Reassessment of the Lethal London Fog of 1952: Novel Indicators of Acute and Chronic Consequences of Acute Exposure to Air Pollution

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This article develops and assesses novel indicators of respiratory and other morbidity and mortality following London's lethal smog in the winter of 1952. Public health insurance claims, hospital admission rates for cardiac and respiratory disease, pneumonia cases, mortality records, influenza reports, temperature, and air pollutant concentrations are analyzed for December–February 1952–1953 and compared with those for the previous year or years. Mortality rates for the smog episode from December 1952 to February 1953 were 50–300% higher than the previous year. Claims that the smog only elevated health risks during and immediately following the peak fog 5–9 December 1952 and that an influenza epidemic accounted fully for persisting mortality increases in the first 2 months of 1953 are rejected. We estimate about 12,000 excess deaths occurred from December 1952 through February 1953 because of acute and persisting effects of the 1952 London smog. Pollution levels during the London smog were 5–19 times above current regulatory standards and guidelines and approximate current levels in some rapidly developing regions. Ambient pollution in many regions poses serious risks to public health. *Key words:* air pollution, history, indicators, London, morbidity, mortality, relative risk. — *Environ Health Perspect* 109(suppl 3):389–394 (2001). *http://ehpnet1.niehs.nih.gov/docs/2001/suppl-3/389-394bell/abstract.html*

In the last half of the twentieth century, several widely publicized acute episodes of lethal smogs spurred public understanding of the hazards of air pollution. One of the earliest such events occurred from 1 December to 5 December 1930 in the Meuse Valley in Belgium (*1*). Stable atmospheric conditions and industrial pollution from steel mills, coke ovens, foundries, and smelters in Liege, Belgium, contributed to the accumulation of air pollutants including sulfur dioxide (SO_2), sulfuric acid mists, and fluoride gases. In the last 2 days of the event, more than 60 persons died, which was more than 10 times the normal mortality rate (*2*).

The first publicly recognized extreme air pollution episode in the United States took place in Donora, a small town in southwestern Pennsylvania. From 27 October to 30 October 1948, an intense, anticyclonic meteorologic inversion settled on the valley town, fueled by pollution from metal works, coal-fired home and industrial facilities, coke ovens, a zinc retort refinery, and iron and steel industries. Seventeen people died on Saturday, 30 October, and three more died within the week. The death rate was more than 6 times the norm for the Donora and Webster Hollow area, which had a population of about 14,000. Pollution was so severe that local funeral homes did not have enough coffins and many residents evacuated (*3,4*).

Long known for its foggy weather and coal-burning homes, power plants, and factories, London, England, experienced a dense smog from 5 December to 9 December 1952. According to official government reports, this lethal fog resulted in about 3,000 more deaths than normal during the first 3 weeks of December 1952. With a death rate more than 3 times the norm for this period (*5*), the London fog of 1952 is widely regarded as a catalyst for the study of air pollution epidemiology.

The official report on the London episode by the Ministry of Health noted morbidity and mortality remained elevated from December 1952 until March 1953 in the region of Greater London. However, the report attributed these increased rates to an influenza epidemic, while recognizing some deaths may have been due to lingering effects from the fog (*5*). In 1954 Wilkins noted this prolonged increase in mortality and suggested it could be related to air pollution. He also indicated the December 1952 fog might have impaired resistance to illness, causing higher mortality in subsequent months (*6*). No follow-up work was done to clarify this idea at the time, and official estimates attributed lingering increased rates of illness and death to influenza.

At the time they occurred, each of these three cases was prominent in the news, signaling in very public ways that severe air pollution could be fatal in a relatively short time. Those events spawned public health research into various impacts of air pollution and the search for mechanisms by which smog might be linked with altered death rates.

Immediate investigations in Donora, London, and Liege confirmed associations between short-term reductions in air quality and increased mortality during acute episodes (*7*). However, no studies were produced within the first several years that addressed persisting public health consequences after each of these episodes. More recently, Schwartz compared the daily mortality and total particulate matter (PM) concentrations for 2–5 December, the 4-day period preceding the episode, to those during the 6–9 December episode and found an increased relative risk (RR) of mortality of 1.06 per 100 $\mu g/m^3$ increase in total suspended matter (TSM) (*8*).

Since these events, public health researchers have provided extensive documentation that acutely elevated exposures do not cause only acutely evident public health effects. These exposures also contribute to chronic health problems (*9*). By extending the period of analysis and looking at novel direct and indirect indicators of respiratory morbidity and mortality for the 3 months after the 1952 London fog, this article establishes that the original assessment was incomplete.

Methods

In an effort to clarify the connections between acute air pollution during the London smog and both acute and chronic health effects linked with this episode, we assembled original information and conducted regression analyses of various direct and indirect indicators of respiratory morbidity and mortality and air pollution, with appropriate lag periods. We included the following:

- Analysis of changes in the RR of daily mortality and measures of SO_2 and smoke or particles (TSM) for the days surrounding and including the fog in December 1952, including consideration of minimum and maximum temperature and relative humidity
- Analysis of weekly mortality and air pollution data including several weeks before and after the episode from October 1952 through March 1953 and corresponding

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weeks from 1951 and 1952, and consideration of temperature, season, autocorrelation of weekly mortality, and exclusion of the week of the severe fog
- Analysis of indirect indicators of morbidity: insurance claims, hospital admissions, and notifications of pneumonia from November 1952 to January 1953 with comparisons for previous years
- Assessment of the potential role of influenza in accounting for elevated rates of morbidity and mortality during the 3 months after the lethal smog episode and calculation of the excess deaths due to the smog
- Examination of levels of key pollutants during the lethal fog compared to modern-day standards and to levels encountered currently in some rapidly developing regions.

Data Sources and Limitations

Air pollutant concentrations used in analysis are averages of measurements from 12 monitors in Greater London. Only TSM and SO_2 air pollutant concentrations were routinely measured at the time. The two pollutants were highly correlated. In much of the analysis, SO_2 is used as a surrogate for air pollution. Daily temperature estimates are averages of measurements at two locations. Daily relative humidity is an average of measurements taken 3 times a day at the same two locations (*5*).

Weekly mortality estimates represent the number of deaths registered with the British General Register Office Statistics. Deaths typically were registered within 2–3 days of their occurrence (*5*).

Weekly insurance claims are based on claims for new illnesses received by the Ministry of Pensions and National Insurance (*5*). Hospital admissions information is based on reports from four metropolitan regional hospital boards in Greater London (5). Applications for emergency bed service refer to applications to the Emergency Bed Service Bureau (*5,10*). Pneumonia was a notifiable disease in England; therefore, notifications of pneumonia cases are available (*5*). Influenza was not a notifiable disease, so the exact number of new cases is unknown, as is the case fatality rate. Influenza incidence and case fatality rate were estimated from observations in general practice in the London area from 1949 to 1968 and from a vaccine study conducted at this time (*11,12*).

The major limitation of this research is data availability. Some information was not collected at the time, such as influenza mortality and air pollutant concentrations for pollutants other than TSM and SO_2. This work is limited to data readily available, which results in different baseline years used for various outcomes. Some health indicators are compared to those of the previous year, the corresponding 1951–1952 time frame, whereas a multiple-year comparison would be preferable. This analysis assumes the 1951–1952 mortality data represent a typical year, as there are no reports of influenza outbreaks or severe fogs during that time (*6,13*).

Results

Acute Effects on Mortality

Daily air pollution concentrations of SO_2 and TSM and mortality from 2 December to 10 December 1952 are highly correlated (Table 1), as are daily mortality and SO_2 and TSM levels from the same and previous day in Greater London and London Administrative County (*5*).

Poisson modeling of mortality, temperature, and the previous day's pollutant concentrations from 3 December to 10 December 1952 for Greater London indicates a significant association between pollutant levels and death for the few days of data that are available. For a 0.10-ppm increase in the previous day's SO_2 level, the estimated RR for daily mortality is 1.19 (95% confidence interval [CI] 1.12–1.27). Alternatively, the estimated RR for a 100 μg/m^3-ppm increase in the previous day's TSM resulted in an estimated relative risk for mortality of 1.08 (95% CI 1.05–1.10). These results are not sensitive to outlier data points. Removing the day of peak pollution gives an SO_2 RR of 1.21 (95% CI 1.12–1.30) and a TSM RR of 1.07 (1.05–1.11). As the two pollutants were highly correlated (0.98), it is difficult to separate their effects.

Backward regression analysis of daily minimum temperature, maximum temperature, relative humidity, and the previous day's TSM and SO_2 concentrations produced a model including SO_2 and maximum temperature. The RR for a 0.10 increase in daily SO_2, adjusted for maximum temperature, is 1.27 (95% CI 1.17–1.38). No interaction was apparent between SO_2 and TSM or between either pollutant and temperature. Associations are even stronger when only the Administrative County of London is considered. Table 2 provides RR for daily mortality.

Regression analysis of approximate weekly mortality and air pollutant concentrations in Greater London included time before and after the severe event. Weekly estimates of mortality from 11 October 1952 to 28 March 1953 are significantly associated with the week's SO_2 concentration. SO_2 is used rather than TSM based on data availability and because the two pollutants were highly correlated. For a 0.10-ppm increase in weekly SO_2, the RR for weekly mortality is 1.35 (95% CI 1.08–1.68). Note, however, if the week of the episode is removed from analysis, the association remains positive, although not significant (RR 1.21, 95% CI 0.83–1.76). When data from corresponding weeks in 1951 and 1952 are also considered, the RR becomes 1.38 (95% CI 1.18–1.60). Omitting the week of the severe event in 1952 provides a positive and significant RR of 1.28 (95% CI 1.03–1.59).

Weekly mortality estimates are correlated with mortality from the previous week (0.58 for the 1952/1953 data). Regression analysis including a term for the previous week's mortality provides a similar but even more significant RR for a 0.10-ppm increase in SO_2. The estimated RR is 1.32 (95% CI 1.16–1.51). When the corresponding 1951 and 1952 weeks also are considered, the RR is 1.25 (95% CI 1.15–1.37). These relationships were not confounded by temperature. The RR for a 0.10-ppm increase in the weekly SO_2 average was 1.26 (95% CI 1.14–1.39), adjusted for weekly average temperature and the previous week's mortality.

To account for possible confounding by season and varying underlying mortality, the analysis was repeated with variables to indicate month. This did not greatly affect the results. The association remained significant with a RR of 1.31 (95% CI 1.11–1.56), adjusted for the previous week's mortality. When the corresponding 1951 and 1952 weeks are included, the RR becomes 1.25 (95% CI 1.13–1.38).

Table 1. Correlations for daily mortality and air pollutant concentration.

	SO_2		TSM	
	Same day (2–10 December 1952)	Previous day (3–10 December 1952)	Same day	Previous day
Mortality in Greater London	0.89	0.94	0.83	0.96
Mortality in London Administrative County	0.89	0.93	0.83	0.95

Table 2. Relative risk for daily mortality and the previous day's pollution levels.

	Greater London	London Administrative County
SO_2 (0.10-ppm increase)	1.19 (1.12–1.27)	1.24 (1.14–1.35)
Adjusted for maximum temperature	1.27 (1.17–1.38)	1.34 (1.20–1.51)
TSM (100-μg/m^3 increase)	1.08 (1.05–1.10)	1.10 (1.06–1.13)
Adjusted for maximum temperature	1.08 (1.06–1.10)	1.09 (1.07–1.12)

Mortality also was associated, although not significantly, with average levels of SO_2 during the previous week. When SO_2 levels of both the current and previous week are considered, the estimated RRs become 1.26 (1.01–1.57) for a 0.10-ppm increase in SO_2 from the same week and 1.20 (0.96–1.50) in SO_2 from the previous week.

Long-Term Effects on Mortality

Information extracted from this period shows the severe air pollution episode of 5–9 December 1952 was followed by a period of elevated pollution for 2 months and an increase in mortality that did not decline to normal levels for several months. In the first 2 months after the fog, both mortality and SO_2 stayed above expected levels and slowly rose.

Figure 1 depicts approximate weekly total mortality and weekly average SO_2 concentrations for Greater London (*5*). Deaths in Greater London in late 1952 before the episode averaged about 1,570 per week, similar to the death rate in 1951. After the December episode, mortality remained elevated for some time. For the week ending 13 December registrations of death were roughly 2.5 times higher than the corresponding weekly averages from 1947 to 1951. Mortality remained almost twice that of pre-episode levels for the next 2 weeks, and rates did not decline to normal levels over the next 2 months. Deaths in January and February 1953 were approximately 50% higher than expected, based on figures available for the same months of the previous year.

Morbidity

Insurance claims received by the Ministry of Pensions and National Insurance for new sickness in the working population of London and Middlesex rose in late December 1952 (*5*). For the week of 3–9 December 1952, which includes the episode, claims were 36% higher than the corresponding weekly average for 1949–1951, resulting in an excess 5,400 claims. The next week, claims were 108% higher than the corresponding 1949–1951 average, with 15,000 excess claims. The number of claims for the following 4 weeks is similar to the corresponding weekly averages for 1949–1951. New illnesses increased sharply during and after the fog, then returned to normal levels.

Figure 2 displays weekly average SO_2 concentrations and the ratio of weekly insurance claims for Greater London to the corresponding weekly average for 1949–1951. A similar pattern can be seen in SO_2 levels and insurance claims for new sickness.

Available weekly insurance data are provided for 7-day periods from Wednesday to Tuesday, whereas weekly SO_2 averages are provided for the 7-day periods from Sunday to Saturday. Excess insurance claims, those above 1949–1951 weekly levels, were analyzed with the SO_2 concentrations averaged across 3–9 days before the insurance claim week ended. For example, excess insurance claims from 12 November to 18 November were paired with SO_2 levels from 9 November to 15 November. The correlation of weekly insurance claims for new illness (29 October 1952–13 January 1953) and weekly average SO_2 (26 October 1952–10 January 1953) is 0.93. If the week containing the episode is omitted, the correlation is 0.71, indicating a strong relationship between illness and air pollution.

Hospital admissions also rose during the fog (*5*). Before the severe episode, hospitals in Greater London admitted approximately 750 cases per day—about 23% for respiratory disease. For the week ending 9 December a total of 1,100 patients were admitted—41% for respiratory disease. Respiratory disease hospital admissions increased 163%, and total hospital admissions increased 48%. Information on admissions for the next 2 months was not available for this analysis.

Applications to the Emergency Bed Service Bureau, which occurred when a patient could not be admitted directly to a particular hospital during and following the episode, far exceeded those of previous years. Weekly averages for London from 1949 to 1952 indicate that a sharp increase in December was not typical and that applications normally peaked in early January. However, on 9 December 1952, applications reached 492, the highest number recorded at that time. Weekly averages in December 1953 peaked around 2,550 applications per week, whereas the highest weekly averages for the December months of previous years were approximately 1,050, 1,600, and 1,200 for 1950, 1951, and 1952, respectively (*10*).

Before the episode the number of applications from 1 December to 4 December averaged 224 per day—47 were for respiratory disease and 15 for cardiac disease (*5*). Figure 3 shows the ratio of the number of admissions for a given 4-day period to those for the 4 days before the episode.

During and after the fog, emergency bed service applications increased, especially for respiratory and cardiac diseases. Total emergency bed service applications and the previous day's SO_2 concentrations have a correlation of 0.78, whereas applications for cardiac disease are even more strongly correlated at 0.89. Applications for admission for respiratory disease were more highly correlated with the previous day's SO_2, with a correlation of 0.90. Figures 4A and 4B depict the rise in SO_2 and associated rise in

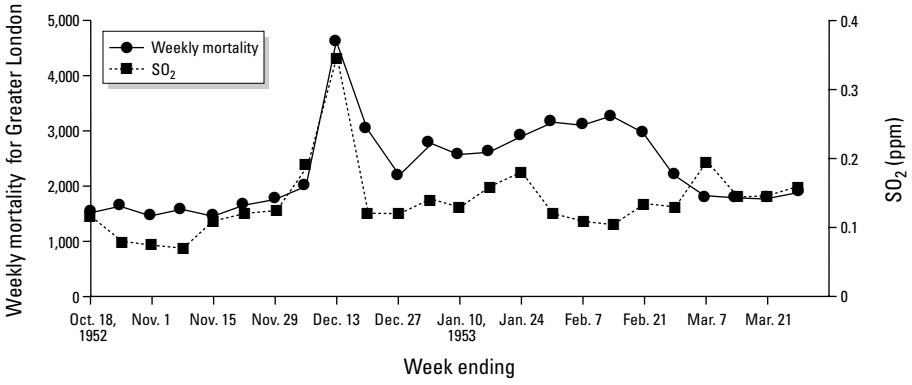

Figure 1. Approximate weekly mortality and SO_2 concentrations for Greater London, 1952–1953.

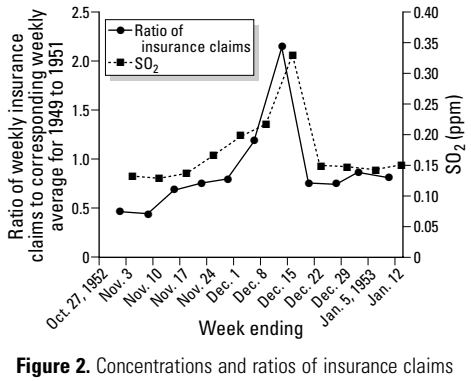

Figure 2. Concentrations and ratios of insurance claims for new sickness in London and Middlesex to corresponding weekly averages for 1949–1951.

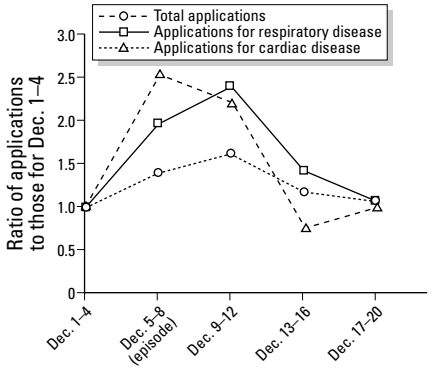

Figure 3. Ratio of applications for emergency bed service in Greater London to those from 1–4 December 1952.

emergency bed applications for respiratory and cardiac disease (*5*).

Notifications, or official reports of pneumonia cases for Greater London from 1 December to 6 December 1952 were 1.4 times the corresponding weekly average for 1947–1951 (*5*). The following 2 weeks they were 2.7 and 2.4 times higher than 1947–1951 levels. Figure 5 shows SO_2 concentrations and the ratio of notifications of pneumonia in 1952 to the corresponding weekly average for 1947–1951. These records show an increase in pneumonia that roughly follows the pattern of SO_2 levels.

The morbidity evidence shows a strong episodic effect but does not appear to show the lingering long-term effects demonstrated with mortality. This may be because of differences in air pollution effects on morbidity or mortality or a lack of data on morbidity impacts.

Potential Role of Influenza in Accounting for Mortality in London, January–March 1953

To clarify the potential role of influenza in accounting for the persisting increases in respiratory morbidity and mortality, it is necessary to consider that at the time of the lethal fog, influenza was a nonnotifiable disease, so no official records were kept of new cases. Thus, the actual incidence and case–fatality rate of influenza are unknown.

Incidence and mortality due to influenza can be reconstructed from other information available for the period. Observations from general practice of medicine in the London area from 1949 to 1968 indicate the overall case–fatality rate from influenza was 0.2%. During this period the incidence of influenza ranged from 3 to 17%, with a mean of 8% annually (*11*). The population of Greater London at the time of this episode was approximately 8.3 million (*12*). If the historical average rate of influenza did occur during this time, namely 8%, with a case–fatality rate of 0.2%, this would have resulted in about 1,328 influenza-related deaths for the entire year, and even fewer for a shorter period. A clinical trial of an influenza vaccine with 12,710 volunteers found attack rates of influenza of 3% in the vaccinated group and 4.9% in the control group during the winter of 1952–1953 (*14*). This could imply an even lower incidence, and therefore fewer deaths, than the estimated 1,328 influenza-related deaths. In theory, the actual incidence and case–fatality rate for the influenza epidemic may have exceeded this range, but there is no empirical evidence to suggest this was the case.

The annual estimate of influenza deaths of about 1,300 could not account for all the excess deaths that took place in the months following the London fog of 1952– 1953. Total mortality in Greater London from January to March 1953 exceeded the previous year's tally by 8,635 deaths, with the vast majority of these deaths occurring in January and February. Figure 6 depicts the unexplained deaths in Greater London using the reconstructed estimated number of influenza deaths. Over 7,000 noninfluenza deaths took place that are not otherwise explained in the months following the fog.

According to yearly reports by the Chief Medical Officer on the State of the Public Health from the Ministry of Health, no major outbreak of influenza occurred in England in 1952 (*13*), and only a relatively mild influenza outbreak took place during the winter of 1953 (*14*). Without providing any clinical support for its reasoning, the British Department of Health reported that 5,655 deaths from influenza occurred in the first 3 months of 1953 (*14*), and another source reported 5,647 influenza deaths during this period (*15*). However, over 8,000 excess deaths occurred in January and February alone. The total number of deaths that occurred during this time was more than 2 times higher than that attributed to influenza under this undocumented scenario. This was because over 13,000 excess deaths occurred from December 1952 through March 1953, when only about 5,600 influenza deaths were officially reported.

Our recalculation of influenza deaths for this period clearly shows the official estimates of influenza deaths in the formal report on this episode were inflated. However, even if the official British Department of Health reports of influenza deaths were accurate, about 3,000 deaths remain unexplained by influenza. Effectively, additional excess deaths remain unexplained.

It is clear from the proceeding analysis not all the excess deaths can be attributed to influenza. On the basis of analyses of the levels of air pollution that occurred and the absence of information confirming an epidemic of influenza, these deaths in January–March of 1953 may stem from high levels of air pollution, which continued for months following the episode. Regression analysis found an association between weekly average SO_2 concentrations and mortality, even when the week of the extreme episode was excluded. For months after the fog, SO_2 concentrations exceeded both pre-episode levels and those from the corresponding 1951–1952 time frame, which had typical air pollution levels. Alternatively, mortality in months after the fog may be related to lingering effects from the episode itself.

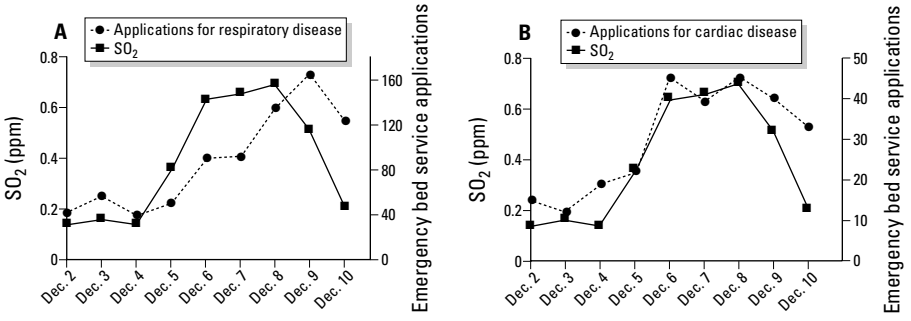

Figure 4. (*A*) SO_2 and emergency bed service applications for respiratory disease for Greater London, 1952. (*B*) SO_2 and emergency bed service applications for cardiac disease for Greater London, 1952.

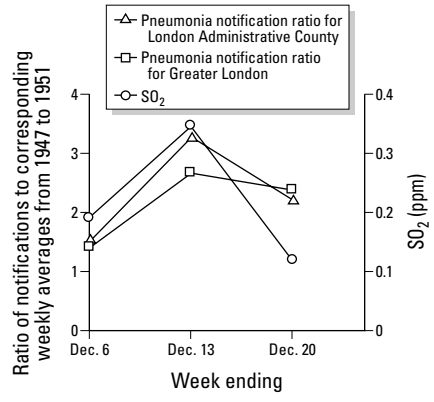

Figure 5. SO_2 concentrations and ratios of notifications of pneumonia in 1952 to corresponding weekly averages for 1947–1951.

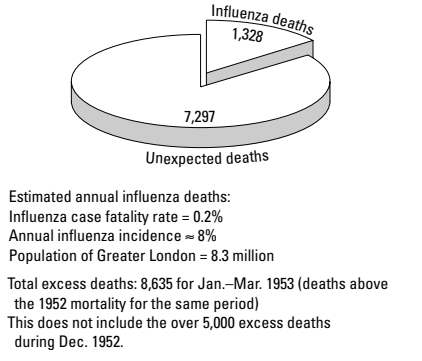

Figure 6. Excess deaths from the London smog for January– March 1953.

If the official estimates of influenza deaths are inflated, as evidenced by our analyses, the number of unexplained excess deaths from 30 November 1952 to 28 February 1953 is about 12,000 (13,375 excess deaths minus 1,328 influenza deaths). This figure is based on the conservative assumption that all influenza deaths for the year occur by the end of February. Even if the official estimates of influenza mortality are accurate, an unexplained excess mortality of about 7,700 remains (13,375 excess deaths minus 5,655 influenza deaths or, using another source for the influenza mortality, 13,375–5,647). If the elevated death rates in the months following the severe episode in early December are attributable to the severe fog, the number of excess deaths would exceed 7,000, which is much higher than the 3,000 or so generally reported (*5,7,16*).

Comparison of Estimated Levels of Pollution during London Lethal Fog with Ambient Levels Recorded in Some Countries

Air pollution levels during the London fog of 1952 greatly exceeded modern-day U.S., British, and European regulations and World Health Organization guidelines (Table 3) (*17–21*). SO_2 concentrations were 12–23 times higher than those set by these regulations and guidelines, and PM concentrations were about 5–19 times higher. Reports from many developing countries indicate past levels in some regions have approximated those reported in London (*22*). Current levels in poorly monitored zones may also approach these levels and have similar consequences. Tables 3 and 4 show that air pollution levels reported during the lethal London fog of 1952–1953 can be found in many cities today and can range 5–20 times higher than modern regulatory requirements (*17–21*). For instance, the average annual level of total suspended particulate (TSP) reported in 1995 in Delhi was over 400 μg/m^3. The annual TSP concentrations for Beijing and Calcutta were about 380 μg/m^3 each (*22*). A number of cities in China have had annual levels in excess of 700 μg/m^3 in the mid-1990s. Current levels in some cities are lower. However, levels measured in many developing countries in the 1990s were much higher than the annual smoke concentration of 270 μg/m^3 for Greater London in 1952 (*23*).

Table 3. Comparison of 1952 air pollution levels in London to current standards.

	SO_2	TSM
Pollution levels for Greater London, 5–8 December 1952		
Episode average	0.57 ppm	1,400 μg/m^3
Highest daily average	0.69 ppm	1,620 μg/m^3
Regulatory standards and guidelines (24-hr average)		
U.S. NAAQS	0.03 ppm	260 μg/m^3 [a] ~250 μg/m^3 [b]
British environmental regulation	125 μg/m^3 (0.047 ppm)	~83 μg/m^3 [c]
WHO guidelines	125 mg/m3	
European Union legislation	125 μg/m^3	~83 μg/m^3 [c]
Ratio of Greater London 1952 levels to standards and guidelines		
For episode average		
U.S. NAAQS	19	5.4–5.6
British standards	12	17
WHO guidelines	12	
European Union legislation	12	17
For highest daily level		
U.S. NAAQS	23	6.2–6.5
British standards	15	19
WHO guidelines	15	
European Union legislation	15	19

Abbreviations: NAAQS, National Ambient Air Quality Standards; WHO, World Health Organization.
[a]TSP NAAQS from 1971 to 1987. [b]TSP estimate based on PM_{10}/TSP of 0.6 and current PM_{10} standard of 150 μg/m^3. [c]TSP estimate based on PM_{10}/TSP of 0.6 and current PM_{10} standard of 50 μg/m^3.

Table 4. Comparison of air pollution levels to current standards for selected cities.

	SO_2	TSP
Approximate annual pollution levels for select cities (1993–1995)		
Guiyand, China	0.16 ppm	–
Chongqing, China	0.13 ppm	–
Delhi	–	410 μg/m^3
Rio de Janeiro	0.05 ppm	140 μg/m^3
Moscow	0.41 ppm	100 μg/m^3
Beijing	0.03 ppm	375 μg/m^3
Regulatory standards and guidelines (annual)		
U.S. NAAQS	80 μg/m^3 (0.03 ppm)	~83 μg/m^3 [a]
British environmental regulation	–	~67 μg/m^3 [b]
WHO guidelines	50 μg/m^3 (0.02 ppm)	–
European Union legislation	–	~83 μg/m^3 [a]
Highest ratio of cities' pollution levels to standards and guidelines		
U.S. NAAQS	14	4.9
British standards	–	6.1
WHO guidelines	21	–
European Union legislation	–	4.9

[a]TSP estimate based on PM_{10}/TSP of 0.6 and current PM_{10} standard of 50 μg/m^3. [b]TSP estimate based on PM_{10}/TSP of 0.6 and current PM_{10} standard of 40 μg/m^3.

Discussion

The analyses presented here indicate that the true scope and scale of the health effects linked with London's lethal smog extended over a longer period than originally estimated. The fact that respiratory deaths and illness in January and February are more highly correlated with average measures of SO_2 and TSM for the previous week than for the current week suggests increased morbidity and mortality may be driven by cumulative exposures or by a lagged effect. This interpretation of the important role of chronic exposures for acute events such as death and respiratory hospitalizations is corroborated by recent studies finding that the RR for cardiac emergency admissions is significantly associated with annual measures of air pollution (*9*).

In London, this lethal episode of pollution was associated with a rise in morbidity, as evidenced by insurance claims for new illness, hospital admissions, applications to emergency bed service, and notifications of pneumonia, all of which have patterns similar to air pollution concentrations. The episode was also accompanied and followed by unusually high mortality rates for a period of 2.5 months, which shows a strong correlation with pollution levels, indicating that the fog increased acute mortality. Long-term mortality may also have been affected, as death rates did not return to normal levels until the end of February.

Although earlier analysis attributed many of these excess deaths to a mild influenza epidemic, a closer look at the number of excess deaths indicates only a fraction of them could have been from influenza. Although the excess deaths reported here cannot be conclusively linked to air pollution, it is highly likely the true tally of this episode is much higher than earlier reported.

Current air pollution research suggests the acute episode of pollution during London's lethal smog would have been expected to have both immediate and chronic public health impacts. New evidence exists on the chronic health impacts of even lower levels of air pollution, particularly its role for cardiac

disease as well as respiratory disease (*9,24*). Every year an estimated 8,100 deaths occur in urban areas of Great Britain due to PM_{10} (PM with a mass median aerodynamic diameter less than 10 μm), and 3,500 deaths result from SO_2. Respiratory hospital admissions are increased by 10,500 from PM_{10} pollution and 3,500 for SO_2 (*25*).

Events as extreme as the 1952 fog are rare in developed countries, although such levels are likely to occur in rapidly growing regions, where monitoring of pollution and health are not widely conducted. In rapidly growing midsize cities of some regions, limited monitoring of air pollution or public health is available, but it is likely conditions exist similar to those of London.

However, air pollution is not restricted to developing regions. From 12 December to 15 December 1991 a dense smog of high levels of nitrogen dioxide and PM settled over London. PM, measured as black smoke, reached a concentration of 228 $\mu g/m^3$, comparable to levels during the 1952 episode. During these days mortality rates rose about 10% (*26*).

Air pollution in London in 1952 differs from modern-day pollution in its severity and pollutant mixture, primarily because of the shift away from coal as a fuel source. Yet reanalysis of this historic event can shed light on the impact of air pollution on premature death and morbidity worldwide. Even with a limited data set and without the use of sophisticated statistical methods, the impact of the London killer fog on mortality is clear. Such extreme events are indicators of what can happen under circumstances of meteorologic inversion and concentrated pollution.

In many rapidly growing midsize cities today, the conditions of London and Donora can arise. Although routine monitoring of health or pollution is not conducted in some areas, this analysis suggests the need for vigilance about public health impacts linked with air pollution in such regions. The absence of public information on the problems of air pollution in industrialized areas should not be mistaken as a signal that such effects are not occurring. Rather, opportunities abound for reducing and controlling such pollution, thereby improving public health and preventing tragedies such as those that occurred in Donora and London.

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