**FINAL SUPPLEMENTAL ENVIRONMENTAL IMPACT STATEMENT FOR THE CROTON WATER TREATMENT PLANT AT THE HARLEM RIVER SITE**

## 7.19. PUBLIC HEALTH

### 7.19.1. Introduction

This section assesses the public health related impacts associated with the proposed Croton Water Treatment Plant at the Harlem River Site and related offsite facilities. Of particular concern are the potential health effects from particulate matter emissions from fuel-burning sources at the plant, as well as diesel emissions from construction-related activities, especially in relation to their effects on asthma rates within New York City and Westchester County. As described in Section 7.11, Air Quality, during construction of the proposed project, construction equipment would generate particulate matter emissions from the combustion of fuel and construction- related activities. These emissions would potentially be of greatest concern in 2009, the peak year of construction, when construction-related on-street truck traffic related to the project would need to traverse the truck routes through the local communities. The planned operation year for the proposed plant is 2011. Section 7.11, Air Quality, assessed the potential particulate matter emissions from the boilers and emergency generators associated with the proposed project. This section presents an overview of the issues related to particulate matter emissions, 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 project-related emissions.

### 7.19.2. Health Effects Related to Emissions of Particulate Matter

#### *7.19.2.1. Overview*[1]

Particulate matter (or PM) is a broad class of air pollutants that exist as liquid droplets or solids, with a wide range of sizes and chemical composition. 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 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 particulate matter 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.

[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.

### *7.19.2.2. $PM_{2.5}$ Issues*

Fine particulate matter, such as $PM_{2.5}$[2], is mainly derived from combustion material that has volatilized and then condensed to form primary particulate matter (often after release from a stack or exhaust pipes) or from precursor gases reacting in the atmosphere to form secondary particulate matter. It is also derived from mechanical breakdown of coarse particulate matter such as pollen fragments.

$PM_{2.5}$ refers to not a single pollutant, but instead 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 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 dramatically. Some particulate matter (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 secondary formation of $PM_{2.5}$ is one determinant of ambient air quality and is, thus far, extremely difficult to model.

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

Data from the Botanical Gardens in the Bronx, NY, and Queens College in Queens, NY, 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). Additional studies confirming the contribution of long-range transport to ambient $PM_{2.5}$ levels compare the data from 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.[3]

An important issue with respect to the health risk associated with $PM_{2.5}$ concentrations is that they have 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 would vary depending on the specific components of the particulate matter in a region. Acid aerosols like sulfuric acid may trigger reactions in pulmonary lung function, while bioaerosols,

[2] Particulates smaller than 2.5 microns.

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

such as mold spores, may result in allergic reactions related to increased incidences of asthma, for example. The United States Environmental Protection Agency's (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 locations and the nature and magnitude of the overall public health risk imposed by ambient particulate matter exposure."

#### *7.19.2.2.1. 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 National Ambient Air Quality Standards (NAAQS) and periodically revise them for such criteria pollutants. Primary NAAQS are mandated to protect public health with an adequate margin of safety. In setting the NAAQS, USEPA must account for uncertainties associated with inconclusive scientific and technical information and potential hazards not yet identified, and the standard must 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 its 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, particularly Chapter V;[4] and USEPA's proposed NAAQS for particulates, found in the December 13, 1996 Federal Register at page 65638. Based on this extensive analysis, in June of 1997, USEPA revised its NAAQS for particulate matter and proposed a new standard for $PM_{2.5}$ consisting of both a long-term (annual) limit of 15 micrograms per cubic meter ($\mu g/m^3$) and a short-term (24-hour) limit of 65 $\mu g/m^3$.[5] 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, and so additional regulation was required. In setting the NAAQS, USEPA was required to account for uncertainties associated with inconclusive scientific and technical information and for potential hazards not yet identified. In setting the value of the annual average NAAQS for $PM_{2.5}$, USEPA found that an annual average $PM_{2.5}$ concentration of 15 $\mu g/m^3$ is below the range of data most strongly associated with both short- and long-term exposure effects. The USEPA Administrator

[4] 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.

[5] 62 Federal Register 38652 (July 18, 1997).

concluded that an annual NAAQS of 15 μg/m$^3$ "would provide an adequate margin of safety against the effects observed in the (se) epidemiological studies."[6] The annual standard is supplemented by a 24-hour standard of 65 μg/m$^3$ to protect against short-term exposures in areas with strong local or seasonal sources.[7]

#### *7.19.2.2.2. Current Status of $PM_{2.5}$ Regulations*

New York State formally recommended that USEPA designate the five counties of the New York City metropolitan area as non–attainment for $PM_{2.5}$. Based on this recommendation, Westchester County is considered to be in attainment of the applicable $PM_{2.5}$ NAAQS. USEPA will finalize the designations by 2005. Once non–attainment designations take effect, the state and local governments will have three years to develop implementation plans designed to meet the standards.

#### *7.19.2.2.3. Public Health Issues Related to Particulate Matter*

The potential for $PM_{2.5}$ to affect public health is dependent on the amount of particulate material in the atmosphere (i.e., the higher the ambient $PM_{2.5}$ concentration, the more likely that it would have an impact), and the composition of the material. The evidence cited by USEPA in establishing the NAAQS for $PM_{2.5}$ is derived from observational epidemiologic studies that found, at typical ambient levels, PM concentrations are statistically correlated with increased levels of morbidity and mortality.[8] It is also 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 simple particle mass concentrations (i.e., total μg/m$^3$), 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

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[6] 62 Federal Register 28652, 38676 (July 18, 1997).

[7] 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/m$^3$ of $PM_{2.5}$ could be present in ambient air without causing adverse health effects.

[8] 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,[9] 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.

As noted above, unlike the other ambient air pollutants regulated at the national level— carbon monoxide, nitrogen dioxide, ozone, lead, and sulfur dioxide—PM ($PM_{10}$ or $PM_{2.5}$) is hardly a single molecule or small set of molecules, but is instead a sundry collection of complex aerosols and microscopic solids with widely varying physical, chemical, and biological properties. The vast differences among various chemical and biological forms of $PM_{2.5}$ mean that these forms also differ significantly in their toxicologic effects.

Considerable research would be required to identify, quantify, and rank the myriad components of $PM_{2.5}$ in terms of their potential importance for public health. The National $PM_{2.5}$ Speciation Program,[10] established under 40 CFR 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 significantly 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).[11] 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.[12]

***$PM_{2.5}$ Rich in Metals.*** Inhalation of metals of various types may harm the upper respiratory tract, lungs, and other organs.[13] 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.[14] In particular, they evaluated ambient PM collected in the late 1980s from the Utah Valley, where PM was rich in copper, zinc, lead, and nickel because of

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

[10] id.

[11] American Lung Association, 2001, http://www.lungusa.org/air/envhayfever.html.

[12] 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.

[13] 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.

[14] 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 dominance of a major steel mill in that valley. Compared with extracts of "ordinary" ambient PM (obtained when the mill was closed), the metal-rich extracts induced several signs of inflammatory injury. The investigators conclude 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.[15]

### *7.19.2.3. Asthma[16]*

Urban populations, such as those in the City, are generally considered to have higher asthma rates than non-urban populations.[17] Given concern that exposure to particulate matter, in particular, $PM_{2.5}$, 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. This discussion would include a review of the risk factors for asthma development and exacerbation; current prevalence, morbidity and mortality estimates of asthma, and whether the scientific literature indicates a causal relationship between truck traffic and the occurrence of asthma.

#### *7.19.2.3.1. Background*

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.[18] During an asthma "attack," an individual experiences difficulty breathing which, if severe enough, and treatment is not rendered, may be fatal in rare instances.[19] Asthmatic episodes may be triggered by specific substances, environmental conditions, and stress, as discussed below.

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[15] 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.

[16] 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.

[17] 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.

[18] 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.

[19] 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.

Although somewhat of a simplification, asthma can be categorized as having either an allergic or a non-allergic basis.[20,21,22] 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.[23] 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.[24]

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. Recently published research 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 NAAQS standards.

#### *7.19.2.3.2. Prevalence of Asthma*

In 1998, the Centers for Disease Control and Prevention (CDC) reported that the estimated self-reported prevalence was between 7 and 10 percent among children.[25] According to the CDC report, over the last two decades the self-reported prevalence of asthma increased 75 percent among all persons of all ages 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. New York is thought to be the state with the second-largest number of affected children. Another report estimated that asthma prevalence in Western countries doubled between 1977 and 1997.[26] 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,[27] decreased physical activity, increasing prevalence of obesity,[28] and increased time spent indoors are hypothesized to be contributing factors to the increase in the prevalence of asthma, the subject is one of continuing research.

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[20] 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.

[21] McFadden, 1987.

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

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

[24] McFadden, 1987.

[25] CDC, 1998.

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

[27] *Ibid*.

[28] 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.

### *7.19.2.3.3. Asthma Morbidity and Mortality*

While asthma morbidity and mortality rates have been rising throughout the United States over the last few decades,[29] New York City has experienced a disproportionate increase. For instance, from 1982 to 1985, the number of deaths from asthma in the City among individuals between 5 and 34 years of age was three times the anticipated number of deaths based on national rates.[30]

In addition, asthma is the leading cause of hospitalization for children ages between 0 and 14 and ranks among the leading causes of hospitalization for all age groups.[31] Asthma exacerbations resulting in hospitalizations appear to be particularly frequent and severe among minority, inner-city children, but the disproportionality 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 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. This difference reflects some combination of variations in asthma prevalence, triggers for asthma exacerbations, access to health care, and hospitalization practices. Between 1988 and 1997, the overall asthma hospitalization rate in New York City increased by 22 percent.[32] This increase was most pronounced in children and in low-income populations.[33] For example, the asthma hospitalization rate for preschool children from low-income areas rose by 63 percent during the 10-year period from 1988 to 1997.[34] In 1997, the hospitalization rate for asthma among children aged 0 to 4 was 16.0 per 1,000 children in New York City, compared to 6.1 per 1,000 in the United States, an almost three-fold difference.[35] In 1998, however, asthma hospitalizations among children decreased approximately 27 percent in New York City compared with 1997 (New York City Department of Health and Mental Hygiene (NYCDOHMH)).

Furthermore, there are striking differences in the number of hospitalizations among the City's boroughs. Compared with the other boroughs, hospitalization and death rates are highest in the Bronx.[36,37] Neighborhood pockets of asthma are also apparent, with East Harlem reporting the highest rate of asthma hospitalizations'–approximately 3,000 hospitalizations per 100,000 persons.[38] The reasons for borough and local disparities in asthma are not known, but may be

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[29] CDC, 1998.

[30] Weiss, K.B., and D.K. Wagener. 1990. "Changing patterns of asthma mortality: identifying target populations at high risk." *Journal of the American Medical Association (JAMA)* 264:1683-1687.

[31] Stevenson, L., Garg, R., and Kaminsky, M., Bijur, P. 1999. *Asthma Facts*. New York City Department of Health, New York City Childhood Asthma Initiative.

[32] *Ibid.*

[33] *Ibid.*

[34] *Ibid.*

[35] United States Department of Health & Human Services. *Healthy People 2010: Understanding and Improving Health.* Washington D.C.: U.S. Government Printing Office, 2000.

[36] Carr, W., L. Zeitel, and K. Weiss. 1992. "Variations in asthma hospitalization and deaths in New York City." *American Journal of Public Health* 82:59-65.

[37] 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.

[38] Stevenson et al., 1999.

due to differences in economic status and ethnicity; exposure to different asthma triggers; or access to medical care.[39,40]

#### *7.19.2.3.4. 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 would 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. 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, 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 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, and by lack of exercise, which might increase the respiratory system's sensitivity to allergens.

[39] 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.
[40] Platts-Mills, 1997.

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. (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). In addition, weather conditions, and cold air in particular, can elicit asthmatic symptoms independent of air pollution.

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. Infectious disease has been dramatically reduced in our society by the use of antibiotics and immunization programs.

#### *7.19.2.4. Potential Public Health Impacts*

##### *7.19.2.4.1. Mobile Sources*

***Potential Public Health Effects.*** As mentioned above, during the peak construction year of the proposed project, construction-related truck traffic is anticipated to increase, potentially contributing to increases in particulate matter levels in the area. Therefore, it is important to determine the relationship between these emissions and asthma. This issue has been addressed, to a limited degree, experimentally and epidemiologically.

Experimentally, exposure to diesel exhaust particles has been shown to increase airways resistance in mice, while other studies of mice and humans have shown that diesel exhaust particles can enhance responses to allergens. Experiments in which non-asthmatic adults were exposed for an hour to diesel exhaust (containing particles and gases) found increased airways resistance and some cellular indicators of inflammatory response; however, these subjects did not experience asthma.

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 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 over the past year, and 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.

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 known, and the triggers for its exacerbation are only partially understood. The potential relationship between vehicular exhaust resulting from increased traffic and asthma, especially in communities with high rates of asthma, requires further study.

***Air Quality Modeling Results.*** As described in Section 7.11, Air Quality, the proposed project involves substantial construction activities for a period of nearly 5.5 to 6 years. The projected period of greatest on- and off-site air quality emissions from construction-related activity would occur in 2009, when an average of up to 634 workers could be on-site. The emissions of airborne particulate matter related to construction would decrease after this year as the project nears completion in 2011.

During construction of the proposed project, construction equipment would generate particulate matter emissions from the combustion of fuel and construction related activities. 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 particulate matter that settles to the ground within a relatively short distance from the source. However, fuel combustion, especially from diesel combustion sources generate particulate matter that mostly consists of $PM_{2.5}$. Heavy construction equipment operating on the site would be dispersed at various locations throughout the 17.5-acre site for the various phases of construction, and much of the time these sources would be located within the site, far from the plant boundaries. However, the construction-related on-street truck traffic related to the project would need to traverse the truck routes through the local communities.

The anticipated construction-related $PM_{2.5}$ impacts associated with the proposed project were predicted in Section 7.11, Air Quality. Analyses were performed for the peak air quality construction analysis year for both on- and off-site emissions. Since future truck trips would be substantially reduced after construction when the proposed project begins operations in 2011, potential $PM_{2.5}$ increments from mobile sources that are related to the operation of the proposed plant are anticipated to be less than those anticipated in the worst-case construction year.

The analysis showed that the maximum predicted incremental daily and annual average $PM_{2.5}$ concentration increments from both off-site construction vehicles and operations of the proposed project were below the interim guidelines used as a threshold for determining significant adverse impacts. In addition, all predicted incremental $PM_{2.5}$ concentrations from on-site construction activities were less than air quality significance thresholds at the nearest sensitive receptors. The total maximum predicted daily (24-hour) concentrations of PM2.5 at all off-site locations are

anticipated to be within the applicable NAAQS. Therefore, since there were no predicted significant adverse air quality impacts, the potential effects of diesel emissions from construction-related on-site activities and off-site truck traffic and operational mobile sources are unlikely to result in a significant adverse impact on public health and local asthma incidents.

#### *7.19.2.4.2. Stationary Sources*

***Potential Public Health Effects.*** Airborne emissions from combustion of distillate fuel oil and natural gas consist primarily of water vapor and carbon dioxide. Also emitted are low levels of PM, nitric oxide (NO) and carbon monoxide (CO), small amounts of $NO_2$, $N_2O$, and $SO_2$, and trace amounts of volatile organic compounds (VOCs), methane, and metals (*AP42*, External Combustion Sources, Section 1.3, September, 1998, and Stationary Internal Combustion Sources Section 3.1, April, 2000). Emissions of sulfur-based compounds (e.g., $SO_2$, sulfur trioxide) are a direct function of the quantity of sulfur in the fuel.

Particulate matter emitted from natural gas boilers consists primarily of organic products of incomplete combustion, and is very low in metal content (*AP42*, Section 1.3, September, 1998 and Section 3.1, April, 2000). Further, this particulate matter contains no biological material. Small amounts of nitrates and sulfates may be present in this particulate matter (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 particulate matter, 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.[41] 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.[42]

#### *Air Quality Modeling Results.*

The air quality modeling analysis in Section 7.11, Air Quality, determined that maximum predicted incremental daily and annual average $PM_{2.5}$ concentration increments from the proposed plant are anticipated to be below the interim guidelines used as a threshold for determining significant adverse air quality impacts. Also, the total maximum predicted daily (24-hour) concentrations of $PM_{2.5}$ at off-site locations are anticipated to be within the applicable NAAQS. Therefore, since there were no predicted significant adverse air quality impacts, the potential effects of airborne emissions from the proposed project are unlikely to result in a significant adverse impact on public health and local asthma incidents.

### 7.19.3. Health Effects Related to Rodent and Pest Control

Urban pest control issues largely encompass vermin infestations from local sewers. These infestations are part of a larger public health issue, for rats and the fleas that they carry are

[41] 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.

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

frequent vectors of communicable diseases. Additionally, rat attacks on urban homeless populations and vulnerable individuals, such as infants, have been a less frequent but extremely grave public health risk. Typically rats are attracted from their underground lairs to search for food in local apartments and piles of garbage that line the sidewalks in New York[43]. However, they can also be driven out of their underground homes by construction activity.

This problem attracted media attention during the 1990s Central Artery/Tunnel project in Boston, subsequently dubbed "the Big Dig" due to the 15 million cubic yards of earth excavated throughout construction[44]. Fears that excavation of this size would unearth a rodent population of 80,000[45] led to the establishment of the Central Artery Rodent control Division, whose jurisdiction covers a 70 mile area around the construction site, and the city-run Inspectional Services Department (ISD)[46]. These efforts were so successful that Integrated Pest Management (IPM) and word of the "Boston Model" has spread worldwide. In fact, the IPM strategy was able to decrease the number of rat complaints from 1,814 in 1995 to 647 in the year 2000. The IPM methods encompasses a wide range of strategies, from the proper containment and timely clean up of garbage, to the minimization of the low-lying shrubbery rodents are partial to making their homes under.

#### *7.19.3.1. NYC Pest Control History*

Pest rodents in urban areas of the northeastern United States include the non-native Norway rat (*Rattus norvegicus*) and house mouse (*Mus musculus*). At times, the native white-footed mouse (*Peromyscus leucopus*) also can be associated with vegetation in urban areas. The Norway rat and house mouse characteristically are referred to as commensal species (i.e., feed from our tables), and both species are well known for their ability to colonize urban infrastructure. Their presence poses a public health concern and suggests a need for improved environmental management (particularly sanitation, refuse storage, and infrastructure repair).

The Bronx itself had this problem in mid 1990s, particularly during housing development construction in vacant lots where the animals had previously been able to live undisturbed[47]. At this time funding for pest prevention had been cut, and the decrease in control allowed for an increase in the rodent population. Once drilling for a project began the animals would be scattered throughout the street, making this underlying infestation increasingly apparent to the public. Subsequently, the Mayors Office redoubled its efforts against vermin in August 1997 with the Comprehensive Rodent Control Initiative[48], including a NYC Health Department

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[43] New York City Department of Health. 2002. Press Release.
http://www.nyc.gov/html/doh/html/public/press00/p1161023.html

[44] Central Artery/Tunnel Project Website. 2002. http://www.bigdig.com/thtml/enviro.htm

[45] South Coast Today. 4/7/02. "Rats! Boston Residents Expect a Rise in Rodent Population this Summer."
http://www.s-t.com/daily/04-07-02/b06sr073.htm

[46] Wright, Chris. 9/18/02. "Rats in Paradise"
http://www.bostonphoenix.com/boston/news_features/top/features/documents/02194425.htm

[47] Padgett, Tina. 1995. Rats Rate Bronx No. 1 Place to Live. Columbia University.
http://www.columbia.edu/cu/bb/oldstuff/bb0501.24.html

[48] New York City Department of Health Bureau of Pest Control. November 1997.
http://www.nyc.gov/html/doh/html/pest/ratwork.html

campaign begun in 2000 titled “You Feed Them, You Breed Them -- Help New York City Send Rats Packing." These efforts utilize the IPM method, focusing on not only eliminating individual complaints, but also reducing the overall rodent population. In fact, between the mid 1990s and the year 2000 the number of both inspections and exterminations has approximately doubled[49]. The number of complaints logged with Pest Control Services (PCS) in 2001 is currently estimated to be 15-16,000;[50] whereas back in 1995 the number of complaints was 27,000.[51]

In order to reduce the overall rodent population, efforts are being made to extinguish food sources and shelter through education of the community about rat-proofing groceries and possible entranceways into apartments, as well as imposing more stringent requirements on local landlords and storeowners. These requirements, which concentrate much of the effort on dining establishments, are defined a set of guidelines for the inspection and enforcement of waste clean up. Storeowners are required to place their refuse in tightly sealed containers, and defined procedures have been established for issuing warnings and fines if these precautions have not been properly adhered to. Procedures have also been implemented by the PCS for the abatement of rodents in public areas, including the assessment of construction activity to determine adequate methodology for specific cases.[52] Areas also under the jurisdiction of PCS maintenance include sewers, street areas and catch basins, which are periodically treated with rodenticides. In addition to efforts that concentrate on depleting the rodent food supply, the aforementioned Department of Health programs target elimination of crevices that make shelter accessible to rodents. These efforts seek to reduce viable nesting places in local residential buildings by urging local residents to block possible entranceways with wire mesh and enforcing requirements on landlords to maintain building integrity. Additionally, these efforts focus on reduction of the rodent population by hindering their ability to leave pre-established nests to hunt for food, therefore, decreasing the population through lack of nutrition, and also hindering the reproductive capability of those individuals not as easily susceptible to starvation.

In the Future Without the Project, no introduction or changes to the current management policy is anticipated.

There are three time periods when rodents can cause impacts during a construction operation: 1) if there is a pre-existing population at the planned work site, they may be displaced during initial excavations; 2) rodents can be attracted to work sites from bordering blocks during construction; and 3) rodents can be displaced from work sites as construction is completed and debris and materials are removed. For these reasons, a pest management program would be implemented to resolve rodent activity in the proposed work areas before construction begins and then the

[49] New York City Department of Health, office of Public Affairs. 2000. http://www.nyc.gov/html/doh/html/public/testi/rats920.html

[50] Information obtained from a phone interview on 9/24/02 with Rick Simon, Director of Operations Department of Health Pest Control Services.

[51] New York City Department of Health and Mental Hygiene-Pest Control Services. 2000. http://www.nyc.gov/html/doh/html/pest/pest1.html

[52] New York City Department of Health and Mental Hygiene-Pest Control Services. 2000. http://www.nyc.gov/html/doh/html/pest/pest1.html

program would be maintained for the duration of construction. Prior to implementation, this pest management program would be reviewed and approved by NYCDOHMH.

The pest management program would be implemented using standard specifications and protocols for construction projects, considering the known behavior of pest rodents. See Appendix I for details regarding the Rodent Control and Management Plan. This entails base-line survey (inspection) of the proposed work area and abutting block, prior to construction, to determine the presence and distribution of rodents. The program would then be tailored based on the survey results. The program would include trapping and rodenticide application by a licensed pest management professional (PMP) within and abutting the proposed work area, prior to construction, in order to eliminate any pest rodents found. During construction, the PMP would monitor and inspect the work site weekly for any new rodent activity and site sanitation, and apply control measures as warranted. The PMP would keep records of observations and activities. Site sanitation would include proper use of refuse containers and minimizing unnecessary debris, and construction contracts would specify housekeeping requirements.

The project would maintain coordination with city agencies responsible for rodent control and code enforcement, and also neighborhood representatives, so that any concerns in adjoining blocks can be effectively communicated. The project would work with the City and abutters concerning investigation and resolution of public concerns during construction, and conform to City regulations.

The Harlem River Site includes waterfront edge, industrial land use, and borders a railroad and highway corridor. Given current conditions, rodent control and prevention of community impacts is not anticipated to be difficult. Oldfield and shrub edge conditions provide some habitat for mice, and edge and sanitation conditions may support limited rat activity at times. The existing Major Deegan Expressway and Metro North railroad tracks separate the closest residences from the construction site, providing a significant barrier between the neighborhood and the proposed site. This restricts the potential for neighborhood impacts. Tunnels to and from the distribution connection would be bored in rock 100 feet and more beneath any buildings. The City's extensive experience with rock tunnels for water supply tunnels has not raised any concerns about causing movements of rodent populations toward human habitations. No significant increase in the emergence of rodent populations is anticipated to arise during construction activities.

### 7.19.4. Conclusion

The causes of asthma and its increase over the last two decades are not known, 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. Air quality modeling results show insignificant increases in the short-term and annual average concentrations of $PM_{2.5}$ from the construction or operation of the proposed Croton project. Therefore, potential $PM_{2.5}$ emissions from mobile and stationary sources related to the construction and operation of the proposed project are not anticipated to result in adverse public health impacts.

During construction activities, the current management policy of the NYCDOHMH is anticipated prevent any significant adverse impacts about causing movements of rodent populations toward human habitations. The contractor would be responsible for the control of rodent populations onsite; therefore no significant adverse impact is anticipated from construction activity.