

# Quantification of Rate of Air Pollution By Means of Statistical Data Collections from Hospitals

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**Abstract-** To develop efficient strategies for pollution control, it is essential to assess both the costs of control and the benefits that may result. These benefits will often include improvements in public health, including reductions in both morbidity and premature mortality. Until recently, there has been little guidance about how to calculate the benefits of air pollution controls and how to use those estimates to assign priorities to different air pollution control strategies. In this work, a method is described for quantifying the benefits of reduced ambient concentrations of pollutants (such as ozone and particulate matter) typically found in urban areas worldwide. The method applies the data on Jakarta, Indonesia, an area characterized by little wind, high population density (8 million people), congested roads, and ambient air pollution. The magnitude of the benefits of pollution control depends on the level of air pollution, the expected effects on health of the pollutants (dose-response), the size of the population affected, and the economic value of these effects. In the case of Jakarta, the methodology suggests that reducing exposure to lead and nitrogen dioxide should also be a high priority. An important consequence of ambient lead pollution is a reduction in learning abilities for children, measured as I.Q. loss. Apart from that, reducing the proportion of respirable particles can reduce the amount of illness and premature mortality.

**Key words:** air pollution, ozone, particulate matter, respirable particles, lead, nitrogen dioxide.

## I. INTRODUCTION

Until recently, there has been little guidance about the calculation of the social costs of air pollution and about using these costs to evaluate alternative air pollution control strategies. With limited resources, rational decision-making requires some quantification of the potential benefits of controlling air pollution. These benefits are dependent on the expected health effects of the pollutant, the magnitude of the effect in response to air pollution (dose-response), the economic valuation of the adverse effect, and the existence of subpopulations particularly sensitive to air pollution. Information about health and economic effects of air pollution needs to be categorized for pollutants commonly discharged by mobile and stationary sources. This work describes a method for determining quantitative estimates of the benefits of reducing ambient concentrations of five pollutants: particulate matter, sulfur dioxide, nitrogen dioxide, ozone and lead. This

methodology is then applied to Jakarta, Indonesia. A brief review of the effects of carbon monoxide and carbon dioxide is also provided. Once the benefits (both quantified and unquantified) of control are calculated, they can be incorporated in decisions about prioritizing control strategies. For cost-benefit analysis of air pollution control, a common denomination for various health effects would be used. It could be based on willingness to pay, medical treatment costs and the value of lost productive days and years. Such valuation is beyond the scope of this paper, however.

It should be acknowledged, however, that large uncertainties about the existence and magnitude of the health effects of air pollution continue to exist. Therefore, the analysis provided below should be viewed as an attempt to present the most likely and well-documented health impacts for which quantitative information exists.

In the past, the U.S. Environmental Protection Agency has estimated the health and welfare effects of air pollution in its Regulatory Impact Analysis for national ambient air quality standards, as required by the Presidential Executive Order #12291 issued in 1981. Additional information and methodological improvements were incorporated in the subsequent analysis of economic benefits of air quality programs in selected U.S. locations. Recently, broad estimates of the health benefits of controlling ozone and particulate matter were provided for both the U.S. and for the ambitious control plans being considered in Southern California.

The analysis reported here uses a similar approach to estimate health effects of criteria pollutants in Jakarta, with two improvements: the most recent set of research findings are utilized and a full range of health endpoints are included. Dose-response functions that relate various health outcomes to air pollution are taken from the available peer reviewed literature. Estimates of selected health effects of air pollution are generated by applying these functions to ambient levels either observed from monitoring stations located throughout the city or estimated from available dispersion models. Using results from both time-series and cross-sectional epidemiologic analyses from the United States, Canada, and Britain, effects are estimated for such health outcomes as premature mortality, hospital visits and admissions, emergency room visits, restrictions in activity, acute respiratory symptoms, acute bronchitis in children, asthma attacks, IQ loss, and blood pressure changes. At this time, however, because of uncertainties about the coverage and representativeness of the existing monitors in the city, and about the applicability of health studies undertaken in the U.S. to the developing world, the results should be viewed as providing only general estimates of the impacts of air pollution.

## II. METHODOLOGY AND BASELINE ASSUMPTIONS FOR ESTIMATING HEALTH EFFECTS

The estimation of the health and economic effects of air pollution involves the use of methodology similar to that used by the U.S. Environmental Protection Agency (EPA) in their Regulatory Impact Analysis for a new national air quality standard for particulate matter. Estimation techniques are also derived from the analyses of economic benefits of air quality control programs in selected U.S. locations. To estimate the economic value associated with changes in air pollution, four factors must be determined: the dose-response relationships, the susceptible populations, the relevant change in air pollution, and an economic valuation of the health endpoints. In this work, health effects for a range of health outcomes are provided, while valuation of these is not performed.

The first step is to develop estimates of the effects of air pollution on various health outcomes. Dose-response functions that relate health impacts to ambient levels of air pollution are taken from the published epidemiologic literature. This step involves calculating the partial derivative (or slope,  $b$ ) of the dose-response function, to provide an estimate of the change in the prevalence of a given health effect associated with a change in outdoor air quality ( $A$ ). Sufficient information is provided in this report to understand the sources of the selected dose-response functions, but a more complete review of the literature can be obtained in the EPA scientific review of the health effects of criteria pollutants?

The next step involves multiplying this slope by the relevant population that is believed to be exposed and susceptible to the air pollutant effect under consideration ( $POP@$ ). For certain pollution-related health effects this may include the entire exposed population; for other effects there may be particularly sensitive subgroups such as children or asthmatics.

A third step in the calculation of health effects of air pollution involves the change in air quality ( $dA$ ) under consideration. The actual change is dependent on both the policy issue under consideration and the available data. For example, it may be relevant to consider the change from current air pollution levels to some ambient air quality standard, either a local one, the EPA standard, or the WHO air quality guideline. A second change that might be relevant for consideration is a given percent reduction, such as 10 percent. A third method of determining the relevant change in air pollution is to assume that air quality changes in some simple proportion to the change in emissions, as in a simple linear rollback model. In that case, a 10 reduction in the total tonnage of particulate emissions, for example, is assumed to reduce ambient particulate air pollution and health effects by 10 percent. Finally, the ambient changes associated with a given change in a stationary or area-wide pollution source can be calculated through use of computer models, if the necessary data are available.

In this report, we examine a change from existing levels to several alternative ambient standards including: (1) proposed Indonesian standards, (2) EPA ambient air quality standards, (3) WHO guidelines, and (4) California state ambient air quality standards. For the case of lead, we also calculate the benefits associated with a 90 percent reduction of ambient lead, assumed to be accomplished through a ban on leaded gasoline. The relevant standards, expressed in terms of the annual average concentration in micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ), are as follows:

TABLE I: AMBIENT AIR QUALITY STANDARDS FOR ANNUAL AVERAGES (MICROGRAMS/M<sup>3</sup>)

Pollutant	Proposed Indonesian	EPA	WHO	California
Total Suspended Particles (TSP)	90	75	60-90	55
Lead	0.5	N/A	0.5-1.0	N/A
Nitrogen dioxide	100	100	N/A	N/A
Sulfur dioxide	60	80	50	N/A
Ozone	200	240	150	180

Where, N/A signifies standards with averaging time other than annual average.

### *2.1 Baseline Assumptions*

An important question in all of the health effects estimates is whether a threshold level exists, below which health effects no longer occur, or whether the slope of the dose-response function diminishes significantly at lower concentrations. There is a presumption by some that a threshold exists at the EPA air quality standard, or at the WHO ambient guidelines for criteria pollutants. Most of the studies reported here have estimated linear or log-linear functions suggesting a continuum of effects down to the lowest levels, and have not specifically identified a threshold level. When efforts have been made to identify a threshold, little conclusive evidence has been found that one exists. In fact, many recent epidemiologic studies report an association between air pollution and health at ambient concentrations at or below the current federal standard. The former Administrator of the EPA has stated, in a heterogeneous population it is unlikely that, for any pollutant, there will be a single scientifically defensible threshold applicable to all people. Instead, there will be a series of thresholds for different sensitive populations and a threshold of zero for some people. Therefore, for this report, we calculate the effects of bringing pollution down to alternative standards, without taking a position on what would happen at even lower pollution levels.

A basic assumption of the model is that the association between air pollution and health estimated in the cited studies can be applied to estimate the health impact in Jakarta. These studies show that when the readings at fixed site monitors change, there is a change in the observed incidence of many health effects. Although the monitors do not measure actual exposures, they do provide a general measure of air quality which is obviously related to ultimate exposure. The use of these results implicitly assumes a similar distribution of baseline factors - health status (e.g., incidence of chronic disease), chemical composition of pollutants, occupational exposures, seasonality, time spent out of doors, general activity and that results from other studies can be applied to the study area. As described in greater detail in Section V, since the baseline health status in developing countries tends to be poorer than that experienced in the western, developed world, this assumption will likely lead to an underestimate of the more severe health outcomes. Another source of underestimation will be present since the population is assigned a pollution concentration based on their residential location. Effects of air pollution will be higher if the person commutes to the central business district and the subsequent higher exposures are incorporated into the analysis. Therefore, the quantitative assessment of health effects presented below is likely to be underestimates.

### *2.2 Review of health effects and provision of dose-response functions*

For this report, dose-response functions have been identified and adapted from published epidemiologic and economics literature. These functions allow the estimation of the change in health effects that would be expected to occur with changes in ambient pollution levels. For each health effect, a range is presented within which the estimated effect is likely to fall. The central estimate is typically selected from the middle of the range reported in a given study, or is based on the most recent study using the most reliable estimation methods available. When several different studies are available for a given health effect, the range reflects the variation in results observed across the studies. When only one study is available, the range is based on the statistical confidence that can be placed on the reported results.

The reported epidemiologic investigations involve two principal study designs: statistical inference based on time-series and cross-sectional data sets. Time-series analysis examines changes in a health outcome within a specific area as air pollution levels fluctuate over time. A cross-sectional analysis compares the rate or prevalence of given health outcomes across several locations for a given point in time. The time-series studies have the distinct advantage of reducing or eliminating the problems associated with confounding or omitted variables, a common concern in the cross-sectional studies. Since the population characteristics are basically constant over the study period, the only factors that may vary with daily mortality are environmental and meteorologic conditions. In general, researchers are able to more easily elicit tie effects of air pollution and weather on health in the time-series studies. Therefore, this review focuses primarily on time-series studies. The use and extrapolation of results from time-series analysis, however, is predicated on its applicability to other areas and for ocher time periods.

### III. DEVELOPMENT OF DOSE-RESPONSE ESTIMATES

#### *3.1 Particulate Matter*

Epidemiologic studies provide dose-response relationships between concentrations of ambient particulate matter and several adverse health outcomes including: mortality, respiratory hospital admissions, emergency room visits, and restricted activity days for adults, lower respiratory illness for children, asthma attacks, and chronic disease. Among these studies, statistically significant relationships have been found using several alternative measures of particulate matter, including TSP, fine particles (particles less than 2.5 microns in diameter), British smoke, coefficient of haze (COH) and sulfates.

##### *3.1.1 Respiratory Hospital Admission*

Plagiannakos and Parker used pooled cross-sectional and time-series data for nine counties in their study for the period 1976 to 1982 Southern Ontario, Canada. A statistically significant relationship was found between the incidences of hospital admissions due to respiratory diseases (RHA). Additional evidence for an effect of particulates on hospital admissions is provided by a study by Pope.' In this study a statistically significant association was found between monthly RHA, including admissions for pneumonia, asthma and bronchitis, and monthly average PM10 in two valleys in Utah studied between 1985 and 1989. Ozone concentrations were close to baseline during the winter seasons when both PM10 and RHA were elevated so the effect appears to be mostly related to particles.

##### *3.1.2 Emergency Room Visits*

Samet et al., analyzed the relationship between emergency room visits (ERV) and air pollution levels in Steubenville, Ohio, an industrial town in the mid western United States. Daily ERV (mean 94.3) at the primary hospital in the area were matched with daily levels of total suspended particulates (TSP), sulfur dioxide levels, and nitrogen dioxide levels for March, April,

October, and November of 1974 through 1977. Daily ERV were regressed on maximum temperature and each of the pollutants in separate runs. The particulates and sulfur dioxide coefficients were statistically significant in separate regression, but these measures were highly correlated.

### *3.1.3 Restricted Activity Days*

Restricted activity days (RAD) include days spent in bed, days missed from work, and other days when activities are significantly restricted due to illness. Ostro examined the relationship between adult RAD in a two week period and fine particles (FP. diameter less than 2.5 microns) in the same two week period for 49 metropolitan areas in the United States. The RAD data were from the Health Interview Survey conducted annually by the National Center for Health Statistics. The FP data were estimated from visual range data available for airports in each area. Since fine particles have a more significant impact on visual range than do large suspended particles, a direct relationship can be estimated between visual range and FP.

### *3.1.4 Lower Respiratory Illness in Children*

Estimates of lower respiratory illness in children are based on an analysis by Dockery et al., of children in six cities in the United States. The study related TSP, PM15, PM2.5 and sulfate levels to the presence of chronic cough, bronchitis, and a composite index of respiratory illness (prevalence of cough, bronchitis, or respiratory illness) as measured during health examinations of samples of children in each city. A logistic regression analysis was used to estimate the relationship between the probability of an illness being present and the average of the 24-hour mean concentrations during the year preceding the health examination. Due to the likely overlap of the health endpoint measures, only the results for bronchitis is used, noting that this could include chronic cough or a more general respiratory illness. The results are applied to the population age 17 and below (17.07 percent of the total population)

### *3.1.5 Asthma Attacks*

Several studies have related air pollution to increases in exacerbation of asthma. For example, in a study of asthmatics in Los Angeles, Whittermore and Korn reported a relationship between exacerbations of asthma and daily concentrations of TSP and ozone using logistic regression analysis. Also, Ostro et al. recently reported an association between several different air pollutants, including sulfates, and increases in asthma attacks among adults residing in Denver. Additional evidence for an effect of particulate matter on asthmatic children is provided by Pope et al., This study examines the effects of air pollution on a clinic-based sample and from a school-based (and relatively untreated) sample. Associations were reported between particulate matter, measured as PM10, and both respiratory symptoms and the use of medication.

### 3.1.6 Respiratory Symptoms

Respiratory symptoms are an additional measure of acute effects of air pollution. Results of Krupnick et al., can be used to determine the effects of particulate matter. This study examined the daily occurrence of upper and lower respiratory symptom among a panel of adults in Southern California. A Markov process model was developed to determine the effects of air pollution on health which incorporated the probability of illness on the prior day and controlled for autocorrelation. Among the pollutants examined independently, coefficient of haze (COH) was found to be statistically associated with the probability of reporting a symptom ( $b=0.0126$ ,  $s.e. = .0032$ ). Data from the study suggest a ratio of COH (units/100 ft) and TSP of 0.116. The marginal effect of COH was calculated by incorporating the stationary probabilities as described in the paper.

### 3.1.7 Chronic Bronchitis

Recent epidemiologic studies have related long-term exposure of air pollution to a higher prevalence of chronic respiratory disease or significant decrements in lung function. For example, Detels et al., found that residents living in the Los Angeles air basin who were exposed over a long period of time to relatively high levels of particulates and oxides of sulfur and nitrogen had significantly lower lung function than a cohort less exposed. Abbey et al., conducted a study on 6,600 Seventh Day Adventists, nonsmokers who had lived for at least 11 years in California. In this study, participants above age 25 ( $n=3914$ ) were matched with 10 years of exposure to ambient pollutants based on their monthly residential location.

### 3.2 Sulfur Dioxide

Effects of sulfur dioxide (SO<sub>2</sub>) on the respiratory system have been observed after either short term (less than one hour average) or longer term (24-hour average or longer) exposures. Several recent epidemiologic studies indicate that changes in 24-hour average exposure to SO<sub>2</sub> may affect lung function, the incidence of respiratory symptoms and diseases, and risks of mortality. These studies have been conducted in different geographic locations and climates, and with different populations and co varying pollutants. Although many of these investigations also indicate that particulate matter or ozone was associated with these adverse health outcomes, several studies appear to show an effect of SO<sub>2</sub> alone. Furthermore, in some of the publications reporting an effect of both SO<sub>2</sub> and particulates, they are highly correlated, but in others, the correlation of the daily levels is only weak to moderate. Thus, it is possible to infer an effect of SO<sub>2</sub> or a sulfur species highly related to SO<sub>2</sub>.

TABLE II: EFFECTS OF 10 PGLM3 CHANGE IN SO<sub>2</sub> CONCENTRATIONS

	Central Estimate	Higher Estimate
Mortality(percent change)	0.48	1.21
Respiratory Symptoms 1,000 child/year	0.18	0.26
Chest Discomfort/adult/year	0.10	0.15

### 3.3 Ozone

Ozone is the primary component of photochemical smog. As such, it has been associated with several adverse respiratory outcomes including increased upper and lower respiratory symptoms, eye irritation (oxidants), restrictions in activity, and exacerbation of asthma. Most of the evidence of the effects of ozone is derived from clinical studies in which subjects are exposed to a known amount of ozone in a controlled setting. For example, healthy individuals may be exposed to moderate levels of ozone in a chamber while engaging in moderate exercise. Unfortunately, these studies usually focus on changes in lung function and less so on increases in symptoms. Also, it is difficult to develop dose-response functions from some of these studies or extrapolate from their findings to the free-living population. However, several epidemiologic studies are available and provide a basis for dose-response estimates.

#### 3.3.1 Respiratory Hospital Admissions

Current evidence indicates that ozone may be associated with hospital admissions related to respiratory disease (RHA)<sup>35</sup>- This possibility is supported by findings from panel studies of asthmatics indicating that exacerbations occur in response to ozone. Clearly, some of these exacerbations may result in either emergency room visits or hospital admissions. Unfortunately, because of the high covariation between ozone and other pollutants in the summer when most of the studies have been undertaken, it is difficult to determine the effect on RHA attributable to ozone alone. However, by using information from several studies, it is possible to begin to apportion the effects of the different pollutants. Thurston et al.<sup>53</sup> found a significant association between RHA and both ozone and sulfates in New York City (the Bronx) and Buffalo in the summer 1988- In this analysis, corrections for autocorrelation and day-of-week effects were made. At the mean, the effect of ozone was approximately twice the effect observed for sulfates. Burnett et al.<sup>4</sup> also reported a statistically significant association between hospital admissions and both ozone and sulfates in Ontario, Canada for the years 1983 through 1988. Their findings suggest that the ozone effect was approximately 3 times that of sulfates, based on a regression equation that included both pollutants. Therefore, it is reasonable to apportion the effects of RHA based on the relative coefficients in Thurston et al. The average of the coefficients for the two cities in that paper is 2L .3 RHA per day/ million/ppm ozone, which becomes the central estimate, with a standard error of 10.9.

TABLE III: EFFECTS OF 1 PPM CHANGE IN OZONE

	Central Estimate	High Estimate
Hospital Admissions/persons	0.0077	0.012
Minor Restrictions in Activity/person	34.0	51.0
Respiratory Symptoms/person	54.75	96.6
Eye Irritation/adult	26.6	29.9
Asthma Exacerbation/asthmatic	68.44	189.8



#### IV. CONCLUSION

The rate of pollution is increasing day by day due to the development of technology and various processes which gives us an inevitable condition is inevitable. It is necessary to keep an eye on the pollution levels and its effects by comparing it with certain standard level so as to make sure that the pollution is subjugated below the parlous level. The above literature gives a brief idea about the pollution levels of certain hospital in an area which could be used further as a reference for other pollution sources to limit the emission below the perilous stage. The state of jeopardy can be brought under control if we follow standardization in pollution levels and give us prior idea about preempting the recurrence of the particular pollution sources. On the whole, pollution is going to be a vital threat for the living beings in our planet and it will ruin various ecosystems explicitly if it is not taken into our serious consideration.

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