Letter Health Consultation

Evaluation of Indoor Air TCE Concentration after
Activation of Mitigation System for Businesses located at the

CHEM FAB SITE

DOYLESTOWN, BUCKS COUNTY, PENNSYLVANIA

Prepared by
Pennsylvania Department of Health

AUGUST 30, 2013

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

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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pennsylvania
DEPARTMENT OF HEALTH

To: Eduardo Rovira, On Scene Coordinator, US Environmental Protection Agency (EPA) Region 3

From: Farhad Ahmed, Health Assessment Program, Division of Environmental Health Epidemiology

Subject: Evaluation of Indoor Air TCE Concentrations after Activation of Mitigation System for Businesses Located at the Chem Fab Site

Date: August 27, 2013

This letter is in response to EPA's request to evaluate air sample results collected after the TCE mitigation system was activated. The U.S. Environmental Protection Agency (EPA) has been conducting TCE vapor intrusion investigation and mitigation at the Chem Fab Superfund Site ("the Site") [1,2] located in Doylestown, Bucks County, Pennsylvania, since October 2011. In June 2012, ATSDR concluded TCE levels found in the main building, which contains several offices and businesses, posed a public health threat to employees of businesses, their patrons, or visitors. This document provides our evaluation of the indoor air sampling results for trichloroethylene (TCE) from August 2012, and compares the TCE concentrations before and after mitigation system activation to determine if the mitigation system is effectively reducing TCE concentrations to below those of health concern.

In July 2012, a vapor mitigation system, similar in design and function to a radon sub-slab mitigation system, was activated. The system was installed under the foundation of the east side of the building approximately six years ago (see map of main building and photo of mitigation system in Appendix). The mitigation system remained idle until approximately three weeks before the last round of indoor air sampling took place (August 2012). This Health Consultation evaluates the data from August 2012.

Environmental Data

Indoor Air. In August 2012, EPA collected indoor air samples from the main building to determine whether TCE concentrations had fallen below levels of health concern following the installation of the mitigation system. Indoor air samples were collected using 24-hour Summa canisters. One or two Summa canisters were placed in each office. One ambient air sample was also collected. Whenever possible, Summa canisters were placed in the same spots as past sampling events. The main building is divided into several office/commercial spaces with

addresses: 300, 310, 314, 320-322, 324, 328, and 330. The table below summarizes TCE concentrations detected in indoor in the offices in the main building for the October 2011, January 2012, and August 2012 rounds of sampling.

Table 1: Indoor Air Sample Results (24-hr) for TCE from the Main Building, Chem Fab NPL site

		Sampling Date		
		Pre-Activation of Gas Mitigation System		Post-Activation of Gas Mitigation System
		October 2011	January 2012	August 2012
300-1	1st Floor-conf. room	NA*	NA*	6.1
300-2	1st Floor-file cab.	80.5/32.1	78	6
300-3	Ambient Air	2.4/1.4/ND	ND	0.31
310	1st Floor	44.7	25.0	6.0
310	2nd Floor	52.7	22.0	5.3
314-1	1st Floor-office	225.0	6.1	4.5
314-2	1st Floor-kitchen	NA*	NA*	3.7
320-322	1st Floor	26.4	18.0	15.0
324	1st Floor	18.3	NA*	15.0
328	1st Floor	29.4	27.0	17.1
330-1	1st Floor	44.7	44.0	30.0

All TCE concentrations in micrograms per cubic meter ($\mu g/m^3$)
*NA: No samples collected from these office spaces.
ND= non detect

The August 2012 sample results show a decrease of TCE concentrations in indoor air at all office/commercial space locations. The maximum indoor air concentration of TCE detected in the August sampling event was $30 \mu g/m^3$. Previous sampling events (pre-activation of gas mitigation system) showed a maximum indoor air concentration of TCE of $225 \mu g/m^3$.

The latest 24-hour indoor air results of TCE are still, however, above the ATSDR Chronic Oral MRL, which is equivalent to the EPA's RfC, of $2 \mu g/m^3$ at all office locations. The RfC is a concentration not likely to result in adverse health effects for a lifetime of exposure. Although the Occupational Safety and Health administration (OSHA) has a standard to evaluate worker exposures to TCE, ATSDR/EPA values are used in commercial/industrial settings where site-related contaminants are not used in the workplace.

To evaluate the potential health effects of TCE, PADOH calculated a range of exposure durations. Indoor air concentrations of TCE (for each office location) were adjusted for 10, 8, 4 and 2 hour exposure durations. The indoor air samples were collected over a 24-hour period,

mostly during non-operating hours. Workers typically are at the facility for eight to ten hours per day. The adjusted exposure durations also account for a part-time worker and visitors. Table 2 shows the adjusted exposure concentrations to TCE.

Table 2: Adjusted TCE exposure concentrations in Main Building, Chem Fab NPL site, pre-mitigation versus post-mitigation system activation.

Offices in main building	**Adjusted TCE exposure concentrations *before* mitigation system activated (Oct. 2011& Jan. 2012), in micrograms per cubic meter (µg/m^3)**						**Adjusted TCE exposure concentrations *after* mitigation system activated (Aug. 2012), in micrograms per cubic meter (µg/m^3)**					
	24-hr	10-hr	8-hr	4-hr	2-hr	1-hr	24-hr	10-hr	8-hr	4-hr	2-hr	1-hr
300	**80.5**	**32.0**	**17.7**	**9.2**	**4.6**	1.7	**6.1**	**2.0**	1.3	0.7	0.3	0.1
310	**52.7**	**18.0**	**12.0**	**6.0**	3.0	1.1	**6.0**	**2.0**	1.3	0.7	0.3	0.1
314	**225.0**	**77.0**	**49.5**	**25.7**	**12.8**	4.9	**4.5**	1.5	1.0	0.5	0.3	0.1
320-322	**26.4**	**8.8**	**5.7**	1.3	0.5	0.5	**15.0**	**5.1**	**3.3**	1.7	0.9	0.3
324	**18.3**	**6.1**	**3.9**	**2.0**	1.0	0.4	**15.0**	**5.1**	**3.3**	1.7	0.9	0.3
328	**29.4**	**9.9**	**6.2**	**3.2**	1.6	0.6	**17.0**	**5.7**	**3.6**	1.9	1.0	0.4
330	**44.7**	**15.0**	**9.8**	**5.0**	**2.5**	1.0	**30.0**	**10.2**	**6.6**	**3.4**	1.7	0.7

Bolded values are adjusted concentrations that exceed the EPA RfC of 2 µg/m^3
Calculations for adjusting exposure concentrations are in the Appendix

The Appendix contains charts that compare adjusted TCE concentrations for pre-mitigation system activation versus post-mitigation system activation. Chart 1 compares TCE concentrations pre-mitigation system activation versus post-mitigation system activation for 24-hour duration (worst-case scenario). Chart 2 compares adjusted TCE concentrations pre-mitigation system activation versus post-mitigation system activation for 8-hour duration (typical employee work day).

Furthermore, the TCE concentrations for offices on the west side of the main building (offices 320-322, 324, 328, 330) are 3 to 5 times higher when compared to the offices on the east side (300, 310, 314). The vapor mitigation system appears to be constructed under only one slab (see Appendix C for picture of vapor mitigation system). The possible effects for workers exposed to TCE at these levels are discussed further in the Public Health Implications section of this document.

Ambient Air. An ambient (outdoor) air sample was collected near office 300 in August 2012. TCE levels found in outdoor air are the result of releases to the atmosphere primarily from vapor degreasing operations, but releases can also occur at treatment and disposal facilities, water treatment facilities and landfills [2.] EPA data suggest that average ambient TCE mean concentrations (i.e., "background" concentrations of TCE) have remained fairly constant since 1999 to 2006 at about 0.3 µg/ m^3 (ranging from non-detect to 18.44 µg/ m^3) [3]. From 1985-1998 EPA also measured outdoor TCE levels across land settings such rural, suburban and urban, and the mean concentrations were 0.42 µg/ m^3, 1.26 µg/ m^3, and 1.61 µg/ m^3 respectively for each land setting. The Chem Fab site is classified as a suburban setting. The ambient air TCE concentration at the Chem Fab site was 0.31 µg/ m^3, within the mean TCE levels EPA has reported for 1999 to 2006, and below reported TCE levels for suburban areas.

Exposure Pathway

It should be noted that when the building was remodeled there was no central HVAC system installed for the building. Each office has its own HVAC system. These systems re-circulate office air; there is no exchange of outside air with indoor office air. The building is used for light commerce and is predominantly an office environment. The building was remodeled and converted to office space approximately ten years ago. Workers, including women of child-bearing age, could have been exposed to TCE in air for as long as 10 years. Dermal exposure to TCE in air is minimal, so the primary route of exposure is from breathing in TCE while in the building. The possible health implications for exposed workers and visitors are discussed in the following section.

Public Health Implications of TCE in Indoor Air

Background Information about TCE Toxicity

ATSDR recently adopted EPA's RfC as our chronic MRL and we use the cMRL for evaluating less than chronic exposures because of health endpoints. EPA identified two animal studies as the basis of the Reference Concentration (RfC) for noncancerous effects[4,5] . In these studies, where animals were exposed to TCE orally via drinking water, the most sensitive adverse effects involved the immune system and the developing fetus[4,5] . EPA used physiologically based pharmacokinetic (PBPK) modeling to convert the oral dose in animals to a human equivalent concentration (HEC) of TCE in air.

In one rat study, EPA used the lower confidence limit of the benchmark dose response ($BMDL_{01}$) to estimate the air concentration that would yield a 1% response rate for fetal cardiac malformations. The result of these transformations is an HEC_{99} of 21 µg/m^3. The HEC_{99} is the human exposure concentration for which there is a 99% likelihood that a randomly selected individual will have an internal dose less than or equal to, in this case, the $BMDL_{01}$. [5]
EPA also used a 30-week mouse study and identified decreased thymus weight as a lowest observed adverse effect level (LOAEL). PBPK modeling was used to derive 190 µg/m^3 as the $HEC_{99,\ LOAEL}$.

In addition to the two animal studies used to determine the RfC for TCE, EPA's Integrated Risk Information System (IRIS) has a supportive RfC, based on effects to the kidneys. A supporting candidate RfC of 3 µg/m3 for kidney impacts include toxic nephropathy and increased kidney weight, is based on HEC_{99} of 30 µg/m^3, derived from a chronic study using BMD modeling. However, there remains substantial uncertainty in the extrapolation of glutathione (GSH) conjugation from rodents to humans due to limitations in the available data. In addition, the candidate RfC based on PBPK modeling for toxic nephropathy had greater dose-response

uncertainty since extrapolation from high response rates (>60%). Therefore, toxic nephropathy is considered supportive but is not used as a principal basis for the RfC. [6]

To summarize, EPA predicts that:

- a small risk of fetal heart malformations exists for pregnant women exposed to TCE at or above 21 $\mu g/m^3$, and
- a small risk of decreased thymus weight exists for humans exposed to TCE at or above 190 $\mu g/m^3$.
- A small risk of for kidney impacts including toxic nephropathy and increased kidney weights for humans exposed to TCE at or above 30 $\mu g/m^3$. Kidney toxicity is considered supportive but is not used as a principal basis for the RfC, due to uncertainties in extrapolating from animal data to humans;
- The most sensitive observed adverse effects, which were used as the primary basis for the RfC, were those affecting the immune system and the developing fetus. The RfC of 2 $\mu g/m^3$ is based on route-to-route extrapolated results from oral studies for the critical effects of heart malformations (rats) and immunotoxicity (mice);

To derive the RfC of 2 $\mu g/m^3$, EPA used an uncertainty factor of 10 for the interspecies extrapolation of fetal heart malformations in rats and an uncertainty factor of 100 for decreased thymus weight in mice (10 fold for interspecies extrapolation and 10 fold for LOAEL).

Exposure to TCE is associated with a number of potential cancerous and non-cancerous health effects, including neurotoxicity, immunotoxicity, developmental, liver, kidney, and endocrine effects in animals. In experimental rodent studies, high doses of TCE administered to mice resulted in tumors of the lungs, liver, and testes. The effects reported at high levels include liver and kidney damage and changes in heartbeat. The levels at which these effects occur in humans are not well characterized. The human evidence of carcinogenicity from epidemiologic studies of TCE exposure is strong for non-Hodgkin lymphoma, but less convincing for kidney cancer, and more limited to liver and biliary tract cancer. [6]

TCE is characterized as "carcinogenic in humans by all routes of exposure" by the U.S. EPA, based on convincing evidence of a causal association between TCE exposure in humans and kidney cancer. The International Agency for Research on Cancer (IARC) has classified TCE as "probably carcinogenic to humans" (Group 2A). The National Toxicology Program determined that TCE is reasonably anticipated to be a human carcinogen.[7] Adverse noncancerous effects associated with TCE exposure by inhalation include hepatic, renal, neurological, immunological, reproductive, and developmental effects. [7]

There is uncertainty in drawing conclusions about the potential health impacts to TCE for workers and visitors. One of the uncertainties is that there are no suitable inhalation studies available, and the RfC is based on animal studies where exposure occurred through drinking water. PBPK modeling was used to convert an oral dose (mg/kg/day) in animals to a human equivalent concentration in air ($\mu g/m^3$), and a bench mark dose model was used to estimate the air concentration that equates to a 1% response rate for the fetal cardiac effects. The exposure

level associated with a 1% response rate is a model prediction and is below the level that has been evaluated in any experimental study or exposed human population.

Possible Non-cancer Health Effects

Unadjusted (24-hr) TCE Concentrations. TCE indoor air concentrations have decreased after the mitigation system was activated with a maximum concentration of 30 $\mu g/m^3$ (office 330) and a minimum of 0.31 $\mu g/m^3$ (office 300). However, the concentrations continue to exceed the EPA RfC of 2 $\mu g/m^3$ for all offices in the main building. The maximum concentration of 30 $\mu g/m^3$ in office 330 is also higher than the modeled concentration of 21 µg/m3 where EPA estimates a 1% response for fetal cardiac malformations.

The maximum, unadjusted, indoor air concentration of TCE (30 $\mu g/m^3$) from office 330 represents a worst-case scenario for TCE exposures currently at this site because workers are not present in their workplace for a 24-hour period. However, if female workers exposed to these levels of TCE for 24 hrs per day, they would be at risk of adverse health effects to a fetus, specifically the development of their babies' hearts. Additionally, exposures to all workers (both male and female) may put them at the increased risk for immune system impacts.

TCE levels found during the air sampling in October 2011 and January 2012, exceeded the modeled concentration of 190$\mu g/m^3$, where EPA estimates effects to the immune system, specifically a decrease in thymus weight and an increase in markers associated with autoimmune disease.

Adjusted TCE Concentrations. It is highly unlikely that workers or visitors will be exposed to the detected TCE levels for 24 hours per day. A more likely scenario is a worker exposed for 8-10 hours per day and visitors or part-time workers from 1-4 hours per day. The TCE concentrations are lower for all the offices when adjusted for these shorter exposure scenarios. No adjusted concentrations exceed any TCE effect levels, however, the west side of the building (offices 320-322, 324, 328, 330) still shows levels exceeding the RfC.

Adjusted exposure doses for TCE for workers exposed for 8-10 hours per day and visitors or part-time workers exposed from 1-4 hours per day in offices 300, 310, 314, 320-322, 324 and 328 are not expected to harm workers health. PADOH made this conclusion because the adjusted exposure doses for TCE are 4 times and at least 30 times lower respectively, than doses reported to show harmful effects (fetal heart malformations- HEC_{99} of 21 $\mu g/m^3$; immune effects- 190 $\mu g/m^3$) in experimental (animal) studies.

Exposure to TCE is a health concern for on-site female workers, particularly pregnant women who are exposed *for 10 hours or more in office 330.* The 10 hour adjusted concentration of TCE for indoor air in office 330 (10.2 $\mu g/m^3$) is closer to the effect level, which puts pregnant women at risk for adverse fetal outcomes (fetal heart defects) for even a three-week period of exposure, and to the possibility of kidney and immune effects.

PADOH concludes that exposure to the highest concentrations of TCE measured in indoor air samples collected in August 2012 is a health concern for all workers, especially for pregnant women, who work at 300- 330 North Broad Street, especially in the offices on the west side of the building. Continued actions should be taken to reduce or eliminate indoor TCE levels.

Possible Cancerous Effects for Workers

EPA has characterized TCE as "carcinogenic to human" by all routes of exposure and has assigned TCE an inhalation unit risk estimate (IUR) of 4×10^{-6} per $\mu g/m^3$. Using the maximum, unadjusted, 24 hour indoor air concentration of TCE (30 $\mu g/m^3$), the theoretical increased cancer risks for workers in the main building with an operating vapor mitigation system, based on a 10 year exposure, is 2×10^{-5}. These levels fall within the range of very low to low increased risk. [9] Using the adjusted 8-10 hour indoor air concentrations of TCE for the offices, the theoretical increased cancer risks for workers in the main building with an operating vapor mitigation system, based on a 10 year exposure, ranged from 1×10^{-6} to 6×10^{-6}. These levels fall within the range of very low to low increased risk.

Conclusions

Based on the August 2012 data, PADOH concludes that:

1. Indoor air concentrations of TCE remain a health concern for workers at office spaces 320-322, 324, 328, and 330. Exposed workers have a small increased risk of immune system impacts, and exposure to pregnant women could result in fetal heart malformations.
2. Indoor air concentrations of TCE in indoor air at office space 300, 310, 314 are not expected to harm workers' health.
3. Visitors and part-time workers are not likely to experience any adverse health effects to TCE anywhere in the building.

Recommendations

PADOH recommends that additional steps beyond the current vapor mitigation system should be taken to stop or further reduce exposure to TCE in the indoor air at office spaces 320-322, 324, 328, and 330 North Broad Street main building complex. Continued air sampling should be conducted to verify that these actions are effective in reducing TCE levels.

Future steps

1. PADOH will continue to review additional environmental sampling data as it becomes available.
2. PADOH remains available to respond to community health concerns, including participation on the community advisory group.
3. PADOH will distribute this health consultation to the community.

Note: *As of the release of this document EPA had taken additional actions to further reduce exposures to TCE. EPA installed portable air filtration units at office/commercial spaces 320-322, 328, and 330.EPA also conducted additional indoor air sampling to evaluate whether the portable units were working adequately. A preliminary review of the data indicates that the portable air filtration units have successfully lowered the concentration of TCE in indoor air where these units have been installed. Indoor air concentration of TCE decreased to concentrations lower than the ones evaluated in this document. PADOH will conduct an evaluation of these results in a separate document.*

References

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[1] US Environmental Protection Agency Region III, Colleen Walling, comp. Organic Data Validation Report of Summa air samples collected in October 2011 and January 2012 for the Chem Fab Site. 2012 Print

[2] Department of Health & Human Services, Agency for Toxic Substances and Disease Registry. Toxicological profile for Trichloroethylene (TCE). September 1997 http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=173&tid=30

[3] US Environmental Protection Agency. Toxicological review of Trichloroethylene (CAS No. 79-01-6). Chapter 2. September 2011. http://www.epa.gov/iris/toxreviews/0199tr/Chapter2_0199tr.pdf

[4] Johnson P, Goldberg S, Mays M, Dawson B. Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat. 2003. *Environ Health Perspective,* 111, 289-292

[5] Keil DE, Peden-Adams M M, Wallace S, Ruiz P, Gilkeson G S. Assessment of trichloroethylene (TCE) exposure in murine strains genetically-prone and non-prone to develop autoimmune disease. 2009. *J Environ Sci Health A Tox Hazard Subst Environ Eng*, 44, 443-453.

[6] US Environmental Protection Agency 2012. Trichloroethylene. Integrative Risk Information System (IRIS), US Environmental Protection Agency. http://www.epa.gov/iris/subst/0199.htm

[7] ATSDR Addendum to the Toxicological Profile for Trichloroethylene January 2013 http://www.atsdr.cdc.gov/ToxProfiles/tce_addendum.pdf (Pages 2 - 10)

Appendix – Figures, Charts and Calculations

Figure 1. Map of Offices, Main Building, Chem Fab Site

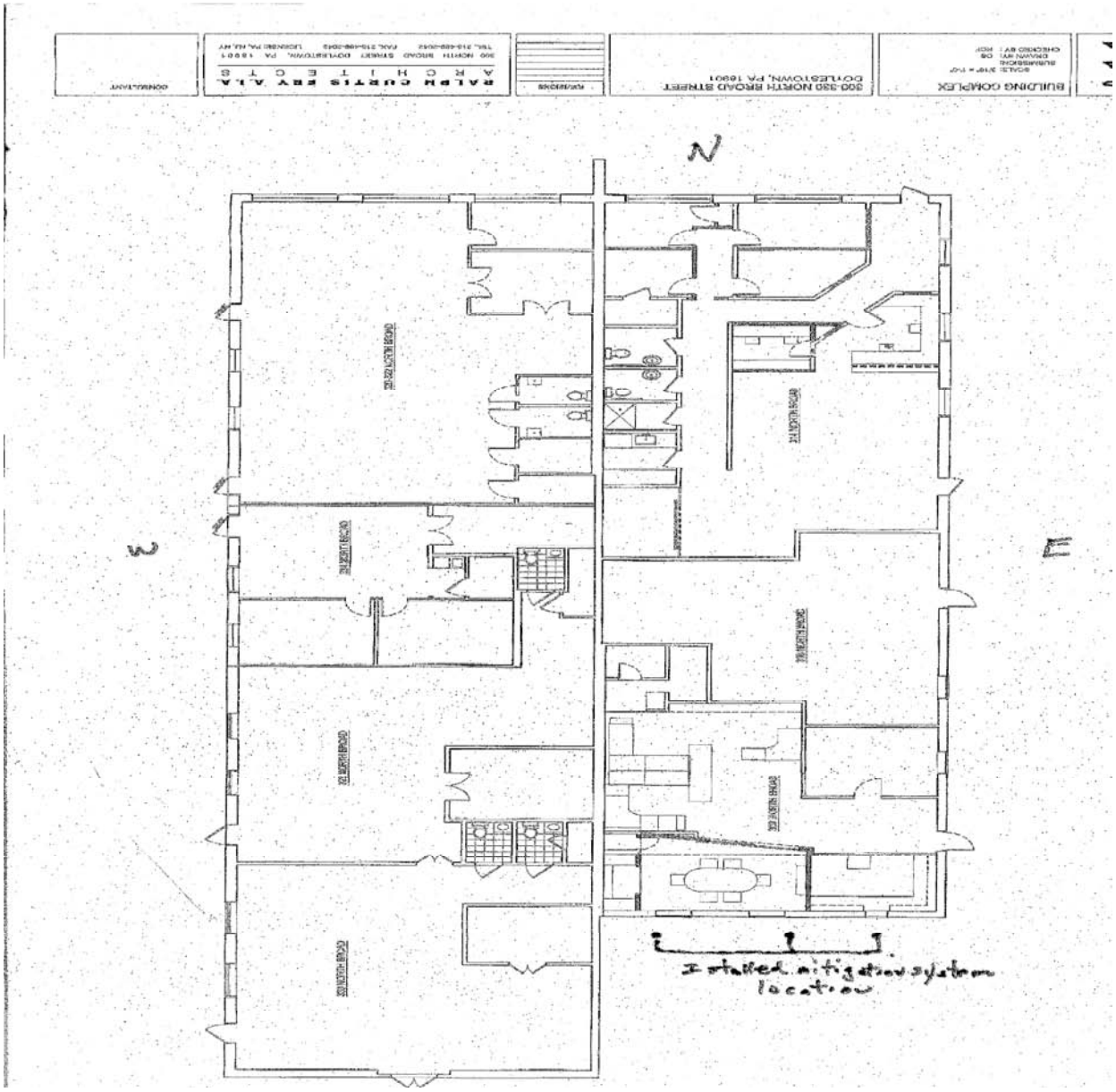


Figure 2. Picture of vapor mitigation system behind office 300, Chem Fab site

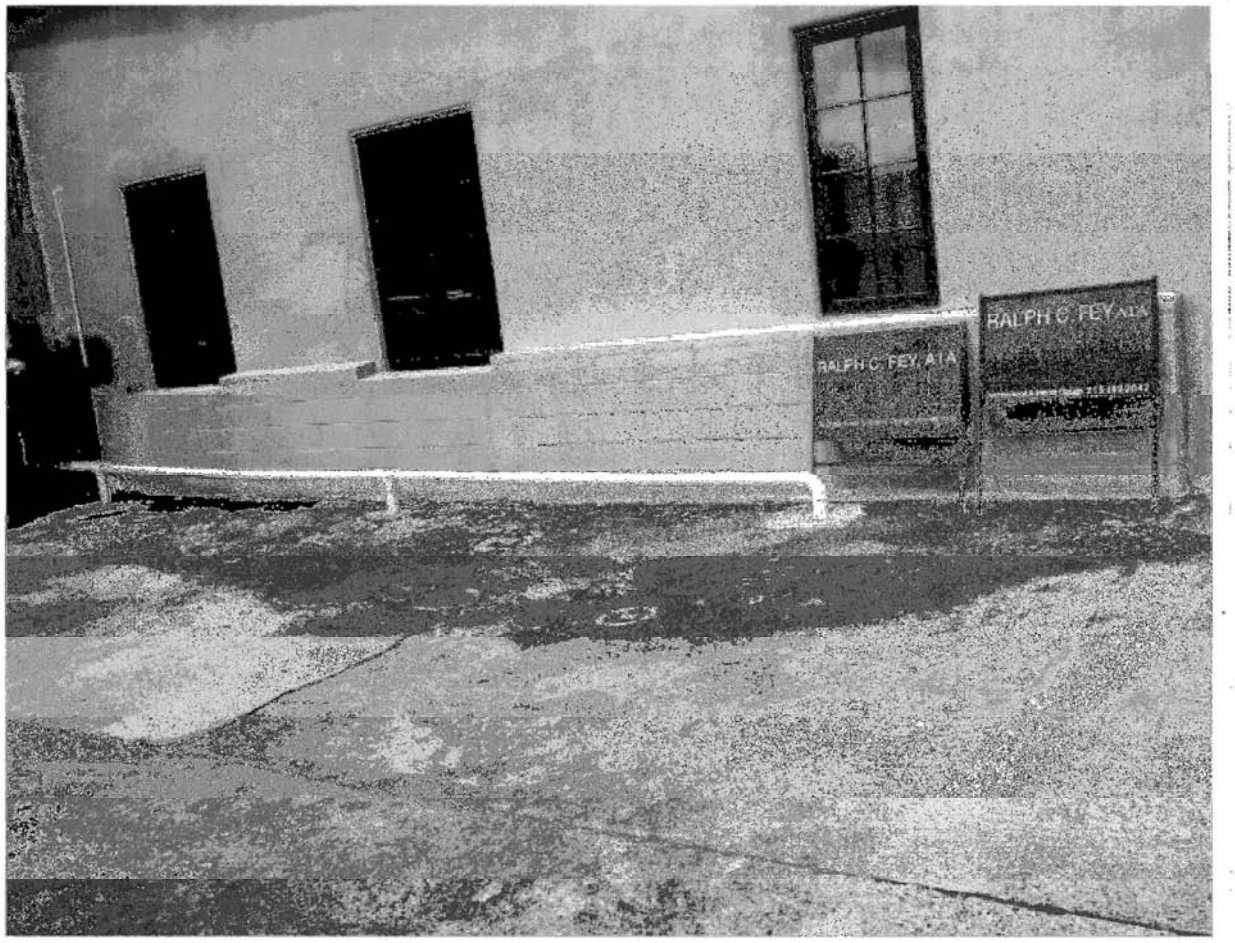


Chart 1

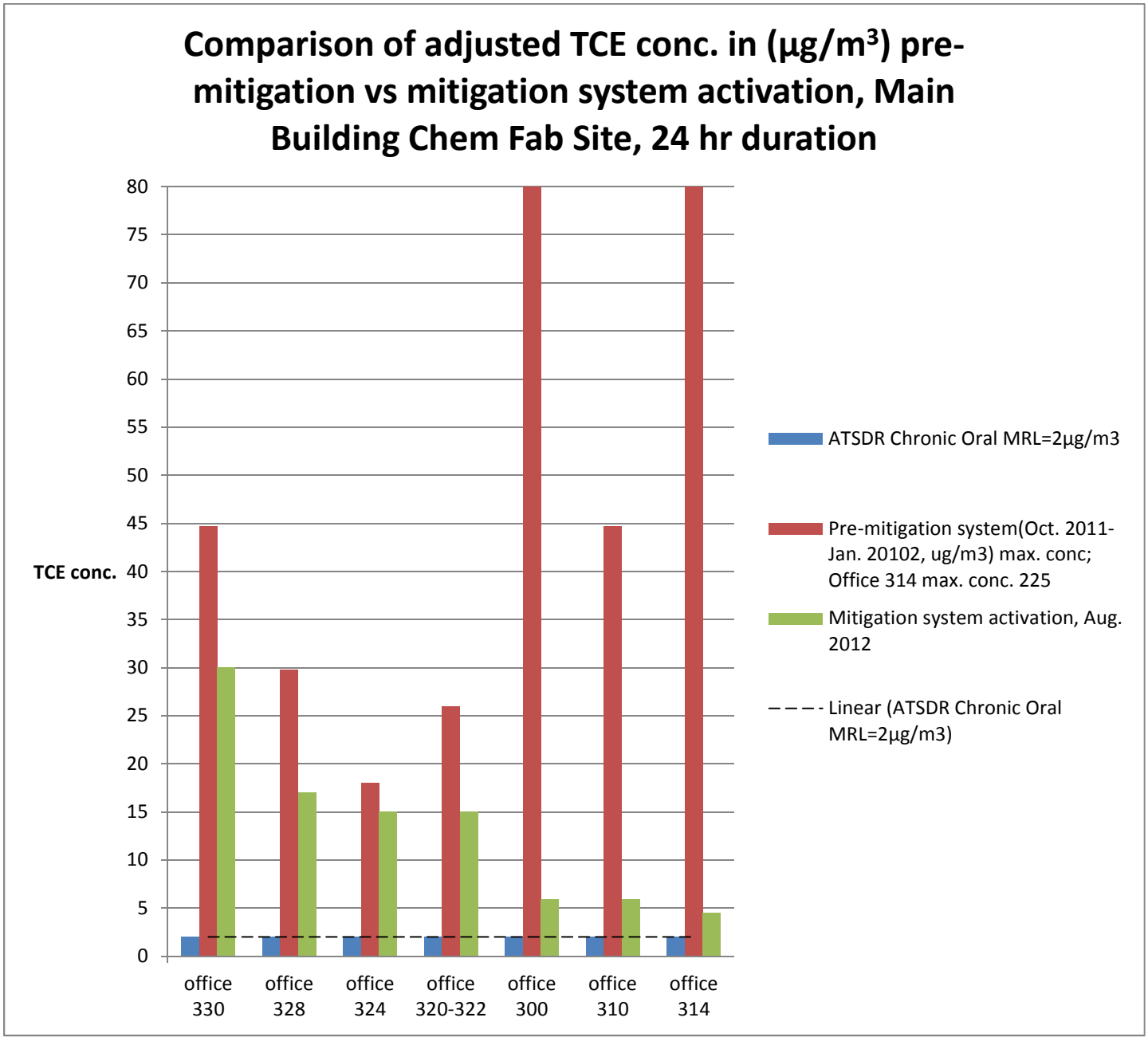
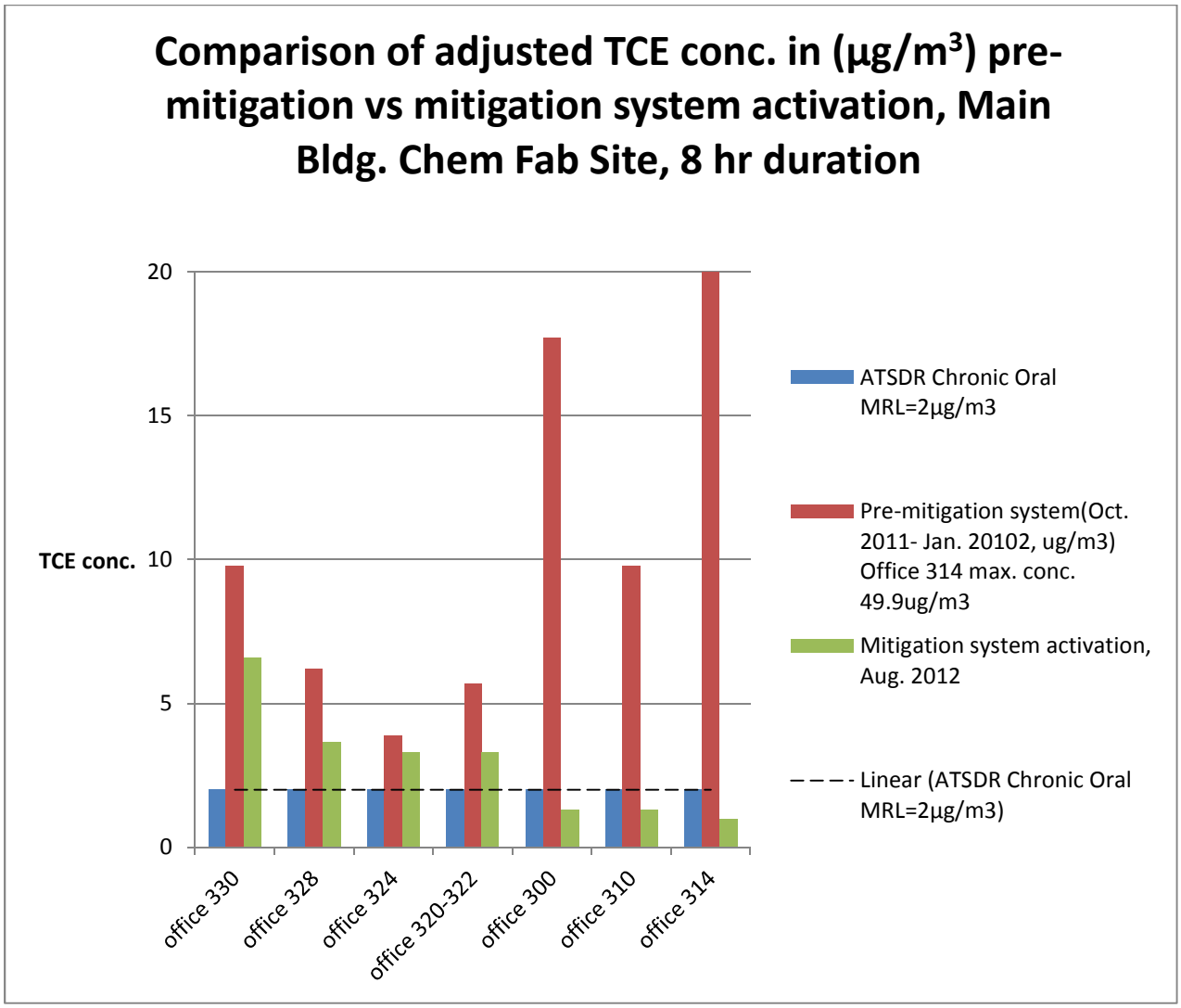

Comparison of adjusted TCE conc. in (µg/m³) pre-mitigation vs mitigation system activation, Main Building Chem Fab Site, 24 hr duration
TCE conc.
80
75
70
65
60
55
50
45
40
35
30
25
20
15
10
5
0
office 330
office 328
office 324
office 320-322
office 300
office 310
office 314
ATSDR Chronic Oral MRL=2µg/m3
Pre-mitigation system(Oct. 2011-Jan. 20102, ug/m3) max. conc; Office 314 max. conc. 225
Mitigation system activation, Aug. 2012
Linear (ATSDR Chronic Oral MRL=2µg/m3)

Chart 2


Comparison of adjusted TCE conc. in (µg/m³) pre-mitigation vs mitigation system activation, Main Bldg. Chem Fab Site, 8 hr duration
TCE conc.
20
15
10
5
0
office 330
office 328
office 324
office 320-322
office 300
office 310
office 314
ATSDR Chronic Oral MRL=2µg/m3
Pre-mitigation system(Oct. 2011- Jan. 20102, ug/m3) Office 314 max. conc. 49.9ug/m3
Mitigation system activation, Aug. 2012
Linear (ATSDR Chronic Oral MRL=2µg/m3)

Calculations

Exposure Calculations

EC = (CA x ET x EF x ED)/AT

Where:
EC ($\mu g/m^3$) = exposure concentration;
CA ($\mu g/m^3$) = contaminant concentration in air;
ET (hours/day) = exposure time;
EF (days/year) = exposure frequency;
ED (years) = exposure duration; and
AT (ED in years x 365 days/year x 24 hours/day) = averaging time

Note: If the duration of the exposure period is less than one year, the units in the above equation can be changed to the following: EF (days/week); ED (weeks/exposure period); and AT (hours/exposure period).

Adjusted Exposure Concentrations

10hrs- 10hours x 6days x 50 weeks/ 24 hrs x 7 days x 52 weeks=.34
8hrs- 8 hours x 5 days x 50 weeks/ 24 hr s x 7 days x 52 weeks= .22
4 hrs- 4 hr x 5 days x 50wks/ 24 hrs x 7days x 52wks= .114
2 hrs- 2 hr x 5 days x 50wks/ 24 hrs x 7days x 52wks= .057
1-hr- 1hr x 4days x 50 wks/ 24hrs x 7days x 52wks= .022

Cancer Risk Calculation

Risk = IUR x EC * (10yrs/70yrs) for 10 years of exposure

Where:
IUR ($\mu g/m^3$)-1 = Inhalation Unit Risk; and
EC ($\mu g/m^3$) = exposure concentration

.000004 * 30 $\mu g/m^3$ (max) * 10/70 = 2 x 10^{-5}