozone value > 39 ppb.
‡Assuming a "high" SO_2 value > 7.8 ppb.
§Assuming a "high" NO_2 value > 22 ppb.

Table 8.—Poisson General Estimating Equation (GEE) Regression Model Results for Total Brisbane Statistical Sub-District Daily Mortality and Quintiles of Same-Day 24-h Average BSP Data

Quintile bsp mean value (10^{-5}/m)	RR*	95% CI†	*t*
1.01	1		0
1.62	1.009	0.973, 1.046	0.50
2.17	1.044	1.007, 1.083	2.43
2.93	1.040	1.004, 1.079	2.16
5.20	1.048	1.009, 1.089	2.69

*RR = relative risk.
†CI = confidence interval.

Table 9.—Poisson General Estimating Equation (GEE) Regression Model Results for Total Brisbane Statistical Sub-District Daily Mortality and Quintiles of Same-Day 1-h Maximum Ozone Data

Quintile ozone mean (ppb)	RR*	95% CI†	*t*
12.7	1		0
17.5	0.986	0.955, 1.018	−0.87
21.5	0.998	0.965, 1.033	−0.10
27.2	0.991	0.958, 1.025	−0.53
42.4	1.040	1.003, 1.078	2.19

*RR = relative risk.
†CI = confidence interval.

maximum temperature in a nonlinear fashion in the analysis. Nevertheless, we ran a model and deleted the days on which maximum temperatures exceeded 32 °C (80 °F) from the analysis; the classical Poisson regression results differed little from those obtained in the previous analysis. Given that classical Poisson regression analysis yielded basically the same results as the GEE analysis, we can conclude that these results were a good indication that the associations did not arise from only a few extreme events.

Biological plausibility. The associations for O_3 and bsp persisted only for the > 65-y age group (Table 7), which dominated the total mortality data (Table 1). The positive associations also persisted for the cardiovascular category, but they were not significant; this was also the case for the respiratory category, although the low counts—especially in this latter category—would have made such tests difficult (Table 1).

This analysis cannot prove a causal mechanism for the effects of these pollutants on mortality; as well, the possibility that these factors could be a surrogate for some other influence cannot be disregarded. The protocols we used in our study, however, were similar to those used in other studies by researchers who sought to control for the effects of other possible confounders. Nevertheless, one must consider the question of biological plausibility when these results are interpreted.

With respect to particulates, the question of biological plausibility remains, although some investigators[4] maintain that plausible mechanisms have been identified, whereas others[2] are dubious. The results we have reported in our study reinforce the plausibility that fine particles (as measured by bsp) have a significant effect on mortality. Researchers[4] are more confident about viable mechanisms that explain the association between O_3 exposure and mortality. However, for *both* O_3 and particulates, one would expect the major impact to be on respiratory mortality—an expectation we could not confirm in our study. Perhaps this occurred because there were low counts. Researchers have suggested that associations with cardiovascular mortality found in other studies[2,4] resulted from misdiagnosis.[28] In our study, the associations in the cardiovascular category were positive, but not always significant, but when the associations were significant (e.g., maximum 1-h bsp), the regression coefficients were much larger than those for total mortality.

The results here, as well as results reported elsewhere,[2,4] indicate that the main impact of pollutants is on elderly people (i.e., > 65 y of age). Anderson et al.[4] suggested that the effects of pollutant exposure may be sufficient to precipitate the death of people already made vulnerable by some preexisting condition (e.g., cardiovascular disease, respiratory disease). The results of our study indicate that Australian cities (e.g., Brisbane) undergoing rapid growth need to study more carefully the impact of present and future air pollution levels on the health of the population because, even at the present levels, significant effects may be occurring. (This is particularly important for a region such as that around Brisbane, which is promoted as a good place for elderly people to retire.) However, more study needs to be done by researchers who must (a) examine biological plausibility; (b) examine the question of coherence[21] (i.e., if there are mortality effects then there should be other health impacts); and (c) measure total exposure (indoor *and* outdoor monitoring).

Conclusions

The results indicated significant associations between O_3 and particulate pollution, and total daily mortality in Brisbane. The association for particulates was similar in magnitude to that found in foreign studies. Sulfur dioxide levels did not appear to be a confounding effect; they were likely too low for this to have occurred. The particulate data did not support a threshold effect, but the O_3 effect did appear to have a threshold, albeit low (30 ppb). We did not note any interaction effects between the pollutants; the impact of O_3 and particulate pollution appeared additive. There was an absence of any confounding effects for very hot days or for "natural" events (e.g., dust storms, bush fires) that might have distorted the results. The associations were significant only for elderly people; we found positive, but generally not significant, associations for cardiovascular categories of mortality.

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The authors acknowledge the support of grants from the Australian Research Council (C59530113 2) in funding part of this project. For the provision of data, the authors thank the staff of the following Queensland State Government Departments: Government State Statistician's Office, Health, Environment. The provision of data from the Federal Government Bureau of Meteorology is also acknowledged.

Submitted for publication October 11, 1996; accepted for publication May 25, 1997.

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