

4.15 PUBLIC HEALTH

4.15.1 Introduction

This Section addresses the public health impacts associated with the proposed construction and operation of Shaft 33B at E. 59th Street and First Avenue. The project-related air, noise, and traffic impacts from construction, along with hazardous materials impacts from construction and chemicals used during activation of the tunnel, would likely have the greatest potential project-related effects on public health. Of particular concern is the potential of diesel emissions from construction-related activities, to impact public health (such as increasing asthma rates). In response to those concerns, the City has recently adopted Local Law 77, which will result in significant reductions in air pollution from construction equipment throughout New York City. Section 4.11 provides the air quality impact assessment, based on the peak air quality emission level increases expected to occur from the preferred Shaft Site, and Section 5.11 provides the air quality impact assessment associated water main connections construction (and potential combined air quality impacts from the construction of the preferred Shaft Site and water main connections). As described in Section 4.11, Air Quality, during the construction of the Shaft 33B at E. 59th Street and First Avenue, construction equipment would generate particulate matter (or PM) emissions from the combustion of fuel and construction-related activities. The maximum short-term emission levels are expected to peak in 2006, while maximum annual average emission levels are expected to peak in 2007-2008 at this Shaft Site. This section presents an overview of the health concerns related to particulate matter emissions, including a discussion of asthma, its prevalence in New York City, and the area most likely to be affected by the Shaft Site, and an assessment of the potential public health effects from the project-related air, noise, traffic and hazardous materials impacts.

4.15.2 Existing Conditions

Existing conditions for traffic, air quality, noise and hazardous materials for the preferred Shaft Site Study Area are discussed within their respective sections in this Chapter. Provided below is additional information on the health effects related to the emissions of particulate matter.

Health Effects Related to Emissions of Particulate Matter

Overview¹

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, $\mu\text{g}/\text{m}^3$). Thus, PM_{10} refers to suspended particles with diameters less than 10 μm , and $\text{PM}_{2.5}$ 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, and 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 particulate 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. 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.²

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, USEPA stated that these statistical associations reflect cause and effect and established the National Ambient Air Quality Standards (NAAQS) for PM primarily on the basis of the associations.⁴ The $\text{PM}_{2.5}$ standard was established to address the shortcomings of the PM_{10} standard and to protect public health.

$\text{PM}_{2.5}$

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. $\text{PM}_{2.5}$ does not

¹ Portions of the text contained in this section are derived from the October 2, 2001 Final Environmental Impact Statement for the Fulton Fish Market at Hunts Point prepared by Urbitran Associates, Inc. and the April 27, 2001 Draft Environmental Impact Statement for the St. Lawrence Cement Greenport Project prepared by AKRF, Inc.

² Kuenzli et al, American Heart Association's Scientific Sessions 2004: New Orleans, Louisiana; 7-10 November, 2004.

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

⁴ USEPA (1996) Air Quality Criteria for Particulate Matter (Vols. I, II, III); EPA/600/P-95/001af. Washington, DC: Office of Research and Development (1997); National Ambient Air Quality Standards for Particulate Matter, Final Rule, Federal Registry: July 18, EPA 2003; Air Quality Criteria for Particulate Matter, Fourth External Review Draft, June 2003.

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 particulate matter 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, SO₂ 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 PM₁₀, 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.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 USEPA 1996 Criteria Document acknowledged this uncertainty:

“There remains 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

⁵ NYSDEC, Report to the Examiners on Consolidated Edison’s East River Article X Project, Case No. 99-F-1314, February, 2002.

locations and the nature and magnitude of the overall public health risk imposed by ambient particulate matter exposure.”

The National Ambient Air Quality Standard for PM_{2.5}

Section 108 of the Clean Air Act (CAA) directs the USEPA to identify criteria pollutants that may reasonably be anticipated to endanger public health and welfare. Section 109 of the CAA requires the USEPA to establish 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, USEPA 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, USEPA conducted a five-year review of the NAAQS for particulate matter, which included an in-depth examination of epidemiologic and toxicological studies. USEPA 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 USEPA's Criteria Document for Particulates, Chapters 10-13 (1996); USEPA's Staff Papers on Particulates, in particular Chapter V⁶; and USEPA's proposed NAAQS for particulates, found in the December 13, 1996 Federal Register on page 65638. Based on this extensive analysis, in June of 1997, USEPA 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 µg/m³ and a short-term (24-hour) limit of 65 µg/m³.⁷

In establishing the NAAQS for PM_{2.5} in 1997, USEPA 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}, USEPA found that an annual average PM_{2.5} concentration of 15µg/m³ is below the range of data most strongly associated with both short- and long-term exposure effects. The USEPA Administrator concluded that an annual NAAQS of 15µg/m³ “would provide an adequate margin of safety against the effects observed in the epidemiological studies.”⁸ The annual standard is supplemented by a 24-hour standard of 65 µg/m³ to protect against short-term exposures in areas with strong local or seasonal sources.⁹

⁶ Many of the studies are found on USEPA's web page at <http://www.epa.gov/ttn/oarpg/t1sp.html>. USEPA's second and third external review draft of the PM criteria document are available on USEPA's website as well.

⁷ 62 Federal Register 38652 (July 18, 1997).

⁸ 62 Federal Register 28652, 38676 (July 18, 1997).

⁹ Although some advocates for a new PM_{2.5} standard identified PM_{2.5} as a “non-threshold” pollutant, and the Appellate Division in its NYPA vs. UPROSE decision agreed with this position, the USEPA Administrator rejected this view when promulgating the PM_{2.5} NAAQS, finding that up to 15 µg/m³ of PM_{2.5} could be present in ambient air without causing adverse health effects.

In addition to the NAAQS, NYCDEP has promulgated an interim guidance for PM_{2.5}. 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 km², 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 µg/m³ within the 1 km² area considered. To put this value in perspective: 0.1 µg/m³ constitutes less than one percent of the annual NAAQS for PM_{2.5}; A mass of 0.1 µg is roughly a million times smaller than the mass of a small bread crumb. A concentration increment that is lower than the incremental neighborhood guidance concentration would not be registered by the ambient air monitors.

On December 17, 2004, USEPA took final action designating the five boroughs of New York City as well as Nassau, Suffolk, Rockland, Westchester, and Orange Counties as PM_{2.5} non-attainment areas under the CAA. State and local governments are required, by early 2008, to develop implementation plans designed to meet the PM_{2.5} annual standard.

Public Health Issues Related to Particulate Matter

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 USEPA 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.^{10,11} 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 PM related public health effects.

Although the NAAQS for PM_{2.5} is based on the measurement of particle mass concentrations (i.e., total µg/m³), the USEPA 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 USEPA's Office of Research and

¹⁰ 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).

¹¹ Some analysts doubt that PM concentrations and these health effects are causal. Compare *Air Quality Criteria for Particulate Matter, Second External Review Draft*, USEPA 600/P-99/002aB (2001). 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, 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.

Development,¹² 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} 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,¹³ 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.

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).¹⁴ 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.¹⁵

PM_{2.5} Rich in Metals

Inhalation of metals of various types may harm the upper respiratory tract, lungs, and other organs.¹⁶ Although such problems have long plagued various occupational settings, environmental scientists at USEPA 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, USEPA scientists have demonstrated that extracts of metal-rich PM cause lung inflammation in human volunteers.¹⁷ 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),

¹² USEPA Office of Research and Development, Research and Development, Fiscal Years 1997-1998 Research Accomplishments, USEPA 60-R-99-106.

¹³ *Ibid.*

¹⁴ American Lung Association, 2001, <http://www.lungusa.org/air/envhayfever.html>.

¹⁵ 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.

¹⁶ 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.

¹⁷ 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.

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.¹⁸

*Asthma*¹⁹

High-density populations, such as those in New York City, are generally considered to have higher asthma rates than non-urban populations.²⁰ Given the concern that exposure to particulate matter emissions, especially PM_{2.5} from activities associated with the construction of the shaft 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 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.²¹ During an asthma “attack,” an individual experiences difficulty breathing which, if severe enough, and treatment is not rendered, may be fatal in rare instances.²² 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.^{23,24,25} Allergic asthma is usually associated with a family history of allergic

¹⁸ 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.

¹⁹ Portions of the text contained in this section are derived from the October 2, 2001 Final Environmental Impact Statement for the Fulton Fish Market at Hunts Point prepared by Urbitran Associates, Inc. the April 27, 2001 Draft Environmental Impact Statement for the St. Lawrence Cement Greenport Project prepared by AKRF, Inc.

²⁰ Andrew, Aligne C., et al. Strong Children’s Research Center, Rochester General Hospital, and American Academy of Pediatrics Center for Child Health Research, Rochester, New York, USA.

²¹ 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.

²² McFadden, Jr., E.R. 1987. Asthma. In *Harrison's Principles of Internal Medicine*. (Eds: E. Braunwald, K.J. Isselbacher, R.G. Petersdorf, J.D. Wilson, J.B. Martin, and A.S. Fauchi), McGraw-Hill Book Company, New York, NY, pp. 1060-1065.

²³ Scadding, J.G. 1985. “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.

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.²⁶ 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.²⁷

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 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 but not other pollutants²⁸

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 percent²⁹ 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. In the United States, 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 percent³⁰. Additionally, it is estimated that asthma prevalence in Western countries doubled between 1977 and 1997.³¹ Other parts of the world have also reported an increase in asthma prevalence in urban areas. Though changes in infectious disease patterns,³² decreased physical activity, increasing prevalence of

²⁴ McFadden, 1987.

²⁵ Sears, M.R. 1997. "Epidemiology of childhood asthma." *Lancet* 350:1015-1020.

²⁶ Centers for Disease Control (CDC). 1998. "Surveillance for Asthma – United States, 1960-1995." *Morbidity and Mortality Weekly Report* 48(4): 1015-1028.

²⁷ McFadden, 1987.

²⁸ The Lancet, Vol 360, October 19, 2002.

²⁹ American Lung Association, May 2005. "Trends in Asthma Morbidity and Mortality."

³⁰ American Lung Association, May 2005. "Trends in Asthma Morbidity and Mortality"

³¹ Cookson, W.O.C.M., and M.F. Moffatt. 1997. "Asthma: an epidemic in the absence of infection?" *Science* 275:41-42.

³² *Ibid.*

obesity,³³ 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,³⁴ with New York City experiencing a disproportionate increase in the early 1990s³⁵. 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.³⁶ Asthma mortality rates between 1990 and 2000 also declined for all age groups.³⁷

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.³⁸ In 2000, the hospitalization rate for asthma among children aged 0 to 4 was 10.2 per 1,000 children in New York City, compared to 6.4 per 1,000 in the United States.³⁹ 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.

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 children⁴⁰ and among adults, 35 years and older, Hunts Point/Mott Haven had the highest rate, 12.6 per 1,000. The borough of Manhattan as a whole has experienced a 50 percent decrease in child hospitalization rates between 1997 and 2004. Child asthma hospitalization rates for the Upper East Side neighborhood in 2004 were reported to be 1.9 cases per 1,000 persons. Directly

³³ 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.

³⁴ CDC, 1998.

³⁵ Garg, R., Karpati, A., Leighton, J., Perrin, M., Shah, M., 2003. *Asthma Facts, Second Edition*. New York City Department of Health and Mental Hygiene.

³⁶ 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>)

³⁷ Garg et al., 2003.

³⁸ Ibid

³⁹ Ibid.

⁴⁰ 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>)

south, in the Gramercy Park-Murray Hill neighborhood, which includes the project site and the neighborhood below E. 60th Street (neighborhoods are reported using the United Hospital Fund definitions), child asthma hospitalization rates in 2004 were 2.9 cases per 1,000 persons. However, between 1997 and 2004, these neighborhoods have also shown a large decrease in hospitalization rates—53 percent for the Upper East Side and 57 percent for the Gramercy Park-Murray Hill neighborhood.⁴¹ The reasons for 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.^{42,43}

The New York City Department of Health and Mental Hygiene (NYCDOHMH) is well aware of the epidemic of childhood asthma in the City's many boroughs and communities, and, under its direction, an aggressive Asthma Initiative was begun in 1997. The goals of the Asthma Initiative are to reduce illness and death from childhood asthma by 1) improving medical standards of care for children with asthma, 2) reducing asthma triggers in both homes and communities, 3) enhancing self-management support for individuals with asthma, 3) enhancing citywide asthma education standards and delivery 4) creating "asthma friendly" schools and daycare settings, 5) monitoring and tracking individuals with asthma, and 6) strengthening the ability of health care facilities, community organizations, schools, government agencies, and academic and research institutions to address asthma by facilitating the New York City Asthma Partnership.

DOHMH promotes the following key messages for individuals with asthma (KICK Asthma):

- Know what worsens your asthma.
- Inform your doctor about frequent asthma symptoms (i.e daytime symptoms more than 2 days per week or nighttime symptoms more than 2 times per month may be an indication of persistent asthma).
- Control frequent symptoms by using long-term control asthma medicines (inhaled corticosteroids are the most effective), and by avoiding tobacco smoke and other triggers.
- Keep regular doctor's visits, and ask your doctor for a written Asthma Action Plan.

In addition, DOHMH recommends that medical providers:

- Assess each patient's asthma severity at every visit
- Prescribe long term control medicine for individuals with persistent asthma. (Inhaled corticosteroids are the most effective treatment for most patients with persistent asthma)
- Partner with patients and develop a written Asthma Action Plan. In addition, complete a school medication authorization form so that children with asthma can receive medication services at school.

⁴¹ Ibid.

⁴² 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.

⁴³ Platts-Mills, 1997.

Since its inception, major childhood asthma initiatives have been implemented in several low income neighborhoods with high hospitalization rates. As mentioned above, 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.

Another successful community-based program has been the Harlem Children's Zone Asthma Initiative, stemming from a partnership between Harlem Children's Zone, Inc., and the Department of Pediatrics at Harlem Hospital Center. Launched in 2001, this initiative was developed out of concern over elevated asthma-related school absenteeism and limitations of existing hospital-based interventions. This program involved the screening of over 3,000 children under the age of 13 who live or go to school within a sixty block area of Central Harlem known as the Harlem Children's Zone Project. Those children with asthma or asthma-like symptoms were invited to participate in the program, which included a series of medical, educational, environmental, social and legal interventions. Following an eighteen month period, preliminary results showed a dramatic impact in reducing the number emergency department and unscheduled doctor visits (from 34 to 16 percent) overnight hospital stays (from 8.6 to 0 percent), and school days missed related to asthma (from 23 to 8 percent)⁴⁴.

Causes and Triggers

The recent dramatic 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 likely 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.⁴⁵

⁴⁴ Centers for Disease Control (CDC). 2005. "Reducing Childhood Asthma Through Community-Based Service Delivery --- New York City, 2001--2004" *Morbidity and Mortality Weekly Report* 54(01):11-4.

⁴⁵ Gentile, D. A. J. *Immunology*, 65, 4, 347-351 (2004).

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 products, 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 PM₁₀ (particulate matter, less than 10 microns in diameter) levels and asthma hospitalizations and emergency room visits others have not.⁴⁶ In addition, weather conditions, and cold air in particular, can elicit asthmatic symptoms independent of air pollution.

Asthma and Traffic Sources of Air Pollution

Scientists have been studying possible links between respiratory diseases or symptoms, such as cough, asthma and bronchitis, and traffic. Particles emitted by diesel engines are generally small enough to be counted as PM_{2.5}. The toxic effects of diesel engine exhaust have been evaluated in numerous studies. Certain experimental studies evaluated the respiratory and systemic effect of diesel particles on laboratory animals.⁴⁷ The 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

⁴⁶ 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

⁴⁷ USEPA (2002, 2003a) IRIS record for diesel engine exhaust, available at www.epa.gov/iris/subst/0642.htm.

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 particulate matter.

In a study conducted in the Netherlands, researchers found that residence 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. Other studies have not found an association between asthma symptoms or hospitalizations and residence near heavy traffic.

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.⁴⁸ Experiments in which non-asthmatic adults were exposed for an hour to diesel engine exhaust containing particles and gases found increased airways resistance⁴⁹ and some cellular indicators of inflammatory response;⁵⁰ 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 effect on allergic individuals.⁵¹ 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 allergy⁵². Infectious disease has been dramatically reduced in our society by the use of antibiotics and immunization programs.

⁴⁸ Brunekreef et al 1997, English et al (1999), Livingstone et al (1996).

⁴⁹ Rudell et al, *Occup. Environ. Med.* 53, 6480652, 1996.

⁵⁰ Slavi et al, *Am. J. Respir. Crit. Care. Med.* 159: 702-709, 1999.

⁵¹ Fujieda et al *Am J. Respir Cell Mol Biol*, 19, 507-12, 1998; Nel et al.

⁵² Cookson et al., 1997

4.15.3 Future Conditions Without the Project

In the Future Without the Project at the preferred Shaft Site, air quality, traffic, noise and hazard materials conditions are anticipated to be relatively similar to those described for existing conditions. Public health initiatives undertaken by the City, along with Federal, State and local regulations outlined in Section 3.15, are expected to continue. Land uses are expected to generally remain the same in this neighborhood. Air quality regulations mandated by the CAA are anticipated to maintain or improve air quality in the region. Therefore, it can be expected that public health conditions related to air quality, noise, traffic and hazardous materials conditions in the Future Without the Project would likely be no worse than those that presently exist.

4.15.4 Future Conditions With the Project

A summary of potential public health impacts from the construction and operation of the shaft at E. 59th Street and First Avenue is provided here. Potential impacts from air, noise, traffic and hazardous materials were assessed, to determine their potential affect on public health from the Shaft Site at E. 59th Street and First Avenue. While the EIS addresses two potential configurations of the preferred Site (see Chapter 2, “Purpose and Need and Project Overview”) there would be no substantive changes in predicted air quality, noise, traffic or hazardous materials impacts between the base and the alternate site configurations for construction or operation of the Shaft Site.

No significant adverse impacts on air quality, noise, and traffic are expected from the operation of Shaft 33B at the preferred Shaft Site. However, since chemicals may be used as part of the activation of the tunnel, the potential for public health impacts from the use of such chemicals are addressed. Thus, the public health analysis for the preferred Shaft Site focuses on the possible impacts on public health from changes in local air quality, noise, and traffic, and the handling of potential hazardous materials during construction activities at the preferred Shaft Site. In addition, the potential impacts on asthma incidences in the community from construction-related activities are determined to the extent possible from the air quality impact assessment.

Construction

Air Quality

The construction of the Shaft Site is expected to result in PM emissions. The sources of these emissions are construction-related truck traffic and on-site construction-related mobile and stationary sources, both of which were included in the air quality assessment near the preferred Shaft Site. PM emitted from combusting distillate fuel oil, such as ultra low sulfur diesel fuel that will be mandated for the contractor to use for on-site diesel-fueled equipment, consists primarily of organic products of incomplete combustion, and is very low in metal content.⁵³ Further, this PM contains no biological material. Small amounts of nitrates and sulfates may be present in this

⁵³ AP42, Section 1.3, September, 1998 and Section 3.1, April, 2000.

PM (given the gas-phase presence of nitrogen oxides and sulfur dioxide), and NO_x, SO₂, and ammonia emissions may lead to further (but much more diffuse) formation of secondary particulate matter, but these constituents, when present at less than 1 µg/m³ levels in air even at the maximally affected locations do not appear to harm health.⁵⁴ 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 function can be observed, even in asthmatic subjects or in sensitive laboratory rodents.⁵⁵ The potential increase of PM that would result from construction at the preferred Shaft Site was evaluated using air quality models for the worst-case stages of construction. The results of this analysis were used to conduct a conservative assessment of the public health affect for this Shaft Site, since the air quality analysis assesses the worst-case increase in emissions at the project site. In reality, the exposure to peak emission levels would rapidly decrease with distance from the Shaft Site and would affect a limited population.

Air Quality Modeling Results

As described in Section 4.11, the maximum short-term emissions levels are expected to peak in the Stage 1 construction phase, while maximum annual average emissions levels are expected to peak in the Stage 3 construction phase at this Shaft Site for the raise bore method (see Section 4.1, “Project Description,” for further descriptions of the construction phases). The emissions of airborne particulate matter related to construction would be less for other stages.

During construction of the Shaft Site, construction equipment would generate particulate matter from the combustion of fuel and construction related activities. With respect to PM_{2.5}, fuel combustion is the primary source of this pollutant. PM generated by construction-related transfer of materials and other fugitive dust sources tends to be larger size particulate matter that settles to the ground within a relatively short distance from the source. However, fuel combustion, especially from diesel sources generates particulate matter that contains a significant amount of PM_{2.5}, which stays aloft for greater distances than larger PM.

The anticipated construction-related PM_{2.5} emission increments associated with the preferred Shaft Site were discussed in Section 4.11. Analyses were performed for the peak air quality short-term (e.g., 24-hours) and long term (e.g., annual average) periods. Potential PM_{2.5} emission increments for other time periods are anticipated to be less than those calculate for the worst-case periods.

The air quality modeling analysis predicted that the maximum daily total PM_{2.5} concentrations from construction vehicles and equipment for the preferred Shaft Site would be less than the applicable ambient air quality standard. The maximum annual average neighborhood-scale incremental concentrations from such sources were less than the interim guideline criterion, used as a threshold for determining significant adverse impacts related to air quality. The predicted

⁵⁴ 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 USEPA 2001 (PM Criteria Document Draft) for extended discussion and references.

⁵⁵ See USEPA 2001 (PM Criteria Document Draft) for extended discussion and references.

effect of construction of the preferred Shaft Site on PM_{2.5} concentrations would be insignificant under all scenarios considered.

The PM_{2.5} emissions from mobile and stationary construction sources associated with the construction of the Shaft Site are not expected to significantly increase the concentration of PM_{2.5} in the nearby community.

Asthma

Hospitalizations from asthma, cardiovascular diseases, and deaths are caused by many things. The causes of asthma, its increase over the last two decades, and the triggers for its exacerbation are only partially understood. New York City health professionals are well aware of the epidemic of childhood asthma. As reported by NYCDOHMH⁵⁶, asthma is a common disease among children and adults. It is a leading cause of missed school among children and the most common cause of hospitalization for children 14 years and younger. Among adults, asthma causes missed work, emergency department visits, and limitation of activity.

The scenarios presented for the preferred shaft site are for a highly localized area, under reasonable worst-case and maximum activity conditions. Based on the air quality modeling results, under these circumstances the resultant air pollution exposure from construction activity would drop off rapidly with distance from the construction area. Thus, the exposure of the population affected by such emissions would be negligible. Though daily and even weekly hospitalization numbers in New York City are numerous from a public health point of view, based on the expected incremental exposures of PM_{2.5} from the construction activities, there would likely be no significant increase in such rates from the construction activities at the preferred Shaft Site.

Air Quality Conclusions

The construction of the preferred Shaft Site would not result in any new predicted exceedances of air quality standards and the predicted neighborhood average incremental concentration of PM_{2.5} would be less than the applicable interim guideline concentration. In addition, the exposure to localized peak emission levels utilized in these analyses would rapidly decrease with distance and would affect a limited population. The construction of the preferred Shaft Site is not anticipated to result in a significant adverse impact on air quality under any scenario. To the extent that it can be determined from the changes in air quality resulting from the construction of the preferred Shaft Site, 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 generated by this Shaft Site.

Noise

As described in Section 4.12, the potential significant adverse noise impacts from the construction of the preferred Shaft Site would occur at a limited number of receptor locations. Based on the noise modeling results, under these circumstances the resultant noise pollution

⁵⁶ On DOHMH website (www.nyc.gov/html/doh).

exposure from construction activity would be for a limited area, and drop off rapidly with distance from the construction area. While the noise impacts during construction were determined to be significant adverse impacts, they would not be expected to result in any permanent loss of hearing. Significant noise activities would not occur overnight during construction, so no additional loss of sleep from noise activities would be expected. NYCDEP is exploring potential mitigation measures that would reduce noise levels at these receptors. However, even without the mitigation measures, the predicted off-site noise levels are not expected to result in a significant adverse impact on public health.

Traffic

Based on the analyses reported in Section 4.9, “Traffic and Parking” and Section 4.11, “Air Quality”, the construction of the preferred Shaft Site is not expected to result in any significant adverse impacts on air quality from increased emissions as a result of construction activities. In addition, any increases in emission levels as a result of vehicular traffic would be transient. Therefore, there are no expected adverse impacts on public health from construction-related traffic.

Hazardous Materials

As described in Section 4.14, during construction, subsurface soils would be excavated from the preferred Shaft Site. The subsurface soils may contain contaminants resulting from a number of sources including deposition and infiltration, contamination from off-site sources, and from historic fill material commonly used throughout the City of New York. Therefore, a number of preventive measures will be implemented to minimize exposure to potentially contaminated soils and groundwater during construction as discussed below. There are no substantive differences between the base configuration and alternate site configuration with regard to hazardous materials impacts and, therefore, the measures presented below would apply to both. The amount of soils disturbed and excavated would be the same under both configurations. With the proposed general procedures and protective measures in place, no significant adverse impact on public health from hazardous materials is expected.

Conclusions

Based on the air quality assessment of the construction stages (including the benefits of the diesel emissions control that NYCDEP will require the contractor to implement), taking into account stationary and mobile impacts from construction, the construction of the preferred Shaft Site would not result in any new predicted exceedances of air quality standards and the predicted neighborhood average incremental concentration of PM_{2.5} would be less than the applicable interim guideline concentration. Therefore, potential PM_{2.5} emissions from mobile and stationary sources related to the construction of the Shaft Site are not anticipated to result in an adverse impact on public health. The principal health effects of airborne particulate matter are on the respiratory system. To the extent that it can be determined from the changes in air quality resulting from the construction of the Shaft Site, no significant increases of asthma incidences in the community would be expected. In addition, the potential impacts from noise, traffic and hazardous materials are also not expected to result in an adverse impact on public health. Further, a comprehensive pest management plan would be developed to address potential rodent

activity on the site. This plan would be submitted to NYCDOHMH for review and approval prior to implementation. Therefore, the construction of the Shaft Site at E. 59th Street and First Avenue is not expected to result in a significant adverse impact on public health. A combined public health assessment from construction of Shaft 33B and its water main connections at the preferred Shaft Site is presented in Section 5.15, “Public Health,” in Chapter 5, “Water Main Connections.”

Operation

Hazardous Materials

As described in Chapter 2, “Purpose and Need and Project Overview,” activating the shaft includes the shaft disinfection process. During the disinfection step, chlorinated water would flow into the shaft from Tunnel No. 3 and be discharged to the local sewer system. However, prior to discharging the chlorinated water to local sewers, the water may need to be de-chlorinated through the delivery of sodium bisulfate at the Shaft Site. Following the procedures outlined in Section 4.14, no significant adverse impacts on public health are expected on the surrounding community from the use of sodium bisulfate.

Following construction and activation of the shaft, no hazardous materials would be used at the site during operation of the shaft. Therefore, no potential significant adverse impacts on public health would be expected from the operation of the shaft at the preferred Shaft Site.

