Ambient Air Pollution and Daily Mortality in Taipei, Taiwan and Hong Kong, China

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Abstract. The relationship between ambient air quality and daily mortality for the period 1997-1998 was examined in Taipei, Taiwan and Hong Kong, China. The observed yearly concentrations of respirable particulate matter (PM_{10}) and sulfur dioxide (SO_2) in each city were below the World Health Organization's 1999 air quality guidelines, though yearly nitrogen dioxide (NO_2) levels in both cities exceeded the WHO's recommendations. Daily death counts—of total mortality, mortality of persons ≥65, mortality due to cardiovascular disease, mortality due to respiratory disease, mortality due to pneumonia, and mortality due to chronic obstructive pulmonary disease (COPD)—were analyzed separately in the two cities by Poisson regressions for the overall two-year period as well as by season. Mortality rate ratios were calculated by identifying the 10% of days in the two-year time period with the highest and lowest concentrations of each air pollutant, and comparing the mortality rates on those days. The analyses demonstrated an overall association between air pollutant levels and mortality in both Taipei and Hong Kong, though the association was generally stronger in Taipei. The study suggests that there may be increased mortality associated with each city's current air pollution levels.

I. Introduction

There is much anecdotal evidence of an association between high levels of ambient air pollution and adverse health effects. In the late 20^{th} century, episodes of extremely high air pollution (e.g. the London smog disaster of 1952 and similar incidents in Donora, Pennsylvania in 1948 and the Meuse Valley, Belgium in 1930), have been strongly correlated with heightened morbidity and mortality. More recently, physiological and toxicological studies have attempted to more quantitatively study the biological responses to air pollution through dose-response tests (Avol et al, 1987; Ostro et al, 1991). Such studies have demonstrated changes in pulmonary function, mean forced expiratory volume over one second (FEV_1), and other physiological responses in human subjects exposed to varying levels of single air pollutants as well as air pollutant mixtures. However, the relatively high doses generally required to induce a measurable response limit the applicability of these results in assessing long-term exposure hazards to air pollution.

More recently, time-series studies have been used with increasing frequency to measure the association between air quality and health at ambient levels. These studies correlate daily counts of an adverse health effect—deaths or admissions to hospitals—to daily average concentrations of pollutants, while accounting for as many potentially confounding factors as possible. In addition to allowing for large sample sizes, these analyses are also unlikely to be confounded by factors such as smoking habits or socioeconomic status (Schwartz, 1994) which are not likely to vary on a day to day basis. Because the "classical compounds"—particulate matter, sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), carbon monoxide (CO), and lead—are regularly measured around the world, most time-series studies have focused upon these pollutants. Of these, particulate matter, measured in either Total Suspended Particulate Matter (TSPM) or PM_{10} (inhalable particles, of which 50% are 10 μg or less), SO_2, and NO_2 have been the focus of the most studies.

Because of the difficulties involved in measuring morbidity, the majority of studies have chosen to use mortality. Studies in Detroit (Schwartz, 1991), Philadelphia (Schwartz and Dockery, 1992), Santa Clara, California (Fairley, 1990), Cincinnati, Ohio (Schwartz, 1994) and Utah Valley (Pope et al, 1992) have shown strong associations between daily mortality and daily particulate concentration, whether measured in TSPM or PM_{10}. In Steubenville, Ohio (Schwartz and Dockery, 1992) a similar relationship was shown, but here, the relationship of TSPM and mortality was independent of SO_2 but the association between mortality and SO_2 was not independent of TSPM. Kinney and Ozkaynak (1991) have similarly shown that mortality in Los Angeles was strongly correlated with NO_2 as well as TSPM, but they also showed that the levels of the pollutants were strongly associated with each other.

Such time-series studies have demonstrated increased morbidity and mortality associated with all three pollutants. From these studies, it is also clear that the correlation is strongest in the most susceptible populations—the elderly and those with pre-existing respiratory problems. Some studies (Kinney and Ozkaynak, 1991) have included persons with cardiovascular disease in these susceptible populations. Unfortunately, these studies have also shown that it is difficult to attribute a specific health effect to a single pollutant.

Based on the combined evidence from such epidemiological and toxicological studies, in 1987, the World Health Organization (WHO) first published its Air Quality Guidelines for Europe to serve as standards for the protection of public health. The guidelines specified 1-hour, 24-hour, and yearly standards for exposure to the "classical compounds." However, despite the title of the guidelines, these standards were promulgated throughout much of the world.

The application of these guidelines to areas outside of Europe was a cause for concern. The studies used for the 1987 WHO guidelines originated almost exclusively from North America and Europe. However, the type and speed of industrialization in many parts of the world is quite different from that which occurred in North America and Europe. This may not fully consider how types and magnitudes of exposures to air pollution, as well as socioeconomic conditions, differ around the world. Perhaps most importantly, these guidelines do not account for differences in previous levels of exposure. Mage (1996), for example, has developed a hypothetical relationship between development and air quality. The relationship can be thought of as a curve with time on the x-axis and air pollution on the y-axis. Before rapid industrial development takes place, air pollution is mainly from domestic sources and light industry. Concentrations of pollutants are low and increase slowly as population growth occurs. During development, both industrial and per capita energy consumption increase. Urban air pollution becomes a serious public health concern and eventually the situation warrants government intervention. Immediate improvement is extremely rare; generally the situation is stabilized, and eventually levels of pollutants begin to decrease. However, a more rapid pace of industrialization will make this curve shorter but steeper. What effects this change may have on human health has yet to be determined. It is for this reason that the 1987 WHO guidelines were devised specifically for Europe.

To remedy this problem, the WHO assembled a task force in December 1997 in order to come up with globally applicable air quality guidelines. The 1999 WHO guidelines recognize that results from studies in one part of the world may not be easily transferable to another. In devising its guidelines for ambient and indoor air quality, the WHO sought to help countries devise their own air quality standards by "provid[ing] a basis for protecting public health from adverse effects of air pollution and for eliminating, or reducing to a minimum, those contaminants that are known to be, or likely to be, hazardous to human health and well being" (WHO, 1999).

But while the 1999 WHO guidelines are more flexible than those from 1987, they also highlight the fact that, to date, few studies have looked at the effects of ambient air pollution in cities in the developing world. In part, this can be attributed to the lack of an infrastructure for collection of health information as well as routine air quality data. Those studies that have looked at air pollution in Santiago, Chile (Salinas, 1995), Rio de Janeiro (Penna and Duchiade, 1991), Seoul (Lee et al, 1999), and Beijing (Xu, 1991) have all reported positive associations between elevated air pollution levels and poor health, including increased mortality. However, it is clear that more studies are needed to determine the relationship between air quality and adverse health effects in areas of the world outside of Europe and North America.

This study aims to examine the relationship between air pollution and excess mortality in two major cities located in regions considered “economies in transition”: Taipei and Hong Kong. Both cities have achieved phenomenal economic growth in an extremely short time. However, accompanying this growth have been dramatic increases in air pollution levels, especially in the urban centers. The magnitude of the problem is such that both cities have recently identified air quality as their most important public health concern. This project therefore undertook a detailed investigation into the relationship between mortality and air pollution in Hong Kong and Taipei. While data on hospital visits or some other measure of morbidity would ideally give a better sense of the overall relationship between air quality and health, such measures are very often culturally defined. For this reason, mortality was used as the health measure. Due to the large body of studies that have established an association between mortality and SO_2, NO_2, and particulate matter, these three pollutants were chosen for analysis. While these cities have recently achieved large reductions in the average yearly levels of these pollutants, there are large daily variations. What effects these episodes of increased air pollution have on mortality have yet to be determined.

II. Methods

Study sites. Taipei is located 27km inland from the northern point of Taiwan. In 1999, Taipei had a population of approximately 2.6 million in an area of 270 km^2. The region experiences a subtropical climate with an average temperature of 16° C in the winter and 28° in the summer. The city is located in a mountain basin and therefore temperature inversions may occur during any season. The average monthly relative humidity ranges from 71-90%.

Hong Kong lies to the southeast of the mainland of China. In 1998, Hong Kong had a population of approximately 6.7 million in an area of 1080 km^2. The city experiences a subtropical climate with an average temperature of 18° C in the winter and 29° C in the summer. The average monthly relative humidity ranges from 69-83%.

Data. Daily deaths in Taipei for the calendar years 1997 to 1998 were extracted from mortality records of the Taiwanese Department of Health's Office of Statistics. Daily mortality data for Hong Kong for the same period were obtained from the Demographic Statistics Section of Hong Kong's Census and Statistics Department. Each mortality record was coded according to age, sex, and cause of death according to the International Classification of Diseases, Injuries, and Causes of Deaths, 9^{th} revision (ICD-9). Deaths due to violent or accidental causes (ICD-9 codes 800-999) were excluded from the study. Daily counts of total mortality and mortality in persons ≥65 were tabulated. Total mortality was also subdivided by cause of death, with an attempt made to balance specificity and sufficient cases to allow analysis. The causes analyzed were cardiovascular disease (ICD-9 codes 390-459), respiratory disease (ICD-9 codes

460-519), pneumonia and influenza (ICD-9 codes 480-487), and chronic obstructive pulmonary disease (COPD) and allied conditions (ICD-9 codes 490-496).

The air quality data for Taipei were obtained from the database of Taiwan's Environmental Protection Agency. Data from the seven ambient air quality monitoring stations (Shi Lin, Zhong San, Song San, Wan Hua, Gu Ting, Da Tung, and Yang Ming) located within Taipei City were averaged to provide a citywide arithmetic mean. Citywide means were available for all but one day of 1997 and 1998 for PM_{10}, and for all days for SO_2 and NO_2. The air quality data for Hong Kong were obtained from the Hong Kong Environmental Protection Department. In 1997 the monitoring network consisted of nine ambient air quality monitoring stations (Central Western, Kwai Chung, Kwun Tong, Sham Shui Po, Sha Tin, Tai Po, Tsuen Wan, and Yuen Long) and one roadside air quality monitoring station (Mong Kok). In 1998 the monitoring network was expanded to include one additional ambient air quality monitoring station (Tap Mun) and two additional roadside monitoring stations (Central and Causeway Bay). Citywide means were calculated as the arithmetic mean of data from all available stations. Data was available for all three pollutants for the entire two-year period.

Statistical Analysis. Data analysis was carried out using STATA statistical software (STATA, 1996). The analysis sought to measure the association between the three air quality indicators and the daily mortality data in each city over the 1997 to 1998 time period. Daily data on total mortality, mortality of persons ≥65, and mortality due to cardiovascular disease were matched to the respective city's 24-hour (midnight to midnight) mean samples of PM_{10}, SO_2, and NO_2. Based on previous studies (Pope, 1992; Schwartz, 1994) that concluded that mortality due to respiratory disease was likely to be lagged, daily data on mortality due to respiratory disease, mortality due to pneumonia, and mortality due to COPD were matched to the city's mean samples of PM_{10}, SO_2, and NO_2 for the previous 24-hour period.

Daily mortality is a classic example of a rare event and was therefore first modeled using Poisson regressions over the combined 2-year period. In an effort to control for confounding seasonal variations in both climate and, the data were then divided into March 21-June 20, June 21-September 21, September 22-December 20, and December 21-March 20 time periods for both 1997 and 1998, and modeled by Poisson regressions.

Finally, mortality rate ratios were calculated for each city to compare mortality rates on days with high and low concentrations of each air pollutant. For each city, data for the combined two-year period were sorted by each pollutant, and the 10% of days with the highest and lowest levels of each pollutant were identified. Using mortality as the measure of incidence, the incidence rate ratios for total mortality, mortality of persons ≥65, mortality due to cardiovascular disease, and mortality due to respiratory disease were calculated for days with high concentrations of the pollutant compared with days with low concentrations of the pollutant. Total mortality, mortality of persons ≥65, and mortality due to cardiovascular disease were matched with the same-day citywide mean of the pollutant, while mortality due to respiratory disease was matched with the previous day's citywide mean for the pollutant. Counts of pneumonia and COPD were insufficient to carry out a mortality rate ratio analysis.

III. Results

Overview. Table 1 presents the mean and percentile distributions of the pollutant and major mortality variables in this study. An examination of the pollutant data indicates that the mean NO_2 levels in both cities exceed the WHO guidelines of 40 $\mu g/m^3$, but are within the recommended guidelines for SO_2 (50 μg/m3). Because no epidemiological studies have yet demonstrated a threshold for particulate concentrations, the WHO does not currently have guidelines for PM_{10} levels. The Hong Kong yearly standards are 80 $\mu g/m^3$ for SO_2, 80 $\mu g/m^3$ for NO_2, and 55 $\mu g/m^3$ for PM_{10}. Air quality in Taipei is measured by a PSI (Pollution Standard Index) generated from measurements of PM_{10}, SO_2, NO_2, ozone (O_3), and carbon monoxide (CO). The average yearly PSI standard is 100, but there is no specific standard for each individual pollutant. The difference in mortality figures between the two cities is generally proportional to their populations. The resulting higher number of mortality counts in Hong Kong within each category resulted in less variability in the Hong Kong data than the Taipei data when the analyses were carried out. It can also be seen that deaths due to respiratory disease constitute a larger proportion of total mortality in Hong Kong than in Taipei.

Table 1: Descriptive statistics of mortality counts and air pollution in Taipei and Hong Kong, 1997-1998

	Taipei						**Hong Kong**					
	Percentile						*Percentile*					
	10	25	50	75	90	Mean	10	25	50	75	90	Mean
Pollutant variable												
PM_{10} ($\mu g/m^3$)	22	30	39	52	72	43	29	35	49	68	87	54
SO_2 ($\mu g/m^3$)	6	8	12	17	22	13	6	8	13	19	28	16
NO_2 ($\mu g/m^3$)	43	53	66	81	96	68	36	47	61	73	88	61
Mortality variable												
Total mortality (counts)	23	26	30	35	39	31	68	74	82	90	98	82
Mortality of those ≥65	15	18	21	25	28	22	50	56	62	69	76	63

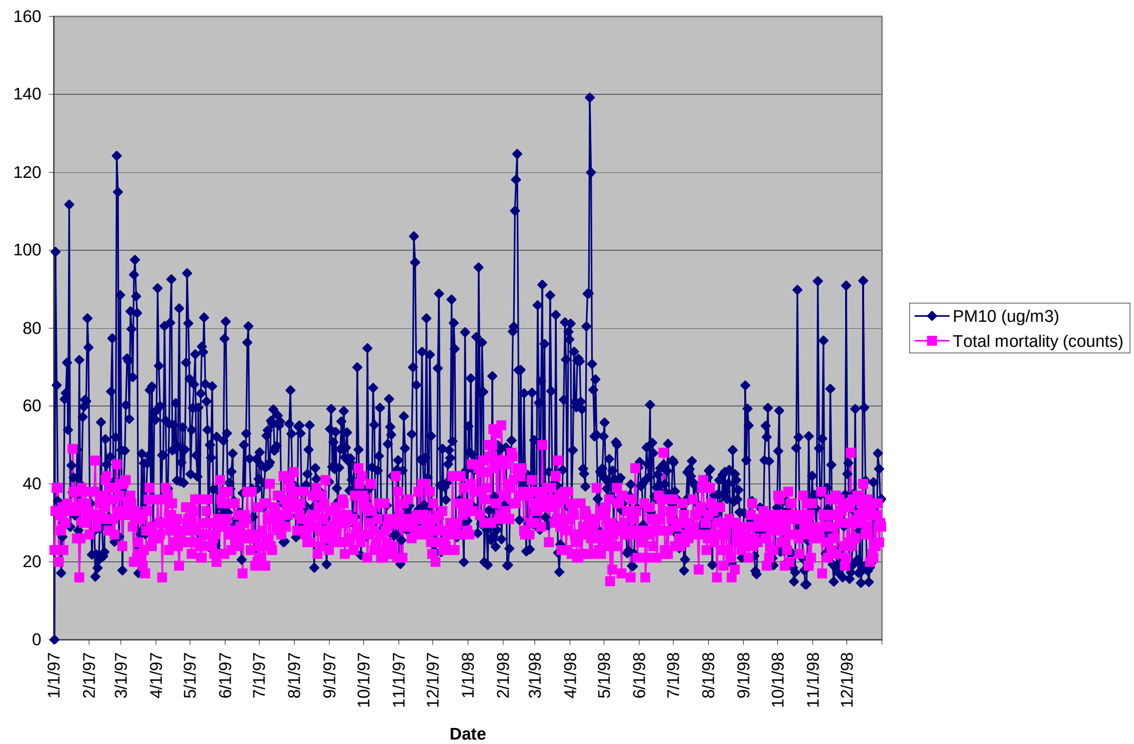
(counts)												
Mortality due to cardio-vascular disease (counts)	5	6	8	10	13	9	15	19	22	27	31	23
Mortality due to respiratory disease (counts)	1	1	2	3	5	3	11	14	17	20	23	17

PM_{10}, SO_2, and NO_2 covaried significantly ($p<0.0005$) in both cities over both the two-year period and the seasonal analyses. Adjusted R^2 values for this covariance ranged from 0.28 to 0.68, and generally were more variable in the Hong Kong data. In addition, there was a high level of variability for all variables. The highest level of variability was seen in the air quality indicators, which show a weekly periodicity. Levels are highest midweek, and lowest on the weekends.

Taipei. The Poisson regression analysis showed a significant association between PM_{10} levels and counts of total mortality (β=0.00077 ± .0013, p=0.023), mortality of persons ≥65 (β=0.0010324 ± 0.00079, p=0.01), and mortality due to cardiovascular disease (β=0.0013 ± 0.00125, p<0.0005) in Taipei during 1997-1998. Total mortality (β=0.00078 ± 0.00066, p=0.015), mortality of persons ≥65 (β=0.00103 ± 0.00075, p<0.0005), mortality due to respiratory disease (β=0.0062 ± 0.0022, p<0.0005), mortality due to pneumonia (β=0.0056 ± 0.0034, p=0.003), and mortality due to COPD (β=0.0061 ± 0.0032, p=0.003) were significantly associated with NO_2 levels in Taipei during the same two-year time period.

The relationship between individual pollutant levels and mortality in Taipei is more complicated when examined on a seasonal basis. The results of the seasonal Poisson regression analyses are shown in Appendix A. Significant associations between pollutant levels and air quality were seen in the spring and fall, and less commonly in the winter and summer. Of these associations, the relationship between mortality due to respiratory diseases (and COPD and pneumonia when analyzed separately) and NO_2 was the most consistently seen, as well as associations between mortality due to respiratory diseases and PM_{10} and SO_2. It is difficult to determine what is driving the associations between pollutant levels and mortality. In Figure 1, the strong seasonal signals in both mortality and PM_{10} levels (which covaried significantly with SO_2 and NO_2) in Taipei over the two-year time period are shown.

Figure 1: Total mortality and PM_{10} levels, Taipei 1997-1998

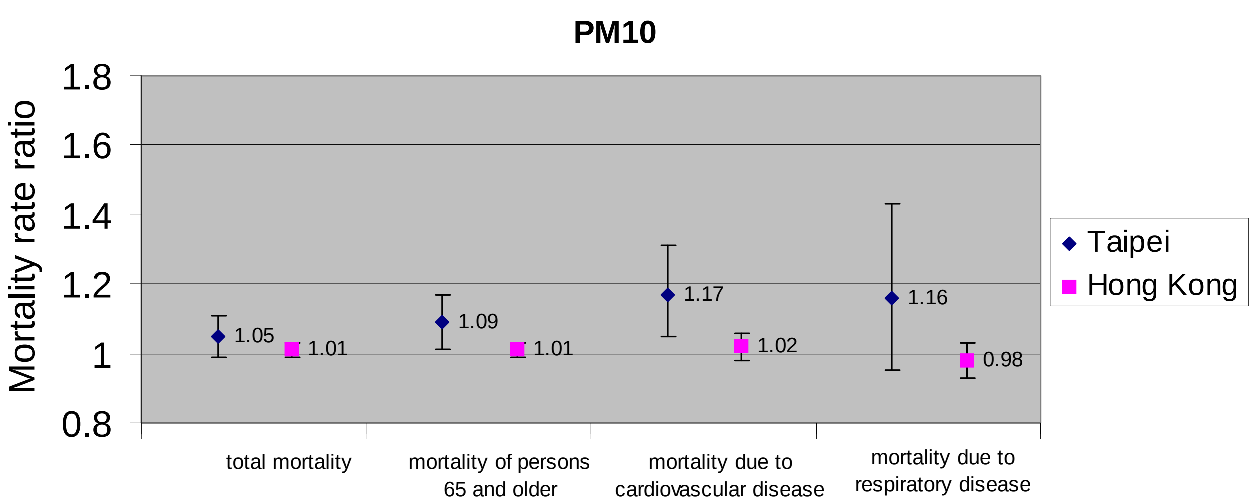


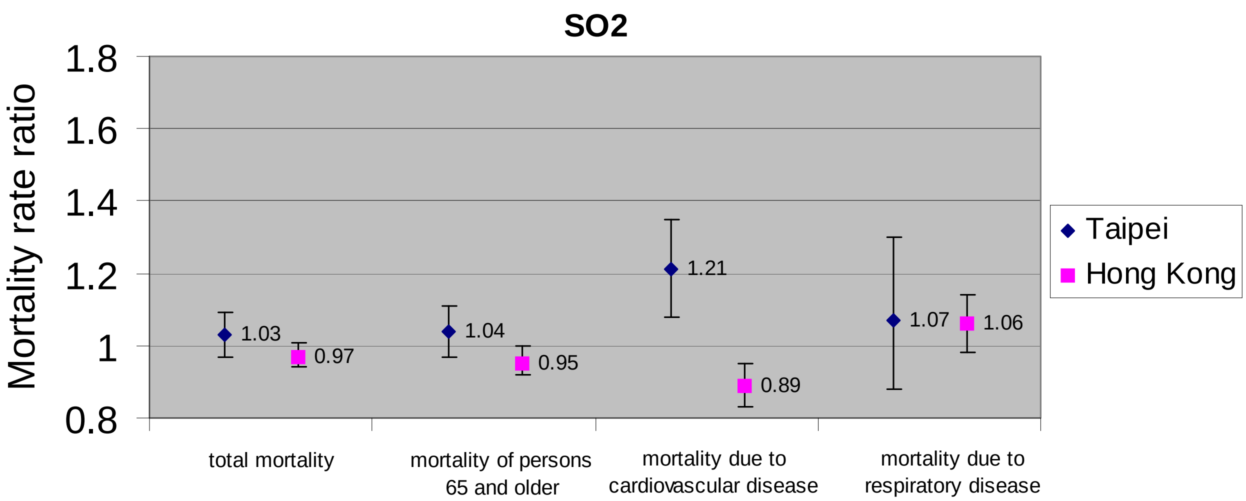
It is possible that a combination of climatic variables is confounding the relationship between pollutant levels and mortality. This may explain the more consistent associations during the spring and fall seasons when climatic conditions are less extreme. However, the results of the seasonal analyses may also have been influenced by the decreased number of samples (days) in each analysis. With a lower number of samples, the variability necessarily increases, which may be reflected in the p-values seen.

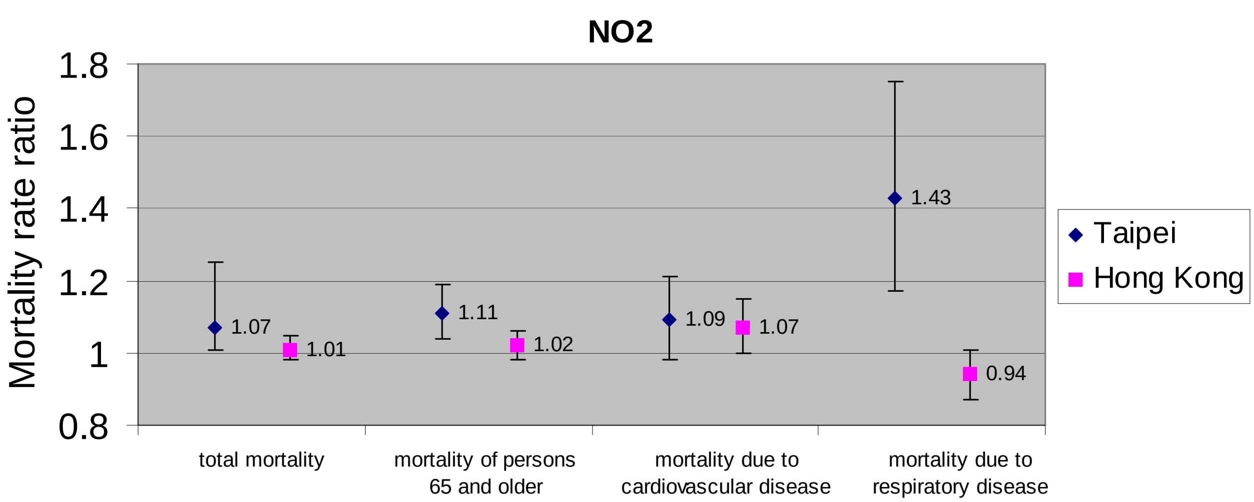
In an attempt to control for the amount of variability due to the smaller seasonal sample size, the data for each season over the two-year period was coupled. While such an analysis benefited from the larger sample size, how it affects differences in other factors, such as infectious disease outbreaks, that may vary between years is not clear. The results are shown in Appendix B. Again, the seasonal relationships between air quality and mortality are difficult to determine. Far more relationships are statistically significant, but in the summer and fall, many of these relationships are negative. However, it can be seen that there is a strong and consistent association between winter mortality and NO_2 that was not evident before.

The mortality rate ratios (MRR) for Taipei 1997-1998 are shown in Figure 2.

Figure 2: Mortality rate ratios for Hong Kong and Taipei, 1997-1998, by pollutant







It can be seen that in all categories the rate ratio is greater than 1 (ranging from 1.03—total mortality and SO_2—to 1.43—mortality due to respiratory disease and NO_2), indicating that the mortality rates on days with high levels of air pollutants are always higher on days when there are low levels of air pollutants. The MRR is consistently the largest for mortality due to respiratory disease. Like the 2-year Poisson regression, the strongest association between pollutant level and mortality was seen for PM_{10} and NO_2. An examination of the data used in the mortality rate ratios also indicated the influence of seasonality may have decreased, as all seasons were represented in both the high and low classes of each pollutant. In addition, despite the strong overall covariance between the three pollutants, the dates when the high and low 10% concentrations occurred for each pollutant did not significantly overlap. Only 13 of the possible 73 days (18%) were in the lowest 10% for all pollutants; only 11 of the possible 73 days (15%) were in the highest 10% for all pollutants.

Hong Kong. The Poisson regression analysis showed a significant association between PM_{10} levels and counts of total mortality (β=0.00394 ± 0.00337, p=0.022), mortality of persons older 65 (β=0.000386 ± 0.00039, p=0.05) and cardiovascular mortality (β=0.0007421 ± 0.00063, p=0.022) in Hong Kong over the 2-year period analyzed. Total mortality (β=0.0006392 ± 0.0004, p=0.002), mortality of persons older than 65 (β=0.007044 ±0.00046, p=0.003), and mortality due to cardiovascular disease (β=.00106 ± 0.00076, p=0.006) were also all significantly associated with NO_2 over the same two-year period. Mortality due to respiratory disease (β=0.00177 ± 0.00152, p=0.022) and mortality due to pneumonia (β=0.00314 ± 0.00189, p=0.001) were significantly associated with levels of SO_2 over this 2-year period in Hong Kong.

The relationship between air pollutant levels and mortality becomes more complicated when analyzed on a seasonal basis. The results of the seasonal Poisson regressions is shown in Appendix C. Significant associations between mortality categories and pollutant variables were seen mostly during the spring and the fall, and only rarely during the summer and winter. However, during these seasons there was no consistent pattern of association. Of these, SO_2 was most significantly associated with all categories of mortality type, in contrast with the results from the two-year data analysis. . This may indicate seasonal confounding by other factors such as climate. The seasonal analyses may be affected by the smaller sample sizes involved in each analysis, which may have increased the variability.

As with Taipei, the combined seasonal analysis shown in Appendix D does not show any consistent pattern of association. In particular, the associations seen in the summer are in fact negative. Whether or not this is due to seasonal confounding is unclear. It is interesting to note that the strong winter association seen in Taipei is not reflected in the Hong Kong data. However, the results do show a strong association between PM_{10} levels and non-respiratory mortality in the spring and fall, supporting the results of the Poisson analysis.

The results of the mortality rate ratios (MRR) for Hong Kong are shown in Figure 2. The majority of the MRRs for Hong Kongi are greater than 1, but range from 0.89 (mortality due to cardiovascular disease and SO_2) to 1.07 (mortality due to cardiovascular disease and NO_2). This indicates that for the most part, the mortality rate on days with high levels of air pollutants was higher than for days with low levels; this relationship is most consistently seen in PM_{10} and NO_2 levels, as the 2-year Poisson regression analysis found. An examination of the data indicates that the effects of seasonality may have been decreased in this analysis, since all seasons were represented in both the high and low class of each pollutant. The decision to analyze mortality rate ratios separately for each pollutant was based on the lack of overlap of days with the highest and lowest 10% concentration of each pollutant. Only 10 of a possible 73 days (14%) of days were in the lowest 10% of days for all pollutants; only 20 of a possible 73 days (27%) of days were in the highest 10% of days for all pollutants.

IV. Discussion

This study measured an association between mean daily values of air pollutants and mortality counts in both Taipei and Hong Kong over the combined 1997-1998 time period. In Taipei, total mortality, mortality due to cardiovascular disease, and mortality of persons 65 and older were significantly associated with PM10, while all mortality categories except for cardiovascular disease were significantly associated with NO_2. In Hong Kong, total mortality, mortality of persons 65 and older, and mortality due to cardiovascular disease were significantly associated with both PM_{10} and NO_2. Mortality due to respiratory disease and mortality due to pneumonia were significantly associated with SO_2 levels in Hong Kong. This is consistent with time-series analyses of air quality and health done elsewhere (Schwartz, 1994; Schwartz and Dockery, 1994).

The association between high levels of air pollutant and excess mortality may be more clearly seen in the mortality rate ratio analysis. The analysis showed that in both cities, high levels of pollutants could be significantly associated with a higher daily mortality. The stronger association between air quality and mortality in Taipei may be a result of its much higher levels of NO_2, or it may be due to its geographic location in a mountain basin, which allows for the formation of temperature inversions. This phenomenon was first seen in Los Angeles, and is now commonly seen in other large cities located in mountain basins. It may be that the geographic features of Taipei make the dispersion of air pollutants difficult. If this is true, then it may be that it is wind speed and not temperature that is confounding the relationship between air quality and mortality.

However, interpretation of the results in this study must be done with caution. The study suffered from an inability to account for climatic variations outside of the seasonal subdivisions that may confound the relationship between individual pollutants and mortality counts. The seasonal analysis revealed that in both cities, the strongest associations between air pollutants and mortality counts occurred in the spring and in the fall, and less frequently in the winter. It seems probable that a combination of climatic factors is driving the mortality data during months with more extreme environmental conditions. Though both cities are located in subtropical climates, there is still a seasonal signal that can be seen in the data.

Combining the seasonal data does not demonstrate a more consistent relationship, however, although there are more significant associations. It seems likely that the smaller sample size is strongly affecting the seasonal analysis. But without knowing what the potential confounders of the relationship between air quality and human health might be, there is an inherent danger in combining the seasonal data. The seasonal analysis was a first attempt at reducing intra-annual variability. Combining the same season of different years, however, may in fact increase potential confounding factors based on inter-annual variability.

Additionally, there may have been issues related to the extensiveness of the air quality monitoring networks in the cities. While the Hong Kong network is very representative of residential areas, several districts in Taipei City are not covered by the monitoring network; when based on population, only 49% of Taipei residents live in districts with monitoring stations. However, computing a weighted average for pollutant levels based on population district did not significantly affect the results. Because any missing data then highly influenced the weight of the existing data, such a method was dropped in favor of an arithmetic mean. Obviously, extending the current air monitoring network to be truly representative is crucial to more accurately gauging the relationship between air quality and human health.

A more detailed model that could account for more of the climatic variations in each city would therefore make the associations measured in this study more robust. In addition, adding more years to the study would have the benefit of increasing the sample size and decreasing the variation. It would also be preferable to use a measure of morbidity rather than mortality. Many recent studies have demonstrated an association between the exacerbation of health effects and high pollutant levels, such as with ozone and asthma (Kehrl et al, 1999). Inclusion of a greater number of air pollutants would also benefit the study by accounting for more elements in real pollutant mixtures. However, the combination of the significant associations seen in the two-year period as well as the mortality risk ratios indicate that it is likely that there is an association between air quality and mortality in both Taipei and Hong Kong. Determining the specific nature of this relationship would be of benefit in making public health decisions, and it is expected that this project will be continued towards that end in the respective cities.

It is to be hoped that similar studies will be done in other parts of the world to more fully understand how the relationship between air quality and human health may vary. Epidemiological studies from diverse environments are critical as they provide information on responses of diverse populations to ambient pollutants and pollutant mixtures (Brunekreef et al, 1995). Now that routine air monitoring of the "classical compounds" is becoming standard in all major cities of the world, it may be possible to determine what the long term health effects of exposure to different levels of air pollution are.

However, it is important to interpret these results with caution. W.S. Robinson coined the term "ecological fallacy" (Robinson, 1950) to describe the problems that may arise from assuming that the results of an ecological study—that is, a study based on populations rather than individuals—apply to the individuals within the populations studied. Because the relationship between the variables being studied may break down at the individual level, many authors (Piantadosi et al, 1988; Brenner et al, 1992) have argued that the individual-level rate ratios from ecological studies should be interpreted with great caution. While this is true, it is also important to recognize that there are situations in which it may be impossible to conduct large-scale studies of individuals and exposure, especially when one of the variables in the study is mortality (Schwartz, 1994). For all of these reasons, time-series analyses can still be extremely beneficial. The potential public health impact of air pollution necessitates a precautionary standpoint.

Acknowledgements

I would like to gratefully acknowledge the following people for their contributions to this thesis: Paul Beggs, who started me on this project and allowed me to find my own way with it; Steve Hamburg, who provided immeasurable support and guidance over the course of this project and this degree; Peter Simon, who offered much insight into the public health aspects of my work; Robert Vanderslice, who provided many helpful comments and revisions; and Joe Hogan, who helped enormously with the statistical methods and interpretation. Many thanks also go to Li-Ling Yu, Philip Ho, and Billy Li for making the data available to me.

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Appendix A: Results of seasonal Poisson regressions for Taipei, 1997-1998

	PM_{10}	SO_2	NO_2
Winter 1997			
Total mortality	β=0.0005, p=0.490	β=0.0012, p=0.654	β=9.3 x 10^{-5}, p=0.945
Mortality of persons ≥65	β=0.0012, p=0.202	β=0.0037, p=0.256	β=0.0012, p=0.433
Mortality due to cardiovascular disease	β= -0.0005, p=0.754	β= -0.0035, p=0.501	β= -0.0018, p=0.478
Mortality due to respiratory disease	β= -0.0007, p=0.812	β= -0.0057, p=0.575	β= -0.0058, p=0.250
Mortality due to pneumonia	β=0.0025, p=0.578	β= -0.0037, p=0.827	β= -0.0079, p=0.359
Mortality due to COPD	β= -0.0031, p=0.518	β= -0.0158, p=0.359	β= -0.0038, p=0.641
Spring 1997			
Total mortality	β=0.0001, p=0.898	β=0.0001, p=0.964	β= -0.0018, p=0.269
Mortality of persons ≥65	β=0.0007, p=0.596	β= -0.0001, p=0.966	β= -0.0024, p=0.216
Mortality due to cardiovascular disease	β=0.0002, p=0.910	β=0.0030, p=0.558	β=0.0030, p=0.334
Mortality due to respiratory disease	β=0.0046, p=0.240	β=0.0072, p=0.471	β= -0.0024, p=0.693
Mortality due to pneumonia	**β=0.0190, p=0.005**	β=0.0072, p=0.471	β= -0.0024, p=0.693
Mortality due to COPD	β= -0.0128, p=0.052	β= -0.0325, p=0.052	**β= -0.0334, p=0.001**
Summer 1997			
Total mortality	β=0.0013, p=0.440	β=0.0009, p=0.783	β= -0.0020, p=0.119
Mortality of persons ≥65	β= -0.0010, p=0.594	β= -0.0004, p=0.927	**β= -0.0036, p=0.017**
Mortality due to cardiovascular disease	β=0.0006, p=0.847	β=0.0053, p=0.409	β= -0.0013, p=0.598
Mortality due to respiratory disease	β=0.0026, p=0.711	β=0.0105, p=0.460	β= -0.0005, p=0.921
Mortality due to pneumonia	β= -0.0090, p=0.402	β=0.0163, p=0.455	β=0.0020, p=0.804
Mortality due to COPD	β= -0.0039, p=0.709	β=0.0114, p=0.592	β= -0.0056, p=0.466
Fall 1997			
Total mortality	β=0.0003, p=0.793	β= -0.0002, p=0.954	β= -0.0004, p=0.817
Mortality of persons ≥65	β=0.0005, p=0.707	β= -0.0004, p=0.913	β=0.0005, p=0.801
Mortality due to cardiovascular disease	β=0.0011, p=0.549	β=0.0001, p=0.976	β= -0.0003, p=0.908
Mortality due to respiratory disease	β=0.0003, p=0.952	β=0.0053, p=0.605	β=0.0021, p=0.729
Mortality due to pneumonia	β= -0.0079, p=0.308	β= -0.0008, p=0.964	β= -0.0036, p=0.738
Mortality due to COPD	β= -0.0006, p=0.926	β=0.0010, p=0.946	β=0.00352, p=0.688
Winter 1997-1998			
Total mortality	β=0.0008, p=0.306	β=0.0034, p=0.214	β=0.0004, p=0.649
Mortality of persons ≥65	β=0.0004, p=0.676	β=0.0026, p=0.414	β=0.0004, p=0.704
Mortality due to cardiovascular disease	β=0.0018, p=0.182	β=0.0099, p=0.042	β=0.0017, p=0.260
Mortality due to respiratory disease	**β=0.0046, p=0.034**	**β=0.0157, p=0.046**	**β=0.0060, p=0.015**
Mortality due to pneumonia	β=0.0042, p=0.256	β=0.0095, p=0.494	β=0.0017, p=0.697
Mortality due to COPD	β=0.0043, p=0.185	β=0.0192, p=0.097	β=0.0067, p=0.067
Spring 1998			
Total mortality	β= -0.0009, p=0.334	β=0.0012, p=0.743	β=0.0018, p=0.126
Mortality of persons ≥65	β= -0.0004, p=0.716	β= -0.0006, p=0.894	β=0.0012, p=0.362
Mortality due to cardiovascular disease	β= -0.0027, p=0.146	β= -0.0007, p=0.927	β= -0.0009, p=0.688
Mortality due to respiratory disease	β= -0.0040, p=0.237	β=0.0083, p=0.514	β=0.0030, p=0.443
Mortality due to pneumonia	β= -0.0099, p=0.105	β=0.0107, p=0.611	β=2.3 x 10^{-5}, p=0.997
Mortality due to COPD	β= -0.0011, p=0.823	β=0.0166, p=0.393	β=0.0032, p=0.596
Summer 1998			
Total mortality	β=0.0014, p=0.502	β= -0.0010, p=0.834	β= -0.0011, p=0.309
Mortality of persons ≥65	β=0.0020, p=0.434	β= -0.0021, p=0.702	β= -0.0010, p=0.415
Mortality due to cardiovascular disease	β=0.0047, p=0.267	β= -0.0088, p=0.343	β= -0.0011, p=0.608
Mortality due to respiratory disease	β= -0.0038, p=0.600	β= -0.017, p=0.277	β= -0.0032, p=0.364
Mortality due to pneumonia	β=0.0050, p=0.694	β=0.0091, p=0.748	β=0.0002, p=0.973
Mortality due to COPD	β= -0.0007, p=0.952	β= -0.0384, p=0.100	**β=0.0003, p=0.048**

Fall 1998			
Total mortality	β= -0.0008, p=0.472	β=0.0018, p=0.759	β=0.0014, p=0.253
Mortality of persons ≥65	β= -0.0002, p=0.856	β=0.0038, p=0.574	β=0.0020, p=0.185
Mortality due to cardiovascular disease	β= -0.0025, p=0.250	β=0.0158, p=0.153	**β=0.0054, p=0.031**
Mortality due to respiratory disease	β=0.0032, p=0.387	β=0.0052, p=0.803	β= -0.0009, p=0.851
Mortality due to pneumonia	β= -0.0100, p=0.243	β= -0.0414, p=0.352	β= -0.0025, p=0.777
Mortality due to COPD	β=0.0094, p=0.054	β=0.0186, p=0.525	β=0.0013, p=0.845

Appendix B: Results of combined seasonal Poisson regressions for Taipei, 1997-1998

	PM_{10}	SO_2	NO_2

Winter			
Total mortality	β=0.0005, p=0.314	β=0.0022, p=0.268	**β=0.0022, p<0.0005**
Mortality of persons ≥65	β=0.0006, p=0.357	β=0.0029, p=0.199	**β=0.0032, p<0.0005**
Mortality due to cardiovascular disease	β=0.0006, p=0.555	β=0.0033, p=0.353	**β=0.0031, p=0.002**
Mortality due to respiratory disease	β=0.0022, p=0.208	β=0.0068, p=0.276	**β=0.0087, p<0.0005**
Mortality due to pneumonia	β=0.0032, p=0.271	β=0.0086, p=0.737	**β=0.0059, p=0.048**
Mortality due to COPD	β=0.0013, p=0.630	β=0.0067, p=0.486	**β=0.0114, p<0.0005**
Spring			
Total mortality	β= -0.0005, p=0.515	β=0.0005, p=0.821	β=0.0003, p=0.636
Mortality of persons ≥65	β= -0.0002, p=0.979	β= -0.0058, p=0.822	β=0.0006, p=0.442
Mortality due to cardiovascular disease	β= -0.0014, p=0.314	β=0.0018, p=0.658	β=2.7 x 10^{-5}, p=0.984
Mortality due to respiratory disease	β= -0.0009, p=0.728	β=0.0059, p=0.439	β=0.0037, p=0.135
Mortality due to pneumonia	β=0.0009, p=0.832	β=0.0228, p=0.071	**β=0.0107, p=0.010**
Mortality due to COPD	β= -0.0021, p=0.126	β=0.0038, p=0.354	β= -0.0012, p=0.360
Summer			
Total mortality	β=0.0022, p=0.070	β=0.0011, p=0.681	**β= -0.0019, p=0.004**
Mortality of persons ≥65	β=0.0013, p=0.363	β=7.8 x 10^{-5}, p=0.981	**β= -0.0026, p=0.001**
Mortality due to cardiovascular disease	**β=0.0048, p=0.043**	β=0.0033, p=0.541	**β= -0.0036, p=0.006**
Mortality due to respiratory disease	β= -0.0025, p=0.598	β= -0.0032, p=0.763	β=0.0004, p=0.861
Mortality due to pneumonia	β=0.0087, p=0.267	β=0.0150, p=0.387	β= -0.0014, p=0.722
Mortality due to COPD	β= -0.0045, p=0.532	β= -0.0126, p=0.424	β=0.0012, p=0.740
Fall			
Total mortality	β= -0.0002, p=0.771	β=0.0002, p=0.929	β=0.0005, p=0.553
Mortality of persons ≥65	β= -5.6 x 10^{-5},p=0.949	β= -0.0005, p=0.837	β=0.0014, p=0.172
Mortality due to cardiovascular disease	β=0.0007, p=0.579	**β=0.0076, p=0.048**	β= -0.0005, p=0.736
Mortality due to respiratory disease	**β=0.0151, p<0.0005**	**β= -0.0171, p<0.0005**	**β=0.0237, p<0.0005**
Mortality due to pneumonia	**β= -0.0124, p=0.003**	**β=0.0075, p=0.003**	**β= -0.0108, p=0.050**
Mortality due to COPD	**β= -0.0489, p<0.0005**	**β=0.0300, p<0.0005**	**β= -0.0625, p<0.0005**

Appendix C: Results of seasonal Poisson regressions for Hong Kong, 1997-1998

	PM_{10}	SO_2	NO_2

Winter 1997			
Total mortality	β=0.0001, p=0.790	β= -0.0012, p=0.340	β=0.0009, p=0.189
Mortality of persons ≥65	β=0.0002, p=0.696	β= -0.0009, p=0.508	β=0.0010, p=0.196
Mortality due to cardiovascular disease	β=0.0003, p=0.776	β= -0.0016, p=0.462	β=0.0006, p=0.632
Mortality due to respiratory disease	β=0.0009, p=0.452	β=0.0007, p=0.793	β= -0.0007, p=0.646
Mortality due to pneumonia	β= -0.0012, p=0.488	β= -0.0071, p=0.083	β= -0.0039, p=0.082
Mortality due to COPD	β=0.0029, p=0.121	**β=0.0079, p=0.050**	β=0.0027, p=0.282
Spring 1997			
Total mortality	β=0.0011, p=0.084	**β=0.0028, p=0.001**	β=0.0011, p=0.089
Mortality of persons ≥65	β=0.0007, p=0.323	**β=0.0022, p=0.026**	β=0.0007, p=0.373
Mortality due to cardiovascular disease	β=0.0017, p=0.140	β=0.0031, p=0.060	β=0.0013, p=0.287
Mortality due to respiratory disease	β=0.0010, p=0.416	β=0.0034, p=0.054	β= -0.0011, p=0.412
Mortality due to pneumonia	β=1.3 x 10^{-5}, p=0.994	**β=0.0050, p=0.021**	β= -0.0012, p=0.481
Mortality due to COPD	β=0.0032, p=0.154	β=0.0013, p=0.696	β= -0.0006, p=0.820
Summer 1997			
Total mortality	β= -0.0007, p=0.188	β= -0.0005, p=0.546	β= -0.0011, p=0.060
Mortality of persons ≥65	β= -0.0005, p=0.395	β= -0.0003, p=0.729	β= -0.0009, p=0.175
Mortality due to cardiovascular disease	β= -0.0013, p=0.240	β= -0.0006, p=0.702	β= -0.0014, p=0.213
Mortality due to respiratory disease	β= -0.0013, p=0.246	β= -0.0018, p=0.259	β= -0.0019, p=0.115
Mortality due to pneumonia	β=0.0002, p=0.907	β= -0.0004, p=0.823	β=0.0002, p=0.906
Mortality due to COPD	β= -0.0034, p=0.122	β= -0.0036, p=0.227	**β= -0.0052, p=0.024**
Fall 1997			
Total mortality	**β=0.0018, p<0.0005**	**β=0.0032, p<0.0005**	**β=0.0029, p<0.0005**
Mortality of persons ≥65	**β=0.0018, p=0.001**	**β=0.0033, p<0.0005**	**β=0.0032, p<0.0005**
Mortality due to cardiovascular disease	β=0.0011, p=0.219	**β=0.0033, p=0.027**	β=0.0023, p=0.057
Mortality due to respiratory disease	**β=0.0027, p=0.010**	β=0.0020, p=0.259	**β=0.0042, p=0.002**
Mortality due to pneumonia	**β=0.0033, p=0.010**	β=0.0028, p=0.194	**β=0.0045, p=0.006**
Mortality due to COPD	β=0.0012, p=0.549	β= -0.0010, p=0.785	β=0.0024, p=0.374
Winter 1997-1998			
Total mortality	β= -0.0009, p=0.146	β= -0.0017, p=0.207	β=0.0009, p=0.297
Mortality of persons ≥65	β= -0.0009, p=0.191	β= -0.0019, p=0.227	β=0.0011, p=0.232
Mortality due to cardiovascular disease	β= -0.0011, p=0.281	β= -0.0028, p=0.249	β= -0.0008, p=0.591
Mortality due to respiratory disease	β= -0.0001, p=0.917	β=0.0011, p=0.708	β=0.0002, p=0.937
Mortality due to pneumonia	β= -0.0003, p=0.863	β=0.0048, p=0.204	β=0.0002, p=0.937
Mortality due to COPD	β= -0.0015, p=0.502	β= -0.0067, p=0.190	β=0.0029, p=0.320
Spring 1998			
Total mortality	β=0.0003, p=0.540	β=0.0003, p=0.806	β=0.0004, p=0.538
Mortality of persons ≥65	β=0.0003, p=0.638	β= -0.0005, p=0.733	β=0.0004, p=0.608
Mortality due to cardiovascular disease	β=0.0004, p=0.664	β= -0.0037, p=0.135	β=0.0002, p=0.839
Mortality due to respiratory disease	β=0.0021, p=0.066	β=0.0034, p=0.241	β=0.0012, p=0.384
Mortality due to pneumonia	β= -0.0005, p=0.780	β= -0.0024, p=0.546	β= -0.0012, p=0.542
Mortality due to COPD	**β=0.0050, p=0.004**	**β=0.0115, p=0.012**	**β=0.0046, p=0.040**
Summer 1998			
Total mortality	β= -0.0005, p=0.520	β= -0.0021, p=0.152	β= -0.0009, p=0.198
Mortality of persons ≥65	β= -0.0004, p=0.629	β= -0.0016, p=0.347	β= -0.0006, p=0.502
Mortality due to cardiovascular disease	β= -0.0010, p=0.494	β= -0.0025, p=0.368	β= -0.0015, p=0.284
Mortality due to respiratory disease	β= -0.0012, p=0.468	β=0.012, p=0.699	β= -0.0020, p=0.220
Mortality due to pneumonia	β= -0.0022, p=0.304	β=0.0039, p=0.309	β= -0.0029, p=0.152
Mortality due to COPD	β= -0.0007, p=0.809	β= -0.0025, p=0.661	β= -0.0019, p=0.520

Fall 1998			
Total mortality	β=0.0005, p=0.326	**β=0.0051, p=0.015**	β=0.0003, p=0.699
Mortality of persons ≥65	β=0.0008, p=0.231	**β=0.0056, p=0.020**	β=0.0007, p=0.469
Mortality due to cardiovascular disease	β= -0.0006, p=0.570	β=0.0046, p=0.264	β= -0.0022, p=0.183
Mortality due to respiratory disease	β= -0.0021, p=0.106	β=0.0085, p=0.069	β= -0.0007, p=0.704
Mortality due to pneumonia	β= -0.0021, p=0.192	β=0.0093, p=0.105	β= -0.0007, p=0.748
Mortality due to COPD	β= -0.0021, p=0.391	β=0.0107, p=0.220	β=0.0005, p=0.891

Appendix D: Results of combined seasonal Poisson regressions for Hong Kong, 1997-1998

	PM_{10}	SO_2	NO_2

Winter			
Total mortality	β= -0.0003, p=0.393	β= -0.0018, p=0.052	β=0.0010, p=0.053
Mortality of persons ≥65	β= -0.0003, p=0.502	β= -0.0018, p=0.086	**β=0.0012, p=0.042**
Mortality due to cardiovascular disease	β= -0.0004, p=0.537	β= -0.0029, p=0.072	β=0.0003, p=0.731
Mortality due to respiratory disease	β=0.0003, p=0.698	β= -3.5×10^{-5},p=0.986	β=0.0008, p=0.511
Mortality due to pneumonia	β= -0.0009, p=0.477	β= -0.0023, p=0.402	β= -0.0015, p=0.364
Mortality due to COPD	β=0.0010, p=0.483	β=0.0016, p=0.604	β=0.0028, p=0.137
Spring			
Total mortality	**β=0.0042, p<0.0005**	**β= -0.0032, p<0.0005**	**β= -0.0019, p<0.0005**
Mortality of persons ≥65	**β=0.0130, p<0.0005**	**β= -0.0146, p<0.0005**	**β= -0.0129, p<0.0005**
Mortality due to cardiovascular disease	**β=0.0059, p<0.0005**	**β= -0.0046, p<0.0005**	**β= -0.0031, p=0.001**
Mortality due to respiratory disease	β=0.0016, p=0.053	**β=0.0045, p=0.003**	β=0.0005, p=0.592
Mortality due to pneumonia	β= -0.0001, p=0.901	**β=0.0051, p=0.008**	β= -0.0003, p=0.820
Mortality due to COPD	**β=0.0043, p=0.002**	β=0.0045, p=0.081	β=0.0022, p=0.184
Summer			
Total mortality	β= -0.0007, p=0.120	β= -0.0013, p=0.051	**β= -0.0013, p=0.003**
Mortality of persons ≥65	β= -0.0006, p=0.277	β= -0.0011, p=0.128	**β= -0.0011, p=0.026**
Mortality due to cardiovascular disease	β= -0.0013, p=0.148	β= -0.0012, p=0.340	**β= -0.0022, p=0.015**
Mortality due to respiratory disease	β= -0.0013, p=0.181	β= -0.0008, p=0.537	β= -0.0017, p=0.079
Mortality due to pneumonia	β= -0.0005, p=0.664	β=0.0009, p=0.562	β= -0.0005, p=0.089
Mortality due to COPD	β= -0.0025, p=0.162	β= -0.0032, p=0.219	**β= -0.0038, p=0.034**
Fall			
Total mortality	**β=0.0013, p=0.001**	**β=0.0033, p<0.0005**	**β=0.0020, p<0.0005**
Mortality of persons ≥65	**β=0.0014, p=0.001**	**β=0.0035, p<0.0005**	**β=0.0023, p<0.0005**
Mortality due to cardiovascular disease	β=0.0003, p=0.635	**β=0.0035, p=0.012**	β=0.0007, p=0.484
Mortality due to respiratory disease	β=0.0007, p=0.418	β=0.0029, p=0.078	**β=0.0025, p=0.026**
Mortality due to pneumonia	β=0.0010, p=0.311	β=0.0036, p=0.066	**β=0.0027, p=0.049**
Mortality due to COPD	β= -0.0001, p=0.942	β=0.0005, p=0.867	β=0.0017, p=0.428