3.20 PUBLIC HEALTH

INTRODUCTION AND METHODOLOGY

The proposed action would not result in significant adverse impacts to public health.

The *City Environmental Quality Review (CEQR) Technical Manual* states that a public health assessment may not be necessary for many proposed actions, but a thorough consideration of health issues should be documented. In determining whether a public health assessment is appropriate, the following has been considered:

Whether increased vehicular traffic or emissions from stationary sources would result in significant adverse air quality impacts.

The potential for these impacts was examined in Chapter 3.17, "Air Quality." A total of three receptor locations were selected for carbon monoxide (CO) microscale analysis. The highest project-generated CO increment would occur at the intersection of East 126th Street and Second Avenue during the AM peak period. The NYCDEP CO de minimis values would not be exceeded at this site or any other analysis site, indicating that the proposed action does not have the potential to cause CO impacts that are considered to be significant. The proposed action would also not result in any violations of the CO standard and therefore would not result in significant CO impacts at the analyzed locations. Estimated CO concentrations near the two new underground parking facilities were not estimated to cause or exacerbate the NAAQS of 9.0 ppm. Additionally, the proposed action would not cause increases in concentrations above the 24-hour and annual $PM_{2.5}$ significant threshold values, and therefore the proposed action would not result in significant $PM_{2.5}$ impacts at the analyzed receptor location. Particulate matter smaller than or equal to 10 microns in size (PM_{10}) was not analyzed in detail for the mobile source analysis given the small affect the proposed action would have on the number of heavy duty and/or diesel fueled vehicles in the study area. Therefore, the proposed action would not have the potential to result in significant PM_{10} impacts. As such, the results show that the development of the proposed project would not result in any significant adverse air quality impacts from mobile sources for CO, $PM_{2.5}$ and PM_{10}.

No exceedances of the NAAQS are predicted as a result of emissions associated with HVAC systems including: (1) the impact of the proposed action, or the proposed action with the MTA Bus Depot Expansion Alternative, on existing and future buildings, or on other sensitive receptor locations within the project area; and, (2) the impact of two existing large scale residential developments located to the south of the project site on the project site. In addition, there would be no exceedances of the NAAQS as a result of the emissions from the existing 126th Street MTA Bus Depot.

An analysis of the cumulative impacts of industrial sources on the development site was also performed, as detailed in Chapter 3.17. Searches of three databases did not identify any air toxics facilities, so no further analysis of air toxics is required since air quality impacts from air toxics on the proposed development would not occur.

If there is an increased potential for exposure to contaminants in soil or dust or vapor infiltration from contaminants within a building or underlying soil that may result in significant adverse hazardous materials or air quality impacts.

As described in detail in Chapter 3.10, "Hazardous Materials," the proposed action has the potential to result in an increased human exposure to potential contaminants in soil or dust during construction and potentially during occupancy of the project site. Prior to construction, further investigation would be performed on the project site to determine the presence and nature of contamination of concern and the proper remedial and/or health and safety measures that would be employed during development of the project site.

Under conditions with the proposed action, the developer would be obliged to prepare and submit plans for site remediation, for New York City Department of Environmental Protection (NYCDEP) approval. Along with these plans, a Site Management Plan (SMP) and a Construction Health and Safety Plan (CHASP) would be required, in accordance with standard industry practice. In addition, it is expected that the selected developer would apply for inclusion in the New York State Department of Environmental Conservation (NYSDEC) Brownfields Cleanup Program (BCP), and would also be required to prepare documentation required by NYSDEC to support that application. NYSDEC would provide oversight for spill remediation. Requirements for vapor mitigation would follow the New York State Department of Health (NYSDOH) Final "Guidance Evaluating Soil Vapor Intrusion in the State of New York," dated October 2006.

Solid waste management practices that could attract vermin and result in an increase in pest populations.

No solid waste management practices are proposed beyond those which occur at most residential and commercial uses found in the City. These practices would include all contemporary solid waste collection and containment practices and conformance with the laws of the New York City Board of Health. Development pursuant to the proposed action would occur in an area that is currently served by NYC Department of Sanitation residential trash and recycling pickups. As discussed in Chapter 3.13, "Solid Waste and Sanitation Services," the proposed action would not affect the delivery of these services, or place a significant burden on the City's solid waste management system.

Potentially significant adverse impacts to sensitive receptors from noise.

The proposed action would facilitate a new mixed-use development in an area with high ambient noise levels, due to the presence of transportation infrastructure, commercial and transportation land uses, and proximity to the busy 125th Street traffic corridor. No new significant sources of noise would be generated by the proposed action. Traffic generated by the proposed action would not produce any significant adverse noise impacts.

The maximum existing L_{10} noise levels at the project site exceed 70 dBA, and the future noise levels at the facades of the development site would exceed 70 dBA. The project site would be suitable for residential, commercial and cultural uses only by providing window-wall attenuation ranging from 30 dBA to 40 dBA for the affected exterior façades of the proposed development in

order to achieve a 45 dBA interior noise level or lower. The closed window condition of the proposed development can be maintained only by providing an alternate means of ventilation for the interior spaces. Details of window insulation are as follows.

- Sound attenuation of 30 dBA would be needed for sites where future noise levels would be between 70 and 75 dBA. This can be achieved through installing ¼-inch laminated single-glazed windows or double-glazed windows with $^1/_8$-inch glass panes, with ¼-inch air space between them mounted in a heavy frame.

- Sound attenuation of 35 dBA would be required for sites where future noise levels would be between 75 and 80 dBA. This can be achieved through installing double-glazed windows on a heavy frame in masonry structures or windows consisting of laminated glass.

- Sound attenuation of 40 dBA would be required where future noise levels would be between 80 and 85 dBA. This requires the use of noise attenuation measures that typically exceed standard practice for new construction. Achieving the 40 dBA attenuation would require the placement of acoustically well-sealed ¼-inch laminated storm sash 1.5 to 3 inches from single-glazed windows on wood or metal frame.

With the attenuation measures specified above, the proposed action would not have any significant adverse noise impacts and would meet CEQR guidelines. It is anticipated that "E" designations, a restrictive declaration, restrictions in the property deed, or other similar techniques would be used to enforce these noise abatement measures.

Potentially significant adverse impacts to sensitive receptors from odors.

No new odor sources would be created as a result of the proposed action.

No activities are proposed that would exceed accepted City, State, or federal standards with respect to public health or result in activities which result in significant public health concerns. For the reasons stated above, a full assessment of potential impacts on public health is not necessary and no significant adverse impacts are expected as a result of the proposed action. While the proposed project would not meet any of the thresholds warranting a public health assessment, 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.

HEALTH EFFECTS RELATED TO ASTHMA[1]

Certain neighborhoods in New York City -- especially the South Bronx, East and Central Harlem, and Central Brooklyn -- have much higher rates of childhood asthma than others.[2] Given comments from the public expressing concern that exposure to PM -- in particular, emissions of fine particulate matter with an aerodynamic diameter less than 2.5 micrometers in diameter ($PM_{2.5}$) 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 or 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 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; and 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.[3]

Researchers found a 0.5 percent increase in death rates for every increase in the PM_{10}

[1] Portions of the text contained in this section are derived from the December 7, 2005 *Final Environmental Impact Statement for the Gateway Center at Bronx Terminal Market*, prepared by AKRF, Inc., Wachtel & Masyr, LLP, Eng-Wong Taub & Associates, Langan Engineering and Environmental Services, and Sive, Paget & Riesel, P.C., and the November 16, 2007 *Final Environmental Impact Statement for the Proposed Manhattanville in West Harlem Rezoning and Academic Mixed-Use Development*, prepared by AKRF, Inc., Emisstar LLC, HydroQual, Inc., and The Sam Schwartz Company.

[2] Karpati AM, Matte T, Kass D, Garg R, Mostashari F, Thorpe L, Frieden TR. Asthma can be controlled. NYC Vital Signs 2003: 2(4);1-4. New York City Department of Health and Mental Hygiene.

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

concentration level of 10 μg/m, even where ambient levels were well below the National Ambient Air Quality Standards[4] (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.[5] 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}-$PM_{2.5}$).[5] 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.[6]

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.[7] 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.[8] The $PM_{2.5}$ NAAQS were established to address the shortcomings of the PM_{10} NAAQS standard, and to protect public health.

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.[9] During an asthma "attack," an individual experiences difficulty breathing which, if severe enough, and treatment is not rendered, may be fatal in rare instances.[10] Asthmatic episodes may be triggered by specific substances, environmental conditions, and stress.

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

[4] Samet, J.M. et al. N. Engl J. Med, 343, 24, 1742-1749 (2000).
[5] Schwartz, J. et al. J. Air Waste Manag. Assoc 46, 927-939 (1996).
[6] Ware, J. H. Harvard School of Public Health, N. Engl. J. Med., 343, 24, 1798-1799 (2000).
[7] CEPA/FPAC Working Group on Air Quality Objectives and Guidelines. National Ambient Air Quality Objectives for Particulate Matter. Part 1: Science Assessment Document.
[8] USEPA (1996) Air Quality Criteria for Particulate Matter (Vols. I, II, II); EPA/6000/P-95/001af. Washington, DC: Office of Research and Development (1997); National Ambient Air Quality Standards.
[9] 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.
[10] 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.

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 in California have suggested that some incident asthma cases could be related to ozone but not other pollutants.[11]

Prevalence of Asthma

In the US, approximately 6.8 million children (9.3 percent of children under age 18) had asthma. Asthma prevalence in children in New York State is estimated at approximately 9.9 percent.[12] According to the Centers for Disease Control and Protection (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.[13] In 2006, approximately 22.9 million Americans had asthma and the condition accounted for an estimated 12.8 million lost school days in children and 10.1 million lost work days in adults.[14]

Asthma Morbidity and Mortality

Asthma morbidity and mortality rates have been rising throughout the United States over the last few decades,[15] with New York City experiencing a disproportionate increase in the early 1990s.[16] However, hospitalization rates in New York City have been gradually declining since the peak rates in the mid-1990s. Between 1997 and 2005, asthma hospitalization rates among children aged 0-14 years decreased in all New York City boroughs.[17] East Harlem in Manhattan, while continuing to have the highest rate of childhood asthma in New York City, had a significant decrease in rates between 1997 and 2005.[18]

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.[19] In 2005, the hospitalization rate for asthma among children aged 0 to 14 was 5.4 per 1,000 children in New York City.[20] Asthma exacerbations resulting in hospitalizations appear to be particularly frequent and severe among minority, inner-city children, but the disproportionate rates

[11] *The Lancet*, Vol 360, October 19, 2002.
[12] American Lung Association, November 2007. "Trends in Asthma Morbidity and Mortality."
[13] Cookson, W.O.C.M., and M.F. Moffatt. 1997. "Asthma: an epidemic in the absence of infection? *Science* 275:41-42.
[14] American Lung Association, 2007.
[15] Centers for Disease Control (CDC). 1998. "Surveillance for Asthma – United States, 1960-1995." *Morbidity and Mortality Weekly Report* 48(4):1015-1028.
[16] Garg, R., Karpati, A., Leighton, J., Perrin, M., Shah, M., 2003. Asthma Facts, Second Edition. New York City Department of Health and Mental Hygiene.
[17] 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-hospit06-table1.pdf, January, 2008.
[18] Ibid.
[19] Garg et al., 2003.
[20] New York City Department of Health and Mental Hygiene website accessed January, 2008.

among affected groups is likely to be due to factors other 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. Children who live in poor neighborhoods are almost three times as likely to be hospitalized for asthma as those who live in wealthier neighborhoods.[21] This difference in hospitalization rates is a result both of asthma being more common among children living in poorer neighborhoods, and of inadequate control among children with asthma in those communities.[22]

As such, there are striking differences in the number of hospitalizations among New York City boroughs and specific neighborhoods within each borough. Compared with other boroughs, in 2005, hospitalization rates were highest in the Bronx (8.9 cases per 1,000 children).[23] On a neighborhood scale, in 2005, the East Harlem area of Manhattan reported the highest rate of asthma hospitalizations, or approximately 11.9 hospitalizations per 1,000 children.[24] This is higher than the reported average for Manhattan (5.5 per 1,000 children), and higher than the average rate for New York City (5.4 cases per 1,000 children).[25] However, between 1997 and 2005, the area of East Harlem had also shown one of the largest decreases in hospitalization rates (59 percent) among all New York City neighborhoods.[26]

New York City officials are well aware of the epidemic of childhood asthma in the City's boroughs and communities and, under the direction of the New York City Department of Health (NYCDOH), began the New York City Asthma Initiative (NYCAI) 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; and, (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; and, (6) help improve the environment.

The NYCAI's strategic focus has shifted away from administering direct services to activities that support broader system improvements. The NYCAI's primary target continues to be children 0-14 years old in communities with the highest asthma rates. However its program scope includes enhancement of clinical and self-management support for adults with asthma. NYCAI is working to: improve medical standards of care for children and adults with asthma; reduce asthma triggers in both homes and communities; enhance self-management support for individuals with asthma; enhance citywide asthma education standards and delivery; create

[21] Karpati et al., 2003.
[22] Ibid.
[23] New York City Department of Health and Mental Hygiene website, January 2008.
[24] Ibid.
[25] Ibid.
[26] Ibid.

"asthma friendly" schools and daycare settings; and, monitor and track individuals with asthma. The NYCAI continues to coordinate the New York City Asthma Partnership (NYCAP), a citywide coalition of over 300 organizations and individuals initiated in 1999. The New York City Asthma Partnership brings together representatives from schools, daycare, health care institutions, pharmacies, community based organizations, government, and others who make recommendations to improve citywide policies and systems that affect people with asthma. The New York City Asthma Partnership addresses the following: the environment, asthma education, data and research, health care delivery, and issues affecting children in schools, childcare, and recreation programs.[27]

Since the inception of NYCAI, major childhood asthma initiatives have been implemented in several low income neighborhoods having high hospitalization rates. As mentioned above, between 1997 and 2005, 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 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).[28]

In May 2007, it was announced by Mayor Bloomberg that the NYC Department of Health and Mental Hygiene will create the city's first asthma center, the East Harlem Asthma Center of Excellence, to improve the treatment of asthma in East Harlem.[29] The new Asthma Center will be placed in a storefront location, will train medical providers in the latest and most effective treatments and medications, and will help reduce exposure to the environmental triggers that exacerbate asthma. The new facility will offer a variety of education services with the goal of cutting asthma hospitalizations by 50 percent by 2010 – from twelve hospitalizations per 1,000 children to six hospitalizations per 1,000 children. The center will train health care workers in the most effective treatments, provide walk-in screening and help reduce exposure to environmental factors that worsen the condition. The city has many different treatment programs already, but the new center will offer more individualized services and counselors. This includes increasing the number of children who have appropriate medications prescribed, increasing the number of children who will receive assistance to

[27] New York City Department of Health and Mental Hygiene website, January 2008.
[28] 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.
[29] News from the Blue Room from The City of New York website (www.nyc.gov), June, 2007.

address home environmental triggers, increasing the number of hospitalized children who receive asthma counseling services, and working to reduce the causes of asthma.

With the announcement of the new Asthma Center, the "Go Green East Harlem" initiative was also launched.[30] The Go Green initiative is a collaborative community based initiative aimed at breaking the pattern of harmful environmental conditions that have been part of East Harlem's history for decades. Go Green will focus on addressing six core problem areas: public health and asthma, parks and open space, sustainable business, farmers markets and healthy eating, green buildings, and transportation. It will seek community improvements through neighborhood partnerships and programs, legislative change, policy reform and the leveraging of collective resources. The Go Green initiative will be a landmark model for green living in East Harlem and New York City as a whole.

PlaNYC, the Mayor's long-term sustainability plan, aims to give New York City the cleanest air of any City in America by reducing the harmful pollutants that decrease lung function and aggravate asthma. In keeping with the clean air initiatives of PlaNYC, NYSDEC (in cooperation with the City of New York) started a major enforcement action in November 2007 to address urban outdoor air quality in East Harlem.[31] The Stop Smoking Initiative for Trucks and Boilers program is focusing on polluting diesel trucks and boilers, and is centered on East Harlem from 96th Street to 116th Street and from FDR Drive to Fifth Avenue. As part of the initiative, DEC law enforcement officers have been issuing tickets to diesel trucks that fail to comply with state emissions standards on emissions, trucks or buses illegally idling, and boilers found emitting black smoke and polluting the neighborhood. An air monitoring unit has been placed on the grounds of Metropolitan Hospital, on the west side of First Avenue between 97th Street and 99th Street, to monitor the air in the area for two months. Data from these enforcement actions will be used to develop a long term strategy for addressing local outdoor air pollution sources in NYC and other urban areas.

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. 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.

[30] News from the Blue Room from The City of New York website (www.nyc.gov). Site accessed June, 2007.
[31] Business News from the MetroGreen+Business website (www.metrogreenbusiness.com). Site accessed November, 2007.

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.[32] 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.

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, and pet and rodent dander and urine, are the principal indoor air quality triggers of asthma attacks in children. 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.[33] 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

[32] Gentile, D. A. J. Immunology, 65, 4, 347-351 (2004).
[33] 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.

numerous studies. Certain experimental studies evaluated the respiratory and systemic effect of diesel particles on laboratory animals.[34] 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. 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.

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.[35] Experiments in which non-asthmatic adults were exposed for an hour to diesel engine exhaust containing particles and gases found increased airways resistance[36] and some cellular indicators of inflammatory response;[37] 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.[38] 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.[39] Infectious disease has been dramatically reduced in our society by the use of antibiotics and immunization programs.

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.

[34] USEPA (2002, 2003a) IRIS record for diesel engine exhaust, available at www.epa.gov/iris/ subst/0642.htm.
[35] Brunekreef et al 1997, English et al (1999), Livingstone et al (1996).
[36] Rudell et al, Occup. Environ. Med. 53, 6480652, 1996.
[37] Slavi et al, Am. J. Respir. Crit. Care. Med. 159: 702-709, 1999.
[38] Fujieda et al Am J. Respir Cell Mol Biol, 19, 507-12, 1998; Nel et al.
[39] Cookson et al., 1997.

As described in Chapter 3.17, "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 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. It is conservatively assumed that the proposed heating systems in the new development would use No. 2 fuel oil. As part of the HVAC analysis, vehicle exhaust emissions from the proposed bus garage were included.

As described in Chapter 3.17, "Air Quality," an air quality screening analysis was conducted that 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.

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 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. 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, including impacts on asthma rates.