Chapter 18: Public Health

A. INTRODUCTION

This chapter presents an assessment of the potential health concerns related to air quality and noise during the construction and operational phases of the proposed project. During construction, potential health impacts due to noise and air pollutant emissions can stem from construction equipment and construction vehicles. Potential health effects during operations are related to noise and pollutant emissions from traffic, as well as pollutant emissions from heating, ventilation and air conditioning (HVAC) systems. Of particular concern is the potential for diesel emissions from construction-related activities to impact public health (such as increasing asthma rates). Therefore, this chapter also provides an overview of health concerns related to traffic and construction equipment, particulate matter (PM) emissions, and a discussion of asthma, its prevalence in New York City and the area most likely affected by the proposed project.

PRINCIPAL CONCLUSIONS

Since the issuance of the DEIS, the building program of the proposed project has been modified to reflect a reduction in the proposed program. These modifications do not result in changes to the conclusions of the public health analysis. No significant adverse impacts to public health are anticipated as a result of the proposed project.

AIR QUALITY

As set forth in Chapter 14, "Air Quality," and the air quality analysis in Chapter 17, "Construction," the project would result in no significant adverse impacts on air quality for any of the relevant pollutants. The discussion in this chapter focuses on $PM_{2.5}$ as explained below.

During construction, predicted exceedances of the New York State Department of Environmental Conservation's (NYSDEC's) $PM_{2.5}$ interim guidance thresholds would be limited in extent, duration and severity. The increments in excess of the thresholds were predicted to be highly localized, i.e., almost entirely due to construction activities in close proximity to the affected location and not a result of cumulative impacts from the larger project site. At a small number of sidewalk or ground-floor residential locations, exceedances of the annual threshold were predicted, but only for a single year of construction. Short-term exceedances of the thresholds were not predicted for more than a single day at any one ground-floor residential location throughout duration of construction. At sidewalk locations where short-term exceedances were predicted (all of which would be in close proximity to the site), the exceedances would occur multiple days in a year of construction, but not for the entire construction period. The levels of $PM_{2.5}$ increment are comparable to increments predicted for many standard small-scale construction operations and would be much lower than those associated with standard construction operations of a similar size due to the incorporation into the proposed project of extensive measures to reduce $PM_{2.5}$ emissions.

During project operations, the results of the air quality analysis showed that near study area intersections for mobile sources, and at any off-site receptor location for stationary sources, the maximum 24-hour and annual incremental concentrations would be less than the applicable NYSDEC interim guidance criteria. While modeled increments were predicted to exceed the annual threshold at some locations on the exterior of on-site buildings, the potential exposure to $PM_{2.5}$ at these locations would be limited since occupants would not be expected to have their windows open continuously and be exposed to outdoor concentrations throughout the year (boiler emissions are highest in the winter when windows would least likely be opened).

During both the construction and operational periods, the predicted neighborhood-scale average incremental concentrations from the proposed project were less than the applicable interim guideline concentration. Therefore, no significant adverse impacts on public health from $PM_{2.5}$ emissions would be expected from the construction or operation of the proposed project.

NOISE

While potential noise impacts during construction were determined to be significant at certain locations, the noise levels produced by construction activities are low for construction of a project of this magnitude and would be below those typically experienced by residents living adjacent to large construction projects, because of the project sponsors' commitment to implement extensive measures during project construction to reduce the noise levels emanating from the project site. During construction, noise levels in the existing Brooklyn Bear's Community Garden, South Oxford Park, and Dean Playground would be above the *CEQR Technical Manual's* impact criteria for noise levels. During operations, noise levels at Dean Playground and the proposed project's new open spaces areas created on-site would also experience levels above the CEQR impact criteria, but would be comparable to noise levels in a number of open space areas, and parks in New York City, including Hudson River Park, Riverside Park, Bryant Park, Fort Greene Park, and other urban open space areas. Potential significant adverse impacts during operation on certain streets would result in noise levels that fall in the marginally unacceptable range, which is not unusual for New York City residential areas. The overall changes in noise level due to the project are not of a magnitude that would significantly affect public or mental health. Therefore, no significant adverse health impacts due to noise are expected due to construction and operation of the proposed project.

B. METHODOLOGY

For determining whether a public health assessment is appropriate, the 2001 *City Environmental Quality Review (CEQR) Technical Manual* lists the following as public health concerns for which a public health assessment may be warranted:

- Increased vehicular traffic or emissions from stationary sources resulting in significant adverse air quality impacts;
- Increased exposure to heavy metals (e.g., lead) and other contaminants in soil/dust resulting in significant adverse impacts;
- The presence of contamination from historic spills or releases of substances that might have affected or might affect groundwater to be used as a source of drinking water;
- Solid waste management practices that could attract vermin and result in an increase in pest populations (e.g., rats, mice, cockroaches, and mosquitoes);

- Potentially significant adverse impacts to sensitive receptors from noise or odors;
- Vapor infiltration from contaminants within a building or underlying soil (e.g., contamination originating from gasoline stations or dry cleaners) that may result in significant adverse hazardous materials or air quality impacts;
- Actions for which the potential impact(s) result in an exceedance of accepted federal, state, or local standards; or
- Other actions, which might not exceed the preceding thresholds, but might, nonetheless result in significant public health concerns.

Because the proposed project is recognized to be of a large scale, has an anticipated construction period of approximately 10 years, and is adjacent to heavily-trafficked intersections in New York City, this chapter provides an assessment of the potential health concerns related to air quality and noise during the construction and operational phases of the proposed project.

The public health assessment first identifies the pollutants of concern relating to air quality, then outlines the applicable standards and thresholds to which potential emissions from construction and operational activities associated with the proposed project will be compared. A description of the sources of air and noise pollutants during construction and operation are then presented, followed by a literature review of the health effects associated with diesel engine exhaust and emissions of PM in particular.

Given the public's concern over asthma rates in New York City, and concern that exposure to PM emissions could aggravate or induce asthma episodes in an individual, this chapter also provides an in-depth review of relevant asthma-related studies, provides an overview of the prevalence of asthma in New York City, and presents current asthma hospitalization data for neighborhoods representing the potentially affected population surrounding the proposed project.

A summary of the air quality and noise impact assessments during the construction and operational periods of the proposed project is then presented, and the potential for public health impacts due to the proposed project is determined. Rodent control measures during construction are also summarized.

C. POLLUTANTS OF CONCERN

PARTICULATE MATTER

PM is a broad class of air pollutants that exist as liquid droplets or solids, with a wide range of sizes and chemical composition. Generally, airborne concentrations of PM are expressed as the total mass of all material (often smaller than a specified aerodynamic diameter) per volume of air (in micrograms per cubic meter, $\mu g/m^3$). Thus, PM_{10} refers to suspended particles with diameters less than 10 μm, and $PM_{2.5}$ to suspended particles with diameters less than 2.5 μm.

PM is emitted by a variety of natural and man-made sources. Natural sources include the condensed and reacted forms of natural organic vapors, salt particles resulting from the evaporation of sea spray; wind-borne pollen, fungi, molds, algae, yeasts, rusts, bacteria; debris from live and decaying plant and animal life; particles eroded from beaches, desert, soil and rock; particles from volcanic and geothermal eruptions; and, forest fires.

Major man-made sources of PM include the combustion of fossil fuels, such as vehicular exhaust, power generation and home heating, chemical and manufacturing processes, all types of

construction, agricultural activities and wood-burning fireplaces. Since the chemical and physical properties of PM vary widely, the assessment of the public health effects of the airborne pollutants in ambient air is extremely complicated.

$PM_{2.5}$

As mentioned above, PM is a byproduct of fossil fuel combustion. It is also derived from mechanical breakdown of coarse particulate matter such as pollen fragments. $PM_{2.5}$ does not refer to a single pollutant, but to an array of fine inhalable materials. There are, for example, thousands of forms of natural ambient $PM_{2.5}$ and perhaps as many forms of man-made $PM_{2.5}$, which include the products of fossil fuel combustion (such as diesel fuel), chemical/industrial processing, and burning of vegetation. While all the disparate forms of $PM_{2.5}$ can be inhaled, their toxicological properties can differ. Some PM is emitted directly to the atmosphere (i.e., primary PM), while other types of PM are formed in the atmosphere through various chemical reactions and physical transformations (i.e., secondary PM). The formation of secondary $PM_{2.5}$ is one determinant of ambient air quality and is, thus far, extremely difficult to model.

The major constituents of $PM_{2.5}$ are typically sulfates, nitrates, organic carbon, elemental carbon (soot), ammonium, and metallic elements (not including sulfur). Secondary sulfates and nitrates are formed from their precursor gaseous pollutants, SO_2, and NO_x at some distance from the source due to the time needed for the chemical conversion within the atmosphere. Elemental carbon and metallic elements are components of primary PM, while organic carbon can be either emitted directly from a source or formed as a secondary pollutant in the atmosphere. Due to the influence of these "secondary" pollutants from distant or regional sources, regional ambient levels of $PM_{2.5}$ are typically more evenly distributed than their related class of pollutants PM_{10}, which is more highly influenced by local sources.

Data from the Botanical Gardens in the Bronx and Queens College in Queens, New York City indicate that the greatest contributors to ambient $PM_{2.5}$ concentrations are sulfates and organic carbon (approximately two thirds of the total $PM_{2.5}$ mass). Studies confirming the contribution of long-range transport to ambient $PM_{2.5}$ levels compared the data from New York City monitors to monitors from a remote site within the state, downwind from other states. These data show that high levels of sulfate and other pollutants come into New York State from areas to the west and south of New York. The data also indicate that urban sites are more likely to experience increased nitrate and carbon levels than rural sites.[1]

D. BENCHMARKS FOR DETERMINING THE SIGNIFICANCE OF AIR QUALITY AND NOISE IMPACTS

The potential public health impacts of PM emissions and noise levels due to the proposed project are based on the results of the air quality and noise impact assessments presented in Chapters 14, "Air Quality," 15, "Noise," and 17, "Construction Impacts." The following discussion presents the applicable standards and thresholds to which the results of the air quality and noise modeling are compared in determining the significance of air quality impacts.

[1] New York State Department of Environmental Conservation (NYSDEC), Report to the Examiners on Consolidated Edison's East River Article X Project, Case No. 99-F-1314, February, 2002.

AIR QUALITY

As mentioned in Chapter 14, "Air Quality," the State Environmental Quality Review Act (SEQRA) regulations and the *City Environmental Quality Review (CEQR) Technical Manual* state that the significance of a likely consequence (i.e., whether it is material, substantial, large or important) should be assessed in connection with:

1) Its setting (e.g., urban or rural);
2) Its probability of occurrence;
3) Its duration;
4) Its irreversibility;
5) Its geographic scope;
6) Its magnitude; and
7) The number of people affected.

In terms of the magnitude of air quality impacts (bullet 6 above), any action predicted to increase the concentration of a criteria air pollutant to a level that would exceed the concentrations defined by the NAAQS would be deemed to have a potential significant adverse impact. In addition, to maintain concentrations lower than the NAAQS in attainment areas, or to ensure that concentrations will not be significantly increased in non-attainment areas, threshold levels have been defined for certain pollutants. Any action predicted to increase the concentrations of these pollutants above the thresholds would be deemed to have a potential significant adverse impact, even in cases where violations of the NAAQS are not predicted, requiring a detailed analysis of air quality impacts for that pollutant.

THE NATIONAL AMBIENT AIR QUALITY STANDARD FOR $PM_{2.5}$

Section 108 of the Clean Air Act (CAA) directs EPA to identify criteria pollutants that may reasonably be anticipated to endanger public health and welfare. Section 109 of the CAA requires the EPA to establish National Ambient Air Quality Standards (NAAQS) and periodically revise them for such criteria pollutants. Primary NAAQS are mandated to protect public health with an adequate margin of safety. In setting the NAAQS, EPA must account for uncertainties associated with inconclusive scientific and technical information, and potential hazards not yet identified. The standard must also be adequate to protect the health of any sensitive group of the population. Secondary NAAQS are defined as standards that are necessary to prevent adverse impacts on public welfare, such as impacts to crops, soils, water, vegetation, wildlife, weather, visibility, and climate.

Beginning in 1994, EPA conducted a five-year review of the NAAQS for particulate matter, which included an in-depth examination of epidemiologic and toxicological studies. EPA also held public meetings across the nation and received over 50,000 oral and written comments regarding these studies, particularly as to whether $PM_{2.5}$ is correlated with adverse health effects, and at what ambient air concentrations of $PM_{2.5}$ these correlations hold. The studies are summarized in EPA's Criteria Document for Particulates, Chapters 10-13 (1996); EPA's Staff Papers on Particulates, in particular Chapter V[1]; and EPA's proposed NAAQS for particulates,

[1] Many of the studies are found on EPA's website at http://www.epa.gov/ttn/oarpg/t1sp.html.

found in the December 13, 1996 Federal Register on page 65638. Based on this extensive analysis, in June of 1997, EPA revised the NAAQS for particulate matter and proposed a new standard for $PM_{2.5}$ consisting of both a long-term (annual) limit of 15 $\mu g/m^3$ and a short-term (24-hour) limit of 65 $\mu g/m^3$.[1]

In establishing the NAAQS for $PM_{2.5}$ in 1997, EPA conservatively assumed that moderate levels of airborne PM of any chemical, physical, or biological form might harm health. In setting the value of the annual average NAAQS for $PM_{2.5}$, EPA found that an annual average $PM_{2.5}$ concentration of 15$\mu g/m^3$ is below the range of data most strongly associated with both short- and long-term exposure effects. The EPA Administrator concluded that an annual NAAQS of 15$\mu g/m^3$ "would provide an adequate margin of safety against the effects observed in the epidemiological studies."[2]

Since the DEIS was published, EPA has revised the NAAQS for PM, effective December 18, 2006. The revision included lowering the level of the 24-hour $PM_{2.5}$ standard from the current level of 65 $\mu g/m^3$ to 35 $\mu g/m^3$, and retaining the level of the annual $PM_{2.5}$ standard at 15 $\mu g/m^3$.

INTERIM GUIDANCE CRITERIA (THRESHOLD LEVELS) REGARDING $PM_{2.5}$ IMPACTS

In addition to the NAAQS, the New York City Department of Environmental Protection (DEP) has promulgated an interim guidance for $PM_{2.5}$, a threshold value that is used for comparison when determining potential significance of air quality and public health impacts.[3] The interim guidance requires a $PM_{2.5}$ neighborhood analysis for actions that have potential for a significant impact. In the neighborhood analysis, an area of 1 km^2, centered at the maximum predicted ground-level concentration, is considered. According to the interim guidance, actions should not exceed an average annual $PM_{2.5}$ concentration increment of 0.1 $\mu g/m^3$ within the 1 km^2 area considered. To put this value in perspective: 0.1 $\mu g/m^3$ constitutes less than one percent of the annual NAAQS for $PM_{2.5}$. A concentration increment that is lower than the incremental neighborhood guidance concentration would not be registered by the ambient air monitors. $PM_{2.5}$ impacts below this threshold are considered to be insignificant with regards to public health impacts.

NOISE

As discussed in Chapter 15, "Noise," noise levels associated with the construction and operation of the proposed project would be subject to the emission source provisions of the New York City

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[1] 62 Federal Register 38652 (July 18, 1997).

[2] 62 Federal Register 28652, 38676 (July 18, 1997).

[3] NYSDEC has also published a policy to provide interim direction for evaluating $PM_{2.5}$ impacts. This policy would apply only to facilities applying for permits or major permit modification under the State Environmental Quality Review Act (SEQRA) that emit 15 tons of PM_{10} or more annually. The policy states that such a project will be deemed to have a potentially significant adverse impact if the project's maximum impacts are predicted to increase $PM_{2.5}$ concentrations by more than 0.3 $\mu g/m^3$ averaged annually, or more than 5 $\mu g/m^3$ on a 24-hour basis (these thresholds have also been referenced by DEP in its interim guidance policy). The proposed project's annual emissions of PM_{10} are estimated to be well below the 15 ton per year threshold under the NYCDEC's $PM_{2.5}$ guidance. The DEP community-based threshold of 0.1$\mu g/m^3$ is considered more relevant and appropriate when determining potential public health impacts than the above-mentioned NYSDEC thresholds, since it represents maximum ground-level concentrations averaged over a wider "neighborhood-scale" area.

Noise Control Code and to Noise Standards set for the CEQR process. Construction equipment is regulated by the Noise Control Act of 1972 and the New York City Noise Control Code.

E. SUMMARY OF AIR AND NOISE POLLUTION SOURCES

CONSTRUCTION

AIR QUALITY

Construction activities have the potential to impact public health as a consequence of emissions from on-site construction engines as well as emissions from on-road construction related vehicles and their impact on traffic conditions. In general, most construction engines are diesel powered, and produce relatively high levels particulate matter. Construction activities also emit fugitive dust. Impacts on traffic could also increase mobile source-related emissions.

In recognition of the potential construction-related air quality and public health effects of emissions from diesel engines, the project sponsors have committed to implementing a state-of-the-art emissions reduction program, as detailed in Chapter 17, "Construction Impacts."

In addition, to address health and safety procedures which minimize exposure to workers and the public to airborne dust and volatile organic compounds during construction activities, a Construction Health and Safety Plan (CHASP) would be prepared in accordance with Occupational Safety and Health Administration (OSHA) regulations and guidelines, also detailed in Chapter 17, "Construction Impacts."

Additional measures would be taken to reduce pollutant emissions during construction in accordance with all applicable laws, regulations, and building codes. These include dust suppression measures and the restriction of on-road vehicle idle time to three minutes for all vehicles that are not using the engine to operate a loading, unloading or processing device (e.g., concrete mixing trucks).

NOISE

Community noise levels during construction of the proposed project can result from noise and vibration from construction equipment operation and from construction vehicles and delivery vehicles traveling to and from the site. Noise levels caused by construction activities would vary widely, depending on the phase of construction and the location of the construction relative to receptor locations. The most significant construction noise sources related to the proposed project are expected to be impact equipment such as jackhammers, impact wrenches, and paving breakers, as well as the movements of trucks and cranes.

PROJECT OPERATIONS

AIR QUALITY

The primary source of mobile source pollutant emissions during project operations would be from project-generated vehicles using nearby intersections in the study area. The proposed project would increase traffic in the vicinity of the project site and along feeder streets to and from the project study area, potentially increasing pollutant emissions.

Potential stationary source emissions associated with operation of the proposed project would primarily be from fuel burned on site for heating, ventilation, and air conditioning (HVAC) systems.

NOISE

The primary source of noise during project operations (i.e., when construction of Phase I and Phase II have been completed in the years 2010 and 2016, respectively) would be attributable to increased traffic in the area stemming from the proposed project.

F. AIR QUALITY-RELATED HEALTH EFFECTS

Scientists have been studying possible links between various health effects, particularly respiratory diseases or symptoms, such as cough, asthma, and bronchitis, and traffic sources of air pollution. The toxic effects of diesel engine exhaust, in particular, have been evaluated in numerous studies. Increases in airborne particle matter (PM) emitted by such sources may account for potential impacts on public health. The following section provides a general discussion of the health effects from traffic and construction equipment sources of air pollution, such as engine exhaust, then focuses specifically on the characteristics of PM, especially $PM_{2.5}$ (suspended particles with diameters less than 2.5 μm) and the public health effects related to human exposure to airborne concentrations of $PM_{2.5}$. Because New York City, and the project area in particular, are considered high-density areas with asthma rates that are generally higher than in less urban areas, a detailed discussion of asthma is presented, including its prevalence in New York City and the area most likely to be affected by the proposed project.

DIESEL ENGINE EXHAUST

The United States Environmental Protection Agency's (EPA's) *Health Assessment Document for Diesel Engine Exhaust, 2002,* evaluates available evidence of the health hazards associated with exposure to diesel engine exhaust (DE).[1] The assessment categorizes the possible health hazards as either acute (short-term exposure) effects, chronic (long-term exposure) noncancer respiratory effects, or chronic (long-term exposure) carcinogenic effects.

EPA's assessment notes that there is available, but limited, human and animal evidence to suggest that exposure to diesel exhaust can cause acute irritation (e.g., eye, throat, and bronchial), neurophysiological symptoms (e.g., lightheadedness and nausea), and respiratory symptoms (e.g., cough, and phlegm). There is also evidence of the exacerbation of allergenic responses to known allergens and asthma-like symptoms.

Toxicological information from human studies does not provide a definitive evaluation of possible noncancer health effects; however, there is extensive animal evidence. Based on the available animal evidence, EPA has concluded that diesel exhaust exposure may pose a chronic respiratory hazard to humans. In several animal species, including rats, mice, hamsters and monkeys, chronic-exposure animal inhalation studies show a range of dose-dependent inflammation and histopathological changes in the lungs.

[1] EPA National Center for Environmental Assessment, 2002, *Health Assessment Document for Diesel Engine Exhaust*, EPA/600/8-90/057F.

Based on the evaluation of evidence from human, animal, and other supporting studies, EPA has concluded that diesel engine exhaust is "likely to be carcinogenic to humans by inhalation" and that this hazard applies to environmental exposures. EPA's assessment states that:

> *Although the available human evidence shows a lung cancer hazard to be present at occupational exposures that are generally higher than environmental levels, it is reasonable to presume that the hazard extends to environmental exposure levels.*
>
> *Given a carcinogenicity hazard, EPA typically performs a dose-response assessment of the human or animal data to develop a cancer unit risk estimate that can be used with exposure information to characterize the potential cancer disease impact on an exposed population. The DE human exposure-response data are considered too uncertain to derive a confident quantitative estimate of cancer unit risk, and with the chronic rat inhalation studies not being predictive for environmental levels of exposure, EPA has not developed a quantitative estimate of cancer unit risk.*

Although there is convincing evidence for potential human health hazards related to diesel engine exhaust, EPA's assessment acknowledges that uncertainties exist because of the use of assumptions to bridge data and knowledge gaps about human exposures to DE and the underlying mechanisms by which DE may cause the observed toxicities in humans and animals:

> *A notable uncertainty of this assessment is how the physical and chemical nature of DE emissions has changed over the years because the toxicological and epidemiologic observations are based on older engines and their emissions, yet the desire is to focus on the potential health hazards related to exposure from present-day or future emissions.*
>
> *Other uncertainties include the assumptions that health effects observed at high doses may be applicable to low doses, and that toxicologic findings in laboratory animals are predictive of human responses. Also, the available data are not sufficient to demonstrate the absence or presence of an exposure/dose-response threshold in humans from DE toxicity at environmental exposures.*

As mentioned in the above, the results of the EPA study are based on data for older engines. As part of the project sponsors' commitment to implementing a state-of-the-art emissions reduction program, ultra-low sulfur diesel (ULSD) fuel would be used exclusively for all diesel engines throughout the site during construction. This would enable the use of tailpipe reduction technologies, and would directly reduce diesel PM emissions, which would reduce the potential for public health impacts. The PM emitted from combusting ULSD consists primarily of organic products of incomplete combustion, and is very low in metal content.[1] Further, this PM contains no biological material. Small amounts of nitrates and sulfates may be present in this PM and NO_x, SO_2, and ammonia emissions may lead to further (but much more diffuse) formation of secondary particulate matter in the region, although chemical reactions that result in secondary PM are typically too slow to cause an increase in secondary PM near the source. Many toxicological studies have shown that concentrations of hundreds of micrograms of sulfate or

[1] *AP42*, Section 1.3, September, 1998 and Section 3.1, April, 2000.

nitrate per cubic meter of air are required before even minimal changes in respiratory or other functions can be observed, even in asthmatic subjects or in sensitive laboratory rodents.[1]

$PM_{2.5}$

An important issue associated with $PM_{2.5}$ is that it has a direct causal effect on human health. Since particulate matter in the ambient air is comprised of a combination of discrete compounds or elements, its possible public health effects could vary depending on the specific components of particulate matter in a region. Acid aerosols such as sulfuric acid may trigger reactions in pulmonary lung function, while bioaerosols, such as mold spores, may result in allergic reactions related to increased incidences of asthma, for example. The EPA 2004 Criteria Document acknowledged the uncertainty regarding the shapes of particulate matter exposure-response relationships; magnitude and variability of risk assessments for particulate matter; the ability to attribute observed health effects to specific particulate matter constituents; the time intervals over which particulate matter health effects are manifested; the extent to which findings in one location can be generalized to other locations and the nature and magnitude of the overall public health risk imposed by ambient particulate matter exposure.

Studies have shown the importance of separating total personal exposure to $PM_{2.5}$ into its two major components.[2] Ambient (or outdoor) exposure includes the ambient PM concentration while outdoors, usually estimated by measurements at local air monitoring stations. Non-ambient exposure is the result of indoor sources (cooking, cleaning) and personal sources (smoking, hobby). Non-ambient exposure levels are independent of outdoor ambient PM concentrations. Among subjects of a large study of three cities, personal exposures to $PM_{2.5}$ were significantly higher than outdoor $PM_{2.5}$ concentrations.[3] The fact that personal PM exposures were higher than outdoor concentrations indicates that indoor sources of $PM_{2.5}$ contribute to, and in some cases, dominate personal exposures.

The potential for $PM_{2.5}$ to affect public health is dependent on the composition and the amount of PM in the atmosphere (i.e., the higher the ambient $PM_{2.5}$ concentration, the more likely that it would have an effect). The evidence cited by EPA in establishing the NAAQS for $PM_{2.5}$ is derived from epidemiologic studies that found, at typical ambient levels, a statistical correlation of PM and increased levels of morbidity and mortality.[4,5] It is unclear what forms of PM and

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[1] Concentrations of at least 100 micrograms of sulfate or nitrate per cubic meter of air are required before even minimal changes in respiratory function can be observed, even in asthmatic subjects or in sensitive laboratory rodents. See EPA's 2004 PM Criteria Document for extended discussion and references.

[2] Wilson, W.E., Brauer M., 2006. Estimation of ambient and non-ambient components of particulate matter exposure from a personal monitoring panel study. J Exp Sci Env Epid 16:264-74.

[3] Weisel, C.P., Zhang., J., Turpin, B.J., et al. 2005. Relationships of indoor, outdoor, and personal air (RIOPA), Part I. Collection methods and descriptive analyses. Health Effects Institute No. 130 Part I. Available at: http://www.healtheffects.org/Pubs/RIOPA-I.pdf (Accessed July 5, 2006).

[4] Krewski et al (2000); Dockery et al. N. Engl. J. Med. 329, 1753-1759 (1995); Pope et al Am. J. Respir. Crit. Care Med., 151:669-674 (1995), Burnett et al, JAMA 287(9), 1132-41 (2002); Dominici et al, Am. J. Epidemiol. 157 (12), 1055-1065 (2003).

[5] Some analysts doubt that PM concentrations and these health effects are causal. Compare. Pope, III, C. A. (2000), "Epidemiology of fine particulate air pollution and human health: Biologic mechanisms and who's at risk?" *Environ Health Perspect*, 108(4), 713-23; and Samet, J. M., Dominici, F., Curriero, F., C., Coursac, I., & Zeger. S. L. (2000), "Fine particulate air pollution and mortality in 20 U.S. cities,

what physiological mechanisms are responsible for the observed health effects. However, the extent of any adverse public health effect related to an increase in PM concentrations is anticipated to be proportional in some way to the concentration increase. A small increase in PM concentrations can, at most, lead to a small increase in PM related public health effects.

Although the NAAQS for $PM_{2.5}$ is based on the measurement of particle mass concentrations (i.e., total $\mu g/m^3$), the EPA recognized the need for further research into the relationships between PM composition and PM-related health effects. Indeed, a major requirement of 40 CFR Part 58 (Ambient Air Quality Surveillance for Particulate Matter, Final Rule) is the chemical speciation of $PM_{2.5}$ at 50 monitoring sites across the country. A great deal of current PM research, including studies conducted under the EPA's Office of Research and Development,[1] is focused on attempting to better understand the biological, chemical, and physical characteristics of PM underlying its potentially toxic effects. A basic finding among these studies is that different forms of $PM_{2.5}$ may differ substantially in their toxicologic significance.

Considerable research would be required to identify, quantify, and rank the myriad components of $PM_{2.5}$ in terms of their potential effect on public health. The National $PM_{2.5}$ Speciation Program,[2] established under 40 Code of Federal Regulations Part 58 as mentioned above, would serve as only a modest, first-cut analysis, as it would provide no information on the biologic content of ambient air PM, and only limited information on some metallic, ionic, and organic constituents of ambient PM. Although chemical and toxicologic knowledge of ambient $PM_{2.5}$ is limited, current evidence, as outlined below, suggests that $PM_{2.5}$ that is rich in either biologically-active material or in various metals is more harmful than $PM_{2.5}$ that has little to no biologic or metallic content.

The principal health effects of airborne particulate matter are on the respiratory system, although recent research investigated the possible link between particulate matter pollution and cardiovascular disease.[3]

Respiratory

General Respiratory Effects of $PM_{2.5}$. Numerous studies have correlated increased rates of hospital admissions for respiratory conditions, small decreases in lung function in children with or without asthma, and absences from school with changes in PM concentrations.[4] As a result, EPA stated that these statistical associations reflect cause and effect and established the NAAQS

1987-1994," *N Engl J Med*, 343(24), 1742-1749; with Lipfert, F.W., Perry, Jr., H. M., Miller, J. P., Baty, J. D. Wyzga, R. E., & Carmody, S. E. (2000), The Washington University-EPRI Veteran's "Cohort Mortality Study: Preliminary Results," *Inhalation Toxicology*, 12(4), 41-73; and Gamble, J. F. (1998). "$PM_{2.5}$ and mortality in long-term prospective cohort studies: Cause-effect or statistical associations?" *Environ. Health Perspect*. 106, 535-549.

[1] EPA Office of Research and Development, Research and Development, Fiscal Years 1997-1998 Research Accomplishments, EPA 60-R-99-106.

[2] Ibid.

[3] Künzli, N., Tager I.B. 2005. Air pollution: from lung to heart. Swiss Med Wkly 135:697-702. Available at http://www.smw.ch/docs/pdf200x/2005/47/smw-11025.pdf (accessed July 2006).

[4] CEPA/FPAC Working Group on Air Quality Objectives and Guidelines. National Ambient Air Quality Objectives for Particulate Matter. Part 1: Science Assessment Document.

for PM primarily on the basis of the associations.[1] The $PM_{2.5}$ standard was established to address the shortcomings of the PM_{10} standard and to protect public health.

Biologically Active $PM_{2.5}$. Particulate matter rich in pollen and other aero-allergens is well known to exacerbate respiratory problems, especially among people with allergic asthma and sufferers of hay fever (also called seasonal allergic rhinitis).[2] Other common forms of PM, present year-round, may aggravate respiratory problems because of their biologic content. Fine particulate matter from "ordinary" resuspended dust, for example, is a complex mixture of biologically and immunologically active materials, such as macromolecules, derived from molds, grasses, trees, cat and dog dander-epithelium, and latex rubber.[3]

$PM_{2.5}$ Rich in Metals. Inhalation of metals of various types may harm the upper respiratory tract, lungs, and other organs.[4] Although such problems have long plagued various occupational settings, environmental scientists at EPA and elsewhere are now focusing on whether the heavy metal content of some forms of respirable PM may be responsible for correlations between ambient air PM and morbidity and mortality in studied populations. For example, EPA scientists have demonstrated that extracts of metal-rich PM cause lung inflammation in human volunteers.[5] In particular, they evaluated ambient PM collected in the late 1980s from Utah Valley, where PM was rich in copper, zinc, lead, and nickel because of the dominance of a major steel mill in that valley. Compared with extracts of "ordinary" ambient PM (obtained when the mill was closed), the metal-rich extracts induced several signs of inflammatory injury. The investigators concluded that "metal content, and consequent oxidative stress that paralleled metal concentrations" caused the injury they observed, so that "mass may not be the most appropriate metric to use in assessing health effects after PM exposure, but rather specific components must be identified and assessed." Similar studies have been carried out in laboratory rats, with similar results reported.[6]

Asthma

High-density populations, such as those in New York City, are generally considered to have higher asthma rates than non-urban populations.[7] Given the concern that exposure to particulate matter emissions, especially $PM_{2.5}$, from activities associated with the proposed project could

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[1] EPA (2004) Air Quality Criteria for Particulate Matter (Vols. I and II); EPA/600/P-99/002af.Washington, DC: Office of Research and Development (1997); National Ambient Air Quality Standards for Particulate Matter, Final Rule, Federal Registry: July 18, EPA 2003.

[2] American Lung Association, 2001.

[3] Miguel, A.G., Cass, G.R., Glovsky, M.M., and Weiss, J. 1999. Allergens in Paved Road Dust and Airborne Particles. *Environ. Sci. Technol.*, 33:4159-4168.

[4] Kelleher, P.T., Pacheco, K., and Newman, L.S. (2000), Inorganic Dust Pneumonia: The Metal-Related Parenchymal Disorders, Environ. Health Perspect. 108, Supplement 4, 685-696.

[5] Ghio, A. J. and Devlin, R.B. (2001), Inflammatory Lung Injury after Bronchial Instillation of Air Pollution Particles, Am J Respir Crit Care Med 164: 704-708.

[6] Dye, J. A., Lehmann, J. R., McGee, J. K., Winsett, D. W., Ledbetter, A. D., Everitt, J. I., Ghio, A. J., & Costa, D.L. (2001), Acute pulmonary toxicity of particulate matter filter extracts in rats: Coherence with epidemiologic studies in Utah Valley Residents. EHP Supplement, 109(3), 395 - 404.

[7] Aligne C.A., Auinger P., Byrd R.S. 2000. Risk factors for pediatric asthma: contributions of poverty, race, and urban residence. Am J Resp Crit Care Med 162:873-877.

either aggravate pre-existing asthma or induce asthma in an individual with no prior history of the disease, the potential for emissions of $PM_{2.5}$ to precipitate the onset or exacerbation of asthma is examined in the following discussion. The discussion includes a review of the risk factors for asthma development and exacerbation; current prevalence, morbidity and mortality estimates of asthma, and a survey of the scientific literature that discusses the relationship between truck traffic and the occurrence of asthma.

Background. Asthma is a complex disease with multiple causes and substantial inter-individual variation in the severity of symptoms. It is a chronic inflammatory disorder of the airways characterized by variable airflow obstruction and airway hyper-responsiveness in which prominent clinical manifestations include wheezing and shortness of breath.[1] During an asthma "attack," an individual experiences difficulty breathing which, if severe enough, and treatment is not rendered, may be fatal in rare instances.[2] Asthmatic episodes may be triggered by specific substances, environmental conditions, and stress, as discussed below.

Although somewhat of a simplification, asthma can be categorized as having either an allergic or a non-allergic basis.[3,4,5] Allergic asthma is usually associated with a family history of allergic disease, increased levels of certain immune system proteins, and/or positive responses to specific diagnostic tests. Although exercise, cold air, and respiratory infections may also exacerbate asthma for allergic asthmatics, allergen exposure may be most important for eliciting airway inflammation and hyper-responsiveness. About 75 percent of people suffering from asthma have allergic asthma.[6] In contrast, people suffering from non-allergic asthma experience symptoms in their airways when confronted with such conditions as exercise, breathing cold air, or respiratory infections.[7]

Studies have demonstrated an increase in daily mortality, hospitalizations and emergency department utilization for asthma, attributable to air quality diminution from increased levels of sulfur dioxide, ozone and particulate matter. However, in children living in 24 US and Canadian communities, significant associations were reported between exposure to fine particles and their acidity and reduced lung function, symptoms of bronchitis, but not asthma. Children relocating from high to low pollution areas (or vice versa) were shown to experience changes in lung function growth that mirrored changes in exposure to particulate matter. The relation of

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[1] Sheffer, A.L., and V.S. Taggart. 1993. The National Asthma Education Program: expert panel report guidelines for the diagnosis and management of asthma. Med Care 1993:31 (suppl):MS20-MS28.

[2] McFadden, Jr. E.R. 2004. Asthma. In Harrison's Principles of Internal Medicine. (Eds: D.L. Kasper, E. Braunwald, A. Fauci, S. Hauser, D. Longo, J.L. Jameson), McGraw-Hill, New York, pp. 1508-1516.

[3] Scadding, J.G. 1993. "Chapter 1: Definition and clinical categorization." In *Bronchial Asthma: Mechanisms and Therapeutics*. Second Edition (Eds: Weiss, E.B, M.S. Segal, and M. Stein), Little, Brown, and Company, Boston, MA, pp. 3-13.

[4] McFadden, 2004.

[5] Sears, M.R. 1997. "Epidemiology of childhood asthma." *Lancet* 350:1015-1020.

[6] Centers for Disease Control (CDC). 2002. "Surveillance for Asthma – United States, 1980-1999." *Morbidity and Mortality Weekly Report* 51(SS01): 1-13. Available at http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5101a1.htm (accessed July 2006).

[7] McFadden, 2004.

variations in asthma prevalence to air pollution has been difficult; although, prospective studies in California have suggested that some incident asthma cases could be related to ozone.[1]

Prevalence of Asthma. In the US, approximately 6.4 million children (8.8 percent of children under age 18) have asthma. Asthma prevalence in New York State is estimated at approximately 9.9 percent.[2] According to the CDC, over the last two decades the self-reported prevalence of asthma increased 75 percent in all age groups and 160 percent in children between 0 and 4 years of age. The rate of asthma is increasing most rapidly in children under age 5. Additionally, it is estimated that asthma prevalence in Western countries doubled between 1977 and 1997.[3] Other parts of the world have also reported an increase in asthma prevalence in urban areas. Though changes in infectious disease patterns,[4] decreased physical activity, increasing prevalence of obesity,[5] and increased time spent indoors are hypothesized to be contributing factors to the increase in the prevalence of asthma, the subject is one of continuing research.

Asthma Morbidity and Mortality. Asthma morbidity and mortality rates have been rising throughout the US over the last few decades,[6] with New York City experiencing a disproportionate increase in the early 1990s[7]. However, hospitalization rates in New York City have been gradually declining since the peak rates in the mid-1990s. Between 1997 and 2004, asthma hospitalization rates among children aged 0-14 years decreased in most New York City boroughs.[8] Asthma mortality rates between 1990 and 2000 also declined for all age groups.[9]

Asthma is the leading cause of hospitalization in New York City for children aged 0 to 14 and ranks among the leading causes of hospitalization for all age groups.[10,11] In 2000, the hospitalization rate for asthma among children aged 0 to 4 was 10.2 per 1,000 children in New

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[1] The Lancet, Vol 360, October 19, 2002.

[2] American Lung Association, May 2005. "Trends in Asthma Morbidity and Mortality."

[3] Cookson, W.O.C.M., and M.F. Moffatt. 1997. "Asthma: an epidemic in the absence of infection?" *Science* 275:41-42.

[4] Ibid.

[5] Platts-Mills, T.A.E., R.B. Sporik, M.D. Chapman, and P.W. Heymann. 1997. "The role of domestic allergens." In: *The Rising Trends in Asthma.* Ciba Foundation Symposium 206. John Wiley and Sons, New York, NY, pp. 173-189.

[6] CDC, 2002.

[7] Garg, R., Karpati, A., Leighton, J., Perrin, M., Shah, M., 2003. *Asthma Facts, Second Edition*. New York City Department of Health and Mental Hygiene.

[8] New York City Department of Health and Mental hygiene. *Updated Asthma Hospitalization Data by NYC Neighborhood* from website http://www.nyc.gov/html/doh/downloads/pdf/asthma/asthma-hosprates-children.pdf. Site accessed June, 2006.

[9] Garg et al., 2003.

[10] Ibid.

[11] It should be noted that although hospitalization data is useful in characterizing the population severely affected by asthma, it is not necessarily directly correlated with asthma prevalence in a community (e.g., individuals who seek private care or those who effectively self manage asthma symptoms would not appear in hospitalization data).

York City, compared to 6.4 per 1,000 in the United States.[1] Asthma exacerbations resulting in hospitalizations appear to be particularly frequent and severe among minority, inner-city children. A recent study by investigators at the Mount Sinai School of Medicine found an enormous difference in the rate at which children living in poor New York City neighborhoods were hospitalized for asthma, compared to children in wealthy neighborhoods. Another recent study conducted in New York City found that children living in neighborhoods of low socioeconomic status had more than 70 percent increased risk of current asthma (diagnosis and symptoms during the previous 12 months), when compared to children of their same ethnicity and income level living in communities of greater economic affluence.[2] These findings suggest that characteristics of the urban environment, apart from the ethnicity and income level of the residents, contribute to high asthma prevalence. The study noted that areas with high asthma hospitalization rates are geographically clustered in low socioeconomic status areas. These areas tend to contain a number of potential pollution sources that could affect respiratory health, including designated truck routes and high traffic roads, waste transfer stations, and nearby power plants.

As such, there are striking differences in the number of hospitalizations among New York City boroughs and specific neighborhoods within each borough. On a borough level, hospitalization and death rates that are associated with asthma are highest in the Bronx. On a neighborhood scale, in 2004, the East Harlem area of Manhattan reported the highest rate of asthma hospitalizations among children 0-14 years old—approximately 13.1 hospitalizations per 1,000 children[3] and among adults 35 years and older, Hunts Point/Mott Haven had the highest rate, 12.6 per 1,000.

The borough of Brooklyn as a whole has experienced a 30 percent decrease in child hospitalization rates between 1997 and 2004.[4] A comparison of asthma hospitalization rates in 1997 and 2004 among children ages 0-14 is presented in Table 18-1, for zip codes surrounding the proposed project (See Figure 18-1), and for Brooklyn and New York City as a whole.

The reasons for the borough and local disparities in asthma are not known, but may be due to differences in economic status and ethnicity; exposure to different asthma triggers; or access to medical care.[5,6]

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[1] Ibid.

[2] Claudio L, Stingone JA, Godbold J. Prevelence of Childhood Asthma in Urban Communities: The Impact of Ethnicity and Income. Ann Epidemiol 2006; 16: 332-340.

[3] New York City Department of Health and Mental hygiene. *Updated Asthma Hospitalization Data by NYC Neighborhood* from website http://www.nyc.gov/html/doh/downloads/pdf/asthma/asthma-hosprates-children.pdf. Site accessed June, 2006.

[4] Under the direction of the New York City Department of Health and Mental Hygiene (DOHMH), an aggressive Asthma Initiative was begun in 1997, with goals of reducing illness and death from childhood asthma. Since its inception, major childhood asthma initiatives have been implemented in several low income neighborhoods with high hospitalization rates. Between 1997 and 2004, many of these neighborhoods have experienced substantial decreases in hospitalization rates, which may be an indication of success from extensive efforts by medical providers and community organizations participating in such initiatives.

[5] Weiss, K.B., P.J. Gergen, and E.F. Crain. 1992. Inner-city asthma: the epidemiology of an emerging U.S. public health concern. *Chest* 101:362S-367S.

[6] Platts-Mills, 1997.

Table 18-1
1997 and 2004 Hospitalization Rates per 1,000 Persons (Aged 0-14 Years)*

Location	1997	2004
Downtown Brooklyn/Brooklyn Heights/Park Slope (includes zip codes 11201, 11205, 11215, 11217, and 11231)	9.1	4.5
Bedford-Stuyvesant/Crown Heights (includes zip codes 11212, 11213, 11216, 11233, and 11238)	14.0	10.2
Borough of Brooklyn	8.3	5.8
New York City	9.5	6.0

* New York City Department of Health and Mental hygiene. *Updated Asthma Hospitalization Data by NYC Neighborhood* from website http://www.nyc.gov/html/doh/downloads/pdf/asthma/asthma-hosprates-children.pdf. Site accessed June, 2006.

Causes and Triggers. The increase in asthma among children has spurred scientists and clinicians to search for causes and risk factors for the disease. The rapidity of the increase points away from a significant change in population genetics, which would evolve over a much longer time scale, and towards some characteristic(s) of modern life. Factors that have been investigated epidemiologically (and sometimes experimentally) include indoor air pollution, outdoor air pollution, behaviors, food and food additives, medical practices, and illness in infancy. The reasons for the dramatic increase in asthma prevalence are currently unknown, although a number of hypotheses have been developed and investigated. Current hypotheses tend to focus on three areas: (1) increases in individual sensitivity (possibly due to reduced respiratory infections); (2) increases in exposures to allergens (due to change in ambient air pollution and/or indoor air quality); and (3) increases in airway inflammation of sensitized individuals (due to factors such as viral infections). No single factor is likely to explain the increased rates of asthma, however, and different factors are likely to dominate in different areas, homes, and individuals.

In theory, one can distinguish between "causes" and "triggers" of asthma. Causes are those factors that make a person susceptible to asthmatic attacks in the first place, while triggers are those factors that elicit asthmatic symptoms at a particular time. Immunologists are increasingly coming to understand asthma as a genetic disorder. While genetic predisposition seems to be necessary for the onset of asthma, it is not sufficient. Asthma attacks typically occur when a genetically predisposed person encounters one or more environmental triggers.[1]

Triggers are more easily studied, but may not be the underlying causes of the disease. For example, although a genetic predisposition to allergy is an important risk factor for developing asthma, there may have been no real increase in the number of genetically susceptible children, but rather a growth in the prevalence of factors that promote asthma development or trigger an attack. For a person suffering from asthma, however, the identification and elimination of triggering factors is of greatest practical importance.

Allergens in the indoor environment are important triggers of asthma in the US. Organic materials that cause the immune system to overreact, such as cockroach antigens, dust mite antigens, molds, pet and rodent dander and urine, are the principal indoor air quality triggers of asthma attacks in children. Some of these antigens are probably more common in poor quality housing, which could explain, in part, why poor children suffer high rates of asthma. Other indoor pollutants, such as tobacco smoke and natural gas combustion from household appliances

[1] Gentile, D. A. J. Immunology, 65, 4, 347-351 (2004).

can also exacerbate asthma symptoms. "Improvements" in housing, such as increased insulation and reduced ventilation to save on energy costs, and increased amounts of wall-to-wall carpeting and stuffed furniture, may have the unintended effects of promoting growth of dust mites and molds, and of concentrating antigens, irritants, and particulate matter indoors. These changes in housing over recent decades could help explain the widespread increases in asthma rates. In addition, the effect of indoor pollutants may be increased by the growing amount of time that children spend indoors, which increases a child's exposure to antigens. The lack of exercise might also increase the respiratory system's sensitivity to allergens.

Some natural aspects of outdoor air, such as pollens, are capable of triggering asthma attacks. On a local scale, air pollution may be important and, on a larger scale, it is possible that specific pollutants, such as ozone or diesel exhaust, enhance the effects of other factors, such as allergens, even if the pollutants themselves are not triggers of asthma. Though some epidemiologic studies have found an association between 24-hour average PM_{10} (particulate matter, less than 10 microns in diameter) levels and asthma hospitalizations and emergency room visits, others have not.[1] In addition, weather conditions, and cold air in particular, can elicit asthmatic symptoms independent of air pollution.

Asthma and Traffic and Construction Equipment Sources of Air Pollution. Most of the particles emitted by diesel engines are small enough to be counted as $PM_{2.5}$. Their small size makes them highly respirable and able to reach deep within the lung.

Certain experimental studies have evaluated the respiratory and systemic effect of diesel particles on laboratory animals.[2] These studies revealed that chronic and/or prolonged continuous exposures of the animals to large concentrations cause inflammation, fibrosis and functional changes in the respiratory system, and that very large concentrations cause premature death. The lowest observed adverse effect levels, as well as no observed adverse effect levels, occurred at concentrations that were considerably in excess of ambient concentrations. Specifically, the levels at which these effects were not observed ranged from 100 to 500 μg of diesel particulates per cubic meter, concentrations that are above allowable average daily values.

Epidemiologically, a few studies have addressed childhood asthma in relation to distance from roads and, hence, from vehicle exhaust. For example, young children in Birmingham, England admitted to hospitals with a diagnosis of asthma were more likely to live close to busy roads than children admitted for other reasons. The apparent risk of admission for asthma was increased by almost two-fold for children who live close to busy roads. Undercutting the significance of these findings was the lack of information about their socioeconomic status, family history of asthma, and the indoor environment. Other epidemiological studies have demonstrated an increase in daily mortality, hospitalizations, and emergency department utilization attributable to air quality diminution from increased levels of sulfur dioxide, ozone and PM. [3,4,1]

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[1] Norris et al., 1999; Schwartz et al., 1993; Sheppard et al., 1999; Tolbert et al., 2000; Henry et al., 1991; Hiltermann et al., 1997; Roemer et al., 1998; Roemer et al., 1999; Roemer et al., 2000

[2] EPA (2002, 2003a) IRIS record for diesel engine exhaust, available at www.epa.gov/iris/subst/0642.htm.

[3] Kunzli, et al., Public health impact of outdoor and traffic-related air pollution: a European assessment, Lancet 2000 2:356 (9232); 795-801

[4] Schwela, D. Air Pollution and Health in Urban Areas. Rev Environ Health. 2000 Jan-Jun; 15(1-2): 13-42

In a study conducted in the Netherlands, researchers found that living near busy streets was associated, in children, but not adults, with a one and a half fold increase in wheezing symptoms in the past, with a 4.8 fold higher use of asthma medications among children after controlling for various socioeconomic and indoor environmental exposures.[2] Other studies have not found an association between asthma symptoms or hospitalizations and residence near heavy traffic.[3]

Most studies found associations between some indicator of traffic (distance to roads, traffic volumes, or truck traffic volumes) near a residence or school and some indicator of respiratory disease (allergic rhinitis, wheezing or cough), while a few found no evidence of an association.[4] Experiments in which non-asthmatic adults were exposed for an hour to diesel engine exhaust containing particles and gases found increased airways resistance[5] and some cellular indicators of inflammatory response;[6] however, these subjects did not experience asthma. Diesel particulates and ozone have been shown to increase the synthesis of the allergic antibody IgE in animals and humans, which would increase sensitization to common allergens. By interacting together and with other environmental factors, particulates and gaseous air pollutants can have an effect on allergic individuals.[7] An additional hypothesis described by Cookson and Moffatt suggests a link between the increase in asthma and the decline of respiratory infections in modern society, which could shift the balance of the immune system in favor of factors that predispose persons to asthma and allergy[8]. Infectious disease has been dramatically reduced in our society by the use of antibiotics and immunization programs.

Other Health Effects including Cardiovascular, Lung Cancer, and Premature Mortality

People with heart disease such as coronary artery disease and congestive heart failure are at risk of serious cardiac effects.[9] In people with heart disease, very short-term exposures of one hour to elevated fine particulate matter concentrations have been linked to irregular heart beats and heart attacks.[10]

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[1] Edwards et al., (1994). Hospital Admissions for Asthma in Preschool Children; Relationshiop to Major Roads in Birmingham, United Kingdom. Arch. Environ. Health 49 (4); 223-227

[2] Oosterlee, A. et al., (1996). Chronic Respiratory Symptoms in Children and Adults Living Along Streets with High Traffic Density. Occup. Environ. Med. 53:241-247.

[3] Wilkinson, P. et al., (1999). Case-control Study of Hospital Admission with Asthma in Children Aged 5-14 Years: Relations with Road Traffic in North West London. Thorax. 54(12); 1070-1074.

[4] Brunekreef et al 1997, English et al (1999), Livingstone et al (1996).

[5] Rudell et al, Occup. Environ. Med. 53, 6480652, 1996.

[6] Slavi et al, Am. J. Respir. Crit. Care. Med. 159: 702-709, 1999.

[7] Fujieda et al Am J. Respir Cell Mol Biol, 19, 507-12, 1998; Nel et al.

[8] Cookson et al., 1997

[9] Goldberg MS, Bailar JC 3rd, Burnett RT, Brook JR, Tamblyn R, Bonvalot Y, Ernst P, Flegel KM, Singh RK, Valois MF. Identifying subgroups of the general population that may be susceptible to short-term increases in particulate air pollution: a time-series study in Montreal, Quebec. Res Rep Health Eff Inst 2000 Oct;(97): 7-113; discussion 115-20; and Zanobetti A, Schwartz J. Cardiovascular damage by airborne particles: are diabetics more susceptible? Epidemiology 2002 Sep; 13(5):588-92.

[10] Peters A, Liu E, Verrier RL, Schwartz J, Gold DR, Mittleman M, Baliff J, Oh JA, Allen G, Monahan K, and Dockery DW. Air pollution and incidence of cardiac arrhythmia. Epidemiology 2000 Jan; 11(1):11-

New epidemiological re-analyses of studies of long-term ambient PM exposure also show substantial evidence for increased lung cancer risk being associated with such PM exposures, especially exposure to fine PM or specific fine particles subcomponents.[1]

The elderly are at increased risk from fine particulate matter air pollution. Numerous community health studies have shown that when particle levels are high, senior citizens are more likely to be hospitalized for heart and lung problems, and some may die prematurely.[2]

Inhaling fine particulate matter has been attributed to increased hospital admissions, emergency room visits and premature death among sensitive populations with pre-existing heart or lung disease. Studies estimate that tens of thousands of elderly people die prematurely each year from exposure to ambient levels of fine particles.

G. PROBABLE IMPACTS OF THE PROPOSED PROJECT

The following discussion summarizes the potential public health impacts related to air quality and noise during construction and operation of the proposed project. A discussion of rodent control practices during construction is also discussed. As discussed in Chapter 10, "Hazardous Materials," no significant adverse impacts related to hazardous materials would be expected to occur as a result of construction and operation of the proposed project.

As discussed above and in Chapters 14, "Air Quality," and 17, "Construction Impacts," the discussion of significance of $PM_{2.5}$ air quality impacts is based on the NYSDEC incremental threshold guidance levels and EPA significant impact levels. In addition to the NYSDEC thresholds which are applicable for new stationary sources emitting 15 or more tons per year of PM_{10}, DEP has promulgated an interim guidance for $PM_{2.5}$, a neighborhood-scale threshold value that is used for comparison when determining potential significance of air quality and public health impacts. Based on SEQRA regulations and CEQR guidance, determination of the significance of impacts should also be made in connection with the intensity, duration, geographic extent, reversibility, and the number of people that would be affected by the predicted impacts.

CONSTRUCTION

AIR QUALITY

As described in air quality impact assessment section of Chapter 17, "Construction Impacts," The results of the analyses showed predicted exceedances of the NYSDEC $PM_{2.5}$ thresholds; however these were limited in extent, duration, and severity. The increments in excess of interim guidance thresholds were predicted to be highly localized, i.e., almost entirely due to construction activities

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7; and Peters A, Dockery DW, Muller JE, and Mittleman MA. Increased particulate air pollution and the triggering of myocardial infarction. Circulation 2001 Jun 12; 103(23):2810-5.

[1] EPA Air Quality Criteria for Particulate Matter (Vols II); October 2004, EPA/600/P-99/002bf.

[2] Pope CA 3rd. Epidemiology of fine particulate air pollution and human health: biologic mechanisms and who's at risk? Environ Health Perspect 2000 Aug; 108 Suppl 4:713-23; and Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW, Schwartz J, and Zanobetti A. The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity, Mortality and Air Pollution in the United States. Health Effects Institute Research Report 94, Part II, June 2000.

in close proximity to the affected location and not due to cumulative impacts from the larger project site. At a small number of sidewalk or ground-floor residential locations, exceedances of the annual threshold were predicted, but only for a single year of construction. Short-term exceedances of the thresholds were not predicted for more than a single day at any one ground-floor residential location throughout duration of construction. At sidewalk locations where short-term exceedances were predicted (all of which would be in close proximity to the site), the exceedances would occur multiple days in a year of construction, but not for the entire construction period. The levels of $PM_{2.5}$ increment are comparable to increments predicted for many standard small-scale construction operations and would be much lower than those associated with standard construction operations of a similar size due to the incorporation into the proposed project of extensive measures to reduce $PM_{2.5}$ emissions. In addition, the neighborhood-scale analysis resulted in no predicted exceedances of the $PM_{2.5}$ interim guidance threshold. Therefore, no significant adverse impacts on air quality are predicted during the construction of the proposed project. To the extent that it can be determined from the projected changes in air quality resulting from the construction of the proposed project, no significant adverse impacts on public health or increases of asthma rates in the community would be expected as a result of the temporary increases in airborne emissions from construction activities.

NOISE

As described in the noise impact assessment section of Chapter 17, "Construction Impacts," potential significant adverse noise impacts due to construction were predicted to occur at a large number of residential locations adjacent to the project site. However, because of the construction noise mitigation measures that have been incorporated into the project and committed to by the project sponsors, the magnitude of the noise levels produced by construction activities for this project are below those typically produced by major construction projects in New York City. While construction activities would be noticeable and intrusive, the noise levels produced by construction activities with the incorporated noise reduction measures would be relatively low for construction of a project of this magnitude. Significant noise activities would not occur overnight (after 11PM) except for infrequent circumstances such as continuous concrete pours during construction. During construction, noise levels in the existing Brooklyn Bear's Community Garden, South Oxford Park, and Dean Playground would be above the *CEQR Technical Manual's* impact criteria on noise levels. While the noise impacts during construction were determined to be significant, the changes in noise level due to the project are not of a magnitude that would significantly affect public health.

Moreover, the majority of buildings near or adjacent to the project site either have double-glazed windows or storm windows. In addition, a large number of residences have some form of alternative ventilation, either window, through-the-wall (sleeve), or central air conditioning. As described in Chapter 15, "Noise" and Chapter 19, "Mitigation", at locations where significant adverse noise impacts are predicted to occur, and where the residences do not contain either double-glazed or storm windows and alternative ventilation (i.e., air conditioning), the project sponsors would make these mitigation measures available, at no cost for installation, to owners of residences. With this level of mitigation, interior L_{10} noise levels at most, if not all, residences during most periods of time where significant noise impacts are predicted to occur would generally be below the CEQR 45 dBA L_{10} recommended level. With the implementation of the noise mitigation measures, the predicted absolute off-site noise levels during construction would be below those typically experienced by residents living adjacent to large construction projects.

RODENT CONTROL

As discussed in Chapter 17, "Construction Impacts," construction contracts would include provisions for a rodent (mouse and rat) control program. Prior to the start of construction, the contractor would survey and bait the appropriate areas. The contractor would provide for proper site sanitation, including trash containers and regular pickup throughout the construction period. As necessary, the contractor would carry out a maintenance program of baiting. Coordination would be maintained with appropriate public agencies. Only EPA- and NYSDEC-registered rodenticides would be permitted, and the contractor would be required to perform rodent control programs in a manner that avoids hazards to persons, domestic animals, and non-target wildlife.

PROJECT OPERATIONS

AIR QUALITY

As mentioned in Chapter 14, "Air Quality," the results of the analysis showed that near study area intersections for mobile sources, and at any off-site receptor location for stationary sources, the maximum 24-hour and annual incremental impacts would be less than the applicable NYSDEC interim guidance criteria. While the proposed project's stationary source emissions of $PM_{2.5}$ would be deemed insignificant under NYSDEC interim guidance, modeled increments of $PM_{2.5}$ were predicted to exceed the annual impact threshold at some locations on the exterior of on-site buildings. The potential exposure to $PM_{2.5}$ at these locations would be limited since occupants would not be expected to have their windows open continuously and be exposed to outdoor concentrations throughout the year (boiler emissions are highest in the winter when windows would least likely be opened). Therefore, it was concluded that no significant adverse air quality impacts would be expected from the proposed project during project operations. In addition, the results of the neighborhood-scale analysis demonstrated the maximum neighborhood-scale increment would be below the DEP interim guidance criterion of 0.1 $\mu g/m^3$. Therefore, to the extent that it can be determined from the projected changes in air quality resulting from the operation of the proposed project, no significant adverse impacts on public health or increases of asthma rates in the community would be expected as a result of the increases in airborne emissions from operational activities.

NOISE

As described in Chapter 15, "Noise," noise levels during operation of the proposed project from project-generated traffic would exceed the CEQR impact criteria and result in significant adverse noise impacts during one or more time periods at certain receptor locations. These locations would be the principal feeder streets to and from the parking facilities for project elements. While exceeding the CEQR impact criteria, the impacts would be localized and occur on street segments immediately adjacent to the project site (Flatbush Avenue, Dean Street, and 6th and Carlton Avenues). On Dean Street, existing and No Build noise levels are relatively low. Project-generated traffic would cause significant increases on this street, but would not be for prolonged periods of time and would still result in noise levels that fall in the marginally unacceptable range, which is not unusual for New York City residential areas. Therefore, no significant adverse health impacts are expected due to operation of the proposed project.

Noise levels within the newly created open space areas would also be above the CEQR guideline noise level. These noise levels would result principally from the noise generated by traffic on Atlantic Avenue. The open space, except for the portion immediately adjacent to Atlantic

Avenue, would be in the "marginally acceptable" range for residential areas and would experience noise levels similar to those experienced throughout the surrounding residential neighborhoods under Existing, No Build, and Build conditions. However, based on *CEQR Technical Manual* criteria, the noise levels at these new open space areas would result in potentially significant adverse noise impacts on their users. Since the noise levels are comparable to those in many residential neighborhoods of the City, they are not of a magnitude that would significantly affect public health.

At most locations in the area where project impacts would be predicted to occur, most residences already have either double-glazed windows or storm windows, and many have some form of alternative ventilation (air conditioning). At all of the impacted locations the project sponsors would make these types of noise mitigation measures available at no cost for purchase and installation to owners of residences (i.e., storm windows and alternative ventilation). While the CEQR criteria would be exceeded at some locations, the overall changes in noise level due to the project are not of a magnitude that would significantly affect public health. ✷