Chapter 21: Public Health

A. INTRODUCTION AND METHODOLOGY

This chapter assesses the potential for public health related impacts associated with the Proposed Project.

For determining whether a public health assessment is appropriate, the *2001 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 ground water to be used as a source of drinking water;
- Solid waste management practices that could attract vermin and result in an increase in pest populations (e.g. rats, mice, cockroaches, and mosquitoes);
- Potentially significant adverse impacts to sensitive receptors from noise or odors;
- Vapor infiltration from contaminants within a building or underlying soil (e.g., contamination originating from gasoline stations or dry cleaners) that may result in significant adverse hazardous materials or air quality impacts;
- Actions for which the potential impact(s) result in an exceedance of accepted federal, state, or local standards; or
- Other actions, which might not exceed the preceding thresholds, but might nonetheless result in significant public health concerns, including projects such as the New York City Adult Mosquito Control Programs, the Williamsburg Bridge Lead Removal Project, and the New York City Comprehensive Solid Waste Management Plan.

The Proposed Project would not meet any of the thresholds warranting a public health assessment. As discussed in Chapter 18, "Air Quality," the Proposed Project would not result in any significant adverse air quality impacts. The Proposed Project would not engage in any solid waste management practices that could attract vermin and result in an increase in pest populations. The project sponsor has entered into agreements with NYSDEC under the auspices of the New York State Brownfield Cleanup Program (BCP) to investigate and, where necessary, remediate contamination on large portions of the site as part of its redevelopment. Under this program, a draft Remedial Work Plan (RWP) would be submitted to NYSDEC and the New York State Department of Health (NYSDOH), including remedial actions, as necessary, to be performed before, during, and/or after construction of the Proposed Project. All activities

involving disturbance of existing soils would be conducted in accordance with a Health and Safety Plan (HASP) that would detail measures, including health and safety guidelines and work practices, to reduce the potential for exposure (e.g., dust control). With the implementation of the RWP, no significant adverse impacts related to hazardous materials would result from demolition and/or construction activities on the project site or operation of the Proposed Project. Although noise levels in the 2-acre off-site public open space that would be developed by the City would be above the *CEQR Technical Manual's* Table 3R-3, "Noise Exposure Guidelines for Use in City Environmental Impact Review" guideline level of 55 dBA $L_{10(1)}$ for outdoor areas requiring serenity and quiet, they would be comparable to noise levels in a number of well-used and attractive open spaces in New York City that are also located adjacent to heavily trafficked roadways, such as the Hudson River Park, Empire State Park, and the East River Esplanade.

While the Proposed Project would not meet any of the thresholds warranting a public health assessment, in order to address comments made during the scoping of the Proposed Project, this chapter presents a discussion of asthma, its prevalence in New York City and its possible causes and triggers, and then presents an assessment of the potential public health effects from the Proposed Project.

This analysis concludes that potential $PM_{2.5}$ emissions from mobile and stationary sources related to the Proposed Project are not expected to result in adverse public health impacts, including impacts on asthma rates.

B. HEALTH EFFECTS RELATED TO ASTHMA[1]

Urban populations, such as those in New York City, are generally considered to have higher asthma rates than non-urban populations.[2] Given concern that exposure to PM—in particular, emissions of fine particulate matter with an aerodynamic diameter less than 2.5 micrometers in diameter ($PM_{2.5}$), emissions from activities associated with the Proposed Project—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 onset of an exacerbation is examined in the following discussion.

BACKGROUND

PARTICULATE MATTER

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

[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 November 2004 Final Environmental Impact Statement for the Catskill/Delaware UV Facility, prepared by Hazen and Sawyer, P.C., CDM, Inc., and AKRF, Inc.

[2] 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.

particles with diameters less than 10 µm, and $PM_{2.5}$ to suspended particles with diameters less than 2.5 µm.

Particulate matter is emitted by a variety of sources, both natural and man-made. 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 matter 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 PM are on the respiratory system, although recent research investigated the possible link between particulate matter pollution and cardiovascular disease.[1]

Researchers found a 0.5 percent increase in death rates for every increase in the PM_{10} concentration level of 10 $\mu g/m^3$, even where ambient levels were well below the National Ambient Air Quality Standards[2] (NAAQS). The authors of that research recognized the limitations of their work. They explained that they used PM_{10} data in their study because at that time $PM_{2.5}$ data were not yet available nationally.[3] Some studies have found that the daily mortality rate is associated with the concentration of fine particles ($PM_{2.5}$) but not coarse particles (PM_{10}-$PM_{2.5}$).[3] Responding to a substantial body of epidemiologic evidence, the U.S. Environmental Protection Agency (USEPA) stated in 1996 that the PM_{10} standards alone may not be sufficient to protect the public health with an adequate margin of safety, and that $PM_{2.5}$ is a better surrogate for particulate components linked to mortality and morbidity at the levels below the PM_{10} standards. To address these issues in 1997, the USEPA retained the PM_{10} standards and promulgated the 24-hour and annual standards for $PM_{2.5}$, based on the consistency with the literature on health effects.[4]

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.[5] As a result, USEPA stated that these statistical associations reflect cause and effect and established the PM NAAQS primarily on the basis of the associations.[6] The $PM_{2.5}$ NAAQS were established to address the shortcomings of the PM_{10} NAAQS standard and to protect public health.

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

[2] Samet, J.M. et al. N. Engl J. Med, 343, 24, 1742-1749 (2000).

[3] Schwartz, J. et al. J. Air Waste Manag. Assoc 46, 927-939 (1996).

[4] Ware, J. H. Harvard School of Public Health, N. Engl. J. MEd., 343, 24, 1798-1799 (2000).

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

[6] USEPA (1996) Air Quality Criteria for Particulate Matter (Vols. I, II, II); EPA/6000/P-95/001af. Washington, DC: Office of Research and Developemtn (1997); National Ambient Air Quality Standards

ASTHMA

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

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

Studies have demonstrated an increase in daily mortality, hospitalizations, and emergency department utilization for asthma, attributable to air quality diminution from increased levels of sulfur dioxide, ozone, and PM. 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 and 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 to determine; although, prospective studies

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for Particulate Matter, Final Rule, Federal Registry; July 18, EPA 2003; Air Quality Criteria for Particulate Matter, Fourth External Review Draft, June 2003.

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

[2] McFadden, Jr., E.R. 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.

[3] 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.

[4] McFadden, 1987.

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

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

[7] McFadden, 1987.

in California have suggested that some incident asthma cases could be related to ozone but not other pollutants.[1]

Prevalence of Asthma

In the US, approximately 6.4 million children (8.8 percent of children under age 18) have asthma. Asthma prevalence in New York State is estimated at approximately 9.9 percent.[2] According to 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. Another report estimated that asthma prevalence in Western countries doubled between 1977 and 1997.[3] Other parts of the world besides the West have also reported an increase in asthma prevalence in urban areas. Though changes in infectious disease patterns,[4] decreased physical activity, increasing prevalence of obesity,[5] and increased time spent indoors are hypothesized to be contributing factors to the increase in the prevalence of asthma, the subject is one of continuing research.

Asthma Morbidity and Mortality

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

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.[9] 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.[10] Asthma exacerbations resulting in hospitalizations appear to be particularly frequent and severe among minority, inner-city children, but the disproportionate rates among affected groups is likely to be due to factors other

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

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

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

[4] Ibid.

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

[6] CDC, 1998.

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

[8] Ibid.

[9] Garg et al., 2003.

[10] Ibid.

than genetic differences. A recent study by investigators at the Mount Sinai School of Medicine found a significant 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. Compared with the other boroughs, hospitalization and death rates are highest in the Bronx.[1] On a neighborhood scale, in 2000, the East Harlem area of Manhattan reported the highest rate of asthma hospitalizations —approximately 1,718 hospitalizations per 100,000 persons.[2] Asthma among children is also a serious medical concern in the Hunts Point area of the Bronx. A survey completed by the New York City Department of Health of students of the three elementary schools in Hunts Point in the spring of 2000 revealed that over 20 percent of local children have asthma, over three times the national average. Asthma hospitalization rates among children in this neighborhood in 2000 were reported to be 10 cases per 1,000 persons. This is slightly higher than the reported average for the borough of the Bronx (9.16 cases per 1,000 persons), and higher than the average rate for the city as a whole (6.06 cases per 1,000 persons).[3] However, between 1997 and 2000, the Hunts Point-Mott Haven neighborhood had also shown the largest decrease in hopitalization rates—56 percent—among all New York City neighborhoods.[4] In the project area, child asthma hospitalization rates in 2000 were reported to be 11.02 cases per 1,000 persons, a 41 percent decrease from 1997 rates. The reasons for the borough and local disparities in asthma are not known, but may be due to differences in economic status and ethnicity; exposure to different asthma triggers; or access to medical care.[5,6]

New York City officials are well aware of the epidemic of childhood asthma in the City's many boroughs and communities, and, under the direction of the New York City Department of Health (NYCDOH), began an aggressive Asthma Initiative in 1997. The goals of the Asthma Initiative are to reduce illness and death from childhood asthma by: (1) consulting with a physician to determine the appropriate regimen of preventative and rescue medications to obtain an asthma action plan; (2) strengthening the ability of institutions, such as schools and medical facilities, to respond to the disease; (3) encouraging and coordinating asthma research; (4) facilitating interactions among health care facilities, schools, communities, and governments agencies; (5) giving special attention to high-risk populations. Among the Initiative's recommendations for preventing asthma episodes are: (1) avoid cigarette smoke; (2) reduce exposure to dust mites; (3) avoid furred pets and birds; (4) eliminate or reduce roaches; (5) close windows and use an air conditioner when pollen or air pollution is bad; (6) help improve the environment.

Since its inception, major childhood asthma initiatives have been implemented in several low income neighborhoods having high hospitalization rates. As mentioned above, between 1997

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[1] De Palo, V.A., P.H. Mayo, P. Friedman, and M.J. Rosen. 1994. "Demographic influences on asthma hospital admission rates in New York City." *Chest* 106:447-451.

[2] Garg et al., 2003.

[3] Garg et al., 2003.

[4] Ibid.

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

[6] Platts-Mills, 1997.

and 2000, many of these neighborhoods have experienced substantial decreases in hospitalization rates, which may be a positive 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 deparment 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).[1]

Causes and Triggers

The 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 increase rates of asthma, however, and various factors will dominate in specific 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. 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.[2] 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 child suffering from asthma, however, identification and elimination of triggering factors is of greatest practical importance.

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[1] Centers for Disease Control (CDC). 2005. "Reducing Childhood Asthma through Community-Based Service Delivery – New York City, 2001–2004" *Morbidity and Mortality Weekly Repor*t 54(01):11-4.

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

Allergens in the indoor environment are definitely important triggers of asthma in the US. Organic material 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 affects of promoting growth of dust mites and molds, and of concentrating antigens, irritants, and PM 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, and by lack of exercise, which might increase the respiratory system's sensitivity to allergens.

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

ASTHMA AND TRAFFIC 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.[2] 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, 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

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

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

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.[1] Experiments in which non-asthmatic adults were exposed for an hour to diesel engine exhaust containing particles and gases found increased airways resistance[2] and some cellular indicators of inflammatory response;[3] 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.[4] An additional hypothesis described by Cookson and Moffatt suggests a link between the increase in asthma and the decline of respiratory infections in modern society, which could shift the balance of the immune system in favor of factors that predispose persons to asthma and allergy.[5] Infectious disease has been dramatically reduced in our society by the use of antibiotics and immunization programs.

C. PROBABLE IMPACTS OF THE PROPOSED ACTIONS

MOBILE SOURCES

As mentioned above, asthma among children is a major public and individual health problem in the City. However, the causes of asthma and its increase over the last two decades are not certain, and the triggers for its exacerbation are only partially understood. The potential relationship between vehicular exhaust resulting from increased truck traffic and asthma, especially in communities with high rates of asthma, will continue to be studied by epidemiologists.

As described in Chapter 18, "Air Quality," the Proposed Project would result in PM emissions from the combustion of fuel from mobile sources. With respect to $PM_{2.5}$, fuel combustion sources are the primary components of this pollutant. Particulate matter generated by

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

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

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

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

[5] Cookson et al., 1997.

construction-related transfer of materials and other fugitive dust sources tend to be larger size PM that settles to the ground within a relatively short distance from the source. However, fuel combustion, especially from diesel combustion sources, generates PM that mostly consists of $PM_{2.5}$. An analysis of $PM_{2.5}$ from mobile sources was performed and indicated that the incremental increases of $PM_{2.5}$ concentrations with the Proposed Project would be less than the interim guidance levels employed by the New York City Department of Environmental Protection (NYCDEP). Therefore, the Proposed Project is not considered to have significant $PM_{2.5}$ impacts, and diesel emissions from project-related truck traffic are unlikely to significantly affect public health and local asthma incidents.

STATIONARY SOURCES

The Proposed Project would also result in the emission of PM from stationary sources associated with the Proposed Project, such as emissions from fuel burned on-site for heating and hot water systems. These proposed heating systems would use natural gas as fuel.

Particulate matter emitted from sources combusting natural gas 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 (given the gas-phase presence of nitrogen oxides and sulfur dioxide), and NO_x, SO_2, and ammonia emissions may lead to further (but much more diffuse) formation of secondary PM, but these constituents, when present at less than 1 $\mu g/m^3$ levels in air—even at the maximally affected locations—do not appear to harm health.[2] 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.[3] The specific types and amount of $PM_{2.5}$ associated with combustion of natural gas are not known to adversely impact health, and are expected to be benign at the concentrations that would be in ambient air with the operation of the combustion sources.

As described in Chapter 18, "Air Quality," an air quality screening analysis was conducted which determined that the Proposed Project is not likely to result in significant impacts from stationary sources. Although the issue of health effects due to $PM_{2.5}$ is complex, it is reasonable to infer that the Proposed Project would not result in potentially significant adverse health impacts from PM. The specific types and amount of $PM_{2.5}$ associated with combustion of natural gas are not known to adversely impact health, and are expected to be benign at the concentrations that would be in ambient air with the operation of the combustion sources.

D. CONCLUSION

The causes of asthma and its increase over the last two decades are not certain, and the triggers for its exacerbation are only partially understood. The potential relationship between vehicular

[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 U.S. EPA 2001 (PM Criteria Document Draft) for extended discussion and references.

[3] See U.S. EPA 2001 (PM Criteria Document Draft) for extended discussion and references.

exhaust resulting from increased truck traffic and asthma, especially in communities with high rates of asthma, requires further study. Since the Proposed Project is not considered to have significant $PM_{2.5}$ impacts, diesel emissions from project-related truck traffic are unlikely to significantly affect public health and local asthma incidents. Also, the specific types and amount of $PM_{2.5}$ associated with combustion of natural gas are not known to adversely impact health, and are expected to be benign at the concentrations that would be in ambient air with the operation of the Proposed Project's stationary sources of combustion. Therefore, potential $PM_{2.5}$ emissions from mobile and stationary sources related to the Proposed Project are not expected to result in adverse public health impacts, <u>including impacts on asthma rates</u>. ✷