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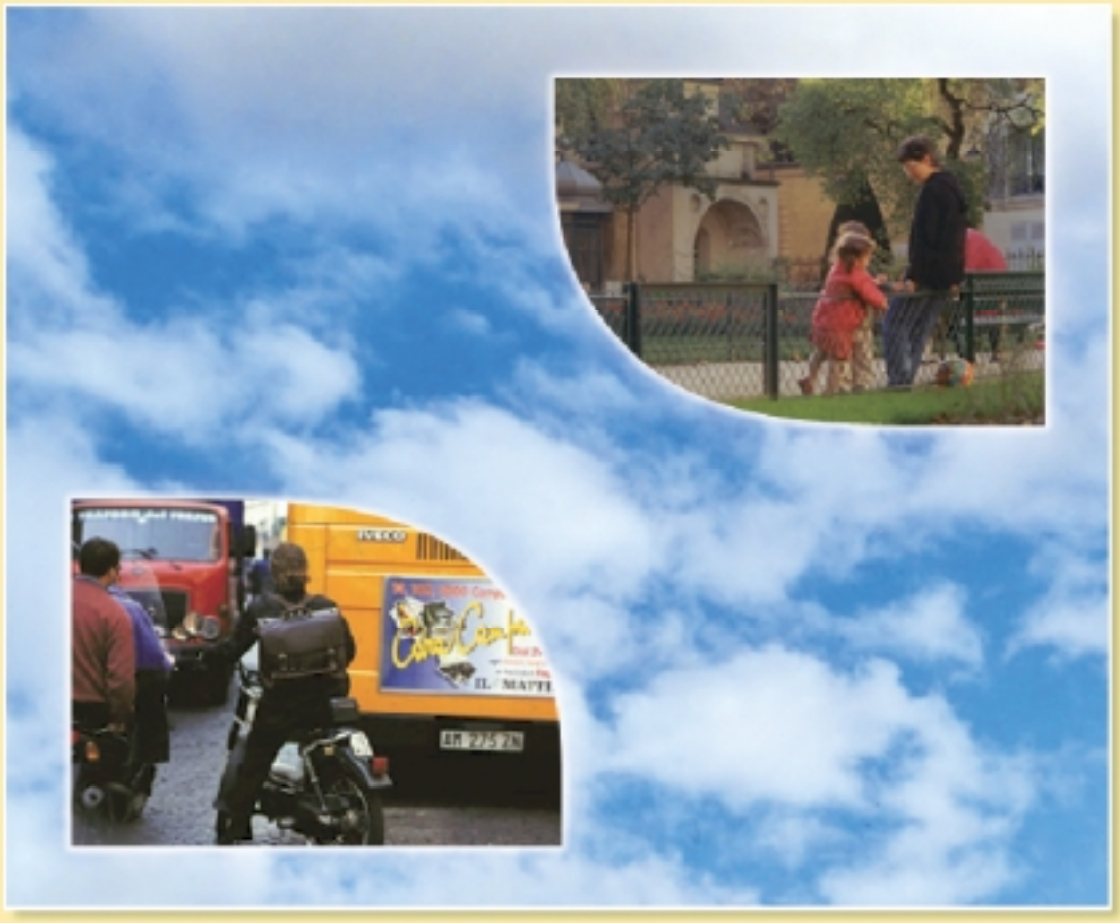
HEALTH IMPACT ASSESSMENT OF AIR POLLUTION IN THE EIGHT MAJOR ITALIAN CITIES



HEALTH IMPACT ASSESSMENT OF AIR POLLUTION IN THE EIGHT MAJOR ITALIAN CITIES

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ABSTRACT

The report contains the health impact assessment of urban air pollution in the eight major Italian cities; it gives estimates of mortality, morbidity and numbers days of restricted activity associated with air pollution level. The report illustrates the methodology, discusses scientific uncertainty and implications for findings as well as for the need for further research.

Due to the methodological discussion and to its practical application in quantifying health effects of air pollution exposure, the report is also recommended as a handbook for local health officers.

The case study and methodological tools can support Member States in implementing and developing environmental health policies. The dissemination of the report, among health officers and local government officers in Europe will increase awareness of air pollution related health effects and improve the knowledge of management of air quality data for air pollution monitoring and exposure assessment.

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Foreword

This report describes a research project carried out by the European Centre for Environment and Health of the World Health Organization to assess the health impact of urban air pollution in Italy. The project was based on the eight cities in Italy with a population greater than 400 000 people and was developed in two phases, beginning in 1998 with a financial contribution from the Italian Ministry of Environment. The first phase of the project gathered data on urban air pollution, and the second phase assessed its health impact.

In the first phase, the difficulties of using the data as recorded in the local databases became apparent: the collection and storage methods differed from city to city and the classification criteria of the air quality monitoring stations are not standardised. Although the air quality monitoring networks (i.e. the location and number of the monitoring stations) have been designed to monitor a large number of pollutants, the data have not been extensively used. Thus, an expert group ("Itaria") was set up to support the process. The group provided hourly data for a six-year period and informed the choice and weight of monitoring stations for exposure assessment. The work of the group is still in progress and the association itself is currently a very important consultancy working group for issues relating to air quality monitoring.

The second phase of the project (the health impact assessment of urban air pollution) has also been based on a collaborative effort, involving a large group of advisors and consultants.

As a result, the report draws on existing expertise and literature to quantitatively estimate the current health effects attributable to air pollution in the eight major Italian cities, using PM10 as a proxy for concurrent exposures to different pollutants. These eight cities constitute 15% of the population of Italy and include: Genoa, Turin, Milan, Bologna, Florence, Rome, Naples and Palermo.

Executive summary

Urban outdoor air in western countries is polluted with a variety of noxious agents that can result in increased morbidity and mortality, through short- and long-term exposures. Many studies have demonstrated that PM10 (particulate matter less than 10 microns in diameter) is associated with a wide range of adverse health outcomes. Given the high correlation between pollutants PM10 may also serve as a surrogate measure for other pollutants including very fine particles (less than 2.5 microns) and a host of traffic-related toxins. A recent study conducted in Austria, France and Switzerland has estimated a large impact on population health with, for example, some 40,000 deaths per year attributable to PM10 in the three countries.

In 1998, the WHO European Centre for Environment and Health - Rome Division was asked by the Italian Ministry of Environment to carry out a health impact assessment study for the eight largest cities in Italy: Turin, Milan, Bologna, Genoa, Florence, Rome, Naples and Palermo. The population in 1991 was 8.3 million, 15% of the total national population. Mean concentration PM10 levels for 1998-99 were determined in each city using the existing monitoring network; yearly average PM10 concentrations ranged between 44.4 and 53.8 $\mu g/m^3$, with a population-weighted mean value of 52.6. Mortality, hospital admission and morbidity were studied; data were obtained from national statistics sources. Dose-response coefficients for exposure to PM10 were derived from published literature, including studies conducted in Italy, through meta-analysis. These coefficients were used to calculate the rates that would prevail at a given arbitrary reference concentration level chosen *a priori*. These rates were then compared with the observed ones to derive the proportion and the number of cases per year attributable to exposure to PM10 in excess of the reference level. Two reference PM10 levels were used, 20 and 30 $\mu g/m^3$. These are values realistically achievable through limitations on emissions and the former is the limit for the EU countries for 2010 (while the limit for 2005 is $40\mu g/m^3$) (EC, 1999). The analysis is based on conservative assumptions and estimates of the number of cases attributable to PM10 exposure are likely to describe only part of the overall air pollution health impact (for example, effects of other pollutants are not included). The methodology of analysis was similar to that used by Kuenzli et al, although they used $7.5\mu g/m^3$ as reference and a higher dose-response coefficient for mortality.

Results indicated that: 4.7% of mortality (95%CI: 1.7 – 7.5) is attributable to PM10 concentrations higher than 30 $\mu g/m^3$. This proportion increases to 7.0% using 20 $\mu g/m^3$ as reference. The numbers of yearly attributable deaths are 3,472 and 5,108 respectively. Attributable hospital admissions are in the same order of magnitude, with some 4,500 cases per year (combining respiratory and cardiovascular causes; almost 7,000 with the lower reference value). Tens of thousands of attributable cases of childhood bronchitis, and asthma exacerbation cases were estimated, as well as millions of days of restricted activity and episodes of respiratory symptoms.

These data show that the societal burden of urban air pollution in Italian cities, in terms of mortality, morbidity and hospitalisation, is very high. These figures should be interpreted with caution when estimating the direct health benefits of reducing PM10 concentrations. Some long-term effects are included in these estimates and the relative importance of risk factors other than air pollution might change over the time necessary to achieve the expected health

gains. On the other hand, the true global impact of air pollution is likely to be larger, since only one pollutant has been considered in this study, analysis has been limited to selected known health outcomes, and conservative dose-response relationships have been employed.

The main source of PM10 in Italian cities is motor vehicle traffic, including diesels and two-stroke motorcycles. In light of these evaluations, intervention to curb motor vehicle traffic volume in urban areas is warranted. The health benefits of transport policies to reduce air pollution would be further increased by the consequent reduction in road accidents, noise, and psychosocial effects, and other indirect beneficial consequences (such as more walking and cycling) would ensue. Compliance with the forthcoming European Union standard of $20 \mu g / m^3$ will result in substantial heath gains in large Italian cities, but the limit will be in place no sooner than 2010.

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1. Introduction

Over the last decade, several dozen scientific studies have been published indicating an association between air pollutants to which people are routinely exposed and a wide range of adverse health outcomes. These studies have been undertaken at usual (i.e., non-episodic) levels of air pollution in cities throughout the world. As a consequence, they involve a wide range of climatic conditions, human behaviours, baseline health levels, and exposure conditions. The majority of these studies have used observational epidemiology where associations between air pollution and readily observed outcomes such as mortality or hospital admissions can be statistically documented.

One pollutant, particulate matter (PM), appears to be most consistently associated with adverse health outcomes ranging from acute respiratory symptoms to premature mortality.

PM includes particles directly emitted into the air such as diesel soot, agricultural and road dust, and emissions from mechanical scrapings. PM is also produced through photochemical reactions involving pollutants that are a by-product of fuel combustion from motor vehicles, power plants and industrial boilers.

Taken together, the studies provide strong evidence of a causal association between PM and health and provide a quantitative basis from which to generate a risk assessment. Given the widespread presence of PM air pollution in Western cities, large groups of people are exposed, and the overall burden on health, as estimated in North America and some parts of Europe, is severe. The objective of this study was to conduct a risk assessment of PM for large Italian cities, where high concentrations of various air pollutants have been documented in recent years.

This report is organized as follows: Section 2 describes the rationale and provides background information for the field of quantitative estimates of health effects of PM. Section 3 outlines the available data on exposure and baseline population health for the eight Italian cities, and describes the derivation and use of the dose-response information from the epidemiological studies. Section 4 summarizes the results of the quantitative estimates applied to Italy. Sections 5 and 6 provide the conclusions and detail the uncertainties and potential biases in the study, outlining future research that could be undertaken to reduce them.

2. Rationale

2.1. Prior studies of risk assessment

There is abundant evidence, accumulated over the last two decades, that exposure to current levels of ambient air pollution has significant implications for public health (U.S. EPA, 1996, WHO 1999). In light of this fact, government decisions about the levels of pollution control that will be mandated have become increasingly important. More informed decisions about pollution control require information about the expected benefits of such efforts. This can aid in prioritizing among alternative pollution control levels and strategies. It is also then possible for public health agencies to compare the benefits obtained from controlling air pollution with other social investments aimed at protecting or improving public health.

Early efforts at estimating the health and economic benefits of reducing air pollution relating to alternative ambient air pollution concentrations were undertaken by the United States Environmental Protection Agency (EPA) (EPA, 1994). Broad estimates of the health benefits of controlling particulate matter or ozone were provided for both the United States as a whole and for the pollution control plans under consideration in Southern California (Natural Resources Defense Council, 1996, American Lung Association, 1996; Hall et al. 1992; Krupnick and Portney, 1991). Methodology was developed to apply many studies conducted primarily in the United States to other parts of the world, where less epidemiological information on dose-response was available (Ostro, 1994, 1996). Epidemiological research over the last few years has provided additional evidence on the health effects of air pollution and provides a rich basis for predicting several adverse outcomes that are associated with exposure to air pollution, especially particulate matter. Using these more recent studies, conducted in both the United States and Europe, Sommer et al. (1999) provided estimates of the health and economic consequences of air pollution associated with road traffic in Austria, France and Switzerland.

2.2. Use of epidemiology

Air pollution epidemiology studies typically involve the determination of a statistical association between the frequency or probability of a given health outcome and concurrent air pollution concentrations. Usually, the pollution concentrations are measured at fixed site monitors located in proximity to the study population in the course of their usual activities and consequent pollution exposures. Thus, extrapolations between species or to lower doses, necessary for most toxicological or human clinical studies, are not required to develop dose-response functions. The reported epidemiological investigations are mainly based on three types of study designs: time-series, cross-sectional and cohort.

Time-series analysis examines changes in a given health outcome over time within a specific area as air pollution levels fluctuate. An example of this study design would be daily observations of emergency room visits and air pollution in a community over several years.

A cross-sectional analysis compares differences in health outcomes among individuals living in areas or cities with different pollution levels at a selected point or period of time. This would include, for example, studies that compare chronic bronchitis rates and associated air pollution concentrations at several locations at a single period of time.

Cohort studies follow up a defined population over time and compare occurrence of disease by exposure levels. They are suited to studying long-term health effects of air pollution.

The primary advantage of time-series studies is that many factors that could potentially confound the association between air pollution and health are constant during the study period and are therefore not relevant. For example, while daily counts of emergency room visits are being investigated, most relevant population characteristics (e.g., age, smoking habits, occupational exposure and diet) do not change on a daily basis; they can be considered constant over the study period. As a result, they will not influence the observed statistical association between air pollution and health. The only factors likely to vary with daily mortality and morbidity are environmental and meteorological conditions, which typically are accounted for in the statistical analysis. Therefore, for many of the health endpoints, dose-response estimates are based primarily on time-series studies. However, carefully conducted studies according to a cross-sectional design are also drawn upon for dose-response estimates. In addition, information on long-term mortality following exposure to air pollutants is largely based on longitudinal cohort studies (Pope et al., 1995; Abbey et al., 1995).

One of the issues in the use of time-series studies is whether those conducted in one city are applicable to other cities with potentially different characteristics. Fortunately, studies of air pollution epidemiology have now been undertaken in dozens of cities around the world. The weight of evidence suggests that it is reasonable to extrapolate the findings from the United States and Western Europe to the Italian cities under study. In addition, several air pollution studies have now been undertaken in Italy. Since the findings of these studies are generally similar to those from other parts of the world, they provide additional support for the extrapolation of previous studies to Italy.

2.3. Uncertainties in studies of risk assessment

There are several uncertainties and limitations associated with using epidemiological evidence to predict how health may change as a result of changes in ambient pollution concentrations.

A key assumption is that the air pollution - health effects relationship is causal. Then, the observed association can be used to predict how changes in pollution concentrations will influence the incidence of health effects. Observational epidemiological studies are able to demonstrate whether a statistically significant relationship exists between health effects and pollution concentrations, but the studies do not prove conclusively that the relationship is causal. It is possible that a statistically significant relationship is due to some unidentified factor correlated to pollution concentrations. However, the likelihood of causation is strengthened when epidemiological results are replicated by similar findings in

different studies and when multiple health outcomes appear to be affected. This is the case of PM, where numerous studies have found a similar magnitude of effect for mortality, and have also linked it with a wide spectrum of adverse health outcomes. At present, there is limited laboratory and clinical evidence regarding the health effects associated with exposure to PM, and biological mechanisms that underlie the observed epidemiological associations have not been firmly established.

There may also be uncertainty about possible non-linearity or thresholds in the concentration-response functions. It remains uncertain whether there is a threshold concentration below which health effects no longer occur, or whether the slope of the concentration-response function diminishes significantly at lower concentrations. Available epidemiological studies have not usually addressed the question of thresholds, and epidemiological data are not always sufficient for making such a determination. Many recent epidemiological studies show a statistically significant association between PM concentrations and health endpoints over ranges of concentrations that are typical of current conditions in most Italian cities. For this assessment the assumption that concentration-response functions are linear down to each of the target PM levels considered is adopted.

Another source of uncertainty is error in measuring exposure. For most epidemiological studies of air pollution, exposure is based on readings from nearby fixed-site monitors. To the extent that there are errors in measuring pollution concentrations to which individuals are actually exposed, additional errors may be introduced into the analysis. However, most examinations of this measurement error suggested that it is more likely to result in an underestimation of the effects of pollution (Thomas, 1994, Zeger et al., 1999).

2.4. Measures of air pollution

Particulate matter air pollution is typically monitored in the ambient air as TSP (total suspended particulate) or PM10 (particles 10 microns or less in diameter), or as black smoke. Particles included in PM10 or even smaller particles such as PM2.5 (fine particles that are below 2.5 microns in diameter) are of greater health concern than particles of all sizes (TSP), since smaller particles are more likely to penetrate into the deep lung. Currently, the U.S. EPA uses national ambient air quality standards for PM10 and PM2.5, while the World Health Organization established air quality guidelines (AQGs) for both measures of PM (WHO, 1999). The European Commission has also proposed limit values for PM10 to be adopted by the 15 European Union countries (EC, 1999). By far, most of the epidemiological studies undertaken during the last decade have implicated PM as a pollutant of concern. In many cases, however, PM may be serving as a surrogate measure for the complex mix of particles and gases that result from fuel combustion from automobiles or power generators.

For these reasons, the estimates provided in this report rely on effects of PM only, in order to preclude double counting of health effects related to air pollution. As a result, it is likely that the total effect of air pollution is underestimated. For example, there is evidence of effects of ozone on hospital admissions for respiratory disease, exacerbation of

asthma, and even mortality (Spix et al., 1998; Touloumi et al., 1997; Burnett et al., 1997a, Ostro et al., 1993).

2.5. Assumed standards

There is considerable latitude concerning the choice of a "target" concentration, i.e. the reference value beyond which attributable risks are estimated. Currently, the existence of threshold level PM10, below which no effects related to exposure are expected to occur, has not been identified. There is also little evidence, based on reviewing studies conducted at a wide range of air pollution concentrations, that the slope of the dose-response function diminishes significantly at lower concentrations (U.S. EPA, 1996). Most of the epidemiological studies have estimated linear or near-linear functions that suggest a continuum of effects down to the lowest PM levels observed in the study sample. For example, for mortality, most studies report a linear association between the relative risk in a population (percent increase in mortality) and the concentration of PM10. When efforts have been made to identify a threshold, little conclusive evidence has been found that one exists (Ostro, 1984). An analysis conducted by Cakmak et al. (1999) indicated that if threshold concentrations did exist, the current statistical models are sufficiently robust to be able to identify them. Many recent epidemiological studies show a consistent association between particulate matter and health effects across the entire range of measured particulate levels, including those well below the current U.S. standards for particulate matter. For example, Schwartz and Dockery (1992a, 1992b) have found that the observed relationship between mortality and particulate matter in two eastern U.S. cities is similar across all four quartiles of daily particulate matter. The lowest 5 to 10 percentile concentrations of PM in these studies were in the range of 30 to 40 $\mu g/m^3$ TSP (24-hour average) or roughly 15 to 20 $\mu g/m^3$ as PM10. Likewise, other studies involving PM10, including those carried out in Santiago, Chile (Ostro et al., 1996) and the Utah Valley (USA) (Pope et al., 1992) observed continuous effects down to 10 $\mu g/m^3$.

3. Methods

The study is based on the existing epidemiological literature to quantitatively estimate the current health effects attributable to air pollution in the eight major Italian cities (those with a resident population of more than 400 000 people), using PM10 as an indicator of urban air quality and a proxy for concurrent exposures to different pollutants. These eight cities are Rome, Milan, Florence, Bologna, Palermo, Genoa, Naples, and Turin and host 15% of the population of Italy (see Appendix A1).

This study draws heavily on the methodology of Ostro (1996) and on the dose-response estimates provided by Künzli et al. (1999). The results provide estimates of the range of effects that may be prevented by reducing current levels of PM10.

Three factors are required for the quantification of the health burden of air pollution. The first is the dose-response functions, obtained from the epidemiological studies. These functions indicate the expected change in a given outcome per unit change of pollutant (PM10, in this case). The second factor in the quantification process is the exposure of the population under consideration, more specifically the change in air quality (i.e., the difference between observed levels and some target). Thirdly, the size of the population that will be exposed to this change in air pollution needs to be determined, together with the frequency of occurrence of the health outcome.

The proportion and the absolute number of adverse health events attributable to exposure above the commonly accepted international ambient air quality standards of air pollution is derived on the basis of these three factors. Detailed description of the methods of analysis is given in 3.2.4.

The adverse health outcomes used in this analysis include: mortality (all causes except external causes, adults) and morbidity (acute bronchitis, hospital admissions, restricted activity days for adults and asthma attacks). Some evidence linking long-term exposure to PM to increases in cancer is also available (Abbey et al. 1999, Pope et al., 1995). However, cancer effects are not studied separately in this analysis.

Data on pollution levels were available for the period 1994-99 and were reviewed for completeness and comparability. PM10 concentrations for risk assessment were estimated as average for the biennium 1998-99. Data from the cities provided information on population, mortality and morbidity and pollution levels that were then combined with dose-response information from epidemiological studies. Finally, estimates are provided for the total number of cases for the considered health effects attributable to exposure above the selected reference levels described in 3.2.4. The proportion and number of cases attributable to PM10 were estimated for one calendar year.

3.1. Dose-response functions

There is currently a considerable number of studies providing a solid basis for risk assessment. The central estimate of the necessary relative risks and 95% confidence bounds were derived using a meta-analytic approach. This approach incorporates information from all relevant studies by weighting each study's estimate by its associated uncertainty, i.e., the

standard error of the effect estimate. In order to allow for the uncertainties referred to in 2.3, conservative dose-response coefficients were selected for estimating the attributable number of cases. For example, the relative risk for mortality was taken to be the lower bound of the confidence limit around the overall relative risk estimated from the meta-analysis (see 3.1.1 for details).

Both mortality and morbidity outcomes are quantified in this report. For morbidity, effects from both acute and chronic exposure are included. Short-term effects include hospital admissions for cardiovascular and respiratory disease, asthma symptoms, restricted activity days, and acute respiratory symptoms. To avoid double counting, numbers of cases of the more severe endpoint are subtracted from the less severe cases. For mortality, the assessment is based on estimates of the change in mortality from long-term or chronic exposure to PM. However, studies of short-term or acute changes provide additional and compelling evidence of an effect on mortality.

Since different measures of PM are used in the literature, published estimates of relative risks were converted to PM10 equivalent estimates. It was assumed that PM10 = 0.55 of total suspended particles or TSP, that PM2.5 = 0.5 of PM10, and that PM10 equals PM13 or PM15. Analysis of the size distribution of PM suggests that there are not many particles, by weight, between PM10 and PM15 (U.S. EPA, 1996).

Table 1 shows the health endpoints considered and summarizes the dose-response functions used in this report. Details on derivation of these relative risks are given in the following sections.

Table 1 **SUMMARY OF RELATIVE RISKS ESTIMATED BY THE PRESENT STUDY FOR THE HEALTH OUTCOMES CONSIDERED**

Cause	*Central estimate*	*LL 95%*	*UL 95%*	*Notes*
Mortality	1.026	1.009	1.043	Adults age 30+
Hospital admissions for CVD causes	1.009	1.006	1.013	
Hospital admissions for respiratory disease	1.016	1.013	1.020	
Acute bronchitis	1.306	1.135	1.502	Children <15
Asthma exacerbation	1.051	1.047	1.055	Children <15
Asthma exacerbation	1.004	1.000	1.008	Adults 15+
RAD	1.094	1.079	1.109	Adults 20+
Occurrence of respiratory symptoms	1.07	1.02	1.11	

3.1.1. Mortality

Evidence from previous studies

Early studies of 14 winters in London, England showed statistical associations between daily change in PM, measured as black smoke, and daily changes in mortality (Ostro, 1984; Schwartz and Marcus, 1990). These efforts indicated that mortality was associated with air pollution over the entire range of ambient concentrations, not only during the high episodes associated with the earlier London winters. Since these studies, several dozen others conducted throughout the world using multiple regression analysis have indicated an association between acute exposures to PM (measured as TSP, PM10, PM2.5, black smoke, and PM13) and mortality. For example, in the United States, associations have been found in cities including, but not limited to, Philadelphia, Pennsylvania (Schwartz and Dockery, 1992a), Steubenville, Ohio (Schwartz and Dockery, 1992b), Santa Clara County, California (Fairly, 1990), Birmingham, Alabama (Schwartz, 1991), and Utah Valley, Utah (Pope et al., 1992). In Europe, the cities involved include Athens, Barcelona, Bratislava, Krakow, Lodz, London, Lyons, Milan, Paris, Poznan, Rome, and Wroclaw (Katsouyanni et al., 1997; Michelozzi et al., 1998; Zmirou et al., 1998). Also, positive associations have been reported from data in cities in less developed countries, such as Bangkok, Thailand (Ostro et al. 1999) and Santiago, Chile (Ostro et al., 1996). These studies used daily counts of total mortality (excluding accidents and homicides), as well as cardiovascular- and respiratory-specific-mortality, as their outcome measure. Typically, they examined the sensitivity of the regression results to alternative model specifications and carefully controlled for potential confounders such as weather, seasonality, day of the week, and other pollutants. Taken together, the studies suggest a fairly linear association between the PM10 and the percent increase in mortality. This association appears to exist over a PM10 range of from 20 $\mu g/m^3$ to over 400 $\mu g/m^3$ (24-hour average).

Risk estimates used for present study

Several meta-analyses of these studies suggest that after converting the alternative measures of particulate matter used in the original studies to PM10, the effects on mortality are very consistent (Ostro, 1993; Pope and Dockery, 1994; Schwartz, 1994). Specifically, the mean effect of a 10 $\mu g/m^3$ change in PM10 indicated by these studies is approximately 1.0%, with a range of effects of 0.5% to 1.6%. While the use of daily data and acute exposure has many statistical advantages (e.g., reducing confounding and exposure measurement error), the quantitative implications are not without uncertainty. For example, there is uncertainty regarding the extent of the prematurity of mortality (i.e., reduction in life expectancy) resulting from acute exposure. Some of the deaths may merely be displaced by a few days or weeks. It appears, however, that for many of the deaths, particularly those related to cardiovascular outcomes, the prematurity is much greater and may involve several years or more (Schwartz, 1999; Zeger et al. 1999). Although there is a considerable body of evidence indicating an association between short-term changes in PM and subsequent mortality, these studies are not used quantitatively here because the reduction

in life expectancy is unclear. Instead, this assessment will use studies involving long-term exposure to air pollution and measured reductions in life expectancy.

Long-term exposure studies use a prospective cohort design in which a sample is selected and followed over time in many locations. For example, Dockery et al. (1993) published results for a 15-year prospective study based on 8,000 individuals in 6 cities in the United States. Pope et al. (1995) published results of a 7-year prospective study based on 550,000 individuals in 151 cities in the United States. These studies use individual-level data so that other factors that have an impact on mortality can be characterized. Specifically, these studies were able to control for mortality risks associated with differences in body mass, occupational exposures, smoking (present and past), alcohol use, age, and gender. Once the effects of individual-level factors are determined, the models examine whether longer-term citywide averages in PM are associated with differences in life expectancy. Both studies report a robust and statistically significant association between exposure to particulate matter (measured as PM10 or fine particles) and mortality. From these results, it is possible to predict the actual number of years of life lost associated with given levels of air pollution.

To quantify the effects of chronic exposure, estimates from both Pope et al. (1995) and Dockery et al. (1993) were used. The Dockery study suggests a mortality effect of 8.5% per 10 $\mu g/m^3$ of PM10, while the Pope study generates an estimate of 3.8% per 10 $\mu g/m^3$. Following Künzli et al. (1999), the results were combined by weighting each study by the inverse of the variance of the each of the study's estimated coefficient. In this manner, the study with less uncertainty plays a larger role in the central estimate and confidence intervals. The results indicate a relative risk of 1.043 (95% CI = 1.026 – 1.061) for 10 $\mu g/m^3$ of PM10. For the present analysis, there is some uncertainty about the relevant period of exposure, as the latency time and length of the exposure period necessary to generate the observed effects are unclear. It may require 20 years or more of exposure at a given concentration or simply a few years during early childhood. Thus, a more conservative approach to quantification of mortality has been chosen, with recognition that it is more likely to underestimate than to overestimate the effects of long-term exposure to PM10. In addition, Künzli et al. estimated PM10 effects in a population whose majority were exposed to concentrations between 10 and 35 $\mu g/m^3$ (the range on which most information on long term mortality is based), while in the present study the average concentrations are higher. Thus, as this study involves some extrapolation, concentration-response coefficients more conservative than those used by Künzli and colleagues were chosen, to account for such additional uncertainty.

Therefore, the choice made for the central estimate is the lower bound of the relative risk from the meta-analysis of 1.026. The upper bound of the present estimate is assumed to be 1.043, which is the central estimate of the meta-analysis. Assuming a symmetrical range around the central estimate, the lower estimate becomes then 1.009 = 1.026 – (1.043 - 1.026).

It should be noted that the present estimates indirectly include deaths due to cancer, since total mortality is used. Direct associations between long term exposure to PM and increases in lung cancer have been reported by Pope et al. (1995) and Abbey et al. (1999).

However, potential mortality impacts on children were not included in the present estimates. Several cross-sectional studies have reported associations between PM concentrations and neo-natal and infant mortality (Penna and Duchiade, 1991; Bobak and Leon, 1992; Knoebel et al., 1995; Woodruff et al., 1997). Nevertheless, this group of studies appears to be inadequate for quantitative purposes, as uncertainties are large.

Since the original studies only involved adults, the estimates worked out here are applied to adults older than 30, excluding accidental causes. Baseline mortality rates were derived from city-specific data. These data were available for Turin, Milan, Bologna, Florence and Rome. For the remaining three cities for which data were not available, an average of the other cities was used (see Appendix A1).

3.1.2. Morbidity

Dose-response functions and associated relative risks have been developed from studies in the United States and Europe for several morbidity endpoints. Additional risks from PM for hospital admissions, emergency room visits, asthma exacerbation, acute bronchitis in children, restricted activity days, and respiratory symptoms were calculated from studies on acute exposure. Cancer morbidity associated with PM was not estimated because of a lack of studies showing clear associations.

For many health endpoints, multiple studies were available. To combine the study results into one central estimate and associated confidence intervals, a meta-analytic approach similar to that used for mortality was used. Specifically, the overall effect estimate was developed by weighting each single study estimate by the inverse of the variance of the estimated effect. Following Künzli et al. (1999), heterogeneity among the studies was also examined using the Q-test (Petitti, 1994). If the Q-test indicated the studies were fairly similar, a simple weighting or fixed effects model was used. When significant heterogeneity among the studies was apparent, random effect estimates were used based on the method developed by DerSimonian and Laird (1986). This method adds the variance existing between studies to the reported variance within a given study, thereby changing the study weights. This technique can either raise or lower the central estimate of the effect. Ultimately, both European and American studies were pooled to obtain the final estimate of relative risk.

3.1.3. Cardiovascular hospital admissions

Evidence from previous studies

There is evidence from studies conducted in several cities of an association between daily changes in PM and changes in hospital admissions for cardiovascular disease. Following Künzli et al. (1999), four studies conducted in the European cities of Paris, London, Birmingham, and Edinburgh (respectively, Median et al., 1997; Poloniecki et al., 1997; Wordley et al., 1997; Prescott et al., 1988) were selected to calculate a relative risk of 1.013 (95% CI = 1.007 - 1.019) per 10 $\mu g/m^3$. These studies covered all age groups but

different time periods between 1987 and 1995, different cardiovascular outcomes, and different measures of PM (PM10 for Birmingham and Edinburgh and black smoke in the others). Nevertheless, after converting to PM10, the risk estimates were fairly similar across studies.

Three studies from the Unites States and Canada provide additional confirmation of an effect and basis for quantification. Specifically, studies in Detroit (Schwartz and Morris, 1997), Tucson (Schwartz, 1997) and Toronto (Burnett et al., 1997b) were selected. These studies, which all used PM10, provide a joint estimate of 1.008 (95% CI = 1.004 - 1.011) per 10 $\mu g/m^3$. These studies also involved different age groups and different measures of cardiovascular disease.

Risk estimates used for present study

For all studies combined, a fixed effects estimate of 1.009 (95% CI = 1.006 - 1.013) was obtained. To develop baseline data on hospital admissions for cardiovascular disease, regional hospital statistics (see Appendix A2) available for 1997 and 1998 for the cities of Turin, Milan, Bologna, Florence and Rome (with coverage of both public and private hospitals) were used. The average estimates for these five cities were extrapolated to the other cities for which data were not available. All cardiovascular diseases were included (ICD9 390-459), while scheduled admissions and admissions to long-term departments were excluded. The criteria to define non-emergency admissions vary by city, so that the absolute numbers are not directly comparable.

3.1.4. Respiratory hospital admissions

Evidence from previous studies

There is also extensive evidence to indicate that short-term changes in PM affect hospital admissions for respiratory disease. Studies are available from both Europe and the U.S. covering various respiratory endpoints, including all respiratory diseases. From Europe, associations were found in a study combining the results of four cities (London, Amsterdam, Rotterdam and Paris) using similar protocol from the APHEA project (Spix et al., 1998). This effort used hospital admissions for all respiratory diseases for the health endpoint, and black smoke for the PM metric. Additional European studies were conducted by Wordley et al. (1997) and Prescott et al. (1998) for Birmingham and Edinburgh, respectively. These studies used specific respiratory disease outcomes and PM10. The Q test provided evidence of heterogeneity among the studies and the random effects model in general a risk estimate of 1.013 (95% CI = 1.001 – 1.025) per 10 $\mu g/m^3$.

Additional estimates are provided by many studies conducted in the United States and Canada, covering a period from 1986 through 1994, and including Toronto (Thurston et al., 1994; Burnett et al., 1997), Detroit (Schwartz, 1994), Birmingham (Schwartz, 1994), Minneapolis (Schwartz, 1994), New Haven and Tacoma (Schwartz et al., 1995), Spokane (Schwartz, 1996), and Cleveland (Schwartz et al., 1996). PM10 was used in all of these studies and a wide range of ages was included. The Schwartz studies, however, focused on

individuals age 65 and above. Heterogeneity was not present among these studies probably because of similarity in pollution measure and disease coding, and a risk estimate of 1.017 (95% CI = 1.013 – 1.020) was obtained.

Risk estimates used for present study

Combining the U.S., Canadian, and European studies generated a risk estimate of 1.016 (95% CI = 1.013 -1.020) using a random effects model. Baseline data on hospital admissions were derived from regional data for 1997 and 1998 similar to those used for the cardiovascular hospital admissions. All respiratory diseases were included (ICD9 460-519), while scheduled admissions were excluded.

3.1.5. Acute bronchitis

Evidence from previous studies

Several studies indicate an association between annual exposure to PM and the likelihood of chronic bronchitis in children. For example, Dockery et al. (1989, 1996) analyzed data from 6 and 24 U.S. cities, respectively. For each child, the prior one-year exposure to PM was matched with a survey questionnaire asking parents whether their children of 8 to 12 years old had had bronchitis during the preceding 12 months. The first study used PM15 data for the years 1980 and 1981 while the second study used PM10 data for 1988 to 1991. Also, a study from Switzerland (Braun-Fahrländer et al, 1997) reported an association between PM10 and bronchitis among children ages 6 to 15 using data from 1992-1993.

Risk estimates used for present study

A joint estimate from the three studies indicates a risk estimate of 1.306 (95% CI = 1.135 – 1.502) per 10 $\mu g/m^3$. These risk estimates are applied to the population of children below age 15. Baseline estimates were taken from the Italian Studies on Respiratory Disorders in Childhood and the Environment (SIDRIA-ISAAC) conducted in 1995 in five Italian regions (SIDRIA, 1997). A 10.6% prevalence of doctor-diagnosed bronchitis in the last year was observed among a sample of 40,147 children, age 6 to 15 (SIDRIA, unpublished data).

3.1.6. Asthma exacerbations in children

Evidence from previous studies

There have been several panel studies of asthmatics over time conducted in Europe and the U.S. Three (Roemer et al., 1993, Gielen et al., 1997, Ségala et al., 1998) were conducted in Europe during 1990 and 1995 and provide a risk estimate for PM. The Roemer and Gielen studies were conducted in Wageningen and Amsterdam, Netherlands,

respectively and use PM10. The Ségala study was conducted on mild and moderate asthmatics in Paris using PM13. The studies use either asthma attacks or lower respiratory symptoms as measures of asthma exacerbation. The European studies generate a risk estimate of 1.044. Evidence from the U.S. is provided by Pope et al. (1991) in Utah and by Ostro et al. (1995) in Los Angeles. These studies were conducted between 1989 and 1992 using lower respiratory symptoms and shortness of breath as indicators of asthma exacerbation. These two studies generate a risk estimate of 1.051.

Risk estimates used for present study

Combining all of the studies provide a relative risk of 1.051 (95% CI = 1.047 – 1.055) for a 10 $\mu g/m^3$ change in PM10.

These estimates are provided for children under age 15. Baseline estimates were assumed to be equal to those in Künzli et al. (1999). A population baseline frequency of 0.33 asthma attacks per child was assumed based on the results of the ISAAC study completed in Austria in 1995-96 (Eder, 1998; Haidinger, 1998a,b and data from the SCARPOL study in Switzerland, unpublished). This population average corresponds to an average of 3 asthma attacks per asthmatic.

3.1.7. Asthma exacerbation in adults

Evidence from previous studies

Three European panel studies were available for adults: two from the Netherlands (Dusseldorp et al., 1995, Hiltermann et al., 1998) using PM10, and one from Paris (Neukirch et al., 1998) using PM13. Asthma attacks were defined by wheeze or shortness of breath. A joint European estimate was 1.039. Two studies from the United States were also available, one from Utah (Pope et al., 1991) and one from Denver (Ostro et al., 1991), with a relative of 1.002.

Risk estimates used for present study

A random effects model generated a combined estimate of 1.004 (95% CI = 1.000 – 1.008). Baseline estimates were assumed to be equal to those used in Künzli et al. (1999), which were derived from the Swiss SAPALDIA study and the ECRHS study (1991-92, unpublished data). A population average of 0.21 asthma attacks per year per adult was calculated, corresponding to an average of 3.5 asthma attacks per adult asthmatic.

3.1.8. Restrictions in activity

Evidence from previous studies

Several studies have demonstrated an association between PM and restrictions in activity (Ostro, 1990; Ostro and Rothschild, 1989). The Health Interview Survey, an annual

survey of 50,000 individuals conducted in the United States, provides information on the several indicators of morbidity. A restricted activity day (RAD) is one where an individual misses work, spends the day in bed or is otherwise forced to significantly limit normal activity. One study (Ostro, 1990) reported an association between respiratory-related RAD for adults and PM, measured as either PM15 or fine particulates. A multivariate regression model was used, controlling for many individual factors such as age, sex, socio-economic status, and chronic disease.

To correct for double counting of RADs and hospital admissions, each hospital admission was assumed to be equivalent to five RADS, and these five RADS were subtracted from the total RADs in the impact estimates.

Risk estimates used for present study

Using PM15 as the PM metric, a relative risk of 1.094 (95%CI = 1.079 – 1.109) per 10 $\mu g/m^3$ was determined. From this data source, a baseline prevalence of respiratory RAD of 3.23 per year was indicated. These estimates are provided for adults age 21 and above, since this is the population used in the original epidemiological study.

3.1.9. Respiratory symptoms

Evidence from previous studies

Several studies provide estimates of the effect of PM on respiratory symptoms. Ostro et al. (1993) indicate an association between PM and lower, but not upper, respiratory symptoms among adults in Los Angeles. Using a similar data set, Krupnick et al. (1990) reported an association between PM and all symptoms. These studies involved a panel of 300 non-smoking adults followed over a 3-month period. PM was measured as sulfates, which are assumed to equal 0.25 of PM10.

To correct for double counting of symptoms, RADs and asthma attacks, the latter two were subtracted from the total number of symptoms occurrence.

Risk estimates used for present study

Using the 1993 study, the results indicate that a 10 $\mu g/m^3$ change in PM10 generates a relative risk of lower respiratory symptoms of 1.07 (95% CI = 1.02 – 1.11). On average, the baseline prevalence of lower respiratory symptoms was 14.24 days per year. The present estimates are applied to both adults and children.

3.2. Exposure assessment

This section describes how the current ambient concentrations of PM10 were determined for each of the eight cities in the study. The level of urbanization varies considerably among the eight cities: only half of them have one million or more inhabitants

(see Appendix A1). The population age distribution also differs consistently, with a higher proportion of the younger age groups in the cities of the Central-South of Italy.

3.2.1. Air pollution monitoring networks in the eight cities

For many of the cities, PM10 was not measured on a regular basis and the monitors were placed at different points throughout each city. Therefore, it was necessary to determine which monitors were most representative of population exposure for each of the cities. Ideally, the selected monitors would be similar to those used in the original epidemiological studies. Traditionally, the studies have used either the central city monitors or an average of the population-oriented monitors throughout the city. Care is usually taken in the studies not to use monitors that are clearly not relevant to the overall population, such as those near a large stationary source, near a major highway, or far from the population centroid.

In Italy the characteristics of the monitoring stations are indicated by a law that defines four main types, based only on qualitative criteria:

- ✓ type A: urban background station;
- ✓ type B: high density population station;
- ✓ type C: high traffic station;
- ✓ type D: suburban photochemical.

Each city has a different number of stations (ranging from 7 in Bologna and Palermo to 19 in Genoa), with a different distribution of station types. Comparability among networks is overall poor, also given the lack of national standards that define station locations. In particular, type B and C stations, which are the most relevant for the present exercise, are generally not comparable because the two criteria to define them are not mutually exclusive. Moreover, the traffic conditions and local meteorology vary by site.

Measurements of PM10 have been introduced very recently, i.e. January 1998 for most of the cities. Before this date, mainly TSP data are available. For some cities, PM10 measurements are still not available, or they are available for very few monitors of the network. In addition, there are no national rules that define air quality data collection methods for both TSP and PM10. At least two different methods are used to measure TSP (gravimetric and beta-ray absorption) and three for PM10 (gravimetric, TEOM, beta ray absorption). This study attempted to provide a reasonable interpretation of the existing data, which will be updated as monitoring data become more consistent.

3.2.2. Methods to derive estimates of population exposure

To estimate urban population exposure from the existing monitoring data, relevant monitoring stations for each city were selected and the arithmetic means of ambient concentrations from both 1998 and 1999 were taken. This average value was used as an indicator of the current population exposure.

When available, stations measuring PM10 were used; otherwise, estimates were made using TSP data, applying correction coefficients derived from literature or directly calculated from available Italian data. Furthermore, since PM10 is more frequently measured with TEOM analysers or a beta-ray absorption method, a correction factor of 0.77 was applied to PM10 concentrations measured with a gravimetric method (APEG 1999, Stanger Science and Environment, 1999) to derive automatic analyzer-based PM10. For most of the cities, the recently available PM10 data for 1999 are close to the measurements for 1998.

The following general criteria were adopted for the selection of the stations:

1. The stations should be located within the city borders and very close to the population centroid.
2. The stations cannot be placed too close to local source emissions, particularly industrial, and they should be generally located in residential areas.
3. The daily correlation among the selected stations should be reasonably high (~ > 0.7), to exclude outliers or monitors measuring "hot spots" instead of regional background concentrations.
4. The stations should provide a sufficient number of data (~ > 50% of possible data must be valid for a given period of time).
5. To ensure an adequate representation of the population exposure, at least two stations are to be selected for each city.

Details of the exposure assessment specific for each city are provided in Appendix A1.1.

3.2.3. City-specific exposure assessment

A summary of mean annual concentrations of PM10 for the biennium 1998-99 in the eight cities under study is given in Table 2.

Table 2 PM10 ANNUAL MEAN CONCENTRATION IN THE EIGHT LARGEST ITALIAN CITIES FOR 1998-99

	$\mu g/m^3$		$\mu g/m^3$
Turin	53.8	Florence	46.5
Genoa	46.1	Rome	51.2
Milan	47.4	Naples	52.1
Bologna	51.2	Palermo	44.4

3.2.4. Choice of reference values for air pollution

In this study, three different ambient standards were used as reference levels, as follows.

The main reference level was chosen *a priori* at 30$\mu g/m^3$. This value is in between the European Union proposed Stage 1 and Stage 2 limit values of 40 and 20 $\mu g/m^3$ PM10 respectively. These two reference values were also examined.

Using the controlling ambient standards recently promulgated by the U.S. Environmental Protection Agency for PM2.5, the annual average standard for PM2.5 is 15 $\mu g/m^3$. Assuming a ratio between PM2.5 and PM10--approximately 0.5 (U.S. EPA, 1996)--this would be equivalent to 30 $\mu g/m^3$ PM10. To estimate the attributable health effects from air pollution exposure, the change from these three presumed effect levels to the existing ambient concentrations in each city was calculated, based on current monitoring information.

It should be stressed, however, that all impact estimates calculated using these standards understate the overall health burden associated to exposure to PM10 in urban areas. As no threshold seems to exist, health effects are likely to occur down to the background level of approximately 7 $\mu g/m^3$ of PM10 (U.S. EPA, 1996).

3.3. Methods for quantification

For each of the included health endpoints, a relative risk estimate (RR) was determined pooling the estimates from the available studies, as discussed above. The relative risk is the increase in the probability of a given health effect associated with a given increase in exposure (usually 10$\mu g/m^3$ in epidemiological studies of PM).

The attributable proportion (A) of health effects from air pollution for the entire population can be calculated as:

$$(1) \qquad A = (RR - 1) / RR$$

To calculate the number of cases attributable to air pollution (E), the following formulation was used:

$$(2) \qquad E = A * B * C * P$$

where:

B = population baseline rate of the given health effect

C = relevant change in air pollution

P = relevant exposed population for health effect

Following Künzli et al. (1999), the population baseline rate is the proportion of the exposed population that would experience the health outcome assuming a baseline level (or no effects level) of air pollution. This can be calculated as

(3) $B = Bo / [1 + (RR-1)(C/10)]$

where:

Bo = observed rate of the health effect under current exposure

B = baseline rate of the health effect under baseline exposure

C = relevant change in air pollution

RR = relative risk estimate per 10 $\mu g/m^3$ determined from the pooled studies. This number is divided by 10 to obtain the risk per unit.

Bo is obtained from available health statistics, C is obtained from monitoring networks in each city, and P is obtained from census data for the cities under study.

This formula adjusts the current prevalence or incidence level of the health effect, using the relative risk, to the level that would exist with a lower air pollution concentration. As an example of these calculations, assume that (a) the relative risk for respiratory symptoms is 1.10 for a one $\mu g/m^3$ change in PM10; (b) every person in the city is exposed to a PM10 concentration of 50 $\mu g/m^3$ and the projected "standard" is 20 $\mu g/m^3$); (c) people have an average of 10 symptoms per year; and (d) there are a million people in this city who are expected to respond to PM10 with respiratory symptoms.

Using equation (3) it is possible to calculate the baseline prevalence (associated with a lower air pollution level) as: $B = 10 / (1 + (0.1)(30/10)) = 7.7$. The attributable risk per one $\mu g/m^3$ (A) can be calculated from equation (1) as $0.09 = 0.1 / 1.10$. Thus, for this example, roughly 9% of the total number of symptoms in the population can be attributed to a one $\mu g/m^3$ change in PM10. Applying these estimates to equation (2), the number of cases attributable to air pollution are:

E = (0.09) * (7.7) * (30) * (1,000,000) = 20.7 million extra days with symptoms due to air pollution.

4. Results

The analyses provide estimates of the impact of PM10 ambient air pollution on the health of the people living in the eight major Italian cities, as measured by attributable cases for the selected outcomes. This section summarizes the results. Complete information is given in the tables in Appendix A2.

A reference value of 30 $\mu g/m^3$ was assumed here, but other standards (40 and 20 $\mu g/m^3$) have been applied and the results are reported in Appendix A2.

The following table summarizes the results of the study in terms of health impact, expressed as proportion and number of cases attributable to air pollution for the outcomes considered:

Table 3 HEALTH OUTCOMES ATTRIBUTABLE TO PM10 CONCENTRATIONS ABOVE 30 µG/M³. 1998.

	Estimated proportion (%)	*95% Confidence limits*		*Estimated number of attributable cases*
Mortality (excluding accidental causes) (age ≥ 30)	4.7	1.7	7.5	3 472
Hospital admissions for respiratory disease	3.0	2.5	3.7	1 887
Hospital admissions for CVD	1.7	1.2	2.5	2 710
Acute bronchitis (age < 15)	28.6	18.4	32.9	31 524
Asthma attacks (age < 15)	8.7	8.1	9.2	29 730
Asthma attacks (age ≥ 15)	0.8	0	1.5	11 360
RAD (age > 20)	14.3	12.5	15.9	2 702 461
Respiratory symptoms	11.3	3.7	11.0	10 409 836

Details of results by city are given in the following sections.

4.1. Mortality

Results for mortality excluding accidental causes for all the cities, including confidence intervals, are shown in Table 4. Among people older than 30, 4.7% of all deaths are attributable to PM10 concentrations in excess of 30 $\mu g/m^3$. The attributable proportion of mortality range from 3.5% in Palermo to 5.7% in Turin. Turin has the highest attributable proportion and Palermo the lowest since they have the highest and the lowest average PM10 concentration, respectively. A total of 3,472 deaths per year are attributable to air pollution.

Table 4 **MORTALITY ATTRIBUTABLE TO PM10 CONCENTRATIONS ABOVE 30 μG/M³ (EXCLUDING ACCIDENTAL CAUSES). AGE ≥30 YEARS**

	Estimated no. of cases	*95% CI*		*Estimated% of cases*	*95% CI*	
Turin	420	154	658	5.7	2.1	8.9
Genoa	260	94	412	3.9	1.4	6.2
Milan	441	160	698	4.2	1.5	6.7
Bologna	252	92	396	5.1	1.8	8.0
Florence	181	65	286	4.0	1.5	6.4
Rome	1 278	466	2 011	5.1	1.9	8.0
Naples	444	162	697	5.3	1.9	8.3
Palermo	197	71	313	3.5	1.3	5.6
Total	3 472	1 263	5 471	4.7	1.7	7.5

4.2. Morbidity

4.2.1. Hospital admissions

Air pollution is responsible of 1.7% of all hospital admissions due to cardiovascular causes in the cities investigated. Due to the high frequency of cardiovascular diseases, the estimated number of attributable admissions is 2,710 (Table 5).

Table 5 **ADMISSIONS TO HOSPITAL FOR CVD CAUSES ATTRIBUTABLE TO PM10 CONCENTRATIONS ABOVE 30 μG/M³**

	Estimated no. of cases	*95% CI*		*Estimated% of cases*	*95% CI*	
Turin	275	185	391	2.1	1.4	3.0
Genoa	171	115	244	1.4	1.0	2.0
Milan	520	349	742	1.5	1.0	2.2
Bologna	148	99	211	1.9	1.2	2.6
Florence	58	39	83	1.5	1.0	2.1
Rome	1 007	677	1 436	1.9	1.2	2.6
Naples	370	249	528	1.9	1.3	2.8
Palermo	163	110	233	1.3	0.8	1.8
Total	2 710	1 823	3 869	1.7	1.2	2.5

A total of 1,887 hospital admissions for respiratory diseases (3.0%) are estimated as attributable to air pollution in the eight cities. Breakdown by city is given in Table 6.

Table 6 **ADMISSIONS TO HOSPITAL FOR RESPIRATORY CAUSES ATTRIBUTABLE TO PM10 CONCENTRATIONS ABOVE 30 μG/M³**

	Estimated no. of cases	*95% CI*		*Estimated% of cases*	*95% CI*	
Turin	243	200	300	3.6	3.0	4.5
Genoa	119	97	147	2.5	2.0	3.1
Milan	370	303	457	2.7	2.2	3.3
Bologna	107	88	132	3.2	2.6	4.0
Florence	30	25	38	2.5	2.1	3.1
Rome	648	531	800	3.2	2.6	4.0
Naples	257	210	317	3.4	2.8	4.1
Palermo	114	93	141	2.2	1.8	2.7
Total	1 887	1 547	2 332	3.0	2.5	3.7

4.2.2. Acute bronchitis

Acute bronchitis in children was the endpoint with the highest attributable proportion, reaching 32.3% in Turin for example. Overall, 31 524 attributable cases were estimated, amounting to 28.6% of the total (see Table 7).

Table 7 **CASES OF ACUTE BRONCHITIS ATTRIBUTABLE TO PM10 CONCENTRATIONS ABOVE 30 μG/M³. AGE <15 YEARS.**

	Estimated no. of cases	*95% CI*		*Estimated% of cases*	*95% CI*	
Turin	3 360	2 229	3 776	32.3	21.4	36.3
Genoa	1 682	1 046	1 982	25.2	15.7	29.7
Milan	3 723	2 343	4 346	26.6	16.7	31.0
Bologna	1 084	704	1 236	30.1	19.5	34.3
Florence	974	608	1 144	25.7	16.0	30.2
Rome	10 966	7 126	12 504	30.1	19.5	34.3
Naples	6 235	4 081	7073	30.8	20.2	35.0
Palermo	3 500	2 140	4 180	23.3	14.3	27.9
Total	31 524	20 277	36 241	28.6	18.4	32.9

4.2.3. Asthma exacerbation

The proportion of cases of asthma exacerbation was also estimated separately for children (under 15 years old) and adults. For the former group (Table 8) the attributable proportion was 8.7%, or 29 730 extra cases. For the older group, the attributable proportion was lower (0.8%), but the number of attributable cases remained substantial (11360, see Table 9).

Table 8 CASES OF ASTHMA EXACERBATION ATTRIBUTABLE TO PM10 CONCENTRATIONS ABOVE 30 µG/M³. AGE <15 YEARS.

	Estimated no. of cases	*95% CI*		*Estimated% of cases*	*95% CI*	
Turin	3 341	3 117	3 559	10.3	9.6	11.0
Genoa	1 496	1 393	1 598	7.2	6.7	7.7
Milan	3 380	3 147	3 609	7.8	7.2	8.3
Bologna	1 039	969	1 108	9.3	8.6	9.9
Florence	872	812	931	7.4	6.9	7.9
Rome	10 517	9 804	11 214	9.3	8.6	9.9
Naples	6 055	5 646	6 454	9.6	9.0	10.3
Palermo	3 028	2 817	3 236	6.5	6.0	6.9
Total	29 730	27 705	31 709	8.7	8.1	9.2

Table 9 CASES OF ASTHMA EXACERBATION ATTRIBUTABLE TO PM10 CONCENTRATIONS ABOVE 30 µG/M³. AGE >15 YEARS.

	Estimated no. of cases	*95% CI*		*Estimated% of cases*	*95% CI*	
Turin	1 601	0	3 160	0.9	0	1.9
Genoa	774	0	1 531	0.6	0	1.3
Milan	1 700	0	3 363	0.7	0	1.4
Bologna	611	0	1 206	0.8	0	1.7
Florence	468	0	925	0.7	0	1.3
Rome	4 040	0	7 981	0.8	0	1.7
Naples	1 517	0	2 995	0.9	0	1.7
Palermo	651	0	1 289	0.6	0	1.1
Total	11 360	0	22 451	0.8	0	1.5

4.2.4. Other health effects

Finally, Table 10 and Table 11 show the results for the other two health outcomes considered, the number of days with restricted activity (RAD) for respiratory conditions among people more than 20 years of age and the occurrence of respiratory symptoms for all ages. A sizeable proportion of both, 14.3% and 11.3% respectively, are attributable to PM10 concentrations above 30 $\mu g/m^3$. These proportions represent about 3 million extra RADs and 10 million extra episodes.

Table 10 DAYS OF RESTRICTED ACTIVITY (RAD) FOR RESPIRATORY CAUSES ATTRIBUTABLE TO PM10 CONCENTRATIONS ABOVE 30 µG/M³. AGE >20

	Estimated no. of cases	*95% CI*		*Estimated% of cases*	*95% CI*	
Turin	377 305	331 621	419 506	16.7	14.7	18.6
Genoa	195 044	169 998	218 618	12.0	10.5	13.5
Milan	412 567	360 126	461 760	12.9	11.2	14.4
Bologna	143 067	125 396	159 494	15.2	13.3	16.9
Florence	117 482	102 445	131 621	12.3	10.7	13.8
Rome	951 009	833 540	1 060 199	15.2	13.3	16.9
Naples	346 780	304 235	386 243	15.7	13.8	17.5
Palermo	159 206	138 482	178 798	10.9	9.5	12.2
Total	2 702 461	2 365 843	3 016 238	14.3	12.5	15.9

Table 11 OCCURRENCE OF RESPIRATORY SYMPTOMS ATTRIBUTABLE TO PM10 CONCENTRATIONS ABOVE 30 µG/M³. ALL AGES

	Estimated no. of cases	*95% CI*		*Estimated% of cases*	*95% CI*	
Turin	1 391 577	464 487	1 948 848	13.4	4.5	18.7
Genoa	691 277	223 334	989 901	9.5	3.1	13.5
Milan	1 514 944	492 233	2 160 776	10.1	3.3	14.5
Bologna	522 946	172 639	737 790	12.1	4.0	17.0
Florence	415 922	134 616	594 844	9.7	3.1	13.8
Rome	3 681 317	1 215 309	5 193 734	12.1	4.0	17.0
Naples	1 500 668	497 274	2 111 853	12.5	4.1	17.6
Palermo	691 186	221 611	995 104	8.5	2.7	12.3
Total	10 409 836	3 421 504	14 732 851	11.3	3.7	16.0

5. Uncertainties and research needs

There are some uncertainties and omissions in estimating the health effects of air pollution in Italy. The uncertainties involve each of the components of the methodology: dose-response estimates, population exposure, and ambient concentrations. Below is a brief review of some of the major omissions and uncertainties along with suggestions for research that might be undertaken to reduce them.

5.1. Limitations

Certain clinical outcomes such as changes in lung function are not estimated. To the extent that these changes have not been reflected in the estimates of symptoms and other morbidity provided by this study, an underestimation of effects will result. Likewise, outcomes such as physician visits have not been estimated. Finally, since people undertake certain evasive or mitigating behaviour to reduce the effect of air pollution, the effects of air pollution would be underestimated. For example, people may buy air filters, stay indoors, forgo exercise, or take extra medications. These are all social costs related to air pollution that would not be reflected in the present estimates.

Another limitation arises from the use made here of PM10 as a marker for all pollution. Using PM10 may account for the impact of correlated pollutants, which varied with PM10 in the original epidemiological study. However, there may be other pollutant effects related to ozone, nitrogen dioxide, carbon monoxide, and lead, for example, which are not, or are only partially, incorporated into the effects of PM10. Therefore, the total effect of air pollution may be significantly underestimated.

Finally, the estimates from this study only concern the impacts on the eight largest cities in Italy; in order to estimate the total national burden of pollution additional monitoring and/or dispersion modelling would need to be undertaken throughout the country.

5.2. Dose-response estimates

The dose-response estimates made here for mortality and morbidity incorporate some statistical uncertainty associated with the variance in the regression model and data used. However, additional uncertainties may exist. For example, the studies may have omitted confounding variables that could raise or lower the estimated pollution effect. Most of the recent studies have been rigorous in their attempts to control for most major confounders. However, possible confounding will continue to exist, particularly in the long-term cohort studies.

A second issue relates to the transfer of results from studies from the United States and European countries to Italy. When studies have been undertaken in Italy, they appear to confirm the general magnitude of effect found in studies undertaken outside of Italy. However, there may be factors specific to Italy that impact the subsequent dose-response relationship, for example, the weather, housing characteristics, time spent outdoors, habits of exercise and diet, smoking status, socio-economic status, access to health care, and

baseline health status. It is unclear whether the estimates would be higher or lower for Italy-specific studies.

A third uncertainty relates to the possibility of particularly susceptible subgroups among the population such as asthmatics, elderly, or infants. Another arises from the use of the chronic exposure studies for mortality. The exact period and length of exposure necessary to elicit this outcome is uncertain. Again, this could result in either an under- or overestimation of the effect of air pollution. Additional uncertainty in the dose-response estimates results from the use of centrally located, population-orientated monitors in most studies, whose readings are surrogates for the actual exposures of the population, thus resulting in a community-average, rather than true personal exposure time. Generally, this error in measuring exposure will result in an underestimation of the effect of air pollution. The errors in measurement are due to differences between personal and community exposure, instrument and analysis errors in the monitoring itself, spatial variability in the outdoor air concentrations, and differences between outdoor and indoor air quality.

Finally, the choice of a statistical model can affect the dose-response estimate. Each published study has made judgements about the most appropriate model. Often, the results are not sensitive to the model specification or the included covariants. However, it is possible that in certain cases, function form, covariants, and extreme observations may impact the ultimate dose-response function.

The obvious way to reduce these uncertainties related to dose-response is to conduct additional studies in Italy for several adverse health outcomes and subgroups using monitors that measure either PM10 or PM2.5.

5.3. Population exposure

Another potential uncertainty is related to the use of PM10 in the original study as a surrogate for some other pollutants in the atmosphere with which it is correlated. For example, the health effects associated in the model with PM10 may be more a function of fine particles, ultrafine particles, or other agents. Given the weight of evidence for effects related to PM10, however, it is likely that this pollutant by itself has an effect on health.

One method to reduce this uncertainty is to enhance the monitoring network in several Italian cities to include measurement of PM2.5 and ultrafine particles, as well as PM10. Careful and consistent monitor placement among the cities is important in this regard. Only after additional measures of particulate matter and relevant gaseous pollutants are monitored can this issue be effectively addressed. It may be also important to monitor hot spots of pollution such as traffic corridors.

5.4. Valuation of effects

An additional research and policy need relates to the economic assessment of the predicted change in health effects. Although it might be controversial and uncertain, the economic assessment can help put the health impacts into perspective and aid policy makers in prioritizing among different pollution control strategies or among more general options for improving public health. To provide a sense of the magnitude of the total economic effects associated with the assumed changes in air pollution, the physical effects

are valued in monetary terms. These estimates include both mortality and morbidity effects. Ideally, the monetary values reflect the full impact to the affected individuals. This would include both out-of-pocket expenses, such as medical costs and lost income (referred to as "cost of illness"), and less tangible effects on welfare, such as pain and discomfort and restrictions in non-work activities. For mortality, the published literature provides estimates of the value of small changes in the risk of death. Ultimately, there are three general issues that need to be addressed. They are: (1) the appropriate methods and values to be used with regard to environmentally-associated health effects; (2) the appropriateness of utilizing existing monetary values derived mostly from studies in the United States and England; and (3) the adjusting of such values to fit local conditions. Additional studies that take into account the attitudes and perceptions about risk among Italian citizens will help determine the appropriate values and ranges of uncertainty.

6. Conclusions

This study confirms the findings of several investigations worldwide: in large cities of industrialised countries, a sizeable proportion of several adverse health outcomes, including mortality, is due to air pollution. While there are several limitations, discussed in the previous chapter, the estimates suggest, for the population of the eight largest Italian cities, that thousands of deaths, hospital admissions, cases of bronchitis and other respiratory conditions, are in excess, compared to the rates that can be predicted at lower levels of ambient PM10 concentrations. As indicated above, PM10 was used as a summary indicator of air quality, given its importance as a health determinant and the correlation between different urban pollutants. However, while it is currently unclear how to estimate impacts from various pollutants, it is likely that health can be impacted upon additionally by other pollutants. Thus, the figures presented in this report tend to underestimate the real health impact. The same applies for other limitations in the currently available methodology for health impact assessment studies. In other words, the methods used for the study produce estimates that describe at least part of the true health impact, but this is likely to be greater. Even so, the magnitude of the health impact thus estimated is large. To put the thousands of deaths in a relevant context, for example, in the eight cities considered the number of deaths due to road accidents is 488 per year.

Health impact assessment studies of air pollution need to be improved. While these "first generation" studies are valuable, progress is desirable in many aspects including enhancing the methodology and the quality and completeness of data, identifying all relevant health endpoints, considering all pollutants, taking into account all vulnerable subgroups, and characterising uncertainties more reliably. However, the findings cannot be discounted. The health consequences of air pollution in urban areas are large because concentrations are high; large Italian cities, with annual averages of PM10 around $50\mu/m^3$, reach levels even higher than many other European cities. So, reduction of such concentrations is urgent and would produce health gains in the short and medium-long term. Reduction of concentrations is required by European Commission legislation, which has introduced limits of 40 $\mu g/m^3$ to be complied with by 2005 and 20 $\mu g/m^3$ by 2010 (EC, 1999).

Currently, the contribution of air pollutants from different sources has not been quantified systematically, and there is large variability depending on local circumstances and climatic patterns. However, motor vehicle emissions, including those from diesels and two-stroke motorcycles, are among the biggest contributors in Italian cities (Cirillo, in press). The contribution is manifold and includes direct but also indirect pathways, for example via ozone and resuspension of fine particles. Action is urgently needed to curb these emissions if we are to reduce the health burden of air pollution. The gains from reducing motor vehicle traffic would include those characterized by the present evaluation, but, in addition, would likely include other benefits, achievable for example through reduction of noise, or through an increase in physical activities such as cycling and walking.

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APPENDICES

A1. Population and PM 10 pollution in the eight cities

Table 12 POPULATION LIVING IN THE EIGHT MAIN ITALIAN CITIES BY AGE GROUP ACCORDING TO LAST CENSUS (1991)

	Population				
City	**Total**	**≤14 years**		**65+ years**	
		N	**%**	**N**	**%**
Turin	962 507	111 315	11.6	161 477	16.8
Genoa	678 771	69 627	10.3	143 688	21.2
Milan	1 369 231	141 352	10.3	249 204	18.2
Bologna	404 378	33 742	8.3	94 507	23.4
Florence	403 294	40 020	9.9	88 650	22.0
Rome	2 775 250	373 432	13.5	402 485	14.5
Naples	1 067 365	204 694	19.2	128 300	12.0
Palermo	698 556	147 418	21.1	79 545	11.4
Total	**8 359 352**	**1 121 600**	**13.4**	**1 347 856**	**16.1**

Source: 1991 National Census, ISTAT.

A1.1 Calculation of PM10 mean annual concentration

A1.1.1 Turin

1998: Direct measures of PM10 were available for only one station (Consolata) with a concentration of 74.1 $\mu g/m^3$ (gravimetric method). TSP was measured in 4 stations, including Consolata and one station located in a park, using a gravimetric method. The observed PM10/TSP ratio for the station at Consolata is 0.64. The average for the two other stations measuring TSP and located in residential areas is 116.3 $\mu g/m^3$ or 74.4 $\mu g/m^3$ PM10 using the same ratio as observed at Consolata. Averaging these monitors with the PM10 measured at Consolata gives a concentration of 74.2 $\mu g/m^3$ (gravimetric), equivalent to 57.1 $\mu g/m^3$ (automatic).

1999: PM10 measures at Grassi and Consolata give a mean of 65.7 $\mu g/m^3$ (gravimetric), equivalent to 50.5 $\mu g/m^3$ (automatic).

Two-year average: 53.8 $\mu g/m^3$ (automatic).

A1.1.2 Genoa

1998: PM10 concentrations were available from three stations, one of them located in an urban park far from the center of the city (Quarto). Therefore, the two remaining stations

(Piazza Masnata and Via XX Settembre) were selected. This generates a mean concentration of 58.3 µg/m^3 PM10 (gravimetric), or 44.9 µg/m^3 (automatic).

1999: These stations give an annual mean of 47.3 µg/ m^3 (automatic).

Two-year average: 46.1 µg/m^3 (automatic)

A1.1.3 Milan

1998: Two stations (Juvara and Zavattari) measured PM10 using a beta-gauge method. The average of these two was therefore used to estimate Milan exposure, yelding an annual average of 47.7 µg/m^3.

1999: These same two stations had a mean of 47.1 µg/ m^3

Two-year average: 47.4 µg/ m^3.

A1.1.4 Bologna

1998: PM10 was measured only by one station (Malpighi), providing a concentration of 60.7 µg/m^3. TSP was measured by four stations, including Malpighi, using a beta-gauge method. However, one of these stations is far from the population centroid, and was considered not representative. The average for the two other stations measuring TSP is 39.6 µg/m^3 or 33.7 µg/m^3 PM10, assuming a correction coefficient of 0.85 for PM10/TSP. Averaging these monitors with the PM10 measured at Malpighi gives a concentration of 47.2 µg/m^3.

1999: Using these same monitors plus a station at San Felice measuring PM10 gives an annual average of 55.1 µg/ m^3.

Two-year average: 51.2 µg/m^3.

A1.1.5 Florence

1998: Five out of the 7 stations of the network of Florence measured PM10**.** Location and characteristics of these stations are quite different, while the correlations among the stations are low. Two of them are placed very closed to heavily trafficked roads; one is placed in an urban park and another one is far from the center of the city. The remaining station of Mosse (in a center-city residential area) was selected as best representing PM10 exposure of the city. The mean annual PM10 value measured by the Mosse station was 45.4 µg/m^3, which is actually very close to the average value of all five stations (43.7 µg/m^3).

1999: The same station measured 47.6 µg/m^3.

Two-year mean: 46.5 µg/m^3.

A1.1.6 Rome

1998: The three stations measuring PM10 located in residential areas of the city were included (beta-ray absorption). Another station (Villa Ada) was excluded since it is located in an urban park. The average PM10 concentration for the three stations is 51.5 μg/m^3.

1999: The same three stations had a mean of 50.8 μg/ m^3.

Two-year average: 51.2 μg/m^3.

A1.1.7 Naples

1998: No PM10 measurements were available for this city. PM10 was therefore estimated from TSP concentrations assuming a PM10/TSP ratio of 0.85. The three stations selected are Museo Nazionale, Ente Ferrovie and Policlinico. The average PM10 concentration for these three stations was 53.6 μg/m^3. This value is close to the average value of all 7 of the monitors that measure TSP, after applying the 0.85 ratio, of 54.6 μg/m^3.

1999: The three stations selected for 1998 averaged 50.5 μg/m^3.

Two-year average: 52.1 μg/m^3.

A1.1.8 Palermo

1998: All the stations in Palermo measured both PM10 and TSP (beta-ray method). The two stations Castelnuovo and Unità d'Italia were selected as more representative of the population exposure to PM10, giving an average PM10 concentration of 43.1 μg/m^3. This value is close to the average of the 6 urban stations measuring PM10 of 39.9 μg/m^3.

1999: The Castelnuovo station was the only available, averaging 45.6 μg/m^3.

Two-year average: 44.4 μg/m^3.

A2. Estimates of health impact with different baseline values

A2.1 Mortality

Table 13 MORTALITY, ADULTS (AGED ≥ 30 YEARS, EXCLUDING ACCIDENTAL CAUSES)

	Population 1998			Mortality		Number of cases attributable to air pollution								
	Total	aged ≥30		(aged ≥30 yrs)		Baseline = 30			Baseline = 20			Baseline = 40		
	N	N	%	N	%	Central estimate	95% CI		Central estimate	95% CI		Central estimate	95% CI	
Turin	909 717	642 260	70.6	7 386	1.15	420	154	658	582	216	899	250	90	397
Genoa	641 437	474 022	73.9	6 636	1.40	260	94	412	411	151	642	101	36	162
Milan	1 307 785	941 605	72.0	10 452	1.11	441	160	698	677	249	1 056	192	69	309
Bologna	382 006	289 561	75.8	4 965	1.71	252	92	396	362	134	561	136	49	217
Florence	376 760	278 426	73.9	4 510	1.62	181	65	286	283	104	442	73	26	118
Rome	2 646 408	1 796 911	67.9	25 157	1.40	1 278	466	2 011	1 837	680	2849	691	248	1 104
Naples	1 020 120	599 831	58.8	8 398	1.40	444	162	697	630	233	975	249	89	397
Palermo	686 551	401 632	58.5	5 623	1.40	197	71	313	326	120	511	61	22	99
Total	7 970 784	5 424 248	68.1	73 127		3 472	1 263	5 471	5 108	1 887	7 935	1 752	628	2 802

A2.2 Morbidity

Table 14 RESPIRATORY HOSPITAL ADMISSIONS, ALL AGES

	Population 1998	Respiratory admissions		Number of cases attributable to air pollution								
	Total	1998		Baseline = 30			Baseline = 20			Baseline = 40		
	N	N	%	Central estimate	95% CI		Central estimate	95% CI		Central estimate	95% CI	
Turin	909 717	6 709	0.74	243	200	300	340	280	418	143	117	177
Genoa	641 437	4 811	0.75	119	97	147	190	156	234	46	37	57
Milan	1 307 785	13 863	1.06	370	303	457	573	471	706	160	130	198
Bologna	382 006	3 325	0.87	107	88	132	155	128	191	57	47	71
Florence	376 760	1 198	0.32	30	25	38	48	39	59	12	10	15
Rome	2 646 408	20 065	0.76	648	531	800	940	773	1 156	347	284	430
Naples	1 020 120	7 651	0.75	257	210	317	367	302	452	142	116	177
Palermo	686 551	5 149	0.75	114	93	141	190	156	234	35	29	44
Total	7 970 784	62 770		1 887	1 547	2 332	2 803	2 304	3 451	943	770	1 169

Table 15 CVD HOSPITAL ADMISSIONS, ALL AGES

	Population 1998	CVD admissions		Number of cases attributable to air pollution								
	Total	1998		Baseline = 30			Baseline = 20			Baseline = 40		
	N	N	%	Central estimate	95% CI		Central estimate	95% CI		Central estimate	95% CI	
Turin	909 717	13 206	1.5	275	185	391	387	261	549	161	108	230
Genoa	641 437	12 059	1.9	171	115	244	274	185	390	65	44	93
Milan	1 307 785	34 002	2.6	520	349	742	811	547	1 154	223	149	320
Bologna	382 006	7 977	2.1	148	99	211	216	145	306	79	53	113
Florence	376 760	4 023	1.1	58	39	83	93	63	132	23	16	33
Rome	2 646 408	54 376	2.1	1 007	677	1 436	1 470	992	2 089	535	359	767
Naples	1 020 120	19 178	1.9	370	249	528	533	360	757	204	137	292
Palermo	686 551	12 907	1.9	163	110	233	274	185	391	50	33	72
Total	7 970 784	157 729		2 710	1 823	3 869	4 057	2 737	5 770	1 340	899	1 920

Table 16 ACUTE BRONCHITIS, CHILDREN (< 15 YEARS)

	Population 1998			Annual incidence of acute bronchitis		Number of cases attributable to air pollution								
	Total	aged <15				Baseline = 30			Baseline = 20			Baseline = 40		
	N	N	%	N	%	Central estimate	95% CI		Central estimate	95% CI		Central estimate	95% CI	
Turin	909 717	98 249	10.8	10 414	10.6	3 360	2 229	3 776	4 054	2 872	4 363	2 368	1 440	2 840
Genoa	641 437	62 861	9.8	6 663	10.6	1 682	1 046	1 982	2 264	1 527	2 515	800	445	1 038
Milan	1 307 785	132 086	10.1	14 001	10.6	3 723	2 343	4 346	4 887	3 326	5 398	1 978	1 118	2 525
Bologna	382 006	33 999	8.9	3 604	10.6	1 084	704	1 236	1 346	939	1 464	702	415	861
Florence	376 760	35 792	9.5	3 794	10.6	974	608	1 144	1 300	880	1 442	482	269	622
Rome	2 646 408	344 033	13.0	36 468	10.6	10 966	7 126	12 504	13 622	9 499	14 809	7 099	4 197	8 714
Naples	1 020 120	190 762	18.7	20 221	10.6	6 235	4 081	7 073	7 663	5 373	8 303	4 168	2 489	5 075
Palermo	686 551	141 430	20.6	14 992	10.6	3 500	2 140	4 180	4 899	3 263	5 490	1 347	731	1 789
Total	7 970 784	1 039 212	13.0	110 157		31 524	20 277	36 241	40 036	27 680	43 784	18 943	11 104	23 464

Table 17 ASTHMA EXACERBATIONS IN CHILDREN (< 15 YEARS)

	Population 1998			Asthma exacerbation in the population		Number of cases attributable to air pollution								
	Total	aged <15				Baseline = 30			Baseline = 20			Baseline = 40		
	N	N	%	N	%	Central estimate	95% CI		Central estimate	95% CI		Central estimate	95% CI	
Turin	909 717	98 249	10.8	32 422	0.33	3 341	3 117	3 559	4 537	4 247	4 819	2 030	1 888	2 170
Genoa	641 437	62 861	9.8	20 744	0.33	1 496	1 393	1 598	2 317	2 164	2 467	594	551	637
Milan	1 307 785	132 086	10.1	43 588	0.33	3 380	3 147	3 609	5 085	4 750	5 411	1 508	1 399	1 616
Bologna	382 006	33 999	8.9	11 220	0.33	1 039	969	1 108	1 463	1 369	1 555	574	534	614
Florence	376 760	35 792	9.5	11 811	0.33	872	812	931	1 338	1 249	1 424	361	334	386
Rome	2 646 408	344 033	13.0	113 531	0.33	10 517	9 804	11 214	14 808	13 848	15 740	5 812	5 400	6 218
Naples	1 020 120	190 762	18.7	62 952	0.33	6 055	5 646	6 454	8 415	7 871	8 942	3 468	3 223	3 709
Palermo	686 551	141 430	20.6	46 672	0.33	3 028	2 817	3 236	4 905	4 578	5 225	964	893	1 034
Total	7 970 784	1 039 212	13.0	342 940		29 730	27 705	31 709	42 870	40 075	45 584	15 311	14 222	16 384

Table 18 ASTHMA EXACERBATIONS IN ADULTS (≥ 15 YEARS)

	Population 1998			Asthma		Number of cases attributable to air pollution								
	Total	aged ≥15		exacerbation in the population		Baseline = 30			Baseline = 20			Baseline = 40		
	N	N	%	N	%	Central estimate	95% CI		Central estimate	95% CI		Central estimate	95% CI	
Turin	909 717	811 467.6	89.2	170 408.2	0.21	1 601	0	3 160	2 265	0	4 453	933	0	1 848
Genoa	641 437	578 576.2	90.2	121 501.0	0.21	774	0	1 531	1 250	0	2 464	294	0	584
Milan	1 307 785	1 175 698.7	89.9	246 896.7	0.21	1700	0	3 363	2 666	0	5 254	726	0	1 441
Bologna	382 006	348 007.5	91.1	73 081.6	0.21	611	0	1 206	896	0	1 763	323	0	641
Florence	376 760	340 967.8	90.5	71 603.2	0.21	468	0	925	748	0	1 475	185	0	367
Rome	2 646 408	2 302 375.0	87.0	483 498.7	0.21	4 040	0	7 981	5 927	0	11 663	2 138	0	4 241
Naples	1 020 120	829 357.6	81.3	174 165.1	0.21	1 517	0	2 995	2 196	0	4 319	832	0	1 650
Palermo	686 551	545 121.5	79.4	114 475.5	0.21	651	0	1 289	1 100	0	2 170	198	0	394
Total	7 970 784	6 931 571.7	87.0	1 455 630.1		11 360	0	22 451	17 047	0	33 560	5 629	0	11 166

Table 19 RESTRICTIONS IN ACTIVITY, ADULTS (> 20 YEARS)

	Population 1998			Prevalence of		Number of cases attributable to air pollution								
	Total	aged > 20		NET resp. RAD (days per year)		Baseline = 30			Baseline = 20			Baseline = 40		
	N	N	%	N	%	Central estimate	95% CI		Central estimate	95% CI		Central estimate	95% CI	
Turin	909 717	774 169	85.1	2 253 253	2.91	377 305	331 621	419 506	497 471	441 433	548 131	237 107	206 112	266 452
Genoa	641 437	558 692	87.1	1 621 882	2.90	195 044	169 998	218 618	292 381	257 568	324 375	80 363	69 179	91 184
Milan	1 307 785	1 129 926	86.4	3 205 601	2.84	412 567	360 126	461 760	601 030	530 151	665 980	190 911	164 629	216 239
Bologna	382 006	337 311	88.3	942 279	2.79	143 067	125 396	159 494	195 365	172 940	215 751	81 853	70 923	92 275
Florence	376 760	326 651	86.7	955 603	2.93	117 482	102 445	131 621	174 460	153 751	193 475	50 389	43 400	57 142
Rome	2 646 408	2 175 347	82.2	6 263 597	2.88	951 009	833 540	1 060 199	1 298 650	1 149 581	1 434 155	544 099	471 448	613 377
Naples	1 020 120	759 989	74.5	2 206 249	2.90	346 780	304 235	386 243	467 580	414 249	515 966	205 542	178 294	231 462
Palermo	686 551	503 928	73.4	1 462 904	2.90	159 206	138 482	178 798	249 450	219 367	277 206	52 628	45 197	59 855
Total	7 970 784	6 566 015	82.4	18 911 368		2 702 461	2 365 843	3 016 238	3 776 387	3 339 040	4 175 038	1 442 892	1 249 183	1 627 985

Table 20 RESPIRATORY SYMPTOMS, ALL AGES

	Population	Prevalence of NET		Number of cases attributable to air pollution								
	1998	respiratory symptoms		Baseline = 30			Baseline = 20			Baseline = 40		
	N	N	%	Central estimate	95% CI		Central estimate	95% CI		Central estimate	95% CI	
Turin	909 717	10 421 381.4	11.46	1 391 577	464 487	1 948 848	1 864 096	647 183	2 545 354	858 738	274 680	1 238 421
Genoa	641 437	7 308 744.9	11.39	691 277	223 334	989 901	1 054 674	355 292	1 468 090	279 083	86 161	413 110
Milan	1 307 785	14 929 608.2	11.42	1 514 944	492 233	2 160 776	2 245 483	760 429	3 114 999	687 165	213 466	1 012 426
Bologna	382 006	4 339 030.4	11.36	522 946	172 639	737 790	725 939	249 479	997 603	293 591	92 794	427 064
Florence	376 760	4 298 168.5	11.41	415 922	134 616	594 844	628 553	212 095	873 986	174 818	54 078	258 389
Rome	2 646 408	30 544 947.0	11.54	3 681 317	1 215 309	5 193 734	5 110 302	1 756 225	7 022 703	2 066 758	653 229	3 006 350
Naples	1 020 120	12 008 791.4	11.77	1 500 668	497 274	2 111 853	2 056 535	709 210	2 819 965	873 034	277 060	1 266 189
Palermo	686 551	8 102 125.7	11.80	691 186	221 611	995 104	1 102 705	368 873	1 542 055	223 756	68 510	333 318
Total	7 970 784	91 952 797.4		10 409 836	3 421 504	14 732 851	14 788 287	5 058 784	20 384 755	5 456 944	1 719 978	7 955 269

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This study addresses the health consequences of air pollution in the eight largest cities of Italy. Air quality data were gathered and analyzed using the most recently developed methodologies for assessing pollution levels and health effects. Particulate matter (PM10) was used as a summary indicator of air quality, given its known relationship with health and its correlation with other urban pollutants.

Estimates from the study indicate that deaths, hospital admissions, cases of bronchitis and other respiratory conditions are in excess compared to the rates that can be predicted at lower levels of PM10 concentrations. These results confirm the findings from several investigations worldwide: in large cities of industrialized countries, a sizeable proportion of several adverse health effects, including mortality, is due to bad air quality.

The estimates provided by the study cover, however, only a fraction of the total health impact, as pollutants other than PM10 contribute additional health effects. This suggests that health consequences of urban transport policies largely based on private motor vehicles are likely to be more severe, and that reducing emissions from motor vehicles (the main source of PM10 in cities of industrialized countries) would benefit the health of urban populations.

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This book is also available on the Internet at
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