FINAL ENVIRONMENTAL IMPACT STATEMENT FOR THE CATSKILL/DELAWARE UV FACILITY

4.19. PUBLIC HEALTH

4.19.1. Introduction

This section discusses the public health issues associated with the proposed Catskill/Delaware Ultraviolet Light Disinfection Facility (UV Facility) at the Eastview Site and off-site facilities. Of particular concern is the potential of particulate matter emissions from fuel-burning sources at the plant, as well as diesel emissions from construction-related activities, to impact public health and to increase the already high asthma rates within Westchester County. As described in Section 4.10, Air Quality, during the construction of the proposed UV Facility, construction equipment would generate particulate matter (or PM) emissions from the combustion of fuel and construction-related activities. These emissions are expected to be of greatest concern in 2006, the peak year of on- and off-site construction impacts on air quality. In 2006, the project would also generate the largest amount of construction-related on-street truck traffic that would need to traverse the local community truck and traffic routes. For analysis purposes in this Final Environmental Impact Statement (EIS), 2010 was used as the first full year of operation for the proposed facility. Section 4.10, Air Quality, assessed the potential particulate matter emissions from the natural gas/oil boilers and the diesel emergency generators associated with the proposed UV Facility. This section presents an overview of the health concerns related to particulate matter emissions, a discussion of asthma and its prevalence in Westchester County, and an assessment of the potential public health effects from the project-related emissions. This section also includes a discussion of the West Nile Virus and the potential of the proposed project and project-related activities to impact the local mosquito population.

4.19.2. Health Effects Related to Emissions of Particulate Matter

4.19.2.1. Overview[1]

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

PM is emitted by a variety of natural and man-made sources. Natural sources include the condensed and reacted forms of natural organic vapors, salt particles resulting from the evaporation of sea spray, wind-borne pollen, fungi, molds, algae, yeasts, rusts, bacteria, and debris from live and decaying plant and animal life, particles eroded from beaches, desert, soil and rock, particles from volcanic and geothermal eruptions and forest fires.

Major man-made sources of particulate include the combustion of fossil fuels, such as vehicular exhaust, power generation and home heating, chemical and manufacturing processes, all types of

[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.

construction, agricultural activities and wood-burning fireplaces. Since the chemical and physical properties of PM vary widely, the assessment of the public health effects of the airborne pollutants in ambient air is extremely complicated. The principal health effects of airborne particulate matter are on the respiratory system, although recent research investigated the possible link between particulate matter pollution and cardiovascular disease.[2]

Researchers found a 0.5 percent increase in death rates for every increase in the PM_{10} concentration level of 10 $\mu g/m^3$, even where ambient levels were well below the National Ambient Air Quality Standards[3] (NAAQS). The authors of that research recognized the limitations of their work. They explained that they used PM_{10} data in their study because at that time $PM_{2.5}$ data were not yet available nationally.[3] Some studies have found that the daily mortality rate is associated with the concentration of fine particles ($PM_{2.5}$) but not coarse particles (PM_{10}-$PM_{2.5}$).[4] 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.[5]

Numerous studies have correlated increased rates of hospital admissions for respiratory conditions, small decreases in lung function in children with or without asthma, and absences from school with changes in PM concentrations.[6] .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.[7] The $PM_{2.5}$ standard was established to address the shortcomings of the PM_{10} standard and to protect public health.

4.19.2.2. $PM_{2.5}$

As mentioned above, PM is a byproduct of fossil fuel combustion. It is also derived from mechanical breakdown of coarse particulate matter such as pollen fragments. $PM_{2.5}$ does not refer to a single pollutant, but to an array of fine inhalable materials. There are, for example, thousands of forms of natural ambient $PM_{2.5}$ and perhaps as many forms of man-made $PM_{2.5}$, which include the products of fossil fuel combustion (such as diesel fuel), chemical/industrial processing and burning of vegetation. While all the disparate forms of $PM_{2.5}$ can be inhaled, their

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

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

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

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

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

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

toxicological properties can differ. Some PM is emitted directly to the atmosphere (i.e., primary PM), while other types of particulate matter are formed in the atmosphere through various chemical reactions and physical transformations (i.e., secondary PM). The formation of secondary $PM_{2.5}$ is one determinant of ambient air quality and is, thus far, extremely difficult to model.

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

Data from the Botanical Gardens in the Bronx and Queens College in Queens, New York City indicate that the greatest contributors to ambient $PM_{2.5}$ concentrations are sulfates and organic carbon (approximately two thirds of the total $PM_{2.5}$ mass). Studies confirming the contribution of long-range transport to ambient $PM_{2.5}$ levels compared the data from New York City monitors to monitors from a remote site within the state, downwind from other states. These data show that high levels of sulfate and other pollutants come into New York State from areas to the west and south of New York. The data also indicate that urban sites are more likely to experience increased nitrate and carbon levels than rural sites.[8]

An important issue associated with $PM_{2.5}$ is that it has a direct causal effect on human health. Since particulate matter in the ambient air is comprised of a combination of discrete compounds or elements, its possible public health effects could vary depending on the specific components of particulate matter in a region. Acid aerosols such as sulfuric acid may trigger reactions in pulmonary lung function, while bioaerosols, such as mold spores, may result in allergic reactions related to increased incidences of asthma, for example. The USEPA 1996 Criteria Document acknowledged this uncertainty:

> *"There remains uncertainty regarding the shapes of particulate matter exposure-response relationships; magnitude and variability of risk assessments for particulate matter; the ability to attribute observed health effects to specific particulate matter constituents; the time intervals over which particulate matter health effects are manifested; the extent to which findings in one location can be generalized to other locations and the nature and magnitude of the overall public health risk imposed by ambient particulate matter exposure."*

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

4.19.2.2.1. 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 NAAQS and periodically revise them for such criteria pollutants. Primary NAAQS are mandated to protect public health with an adequate margin of safety. In setting the NAAQS, USEPA must account for uncertainties associated with inconclusive scientific and technical information and potential hazards not yet identified. The standard must also be adequate to protect the health of any sensitive group of the population. Secondary NAAQS are defined as standards that are necessary to prevent adverse impacts on public welfare, such as impacts to crops, soils, water, vegetation, wildlife, weather, visibility, and climate.

Beginning in 1994, USEPA conducted a five-year review of the NAAQS for particulate matter, which included an in-depth examination of epidemiologic and toxicological studies. USEPA also held public meetings across the nation and received over 50,000 oral and written comments regarding these studies, particularly as to whether $PM_{2.5}$ is correlated with adverse health effects, and at what ambient air concentrations of $PM_{2.5}$ these correlations hold. The studies are summarized in USEPA's Criteria Document for Particulates, Chapters 10-13 (1996); USEPA's Staff Papers on Particulates, in particular Chapter V[9]; and USEPA's proposed NAAQS for particulates, found in the December 13, 1996 Federal Register on page 65638. Based on this extensive analysis, in June of 1997, USEPA revised the NAAQS for particulate matter and proposed a new standard for $PM_{2.5}$ consisting of both a long-term (annual) limit of 15 $\mu g/m^3$ and a short-term (24-hour) limit of 65 $\mu g/m^3$.[10]

In establishing the NAAQS for $PM_{2.5}$ in 1997, USEPA conservatively assumed that moderate levels of airborne PM of any chemical, physical, or biological form might harm health. In setting the value of the annual average NAAQS for $PM_{2.5}$, USEPA found that an annual average $PM_{2.5}$ concentration of 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 concluded that an annual NAAQS of 15 $\mu g/m^3$ "would provide an adequate margin of safety against the effects observed in the epidemiological studies."[11] The annual standard is supplemented by a 24-hour standard of 65 $\mu g/m^3$ to protect against short-term exposures in areas with strong local or seasonal sources.[12]

In addition to the NAAQS, NYCDEP has promulgated an interim guidance for $PM_{2.5}$. The interim guidance requires a $PM_{2.5}$ neighborhood analysis for actions that have potential significant impact. In the neighborhood analysis, an area of 1 km^2, centered at the maximum

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[9] 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.

[10] 62 Federal Register 38652 (July 18, 1997).

[11] 62 Federal Register 28652, 38676 (July 18, 1997).

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

predicted ground-level concentration, is considered. According to the interim guidance, actions should not exceed an average annual $PM_{2.5}$ concentration increment of 0.1 $\mu g/m^3$ within the 1 km^2 area considered. To put this value in perspective: 0.1 $\mu g/m^3$ constitutes less than one percent of the annual NAAQS for $PM_{2.5}$; A mass of 0.1 μg is roughly a million times smaller than the mass of a small bread crumb. A concentration increment that is lower than the incremental neighborhood guidance concentration would not be registered by the ambient air monitors.

4.19.2.2.2. 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. Data from the Mamaroneck station in Westchester County show that measured $PM_{2.5}$ concentrations are in compliance with the 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 these standards.

4.19.2.2.3. Public Health Issues Related to Particulate Matter

The potential for $PM_{2.5}$ to affect public health is dependent on the composition and the amount of PM in the atmosphere (*i.e.*, the higher the ambient $PM_{2.5}$ concentration, the more likely that it would have an effect). The evidence cited by USEPA in establishing the NAAQS for $PM_{2.5}$ is derived from epidemiologic studies that found, at typical ambient levels, a statistical correlation of PM and increased levels of morbidity and mortality.[13,14] It is unclear what forms of PM and what physiological mechanisms are responsible for the observed health effects. However, the extent of any adverse public health effect related to an increase in PM concentrations is anticipated to be proportional in some way to the concentration increase. A small increase in PM concentrations can, at most, lead to a small increase in PM related public health effects.

Although the NAAQS for $PM_{2.5}$ is based on the measurement of particle mass concentrations (*i.e.*, total $\mu g/m^3$), 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

[13] Krewski et al (2000); Dockery et al. N. Engl. J. Med. 329, 1753-1759 (1995); Pope et al Am. J. Respir. Crit. Care Med., 151:669-674 (1995), Burnett et al, JAMA 287(9), 1132-41 (2002); Dominici et al, Am. J. Epidemiol. 157 (12), 1055-1065 (2003).

[14] 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.

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 Development,[15] is focused on attempting to better understand the biological, chemical, and physical characteristics of PM underlying its potentially toxic effects. A basic finding among these studies is that different forms of $PM_{2.5}$ differ substantially in their toxicologic significance.

Considerable research would be required to identify, quantify, and rank the myriad components of $PM_{2.5}$ in terms of their potential effect on public health. The National $PM_{2.5}$ Speciation Program,[16] established under 40 Code of Federal Regulations Part 58 as mentioned above, would serve as only a modest, first-cut analysis, as it would provide no information on the biologic content of ambient air PM, and only limited information on some metallic, ionic, and organic constituents of ambient PM. Although chemical and toxicologic knowledge of ambient $PM_{2.5}$ is limited, current evidence, as outlined below, suggests that $PM_{2.5}$ that is rich in either biologically-active material or in various metals is more harmful than $PM_{2.5}$ that has little to no biologic or metallic content.

Biologically Active $PM_{2.5}$. Particulate matter rich in pollen and other aero-allergens is well known to exacerbate respiratory problems, especially among people with allergic asthma and sufferers of hay fever (also called seasonal allergic rhinitis).[17] 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.[18]

$PM_{2.5}$ Rich in Metals. Inhalation of metals of various types may harm the upper respiratory tract, lungs, and other organs.[19] 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.[20] In particular, they evaluated ambient PM collected in the late 1980s from Utah Valley, where PM was rich in copper, zinc, lead, and nickel because of the dominance of a major steel mill in that valley. Compared with extracts of "ordinary" ambient PM (obtained when the mill was closed), the metal-rich extracts induced several signs of inflammatory injury. The investigators concluded that "metal content, and consequent oxidative stress that paralleled metal concentrations" caused the injury they observed, so that "mass may

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

[16] *Ibid*.

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

[18] 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.

[19] 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.

[20] 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.

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.[21]

4.19.2.3. Asthma[22]

High-density populations, such as those in the City and southern Westchester County, are generally considered to have higher asthma rates than non-urban populations.[23] Given the concern that exposure to particulate matter emissions, especially $PM_{2.5}$ from activities associated with the proposed facility could either aggravate pre-existing asthma or induce asthma in an individual with no prior history of the disease, the potential for emissions of $PM_{2.5}$ to precipitate the onset or exacerbation of asthma is examined in the following discussion. The discussion includes a review of the risk factors for asthma development and exacerbation; current prevalence, morbidity and mortality estimates of asthma, and a survey of the scientific literature that discusses the relationship between truck traffic and the occurrence of asthma.

4.19.2.3.1. Background

Asthma is a complex disease with multiple causes and substantial inter-individual variation in the severity of symptoms. It is a chronic inflammatory disorder of the airways characterized by variable airflow obstruction and airway hyperresponsiveness in which prominent clinical manifestations include wheezing and shortness of breath.[24] During an asthma "attack," an individual experiences difficulty breathing which, if severe enough, and treatment is not rendered, may be fatal in rare instances.[25] Asthmatic episodes may be triggered by specific substances, environmental conditions, and stress, as discussed below.

Although somewhat of a simplification, asthma can be categorized as having either an allergic or a non-allergic basis.[26,27,28] 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

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[21] 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.

[22] 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.

[23] 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.

[24] 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.

[25] 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.

[26] 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.

[27] McFadden, 1987.

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

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.[29] 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.[30]

Studies have demonstrated an increase in daily mortality, hospitalizations and emergency department utilization for asthma, attributable to air quality diminution from increased levels of sulfur dioxide, ozone and particulate matter. However, in children living in 24 US and Canadian communities, significant associations were reported between exposure to fine particles and their acidity and reduced lung function, symptoms of bronchitis, but not asthma. Children relocating from high to low pollution areas (or vice versa) were shown to experience changes in lung function growth that mirrored changes in exposure to particulate matter. The relation of variations in asthma prevalence to air pollution has been difficult; although, prospective studies in California have suggested that some incident asthma cases could be related to ozone but not other pollutants[31]

4.19.2.3.2. Prevalence of Asthma

In 1998, the Center for Disease Control and Prevention (CDC) reported that the estimated self-reported prevalence of asthma among children was between 7 and 10 percent.[32] According to the CDC report, 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. 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.[33] Other parts of the world have also reported an increase in asthma prevalence in urban areas. Though changes in infectious disease patterns,[34] decreased physical activity, increasing prevalence of obesity,[35] 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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[29] Centers for Disease Control (CDC). 1998. "Surveillance for Asthma – United States, 1960-1995." *Morbidity and Mortality Weekly Report* 48(4): 1015-1028.

[30] McFadden, 1987.

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

[32] CDC, 1998.

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

[34] *Ibid*.

[35] 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.

4.19.2.3.3. Asthma Morbidity and Mortality

In the US, approximately five million children (7 percent of children under age 18) have asthma[36]. While asthma morbidity and mortality rates have been rising throughout the US over the last few decades,[37] New York City has experienced a disproportionate increase. For instance, from 1982 to 1985, the number of deaths from asthma in New York City among individuals between 5 and 34 years of age was three times the anticipated number of deaths based on national rates.[38] The comparable rates for Westchester County are greater than the national rate, but lower than New York City.

The number of deaths from asthma in Westchester County between 1998 and 2000 was 37, or 1.3 deaths per 100,000 residents.[39] In terms of morbidity, 1,076 hospital discharges in Westchester County were due to "asthma attacks." Among females, asthma-related hospital stays occurred most frequently in the 45- to 49-year-old range, while males seemed to suffer from asthma-related hospitalization more frequently in the 1- to 2-year-old age group. Over one-quarter (26.4 percent) of asthma-related hospital discharges occurred among children ages 10 years or younger. Asthma exacerbations resulting in hospitalizations appear to be particularly frequent and severe among minority, inner-city children.[40].

4.19.2.3.4. 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 increased rates of asthma, however, and different factors likely dominate in different areas, homes, and individuals. In theory, one can distinguish between "causes" and "triggers" of asthma. Causes are those factors that make a person susceptible to asthmatic attacks in the first place, while triggers are those factors that elicit asthmatic symptoms at a particular time. Immunologists are increasingly coming to understand asthma as a genetic disorder. While genetic predisposition seems to be

[36] Centers for Disease Control 1998, 1999, www.cdc.gov/nceh/programs/asthma/default.htm

[37] CDC, 1998.

[38] 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.

[39] Data obtained from the New York State Department of Health—Center for Community Health website on May 5, 2004: http://www.health.state.ny.us/nysdoh/chac/cha00/ast0.htm.

[40] Lobach 1996, New York City Department of Health.

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.[41]

Triggers are more easily studied, but may not be the underlying causes of the disease. For example, although a genetic predisposition to allergy is an important risk factor for developing asthma, there may have been no real increase in the number of genetically susceptible children, but rather a growth in the prevalence of factors that promote asthma development or trigger an attack. For a person suffering from asthma, however, the identification and elimination of triggering factors is of greatest practical importance.

Allergens in the indoor environment are important triggers of asthma in the US. Organic materials that cause the immune system to overreact, such as cockroach antigens, dust mite antigens, molds, pet and rodent dander and urine, are the principal indoor air quality triggers of asthma attacks in children. Some of these antigens are probably more common in poor quality housing, which could explain, in part, why poor children suffer high rates of asthma. Other indoor pollutants, such as tobacco smoke and natural gas combustion products, can also exacerbate asthma symptoms. "Improvements" in housing, such as increased insulation and reduced ventilation to save on energy costs, and increased amounts of wall-to-wall carpeting and stuffed furniture, may have the unintended effects of promoting growth of dust mites and molds, and of concentrating antigens, irritants, and particulate matter indoors. These changes in housing over recent decades could help explain the widespread increases in asthma rates. In addition, the effect of indoor pollutants may be increased by the growing amount of time that children spend indoors, which increases a child's exposure to antigens. The lack of exercise might also increase the respiratory system's sensitivity to allergens.

Some natural aspects of outdoor air, such as pollens, are capable of triggering asthma attacks. On a local scale, air pollution may be important, and on a larger scale, it is possible that specific pollutants, such as ozone or diesel exhaust, enhance the effects of other factors, such as allergens, even if the pollutants themselves are not triggers of asthma. Though some epidemiologic studies have found an association between 24-hour average PM_{10} (particulate matter, less than 10 microns in diameter) levels and asthma hospitalizations and emergency room visits others have not.[42] In addition, weather conditions, and cold air in particular, can elicit asthmatic symptoms independent of air pollution.

4.19.2.3.5. Asthma and Traffic Sources of Air Pollution

Scientists have been studying possible links between respiratory diseases or symptoms, such as cough, asthma and bronchitis, and traffic. Particles emitted by diesel engines are generally small enough to be counted as $PM_{2.5}$. The toxic effects of diesel engine exhaust have been evaluated in numerous studies. Certain experimental studies evaluated the respiratory and systemic effect of diesel particles on laboratory animals.[43] The studies revealed that chronic and/or prolonged continuous exposures of the animals to large concentrations cause

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

[42] 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

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

inflammation, fibrosis and functional changes in the respiratory system, and that very large concentrations cause premature death. The lowest observed adverse effect levels, as well as no observed adverse effect levels occurred at concentrations that were considerably in excess of ambient concentrations. Specifically, the levels at which these effects were not observed ranged from 100 to 500 μg of diesel particulates per cubic meter, concentrations that are above allowable average daily values.

Epidemiologically, a few studies have addressed childhood asthma in relation to distance from roads and hence, from vehicle exhaust. For example, young children in Birmingham, England admitted to hospitals with a diagnosis of asthma were more likely to live close to busy roads; than children admitted for other reasons. The apparent risk of admission for asthma was increased by almost two-fold for children who live close to busy roads. Undercutting the significance of these findings was the lack of information about their socioeconomic status, family history of asthma, and the indoor environment. Other epidemiological studies have demonstrated an increase in daily mortality, hospitalizations and emergency department utilization attributable to air quality diminution from increased levels of sulfur dioxide, ozone and particulate matter.

In a study conducted in the Netherlands, researchers found that residence near busy streets was associated, in children, but not adults, with a one and a half fold increase in wheezing symptoms in the past, with a 4.8 fold higher use of asthma medications among children after controlling for various socioeconomic and indoor environmental exposures. Other studies have not found an association between asthma symptoms or hospitalizations and residence near heavy traffic.

Most studies found associations between some indicator of traffic (distance to roads, traffic volumes, or truck traffic volumes) near a residence or school and some indicator of respiratory disease (allergic rhinitis, wheezing or cough), while a few found no evidence of an association.[44] Experiments in which non-asthmatic adults were exposed for an hour to diesel engine exhaust containing particles and gases found increased airways resistance[45] and some cellular indicators of inflammatory response;[46] 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.[47] 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.

[44] Brunekreef et al 1997, English et al (1999), Livingstone et al (1996).
[45] Rudell et al, Occup. Environ. Med. 53, 6480652, 1996.
[46] Slavi et al, Am. J. Respir. Crit. Care. Med. 159: 702-709, 1999.
[47] Fujieda et al Am J. Respir Cell Mol Biol, 19, 507-12, 1998; Nel et al.

4.19.2.4. Proposed Facility, Air Quality, and Public Health

The construction and operation of the proposed UV facility is expected to result in PM emissions. The sources of these emissions are construction-related truck traffic, on-site construction-related mobile and stationary sources, and the boilers and turbine generators necessary for the operation of the proposed facility. During the peak construction year of the proposed facility, construction-related truck traffic is anticipated to increase, potentially contributing to increases in particulate matter levels in the area. PM emitted from turbine generators combusting distillate fuel oil consists primarily of organic products of incomplete combustion, and is very low in metal content.[48] Further, this PM contains no biological material. Small amounts of nitrates and sulfates may be present in this PM (given the gas-phase presence of nitrogen oxides and sulfur dioxide), and NO_x, SO_2 and ammonia emissions may lead to further (but much more diffuse) formation of secondary 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.[49] 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.[50] The potential increase of PM that would result from the project was evaluated using air quality models for several scenarios, with and without the Croton project.

4.19.2.4.1. Air Quality Modeling Results

As described in Section 4.10, Air Quality, the proposed facility would involve substantial construction activities for a period of five years. The projected period of greatest on- and off-site air quality emissions from construction-related activity would occur in 2006. The emissions of airborne particulate matter related to construction would decrease after this year as the project nears completion in 2009.

During construction of the proposed facility, construction equipment would generate particulate matter from the combustion of fuel and construction related activities. With respect to $PM_{2.5}$, fuel combustion is the primary source of this pollutant. PM generated by construction-related transfer of materials and other fugitive dust sources tends to be larger size particulate matter that settles to the ground within a relatively short distance from the source. However, fuel combustion, especially from diesel sources generates particulate matter that contains a significant amount of $PM_{2.5}$. Heavy construction equipment operating on the site would be dispersed throughout the site during the construction period. 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 local community traffic routes.

The anticipated construction-related $PM_{2.5}$ impacts associated with the proposed facility were discussed in Section 4.10, Air Quality, and Section 5, Off-Site Facilities. Analyses were

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

[49] Concentrations of at least 100 micrograms of sulfate or nitrate per cubic meter of air are required before even minimal changes in respiratory function can be observed, even in asthmatic subjects or in sensitive laboratory rodents. See USEPA 2001 (PM Criteria Document Draft) for extended discussion and references.

[50] See USEPA 2001 (PM Criteria Document Draft) for extended discussion and references.

performed for the peak air quality construction year for both on- and off-site emissions. Since future truck trips would be substantially reduced after construction when the proposed facility begins operations in 2009, potential $PM_{2.5}$ increments from mobile sources that are related to the operation of the facility are anticipated to be less than those anticipated in the worst-case construction year.

The air quality modeling analysis predicted that the maximum daily and annual average $PM_{2.5}$ concentration increments from construction vehicles and equipment would be lower than the interim guideline concentration increments, used as a threshold for determining significant adverse impacts. The predicted impact of the UV project on $PM_{2.5}$ concentrations at the nearest sensitive receptor (neighborhood annual and 24-hour) is insignificant under all scenarios considered, with and without the Croton project.

The air quality modeling analysis in Section 4.10, Air Quality, predicted daily and annual average $PM_{2.5}$ concentration increments from operations of boilers and generators at the proposed UV Facility. These concentration increments were below the interim guidelines used as a threshold for determining significant adverse air quality impacts. For the neighborhood analysis, the maximum annual predicted incremental impact was in compliance with the NYCDEP interim guidance criterion of 0.1 $\mu g/m^3$. There would be no additional increases in the airborne emissions from the operational boilers and generators for the proposed facility if the Croton project were also constructed and operated at the Eastview Site. However, the potential effect of the dispersion of the air pollutants discharged by the proposed facility by the structure of the Croton project (*i.e.*, structures may affect local wind fields, resulting in different localized air flows) was assessed in Section 4.10, Air Quality. Under this scenario, the air quality modeling analysis presented in Section 4.10, Air Quality, also showed that the maximum daily and annual average $PM_{2.5}$ concentration increments from operations of boilers and generators at the facility were below the interim guidelines used as a threshold for determining significant adverse air quality impacts.

Therefore, the $PM_{2.5}$ emissions from mobile (on-site and off-site) and stationary construction sources and from the boilers and generators associated with the operation of the UV facility are not expected to increase the concentration of $PM_{2.5}$ by an amount that would have an adverse impact on public health under any scenario.

4.19.3. Detention Basins and Public Health

4.19.3.1. Detention Basin Concerns

Conventional detention basins of standing freshwater can quickly turn into a prime breeding habitat for a variety of mosquito species; however, vegetated detention basins with a functioning constructed wetland and wet weather-associated flushing typically do not provide a prime breeding habitat for mosquitoes. The diverse and complex vegetative and wildlife habitat associated with the constructed wetland extended detention basins includes an abundance of mosquito larvae predators, which do limit the potential for exposure. One of the most populous species in Westchester County is the *Culex pipens*, a carrier of the West Nile Virus. Over the past few years the spread of the virus has raised public awareness forcing government officials to

treat its spread as a legitimate public health issue. This section examines the potential for detention basins at the Eastview Site to serve as a breeding site for these mosquitoes and any mitigation needed to prevent potential adverse impacts on public health.

4.19.3.2. Background

The West Nile Virus belongs to the *Flaviviridae* family (genus Flavivirus) and is part of the Japanese Encephalitis (JE) serocomplex.[51] JE viruses are maintained in nature by a mosquito vector and bird reservoir host. Humans and other mammals (often equine) infected with West Nile Virus are considered "dead-end" hosts, as they are not anticipated to contribute to the transmission cycle. The greatest mortality due to West Nile Virus in New York has occurred in the bird and horse population. The chance of humans becoming seriously ill from West Nile Virus is very small. This is because most people are able to overcome any infection from West Nile Virus by the normal response of their immune system.

West Nile Virus was first discovered in a woman from the West Nile District of Uganda in 1937. It has a wide geographic distribution, with outbreaks in Africa, southwest Asia and Europe. Although the exact origin of the West Nile Virus found in the U.S. remains unknown, the strain isolated from the 1999 outbreak is most closely related to that isolated in Israel in 1998 from a dead goose.[53]

Symptoms of West Nile Virus in humans generally appear within 3-14 days of infection, following a period of incubation. Symptoms are typically flu-like, with a mild fever, headache, and body ache. Skin rash and swollen lymph glands may also accompany infection. Some individuals who become infected with West Nile Virus (less than 1 percent) develop encephalitis, which is an inflammation of the brain. West Nile Virus encephalitis primarily affects persons over 50 years of age. Those with a compromised immune system may also be more susceptible to acquiring encephalitis. Symptoms of this disease develop rapidly and may include high fever, headache, muscle weakness, stiff neck, confusion, and coma. The rate of fatality is 3 to 15 percent among those who develop the disease. Currently no approved human vaccine is available for West Nile Virus.

4.19.3.2.1. Geographic Distribution

West Nile Virus has already spread throughout Northern Africa and portions of Western Europe. The climate changes and weather trends associated with global warming are suggested as a reason for the gradual expansion of this and other viral diseases across larger areas of the planet, including areas with large urban populations.

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[51] National Pesticide Information Center (NPIC) website, (http://ace.orst.edu/info/npic/wnv/virus.htm).

[53] Briese, T., Rambaut, A., Pathmajeyan, M., Bishara, J., Weinberger, M., Pitlik, S., and Lipkin, I. Phylogenetic Analysis of a Human Isolate from the 2000 Israel West Nile virus Epidemic, *Emerging Infectious Diseases.* 8 (5) 528-531. May 2002.

While West Nile Virus was first identified in the U.S. in the fall of 1999, it appears to be well established in the United States and continues to spread. As of November 20, 2002, there were 3,698 confirmed human cases of West Nile Virus, and 212 reported deaths. The virus has already been detected in 44 states and the District of Columbia.

4.19.3.2.2. Causes and Aggravating Factors

Mosquitoes commonly carry the West Nile Virus. Of the 159 types of mosquito species present in New York State, only one, *Culex pipiens* or the household mosquito, is the central focus of the emergence and spread of West Nile Virus in the United States. There are five other species of mosquitoes known to carry West Nile Virus. They include *Culex restuans, Culex salinarius, Aedes triseriatus, Aedes vexans, and Aedes japonicus.*

Any object that can hold water can become a breeding area for mosquitoes. Mosquitoes breed and lay their eggs in standing water. Standing water can be found in gutters, abandoned tires, pools, birdbaths, trashcans, window boxes, wheelbarrows, rowboats, planters, and children's toys. Weeds, tall grass, and shrubs also provide a home for mosquitoes.

Mosquitoes are most likely to bite during evening, nighttime, and early morning hours. During these hours it is important for people to be cautious of the local mosquito population. As temperatures fluctuate viruses, such as the West Nile Virus, can survive in their host organisms, or vectors, through the winter and reemerge in the spring.

4.19.3.2.3. Prevention & Management Plans

The unprecedented introduction of West Nile Virus into the metropolitan area of New York City (NYC) in the late summer of 1999 and its subsequent statewide expansion in 2000 has resulted in a large-scale review of existing programs and required resources to address the threat.[54] Representatives from local health units, state and federal agencies have been meeting on a regular basis to address each of the significant surveillance and response issues associated with the mosquito borne disease. As a result of the discussions and consultation with community groups, the New York State Department of Health (NYSDOH) has developed a set of complementary action plans designed to offer a degree of consistency in the approach taken among municipalities to minimize cases of this or other related arbovirus infections.

These management plans are based on four criteria: educating the public; destroying larval stage mosquitoes; destroying adult mosquitoes; and effective public communication. The plans include a preventative, phased-response strategy to minimize the potential re-appearance of West Nile Virus. Biological pesticides that kill mosquito eggs and larvae (larvicides) are used first, followed by a full-scale aerial spraying program for adult mosquitoes if West Nile Virus occurs in human populations again.
Many different pesticides are registered in New York State for mosquito control. Broadly, these products can be broken down into either adulticides (those products that control adult mosquitoes) or larvicides (those products that control immature forms of mosquitoes). While

[54]New York State West Nile Virus Response Plan– Guidance Document: (http://www.health.state.ny.us/nysdoh/westnile/2001/responseplan/2001wnv_responseplan.pdf).

pesticides are inherently toxic, if used properly, the potential for significant human exposure to the mosquito adulticides or larvicides is low. Hence, the risk of health effects to the general public is also low.

4.19.3.2.4. West Nile Virus Status in 2004

Table 4.19-1 shows the current numbers of dead birds, mosquito pools, and number of humans afflicted with West Nile Virus to date for 2004.

The only pool of standing water on the site is a pool at the north end of the site where storm drains from the adjacent Grasslands Reservation property discharge. The Mine Brook corridor does not provide good mosquito habitat. Mosquitoes need stagnant water to lay their eggs. Mosquitoes most commonly associated with the West Nile Virus often breed in artificial containers that hold stagnant water. They are much less frequently found in natural habitats such as salt marshes, wetlands, large bodies of water, or streams. Wildlife within such bodies of water, such as fish, frogs, turtles, or waterfowl, feeds on the mosquito larvae before the larvae are able to complete their life cycle and become adult mosquitoes.[55] Therefore, the Mine Brook corridor, being a moving stream, does not provide good mosquito breeding habitat.

TABLE 4.19-1. NEW YORK CITY & WESTCHESTER COUNTY WEST NILE VIRUS: 2004 POSITIVE RESULTS SUMMARY TO DATE

West Nile Virus Positive Results	Westchester Totals	Citywide Totals	Bronx	Brooklyn	Man-hattan	Queens	Staten Island
Dead Birds	1	37	2	1	2	13	19
Mosquito Pools	14	174	9	24	1	29	111
Human Cases	1	4	0	0	3*	0	1

Notes:
New York City information obtained on October 12, 2004 from http://www.nyc.gov/html/doh/html/wnv/wnvr1-2004.html. Westchester County information verified on October 12, 2004 from http://www.westchestergov.com/health/PHU_06_26_2003(WNV)_files/PublicHealthUpdate (WNV_1stHumanCase).htm.
Dead Birds: Dead birds are tested by the New York State Department of Environmental Conservation and the New York State Department of Health.
Mosquito Pools: Mosquitoes are collected from over 90 locations Citywide and tested by the New York City Department of Health & Mental Hygiene. New York State Department of Health tests for Westchester County.
Human Cases: Healthcare providers in New York City are required to report all patients hospitalized with viral encephalitis and meningitis to the New York City Department of Health & Mental Hygiene. Blood and spinal fluid specimens are tested for West Nile Virus by the New York City Department of Health & Mental Hygiene. In New York City, the Human cases reported were hospitalized cases of West Nile Encephalitis or Meningitis.
*Did not acquire West Nile infection in New York City; infection likely associated with travel.

[55] Information obtained on May 4, 2004, from www.westchestergov.com/health/WNV2001/WNV%20and%20your%20pond.pdf.

4.19.3.3. Potential Public Health Impacts

The first full year of operation of the proposed UV Facility would be 2010. Therefore, potential project impacts have been assessed by comparing the Future With the Project conditions against the Future Without the Project conditions for the year 2010.

In the Future With the Project, the proposed facility is not anticipated to result in any significant adverse impacts to the surface water, stormwater, or groundwater. A pretreatment forebay, along with on-line storage, would be utilized to ensure that flows are maintained to adjacent natural resource areas and Mine Brook at or near the existing rates and volumes. These areas would be diversely vegetated in order to provide a natural habitat for known vector predators for mosquito larvae. Generally, the flow-through characteristics of on-line storage does not lead to stagnant water, which is a prime mosquito breeding habitat. The absence of stagnant water coupled with the periodic flushing associated with wet weather events and the presence of potential predators minimizes the viability of mosquito larvae survival, thereby minimizing the potential for a mosquito infestation.

In addition, the advantage of utilizing the pretreatment forebay to improve the water quality would further limit the potential existence of mosquito larvae. The potential for limiting the mosquito populations within this area would aid with minimizing the spread of the West Nile Virus and other mosquito-borne diseases. In addition, the site would also comply with Westchester County's *Comprehensive Mosquito-Borne Disease Surveillance and Control Plan*. The construction of the proposed facility would take place from fall 2005 to fall 2009. During the peak year of construction, no adverse public health risks are anticipated with respect to the natural resources affecting the project site. No impacts are anticipated to the natural and surrounding structural resources from the quality and quantity of stormwater runoff due to the construction activities at the Eastview Site.

During construction, artificially created detention basins could provide a breeding habitat for mosquitoes that are capable of carrying West Nile Virus. These detention basins would be designed to not accumulate standing water and be periodically drained. The contractor would be responsible for removing any containers that could temporary store water; therefore, mosquito growth would not be increased because there would be no increase in standing water.

The Croton project is not anticipated to result in any significant adverse impacts to the surface water, stormwater, or groundwater. A stormwater detention basin would be installed to ensure that flows are maintained to adjacent natural resource areas at or near the existing rates and volumes. The Croton project detention basin was designed so that the outfall would be fitted with a low volume drain that would completely empty the pond and prevent the pooling of standing water.

4.19.4. Conclusion

The causes of asthma, its increase over the last two decades, and the triggers for its exacerbation are only partially understood. Government officials are well aware of the epidemic of childhood asthma. As reported by the New York City Department of Health and Mental Hygiene (DOHMH)[56], asthma is a common disease among children and adults. It is a leading cause of missed school among children and the most common cause of hospitalization for children 14 years and younger. Among adults, asthma causes missed work, emergency department visits, and limitation of activity.

Any impact from construction activities related to the proposed project is likely to remain local and temporary. Based on the air quality modeling of various proposed scenarios with and without the Croton project, taking into account stationary and mobile impacts from construction, the predicted concentrations of $PM_{2.5}$ at the nearest sensitive receptors would be in compliance with the interim guideline incremental concentrations. Thus, the construction of the proposed project is unlikely to have an adverse impact on public health. Air quality modeling results also show insignificant increases in the short-term and annual average concentrations of $PM_{2.5}$ from the operation of the proposed facility. Therefore, potential $PM_{2.5}$ emissions from mobile and stationary sources related to the construction and operation of the proposed facility are not anticipated to result in an adverse impact on public health.

Current and proposed regulations will substantially reduce the sulfur content of diesel fuel and will help control emissions of several pollutants from diesel equipment and vehicles. These are welcome improvements that will provide additional margins of safety to help reduce the impact of pollution on public health.

The operation of the proposed facility is not anticipated to result in any significant adverse impacts to the surface water, stormwater, or groundwater. A pretreatment forebay, along with on-line storage, would be utilized to ensure that flows are maintained to adjacent natural resource areas and Mine Brook at or near the existing rates and volumes. These areas would be diversely vegetated in order to provide a natural habitat for known vector predators of mosquito larvae. During construction, detention basins at the Eastview Site could provide a breeding habitat for mosquitoes that are capable of carrying West Nile Virus. However, these detention basins would be designed to prevent the accumulation of standing water and to be periodically drained. During construction, the contractor would be responsible for removing any containers that could temporary store water; therefore, mosquito growth would not be increased because there would be no increase in standing water.

[56] on NYCDOH website (www.nyc.gov/html/doh).