Health Consultation

PUBLIC SCHOOL 51X
BRONX BOROUGH, CITY OF NEW YORK

Prepared by:
New York State Department of Health

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Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

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INTRODUCTION

The New York State Department of Health (NYS DOH), under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) reviewed environmental data (sub-slab soil vapor, indoor air and outdoor air data) and evaluated the public health implications of the indoor air data that were collected at the former Bronx New School (PS 51X) at 3200 Jerome Avenue in Bronx County, New York. The United Federation of Teachers (UFT) requested a public health consultation and assessment in an August 5, 2011 letter from the President of the UFT to the Petition Coordinator for ATSDR (Mulgrew, 2011). This health consultation summarizes the NYS DOH's public health evaluation of the exposures to contaminants in indoor air at the former school resulting from soil vapor intrusion by chlorinated solvents under the school.

BACKGROUND AND STATEMENT OF ISSUES

The Bronx New School (PS 51X) was located at 3200 Jerome Avenue in Bronx County from 1993 until June 2011. The New York City Department of Education (NYC DOE) leased the building as a school for about 270 students in grades kindergarten through five. As part of the NYC DOE's lease renewal, the New York City School Construction Authority (NYC SCA) performed an audit and environmental investigation of the building (AKRF, 2011a, 2011b). The investigation found that the chemical trichloroethene (TCE), also known as trichloroethylene, was present in the indoor air of areas routinely occupied by faculty, staff, and students at levels up to 53 micrograms per cubic meter (mcg/m^3), which exceeds the NYS DOH's air guideline for TCE of 5 mcg/m^3 (NYS DOH, 2006a). Subsequent sampling of soil vapor beneath the foundation of the school showed up to 53,300 mcg/m^3 of TCE, indicating that soil vapor intrusion was the likely source of the indoor air contamination.

The investigation also found that the property had been used previously as an automotive garage from the 1940's to the 1950's and as a lamp manufacturer from 1957 to 1991. The lamp manufacturer, Nessen Lamps, used TCE in its manufacturing process. The federal Resource Conservation and Recovery Act (RCRA) requires that businesses that use certain chemicals, such as TCE, report their use of regulated chemicals. RCRA records show that Nessen Lamps used TCE between 1982 and 1987. In addition to Nessen Lamps, TCE may have been used by some automotive garages in the area to remove grease from metal parts.

PS 51X has been relocated and the building at 3200 Jerome Avenue is vacant at the time of this report. Due to the contamination present in soil vapor at the property, the New York State Department of Environmental Conservation designated 3200 Jerome Avenue as a potential inactive hazardous waste site. Designation of the property as a potential inactive hazardous waste site allows the state to investigate the property using

State resources. In cooperation with the State, the property owner (a private corporation) has initiated an investigation to find the source of the contamination.

DISCUSSION

The NYC SCA investigated PS 51X at 3200 Jerome Avenue in Bronx County as part of a lease renewal process. NYC SCA's consultant, AKRF, performed the investigations and its findings are documented in two reports: "Phase I Environmental Site Assessment of the Bronx New School" (AKRF, 2011a), and "Indoor Air Quality and Vapor Intrusion Survey of the Bronx New School (PS 51X)" (AKRF, 2011b). These two reports summarize several rounds of sampling at the property in 2011[1]. On January 22, two indoor air samples and one outdoor air sample were collected at the school. On March 27, three sub-slab soil gas samples and one outdoor air sample were collected. On April 22, three indoor air samples, three sub-slab soil gas samples, and one outdoor air sample were collected. On May 14, after enhanced overnight ventilation of the building[2], three indoor air samples and one outdoor air sample were collected. The data are summarized in Table 1.

Several chemicals in addition to TCE were found at low levels in the indoor air of the school. The presence of chemicals in indoor air at low concentrations is not unusual because chemicals are found in products used every day in homes and schools. The levels did not exceed typical levels found in indoor air, based on a review of several databases on background levels of chemicals in indoor air of homes and offices (NYS DOH, 2005, 2006b).

The indoor air sample locations included the first floor cafeteria, the hallway near the entrance, and the partial basement. The first floor cafeteria and the hallway are common areas frequented by faculty, staff, and students during the school day. Samples from these areas would represent air that faculty, staff, and students would breathe during a school day. Based on discussions with staff from the NYC DOE, the partial basement was not routinely occupied. Therefore, exposure to the TCE levels found in the partial basement would be infrequent and intermittent, and are unlikely to be representative of the exposures to faculty, staff, and students.

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[1] All samples were collected using Summa® canisters. Collection and analyses using this method allow measurement of small quantities of chemicals in air. According to the report: "The samples collected on January 22, March 27, and May 14, 2011 were collected for approximately six hours, and the samples collected on April 22, 2011 were collected for eight hours. The indoor air samples were collected at a height of approximately 3 to 5 feet to simulate a typical Indoor Air Quality and Vapor Intrusion Survey breathing zone. Samples collected on January 22 and May 14, 2011 were analyzed by TestAmerica-Burlington of South Burlington, Vermont; samples collected on March 27, 2011 were analyzed by Alpha Analytical, Inc. of Mansfield, Massachusetts; and samples collected on April 22, 2011 were analyzed by Con-Test Analytical Laboratory (Con-Test) of East Longmeadow, Massachusetts. TestAmerica-Burlington, Alpha Analytical, Inc. and Con-Test are all NYS DOH Environmental Laboratory Approval Program (ELAP)-certified analytical laboratories for air quality sample analyses."

[2] Enhanced overnight ventilation is defined in AKRF (2011b) as ventilating the school building overnight by completely opening several windows in all first and second floor classrooms, operating the building HVAC system in summer mode, and activating a roof-mounted, 8,000 cubic foot per minute ventilation fan between 4 PM on May 13, 2011 and 9:45 AM on May 14, 2011.

Table 1. 2011 Trichloroethene Air Sampling Data for 3200 Jerome Ave (PS 51X).*

All values in micrograms per cubic meter (mcg/m^3)

Sample Location	Date in 2011			
	1/22	3/27	4/22	5/14**
Indoor Air				
First Floor Cafeteria	53	NS	22	2.4
Hallway Near Entrance	49	NS	10	1.7
Partial Basement	NS	NS	310	580/670***
Sub-Slab Soil Vapor				
First Floor Cafeteria	NS	53,300	21,000	NS
Hallway Near Entrance	NS	706	730	NS
Partial Basement	NS	34,900	31,000	NS
Outdoor Air				
Roof	4.4	NS	0.43	ND (0.21)
Sidewalk	NS	ND (0.107)	NS	NS

NS – not sampled
ND – not detected at the detection limit shown in parentheses.
*The New York State air guideline for trichloroethene is 5 micrograms per cubic meter.
**Sampled after enhanced ventilation
***Duplicate sample

A. Exposure Pathways

Based on the results of the indoor air sampling, faculty, staff, and students at PS 51X were likely exposed to elevated levels of TCE by breathing indoor air. The enhanced overnight ventilation reduced the concentrations of TCE to levels near those we might expect to find in indoor air. However, we do not know how often and under what conditions the building may have been similarly ventilated, if at all. We have data for a limited number of air samples and we do not know what level might be representative of levels found over the long-term occupancy of the school, whether the levels fluctuated seasonally, or whether they have fluctuated over the course of the school occupancy.

B. Public Health Implications

The NYS DOH evaluated the risks for cancer and noncancer health effects for TCE and the exposure pathway identified for PS 51X. These risks depend primarily on how much TCE was in the air, and how often and how long people were exposed. The following section summarizes information on the health effects of TCE and characterizes the risk associated with exposure to TCE in air at PS 51X. This risk characterization cannot be used to predict whether or not a specific person will have health effects. The risk characterization is an expression of the probability a person might have health effects

based on limited information about the levels of TCE in the indoor air and assumptions about how long and how often people might have been exposed.

Past Exposure to TCE in Air at PS 51X

Faculty, staff, and students were exposed to TCE in indoor air at PS 51X. Faculty and staff could have been exposed from 1993 to 2011, when the building was used as a school, and students could have been exposed for up to six years (i.e., kindergarten to 5th grade). Air samples taken in 2011 from areas in the building likely to be occupied by people, first floor hallway and cafeteria, prior to increased ventilation of the building, contained TCE ranging from 10 mcg/m^3 to 53 mcg/m^3 and averaging 33.5 mcg/m^3.

We first compared the indoor air sampling results for TCE (from Table 1 in the text) to levels we would typically expect to find in indoor air (background levels), ATSDR public health comparison values, and the NYS DOH TCE air guideline of 5 mcg/m^3 (see Appendix A, Table 1). The ATSDR comparison values (0.24 mcg/m^3 and 2 mcg/m^3; ATSDR 2013a) are based solely on health-based criteria, while the NYS air guideline considers other factors including the ability to reliably detect the chemicals, background levels, and gaps in the toxicological databases. Both the comparison values and the air guideline are air concentrations of TCE at which we do not expect adverse health effects to occur. Because the TCE air levels at the school exceeded background levels, the NYS DOH air guideline and health-based comparison values, we further evaluated the health risks for exposure to TCE.

Health Effects of TCE

Lastly, studies of people exposed to TCE provide convincing evidence of a cause-effect relationship between TCE exposure and cancer. The strongest evidence comes from several well-designed studies that found increased risks of kidney cancer among workers exposed to TCE during the degreasing of metal parts, with more limited evidence for non-Hodgkin's lymphoma (NHL) and liver cancer. (ATSDR, 1997; EPA, 2011a; NTP, 2011; NYS DOH, 2006a). In laboratory animals, lifetime exposure to high levels of TCE has caused cancer (including kidney, liver, lung cancers, and lymphomas). Based on the evidence that TCE causes kidney cancer in people and the results of animal studies, the EPA has concluded that TCE is carcinogenic (causes cancer) in humans by all routes of exposure (EPA, 2011a). Overall, the studies of humans and animals exposed to high levels of TCE suggest that there may be an increased risk of cancer in people who are exposed to lower levels over long periods of time.

Long-term exposure to high levels of TCE in workplace air has also caused effects on the central nervous system and irritation of the mucous membranes in humans (ATSDR, 1997). Some studies also reported an increased risk for adverse effects on human fetal development in the offspring of women who lived in areas with elevated levels of TCE in air or drinking water (Goldberg *et al*., 1990; Forand *et al*., 2012). Due to

limitations in the studies, we do not know if the observed effects on fetal development are due to TCE or some other factor. In laboratory animals, exposure to high levels of TCE has damaged the central nervous system, immune system, liver and kidneys, and adversely affected reproduction and development of offspring (NYS DOH, 2006a). Taken together, the human and animal studies indicate that human exposure to high levels of TCE causes effects on the nervous system, and suggest that human exposure to high levels of TCE may increase the risk for immune and developmental health effects. A more detailed discussion of the studies of TCE health outcomes in humans is found in Appendix C of this document.

Risk Characterization

We evaluated the health risks of TCE exposure for people who worked or attended school at PS 51X since the TCE air levels at the school exceeded background levels, the NYS DOH air guideline and health-based comparison values. Adults and children were exposed to TCE in indoor air for different lengths of time depending on whether they were faculty, staff, or students, and exposure to TCE was intermittent since only a portion of the day is spent in school and school is not attended on weekends and during summer months. We discuss the results of assuming longer periods of exposure to account for after school or summer school programs in Appendix D, Comment # 7. Therefore, for our characterization of cancer and noncancer risks for effects on the central nervous and immune systems, we assumed that faculty, staff, and students were exposed for a portion of the day and year as summarized in Table 2. For evaluating cancer risks, we assumed faculty/staff, and students were exposed for 18 and 6 years, respectively. These assumptions are based on information obtained for PS 51X (Mulgrew, 2011) and assumptions used by the EPA for schools (EPA, 2011b). We used different exposure assumptions (see below) for our characterization of the noncancer risk for developmental effects.

Table 2. Exposure Assumptions for Faculty, Staff, and Students at PS 51X.

Group	Hours per Day*	Days per Year*	Years**
Faculty/Staff	8	185	18
Students	6.5	180	6

* EPA, 2011b; school year, not including weekend and summer vacations.
** Mulgrew, 2011.

1. Cancer Risk Characterization

We calculated the estimated increased risk of developing cancer, above the background lifetime cancer rate for humans, for faculty, staff, and students at the school using the exposure estimates and the EPA's inhalation unit risk value for TCE (4.1×10^{-6} per mcg/m^3; EPA, 2011a). The inhalation unit risk value is a numerical estimate of the carcinogenic strength of a chemical. The calculated increased cancer risk for long-term

exposure (6 years for students and 18 years for faculty and staff) to the highest (53 mcg/m^3) and average (33.5 mcg/m^3) TCE levels detected in the indoor air of the hallway and cafeteria ranges from 2 to 9 extra cases of cancer for every 1 million persons exposed. We consider this increase in cancer risk to be low. An example of calculations used in our evaluation of cancer risks is presented in the footnote at the bottom of this page (adults)[3] and in Appendix A, Table 2 (children).

2. Noncancer Risk Characterization for Nervous System Effects

To evaluate the noncancer risk for effects on the nervous system, we compared the exposure estimates for faculty, staff, and students at PS 51X to the NYS DOH reference concentration for TCE (10 mcg/m^3; NYS DOH, 2006a). A reference concentration is the concentration of a contaminant in air that is not expected to result in adverse noncancer health effects, assuming up to a lifetime of exposure. We based our reference concentration for TCE on central nervous system effects (reduced scores on motor coordination tests) reported in an occupational study of workers exposed to TCE by inhalation for an average of seven years. Since the exposure estimates (7.1 and 9.0 mcg/m^3 for children and adults, respectively), assuming exposure occurs for a portion of the day and year as summarized in Table 2, do not exceed the NYS DOH reference concentration for TCE, the risk for noncancer health effects on the nervous system for faculty, staff and students at PS 51X is minimal. Calculations used in our evaluation of the risk for noncancer effects on the nervous system is presented in the footnote at the bottom of this page[4].

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[3]Sample cancer risk calculation for adults:

$$\text{Cancer Risk} = 53\ \text{mcg/m}^3 \times \frac{8\ \text{hours}}{24\ \text{hours}} \times \frac{185\ \text{days}}{365\ \text{days}} \times \frac{18\ \text{years}}{70\ \text{years}} \times \frac{4.1 \times 10^{-6}}{\text{mcg/m}^3} = 9.4 \times 10^{-6}\ \text{(or about 9 in one million [low])}$$

[4]Noncancer risk calculation for students (central nervous system effects):

$$\text{Time-weighted Air Concentration} = 53\ \text{mcg/m}^3 \times \frac{6.5\ \text{hours}}{24\ \text{hours}} \times \frac{180\ \text{days}}{365\ \text{days}} = 7.1\ \text{mcg/m}^3$$

$$\text{Hazard Quotient} = \frac{\text{school concentration}}{\text{reference concentration}} = \frac{7.1\ \text{mcg/m}^3}{10\ \text{mcg/m}^3} = 0.7\ \text{(minimal)}$$

Noncancer risk calculation for adults (central nervous system effects):

$$\text{Time-weighted Air Concentration} = 53\ \text{mcg/m}^3 \times \frac{8\ \text{hours}}{24\ \text{hours}} \times \frac{185\ \text{days}}{365\ \text{days}} = 9.0\ \text{mcg/m}^3$$

$$\text{Hazard Quotient} = \frac{\text{school concentration}}{\text{reference concentration}} = \frac{9.0\ \text{mcg/m}^3}{10\ \text{mcg/m}^3} = 0.9\ \text{(minimal)}$$

3. Noncancer Risk Characterization for Developmental and Immune Effects

To evaluate the noncancer risk for developmental and immune system effects, we compared the exposure estimates to the EPA reference concentration[5] for TCE (2 mcg/m^3). The EPA derived its TCE reference concentration based on two studies of laboratory animals exposed to TCE in drinking water (EPA, 2011a). One study reported an increase in fetal heart defects in the offspring of exposed female rats and the other reported a decrease in thymus weights in exposed adult mice. The EPA and ATSDR use this reference concentration to evaluate the risks for developmental toxicity (fetal heart defects) and immune toxicity (decreases in thymus weights) for people exposed to elevated levels of TCE. The EPA used models of the metabolism of TCE in animals and humans to calculate internal doses[6] corresponding to TCE oral doses from drinking water and then used the same models to convert those internal doses to corresponding air concentrations. This enabled EPA to estimate the reference concentration from studies based on oral exposures.

As part of its reference concentration derivation, the EPA calculated the TCE air concentration (21 mcg/m^3) at which there is a 1% likelihood (chance) that a randomly selected person would have an internal dose higher than the 95% lower confidence bound on the rat internal dose corresponding to a 1% increased risk for fetal heart defects. This air concentration (called a "point-of-departure"[7]) was divided by a total uncertainty factor of 10 to obtain the reference concentration of 2 mcg/m^3. The EPA used an uncertainty factor of 10 to account for the possibility that humans may be more sensitive than rats to the same internal dose and for the possibility that some humans may be more sensitive than others to the same internal dose. The EPA did not use an uncertainty factor to account for the use of a lowest-observed-adverse-effect level (LOAEL) because the point of departure was a 95% lower bound on a dose associated with a 1% increased risk. Both the point of departure and the reference concentration are estimated values and are well below levels that have been evaluated in any human study of TCE toxicity.

We evaluated the risk for developmental effects assuming women at PS 51X are exposed for 8 hours per day and 5 days per week because these effects can occur after relatively short periods of exposure (e.g., early pregnancy). In other words, we did not, as we did when characterizing the risks for central nervous and immune systems, time weight the highest measured TCE exposure level (53 mcg/m^3) for the fraction of the year not exposed (i.e., summer vacation). This increases the TCE exposure level used in the risk characterization of developmental effects. The exposure to TCE estimated in this manner (12.6 mcg/m^3) is about 6.3 times higher than the EPA reference

[5]The Agency for Toxic Substances and Disease Registry has adopted the EPA reference concentration as its chronic inhalation minimal risk level for TCE (ATSDR, 2013b).

[6] The internal dose is the amount of TCE in the blood or the amount of TCE metabolized in the body.

[7]The point of departure is a point on a dose-response curve for an effect of TCE that is within or near the range of experimental or observational data for the effect. It is the starting point for the extrapolation (using uncertainty factors) from the range of observation in human or animal studies to the human doses at or near the TCE reference concentration.

concentration (2 mcg/m^3). Exposure to these levels at PS 51X is therefore estimated to pose a moderate risk for developmental effects (fetal heart defects in infants born to faculty and staff who were pregnant while working at the school). Another way of evaluating the risk for developmental effects is to recognize that 12.6 mcg/m^3 is lower than the air concentration (21 mcg/m^3) that corresponds to roughly a 1% increased risk for fetal heart malformations in rats. An example of calculations used in our evaluation of the risk for noncancer developmental effects is presented in the footnote at the bottom of this page.[8]

In addition to developmental toxicity (fetal heart defects), EPA also based its reference concentration of 2 mcg/m^3 on immune system toxicity in animals. EPA first calculated the TCE point-of-departure air concentration in humans (190 mcg/m^3) at which there is a 1% chance that a randomly selected person would have an internal dose higher than the 95% lower confidence bound on the mouse internal dose that corresponds to a LOAEL for decreased thymus gland weights. This point-of-departure air concentration was divided by a total uncertainty factor of 100 to account for using a LOAEL instead of a no-effect-observed level to derive the reference concentration, the possibility that humans may be more sensitive than mice to the same internal dose, and the possibility that some humans may be more sensitive than other humans to the same internal dose.

To evaluate the noncancer risk for immune toxicity, we compare the estimated exposures to TCE at PS 51X (assuming faculty, staff, and students are exposed a portion of the day and year, as summarized in Table 2) to the EPA reference concentration (2 mcg/m^3). The estimated TCE exposures (7.1 and 9.0 mcg/m^3 for children and adults, respectively) are above the EPA reference concentration by 3.5- and 4.5-fold, respectively. However, these exposures are about 20- to 25-times lower than EPA's estimate of the air concentration that corresponds to exposures that cause immune toxicity in mice (190 mcg/m^3). Given this margin of exposure and the mild nature of the immune effects in exposed mice, the exposure to TCE for children and adults (7.1 and 9.0 mcg/m^3, respectively) is estimated to pose a low risk for effects on the immune system. An example of calculations used in our evaluation of the risk for noncancer immune effects is presented in the footnote at the bottom of this page[9].

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[8]Sample noncancer risk calculation for women (developmental effects to the fetus):

$$\text{Time-weighted Air Concentration} = 53\ \text{mcg/m}^3 \times \frac{8\ \text{hours}}{24\ \text{hours}} \times \frac{5\ \text{days}}{7\ \text{days}} = 12.6\ \text{mcg/m}^3$$

$$\text{Hazard Quotient} = \frac{\text{school concentration}}{\text{reference concentration}} = \frac{12.6\ \text{mcg/m}^3}{2\ \text{mcg/m}^3} = 6.3\ \text{(moderate)}$$

[9] Sample noncancer risk calculation for children (immune effects):

$$\text{Time-weighted Air Concentration} = 53\ \text{mcg/m}^3 \times \frac{6.5\ \text{hours}}{24\ \text{hours}} \times \frac{180\ \text{days}}{365\ \text{days}} = 7.1\ \text{mcg/m}^3$$

$$\text{Hazard Quotient} = \frac{\text{school concentration}}{\text{reference concentration}} = \frac{7.1\ \text{mcg/m}^3}{2\ \text{mcg/m}^3} = 3.5\ \text{(low)}$$

Following release of the public comment draft version of this health consultation, we received information that children may have attended after-school and summer-school programs at PS 51X. Assuming longer periods of exposure to account for after-school or summer-school programs results in slightly higher quantitative estimates of cancer and noncancer risk. The qualitative descriptors for cancer risk and for developmental toxicity are unchanged (low and moderate, respectively). The risk descriptor for nervous system toxicity changes from minimal to low, and the descriptor for immune system toxicity changes from low to moderate. Thus, children who attended these after-school or summer programs and may have had additional exposure to TCE were at slightly greater risk for the previously described health effects than children who did not. These changes in the risk characterization for past exposures at PS 51X do not change the public health action that was already taken, specifically, to relocate the school to eliminate the exposure of faculty, staff, and students to elevated levels of TCE.

A significant uncertainty in our risk characterization of cancer and noncancer health effects is that due to limited indoor air sampling data, we do not know for how long or to what levels people were actually exposed to prior to the time that TCE was detected in PS 51X indoor air. Another significant uncertainty in the noncancer risk characterization is the ability of the models used in the derivation of the EPA reference concentration to accurately predict air concentrations for humans that correspond to effect levels in studies where animals were exposed to TCE in drinking water.

C. Child Health Considerations

ATSDR and NYS DOH consider children when evaluating exposure pathways and potential health effects for environmental contaminants. Children are of special concern because their behavior patterns, play activities and physiological differences can result in more exposure than adults. Children sometimes differ from adults in their sensitivity to the effects of chemicals, but this depends on the chemical. Whether or not there is a difference can also change as the child gets older.

We considered the possibility that children may be more sensitive to the health effects of TCE when we evaluated the indoor air sampling results for PS 51X. The EPA recently revised its estimate of carcinogenic strength (potency) of TCE, as well as its guidance for evaluating TCE cancer risks for children (EPA, 2011a,c). We incorporated these revisions into our cancer risk evaluation of TCE air levels at PS 51X. TCE is identified by the EPA as a chemical that causes cancer by a mutagenic mode-of-action (EPA, 2005; 2006; 2011a,c). A mutation is a permanent change in the DNA (deoxyribonucleic acid, the main component of chromosomes and material that transfers genetic characteristics) sequence of a gene. A carcinogenic chemical with a mutagenic mode of action causes cancer by interacting with DNA and inducing permanent, heritable mutations early in the carcinogenic process. In other words, mutagenicity is an early and necessary event in the process of how such carcinogens cause cancer to develop. This is in contrast to other mode-of-actions where mutations

are acquired after other key events (e.g., cell damage and subsequent repair). Carcinogens with a mutagenic mode-of-action are considered to pose a higher risk for cancer if exposure occurs early in childhood compared to the risk from exposure during adulthood (EPA, 2005). Therefore, children may be more sensitive than adults to the carcinogenic effects of TCE. To account for this possible greater sensitivity, we followed the EPA guidance and increased our theoretical cancer risk calculations for TCE by a factor of three when we evaluated the risk to students at PS 51X (EPA, 2005; 2006; 2011a,c; see also Appendix A, Table 2). Taking into account this potential additional sensitivity, the theoretical increased risk for getting cancer is low for students exposed to the highest measured level of TCE in areas likely occupied by students at PS 51X.

D. Health Outcome Data

NYS DOH staff received information about health concerns from parents of students who attended PS 51X, the UFT and local elected officials. Parents and their representatives have emphasized that all former students and/or their parents should be notified about the history of potential exposures at the school. All the groups involved expressed concerns about potential health effects among the faculty, staff, and students.

NYS DOH considers a variety of factors when determining if a review of health outcome data is warranted, and if so, what type of review is appropriate and feasible. When an exposure has occurred and/or concerns have been raised about potential health effects of the exposure, NYS DOH staff considers whether a study of health outcomes is warranted, what types of health outcomes would be appropriate to study, what types of data and methods would be useful for assessing these outcomes, and whether such studies are feasible.

In some situations where relatively low-level exposures have occurred, NYS DOH has still proceeded with health outcome studies, despite the fact that, based on information from existing studies, the exposures are not expected to lead to detectable increases in health outcomes. These health studies were conducted in response to the large gaps in knowledge about human health effects and to assist in addressing the high level of concern among the exposed people.

In Appendix C, we briefly summarize findings from specific human epidemiology studies that evaluated health outcomes in TCE-exposed populations. Higher risks for some specific types of cancer and adverse birth outcomes have been suggested by such studies. NYS DOH researchers are able to study these types of outcomes because we have access to comprehensive and accurate statewide data for cancer and adverse birth outcomes diagnosed among NYS residents.

Regarding feasibility, we have learned from many years of conducting health outcome studies that the use of existing records from statewide databases is preferable to using questionnaires or other types of data collection tools that rely on individuals' active

participation. As a first step in conducting a valid and successful health outcome follow-up study for a population where the exposures occurred at a school, we would need complete lists of faculty, staff and students, with detailed specific information for each individual. Feasibility of the study would depend on whether these detailed listings are available.

For any type of follow-up study for the small numbers of adults and children exposed at PS 51X, a study is unlikely to support strong conclusions.

If former students, parents, faculty, and staff are interested in discussing the feasibility and usefulness of specific types of health outcome follow-up, NYS DOH is gathering additional input from the PS 51X community about their concerns, and are sharing additional information about the types of investigations we can conduct.

CONCLUSIONS

NYS DOH and ATSDR conclude that, currently, because faculty, staff, and students have been re-located out of the 3200 Jerome Avenue building, and the building has not been occupied at the time of the development of this report, elevated levels of TCE in indoor air of the former PS 51X are not expected to harm people's health. Re-location of the school was an effective measure to eliminate exposure of faculty, staff, and students to TCE in the indoor air of PS 51X.

However, TCE in the indoor air of PS 51X, while the school occupied 3200 Jerome Avenue, were at levels that could harm people's health (see Appendix B). Based on the limited available sampling data, past exposure to indoor air containing TCE is estimated to pose a low increased risk for cancer (i.e., between 2 to 9 extra cases of cancer for every 1 million persons exposed) among faculty, staff, and students who attended the school. Past exposure to TCE in the indoor air of PS 51X is also estimated to pose a minimal risk for noncancer central nervous system effects and a low risk for immune system effects among faculty, staff, and students who attended the school. A moderate risk is estimated for developmental toxicity (fetal heart defects) for the children born to faculty and staff who were pregnant while working at PS 51X.

Information indicates that some children may have attended after school and summer school programs at PS 51X. Assuming longer periods of exposure to account for after school or summer school programs results in slightly higher quantitative estimates of cancer and noncancer risk. The qualitative descriptors for cancer risk and for developmental toxicity are unchanged (low and moderate, respectively). The risk descriptor for nervous system toxicity changes from minimal to low, and the descriptor for immune system toxicity changes from low to moderate. These changes in the risk characterization for past exposures at PS 51X do not change the public health action that was already taken, specifically, to relocate the school to eliminate the exposure of faculty, staff, and students to elevated levels of TCE.

NYS DOH and ATSDR recognize that current knowledge about potential health effects that might be associated with these past exposures is based on incomplete information.

While many scientific studies of TCE exposure in animals and humans exist, the health risks associated with exposure to environmental levels of TCE are not completely understood. We also do not know how long or to what levels people were actually exposed to prior to the time that TCE was detected in PS 51X indoor air. Overall, the conclusions NYS DOH made about whether people's health has been harmed are stated, as above, as expectations, and a degree of uncertainty remains. NYS DOH and ATSDR recognize the remaining uncertainty affects the people involved and also raises questions for the scientific community.

RECOMMENDATIONS

NYS DOH and ATSDR recommend that the building that formerly housed PS 51X not be reoccupied until remedial measures have been taken to reduce potential exposures to future occupants.

PUBLIC HEALTH ACTION PLAN

Completed

NYS DOH and ATSDR shared this health consultation with the people potentially affected by past exposures at PS 51X and with other government agencies and stakeholders. NYS DOH and ATSDR held a public meeting on July 1, 2013 to discuss the findings of the health consultation. Comments from the public and NYS DOH responses are presented in Appendix D.

Planned

NYS DOH and ATSDR will continue to seek input regarding interest in conducting follow-up health outcome studies and will provide additional information about the usefulness and feasibility of various follow-up study options. NYS DOH is planning additional meetings with the community.

The NYS DOH will prepare and distribute a packet of information targeted to health care providers. The packet will contain site-specific and TCE-specific information. The NYS DOH will make these packets available to students, parents, staff, and faculty from Public School 51X, so that they can share them with their health care provider. Alternatively, at the request of students, parents, staff and faculty, the NYS DOH will mail a package out to their health care provider. NYS DOH will place these materials on the NYS DOH website so that they can be accessed more readily by community members and health care providers. Also, NYS DOH will work with the Community Board to find additional ways to reach more community members in the distribution of this final health consultation.

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APPENDIX A

Table 1. Indoor Air Levels and Public Health Comparison Values for Trichloroethene (TCE) at the New York City PS 51X (the Bronx New School at 3200 Jerome Avenue) Building.
All values in micrograms per cubic meter (mcg/m^3)

Contaminant	Maximum Detection	Average Detection	Background[1]	New York State Air Guideline	ATSDR Comparison Values	
					Cancer[2]	Noncancer[3]
trichloroethene	53	33.5	< 1	5	0.24	2

[1] The NYS DOH reviewed and compiled information from studies in New York State as well as from homes and office buildings across the United States on typical levels of TCE in indoor air. Levels of TCE in the indoor air of homes and office settings are expected to be less than 1 mcg/m^3.
[2] The ATSDR cancer comparison value is the EPA estimate of the TCE air concentration corresponding to an increased lifetime cancer risk of one-in-one million (EPA, 2011a).
[3] The ATSDR noncancer comparison value is the EPA reference concentration (EPA, 2011a).

Table 2. Calculation[a] of Estimated Cancer Risk for Students Exposed to Trichloroethene (TCE) in Indoor Air at the New York City PS 51X (The Bronx New School at 3200 Jerome Avenue) Building.

Age Group	Air Concentration (mcg/m^3)	Duration (years)	Fraction of Lifetime	Unadjusted Kidney Unit Risk $(mcg/m^3)^{-1}$	ADAF	Adjusted Kidney Cancer Risk	NHL and Liver Unit Risk $(mcg/m^3)^{-1}$	NHL and Liver Cancer Risk	Total Cancer Risk: Adjusted Kidney and Unadjusted NHL and Liver
5 to <11 years	**7.1**	**6**	**0.0857**	**1×10^{-6}**	**3**	**1.8×10^{-6}**	**3.1×10^{-6}**	**1.9×10^{-6}**	**3.7×10^{-6}**

ADAF = age-dependent adjustment factor; NHL = non-Hodgkin lymphoma; mcg/m^3 = micrograms per cubic meter.

[a]We calculated the cancer risk estimates using the guidance provided in Section 5.2.3.3.1 of EPA (2011c), using age-dependent adjustment factors of 3 for ages 5 to <11 years. For students attending school from kindergarten to 5th grade, we first time-weighted the highest TCE air concentration of 53 mcg/m^3 to adjust for the intermittent nature of the exposure, assuming a child attends school for 6.5 hours a day and 180 days per year:

$$53\ mcg/m^3 \times \frac{6.5\ hours}{24\ hours} \times \frac{180\ days}{365\ days} = 7.1\ mcg/m^3$$

The estimated cancer risk is calculated as follows (EPA, 2011c):

$$7.1\ mcg/m^3 \times \frac{6\ years}{70\ years} \times \frac{1.0 \times 10^{-6}}{mcg/m^3} \times 3 + 7.1\ mcg/m^3 \times \frac{6\ years}{70\ years} \times \frac{3.1 \times 10^{-6}}{mcg/m^3} = 3.7 \times 10^{-6}$$ (or about 4 in one million [low])

APPENDIX B

Conclusion Categories and Hazard Statements

ATSDR has five distinct descriptive conclusion categories that convey the overall public health conclusion about a site or release, or some specific pathway by which the public may encounter site-related contamination. These defined categories help ensure a consistent approach in drawing conclusions across sites and assist the public health agencies in determining the type of follow-up actions that might be warranted. The conclusions are based on the information available to the author(s) at the time they are written.

1. Short-term Exposure, Acute Hazard "ATSDR concludes that...could harm people's health."

This category is used for sites where short-term exposures (e.g. < 1 yr) to hazardous substances or conditions could result in adverse health effects that require rapid public health intervention.

2. Long-term Exposure, Chronic Hazard: "ATSDR concludes that...could harm people's health."

This category is used for sites that pose a public health hazard due to the existence of long-term exposures (e.g. > 1 yr) to hazardous substance or conditions that could result in adverse health effects.

3. Lack of Data or Information: "ATSDR cannot currently conclude whether...could harm people's health."

This category is used for sites in which data are insufficient with regard to extent of exposure and/or toxicologic properties at estimated exposure levels to support a public health decision.

4. Exposure, No Harm Expected: "ATSDR concludes that ... is not expected to harm people's health."

This category is used for sites where human exposure to contaminated media may be occurring, may have occurred in the past and/or may occur in the future, but the exposure is not expected to cause any adverse health effects.

5. No Exposure, No Harm Expected: "ATSDR concludes that ...will not harm people's health."

This category is used for sites that, because of the absence of exposure, are not expected to cause any adverse health effects.

Appendix C: Human Epidemiology Studies Review

Almost all published human epidemiology studies are either of occupational groups exposed to relatively high levels of TCE and other chemicals through inhalation or of people exposed to relatively low levels of TCE in residential drinking water, which also results in exposure to TCE via inhalation of indoor air. The occupational studies of relatively high TCE exposures have shown increased risks for several types of cancer. The most consistent evidence has been for kidney, liver, and esophageal cancers and non-Hodgkin's lymphoma (Alexander *et al*., 2007; Charbotel *et al*., 2006; Siegel Scott and Chiu, 2006; Mandel *et al*., 2006; Zhao *et al*., 2005; Raaschou-Nielsen *et al*., 2003; Wartenberg and Siegel Scott, 2002; Wartenberg *et al*., 2000; Hansen *et al*., 2001; ATSDR, 1997; EPA, 2011). Additional evidence from occupational studies points to possible relationships between TCE exposure and increased risk of Hodgkin's disease, cervical cancer, multiple myeloma, bladder cancer, female breast cancer, and prostate cancer (Krishnadasan *et al*., 2007; Sung *et al*., 2007; Siegel Scott and Chiu, 2006; Zhao *et al*., 2005; Hansen *et al*., 2001; Wartenberg *et al*., 2000; ATSDR, 1997). Many of these studies have strong limitations including unknown exposure levels and small sample sizes. In addition, many of these studies were unable to adequately separate the effects of TCE from other solvents present in the workplace.

Community studies have shown an increased risk of certain cancers where the public drinking water was contaminated with TCE and other VOCs, with the strongest evidence for leukemia (Wartenberg *et al*., 2000). The Wartenburg review article also describes the evidence for possible associations of exposure to TCE in public drinking water supplies and non-Hodgkin's lymphoma and bladder cancer. These community studies also have strong limitations. They lack precise or individual-level information about exposures and they usually have very limited or no information on confounding factors such as smoking. In addition, public water supplies are often contaminated with a mixture of solvents making it difficult to determine whether an observed effect was due to TCE exposure, some other contaminant in the drinking water, or a combination of both. A study conducted by the NYS DOH of an area in the Village of Endicott affected by soil vapor intrusion of TCE showed elevated levels of two types of cancer, kidney and testicular (NYS DOH, 2006).

Some studies have shown associations between adverse reproductive and developmental health outcomes and exposure to TCE. Previous epidemiologic studies of women living in areas where the drinking water was contaminated with TCE or perchloroethylene (PCE, also known as tetrachloroethene) have suggested an increased risk of several types of birth defects. Studies in Arizona and New Jersey suggested an association between TCE contamination in public drinking water wells and cardiac defects, and the New Jersey study also found an increased risk of oral clefts and neural tube defects (NTDs) (Bove *et al*., 1995; Goldberg *et al*., 1990). The New Jersey study also included an analysis of women exposed to PCE in drinking water and found increased risks for oral clefts. In Woburn, Massachusetts, where public drinking wells were contaminated with TCE and to a lesser extent PCE, there was an unusually high number of infants born with choanal atresia, a defect of the nasal airway

(MDPH *et al*., 1996). One study in Milwaukee, Wisconsin, showed associations between potential inhalation exposures to TCE in outdoor air and an increased risk of certain congenital heart defects in infants born to older women (Yauck *et al*., 2004).

Studies of women exposed to TCE-contaminated drinking water have shown some evidence of increased risks of low or very low birth weight, term low birth weight, and small for gestational age (ATSDR, 1998; Bove *et al*., 1995; MDPH *et al*., 1996; Rodenbeck *et al*., 2000). The NYS DOH study conducted in an area in the Village of Endicott affected by soil vapor intrusion of TCE showed elevated levels of low birth weight outcomes and congenital heart defects (NYS DOH, 2006; Forand *et al*., 2012).

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APPENDIX D

Summary of Public Comments and Responses

The NYS DOH prepared this summary to address comments and questions on the public comment draft of the Bronx Public School 51X Health Consultation. NYS DOH invited the public to review the draft during the public comment period, which ran from June 28, 2013 to August 9, 2013. NYS DOH held a public meeting to discuss and receive comments on the draft health consultation. The meeting was held on July 1, 2013 at the Parish Hall of the St. Phillip Neri Church, 3025 Grand Concourse Bronx. NYS DOH also received written comments. In response to these comments, we reworded some statements for clarity and brevity. If you have any questions about the summary, please contact the NYS DOH at (518) 402-7860.

Comment 1: Please explain possible effects on the nervous system that might result from TCE exposure.

Response: Exposure to high levels of TCE (much higher than measured in the school) for short periods of time can cause nervous system effects such as dizziness, headache, sleepiness, nausea, confusion, blurred vision and fatigue. Exposure to lower levels of TCE for long periods of time (for example, in workplace air, still higher than measured in the school) can cause many of these same effects and is also linked to reduced scores on tests evaluating motor coordination. The risk for nervous system effects depends on the actual concentration of TCE in air, and how often and how long people are exposed. The risk also depends on a person's individual characteristics, including inherited traits, sensitivity to chemicals, whether the person is young or elderly, and their general health. The short-term effects on the nervous system are often reversible, meaning that the effects diminish once the exposure is stopped.

Comment 2: The conclusions were misleading and some parents were under the impression that because the children's risk was "low" it means the child will never get "sick" but the information presented at the meeting indicates that the NYS DOH cannot predict health outcomes. The document should do a better job of stating that we are not predicting health outcomes and the level of risks are used more as guidance for actions that should be taken.

Response: The commenter is correct in that the health consultation cannot be used to predict health outcomes. A health consultation presents its findings in terms of risk based on exposure assumptions. The comment about the clarity of this point in the document is noted and we will include additional language about the limitations and scope of the health consultation in the revised version.

Comment 3: Please elaborate on the statement in the Child Health Considerations section of document that mentions that EPA's guidance says that TCE makes permanent changes to DNA.

Response: The Child Health Considerations section referred to EPA guidance on assessing cancer risks for environmental contaminants that cause cancer by a mutagenic mode of action. The EPA identified TCE as one of the chemicals that causes cancer in this way. A mutation is a permanent change in the DNA (deoxyribonucleic acid, the main component of chromosomes and material that transfers genetic characteristics) sequence of a gene. A carcinogenic chemical with a mutagenic mode of action causes cancer by interacting with DNA and inducing permanent, heritable mutations early in the carcinogenic process. In other words, mutagenicity is an early and necessary event in the process of how such carcinogens cause cancer to develop. This is in contrast to other mode-of-actions that do not damage DNA but rather promote the DNA-damaged cell through the cancer process. Carcinogens with a mutagenic mode-of-action are considered to pose a greater cancer risk to children than to adults because children have longer to live and thus there is a longer time for DNA-damaged cells to go through the cancer process. We therefore considered the possibility of greater child vulnerability to the cancer effects of TCE by following the EPA's guidance, which calls for increasing the cancer risk calculations by a factor of three when evaluating the risk for school-aged children.

Comment 4: Have any mental health issues such as depression, mood disorders or attention deficit hyperactivity disorder (ADHD) been connected to TCE exposure?

Response: We searched the scientific literature for studies about TCE exposure and mental health issues, and found one limited study which reported an association between residential TCE exposure in drinking water and higher scores on tests used to measure confusion, depression, and tension. Since the exposure information in this study is limited, we do not know if the test results were related to TCE or some other factor. This one study does not provide enough evidence to conclude that TCE exposure causes mental health issues or behavioral problems.

Comment 5: Does TCE cause breathing and or behavioral problems? Do the levels of TCE found at the school actually cause cancer?

Response: Exposure to TCE at environmental levels is unlikely to result in direct effects on the respiratory system that would cause breathing problems. We addressed the evidence on TCE and behavioral problems in the previous response. There is no way to know with certainty whether the levels of TCE found at the school will cause cancer. However, based on our TCE exposure estimates and the available information on the ability of TCE to cause cancer in animals, the health consultation concluded that the risk for getting cancer from past TCE exposure at PS 51X is low, meaning that we do not expect these effects to occur from past exposure.

Comment 6: My son had a benign growth removed 2½ years after attending this school for 6 years. How would I ever know there is a connection?

Response: Generally, we are unable to link the cause of a specific medical condition with a specific environmental agent such as TCE, because most medical conditions can have many or unknown causes, and because a person's past exposure to an environmental agent is usually very difficult to know.

Comment 7: The health consultation does not contain important information regarding the amount of time kids spent at school on a daily basis. Many kids attended a daily afterschool program until 6 pm. Some kids also attended summer school programs at PS 51X. The time spent at school was approximately 4 to 5 hours. Can the NYS DOH re-evaluate exposures using this site specific information?

Response: Assuming longer periods of exposure to account for after school or summer school programs (e.g., assuming 12 hours per day in school during the school year and 5 hours per day in school for two months during the summer) would result in slightly higher quantitative estimates of cancer and noncancer risk. The qualitative descriptors for cancer risk and for developmental toxicity would be unchanged (low and moderate, respectively). The risk descriptor for nervous system toxicity would change from minimal to low, and the descriptor for immune system toxicity would change from low to moderate. These changes in the risk characterization for past exposures at PS 51X have no bearing on the public health action that was already taken, specifically, to relocate the school to eliminate the exposure of faculty, staff, and students to elevated levels of TCE.

Comment 8: Your explanation of consultation conclusions was misleading because it was based on three samples in one year. Therefore, the graphical representation is not accurate!

Response: The uncertainties related to the limited nature of the sampling data were discussed in the Public Health Implications section of the health consultation:

> "A significant uncertainty in our risk characterization of cancer and noncancer health effects is that due to limited indoor air sampling data, we do not know for how long or to what levels people were actually exposed to prior to the time that TCE was detected in PS 51X indoor air."

We reiterated these uncertainties when we presented the conclusions of the health consultation at the public meeting. Our goal was to present the results and conclusions in the context of the uncertainties, including the limited number of indoor air samples.

Comment 9: Who is going to take responsibility? Does the State or City feel responsible since I presume they test the building to renew the lease? Who's responsible if my kids get sick? What if NYS DOH is sued? Does that make them responsible? Many people stated that it is NYC DOE's responsibility.

Response: The level of oversight that occurred in the past is outside the scope of this Health Consultation. Responsibility is an issue that the NYS DOH cannot address for any other organization. NYS DOH assesses potential and known exposures to contamination and recommends measures to minimize or prevent exposures. When the NYS DOH became aware of exposures at P.S. 51X, the NYS DOH worked closely with the involved agencies to take actions to reduce exposure to the TCE that was detected in the indoor air of the school.

Comment 10: The mailing lists used by NYS DOH need to be improved. Request that NYS DOH and NYC DOE work with the community to build a more comprehensive list of faculty, staff, parents, and alumni of PS 51X to make the mailing lists more accurate and up-to-date. Can you please detail the next steps you will be taking to ensure the list is built upon and information is distributed? We would also like data about their outreach to parents in terms of the number of families contacted, the number of letters that were returned, and the number of letters sent to the incorrect address. Attendees of the public meeting indicated that several parents have received letters that are addressed to a student that is not theirs, while receiving no letter for their student. To better reach people, NYS DOH should publish announcements and information in local papers. NYS DOH should collect information from local organizations like Northwest Bronx Community and Clergy Coalition (NWBCC), Community Board, etc.

Response: The NYC DOE mailed the meeting information for the NYS DOH and the NYS DOH received the mail that was returned by the post office as not deliverable. The NYS DOH organized the returns and retained the returned mail until it was all received. The majority of the returns did not include a forwarding address. NYS DOH returned these to the NYC DOE. NYS DOH will work with the Community Board to find additional ways to reach more community members in the distribution of future mailings.

The NYC DOE indicated to us that they notified over 2,000 PS 51X former faculty, staff, and students that appear in their records and is committed to reaching as many former faculty, staff, and students as possible. NYC DOE indicated that they will immediately update faculty, staff, and students' records upon notification of any address changes by the NYS DOH or PS 51X community members.

NYC DOE also indicated that they would support NYS DOH in sharing information with the community, including mailing notices for any additional meetings proposed or any other information NYS DOH wishes to share with the individuals identified by the NYC DOE database.

Comment 11: - The public wants to have another public meeting in the fall when school starts again. In the report, you indicated that a potential next step would be to hold a meeting with NYS DOH and NYC DOHMH to discuss the feasibility of further health investigations that could be conducted to learn more about the health implications of our children and staff's exposure. We would like to discuss these options. However, we would like to request that such a meeting be held in the early fall after school is in session again, when more families would be available to participate in the discussion. Will you commit to work with us to plan this meeting for the September or October?

Response: NYS DOH was agreeable to coming in the fall of 2013 to meet with the community about health outcome issues/concerns/needs, however, NYS DOH and the community were not able to schedule the meeting during this time. NYS DOH met with the community on April 24, 2014.

Comment 12: Parents have taken their kids to local doctors/pediatricians who aren't familiar with the issues or understand how environmental exposures may have impacted our kids. The burden shouldn't be on the parents to get the information to their doctors/pediatricians. The information should already be available, and be provided directly to the doctors/pediatrician.

Can NYS DOH send a report to these local doctors? Why do parents and doctors have to go to Mt. Sinai when there are medical services resources in their own areas? Mt. Sinai is too far. Can you send the final health consultation report out to all physicians in the Bronx via the City Health Information (CHI) or using other mailing/listserv capabilities?

Response: The NYS DOH will prepare and distribute a packet of information targeted to health care providers. The packet will contain site-specific and TCE-specific information. The NYS DOH will make these packets available to students, parents, staff and faculty from Public School 51X, so that they can share them with their health care provider. Alternatively, at the request of students, parents, staff and faculty, the NYS DOH will mail a package out to their health care provider. NYS DOH will place these materials on the NYS DOH website so that they can be accessed by community members and health care providers. Also, NYS DOH will work with the Community Board to find additional ways to reach more community members in the distribution of this final health consultation.

Comment 13: Several audience members want the agencies to explore the possibility of tracking health status of former students into adulthood to see if they develop illnesses associated with TCE exposures. Audience members noted that UFT teachers are getting medical monitoring done through the teachers union. Although NYS DOH noted that they are not set up to do medical monitoring, why can't our kids get the same medical monitoring services? Can the NYS DOH make recommendations to the

NYC DOE regarding follow-up with former faculty, staff, and students via a health registry? How are they going to keep track of health of who was exposed?

Response: NYS DOH held a public meeting on April 24, 2014 to discuss community questions and concerns related to health studies for faculty, staff, and students who were exposed to TCE in the former Bronx PS 51X school building. During this meeting, the NYS DOH addressed the types of questions that could and could not be answered by a health study of the PS 51X community, as well as what types of information would be required to conduct such a health study. At the conclusion of that meeting, NYS DOH agreed to look into the completeness and availability of information about the individuals who are a part of the PS 51X community and to evaluate the feasibility of conducting a health outcomes review. NYS DOH staff are available to answer questions about this meeting or these topics, and can be reached at (518) 402-7950.

Response: A meeting to discuss the possibility of a health study is currently being planned. If you are interested in participating in that meeting, please contact the NYS DOH at (518) 402-7950 to be placed on the mailing list for that meeting.

Comment 14: Do levels of TCE in the indoor air of the school that were recorded in 2011 have any relevance to levels in 1993, or what they will be in future? Are there soil vapor intrusion studies that show TCE levels diminish over time? Can't you find out? Does TCE level decrease over time, i.e., were levels likely higher in the past?

Response: We do not, and cannot, know what the levels of TCE were in the indoor air of the school before the air was sampled. Although we know that TCE is present in the soil and groundwater below the building, a variety of environmental factors could have affected the timing and extent of past TCE movement into the building.

Comment 15: What are the NYC DOE and NYC SCA's current policies around the testing of sites NYC DOE leases and notifying a school community of the results of an environmental investigation? We believe that the NYC DOE and NYC SCA's current policy regarding testing of leased sites poses a significant public health concern that creates unnecessary risk for students and staff of NYC DOE schools, like at PS 51X.

NWBCC is working to create legislation (via disclosure petition) to hold NYC DOE responsible and make them notify parents if environmental investigations are conducted at schools. NWBCC wants NYC DOE to do something different, and wants agencies in attendance NYS DOH, NYC DHMH, ATSDR, NYC DOE to support such legislation.

Response: NYS DOH does not have a position on these policies, but will continue to work with the NYS DOE to make sure that contamination issues are resolved and identified exposures reduced or eliminated.

The NYC DOE provided the following information for inclusion in this document:

The NYC SCA posts test results and communicates with the school community in accordance with the following posted protocols:
Lease Renewals:
http://www.nycsca.org/Community/Programs/EnvironmentalDueDiligence/Pages/LeaseRenewals.aspx
New Leases:
http://www.nycsca.org/Community/Programs/EnvironmentalDueDiligence/Pages/NewLeases.aspx

Comment 16: What happens next? What are former students and families to do now?

Response: NYS DOH is committed to continuing to work with the PS 51 community to help address the health concerns of former students, parents, and staff. Concerned individuals are encouraged to continue participating in the ongoing dialogue between NYS DOH and the PS 51 community. If you are interested in being placed on the mailing list to receive information about future meetings, please contact the NYS DOH at (518) 402-7950 to be placed on the mailing list for that meeting. NYSDOH staff are also available to help answer specific environmental health questions.