CHAPTER 33 PUBLIC HEALTH EVALUATION

33.1 Introduction

The potential impacts of the Proposed Plan Facilities on public health are evaluated in this chapter. The chief public health concerns are (1) potential health effects (including asthma) of air pollutants released by the Proposed Plan Facilities; (2) effects of noise related to the Proposed Plan Facilities; (3) effects of odors related to the Proposed Plan Facilities; and (4) the potential for vermin (such as rats and insects) to infest areas near Proposed Plan Facility sites.

33.2 Air Pollution

33.2.1 Air Pollutants of Concern

Project-related air pollutants of two kinds have been directly assessed in this DEIS. One set of pollutants, called criteria pollutants, includes compounds for which NAAQS have been established by the USEPA: CO, NO_2, SO_2 and PM. Two overlapping categories of PM are often measured or modeled in environmental health science, namely fine PM ($PM_{2.5}$) and coarse PM (PM_{10}). Much of the PM associated with the Proposed Plan Facilities would be in the form of diesel particulate matter (DPM). Airborne concentrations of the other set of pollutants, termed hazardous air pollutants (HAPs), are not limited nationally. The HAPs evaluated in this DEIS include benzene, formaldehyde, 1,3-butadience, benzo(a)pyrene and other chemicals emitted from diesel fuel and/or exhaust.

33.2.2 Health Effects of Air Pollutants of Concern

33.2.2.1 ***Carbon Monoxide***

CO is a colorless, odorless gas released during combustion of many substances, including gasoline, diesel fuel and home heating oil. CO is deadly at high concentrations in air; hence, the need for CO detectors in homes where malfunctioning furnaces or boilers may cause a build up of the gas. At lower concentrations, CO causes fatigue and confusion. CO exerts toxicity by binding to the blood's hemoglobin, thereby creating carboxyhemoglobin (COHb) and displacing oxygen. COHb is a very stable molecule and, thus, the body's tissues become starved for oxygen when COHb levels accumulate. For example, a COHb concentration of 65% or more may be lethal, a concentration of 30% may cause severe headache and a concentration of 10% may cause slight headache and fatigue.[1] USEPA's review of CO toxicity at ambient concentrations determined that the most sensitive effects of exposure are on the cardiovascular system in persons with pre-existing heart disease, namely a quicker onset of angina and

[1] Clayton, G. and Clayton, F., editors. *Patty's Industrial Hygiene and Toxicology, fourth edition, Volume II*. John Wiley and Sons: New York, NY. 1994.

electrocardiogram (EKG) changes.[2] The NAAQS for CO (9 ppm for an 8-hour average and 35 ppm for a 1-hour average) are set to keep COHb levels in the blood low enough to reduce the risk of these cardiovascular effects. The City is in attainment for the CO air quality standards.

33.2.2.2 ***Nitrogen Dioxide***

NO_2 is one of several related oxides of nitrogen, collectively termed "NO_X," found in ambient air. USEPA decided to issue a NAAQS only for NO_2, however, as it is found at the highest concentrations. NO_2 is an irritant gas, and it is regulated in air based on its potential effects on respiratory health of children (who might be made more vulnerable to respiratory illnesses) and on pulmonary function in asthmatics and persons with chronic obstructive pulmonary disease.[3] Long-term exposure to NO_2 at much higher concentrations than are found in ambient air has produced emphysema-like changes in laboratory rodents. As of 1996, when the USEPA last reviewed the NAAQS for NO_2, (53 ppb as an annual average), the entire country was in compliance with the standard.[4]

33.2.2.3 ***Sulfur Dioxide***

Analogous to NO_2, SO_2 is one of several oxides of sulfur, collectively termed SO_X, and is the one most present in ambient air. It is created primarily by combustion of fossil fuels and processing of ores. Air quality standards for SO_2 are intended to protect against possible mortality, aggravation of bronchitis, decreased lung function in asthmatics and/or children, and reduced capacity to respond to respiratory infections.[5] The standards are 30 ppb as an annual average and 140 ppb as a 24-hour average. The City is in compliance with the SO_2 NAAQS.

[2] USEPA (1994). "National Ambient Air Quality Standards for Carbon Monoxide – Final Decision." *Federal Register*: August 1.
[3] USEPA (1995). "National Ambient Air Quality Standards for Nitrogen Dioxide: Proposed Decision." *Federal Register*: October 11.
[4] USEPA (1996). "National Ambient Air Quality Standards for Nitrogen Dioxide: Final Decision." *Federal Register*: October 8.

33.2.2.4 ***Particulate Matter***

33.2.2.4.1 PM_{10} and $PM_{2.5}$

Unlike the other criteria pollutants, which are specific chemical molecules, PM refers to any of thousands of different solid particles or liquid droplets suspended in outdoor air. Various forms of airborne PM differ with respect to (1) size (with diameters ranging from about 0.001 to 100 microns [µm]), shape and surface characteristics; (2) water solubility and pulmonary persistence; (3) chemical composition, pH, and metal content; and (4) biologic and immunologic properties and potencies. 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 units of micrograms per cubic meter, $\mu g/m^3$). Thus, PM_{10} refers to all particles and aerosols with diameters less than 10 µm, and $PM_{2.5}$ to all particles with diameters less than 2.5 µm.

In practice, $PM_{2.5}$ and PM_{10} are defined as all material collected and weighed using specific types of equipment and under specified conditions.[6] When samples of ambient air are collected and analyzed for purposes of NAAQS compliance, the specific physical, chemical and biological forms of PM are not determined.

Many observational, epidemiologic studies have reported weakly positive statistical associations between rates of mortality or morbidity in populations and moderate concentrations of total $PM_{2.5}$ and PM_{10} measured in ambient air near those populations.[7] These observational studies include cross-sectional studies[8] in which mortality in various metropolitan areas is associated

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[5] USEPA (1988). "Proposed Decision not to Revise the National Ambient Air Quality Standards for Sulfur Oxides (Sulfur Dioxide)." *Federal Register*: April 26; USEPA (1996). "National Ambient Air Quality Standards for Sulfur Oxides (Sulfur Dioxide) – Final Decision." *Federal Register*: May 22.

[6] USEPA (1997). "National Ambient Air Quality Standards (NAAQS) for Particulate Matter; Final Rule." *Federal Register*: July 18.

[7] See Krewski, D., Burnett, R., Goldberg, M., *et al*. (2000). "Reanalysis of the Harvard Six Cities Study and the American Cancer Society study of Particulate Air Pollution and Mortality." Health Effects Institute: Cambridge, MA; and Lipfert, F. and Wyzga, R. (1995). "Air Pollution and Mortality: Issues and Uncertainties." J. Air Waste Manage. Assoc. 45:949-966 for reviews.

[8] Dockery, D., Pope, C., Xy, X., *et al*. (1993). "An Association between Air Pollution and Mortality in Six U.S. Cities." N. Engl. J. Med. 329:1753-1759; Pope, C., Thun, M., Namboodiri, N., *et al*. (1995). "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults." Am. J. Respir. Crit. Care Med. 151:669-674; Pope, C., Burnett, R., Thun, M., *et al*. (2002). "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution." JAMA 287(9):1132-41.

with ambient concentrations of PM in those areas, and time-series studies[9] in which daily mortality within a metropolitan area is associated with concurrent or lagged daily fluctuations in ambient PM concentrations. Similarly, some 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.[10] USEPA[11] stated that these statistical associations reflect cause and effect, and has established the PM NAAQS primarily on the basis of the associations.

For purposes of public health assessment, however, it is important to recognize that different forms of PM may pose markedly different risks to health. Airborne PM includes countless naturally occurring materials, such as thousands of species of viruses and bacteria, various molds and pollen fragments (from thousands of species of flowering plants), fragments of innumerable species of insects and bits of different types of sand and soil. Clearly, small concentrations of some forms of natural PM, such as tuberculosis bacillus, can be deadly, while other forms, such as suspended sea salt, are benign.

Pollution-derived PM is also a complicated mixture. Standard characterizations separate such PM into five categories – sulfates, nitrates, organic compounds, elemental carbon and "other."[12] Such characterizations belie substantial, underlying heterogeneity, however. For example, members of the "organic compounds" class of PM number in the thousands and are quite diverse in their structures and expected toxicities. Even members of a category as seemingly simple as the first (sulfates) differ in important features. Thus, most ambient sulfates (such as ammonium sulfate and sodium sulfate) are water-soluble, but a few (such as calcium sulfate) are not. The

[9] Samet, J., Dominici, F., Curriero, F., *et al*. (2000). "Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 1987-1994." New Engl. J. Med. 343:1742-1749; Dominici, F., McDermott, A., Zeger, S., and Samet, J. (2003). "Airborne Particulate Matter and Mortality: Timescale Effects in Four U.S. Cities." Am. J. Epidemiol. 157(12):1055-1065.

[10] CEPA/FPAC Working Group on Air Quality Objectives and Guidelines. National Ambient Air Quality Objectives for Particulate Matter. Part 1: Science Assessment Document. Environmental Health Directorate, Canada. 1999.

[11] USEPA (1996). Air Quality Criteria for Particulate Matter (Vols. I, II, & III). EPA/600/P-95/001af. Washington, DC: Office of Research and Development. [http://www.epa.gov/ncea/archive/pdfs/ partmatt/vol1/0671v1fm.pdf]; USEPA (1997). "National Ambient Air Quality Standards for Particulate Matter, Final Rule." *Federal Register*: July 18; EPA (2003). Air Quality Criteria for Particulate Matter, Fourth External Review Draft. EPA/600/P-99/002aD June 2003.

[12] USEPA (1996).

solubility or insolubility of aerosols and particles is expected to be an important determinant of toxicity, as it is for airborne fibers.[13] Solubility aside, sulfate salts range widely in their effects on respiratory function and structure.[14]

Most of the PM emitted by the activities related to the Proposed Plan Facilities would be from diesel engine exhaust and, hence, in the form of DPM. This mixture of gases and particles has been unusually well-studied.

33.2.2.4.2 DPM

DPM consists primarily of soot (carbon particles) within which various organic compounds are absorbed.[15] Diesel particles are generally small enough to be counted as $PM_{2.5}$ and are emitted by diesel engines of all kinds, although different engines, loads, specific fuels and other factors result in DPM mixtures with varying chemical constituents. DPM is not a criteria pollutant, so there are no NAAQS for it, nor is it generally considered an HAP. Therefore, DPM impacts have not been quantitatively assessed in this DEIS except as a component of $PM_{2.5}$.

The toxic effects of diesel engine exhaust – both DPM and the gases and vapors that comprise the bulk of the exhaust – have been evaluated in numerous acute and chronic studies. Laboratory animals are believed to be good models for humans with regard to their responses to DPM,[16] and some 17 chronic studies involving laboratory rats, mice, hamsters, guinea pigs, cats and monkeys have evaluated the respiratory and systemic effects of exposure to DPM.[17] Chronic exposures to large concentrations of DPM (in the presence of diesel engine exhaust gases) cause inflammation, fibrosis and functional changes in the respiratory system, and very large

[13] McConnell, E. (2000). "A Science-Based Paradigm for the Classification of Synthetic Vitreous Fibers." Regul. Toxicol. Pharmacol. 32:14-21.
[14] Amdur, M.O. "Air Pollutants" in Casarett and Doull's Toxicology, 3rd edition, Macmillan Publishing Co.: New York, NY. 1986.
[15] This discussion of DPM is taken, with the permission of the authors, from Green, L. and Armstrong, S. (2003). "Particulate Matter in Ambient Air and Mortality: Toxicologic Perspectives." Regul. Toxicol. Pharmacol. 38:326-335.
[16] International Life Sciences Institute (ILSI) (2000). "ILSI Risk Science Institute Workshop: The Relevance of the Rat Lung Response to Particle Overload for Human Risk Assessment." Inhal. Toxicol. 12(1-2):1-17; USEPA (2002). Health Assessment Document for Diesel Engine Exhaust. EPA/600/8-90/057F.
[17] USEPA (2002); USEPA (2003a). IRIS record for diesel engine exhaust. Available at www.epa.gov/iris/subst/0642.htm.

concentrations cause premature death. The lowest observed adverse effect levels (LOAELs) and no observed adverse effect levels (NOAELs) for these effects are considerably in excess of ambient concentrations. Thus, the experimentally-derived LOAELs for pulmonary changes in rats are in the range of 800 to 3,000 µg DPM/m^3, while the levels at which these effects are not observed – that is, the NOAELs – range from about 100 to 500 µg DPM/m^3.[18] With regard to premature mortality due to lifetime exposure to DPM, the LOAELs are about 6,000 µg DPM/m^3 in rats[19] and 4,000 µg DPM/m^3 in mice[20], although other rodents tested in other laboratories showed no decreased survival even given lifetime exposures of some 7,000 µg DPM/m^3.[21] For purposes of public health assessment, application of typical safety factors to these data from laboratory rodents suggests that current ambient concentrations of diesel engine exhaust in New York State are not harmful. Nonetheless, current and proposed regulations that will substantially reduce the sulfur content of diesel fuel and will substantially control emissions of several pollutants from diesel equipment and vehicles are welcome improvements that will provide additional margins of safety and help to reduce regional haze.

Laboratory rats, though not necessarily other test species, develop lung tumors during lifetime exposures to very high concentrations of DPM. As noted by USEPA[22], the mechanism by which these tumors arise involves "particle overload and consequent persistent inflammation and cell proliferation, [which] supports a nonlinear mode of action for lung cancer in the rat (ILSI, 2000). The nonlinear cancer response is further characterized as occurring at relatively high exposures of diesel exhaust (>3500 µg DPM/m^3), which is far beyond the range of environmental levels. The rat tumor occurrences, thus, are not particularly influential in judging the hazards at

[18] USEPA (2003a). (See footnote 17.)

[19] Nikula, K., Snipes, M., Barr, E., *et al*. (1995). "Comparative Pulmonary Toxicities and Carcinogenicities of Chronically Inhaled Diesel Exhaust and Carbon Black in F344 rats." Fundam. Appl. Toxicol. 25:80-94.

[20] Heinrich, U., Muhle, H., Takenaka, S., *et al*. (1986). "Chronic Effects on the Respiratory Tract of Hamsters, Mice, and Rats After Long-Term Inhalation of High Concentrations of Filtered and Unfiltered Diesel Engine Emissions." J. Appl. Toxicol. 6:383-395.

[21] Mauderly, J., Benson, J., Rice, D., *et al*. (1984). "Life Span Study of Rodents Inhaling Diesel Exhaust: Effects on Body Weight and Survival" in: Guilmette, R., Medinsky, M., editors. Inhalation Toxicology Research Institute Annual Report. ITRI:Albuquerque, NM, pp. 287-291; Mauderly, J., Bice, D., Carpenter, R., *et al*. (1987). Effects of Inhaled Nitrogen Dioxide and Diesel Exhaust on Developing Lung. Health Effects Institute, Report No. 8, Cambridge, MA; Mauderly, J., Banas, D., Griffith, W., *et al*. (1996). "Diesel Exhaust is not a Pulmonary Carcinogen in CD-1 Mice Exposed Under Conditions Carcinogenic to F344 Rats." Fundam. Appl. Toxicol. 30:233-242; Heinrich, U., Muhle, H., Takenaka, S., *et al*. (1986). "Chronic Effects on the Respiratory Tract of Hamsters, Mice, and Rats After Long-Term Inhalation of High Concentrations of Filtered and Unfiltered Diesel Engine Emissions." J. Appl. Toxicol. 6:383-395; all as reviewed in USEPA (2002).

environmental levels of exposure." USEPA also notes that "while the weight of evidence indicates that DE [diesel engine exhaust] has the potential to pose a lung cancer hazard to humans at anticipated levels of environmental exposure, as shown by occupational epidemiology studies, a confident dose-response relationship based on occupational exposure levels is currently lacking." The National Toxicology Program classifies DPM as "reasonably anticipated to be a human carcinogen," but notes that the increased risk of lung cancer seen in epidemiologic studies of workers "cannot always be clearly ascribed to diesel exhaust exposure . . . [and] most studies used inadequate measures of exposure."[23]

Current ambient concentrations of DPM typically average about 1 to 10 μg DPM/m^3. The Proposed Plan Facilities analyzed would add less than 1 μg DPM/m^3, measured as $PM_{2.5}$, on an annual basis (see Section 33.2.3).

33.2.2.5 HAPs

Acetaldehyde, a widely used industrial chemical, is a component of vehicle exhausts and tobacco smoke, and is a metabolite of ethanol. That is, everyone who consumes alcohol generates acetaldehyde. At high concentrations of vapor, acetaldehyde is irritating to the respiratory tract. In animal studies, the most sensitive indicator of acetaldehyde toxicity is damage to the olfactory epithelium (i.e., part of the lining of the nasal passages). At higher exposure levels, animals also lost weight, showed signs of irritation and showed altered activity of certain immune cells in the lungs. USEPA considers acetaldehyde a "B2," or "probable" human carcinogen. No reliable human data indicate that acetaldehyde is carcinogenic, but animals forced to breathe acetaldehyde for at least one year developed nasal and laryngeal tumors. For non-cancer effects, USEPA has derived a concentration of acetaldehyde of 9 μg/m^3 (16 ppb) as a safe, long-term limit for the general population.[24] Recommended occupational limits for the workplace are 14 mg/m^3 (25 ppm) or less, as a ceiling concentration.[25]

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[22] USEPA (2003a).
[23] National Toxicology Program (2002). 10th Report on Carcinogens. U.S. Department of Health and Human Services.
[24] USEPA (2003b). IRIS record for acetaldehyde. Available at www.epa.gov/iris/subst/0290.htm.
[25] American Conference of Governmental Industrial Hygienists (ACGIH). Documentation of the Threshold Limit Values and Biological Exposure Indices, seventh edition and supplement. ACGIH: Cincinnati, OH. 2002; National

Acrolein is a flammable, volatile liquid. It is emitted in vapor form in fuel exhaust and cigarette smoke, and is found in very low levels in some foods and beverages. Because acrolein is chemically reactive, its toxicity in experimental animal studies has usually been exerted at the point of entry, for example, in the nasal passages and respiratory tract if exposure is by inhalation, or in the stomach if exposure is oral. Severity of lesions at these sites depends on the dose of acrolein. At the higher concentrations tested in such studies, acrolein increased the mortality rate of animals. USEPA does not consider acrolein a carcinogen because there is no useful human data on the subject, and cancer studies in animals have been deficient in design and/or conduct. USEPA has derived a concentration of acrolein of 0.02 $\mu g/m^3$ (0.008 ppb) as a safe, long-term limit for the general population.[26] The recommended occupational limit for the workplace is 0.25 mg/m^3 (0.1 ppm) as a ceiling concentration according to one group and an 8-hour average concentration according to another.[27]

Benzene, a volatile organic compound present in fuels and emitted in fuel exhaust, is one of the two known human carcinogens included in the HAPs analysis. Benzene is ubiquitous in outdoor and indoor air, occurs in cigarette smoke and is used in many heavy and light industrial processes. The damaging effects of exposure to high airborne concentrations of benzene have been known for about a century — high exposures cause severe blood diseases, such as aplastic anemia, and can be rapidly fatal. Lower airborne concentrations in workplaces (still much higher than those in outdoor air) have caused many cases of leukemia, principally acute myelogenous leukemia (AML).[28] The maximum allowable concentration of benzene in workplace air is now 1 ppm to guard against cancer risk and the risk of non-cancer blood diseases. There is no demonstrable adverse effect of ppb levels (or less) of benzene in air, such as occurs around the U.S., although cancer remains a theoretical concern.

The gas 1,3-butadiene is manufactured for various uses, including production of styrene-butadiene rubber. It is also present in vehicle exhausts. In long-term animal studies, the most sensitive indicator of 1,3-butadiene toxicity was atrophy of ovaries in female mice exposed

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Institute of Occupational Safety and Health (NIOSH). Pocket Guide to Chemical Hazards. U.S. Department of Health and Human Services. 2003.

[26] USEPA (2003c). IRIS record for acrolein. Available at www.epa.gov/iris/subst/0364.htm.

[27] ACGIH (2002); NIOSH (2003).

by inhalation to 6.25 ppm for up to two years. Testicular atrophy occurred in male mice exposed to even higher concentrations of 1,3-butadiene. In reproductive studies, the most sensitive indicator of 1,3-butadiene toxicity was decreased weight of mouse fetuses in dams exposed by inhalation during pregnancy. USEPA considers 1,3-butadiene to be carcinogenic to humans by inhalation based both on animal studies (in which various tumor types were produced) and on epidemiologic studies of workers in industries producing or using the chemical. The epidemiologic studies have found increased rates of lymphohematopoietic cancers (cancers of the blood and lymph systems).[29] For non-cancer effects, USEPA has derived a concentration of 1,3-butadiene of 2 $\mu g/m^3$ (0.9 ppb) as a safe, long-term limit for the general population, even for a lifetime. Recommended occupational limits in workplaces are 2 ppm (4.4 mg/m^3) or less averaged over eight hours.[30]

The toxicity of formaldehyde is qualitatively similar to that of a related chemical, acetaldehyde (discussed above). An irritating vapor, formaldehyde causes similar irritative symptoms and lesions in the respiratory tract.[31] It is considered by USEPA to be more likely than acetaldehyde to be a human carcinogen ("B1" rather than "B2") on the basis of several epidemiologic studies. Increased incidences of or death from lung, nasopharyngeal and buccal tumors in people with occupational exposure to formaldehyde were observed in some of these studies. Overall, however, USEPA characterizes the human evidence as "limited." Squamous cell carcinomas of the nasal cavities occurred in animals inhaling formaldehyde in several long-term experiments.[32] Recommended limits for formaldehyde exposure in workplaces are 0.3 ppm or less, as a ceiling concentration.[33]

Several of the HAPs evaluated in this DEIS – benzo(a)pyrene, anthracene, benzo(a)anthracene, chrysene, dibenz(a,h)anthracene, phenanthrene and pyrene – are collectively known as polycyclic aromatic hydrocarbons (PAHs). PAHs are typically produced through combustion, whether of diesel fuel, wood, cigarettes or other organic matter. While no single PAH has been

[28] Graham, J., Green, L., and Roberts. M. In Search of Safety. Harvard University Press: Cambridge, MA. 1988.
[29] USEPA (2003d). IRIS record for 1,3-butadiene. Available at www.epa.gov/iris/subst/0139.htm.
[30] ACGIH (2002); NIOSH (2003).
[31] International Programme on Chemical Safety (IPCS). Environmental Health Criteria 89: Formaldehyde. World Health Organization: Geneva, Switzerland. 1989.
[32] USEPA (2003e). IRIS record for formaldehyde. Available at www.epa.gov/iris/subst/0419.htm.

shown to cause cancer in humans, they are collectively suspect because of their presence in cigarette smoke, soot and other materials known to be carcinogenic to people. In addition, some specific PAHs can cause cancer in laboratory animals. According to USEPA's traditional classification of chemicals in terms of carcinogenicity, benzo(a)pyrene, benzo(a)anthracene, chrysene and dibenz(a,h)anthracene are termed "B2" or "probable" human carcinogens, and anthracene, phenanthrene and pyrene are "D" or "unclassifiable."[34] However, NYSDEC or USEPA have made an estimate of cancer risk for only one of the PAHs evaluated in this DEIS, that being benzo(a)pyrene.

Naphthalene is an odorous solid chemical used in certain types of mothballs and toilet bowl cleaners, and is emitted in fuel exhaust. Humans exposed to naphthalene vapors have developed headache, nausea, loss of appetite, acute hemolysis and lens opacities.[35] In animal studies, the more serious of these effects have not been observed following vapor exposure. Rather, in laboratory rodents, naphthalene vapors produce inflammation and regeneration in the respiratory epithelium. Unlike similar effects produced by acetaldehyde and formaldehyde, naphthalene's toxicity is probably not due to direct chemical attack on tissues; rather, it appears to require metabolism. There is no information about a risk of cancer in humans from naphthalene exposure, and USEPA states that the carcinogenic potential "cannot be determined" at this time. USEPA has derived a concentration of naphthalene of 3 $\mu g/m^3$ (0.6 ppb) as a safe, long-term limit for the general population.[36] The recommended occupational limit in the workplace is 50 mg/m^3 (10 ppm), averaged over eight hours.[37]

Propylene, a gas, is present in vehicle exhausts. At high concentrations (more than 20,000 ppm), propylene is explosive. Otherwise, propylene has no significant toxicity except as a simple asphyxiant when it is so concentrated that it reduces the amount of oxygen available for inhalation.

[33] ACGIH (2002); NIOSH (2003).
[34] USEPA (2003f). IRIS record for benzo(a)pyrene. Available at www.epa.gov/iris/subst/0136.htm.
[35] ACGIH (2003).
[36] USEPA (2003g). IRIS record for naphthalene. Available at www.epa.gov/iris/subst/0436.htm.
[37] ACGIH (2002); NIOSH (2003).

Toluene is a common solvent used in many industries and found in many consumer products, as well as in fuels and exhaust. Inhalation of large concentrations of toluene vapors is well known to affect the human central nervous system. In the case of people who abusively inhale toluene-containing products and are exposed to thousands of ppm vapor, significant brain damage has occurred. However, at lower levels found in many occupational settings, effects are either subtle (revealed by neuropsychologic testing) or transient (such as headache, dizziness and irritation). Abusive inhalation of toluene vapors has also been linked to birth defects. Toluene is not classified as a carcinogen (known, probable or possible) by USEPA. USEPA has derived a concentration of toluene of 400 $\mu g/m^3$ (105 ppb) as a safe, long-term limit for the general population.[38] Recommended occupational limits in the workplace range from 190 to 375 mg/m^3 (50 to 100 ppm) averaged over eight hours.[39]

Xylene is an aromatic solvent related to benzene and toluene and, like them, is found in fuels, fuel exhausts, and various commercial and industrial products. High concentrations of xylene in air (200 ppm or more) can cause watering eyes, sore throat, headache and mild nausea. In several studies of xylene exposure in rats, neurological effects were found to be the most sensitive indicator of possible toxicity. Rats repeatedly exposed to xylene vapors made fewer spontaneous movements than did unexposed animals, were unable to maintain their footing on rotating rods and performed worse in mazes. Xylene caused developmental toxicity in offspring of rats and rabbits exposed to vapors during pregnancy, but only at concentrations higher than those that cause the neurological effects. Xylene is not considered a carcinogen. USEPA has derived a concentration of xylene of 100 $\mu g/m^3$ (23 ppb) as a safe, long-term limit for the general population.[40] The recommended occupational limit in workplaces is 435 mg/m^3 (100 ppm), averaged over eight hours.[41]

[38] USEPA (2003h). IRIS record for toluene. Available at www.epa.gov/iris/subst/0118.htm.
[39] ACGIH (2002); NIOSH (2003).
[40] USEPA (2003i). IRIS record for xylenes. Available at www.epa.gov/iris/subst/0270.htm.
[41] ACGIH (2002); NIOSH (2003).

33.2.3 Public Health Assessment of Air Pollutants of Concern

In this DEIS, the potential health impacts of the two sets of pollutants – criteria pollutants and HAPs – are assessed differently. In addition to having established NAAQS for the criteria air pollutants, USEPA has set significant impact levels (SILs) for CO, NO_2, SO_2 and $PM_{10.}$ For these criteria pollutants, estimated Proposed Plan Facility-related increases in concentrations can be added to the existing (background) concentrations and the total compared to the NAAQS (see table entitled "Highest Estimated Concentrations of the Criteria Pollutants from On-Site Emission" of Chapters 4 through 16). The regulatory program for $PM_{2.5}$ is still under development and SILs have not yet been set. NYCDEP has proposed a draft policy for the assessment of $PM_{2.5}$ impacts; however, and NYSDEC has issued a final policy. In particular, the draft NYSDEC policy is that a "project . . .[with $PM_{2.5}$] air quality impacts equal to or less than two percent of the annual NAAQS standard of 15 $\mu g/m^3$ (0.3 $\mu g/m^3$) and equal to or less than 5 $\mu g/m^3$ on a 24-hour basis, would be considered to have insignificant impacts."[42] Similarly, NYCDEP's interim guidance value indicates that facility-related impacts on the nearest neighborhood no larger than 0.1 $\mu g/m^3$ assessed annually are to be considered insignificant. These all are referred to as interim screening threshold values (STVs) that are to be used as screening thresholds in impact evaluations until USEPA establishes national SILs. For a HAPs analysis, estimated increases are compared to benchmark concentrations established by NYSDEC that protect against cancer and/or non-cancer health risks.

33.2.3.1 Criteria Pollutants

Section 3.17 describes the air quality modeling methodologies used to estimate increases in airborne concentrations of criteria pollutants stemming from on-site activities (e.g., operation of heavy machinery) and off-site activities (e.g., emissions from truck traffic at critical intersections). For on-site analyses, sources of criteria pollutant emissions, such as collection vehicles, front-end loaders and tugboats, were catalogued for each Proposed Plan facility. The emission rates of criteria pollutants from each on-site source were combined in a USEPA-approved computer model with meteorological data (e.g., describing wind speed and wind directions) and maps of local land use to predict the increase in ambient pollutant

[42] Assessing and Mitigating Impacts of Fine Particulate Matter Emissions" undated. NYSDEC's Commissioner's Policy Proposed Draft available at: http://www.dec.state.ny.us/website/dar/drpm25.html.

concentrations at various off-site locations, called receptors. The time scale over which pollutant increments were calculated was dictated by the NAAQS for each pollutant. For example, 1-hour and 8-hour periods were evaluated for CO, while 24-hour and year-long periods were considered for PM_{10}.

The receptors with the greatest estimated pollutant increments due to on-site activities were then identified for each Proposed Plan Facility. Except for $PM_{2.5}$, these maximum increments were added to the existing (background) concentrations of criteria pollutants and the totals were compared to the appropriate NAAQS. The $PM_{2.5}$ increments were compared to the draft NYCDEP and final NYSDEC interim STVs. The summary tables of on-site results for each Proposed Plan Facility analyzed are presented in the table entitled "Highest Estimated Concentrations of the Criteria Pollutants from On-Site Emission" in Chapters 4 through 16, , as applicable.

As the tables demonstrate, in no case do the predicted total concentrations of criteria pollutants from on-site operations at the Proposed Plan Facilities analyzed exceed the NAAQS (for CO, NO_2, SO_2 and PM_{10}), and in no case do the maximum increments for $PM_{2.5}$ exceed the NYCDEP and NYSDEC screening thresholds proposed or established as the interim STVs. In the case of CO, NO_2, SO_2 and PM_{10}, the total worst-case pollutant concentrations due to on-site activities are well below all health-based limits. Because the USEPA establishes these limits with an ample margin of safety, no adverse effects are expected from on-site emissions of CO, NO_2, SO_2 and PM_{10}. In the case of $PM_{2.5}$, the increments are deemed acceptably small, according to the draft or final policies of NYCDEP and NYSDEC.

Similar analyses were performed for traffic-generated criteria pollutants at critical intersections, focusing on CO, PM_{10} and $PM_{2.5}$. The summary tables of off-site results for each Proposed Plan Facility analyzed are presented in the table entitled, "Maximum Estimated Pollutant Concentrations Near Selected Roadway Intersections" in Chapters 4 through 16, as applicable. Air quality impacts were evaluated for at least two intersections near each Proposed Plan Facility analyzed. In all instances, the anticipated total concentrations of CO and PM_{10} (traffic plus background) at these intersections are less than the applicable NAAQS. For all the Proposed

Plan Facilities analyzed, then, no adverse health consequences are expected from traffic-related CO or PM_{10} emissions, because the expected concentrations are below the health-based limits.

For the $PM_{2.5}$ analyses, the incremental concentrations contributed by traffic related to the Proposed Plan Facilities were modeled, but not added to existing background levels. Rather, the increments were compared to the STVs described above. At all critical intersections, incremental concentrations of $PM_{2.5}$ are less than or equal to the STV values. In addition, the $PM_{2.5}$ contributions at these same intersections from the on-site operations of the Proposed Plan Facilities analyzed were evaluated,. On-site operations from the Proposed Plan Facilities analyzed are not expected to cause exceedances of $PM_{2.5}$ STVs at any intersection. Furthermore, the combined $PM_{2.5}$ impacts at critical intersections from traffic and Proposed Plan Facility operations analyzed also do not exceed the STVs.

33.2.3.2 HAPs

The method used to estimate increases in airborne concentrations of HAPs is similar to that used to assess the impacts of on-site operations on off-site concentrations of criteria pollutants, except that emissions rates of the various HAPs from the different types of equipment were evaluated. In addition, only two time periods were used, a 1-hour average and an annual average. Then, where toxicologic benchmarks have been established by NYSDEC, they were compared to the predicted increases in HAPs concentrations over the 1-hour and annual averaging periods. NYSDEC's method of developing these benchmark concentrations ensures that the most health-protective values are selected from the range of possibilities, including the USEPA reference concentrations noted in the HAPs discussions above, and takes into account the carcinogenic and non-carcinogenic effects discussed in Section 33.2.2.5 above.[43] Note that cancer risk is calculated for both known and probable human carcinogens. The NYSDEC toxicity benchmarks are presented in Table 33.2-1.

[43] New York State Department of Environmental Conservation. (2000). DAR-1 AGC/SGC Tables. Division of Air Resources, Bureau of Stationary Sources.

Table 33.2-1
NYSDEC Toxicity Benchmarks For HAPs

HAP	NYSDEC Short-Term (1-hr) Guideline Concentration ($\mu g/m^3$)	NYSDEC Long-Term (Annual) Guideline Concentration ($\mu g/m^3$)	NYSDEC Unit Cancer Risk ($m^3/\mu g$)
Known or probable carcinogens			
Acetaldehyde	4,500	0.450	2.2×10^{-6}
Benzene	1,300	0.130	8.3×10^{-6}
Benzo(a)pyrene	NA	0.002	1.7×10^{-3}
1,3-Butadiene	NA	0.0036	2.8×10^{-4}
Formaldehyde	30	0.060	1.3×10^{-5}
Non-carcinogens			
Propylene	NA	3000	NA
Acrolein	0.19	0.020	NA
Anthracene	NA	0.020	NA
Benzo(a)anthracene	NA	0.020	NA
Chrysene	NA	0.020	NA
Dibenz(a,h)anthracene	NA	0.020	NA
Naphthalene	7,900	3.0	NA
Phenanthrene	NA	0.020	NA
Pyrene	NA	0.020	NA
Toluene	37,000	400	NA
Xylene	4,300	700	NA

Notes:

NA = Not applicable.

At all Proposed Plan Facilities analyzed, all 1-hour increments of HAPs are substantially below corresponding NYSDEC SGCs; similarly, all annual-average increments are substantially below AGCs. More importantly, in using the hazard index approach to sum the impacts of all HAPs, this study finds that the total hazard index at each Proposed Plan Facility is acceptable (less than 1.0) for non-cancer effects over both short-term and annual periods. (The site-specific HAPs results are presented in the table entitled "Highest Estimated Concentrations for the Criteria Pollutants from On-Site Emissions" in Chapters 4 through 16, as applicable.) In addition, the total (multi-pollutant) increase in estimated cancer risk (which assumes 70 years of continuous exposure) from exposure to carcinogenic HAPs is below the allowable limit at each Proposed Plan Facility. This HAPs analysis indicates, thus, that emissions of the chemicals studied from the on-site operations at all Proposed Plan Facilities analyzed are very unlikely to adversely affect health.

33.3 Public Health Evaluation of Noise

The major health concern posed by noise is hearing impairment, which can develop, usually over many years, following either continuous loud noise or brief exposures to extremely loud noise. (There are other causes of hearing impairment and loss, of course, such as injury, congenital defect and age.) Loudness of noise is measured in units called A-weighted decibels, or dBA, and the noise analysis methodology described in Section 3.19 quantifies facility-related noise in this unit. Long-term exposure to noise averaging 70 dBA or less is not thought to pose a risk of hearing impairment.[44]

Other health conditions that have been researched in relation to noise exposure include hypertension, heart disease, exacerbation of mental disorders and impairment of performance on cognitive tasks, such as reading and problem solving. Other adverse effects of noise include sleep disturbance, annoyance and inhibition of spoken communication. Production of such effects by noise is highly dependent on the individual and in some cases is not well understood. Adaptation to noise, even loud noise, can often occur. An international group of reviewers assessed as "sufficient" the evidence of a connection between noise and hearing impairment, hypertension, ischemic heart disease, annoyance and sleep disturbance.[45] The reviewers concluded that risk of hearing impairment, hypertension and ischemic heart disease was increased at average noise levels of 70 dBA or more.

The chief criterion by which noise impacts were assessed near the Proposed Plan Facility sites analyzed was whether on-site operations at peak levels were likely to increase the total noise at near-by receptors by 3 dBA or more during what is otherwise the quietest hour of the day or night. This procedure determines the greatest (i.e., most noticeable) impact on noise levels At one Proposed Plan Facility, Hamilton Avenue Converted MTS, no receptors were located within or near the 55 dBA contour of facility related noise, so no further assessment was required. At three of the Proposed Plan Facilities, Southwest Brooklyn Converted MTS, East 91st Street Converted MTS, and North Shore Converted MTS an on-site noise analysis was performed and

[44] Berglund, B., Lindvall, T., and Schwela, D., editors. Guidelines for Community Noise. World Health Organization: Geneva, Switzerland. 1999.

incremental noise was estimated to be below the 3 dBA limit. At none of the Proposed Plan Facilities was the average noise level, including the contribution from on-site operations, likely to exceed Zoning Code Performance Standards at the facility boundary. At none of the Proposed Plan Facilities was the average noise level, including the contribution from on-site operations, likely to exceed the 70 dBA at the facility boundary. With mitigation measures in place, on-site operations would contribute only minor increases in overall noise levels. Therefore, no adverse health impact from on-site noise from the Proposed Plan Facilities is anticipated.

The potential for adverse levels of noise due to facility-related traffic through critical intersections was also assessed for each Proposed Plan Facility analyzed. The potential for off-site noise impacts from deliveries of commercial waste to the Converted MTSs exists at certain hours and Converted MTSs between 8:00 p.m. and 8:00 a.m. The impacts are avoided by restricting the deliveries of commercial waste past specific sensitive receptors along the delivery routes during certain hours at the Converted MTSs. The limitations on specific hours, locations and Converted MTSs required to prevent the potential for off-site noise impacts of collection vehicles delivering waste to these facilities are included in the summary tables for the Commercial Waste Management Study in Appendix D to this DEIS. For the remaining Proposed Plan Facilities analyzed, off-site noise from the DSNY and other agency collection vehicles would not cause an increase in noise levels above 3dBA. Therefore, no adverse health impacts from off-site noise is anticipated.

[45] Passchier-Vermeer, W. and Passchier, W. (2000). "Noise Exposure and Public Health." Environ. Health Perspect. 108 (Suppl. 1):123-131.

33.4 Public Health Evaluation of Odors

Non-living organic matter, such as food waste, is subject to bacterial degradation, especially if wet and/or exposed to air. This decay inevitably produces odors. While the potential for odorous emissions from the Proposed Plan Facilities are evaluated here primarily because of the nuisance and annoyance odors can cause, it is also the case that some odors, when sufficiently intense, can adversely affect health. The quality of an odor can be so obnoxious as to cause nausea, for instance. However, at sufficient concentrations, odorous chemicals can also irritate nerve endings in the respiratory tract, mouth or eyes and cause changes in breathing patterns, sneezing, swelling of nasal membranes, tearing of the eyes and other effects.[46] In most people, these effects disappear fairly soon after the odor dissipates, but in sensitive persons, such as those with asthma, the effects may be longer lasting. Of course, chemicals may adversely affect health independent of their odorous and irritating properties.

Transfer stations, such as the Proposed Plan Facilities, must incorporate design and operational features that would reduce both the potential for odors to develop and the potential for those odors to reach neighboring properties. Maintaining negative air pressure within the facility to prevent escape of odors, installing odor neutralizing systems to treat indoor air before it is exhausted from the waste processing building through vents, and practicing good housekeeping are three such features; these and other features are discussed in Section 3.18. In addition, the potential for noxious odors to reach receptors near each Proposed Plan Facility analyzed was examined in Section 16 of Chapters 4 through 16. For each Proposed Plan Facility analyzed, the analysis suggested that emissions would not pose a risk of detectable, let alone obnoxious, odors at the property boundary or nearby receptors. This being the case, the likelihood of chemical irritative effects is also small, and no adverse odor impacts are expected.

[46] Schiffman, S., Walker, J., Dalton, D., *et al.* (2000). "Potential Health Effects of Odor from Animal Operations, Wastewater Treatment, and Recycling of Byproducts." J. Agromed. 7(1):7-81.

33.5 Vermin Control Measures

Procedures to control vermin, such as rats and insects, would be or, in the case of existing facilities, are incorporated into the operating permit of each Proposed Plan Facility. Licensed exterminators would service each Converted MTS monthly. Exterminating logs kept at the Converted MTSs would provide documentation of their activities. The exterminators would evaluate potential pest and vector problems and apply bait and/or spray throughout the refuse handling area, the tipping floor, the lunch and locker rooms and administrative areas. Standing water in barges not being used would be treated with larvicide and pesticide spray when necessary. During normal operations, exterminating jobs are undertaken as part of a preventive maintenance cycle. Should additional emergency service be needed, additional visits by the exterminators would be made within two to three days. An inspection would then be conducted at the location to determine if baiting and spraying are required. If droppings were present in an area or areas, those areas would be baited. The NYSDEC and DSNY issue operating permits to the existing Proposed Plan Facilities that require procedures to control vermin at these sites. The effectiveness of these procedures is evaluated during facility inspections.

33.6 Public Health Concerns of Host Communities

33.6.1 Introduction

Asthma, especially among children, is a significant medical problem. Parents and public health officials have expressed concern that new industrial facilities might cause or exacerbate asthma either directly, due to emissions from industrial operations, for example, or indirectly, due to increases in vehicular traffic and emissions.

33.6.2 Traffic and Respiratory Health

A search of the scientific literature has not identified studies of the effects of municipal solid waste transfer stations on public health. However, during the last decade, scientists have been studying possible links between respiratory diseases or symptoms, such as cough, asthma and bronchitis, and levels of traffic nearby. Because the Proposed Plan Facilities would require that all solid waste be brought to them by truck, this "traffic literature" is relevant to the public health analysis.

The studies of traffic and respiratory health pertain to children[47] and occasionally to adults.[48] They were performed mostly outside the U.S.,[49] although two studies were performed recently in this country.[50] All of these studies are cross-sectional in design; that is, the respiratory health of the subjects and the levels of traffic nearby were assessed at the same time. Traffic studies that examine diesel traffic in particular are most relevant to this evaluation because the collection vehicles that would transport solid waste to the Proposed Plan Facilities are diesel-powered, as is the equipment used in waste processing operations, such as front-end loaders and tugboats. About half of the studies identified quantified diesel traffic in some manner.[51] In most cases, the health endpoints, such as asthma or allergic rhinitis, were investigated by asking either children or their parents to complete questionnaires that inquired about symptoms or diagnoses of

[47] Brunekreef, B., Janssen, N., de Hartog, J., *et al*. (1997). "Air Pollution from Truck Traffic and Lung Function in Children Living near Motorways." Epidemiol. 8:298-303; Buckeridge, D., Glazier, R., Harvey, B., *et al*. (2002). "Effect of Motor Vehicle Emissions on Respiratory Health in an Urban Area." Environ. Health Perspect. 110(3):293-300; Ciccone, G., Forastiere, F., Agabiti, N., *et al*. (1998). "Road Traffic and Adverse Respiratory Effects in Children." Occup. Environ. Med. 55:771-778; Duhme, G., Weiland, S., Keil, U., *et al*. (1996). "The Association between Self-Reported Symptoms of Asthma and Allergic Rhinitis and Self-Reported Traffic Density on Street of Residence in Adolescents." Epidemiol. 7:578-582; Edwards, J., Walters, S., and Griffiths, R. (1994). "Hospital Admissions for Asthma in Preschool Children: Relationship to Major Roads in Birmingham, United Kingdom." Arch. Environ. Health 49(4):223-227; English, P., Neutra, R., Scalf, R., *et al*. (1999). "Examining Associations between Childhood Asthma and Traffic Flow Using a Geographic Information System." Environ. Health Perspect. 107(9):761-767; Kramer, U., Koch, T., Ranft, U., *et al*. (2000). "Traffic-Related Air Pollution is Associated with Atopy in Children Living in Urban Areas." Epidemiol. 11:64-70; Lee, Y-L., Shaw, C-K., Su, H-J., *et al*. (2003). "Climate, Traffic-Related Air Pollutants and Allergic Rhinitis Prevalence in Middle-School Children in Taiwan." Eur. Respir. J. 21:964-970; Lin, S., Munsie, J., Hwang, S-A., *et al*. (2002). "Childhood Asthma Hospitalization and Residential Traffic Exposure to State Route Traffic." Environ. Res. Sect. A. 88:73-81; Livingstone, A., Shaddick, G., Grundy, C., and Elliott, P. (1996). "Do People Living near Inner City Main Roads have More Asthma Needing Treatment? Case-Control Study." BMJ 312:676-677; Nicolai, T., Carr, D., Weiland, S., *et al*. (2003). "Urban Traffic and Pollutant Exposure Related to Respiratory Outcomes and Atopy in a Large Sample of Children." Eur. Respir. J. 21:956-963; Oosterlee, A., Drijver, M., Lebret, E., and Brunekreef, B. (1996). "Chronic Respiratory Symptoms in Children and Adults Living along Streets with High Traffic Density." Occup. Environ. Med. 53:241-247; van Vliet, P., Knape, M., de Hartog, J., *et al*. (1997). "Motor Vehicle Exhaust and Chronic Respiratory Symptoms in Children Living near Freeways." Environ. Res. 74:122-132; Venn, A., Lewis, S., Cooper, M., *et al*. (2000). "Local Road Activity and the Prevalence, Severity, and Persistence of Wheeze in School Children: Combined Cross Sectional and Longitudinal Study." Occup. Environ. Med. 57(3):152-158; Venn, A., Lewis, S., Cooper, M., *et al*. (2001). "Living near a Main Road and the Risk of Wheezing Illness in Children." Am. J. Respir. Crit. Care Med. 164:2177-2180; Waldron, G., Pottle, B., and Dod, J. (1995). "Asthma and the Motorways – One District's Experience." J. Pub. Health Med. 17(1):85-89; Weiland, S., Mundt, K., Ruckmann, A., and Keil, U. (1994). "Self-Reported Wheezing and Allergic Rhinitis in Children and Traffic Density on Street of Residence." Ann. Epidemiol. 4:243-247; Wilkinson, P., Elliott, P., Grundy, C., *et al*. (1999). "Case-Control Study of Hospital Admission with Asthma in Children Aged 5-14 Years: Relation with Road Traffic in Northwest London." Thorax 54:1070-1074; Wjst, M., Reitmeir, P., Dold, S., *et al*. (1993). "Road Traffic and Adverse Effects on Respiratory Health in Children." BMJ 307:596-600.

[48] Buckeridge *et al*. (2002); Livingstone *et al*. (1996); Oosterlee *et al*. (1996).

[49] All articles initially cited except English *et al*. (1999) and Lin *et al*. (2002).

[50] English *et al*. (1999) and Lin *et al*. (2002).

[51] Brunekreef *et al*. (1997); Buckeridge *et al*. (2002); Ciccone *et al*. (1998); Duhme *et al*. (1996); Lin *et al*. (2002); Nicolai *et al*. (2003); Oosterlee *et al*. (1996); van Vliet *et al*. (1997); Weiland *et al*. (1994).

respiratory illness that occurred either in the last year or at any time. Some investigations used medical databases containing information on hospital visits for asthma[52] or prescriptions for asthma medication,[53] or had children undergo pulmonary function tests or tests for skin sensitivities (an indicator of allergy).[54]

Various methods of gauging traffic flow were used, and while some distinguished between truck and car traffic or focused on car traffic, others did not distinguish between these kinds of vehicles.[55] Regardless, when children were studied, traffic flow was estimated near either the child's school or home. In some studies, children were asked to rate the level of truck traffic near their homes, while in others, investigators used traffic counts made by cities and towns on specific roads, maps of traffic flows, or distances from home or school to highways or to the nearest busy street. In half a dozen investigations, air pollutants were either measured in air near schools and homes and then correlated to traffic flows, or estimated using information on local traffic. Indoor concentrations of air pollutants were determined in only a few studies.[56]

Most traffic studies found associations between some indicator of traffic near a child's home or school and some indicator of respiratory disease; a few found no evidence of an association.[57] Studies that found positive associations, however, were not necessarily consistent, and increases in the risk of wheeze, rhinitis, asthma, etc. were usually fairly small. The apparent effect of nearby traffic on health was frequently stronger in girls than boys.[58]

Studies of particular interest are those conducted in the United States and those in which truck traffic was quantified in some manner. Lin and colleagues (2002) studied white children aged 0 to 14 in Erie County, NY (excluding Buffalo) who were hospitalized for asthma between January 1990 and December 1993. Characteristics of traffic on state routes near the homes of these children were compared to such characteristics for children who were hospitalized during the same period for gastrointestinal illnesses, falls or other non-traffic-related accidents. The characteristics considered included: (1) distance from the child's home to a major state route;

[52] Edwards *et al*. (1994); English *et al*. (1999); Lin *et al*. (2002).
[53] Livingstone *et al*. (1996).
[54] Brunekreef *et al*. (1997); Wjst *et al*. (1993).
[55] Edwards *et al*. (1994); English *et al*. (1999); Kramer *et al*. (2000); Lee *et al*. (2003); Livingstone *et al*. (1996); Venn *et al*. (2000, 2001); Waldron *et al*. (1995); Wilkinson *et al*. (1999).
[56] Brunekreef *et al*. (1997); Kramer *et al*. (2000); van Vliet *et al*. (1997).
[57] Livingstone *et al*. (1996); Waldron *et al*. (1995); Wilkinson *et al*. (1999).

(2) vehicle miles traveled on major state routes within 200 meters or 500 meters of the home; or (3) the proportion of heavy trucks passing within 200 meters or 500 meters of the home on a major state route. In comparisons between the two groups of children, age, sex, poverty level and lower education (the last two determined at the census-tract level) were controlled. Distance of the home from the nearest major state route did not significantly differ for children hospitalized for asthma or for other reasons, nor did traffic density on routes within 500 meters of home. However, the odds ratio (OR)[59] for an asthma hospitalization was statistically significantly increased by the presence of heavy trucks passing within 200 meters of home (OR = 1.43), and for high overall traffic density within 200 meters of home (OR = 1.93).

Children 14 years of age or younger in San Diego County, California, were studied by English *et al*. (1999). Children admitted to hospitals for asthma were compared to other children hospitalized for reasons other than respiratory disease or cancer. Information on traffic flow on virtually all county roads was collected by the county itself and seems to have included only cars. The distance from each child's home to each street within a 550-meter radius was determined, as were the number of cars per day on each of those streets. In contrast to expectations, children hospitalized for asthma were less likely than other children to live nearer to streets with the highest traffic flows or to have higher traffic flows nearby. No difference was found between groups of children for the average traffic volume on all streets within 550 meters of home, nor for the traffic volume on the busiest nearby street. However, among children hospitalized for asthma, children with two or more hospital admissions tended to have higher traffic volumes at the nearest street than did children with only one admission. This tendency was much stronger for girls than boys.

Several other investigations, but not all, found statistically significantly increased ORs for asthma (measured, for example, as current asthma, asthma ever, doctor-diagnosed asthma or hospital admissions for asthma) and various measures of traffic near homes or schools. For example, Nicolai *et al*. (2003) found an OR of 1.8 for asthma among children exposed to the

[58] Brunekreef *et al*. (1997); Kramer *et al*. (2000); Oosterlee *et al*. (1996); van Vliet *et al*. (1997); Venn *et al*. (2001).
[59] The odds ratio (OR) compares the chance of having the disease of interest in a group with an exposure of interest to the chance of having the disease in a group without the exposure. If the odds are the same, meaning there is no effect of exposure on disease, then the OR is 1.0. An OR greater than 1.0 indicates an increased risk of disease, given exposure. An OR of 1.5, for example, indicates a 50% increase in risk.

highest of three categories (i.e., tertile) of car traffic counts and an OR of 1.8 for those exposed to the highest tertile of soot concentration. Buckeridge *et al*. (2002) measured an OR of 1.2 for respiratory hospital admissions per $\log_{10}$ vs. order of magnitude increase in modeled $PM_{2.5}$ concentrations. Wheezing was often assessed separately from asthma. For example, ORs for wheezing of about 5 were found for girls but not boys living near busy streets compared to children living along quiet streets, according to Oosterlee *et al*. (1996). Nicolai *et al*. found an odds ratio of 1.7 for wheezing among children exposed to the highest tertile of car traffic counts. An OR for wheezing of 15 was found by Kramer *et al*. (2000) in association with an increase in outdoor, urban NO_2 of 10 $\mu g/m^3$.

Overall, most studies of traffic and children's respiratory health find some associations between traffic characteristics (such as distance to roads, traffic volumes or truck traffic volumes) and respiratory morbidity measures (such as allergic rhinitis, wheezing or cough), although results can vary a good deal from study to study. However, some weaknesses in the literature must be mentioned.

First, an association, even if statistically significant, does not necessarily indicate cause and effect, particularly in a cross-sectional study. There may be factors, called confounders, that are both associated with residence or schooling near heavy (truck) traffic and that cause or aggravate disease. For example, it is possible that people living near busy streets or highways keep windows closed more than do people who live in quieter neighborhoods. Concentrations of indoor pollutants and agents that may contribute to respiratory illness, such as pet allergens or cigarette smoke, might therefore be higher in homes near heavily trafficked streets. Some of the traffic studies cited in this discussion (particularly those that studied hospitalization rates) were not able to gather information on personal exposure to indoor pollutants. There is also a general concern that differences in socioeconomic status, which likely varies with distance of residence to heavily traveled streets and is associated with health, may not have been adequately controlled.

Second, studies in which information on the exposure of interest and/or the health endpoints of interest are gathered in questionnaires can be vulnerable to bias. If people living near busy streets are already concerned about a potential effect of air pollution on health, they may unconsciously overestimate the level of traffic or severity of illness.

Third, most studies did not distinguish between truck traffic and car traffic. We cannot determine from these studies if associations between car traffic and illness would be relevant to concerns about truck traffic, given differences in the pollutants emitted.

Finally, as most studies were performed outside of the U.S., relevance to the U.S. situation depends on essential similarity between the types of fuels and engines used and pollutants emitted in these parts of the world.

33.6.3 Asthma Causes and Triggers

Asthma is a chronic, inflammatory disease of the small airways characterized by episodic and reversible restriction of breathing passages. Symptoms include difficulty in breathing (which may range from mild to life-threatening), wheezing and coughing. Asthmatic episodes may be triggered by specific substances, environmental conditions and stress, as is discussed below.

The prevalence of asthma and the amount of poorly controlled asthma requiring hospitalization among children has risen significantly in recent decades.[60] In the U.S., approximately five million children (7% of children under age 18) have asthma, and New York is thought to be the state with the second-largest number of affected children.[61] The rate of asthma is increasing most rapidly in children under age five.[62] Asthma exacerbations resulting in hospitalizations appear to be particularly frequent and severe among minority, inner-city children.[63] In the City in

[60] Crater, S. and Platts-Mills, T. (1998). Searching for the Cause of the Increase in Asthma. Curr. Opin. Pediatr. 10:594-599.
[61] Centers for Disease Control (CDC) (1998). Forecasted State-Specific Estimates of Self-Reported Asthma Prevalence – United States, 1998. MMWR 47(47):1022-1025 and Centers for Disease Control (CDC) (1999). Asthma: A Public Health Response. Available at http://www.cdc.gov/nceh/programs/asthma/default.htm.
[62] President's Task Force on Environmental Health Risks and Safety Risks to Children (PTF). (1998). Asthma and the Environment: A Strategy to Protect Children.
[63] Lobach, K. (1996). Providing a "Medical Home." City Health Information: Childhood Asthma. New York City Department of Health.

particular, several groups of researchers have analyzed the distribution and factors affecting asthma hospitalizations and mortality.[64] Asthma prevalence in the City correlates strongly with socioeconomic status, and several factors link asthma with poverty. Factors that related to asthma risk in low-income areas were the number of occupants per apartment (related to bacterial and viral exposures), water leaks (related to fungal exposures), moist basements (related to fungal exposures), deteriorating building materials (related to fungal and mite exposures) and house dust exposure (containing insect parts, animal dander and rodent excreta). Recent statistics on childhood and adult asthma prevalence in the City boroughs are given in Section 33.6.4 below.

The dramatic increase in asthma among children has spurred scientists and clinicians to search for causes and risk factors for the disease, as well as therapies and interventions. The reasons for the rise in the prevalence and severity of asthma are not understood. Suspected factors include changing patterns of childhood illnesses, changing diet, increasing rates of obesity, changing exercise patterns, changing housing, increased vaccinations against childhood respiratory disease, increased survival of very low birth weight babies and increased exposure to indoor-air allergens. Current hypotheses tend to focus on three areas: (1) increases in individual sensitivity (possibly due to reduced respiratory infections); (2) increases in exposure to allergens (due to changes 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 various factors would dominate in specific areas, homes and individuals.

In theory, one can distinguish between "causes" and "triggers" of asthma. Causes would be those factors that make a person susceptible to asthmatic attacks in the first place, while triggers would be 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 allergies 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

[64] Carr, W., Zeitel, L., and Weiss, K. (1992). Variations in Asthma Hospitalization and Deaths in New York City. *Am J Public Health* 82:59-65. de Palo, V.A., Mayo, P.H., Friedman, P., and Rosen, M.J. (1994). Demographic Influences on Asthma Hospital Admission Rates in New York City. *Chest 106:*447-451.Claudio, L., Tulton, L.,

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 United States. Organic materials that cause the immune system to overreact, such as cockroach antigen, dust mite antigens, molds, pet and rodent dander and urine, are the principal 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 had the unintended effects of promoting the growth of dust mites and molds, and of concentrating antigens, irritants and PM indoors.[65] 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.[66]

Some aspects of outdoor pollution are capable of triggering asthma attacks, such as pollens. Some researchers have suggested that outdoor air pollution *per se* is not likely to contribute significantly to the asthma epidemic, however, because air pollution has decreased on the whole while asthma rates have increased.[67] It is nonetheless 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. In addition, weather conditions, and cold air in particular, can elicit asthmatic symptoms independent of air pollution.

Doucette, J., and Landrigan, P. (1999). "Socioeconomic Factors and Asthma Hospitalization Rates in New York City." Journal of Asthma. 36(4):343-350.

[65] Bielory, L. and Deener, A. (1998). Seasonal Variation in the Effects of Major Indoor and Outdoor Environmental Variables on Asthma: Review Article. J. Asthma 35(1):7-48.

[66] Crater, S. and Platts-Mills, T. (1998). Searching for the Cause of the Increase in Asthma. Curr. Opin. Pediatr. 10:594-599.

An additional hypothesis described by Cookson and Moffatt (1997) 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.[68] Infectious disease has been dramatically reduced in our society by the use of antibiotics and immunization programs.

Experimentally, exposure to diesel exhaust particles increased airways resistance in mice,[69] while other studies of mice and humans showed that diesel exhaust particles can enhance responses to allergens.[70] Experiments in which non-asthmatic adults were exposed for an hour to diesel engine exhaust (containing particles and gases) found increased airways resistance[71] and some cellular indicators of inflammatory response;[72] however, these subjects did not experience asthma.

Causes, triggers and prevention of childhood asthma in the City are the subjects of active research.[73] For example, researchers are investigating the possible influence of prenatal exposure to antigenic materials; collecting air pollution measurements in areas of the City with high rates of asthma; testing infants and children for respiratory symptoms; measuring pollutant levels in urine as an indicator of exposure to diesel exhaust; and cleaning, repairing, and addressing pest infestations in apartments of families with asthmatic children. It is hoped that this research would not only help identify the most significant factors leading to asthma but also identify effective prevention measures.

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[67] Ibid.

[68] Cookson, W.O.C.M. and Moffatt, M.F. (1997). Asthma: An Epidemic in the Absence of Infection? Science 275: 41-42.

[69] Sagai, M., Furuyama, A., Ichinose, T. (1996). "Biological Effects of Diesel Exhaust Particles (DEP) III." "Pathogenesis of Asthma Like Symptoms in Mice." Free Radio Biol. Med. 21:199-201 (abstract).

[70] Diaz-Sanchez, D. (1997). "The Role of Diesel Exhaust Particles and their Associated Polyaromatic Hydrocarbons in the Induction of Allergic Airway Disease." Allergy 52:52-56; Takano, II, Yoshikawa, T., Ichinose, T., Miyabara, Y., Imaoka, K., Sagai, M. (1997). "Diesel Exhaust Particles Enhance Antigen-Induced Airway Inflammation and Local Cytokine Expression in Mice." Am. J Respir. Crit. Care Med. 156:36-42.

[71] Rudell, B., Ledin, M.C., Hammarsurom, U., Stjenberg, N., Lundback, G., Sandstrom, T. (1996). "Effects on Symptoms and Lung Function in Humans Experimentally Exposed to Diesel Exhaust." Occup. Environ. Med. 53:6480652 (Abstract).

[72] Salvi, S., Bloomberg, A., Rudell, B., Kelly, F., Sandstrom, T., Holgate, S.T., Frew, A. (1999). "Acute Inflammatory Response in the Airways and Peripheral Blood after Short-Term Exposures to Diesel Exhaust in Healthy Human Volunteers." Am. J Respir. Crit. Care Med. 159:702-709 (Abstract).

[73] Gergen, P., Mitchell, H., Lynn, H., *et al*. (2002). "Understanding the Seasonal Pattern of Childhood Asthma: Results from the National Cooperative Inner-City Asthma Study (NCICAS)." J. Pediatr. 141(5):631-636; Kinney, P., Northridge, M., Chew, G., *et al*. (2002). "On the Front Lines: An Environmental Asthma Intervention in New York City." Amer. J. Pub. Health 92(1):24-26; Miller, R., Chew, G., Bell, C., *et al*. (2001). "Prenatal Exposure, Maternal Sensitization, and Sensitization In Utero to Indoor Air Allergens in an Inner-City Cohort." Am. J. Respir. Crit. Care Med. 164:995-2001; Northridge, M., Yankura, J., Kinney, P., *et al*. (1999). "Diesel Exhaust Exposure among Adolescents in Harlem: A Community-Driven Study." Amer. J. Pub. Health 89(7):998-1002; Perera, F., Illman, S., Kinney, P., *et al*. (2002). "The Challenge of Preventing Environmentally Related Disease in Young Children: Community-Based research in New York City." Environ. Health Perspect. 110(2):197-204.

City officials are well aware of the epidemic of childhood asthma in the City's many boroughs and communities. As reported by the New York City Department of Health and Mental Hygiene (DOHMH) on its web site (www.nyc.gov/html/doh), asthma is a common disease among the City's children and adults. Asthma is a leading cause of missed school among school among children and is 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. In the past two decades, the number of people with asthma has increased, although some improvements, such as fewer hospitalizations, have occurred in recent years. Although it is not yet known how to prevent asthma, it is known that asthma can be controlled both by avoiding exposure to triggers and by taking anti-inflammatory medicines. With good control, almost all people with asthma can lead normal, active lives.

Under the direction of the NYCDOH, the City began an aggressive Childhood Asthma Initiative NYCCAI) in 1997. NYCCAI is a public health effort to reduce asthma morbidity among children 0 to 18 years of age. Expected outcomes of the NYCCAI include reductions in hospitalizations, emergency department visits and school absences due to asthma and, relatedly, improvements in management of childhood asthma among families. NYCCAI is building on existing research education and clinical efforts, resulting in a coordinated and comprehensive effort to understand, treat and prevent asthma in New York City.

NYCCAI is currently working to:

- Improve family management of asthma
- Promote state-of-the-art medical diagnosis and treatment
- Reduce exposure to asthma triggers in both homes and communities
- Increase coordination among families, schools, daycares, medical providers, pharmacists, community-based organizations, housing agencies, managed care organizations, and others
- Monitor and track the number of children with asthma. [74]

[74] New York City Department of Health and Mental Hygiene (May 2003). Asthma Facts, Second Edition. Available at http://www.nyc.gov/html/doh/pdf/asthma/facts.pdf.

Clearly, asthma among children is a major public and personal health problem in the City. Yet the causes of asthma and its increase over the last two decades are not known, and the triggers for exacerbation are only partly 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.

33.6.4 Asthma Morbidity and Mortality in Host Communities

The DOHMH provided preliminary, recent statistics on asthma for the City.[75] Information is collected on the fraction of children and adults with asthma (prevalence), discharges from hospitals after asthma-related illness (morbidity) and deaths from asthma (mortality). The numbers of children with asthma are determined from school health examination forms, usually submitted when children are four or five years old, while numbers of adults with asthma are determined from a telephone survey.

A summary of asthma prevalence among children 4 to 5 years old in areas potentially affected by the Proposed Plan Facilities is provided in Table 33.6-1. The Hunts Point-Mott Haven and Williamsburg-Bushwick neighborhoods show child asthma prevalence considerably above the City average.

A summary of asthma prevalence data for adults in areas potentially affected by the Proposed Plan Facilities is provided in Table 33.6-2. Adults are markedly less likely than children to have an asthma diagnosis. Adult asthma is considerably more prevalent in the South Bronx and Brooklyn Downtown-Heights-Slope neighborhoods than in the City overall.

[75] Personal communications from Dan Kass, DOHMH, to Sarah Armstrong, Cambridge Environmental, Inc., 2003.

Table 33.6-1
Asthma Prevalence Among Children 4 to 5 Years Old

New York City Area	Percent with Asthma in 1999
All of New York City	9.1
Bronx	15.5
Hunts Point-Mott Haven neighborhood	17.1
Brooklyn	8.8
Greenpoint neighborhood	8.9
Williamsburg-Bushwick neighborhood	15.5
Downtown-Heights-Slope neighborhood	9.3
Bensonhurst-Bay Ridge neighborhood	5.2
Sunset Park neighborhood	8.9
Manhattan	11.9
Upper East Side neighborhood	6.4
Queens	5.6
Flushing-Clearview neighborhood	2.6
Long Island City-Astoria neighborhood	5.2
West Queens neighborhood	5.7

Table 33.6-2
Asthma Prevalence Among Adults

New York City Area	Percent with Asthma in 2002
All of New York City	4.4
Bronx	6.2
South Bronx, including the Hunts Point-Mott Haven neighborhood	7.1
Brooklyn	3.7
Greenpoint neighborhood	3.1
Williamsburg-Bushwick neighborhood	6.1
Downtown-Heights-Slope neighborhood	8.0
Bensonhurst-Bay Ridge neighborhood	3.3
Sunset Park neighborhood	5.6
Manhattan	4.5
Upper East Side neighborhood	2.7
Queens	3.7
Flushing-Clearview neighborhood	2.3
Long Island City-Astoria-West Queens neighborhood	4.2

Rates of asthma hospitalization among children aged 0 to 14 dropped markedly between 1997 and 2000 in all the neighborhoods that are potentially affected by the Proposed Plan Facilities. In these neighborhoods, decreases in hospitalization rates ranged from 22% to 56%. The rate decreased the most in the Hunts Point-Mott Haven area, in which DOHMH began a major childhood asthma initiative in 1998. The hospitalization rates for specific zip code areas in 2000 are provided in Table 33.6-3. Hospitalization rates in three zip codes, in the Hunts Point-Mott Haven neighborhood (19454 and 10474) and in the Williamsburg-Bushwick neighborhood (11237), are higher than for the City as a whole.

Asthma mortality data for 2000 are not available by neighborhood. By borough, mortality rates from asthma (deaths per 100,000 people) for people of all ages were 4.9 in the Bronx, 2.9 in Manhattan, 2.2 in Brooklyn and 1.6 in Queens. During the 1990s, asthma mortality rates decreased by about 25% in both sexes in the City. Rates of death from asthma increased with age, being highest among people aged 65 or older.

Table 33.6-3
Hospitalization Rates for Selected Zip Codes

New York City Area or Zip Code	Asthma Hospitalization Rate, Per 1,000 Children Ages 0-14, in 2000
New York City	6.1
10128	2.5
10454	11.2
10474	9.0
11101	6.7
11214	1.1
11215	2.3
11222	2.3
11232	2.7
11237	12.8
11354	4.9
11378	2.7

33.7 Conclusions

This chapter presented a review of scientific information regarding the toxicity of various air pollutants and epidemiologic studies relating traffic to respiratory health, as well as the predicted impacts of the Proposed Plan Facilities' operations and associated traffic on air quality, noise and odor. Recent information on rates of asthma in neighborhoods that may be affected by the Proposed Plan Facilities was also presented. None of the air quality, noise or odor impacts predicted in this DEIS are believed to be of public health significance. Regarding existing, permitted facilities in the Proposed Plan not analyzed in this DEIS, it is assumed that these facilities underwent appropriate environmental review and were determined not to create significant impacts on public health. If one of the Alternative Facilities is selected as a Proposed Plan Facility, a supplemental Public Health analysis will address its air quality, odor and noise impacts.