CHAPTER 12 PUBLIC HEALTH EVALUATION

12.1 Introduction

This chapter evaluates the potential impacts of the MTS Conversion Program on public health. The chief public health concerns are (1) potential health effects (including asthma) of air pollutants released by the Converted MTSs, (2) effects of noise related to the Converted MTSs, (3) effects of odors related to the Converted MTSs, and (4) the potential for vermin (such as rats and insects) to infest areas near Converted MTS sites.

12.2 Air Pollution

12.2.1 Air Pollutants of Concern

Project-related air pollutants of two kinds have been directly assessed in this Draft MTS Environmental Evaluation. One set of pollutants, called criteria pollutants, includes compounds for which national ambient air quality standards (NAAQS) have been established by U.S. Environmental Protection Agency: carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO2), and particulate matter (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 Converted MTSs would be in the form of diesel particulate matter (DPM). Airborne concentrations of other pollutants, termed hazardous air pollutants (HAPs), are not limited nationally. The HAPs evaluated in this Draft MTS Environmental Evaluation include benzene, formaldehyde, 1,3-butadience, benzo(a)pyrene, and other chemicals emitted from diesel fuel and/or exhaust.

12.2.2 Health Effects of Air Pollutants of Concern

12.2.2.1 CO

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] EPA's review of CO toxicity at ambient concentrations determined that the most sensitive effects of exposure are on the cardiovascular system in

[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.

persons with pre-existing heart disease, namely a quicker onset of angina and EKG changes.[2] The NAAQS for CO (9 ppm for an eight-hour average and 35 ppm for a one-hour average) are set to keep COHb levels in the blood low enough to reduce the risk of these cardiovascular effects. New York City is in attainment for the CO air quality standards.

12.2.2.2 NO_2

Nitrogen dioxide (NO_2) is one of several related oxides of nitrogen, collectively termed "NOx," found in ambient air. EPA 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] At much higher concentrations than are found in ambient air, long-term exposure to NO_2 has produced emphysema-like changes in laboratory rodents. As of 1996, when EPA last reviewed the NAAQS for NO_2 (53 ppb as an annual average), the entire country was in compliance with the standard.[4]

12.2.2.3 SO_2

Analogous to NO_2, sulfur dioxide (SO_2) is one of several oxides of sulfur, or SOx, 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. New York City is in compliance with the SO_2 NAAQS.

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[2] EPA (1994). "National Ambient Air Quality Standards for carbon monoxide – final decision." *Federal Register*: August 1.

[3] EPA (1995). "National Ambient Air Quality Standards for nitrogen dioxide: proposed decision." *Federal Register*: October 11.

[4] EPA (1996). "National Ambient Air Quality Standards for nitrogen dioxide: final decision." *Federal Register*: October 8.

12.2.2.4 PM_{10} and $PM_{2.5}$

Unlike the other criteria pollutants, which are specific chemical molecules, particulate matter (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 microns (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] EPA (1988). "Proposed decision not to revise the National Ambient Air Quality Standards for sulfur oxides (sulfur dioxide)." *Federal Register*:April 26; EPA (1996). "National Ambient Air Quality Standards for sulfur oxides (sulfur dioxide) – final decision." *Federal Register*: May 22.

[6] EPA (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] EPA[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

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[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 US 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] EPA (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]; EPA (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] EPA (1996).

not. The solubility or insolubility of aerosols and particles is expected to be one 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 this activities related to the Converted MTSs would be from diesel engine exhaust and, hence, in the form of diesel particulate matter (DPM). This mixture of gases and particles has been unusually well-studied.

12.2.2.5 Diesel particulate matter (DPM)

Diesel particulate matter (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 a HAP. Therefore, DPM impacts have not been quantitatively assessed in this Draft MTS Environmental Evaluation 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

[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. In press.

[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; EPA (2002). Health Assessment Document for Diesel Engine Exhaust. EPA/600/8-90/057F.

large 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 - 3,000 µg DPM/m^3, while the levels at which these effects are *not* observed — that is, the NOAELs — range from about 100 – 500 µg/m^3.[18] With regard to premature mortality due to lifetime exposure to DPM, the LOAELs are about 6,000 µg/m^3 in F344 rats[19] and 4,000 µg/m^3 in MMRI mice[20], although other rodents tested in other laboratories showed no decreased survival even given lifetime exposures of some 7,000 µg/m^3.[21]

Laboratory rats, though not necessarily other test species, develop lung tumors during lifetime exposures to very high concentrations of DPM. As noted by EPA[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 environmental levels of exposure." EPA also notes, "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."

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[17] EPA (2002); EPA (2003a). IRIS record for diesel engine exhaust. Available at www.epa.gov/iris/subst/0642.htm.

[18] EPA (2003a).

[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 EPA (2002).

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 1–10 $\mu g/m^3$. The MTS Conversion Program would add less than 1 $\mu g/m^3$, measured as $PM_{2.5}$ (Section 12.2.3 below).

12.2.2.6 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. EPA 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 a year developed nasal and laryngeal tumors. For non-cancer effects, EPA has derived a concentration of acetaldehyde of 9 $\mu g/m^3$ (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]

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

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[22] EPA (2003a).

[23] National Toxicology Program (2002). 10th Report on Carcinogens. U.S. Department of Health and Human Services.

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

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. EPA 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. EPA 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 eight-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 part-per-billion levels (or less) of benzene in air, such as occurs around the U.S., although cancer remains a theoretical concern.

1,3-Butadiene is a gas 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 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. EPA considers 1,3-butadiene to be carcinogenic to humans by inhalation

[26] EPA (2003c). IRIS record for acrolein. Available at www.epa.gov/iris/subst/0364.htm.
[27] ACGIH (2002); NIOSH (2003).
[28] Graham, J., Green, L., and Roberts. M. In Search of Safety. Harvard University Press: Cambridge, MA. 1988.

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, EPA 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 EPA 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, EPA 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 Draft MTS Environmental Evaluation—benzo(a)pyrene, anthracene, benzo(a)anthracene, chrysene, dibenz(a,h)anthracene, phenanthrene, and pyrene—are collectively known as polycyclic aromatic hydrocarbons, or PAHs. PAHs are typically produced through combustion, whether of diesel fuel, wood, cigarettes, or other organic matter. While no single PAH has been 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 EPA's traditional classification of chemicals in terms of carcinogenicity, benzo(a)pyrene,

[29] EPA (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] EPA (2003e). IRIS record for formaldehyde. Available at www.epa.gov/iris/subst/0419.htm.
[33] ACGIH (2002); NIOSH (2003).

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 EPA have made an estimate of cancer risk for only one of the PAHs evaluated in this draft MTS Environmental Evaluation, 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 EPA states that the carcinogenic potential "cannot be determined" at this time. EPA 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]

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 EPA. EPA 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]

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[34] EPA (2003f). IRIS record for benzo(a)pyrene. Available at www.epa.gov/iris/subst/0136.htm.
[35] ACGIH (2003).
[36] EPA (2003g). IRIS record for naphthalene. Available at www.epa.gov/iris/subst/0436.htm.
[37] ACGIH (2002); NIOSH (2003).
[38] EPA (2003h). IRIS record for toluene. Available at www.epa.gov/iris/subst/0118.htm.
[39] ACGIH (2002); NIOSH (2003).

Xylene is an aromatic solvent related to benzene and toluene, and like them found in fuels and fuel exhausts as well as 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 cause the neurological effects. Xylene is not considered a carcinogen. EPA 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]

12.2.3 Public Health Assessment of Air Pollutants of Concern

In this Draft MTS Environmental Evaluation, the potential health impacts of these two sets of pollutants—criteria pollutants and HAPs—are assessed differently. EPA has established NAAQS for the criteria air pollutants and has set Significant Impact Levels (SILs) for CO, NO_2, SO_2, and $PM_{10.}$ For these criteria pollutants, estimated, Converted MTS-related increases in concentrations of criteria pollutants can be added to the Existing ("background") concentrations and the total compared to the NAAQS (see Table 10-3 of Chapters 4 through 10). The regulatory program for $PM_{2.5}$ is still under development, and EPA has not yet set SILs for $PM_{2.5}$, but NYCDEP andNYSDEC have proposed draft policies for the assessment of $PM_{2.5}$ impacts. In particular, the draft NYSDEC policy is that a "project . . .[with $PM_{2.5}$] air quality impacts equal to or less than two percent (2%) of the annual NAAQS standard of 15 $\mu g/m^3$, or 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 has proposed that Converted MTS-related impacts on the nearest neighborhood no larger than 0.1 $\mu g/m^3$, assessed annually, are to be considered

[40] EPA (2003i). IRIS record for xylenes. Available at www.epa.gov/iris/subst/0270.htm.
[41] ACGIH (2002); NIOSH (2003).

insignificant.[43] These are referred to as interim STVs that are to be used as screening thresholds in impact evaluations until EPA 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.

12.2.3.1 Criteria pollutants

Section 3.12 describes the air quality modeling methodologies used to estimate increases in airborne concentrations of criteria pollutants stemming from on-site activities (*i.e.*, operation of heavy machinery) and off-site activities (i.e., emissions from truck traffic at critical intersections). For on-site analyses, sources of criteria pollutant emissions such as collection vehicles, front end loaders and tug boats were catalogued for each site. The emission rates of criteria pollutants from each on-site source were combined in a 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 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}. Then the receptors with the greatest estimated pollutant increments due to on-site activities were identified for each site. 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 City and state interim STVs. The summary tables of on-site results for each converted MTS' are presented in Chapters 4 through 10, Table 10-3 of each chapter.

As the table demonstrates, in no case do the total predicted concentrations of criteria pollutants 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 established as the interim SILs. 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 EPA 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 policies of the City and the state.

Similar analyses were performed for traffic-generated criteria pollutants at critical intersections, focusing on CO, PM_{10}, and $PM_{2.5}$. Air quality impacts were evaluated for at least two intersections near each Converted MTS. With one exception, the total anticipated concentrations of CO and PM_{10} (traffic plus background) at these intersections were less than the applicable NAAQS. The one exception is the intersection of Bay Parkway, Cropsey Ave., and Shore Parkway near the Southwest Brooklyn Converted MTS. Here, the annual average concentration of PM_{10} is anticipated to exceed the NAAQS by 10%. The modeled concentration is anticipated to be the same whether the Southwest Brooklyn Converted MTS is built, however, or whether some other activity occurs. For all the Converted MTSs, then, no adverse health consequences are expected from traffic-related CO or PM_{10} emissions, either because the expected concentrations are below the health-based limits or because the degree of exceedance is small and not worse for Converted MTS-related traffic as opposed to other traffic.

For the $PM_{2.5}$ analyses, the incremental concentrations contributed by traffic related to the Converted MTSs 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 Converted MTSs were evaluated and then added to the traffic-related contributions. On-site operations are not expected to cause exceedances of $PM_{2.5}$ STVs at any intersection. Furthermore, with one exception, the combined $PM_{2.5}$ impacts at critical intersections from traffic and Converted MTS operations do not exceed the STVs either. The exception is the intersection of York Ave and 91st Street near the East 91st Street Converted MTS where the annual average $PM_{2.5}$ concentration assessed 15 m from the intersection is anticipated to be 0.16 $\mu g/m^3$, in contrast to an applicable STV of 0.1 $\mu g/m^3$. This slight exceedance of the STV is not expected to cause adverse health consequences.

12.2.3.2 Hazardous Air Pollutants (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 one-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 one-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 EPA reference concentrations noted in the HAPs discussions above, and takes into account the carcinogenic and non-carcinogenic effects discussed in Section 12.2.2.6 above.[44] Note that cancer risk is calculated for both known and probable human carcinogens. The NYSDEC toxicity benchmarks are presented in Table 12.2-2.

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

Table 12.2-2
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	-	0.002	1.7×10^{-3}
1,3-Butadiene	-	0.0036	2.8×10^{-4}
Formaldehyde	30	0.060	1.3×10^{-5}
Non-carcinogens			
Propylene	-	3000	-
Acrolein	0.19	0.020	-
Anthracene	-	0.020	-
Benzo(a)anthracene	-	0.020	-
Chrysene	-	0.020	-
Dibenz(a,h)anthracene	-	0.020	-
Naphthalene	7,900	3.0	-
Phenanthrene	-	0.020	-
Pyrene	-	0.020	-
Toluene	37,000	400	-
Xylene	4,300	700	-

At all Converted MTS sites, all one-hour increments of HAPs are substantially below corresponding NYSDEC short-term guideline concentrations (SGCs); similarly, all annual-average increments are substantially below annual guideline concentrations (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 site 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 tables 10-4 in Chapters 4 through 10.) 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 site. This HAPs analysis thus indicates that emissions of the chemicals studied from the on-site operations at all sites are very unlikely to adversely affect health.

12.3 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 by 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 dB(A), and the noise analysis methodology described in Section 3.14 quantifies Converted MTS-related noise in this unit. Long-term exposure to noise averaging 70 dB(A) or less is not thought to pose a risk of hearing impairment.[45]

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.[46] The reviewers concluded that risk of hearing impairment, hypertension, and ischemic heart disease was increased at average noise levels of 70 dB(A) or more.

The chief criterion by which noise impacts were assessed near the Converted MTS sites was whether on-site operations at peak levels were likely to increase total noise at near-by receptors by 3 dB(A) 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 three sites (Greenpoint, Hamilton Avenue, and West 59th Street), no receptors were located within or near to the 55-dB(A) contour of Converted MTS-related noise, so no further assessment was required. At three other facilities (Northshore, East 91st Street, and West 135th Street), the additional noise from on-site operations was less than the 3-dB(A) limit. At two other sites (Southwest Brooklyn

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

and South Bronx), incremental noise was estimated to exceed the 3-dB(A) limit, so mitigation measures would be required (see Section 4.12.3.2.1 and 5.12.3.2.1). At none of the Converted MTSs was the average noise level, including the contribution from on-site operations, likely to exceed 70 dB(A) at the facility boundary**.** With mitigation measures in place, on-site operations would make only a minor increase in overall noise levels. No adverse health impact from on-site noise is anticipated.

The potential for adverse levels of noise due to Converted MTS-related traffic through critical intersections was also assessed for each site. Preliminary screening analyses showed that no noise impacts were likely at intersections near the Converted MTSs in the South Bronx, Southwest Brooklyn, Greenpoint, or at West 135th Street or West 59th Street. However, more detailed analyses were required for the Converted MTSs at Hamilton Ave., East 91st Street, and North Shore. In the noise analysis for Hamilton Avenue., a possible noise impact was found for one of five intersection/time-of-day combinations. Further analysis suggested that traffic noise in excess of 3 dB(A) would occur during just one nighttime hour at this location. In the analysis for East 91st Street, possible noise impacts were found for two of four intersection/time-of-day combinations, but more detailed analysis suggested that traffic noise would increase by more than 3 db(A) during only one nighttime hour. In the North Shore analysis, possible noise impacts were found for two of five intersection/time-of-day combinations. At each location, traffic noise in excess of 3 db(A) might occur for two hours during the night or early morning. Such relatively infrequent occurrences of excess noise are not likely to harm health.

[46] Passchier-Vermeer, W. and Passchier, W. (2000). "Noise exposure and public health." Environ. Health Perspect. 108 (Suppl. 1):123-131.

12.4 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 Converted MTSs are evaluated in this Draft MTS Environmental Evaluation 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.[47] 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 Converted MTSs, 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 3.13. In addition, the potential for noxious odors to reach receptors near each Converted MTS was examined in section 11 of Chapters 4 through 11. For each Converted MTS, the analysis suggested that emissions would not pose a risk of detectable, let alone obnoxious, odors at nearby receptors. This being the case, the likelihood of chemical irritative effects is also small, and no adverse odor impacts are expected.

[47] 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.

12.5 Vermin Control Measures

Procedures to control vermin, such as rats and insects, would be incorporated into the operating permit of each Converted MTS. Two in-house, licensed exterminators would service the Converted MTS monthly. Exterminating logs at the Converted MTS would provide documentation of their activities. The exterminators evaluate potential pest and vector problems and apply bait and/or spray. Standing water in barges not being used would be treated with larvicide and pesticide spray when necessary. The control program requires the application of spray and the placement of traps throughout the refuse handling area, the tipping floor, the lunch and locker rooms, and administrative areas. Should additional emergency service be needed, the exterminators would be dispatched from the Converted MTS headquarters from which they are assigned. During normal operations, exterminating jobs are undertaken as part of a preventive maintenance cycle that would be performed every 45 days. Emergency complaints would prompt additional visits within 2 to 3 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.

12.6 Public Health Concerns of Host Communities

12.6.1 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.

12.6.2 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 Converted MTSs 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[48], occasionally also to adults,[49] and were performed mostly outside the U.S.[50], although two studies were performed recently in

[48] 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

this country[51]. 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 Converted MTSs are diesel-powered, as is the equipment used in waste processing operations, such as, front-end loaders and tug boats. About half of the studies identified quantified diesel traffic in some manner[52]. 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 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[53] or prescriptions for asthma medication[54], or had children undergo pulmonary function tests or tests for skin sensitivities (an indicator of allergy)[55].

Various methods of gauging traffic flow were used, and while some distinguished between truck and car traffic or focused on car traffic, some studies did not distinguish between these kinds of vehicles[56]. 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

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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 north west 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.

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

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

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

[52] 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).

[53] Edwards *et al.* (1994); English *et al.* (1999); Lin *et al.* (2002).

[54] Livingstone *et al.* (1996).

[55] Brunekreef *et al.* (1997); Wjst *et al.* (1993).

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[57].

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[58]. Studies that found positive associations, however, were not necessarily consistent, and increases in 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[59].

Studies of particular interest are those conducted in the U.S. and those in which truck traffic was quantified in some manner. Lin and colleagues (2002) studied white children aged 0-14 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, (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[60] 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).

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[56] 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).

[57] Brunekreef *et al*. (1997); Kramer *et al*. (2000); van Vliet *et al*. (1997),

[58] Livingstone *et al*. (1996); Waldron *et al*. (1995); Wilkinson *et al*. (1999)

[59] Brunekreef *et al*. (1997); Kramer *et al*. (2000); Oosterlee *et al*. (1996); van Vliet *et al*. (1997); Venn *et al*. (2001).

[60] 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.

Children 14 years of age or less in San Diego County, CA., were studied by English *et al*. (1999). As in the Lin *et al*. investigation, 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 odds ratios 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 odds ratio of 1.8 for asthma among children exposed to the highest of three categories (*i.e*., tertile) of car traffic counts and an odds ratio of 1.8 for those exposed to the highest tertile of soot concentration. Buckeridge *et al*. (2002) measured an odds ratio of 1.2 for respiratory hospital admissions per log10 vs order of magnitude increase in modeled $PM_{2.5}$ concentrations. Wheezing was often assessed separately from asthma. For example, odds ratios 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 wheeze among children exposed to the highest tertile of car traffic counts. An odds ratio for wheezing of 15 was found by Kramer *et al*. (2000) in association with an increase in outdoor, urban NO_2 of 10 micrograms per cubic meter of air ($\mu g/m^3$).

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. Some weaknesses in the literature must be mentioned, however. 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.

12.6.3 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.[61] In the U.S., approximately 5 million children (7 percent of children under age 18) have asthma, and New York is thought to be the state with the second-largest number of affected children.[62] The rate of asthma is increasing most rapidly in children under age 5.[63] Asthma exacerbations resulting in hospitalizations appear to be particularly frequent and severe among minority, inner-city children.[64] In New York City in particular, several groups of researchers have analyzed the distribution and factors affecting asthma hospitalizations and mortality.[65] 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 New York City boroughs are given in Section 12.6.4 below.

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[61] Crater, S. and Platts-Mills, T. (1998). Searching for the cause of the increase in asthma. Curr. Opin. Pediatr. 10:594-599.

[62] 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.

[63] President's Task Force on Environmental Health Risks and Safety Risks to Children (PTF). (1998). Asthma and the environment: a strategy to protect children.

[64] Lobach, K. (1996). Providing a "medical home." City Health Information: Childhood Asthma. New York City Department of Health.

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

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 allergy is an important risk factor for developing asthma, there may have been no real increase in the number of genetically susceptible children, but rather a growth in the prevalence of factors that promote asthma development or trigger an attack. For a child suffering from asthma, however, identification and elimination of triggering factors is of greatest practical importance.

Allergens in the indoor environment are definitely important triggers of asthma in the U.S. 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 particulate matter indoors.[66] 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.[67]

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.[68] 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.

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

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[66] 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.

[67] Crater, S. and Platts-Mills, T. (1998). Searching for the cause of the increase in asthma. Curr. Opin. Pediatr. 10:594-599.

[68] Ibid.

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

Experimentally, exposure to diesel exhaust particles increased airways resistance in mice,[70] while other studies of mice and humans showed that diesel exhaust particles can enhance responses to allergens.[71] Experiments in which non-asthmatic adults were exposed for an hour to diesel engine exhaust (containing particles and gases) found increased airways resistance[72] and some cellular indicators of inflammatory response;[73] however, these subjects did not experience asthma.

Causes, triggers, and prevention of childhood asthma in New York City are the subjects of active research.[74] 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.

New York City officials are well aware of the epidemic of childhood asthma in the City's many boroughs and communities, and, under the direction of the Department of Health, began an aggressive Asthma Initiative in 1997. The goals of the Asthma Initiative are to reduce illness and death from childhood asthma by: (1) strengthening the ability of institutions, such as schools and

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[70] 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).

[71] 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.

[72] 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).

[73] 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).

[74] 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.

medical facilities, to respond to the disease; (2) encouraging and coordinating asthma research; (3) facilitating interactions among health care facilities, schools, communities and government agencies; and (4) giving special attention to high-risk populations. Among the Initiative's recommendations for preventing asthma episodes are: (1) avoid cigarette smoke; (2) reduce exposure to dust mites; (3) avoid furred pets and birds; (4) eliminate or reduce roaches; (5) close windows and use an air conditioner when pollen or air pollution is bad; and (6) help improve the environment.[75]

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.

12.6.4 Asthma Morbidity and Mortality in Host Communities

The New York City Department of Health and Mental Hygiene (DOHMH) provided preliminary, recent statistics on asthma for City.[76] 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 in areas potentially affected by the Converted MTSs is provided in Table 12.6-1. The Hunts Point-Motts Haven and Washington Heights-Inwood neighborhoods show child asthma prevalence considerably above the City average.

A summary of asthma prevalence data for adults in areas potentially affected by the Converted MTSs is provided in Table 12.6-2. Adults are markedly less likely than children to have an asthma diagnosis. Adult asthma is considerably more prevalent in the South Bronx, Brooklyn, Downtown-Heights-Slope, and Washington Heights-Inwood neighborhoods than in the City overall.

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[75] New York City Department of Health. (1999). "Take Charge of Asthma." Community Asthma Program. Available at http://www.ci.nyc.ny.us/html/doh/html/asthma/atake.html.

[76] Personal communications from Dan Kass to Sarah Armstrong, 2003.

Table 12.6-1

Asthma Prevalence Among Children 4-5 Years Old

New York City Area	% 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
Downtown-Heights-Slope neighborhood	9.3
Bensonhurst-Bay Ridge neighborhood	5.2
Manhattan	11.9
Washington Heights-Inwood neighborhood	12.6
Upper East Side neighborhood	6.4
Chelsea-Clinton neighborhood	9.4
Queens	5.6
Flushing-Clearview neighborhood	2.6

Table 12.6-2
Asthma Prevalence Among Adults

New York City Area	% 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
Downtown-Heights-Slope neighborhood	8.0
Bensonhurst-Bay Ridge neighborhood	3.3
Manhattan	4.5
Washington Heights-Inwood neighborhood	6.7
Upper East Side neighborhood	2.7
Chelsea-Clinton neighborhood	3.6
Queens	3.7
Flushing-Clearview neighborhood	2.3

Asthma is the leading cause of hospitalization of children in New York City. Rates of asthma hospitalization among children aged zero to 14 dropped markedly between 1997 and 2000 in all the neighborhoods that are potentially affected by the Converted MTSs except for one (Flushing-Clearview). In these neighborhoods, decreases in hospitalization rates ranged from 42% 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. In Flushing-Clearview, the rate increased by 7%. The hospitalization rates for specific zip code areas in 2000 are provided in Table 12.6-3. Hospitalization rates in two zip codes, those near the West 59th Street Converted MTS and the South Bronx Converted MTS, 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 1990's, asthma mortality rates decreased by about 25% in both sexes in New York City. Rates of death from asthma increased with age, being highest among people aged 65 or older.

Table 12.6-3
Hospitalization Rates for Selected Zip Codes

New York City Area or Zip Code	Asthma Hospitalization Rate, Per 1000 Children Ages 0-14, in 2000
New York City	6.1
10019	7.4
10031	5.0
10128	2.5
10474	9.0
11214	1.1
11215	2.3
11222	2.3
11354	4.9

12.7 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 Converted MTSs' operations and associated traffic on air quality, noise, and odor. Recent information on rates of asthma in neighborhoods that may be affected by the Converted MTSs were also presented. None of the predicted air quality, noise, or odor impacts are believed to be of public health significance. With rare exceptions, predicted impacts are less than limits established to protect health or prevent nuisance. The exceptions are likely to be too infrequent to be important to health, are likely to exist even if the MTS Conversion Program does not occur, and/or can likely be eliminated through mitigation measures.