Chapter 20: Public Health

A. INTRODUCTION

This chapter assesses the proposed action's potential public health impacts, including those related to air quality, noise, and hazardous materials during construction and operation of the proposed action. Construction equipment and vehicles could cause potential public health impacts related to noise and air pollutant emissions, while potential impacts from hazardous materials could occur from construction-related ground disturbance and demolition. Potential health effects during operation of the proposed action would be related to noise and pollutant emissions from traffic, and pollutant emissions from combustion equipment that provide building heat. Of particular concern is the potential for diesel emissions to impact public health, given the potential effects of PM emissions on asthma. Therefore, this chapter also provides an overview of health concerns related to traffic, diesel equipment, and particulate matter (PM) emissions, and a discussion of asthma, its prevalence in New York City, and the area most likely affected by the proposed action.

B. METHODOLOGY

For determining whether a public health assessment is appropriate, the *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 that might not exceed the preceding thresholds but might, nonetheless, result in significant public health concerns.

Based on this guidance, this chapter assesses the potential health concerns during the construction and operation of the proposed action, including assessments of air quality, noise, hazardous materials and rodent control.

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 action 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 public concern about asthma in New York City, and that exposure to PM emissions could aggravate or induce asthma attacks, this chapter also provides a 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 project site.

A summary of the air quality and noise impact assessments during the construction and operational periods of the proposed action is then presented, and the potential for public health impacts due to the proposed action is determined. Summaries of potential impacts from hazardous materials and rodent control during construction are also presented.

C. SUMMARY OF AIR AND NOISE POLLUTION SOURCES FROM THE PROPOSED ACTION

CONSTRUCTION

AIR QUALITY

Construction activities have the potential to impact public health as a consequence of emissions from on-site construction engines, and emissions from on-road construction-related vehicles and their impact on traffic conditions. Historically, most construction engines have been diesel-powered and have produced relatively uncontrolled emissions of PM. 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, an emissions reduction program will be implemented during construction for the proposed action, as detailed in Chapter 19, “Construction.”

NOISE

Community noise levels during construction of the proposed action could result from noise and vibration from construction equipment operation and from construction vehicles and delivery vehicles traveling to and from the project 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 action 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. However, as described in Chapter 17, "Air Quality," the number of project generated vehicles will be under thresholds for environmental analysis (i.e., 75 peak hour trips for mid-town Manhattan) established in the *CEQR Technical Manual*. Therefore, an analysis of indirect impacts from mobile sources was not warranted, as no significant adverse air quality impacts would be expected from these sources.

Potential stationary source emissions associated with operation of the proposed action would primarily be from fuel combustion equipment that provide heating. The proposed buildings are expected to be heated using natural gas as fuel and therefore, the primary pollutant of concern is nitrogen dioxide (NO_2). Maximum predicted impacts from NO_2 emissions are presented in Chapter 17. An analysis of other pollutants, including potential $PM_{2.5}$ impacts was not warranted, since increases in ambient concentration levels of these pollutants from the proposed natural gas-fueled combustion equipment would be minimal, and would not be expected to result in significant air quality or public health impacts.

NOISE

The primary source of noise during project operations would be attributable to increased traffic in the area generated by the proposed action.

D. POLLUTANTS OF CONCERN

As mentioned above, the primary source of air quality pollutant emissions from the proposed action would be from diesel engines during construction. Increases in airborne PM emitted by such sources may account for potential impacts on public health. Also, given the higher than national asthma prevalence in New York City and the potential effects of PM emissions on asthma, PM has been identified as the primary pollutant of concern as it relates to potential public health impacts from the proposed action. The potential air quality impacts of $PM_{2.5}$ and other pollutants of concern from the proposed action are analyzed in Chapters 17, "Air Quality," and 19, "Construction."

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. PM_{10} refers to suspended particles with diameters less than 10 micrometers (μm), and $PM_{2.5}$ to suspended particles with diameters less than 2.5 μm. 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, $\mu g/m^3$).

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, and bacteria; debris from live and decaying plant and animal life; particles eroded from beaches, desert, soil and rock; and 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 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 PM such as pollen fragments. $PM_{2.5}$ does not refer to a single pollutant, but to an array of fine inhalable materials. For example, there are 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. 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, sulfur dioxide (SO_2), and nitrogen oxides (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.[1,2]

Data from the Botanical Gardens in the Bronx and Queens College in Queens indicate that the greatest contributors to ambient $PM_{2.5}$ concentrations in New York City 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 with 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.[3]

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[1] Ito K., Christensen W.F., Eatough D.J., Henry R.C., Kim E., Laden F., Lall R., Larson T.V., Neas L., Hopke P.K., Thurston G.D. PM source apportionment and health effects: 2. An investigation of intermethod variability in associations between source-apportioned fine particle mass and daily mortality in Washington, DC. J Expo Sci Environ Epidemiol. 2006 Jul;16(4):300-10. Epub 2005 Nov 23.

[2] Lena T.S., Ochieng V., Carter M., Holguin-Veras J., Kinney P.L. Elemental carbon and $PM_{2.5}$ levels in an urban community heavily impacted by truck traffic. Environ Health Perspect. 2002 Oct; 110(10):1009-15

[3] 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.

E. AIR QUALITY AND NOISE REGULATIONS AND STANDARDS

AIR QUALITY

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

Section 108 of the Clean Air Act (CAA) directs the U.S. Environmental Protection Agency (EPA) to identify criteria pollutants that may reasonably be anticipated to endanger public health and welfare. Section 109 of the CAA requires 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, soil, water, vegetation, wildlife, weather, visibility, and climate.

Beginning in 1994, EPA conducted a five-year review of the NAAQS for PM, which included an in-depth examination of epidemiologic and toxicological studies. 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, found in the December 13, 1996, Federal Register on page 65638. Based on this extensive analysis, in June 1997, EPA revised the NAAQS for PM 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 $\mu g/m^3$.[2]

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."[3]

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 65 $\mu g/m^3$ to 35 $\mu g/m^3$, and retaining the level of the annual $PM_{2.5}$ standard at 15 $\mu g/m^3$.

NOISE

Noise levels associated with the construction and operation of the proposed action would be subject to the emission source provisions of the New York City 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.

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

[2] 62 Federal Register 38652 (July 18, 1997).

[3] 62 Federal Register 28652, 38676 (July 18, 1997).

F. DETERMINING THE SIGNIFICANCE OF PUBLIC HEALTH IMPACTS

The State Environmental Quality Review Act (SEQRA) regulations and the *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.

The potential public health impacts of PM emissions and noise levels due to the proposed action are based on the results of the air quality and noise impact assessments in this EIS. The following section presents the applicable standards and thresholds with which the results of the air quality and noise modeling are compared in determining the significance of public health impacts.

AIR QUALITY

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 requires a detailed analysis of air quality impacts for that pollutant. New York County has been designated a non-attainment area for $PM_{2.5}$. To determine the potential significance of impacts from individual projects, the New York State Department of Environmental Conservation (NYSDEC) and the New York City Department of Environmental Protection (DEP) have provided interim guidance criteria, as described below.

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

As mentioned above, DEP is currently recommending an interim guidance for $PM_{2.5}$, a threshold value that is used for comparison when determining potential significance of air quality impacts. A neighborhood analysis is warranted, given that $PM_{2.5}$ is a regional pollutant, with monitored annual background concentrations that are near or above the applicable annual average standard in the New York City metropolitan area. 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 1 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.

In addition, DEP is currently recommending interim guidance criteria for evaluating the potential $PM_{2.5}$ impacts for projects subject to CEQR. The updated interim guidance criteria

currently employed by DEP for determination of potential significant adverse $PM_{2.5}$ impacts under CEQR are as follows:

- 24-hour average $PM_{2.5}$ concentration increments which are predicted to be greater than 5 $\mu g/m^3$ at a discrete receptor location would be considered a significant adverse impact on air quality under operational conditions (i.e., a permanent condition predicted to exist for many years regardless of the frequency of occurrence);
- 24-hour average $PM_{2.5}$ concentration increments which are predicted to be greater than 2 $\mu g/m^3$ but no greater than 5 $\mu g/m^3$ would be considered a significant adverse impact on air quality based on the magnitude, frequency, duration, location, and size of the area of the predicted concentrations;
- Predicted annual average $PM_{2.5}$ concentration increments greater than 0.1 $\mu g/m^3$ at ground-level on a neighborhood scale (i.e., the annual increase in concentration representing the average over an area of approximately 1 square kilometer, centered on the location where the maximum ground-level impact is predicted for stationary sources; or at a distance from a roadway corridor similar to the minimum distance defined for locating neighborhood scale monitoring stations); or
- Predicted annual average $PM_{2.5}$ concentration increments greater than 0.3 $\mu g/m^3$ at a discrete or ground level receptor location.

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 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 action's annual emissions of PM_{10} are estimated to be well below the 15-ton-per-year threshold under the NYSDEC's $PM_{2.5}$ guidance. The DEP community-based annual 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.

As presented in Chapter 17, "Air Quality," both the NYSDEC and DEP interim guidance criteria have been used to evaluate the potential significance of predicted air quality impacts of the proposed action on $PM_{2.5}$ concentrations, and to determine the need to minimize PM emissions from the proposed action. Therefore, the public health analysis considers both the NYSDEC and DEP thresholds in the determination of the public health impacts from the proposed action.

Actions under CEQR that would increase $PM_{2.5}$ concentrations by more than the DEP or NYSDEC interim guidance criteria above will be considered to have potential significant adverse impacts. DEP recommends that its actions subject to CEQR that fail the interim guidance criteria prepare an EIS and examine potential measures to reduce or eliminate such potential significant adverse impacts.

NOISE

As described in Chapter 18, "Noise," in terms of CEQR, a significant noise impacts occurs when there is an increase in the one hour equivalent noise level ($L_{eq(1)}$) of between 3 and 5 dBA, depending upon the noise level without the proposed action. In terms of public health,

significance is not determined based upon the incremental change in noise level, but is based principally upon the magnitude of the noise level and time frame of exposure.

G. AIR QUALITY-RELATED HEALTH EFFECTS

Scientists have studied 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 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 action.

DIESEL ENGINE EXHAUST

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) non-cancer 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 non-cancer 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.

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.*

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

> *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 above, the results of the EPA study are based on data for older engines. As part of the proposed action, Fordham University has committed to implementing an emissions reduction program for all of its construction activities at its Lincoln Center Campus, consisting of the following components: which include: minimizing the use of diesel engines to the extent practicable by using electric engines operating on grid power; exclusively using ultra-low-sulfur diesel (ULSD) for all diesel engines; utilizing best available tailpipe reduction technologies[1] for all nonroad diesel engines with a power rating of 50 horsepower (hp) or greater and for controlled truck fleets (i.e., truck fleets under long-term contract with Fordham University, such as concrete mixing and pumping trucks); and mandating the use of "newer" (i.e., Tier 1[2] or later) construction equipment for nonroad diesel engines greater than 50 hp. These measures would significantly reduce diesel PM emissions, which would reduce the potential for public health impacts.

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[1] Diesel particle filters (DPFs) have been identified as the tailpipe technology currently proven to have the highest reduction capability (construction contracts would specify that all diesel nonroad engines rated at 50 hp or greater would utilize DPFs, either original equipment manufacturer (OEM) or retrofit technology that would result in emission reductions of DPM of at least 90 percent (when compared with normal private construction practices).

[2] The first federal regulations for new nonroad diesel engines were adopted in 1994, and signed by EPA into regulation in a 1998 Final Rulemaking. The 1998 regulation introduces Tier 1 emissions standards for all equipment 50 hp and greater and phases in the increasingly stringent Tier 2 and Tier 3 standards for equipment manufactured in 2000 through 2008. The Tier 1 through 3 standards regulate the EPA criteria pollutants, including particulate matter (PM), hydrocarbons (HC), oxides of nitrogen (NO_x) and carbon monoxide (CO). Prior to 1998, emissions from nonroad diesel engines were unregulated. These engines are typically referred to as Tier 0.

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 PM 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 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.[2]

$PM_{2.5}$

An important issue associated with $PM_{2.5}$ is that it has a direct causal effect on human health. Since PM in the ambient air is composed of a combination of discrete compounds or elements, its possible public health effects could vary depending on the specific components of PM in a region. For example, 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. The EPA 2004 Criteria Document acknowledges the uncertainty regarding the shapes of PM exposure-response relationships; the magnitude and variability of risk assessments for PM; the ability to attribute observed health effects to specific PM constituents; the time intervals over which PM 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 PM exposure.

Studies have shown the importance of separating total personal exposure to $PM_{2.5}$ into its two major components.[3] Ambient (or outdoor) exposure includes the ambient PM concentrations while outdoors, usually estimated by measurements at local air monitoring stations. Non-ambient exposure is the result of indoor sources (e.g., cooking and cleaning) and personal sources (e.g., smoking and materials used for hobbies). 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.[4] 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

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

[2] 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.

[3] 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.

[4] 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).

of PM and increased levels of morbidity and mortality.[1] It is unclear what forms of PM and 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 the risk of PM-related public health effects.

The principal health effects of airborne PM are on the respiratory system, although recent research investigated the possible link between PM pollution and cardiovascular disease.[2]

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.[3] As a result, EPA stated that these statistical associations reflect cause and effect and established the NAAQS for PM primarily on the basis of the associations.[4] The $PM_{2.5}$ standard was established to protect public health.

Asthma

Urban populations in general, and New York City residents, specifically in the greater Harlem area, have a higher prevalence of asthma and higher rates of hospitalization for asthma than non-urban populations.[5] Given the concern that exposure to PM emissions, especially $PM_{2.5}$, from activities associated with the proposed action could 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 below. 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.[6] During an asthma

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[1] 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).

[2] 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).

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

[4] 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.

[5] 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.

[6] 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.

attack, an individual experiences difficulty breathing, which, if severe enough and treatment is not rendered, may be fatal in rare instances.[1] 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.[2,3,4] 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.[5] In contrast, people suffering from non-allergic asthma experience symptoms in their airways when exercising, breathing cold air, or suffering from respiratory infections.[6]

Prevalence of Asthma. Currently in the United States, approximately 6.8 million children (9 percent of children under age 18) have asthma.[7] In 2005, Asthma prevalence in New York State is estimated at approximately 9.9 percent.[8] According to the Centers for Disease Control (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.[9] Other parts of the world have also reported an increase in asthma prevalence in urban areas. Though changes in infectious disease patterns,[10] decreased physical activity, increasing prevalence of obesity,[11] 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.

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[1] 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.

[2] 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.

[3] McFadden, 2004.

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

[5] 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).

[6] McFadden, 2004.

[7] Bloom B, Cohen RA. Summary Health Statistics for U.S. Children: National Health Interview Survey, 2006. National Center for Health Statistics. Vital Health Stat 10(234). 2007.

[8] American Lung Association, November 2007. "Trends in Asthma Morbidity and Mortality."

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

[10] Ibid.

[11] 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.

Asthma Morbidity and Mortality. Asthma morbidity and mortality rates have been rising throughout the U.S. over the last few decades,[1] with New York City experiencing a disproportionate increase in the early 1990s[2]. 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 to 14 years decreased in most New York City boroughs.[3] Asthma mortality rates between 1990 and 2000 also declined for all age groups.[4]

Asthma is the leading cause of hospitalization in New York City for children aged 0 to 14 years and ranks among the leading causes of hospitalization for all age groups.[5] In 2000, the hospitalization rate for asthma among children aged 0 to 4 years was 10.2 per 1,000 children in New York City, compared with 6.4 per 1,000 in the United States.[6] 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 with 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 (a diagnosis with symptoms during the previous 12 months), when compared with children of their same ethnicity and income level living in communities of greater economic affluence.[7] 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 level, in 2005, the East Harlem area of Manhattan reported the highest rate of asthma hospitalizations among children aged 0 to 14 years (approximately 11.9 hospitalizations per 1,000 children[8]). Among adults 35 years and older, Hunts Point/Mott Haven had the highest rate, at 13.2 per 1,000.

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[1] CDC, 2002.

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

[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] Garg et al., 2003.

[5] Ibid.

[6] Ibid.

[7] 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.

[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/html/asthma/asthma.shtml. Site accessed December, 2007.

The borough of Manhattan as a whole has experienced a 55 percent decrease in child hospitalization rates between 1997 and 2005.[1] A comparison of asthma hospitalization rates in 1997 and 2005 among children aged 0 to 14 years is presented in Table 20-1 for zip codes surrounding the project site, and for Manhattan, and New York City as a whole.

Table 20-1
1997 and 2005 Hospitalization Rates per 1,000 Persons (Aged 0 to 14 Years)*

Location	1997	2005
Upper West Side** (includes zip codes 10023, 10024 and 10025)	6.4	3.8
Chelsea-Clinton** (includes zip codes 10001, 10011, 10018, 10019, 10020 and 10036)	14.4	4.3
Borough of Manhattan	12.3	5.5
New York City	9.5	5.4

* New York City Department of Health and Mental hygiene. *Updated Asthma Hospitalization Data by NYC Neighborhood* from website http://www.nyc.gov/html/doh/html/asthma/asthma.shtml. Site accessed December, 2007.
** The project site is located in zip code 10023, in the Upper West Side neighborhood as defined by New York City Department of Health and Mental Hygiene. Zip code 10019, located in the Chelsea-Clinton neighborhood lies directly south of the project site.

The reasons for the borough and local disparities in asthma are not known, but they may be due to differences in economic status and ethnicity, exposure to different asthma triggers, or access to medical care.[2,3]

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 toward 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 and other environmental triggers; 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.

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[1] 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 2005, 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.

[2] 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.

[3] Platts-Mills, 1997.

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 U.S. Organic materials that cause the immune system to overreact, such as cockroach antigens, dust mite antigens, molds, and 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, 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 concentrating antigens, irritants, and PM indoors. 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. Reduced physical activity may 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. 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.

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

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

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. [1,2,3]

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.[4] Other studies have not found an association between asthma symptoms or hospitalizations and residence near heavy traffic.[5]

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.[6] Experiments in which non-asthmatic adults were exposed for an hour to diesel engine exhaust containing particles and gases found increased airways resistance[7] and some cellular indicators of inflammatory response;[8] 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.[9]

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

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

[3] 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

[4] 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.

[5] 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.

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

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

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

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

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.[1] In people with heart disease, very short-term exposures of one hour to elevated fine PM concentrations have been linked to irregular heartbeats and heart attacks.[2]

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.[3]

The elderly are at increased risk from fine PM 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.[4]

Inhaling fine PM 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.

In summary, studies conducted in individual cities and using data pooled from multiple cities have demonstrated that increases in PM, SO_2, and ozone exposures are associated with increases in daily mortality, and hospitalizations and emergency department utilization for asthma with increases in PM. While the epidemiologic literature demonstrates that variation in air quality is associated with these morbidity and mortality events, it does not, in general, demonstrate that air quality differences account for the large increases seen in the prevalence of asthma through the 1980s and 1990s, or the wide variability in the prevalence of asthma and heart disease across and within cities.

H. PROBABLE IMPACTS OF THE PROPOSED ACTION

The following section summarizes the potential public health impacts related to air quality and noise during construction and operation of the proposed action, and hazardous materials during construction.

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[1] 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.

[2] 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):11-7; 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.

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

[4] 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.

AIR QUALITY

As described in Chapter 17, "Air Quality," operation of the proposed action would not result in any significant adverse impacts to air quality from stationary or mobile source emissions.

As presented in Chapter 19, "Construction," the maximum predicted stationary source incremental concentrations of $PM_{2.5}$ for the 24-hour and annual averaging periods during construction activities exceed the applicable DEP interim guidance criteria at some discrete receptor locations using a worst case emissions scenario. However, after taking into the account the temporary nature of construction, the variability of $PM_{2.5}$ emissions over time, the limited frequency of 24 hour impacts, and the limited area-wide extent of the 24 hour and annual impacts, it can be concluded that no significant adverse air quality impacts for $PM_{2.5}$ are expected due to on-site construction sources. Therefore, no significant public health impacts are expected from the construction of the proposed Master Plan.

NOISE

As described in Chapter 18 "Noise," Fordham University's proposed Master Plan for its Lincoln Center Campus would not result in any predicted significant adverse noise impacts from the operation of the proposed action. The analysis presented in Chapter 19, "Construction," shows that the proposed action would result in significant adverse noise impacts during construction at certain discrete locations. These predicted noise levels would be of limited duration, and the predicted overall changes in noise levels would not be large enough to significantly affect public health. While construction activities would produce noise levels of a magnitude that at times are annoying and intrusive, and would be considered undesirable, construction activities would only occur for a limited number of hours per day, and for a limited time period. Based upon the limited durations of these noise levels, the noise produced by construction activities would not result in a significant adverse public health impact.

Therefore, no significant adverse health impacts from noise are expected from construction and operation of the proposed project.

HAZARDOUS MATERIALS

As presented in Chapter 11, "Hazardous Materials," in order to avoid adverse impacts, remedial measures would be undertaken during excavation required for the first phase of construction and during excavation and demolition required for the second phase of construction. With these measures in place, no significant adverse impacts from hazardous material on public health would be expected from construction activities related to the proposed action.

RODENT CONTROL

Construction contracts would include provisions for a rodent (mouse and rat) control program. Before the start of construction, the contractor would survey and bait the appropriate areas and provide for proper site sanitation. During the construction phase, as necessary, the contractor would carry out a maintenance program. 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 humans, domestic animals, and non-target wildlife. ✷