SECTION 3: AIRBORNE PARTICULATE MATTER AND ASSOCIATED HEALTH RISKS

This chapter discusses the effects of air-borne particulate matter on human health. We first assess the current “state-of-the-art” in the literature on human health risks associated with airborne particulate matter (PM), findings to date, and limitations of the available research. Then we provide an analysis of the expected benefits of reducing PM in the state of New Jersey, based on currently available monitoring information and existing models of health effects.

3.1 The Literature on the Health Effects of Airborne PM

Over the last 10 years, an extensive scientific literature has evolved examining the effects of PM on human health, mostly on acute mortality and hospitalizations. The literature on chronic effects of PM has been less extensive and, for reasons discussed in detail below, less conclusive.¹

3.1.1 The Human Health Effects of Airborne PM

Historically, the acute effects of high level particulate matter exposure episodes on human mortality and morbidity is well documented.² Recent studies have linked PM to acute pulmonary problems (such as bronchitis and asthma) and to cardiovascular problems (such as congestive heart failure and ischemic heart disease), which may result in hospitalization or, in some cases, premature mortality (EPA, 1997). This relationship between PM levels and acute health effects has been well investigated and quantified in the epidemiology literature, and will be discussed in some detail below.

In addition, there is some evidence suggesting that PM may result in a range of other conditions, including changes in pulmonary function, altered host defense mechanisms, cancer, chronic respiratory problems, low birth weight and infant mortality (EPA, 1997). The existing epidemiology literature on these latter end-points remains relatively sparse and inconclusive; we discuss these effects but do not attempt to quantify them here.

A common problem with all epidemiological studies of the effects of PM is that the biological mechanisms linking particulate matter and mortality or morbidity end-points remain unspecified. This has meant that identifying the effect of PM on human health has been completely empirical and data driven, presenting some model specification problems and inconsistencies. In addition,

¹ It is beyond the scope of the current report to provide a comprehensive analysis of the existing scientific literature. An excellent comprehensive summary and analysis of literature on the health effects of airborne particulates is contained in an EPA study entitled Air Quality Criteria for Particulate Matter (1996, EPA 600/P-95/001aF). Interested readers can find this study on the EPA web site at: http://www.epa.gov/nceawww1/.
² For example, see the increased mortality and morbidity in London during the historic air pollution episodes of 1952. (Ministry of Public Health, 1954)
as we discuss below, the existence of numerous confounding factors creates significant empirical uncertainty regarding the magnitude of PM’s effect on human health.

### 3.1.2 Dosimetry and Toxicology of PM Related Health Effects

At present, biological models of the mechanistic processes linking particulate matter inhalation to mortality or morbidity end-points are underdeveloped. It is known that various host factors -- age, ventilation patterns, and the presence of airway disease -- influence predicted particle deposition patterns. It is also known that particle retention is a function of deposition site, clearance of particles, and particle characteristics such as solubility. The lack of information on differences in inhalability of particles, airway geometry and clearance mechanisms hamper extrapolations from studies on laboratory animals is (EPA, 1996). While existing dosimetric studies provide some insight into the characteristics of sub-populations with increased susceptibility to PM related effects, they do not currently provide an adequate biologically based exposure dose-response model. This lack of a physiological model severely constrains our ability to specify the functional form of the models used in epidemiologic studies; it also hampers our efforts to separate out the acute and chronic effects of PM air pollution.

The evidence from toxicological studies is also limited. The evidence here, as with that from dosimetric studies, does aid in the identification of highly susceptible sub-populations. However, the mechanistic link between particulate matter deposition and specific pathological processes is again not well established. The lack of useful dosimetric and toxicological models linking PM exposure to health end-points means that there is little scientific basis for the choice of functional form underlying existing epidemiological studies. Therefore, these studies’ findings must be interpreted with caution.

### 3.1.3 Exposure

Most of the epidemiological studies discussed here rely on community level measurements of ambient outdoor PM levels collected generally from a few monitoring stations as a proxy for individual exposures spread out over a city or county. However, this is at best an inaccurate measure of actual exposure to PM air pollution. In fact, outdoor (ambient) and indoor PM levels may not be well correlated. PM in ambient air enters indoor environments at levels approaching outdoor concentrations. Once indoors, the concentration of ambient PM decreases due to deposition on surfaces, with coarse

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3 Interestingly, the lack of such a model also means that we are completely dependent on epidemiologic studies to assess PM health impacts.

4 Acute effects are most likely related to deposited dose, whereas chronic effects are probably related to cumulative or retained doses, or the process of damage and repair to pulmonary tissue (EPA, 1996). Specifying the functional form of these two effects would allow us to begin distinguishing them in epidemiologic studies.

5 For example, studies have found that exposure to acidic aerosols have little effect on the lung function of healthy adults. However, they do result in decreased lung function in asthmatic subjects, particularly in adolescent asthmatics. Other studies have found that lung defense mechanisms and resistance to infection may be altered by exposure to acidic aerosols (EPA, 1996).
PM having a much higher deposition rate than finer particulates. Human indoor activity can re-suspend previously deposited PM, re-elevating indoor exposure levels. In addition, indoor sources (cooking, smoking, fireplace, vacuuming, dusting, etc.) contribute to overall indoor PM exposure (EPA, 1996).  

Since indoor sources and sinks of PM vary considerably between individuals, a cross-sectional correlation between ambient and individual exposures is often very low (EPA, 1996).  

This implies that reliance on ambient PM levels as a proxy for exposure seriously limits the accuracy of most epidemiological studies that seek to establish a dose-response relationship. First, fine particles typically have a longer atmospheric half-life than coarse particles, meaning that relative to PM\textsubscript{10}, indoor concentrations of PM\textsubscript{2.5} are more representative of ambient PM\textsubscript{2.5} (EPA, 1996). Second, since there is some evidence suggesting that PM\textsubscript{2.5} is responsible for a large portion of PM-related morbidity and mortality, focusing on fine particles may offer more accurate results. However, the fact that its greater atmospheric half-life also means that fine particles can travel greater distances poses a different set of public policy challenges. Finally, since most individuals have relatively consistent day-to-day environments, an individual’s exposure to PM is likely highly autocorrelated over time. That is, an individual’s exposure at one point in time will be a good predictor of his or her exposure at another point. This implies that the errors associated with using ambient PM levels to proxy for exposure will compound themselves over time, making studies of long-term exposure or chronic effects of PM even more problematic.

### 3.1.4 Epidemiological Evidence of Acute Effects

With the above limitations in mind, we now turn to the existing epidemiological evidence of PM health effects. Studies of severe air pollution episodes in Europe and the US in the early half of this century indicate that exposure to high ambient urban air pollution produce marked increases in mortality and morbidity (see, for example the Ministry of Health study). More recent studies in industrializing countries show similar results (see, for example, Wells et al. 1994, Chen et al. 1998).

More than 20 recent time-series analyses of PM health effects demonstrated associations between daily mortality and short-term (24 hour) concentrations of ambient PM. Relative risk estimates for daily mortality in relation to increased short-term ambient PM concentration are generally positive and at least marginally statistically significant. On average these studies suggest about a 1% increase in acute total mortality for a 10 \( \mu g/m^3 \) increase in PM\textsubscript{10}, but estimates range from 0.3% to 1.6% (EPA, 1996). While some variation across the studies can be expected, their wide range — the 95% confidence intervals of the lowest and highest estimates do not overlap — gives cause for caution.  

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\[ 6 \] For example, Lipsett et al. (1997) notes that residential wood combustion during the winter months accounts for a significant amount (as high as 45%) of PM in residential areas of Northern California.  

\[ 7 \] This is particularly likely since most adult in the United States spend the bulk of their time indoors. It is estimated that most individuals spend more than 20 out of 24 hours indoors (EPA, 1996).  

\[ 8 \] More detailed discussion below.
These studies contain relatively little information on acute mortality effects associated with fine particles (PM$_{2.5}$). Recent analysis by Schwartz et al (1996) shows that the relative risk of mortality as a result of increases in PM$_{2.5}$ concentration is between 1.026 to 1.055 per 25 µg/m$^3$ of PM$_{2.5}$, and that this relationship is generally statistically significant. However, a related study found that there was a significant relationship between excess mortality and the concentration of sulfates. Thus, it is not clear if the effects of PM$_{2.5}$ on mortality should be attributed principally to its sulfate fraction, or if there is similar risk associated with the non-sulfate fine particles (EPA, 1996).

As for acute morbidity effects, hospital admissions studies show that hospitalization due to chronic obstructive pulmonary diseases and pneumonia show a moderate but significant increase in relative risks in the range of 1.06 to 1.25 resulting from an increase of 50µg/m$^3$ in PM$_{10}$. Admission studies of acute respiratory disease show similar effects (EPA, 1996). There is speculation of a link between PM concentrations and acute cardiovascular diseases, but the empirical evidence is suggestive yet inconclusive. Finally, most studies of acute morbidity resulting from PM exposure to date were conducted using PM$_{10}$. Very few studies have examined the isolated effects of PM$_{2.5}$. In practice, the high correlation between PM$_{10}$ and PM$_{2.5}$ would make it quite difficult to distinguish the independent effects of a specific class of pollutant.

3.1.5 Epidemiological Evidence of Chronic Effects

While a great deal of evidence suggests long-term or chronic effects from PM exposure, there is relatively little data that can be used to quantify these effects. Existing scientific studies do not adequately allow for a separation of the short-term and long-term effects of pollution exposure.

Epidemiologic findings suggest that short-term PM exposure can trigger acute or terminal health events. Long-term PM exposure, however, could promote life-shortening chronic illnesses. Given the potential for “double counting,” determination of years of life lost to PM exposure is not yet possible. Some epidemiologic analyses suggest that a significant portion of PM-induced acute mortality occurs in people who are already ill (see susceptible groups discussion below) and that these individuals would likely soon die even in the absence of PM exposure (EPA, 1996). Cifuentes and Lave suggest that from 37% to 87% of adult deaths occurring during episodic exposures actually may be premature by only a few days. This complicates the attribution of mortality to specific pollutants or pollution episodes, since the relative risk associated with short-term acute PM exposure may be additive or interact with preexisting chronic excess risks from other pollutants (EPA, 1996).

While chronic PM exposure studies do indicate statistically significant positive associations between excess mortality and fine particle indicators, chronic exposure relative risk estimates are based on PM concentrations only during study periods and therefore do not reflect the full impacts of longer past PM exposure. Given that these long-term exposure studies are fairly recent, and that there has been a secular reduction in the ambient PM levels, we would expect these studies to overstate the long term negative effects of PM exposure.

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9 For example, chronic exposure to PM is linked to increased incidence of chronic obstructive pulmonary diseases, and decreases in pulmonary function (EPA, 1996; EPA, 1997).
Thus, the upper limit for PM-associated life shortening remains undetermined. While epidemiologic evidence suggest that both long-term and short-term PM exposure effects are important in assessing the risk of premature mortality, currently available information does not allow us to quantify the number of years lost to PM associated health events. Therefore, the costs of chronic effects are not estimated in the analyses that follow or in subsequent chapters.

3.1.6 Susceptible Groups

The existing evidence suggests that certain sub-populations are particularly susceptible to acute PM exposure. Generally, the elderly (greater than 65 years of age) and those with cardiopulmonary disease appear to be at greatest risk. It is hypothesized that PM impairs ventilation in individuals with chronic obstructive pulmonary diseases, and may cause increased secretion or increased viscosity of mucus. It is further hypothesized that PM can cause inflammatory responses in individuals with respiratory disease. PM may also increase susceptibility to pulmonary infectious disease, and cardiac arrhythmia may be involved in mortality due to acute PM exposure (EPA, 1996). To the extent that smokers constitute a significant portion of patients with chronic obstructive pulmonary diseases and a smaller but notable portion of cardiovascular disease patients, they represent an increased risk group. Finally, epidemiological studies show that PM exposure can exacerbate existing asthmatic conditions (EPA, 1996).

Unfortunately, few studies have performed a meaningful comparison between the relative risks faced by each of these sub-populations and those faced by the population at large. Thus, at this point, we have no measure of the magnitude of the increased risk faced by the more susceptible groups and no means to quantify differences in overall risks faced by different localities with divergent demographic characteristics.

3.1.7 Other Health Effects

There is some evidence suggesting that both pre- and post-natal exposure to ambient PM effects infant birth weight and mortality. A Chinese study reports a statistically and substantively significant adverse relationship between birth weight and maternal exposure to total suspended PM (Wang et al., 1997). Other studies suggest a significant link between exposure to PM and excess infant mortality (Lave and Seskin, 1977; Lipfert, 1978; Woodruff et al., 1997). The Woodruff et al. study, in particular, links $PM_{10}$ exposure to an increased incidence of sudden infant death syndrome.

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10 There is also an indication that children could be susceptible to the mortality effects of air pollution exposure in general. However, the evidence of PM effects on children is limited, and somewhat conflicting (see, for example, Schwartz, 1994; Saldiva et al., 1994).

11 One exception is a study conducted in Chile by Ostro et al., showing that the relative risks of PM exposure faced by the elderly population from was 40% higher than that faced by the entire population during the same period in the same region (1996).
Additional evidence suggests that PM exposure over time can alter lung function, lung tissue and structure, airway responsiveness, and respiratory defense mechanisms, and can increase susceptibility to respiratory infection and damage respiratory cells (EPA, 1996; EPA, 1997). Although the evidence in each of these areas is not yet conclusive enough to quantify excess deaths or relative risks, the omission of these and other unquantifiable health risks from the last part of this chapter is likely create a downward bias in our estimates of the total health impact of PM exposure.

3.1.8 Confounding Factors

It is often difficult to distinguish the effects of particulate matter from those of other criteria pollutants. Many of the processes producing PM also produce these other pollutants, and some pollutants act as precursors to secondary formation of PM. Many studies find that concentrations of other known air pollutants, such as NOx, SOx, CO, and O3, are highly correlated with PM concentrations. In addition, weather conditions and other factors affecting PM emissions or concentrations in certain geographic areas are likely to affect the concentration of other precursor pollutants similarly (EPA, 1996). Since PM does not have a unique chemical composition, there is no biological marker corresponding to the full range of health effects that would allow us to distinguish the effects of PM from other pollutants.

It is important to note that other air pollutants generally have not yet been addressed in studies estimating the increased mortality/morbidity effects of PM. In those few studies that have included other criteria pollutants, their inclusion generally decreases the relative risks of PM, although in many of these the relative risks of PM exposure remain statistically higher than background risks. There were typically only small differences among many pollutants, in part due to the collinearity of concentration levels. Thus, it is extremely difficult to separate out the PM specific effects from the confounding effects of co-pollutants. This problem is particularly severe because we do not know if the effect of PM exposure is additive and/or interactive with other pollutants exposure effects. It is therefore rather difficult to model the behavior of different pollutant mixtures (EPA, 1996). In addition, the PM averaging period used in studies of acute mortality/morbidity effects (e.g., 24 hours, 2 days, 3 days, etc.) affected the significance of PM exposure (EPA, 1996). However, given the lack of biologically based models, it is difficult to assess the appropriate exposure-time in measuring the acute effects of PM exposure.

A final point of importance is that the proportion of fine particulate matter (PM2.5) in PM10 may be a more useful indicator of health effects than the aggregate measure of PM10. The fine fraction appears to contain a large proportion of those reactive pollutants linked to specific health effects. In addition, the PM2.5 fraction accounts for the largest number of particles and the largest aggregate surface area, so that its potential for absorption and deposition in human tissue is especially high (EPA, 1997). However, with the noted exception of the study conducted by Schwartz et al (1996), few epidemiological studies have distinguished the effects of PM2.5 from those of PM10 more generally. In any case, the high degree of correlation between PM2.5 and PM10 makes it

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12 Weather conditions, such as temperature and humidity, affect the physical and chemical behavior particles, as well as human responses to them. (Asthma, for example, tends to be aggravated under certain weather conditions.) However, in the absence of models of the behavior of particulate matter and biological reactions under different weather conditions, it is difficult to separate these effects. (See for example Lipsett et al., 1997; Maier et al., 1997)
very difficult to attribute specific adverse health effects to one portion or another through purely empirical epidemiological studies.

3.1.9 Methodological Issues, Accuracy and Uncertainty

While there exists a consensus that PM poses a human health risk, estimates of the magnitude of this excess risk differ considerably. For example, the relative risk of premature mortality associated with short-term PM exposure typically occupies a range from 1.003 to 1.016 per 10 µg/m³ of PM₁₀ (or 0.3% to 1.6% increase in risk of premature death). While the confidence intervals for these relative risk estimates overlap, they nonetheless represent a factor of 5 difference between the lowest estimate to the highest. Reflecting this uncertainty (and other methodological difficulties of assessing economic impact), the *Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule* notes, without apology:

Partial attainment of the selected PM₂.₅ standard results in estimated monetized annual benefits in a range of $19 to $104 billion per year incremental to the current PM₁₀ standard, including 3,300 to 15,600 incidences of premature mortality avoided. ... The mortality benefits represent about 12% to 70% of the benefits estimates (EPA, 1997; emphasis added).

Indeed, the fact that these studies produce wide variations in the estimated effects of PM exposure despite using similar data is rather troubling. As noted above, there may be genuine confounding factors to explain these discrepancies. The same EPA study is careful to note:

Estimation of public health impacts of ambient airborne particle exposures in the US would most credibly require use of relative risk estimates derived for particular US urban areas, in combination with estimates of exposures to ambient particle concentrations for general population and/or specific susceptible subgroups within those particular areas. In view of geographic differences in ambient PM mixtures, and demographics, broad generalization and application of some single “best estimate” of relative risk for a given increment in concentration of a given particle indicator would be subject to much uncertainty.” (EPA, 1996)

Nonetheless, there is a small but real possibility that the health effects observed in these studies are merely statistical artifacts. The lack of a biologically based mechanistic model has meant that there is no scientific basis for determining a generally acceptable functional form of the exposure-response model used in the epidemiologic studies. In the absence of such a model, most studies use specifications that best fit the data. The state of the science is unclear on even the most basic issues of model specification, such as choosing between linear and non-linear versus threshold models. However, such data driven model specification is generally frowned upon with good reason — it is well established in statistics and econometrics that data driven specifici-

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13 The EPA study notes that while there is little evidence that there is a threshold effect even at very low concentrations, the “evidence for or against threshold effects or other non-linearities in response is as of yet equivocal” (EPA, 1996). However, the lack of accurate information about individual exposure levels greatly hampers the ability to carry out meaningful analysis regarding potential threshold effects.
cation searches (data mining) can distort the significance level of the estimated parameters, and find spurious statistical relationships where no causal relationship exist in reality.\footnote{At least one study is critical of the existing literature on precisely this point. A paper by Gamble and Lewis argues that recent epidemiology studies regarding the adverse mortality effects of PM exposure do not meet the criteria for establishing causality (1996).}

In addition to these model specification issues, other statistical considerations of note include the representativeness of mean concentration-response functions, mean population statistics, and base line incidence rates. All of these serve to make statistical inferences about excess mortality and morbidity related to PM exposure more uncertain.

Clearly, the state of the science regarding the health effects of PM is inexact. While we take some comfort in the fact that the findings of a positive relationship between excess mortality/morbidity and PM exposure is fairly robust to changes in the specification of underlying models, we need to interpret these findings with caution. Resolution of this problem requires the development of a comprehensive biologically based exposure dose-response model — which is unfortunately some time away.

### 3.2 The Health Effects of PM$\textsubscript{10}$ Exposure in New Jersey

In the remainder of this chapter we attempt to quantify the health effects of PM exposure in New Jersey. Because quantitative information on the chronic effects of PM exposure is incomplete, we focus exclusively on acute mortality and morbidity to provide three estimates (low, central and high) for several expected mortality and morbidity effects based on daily PM$\textsubscript{10}$ and PM$\textsubscript{2.5}$ concentrations. We base our estimates on aggregate “national” data from existing epidemiological studies, without controlling for the effects of differential exposure, confounding variables or demographic factors specific to New Jersey. The limitations of these estimates should therefore be clearly understood.\footnote{The collection of additional New Jersey specific data would improve the credibility of this assessment considerably.}

For rural concentrations of particulate matter, we use readings from the monitoring station at Brigantine. For the urban concentrations, we average data from the Camden, Newark, and Elizabeth stations to obtain a single set of urban concentrations. Since no daily data are available for PM$\textsubscript{2.5}$, we employ a simple extrapolation based on the ratio of PM$\textsubscript{10}$ to PM$\textsubscript{2.5}$. Finally, in calculating the mortality and morbidity figures, a straight daily risk is assumed; that is, there is no overlap or averaging of concentrations across days. The estimates of PM$\textsubscript{2.5}$-related mortality rely on the relative risk figures reported by Schwartz et al. (1996). A summary of our results is presented below.
3.2.1 Mortality

Table 3-1: Assumptions in Calculating Morbidity and Mortality

<table>
<thead>
<tr>
<th></th>
<th>Urban Population in New Jersey</th>
<th>Rural Population in New Jersey</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expected Urban</td>
<td>6,910,000</td>
<td>820,000</td>
</tr>
<tr>
<td>Nonaccidental Mortality</td>
<td>104</td>
<td>12</td>
</tr>
<tr>
<td>Baseline Daily</td>
<td>1.5 x 10^{-5}</td>
<td></td>
</tr>
<tr>
<td>Mortality Rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ratio of PM_{2.5}/PM_{10}</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>RR (25 µg/m³ increase in PM_{2.5}) (Schwartz 1996)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minimum</td>
<td>1.026</td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>1.04</td>
<td></td>
</tr>
<tr>
<td>Maximum</td>
<td>1.055</td>
<td></td>
</tr>
<tr>
<td>RR (per 10 µg/m³ increase in PM_{10}) (EPA meta-study)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minimum</td>
<td>1.003</td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>1.01</td>
<td></td>
</tr>
<tr>
<td>Maximum</td>
<td>1.016</td>
<td></td>
</tr>
</tbody>
</table>

Based on New Jersey population data, a PM_{2.5}/PM_{10} ratio of 0.5, and the relative risk figures reported by Schwartz et. al. (1996), the baseline daily mortality in New Jersey due to PM is 1.5 x 10^{-5}. In other words, we expect 15 additional deaths per million people over an average lifetime of 70 years due to exposure to PM. This translates to 104 expected urban deaths and 12 expected rural deaths. For basis of comparison against the expected values, the actual values calculated based on the PM_{10} concentrations are as follows:

Table 3-2: Relative Risk (RR) of Mortality Due to PM Exposure in New Jersey

<table>
<thead>
<tr>
<th></th>
<th>RR (per 10 µg/m³ increase in PM_{10})</th>
<th>RR (per 25 µg/m³ increase in PM_{2.5})</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.003</td>
<td>0.026</td>
</tr>
<tr>
<td>Average</td>
<td>0.010</td>
<td>0.040</td>
</tr>
<tr>
<td>High</td>
<td>0.016</td>
<td>0.055</td>
</tr>
</tbody>
</table>

The relative risk figures range from 0.3% to 1.6% increased risk due to a 10µg/m³ increase for PM_{10}, and from 2.6% to 5.5% increased risk due to a 25 µg/m³ increase in PM_{2.5}. Applying these relative risk figures to New Jersey’s population, the following results are calculated.

Table 3-3: Expected Excess Deaths from Particulate Matter Exposure in New Jersey

<table>
<thead>
<tr>
<th></th>
<th>Excess Deaths from PM_{2.5}</th>
<th>Excess Deaths from PM_{10}</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Urban</td>
<td>Rural</td>
</tr>
<tr>
<td>Low</td>
<td>668</td>
<td>47</td>
</tr>
<tr>
<td>Average</td>
<td>1,027</td>
<td>72</td>
</tr>
<tr>
<td>High</td>
<td>1,412</td>
<td>99</td>
</tr>
</tbody>
</table>
3.2.2 Morbidity Data

For the morbidity results, we applied the relative risk coefficients to data for respiratory- and cardiac-related hospital admissions and emergency room visits. Based on the risk coefficients calculated above and the morbidity data, we calculated the following the number of hospital visits due to the categories above.

<table>
<thead>
<tr>
<th>New Jersey Urban Areas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urban Population</td>
</tr>
<tr>
<td>Respiratory Hospital Admissions</td>
</tr>
<tr>
<td>Cardiac Hospital Admissions</td>
</tr>
<tr>
<td>Emergency Room Visits</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>New Jersey Rural Areas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rural Population</td>
</tr>
<tr>
<td>Respiratory Hospital Admissions</td>
</tr>
<tr>
<td>Cardiac Hospital Admissions</td>
</tr>
<tr>
<td>Emergency Room Visits</td>
</tr>
</tbody>
</table>

3.3 Summary of Findings

The epidemiological literature reviewed here indicates a suggestive link between PM$_{10}$ air pollution and acute pulmonary and cardiovascular problems that may result in hospitalization and even premature mortality. There is also some evidence linking airborne particulates to altered pulmonary and immune functions, chronic respiratory problems, low birth weight, infant mortality, and certain cancers. As the available data are sparse, we do not attempt to quantify these effects.

Our ability to develop credible dose-response models is constrained by several additional factors. One difficulty we known precious little about the physiological mechanisms through which PM-related morbidity and mortality is induced. Another is that measured ambient PM$_{10}$ levels may have little relation to the actual pollution levels individuals are exposed, especially since they spend so much time on average indoors. The principal findings of the literature are as follows.

- **Acute mortality:** Studies suggest that a 10µg/m$^3$ increase in PM$_{10}$ results in approximately a 1% increase in total acute mortality. However, the range of estimates is so wide that the 95% confidence intervals of the lowest and highest estimates do not overlap.

- **Acute Mortality Associated With Fine Particles:** Data on the specific effects attributable to PM$_{2.5}$ are inconclusive.

- **Acute Morbidity:** A 50µg/m$^3$ increase in PM$_{10}$ is associated with a moderate but significant increase in relative risks for chronic obstructive pulmonary diseases and pneumonia,
in the range of 1.06 to 1.25. Similar effects are evident for acute respiratory disease. The data on acute cardiovascular disease are inconclusive.

- **Acute Morbidity Associated With Fine Particles:** Due to the high correlation between PM$_{10}$ and PM$_{2.5}$, it is difficult to distinguish the independent effects of fine particles.

- **Chronic Morbidity:** While epidemiologic evidence suggests that PM exposure may be associated with premature mortality due to chronic illness, currently available information does not allow us to quantify the number of years lost to PM-related ailments.

- **Susceptible Populations:** Although data does suggest that certain groups -- including the elderly, infants, children, and those with cardiopulmonary disease -- are particularly susceptible to the effects of PM exposure, we are unable to quantify the magnitude of the increased risk faced by these groups.

- **Predicted Excess Deaths in New Jersey:** Using a low, medium, and high relative risk (RR) figure, we estimate that acute effects of a 10 µg/m$^3$ increase in PM$_{10}$ exposure would produce between 385 and 2054 excess deaths per year in urban areas and between 27 and 144 excess deaths in rural areas. A similar analysis for an increase of 25 µg/m$^3$ in PM$_{2.5}$ predicts between 668 and 1412 excess deaths in urban areas and 47 to 99 excess deaths in rural areas.

- **Predicted Pollution-Related Acute Morbidity in New Jersey:** Comparing data on daily PM$_{10}$ concentrations with various morbidity indicators over the course of a year, we estimate that PM$_{10}$ pollution is responsible for approximately 1,212 respiratory hospital admissions, 565 cardiac hospital admissions, and 55,426 emergency room visits per year in urban areas; for rural areas, these estimates are 170, 79, and 3,882, respectively.

- **Confounding factors:** Many exist, including the high correlation between PM$_{10}$ and other pollutants; the lack of biologically based models upon which to ground decisions about collecting and categorizing data; and the difficulty of distinguishing the effects PM$_{2.5}$ -- those particles most likely responsible for severe health effects -- from the larger category of PM$_{10}$.

- **Methodological difficulties:** Several limit our ability to draw meaningful conclusions from our and others’ analyses. For example, since most individuals have relatively consistent day-to-day environments, an individual’s exposure to PM$_{10}$ is likely to be highly autocorrelated over time. Thus errors are likely to be compounded over time in long-term studies. Another source of confusion is the difficulty of distinguishing among types of premature deaths. A substantial portion of the deaths associated with exposure to high PM$_{10}$ may be premature by only a few days and it is difficult to compare this sort of risk with that of dramatically decreased life expectancies due to long-term pollution-induced morbidity. Finally, we have little understanding of the possible synergies between the chronic effects of pollution exposure and the acute effects of short-term high exposure.
REFERENCES FOR SECTION 3

Chen, Pau-Chung et al., 1998. “Adverse Effect of Air Pollution on Respiratory Health of Primary School Children in Taiwan” in Environmental Health Perspectives 106.


