

Table 7. VOC sampling locations outside of Ground Zero.

Site Name	Street Location	Sampling Dates for Benzene	Number of Samples Taken
ORD Site A	W. Broadway & Park Place	Sept 22- Jan 3	22
ORD Site B	290 Broadway	Sept 25-Jan 3	14
ORD Site C	Broadway & Liberty	Sept 23-Oct 17	6
ORD Site C'	Cedar & Trinity	Nov 7-Nov 12	4
ORD Site K	Albany & West	Sept 23-Jan 3	24
	140 Broadway	Sept 28	6
	75 Park Place & Greenwich	Sept 28	8
	Albany & Washington	Sept 16	2
	Church & Vessey	Feb 13	2
	Greenwich & Liberty	Sept 16-Feb 23	15
	Liberty & Trinity	Sept 12-Sept 26	6
	Liberty & West	Sept 16-Oct 5	10
Loc A	Barclay & W Broadway	Sept 22-Sept 27	3
Loc B	Church & Dey	Sept 16-Oct 10	3
Loc C	Broadway & Liberty	Sept 27	1
Loc D	Albany & Greenwich	Sept 27	1
Loc E	Liberty & South End	Sept 27	1
Loc F	Vessey & West	Sept 16-Sept 27	2
Loc K	Albany & West	Sept 23-Sept 25	2
Loc N	Pier 25 (southside)	Oct 13	1
Loc P	Albany & South End	Sept 27	1

Table 7. VOC Sampling Locations Outside of Ground Zero (cont'd).

Site Name	Street Location	Sampling Dates for Benzene	Number of Samples Taken
Loc R	EPA TAGA Bus	Sept 23-Jan 1	11
Loc S	Rector Pl & South End	Sept 27	1
	Murray St & W Broadway	Oct 12-Oct 14	3
	Murray St betw West & N End	Nov 5-Mar 31	134
	Rockefeller Park	Sept 18-Nov 7	37
	Park Pl (225 Rector)	Sept 28	6
	Park Row & Spruce	Oct 14	1
Site 1 (NYSDEC)	Park Row	Oct 12-Oct 13	2
Site 16	290 Broadway	Sept 25	1
	130 West (Verizon Building)	Sept 29	1
	South of Building 4	Sept 25	1

Table 8. Locations that showed exceedences of screening benchmarks for VOCs and restrictions to access.

Location	Dates of Exceedence	Restrictions to Access
West Broadway and Park Place	Sept 22, Sept 25, Oct 3, Oct 9, Oct 19, Oct 20, Oct 21, Oct 24	in the restricted zone until Sept 27, was a border of the restricted zone until Oct 24
290 Broadway	Sept 25, Sept 26, Sept 27, Oct 11	in the restricted zone until Sept 19
Broadway and Liberty	Sept 23, Oct 8	in the restricted zone until Jan 28
Albany and West	Oct 1, Oct 20	was in the restricted zone until Oct 24, and became, and still is, a border of the restricted zone as of May 8
Liberty and Trinity	Sept 26	was in the restricted zone until Feb 12, and became, and still is, a border of the restricted zone as of May 8
Greenwich and Liberty	Sept 27, Sept 28, Oct 1	still in the restricted zone as of May 8

Table 10. Benzene grab sample exceedences and 24-hour sample monitoring summary.

I. Grab Samples			
Benzene Screening Benchmarks	Exceedence Concentration	Exceedence Location and Date	Restricted Zone
OSHA PEL 1 ppm ¹ OSHA STEL 5 ppm ¹	11 ppm	Liberty and Trinity Sept 26-	Yes
	19 ppm	Greenwich and Liberty Sept 28	Yes
	49 ppm	Greenwich and Liberty Sept 28	Yes
	1.3 ppm	Greenwich and Liberty Oct 1	Yes
ATSDR Acute MRL 0.05 ppm ²	0.1 ppm	Greenwich and Liberty Sept 27 samples noted above also exceed the ATSDR acute MRL	Yes
ATSDR Intermediate MRL 0.004 ppm ²	0.011 ppm	W. Broadway and Park Pl. Sept 22	Yes
	0.024 ppm	W. Broadway and Park Pl. Sept 25	Yes
	0.026 ppm	W. Broadway and Park Pl. Oct 3	Border
	0.007 ppm	W. Broadway and Park Pl. Oct 9	Border
	0.012 ppm	W. Broadway and Park Pl. Oct 19	Border
	0.008 ppm	W. Broadway and Park Pl. Oct 20	Border
	0.016 ppm	W. Broadway and Park Pl. Oct 21	Border
	0.008 ppm	W. Broadway and Park Pl. Oct 24	Border
	0.005 ppm	290 Broadway Sept 25	No
	0.007 ppm	290 Broadway, Sept 26	No
	0.0043 ppm	290 Broadway, Sept 27	No
	0.005 ppm	290 Broadway Oct 11	No
	0.021 ppm	Broadway and Liberty Sept 23	Yes
	0.007 ppm	Broadway and Liberty Oct 8	Yes
	0.024 ppm	Albany and West Sept 30	Yes
	0.008 ppm	Albany and West Oct 1	Yes
	0.008 ppm	Albany and West Oct 20	Yes
II. 24-Hour Samples			
Location		Concentration (ppm)	Date
Barclay and West Broadway		0.0025	Sep 27
Vesey and West		0.00081	Sep 27
Liberty and South End		<0.0007	Sep 27
Albany and South End		<0.0007	Sep 27
Rector and South End		<0.0007	Sep 27

Table 9. Acetone grab sample exceedences and 24-hour sample monitoring summary.

I. Grab Samples			
Acetone Screening Benchmarks	Exceedence concentration	Exceedence Location and Date	Restricted Zone
OSHA PEL 1000 ppm ¹		None	
ATSDR Acute MRL 26 ppm ²	29 ppm	Greenwich and Liberty Sept 28	Yes
ATSDR Intermediate MRL 13 ppm ²	22 ppm 20 ppm	Greenwich and Liberty Sept 28 Greenwich and Liberty Oct 1	Yes Yes
II. 24-Hour Samples			
Location		Concentration (ppm)	Date
Albany and Greenwich		0.0064	Sep 27
Albany and South End		0.0066	Sep 27
Barclay and West Broadway		0.0071	Sep 27
Church & Dey		0.0078	Sep 27
EPA Taga Bus		0.012 0.0056 0.0040 0.0044 0.0048	Sep 27 Sep 27 Dec 3 Dec 10 Dec 17
Liberty and Broadway		0.0053	Sep 27
Liberty and South End		0.010	Sep 27
Rector and South End		0.0069	Sep 27
Vesey and West		0.0045	Sep 27

¹NIOSH (2002)

²ATSDR (1994)

Table 10. Benzene grab sample exceedences and 24-hour sample monitoring summary (cont'd).

II. 24-Hour Samples		
Location	Concentration (ppm)	Date
Church and Dey	0.005	Sep 27
Liberty and Broadway	0.002	Sep 27
Albany and Greenwich	<0.0007	Sep 27
EPA Taga Lab	<0.0007	Sep 27
	<0.0007	Sep 27
	<0.0007	Dec 3
	0.0007	Dec 10
	0.0007	Dec 17

¹NIOSH (2002)

²ATSDR (1997)

Table 11. 1,3-Butadiene grab sample exceedences and 24-hour sample monitoring summary.

I. Grab Samples			
1,3-Butadiene Screening Benchmarks	Exceedence Concentration	Exceedence Location and Date	Restricted Zone
OSHA PEL 1 ppm ¹ OSHA STEL 5 ppm ¹	1.5 ppm	Greenwich and Liberty Oct 1	Yes
II. 24-Hour Samples			
Location		Concentration (ppm)	Date
Albany and Greenwich		<0.0028	Sep 27
Albany and South End		<0.0027	Sep 27
Barclay and West Broadway		<0.0027	Sep 27
Church & Dey		<0.0026	Sep 27
EPA Taga Bus		<0.0027 <0.0026 <0.0034 <0.0027 <0.0026	Sep 27 Sep 27 Dec 3 Dec 10 Dec 17
Liberty and Broadway		<0.0027	Sep 27
Liberty and South End		<0.0027	Sep 27
Rector and South End		<0.0027	Sep 27
Vesey and West		<0.0027	Sep 27

¹NIOSH (2002)

Table 12. Chloromethane grab sample exceedences monitoring summary.

Chloromethane Screening Benchmarks	Exceedence Concentration	Exceedence Location and Date	Restricted Zone
OSHA PEL 100 ppm ¹		None	
ATSDR Acute MRL 0.5 ppm ²	8.3 ppm 11 ppm 17 ppm 0.82 ppm	Greenwich and Liberty Sept 28 Greenwich and Liberty Sept 28 Greenwich and Liberty Oct 1 Liberty and Trinity, Sept 26	Yes Yes Yes Yes
ATSDR Intermediate MRL 0.2 ppm ²		same as above	
EPA-STSC Provisional Subchronic RfC 0.14 ppm ³		same as above	

¹NIOSH (2002)

²ATSDR (1998)

³EPA (1998)

Table 13. Ethylbenzene grab sample exceedences and 24-hour sample monitoring summary.

I. Grab Samples			
Ethylbenzene Screening Benchmarks	Exceedence Concentration	Exceedence Location and Date	Restricted Zone
OSHA PEL 100 ppm ¹		None	
ATSDR Intermediate MRL 1 ppm ²	4.0 ppm 4.7 ppm 1.7 ppm	Liberty and Greenwich Sep 28 Liberty and Greenwich Sep 28 Liberty and Greenwich Oct 1	Yes
EPA-STSC Provisional Subchronic RfC 0.23 ppm ³	0.4 ppm	same as above Liberty and Trinity Sept 26	Yes
II. 24-Hour Samples			
Location		Concentration (ppm)	Date
Albany and Greenwich		<0.0007	Sep 27
Albany and South End		<0.0007	Sep 27
Barclay and West Broadway		0.0011	Sep 27
Church & Dey		0.0022	Sep 27
EPA Taga Bus		<0.0007 <0.0007 <0.0009 <0.0007 <0.0007	Sep 27 Sep 27 Dec 3 Dec 10 Dec 17
Liberty and Broadway		0.001	Sep 27
Liberty and South End		<0.0007	Sep 27
Rector and South End		<0.0007	Sep 27
Vesey and West		<0.0007	Sep 27

¹NIOSH (2002)²ATSDR (1999)³EPA (1999a)

Table 14. Toluene grab sample exceedences and 24-hour sample monitoring summary.

I. Grab Samples			
Toluene Screening Benchmarks	Exceedence Concentration	Exceedence Location and Date	Restricted Zone
OSHA PEL 200 ppm ¹		None	
ATSDR Acute MRL 1 ppm ²	7.5 ppm 9.0 ppm 3.7 ppm 1.8 ppm	Greenwich and Liberty Sept 28 Greenwich and Liberty Sept 28 Greenwich and Liberty Oct 1 Liberty and Trinity Sept 26	Yes Yes Yes Yes
EPA-STSC Provisional Subchronic RfC 0.25 ppm ³		same as above	
II. 24-Hour Samples			
Location		Concentration (ppm)	Date
Albany and Greenwich		0.0014	Sep 27
Albany and South End		0.0019	Sep 27
Barclay and West Broadway		0.0020	Sep 27
Church & Dey		0.0033	Sep 27
EPA Taga Bus		0.0009 0.0007 0.0018 0.0017 0.0015	Sep 27 Sep 27 Dec 3 Dec 10 Dec 17
Liberty and Broadway		0.0019	Sep 27
Liberty and South End		0.0014	Sep 27
Rector and South End		0.001	Sep 27
Vesey and West		0.0015	Sep 27

¹NIOSH (2002)²ATSDR (2002b)³EPA (1999b)

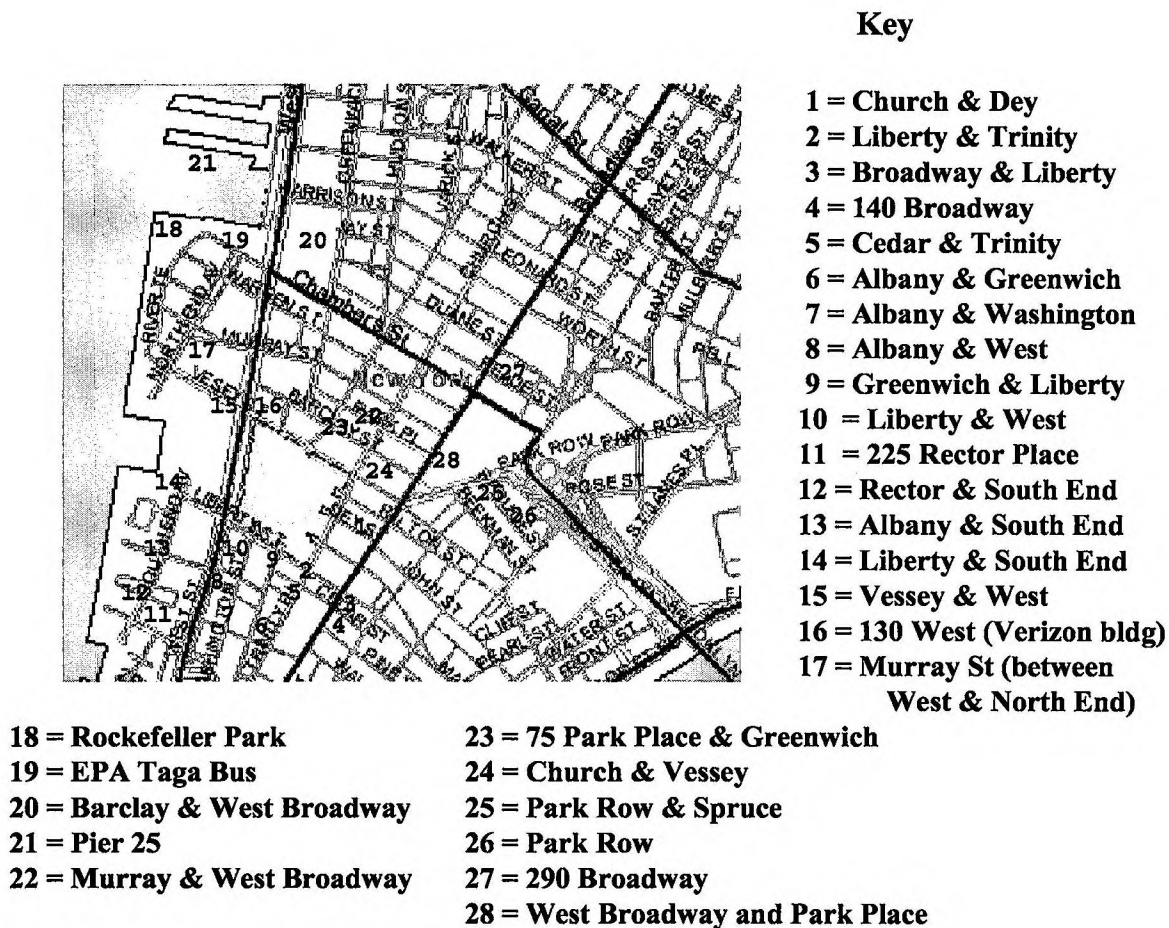


Figure 33. Location of VOC monitoring stations outside Ground Zero.

Section V. Comment on the First Several Days After September 11

An event such as September 11 demonstrates that the greatest environmental impacts occur in the first 24 to 48 hours and in areas close to the site. Difficulties associated with site access and security, power supply sources, equipment availability and analytical capacity hindered efforts by EPA and the New York State Department of Environmental Conservation (NYSDEC) to put air monitors in place immediately after the attack. Region 2 collected numerous samples of dust on September 11 and the next few days, and analyzed them for asbestos and lead. However, the first air samples of some of the critical contaminants from Ground Zero and nearby were not taken until September 14, such as asbestos, while other contaminants were not sampled until September 23, such as dioxin. Rapid initiation of monitoring will allow the measurement of air concentrations that can be very important for evaluation of inhalation exposures and potential short and long-term human health impacts.

Therefore, very little data are available to quantify exposures which could have occurred in the hours and days following the collapse of the WTC towers on September 11. As discussed in all of the individual contaminant sections, the general trend was that air concentrations were elevated in the earliest samples, and that concentrations appeared to return to background within weeks to a few months. The section on particulate matter (PM) went further by speculating on what the air concentrations of PM might have been in the initial plume cloud that occurred on September 11, based on an empirical relationship between visibility and PM concentration. The PM section also included discussions on the findings published by Liroy et al. (2002), who sampled dust which had settled onto cars and other surfaces, and had been undisturbed when their sampling occurred a few days after September 11. The USGS has similarly sampled and reported on measurements of contaminants in dust and debris (<http://speclab.cr.usgs.gov/wtc/>). Modeling studies within EPA to evaluate the movement of plumes in the few days after September 11 are ongoing (preliminary results are presented in the PM section), and these may shed light on exposures which could have occurred during these few critical days. Epidemiological studies may also elucidate information on exposures during the first few days after September 11.

It seems apparent that higher concentrations would have been found in the time frame of about September 11 to September 18 compared to the concentrations that were found when monitors did get in place. The earliest ambient monitoring data within Ground Zero and in the closest monitors are the asbestos sampling results which were measured first on September 14. Benzene and PCB measurements were reported for September 16. Lead was reported starting on September 18, and PM_{2.5} was reported first on September 21. The first measurements for dioxin-like compounds were not available until September 23.

Table 15 lists the first measurements on dioxin, asbestos, and lead in samplers within Ground Zero (the "WTC" sampler) and at locations bordering Ground Zero (e.g., Church & Dey). The data on that table supports the hypothesis that higher concentrations were likely to have been present within the first few days after September 11 as compared to when monitoring did begin. As seen, generally the highest concentrations were the very first ones available or within the first week or two of sampling. It is also noted that for dioxin and lead, the monitoring stations on South End Avenue (Liberty & South End, Albany & South End, and Rector & South

End) were showing very low background measurements on these first days of sampling at the same time the samplers within the plume - the WTC sampler and the Church & Dey sampler - were showing very high concentrations.

These data and similar data for other contaminants underscore the importance of being able to monitor very early after such an event. It is also recognized that a major uncertainty for the evaluations presented in this report is the lack of information on exposures which could have occurred within that first critical week after September 11.

Table 14. Summary of the first measurements of dioxin TEQs, asbestos, and lead at locations within or very near Ground Zero.

Contaminant	Concentration; Location; Sampling Date	Later Measurements and Other Comments
Dioxin TEQ, pg/m ³	160 WTC 9/23 170 WTC 10/2 170 WTC 10/4	All subsequent measurements were less than 100 pg TEQ/m ³ through 5/28/02. Note: Upwind monitors on South End (Liberty, Albany and Rector) all showed NDs on 9/23, indicating that elevations were tied to plume.
	130 Church/Dey 9/23	No further samples > 10 pg/m ³ ; samples through 5/17/02
	100 Liberty/Broadway 9/23	No further samples > 10 pg/m ³ ; samples through 10/26/01
Asbestos, S/mm ²	160 Barclay/W Broadway 9/14 ND Barclay/W Broadway 9/15 128 Barclay/W Broadway 9/15	All subsequent Barclay/W Broadway lower than 100 S/mm ² .
	80 Albany/West 9/22 89 Albany/West 9/23 178 Albany/West 9/27 71 Albany/West 9/30	Concentrations were less than 70 S/mm ² on Sep. 17, 18, 20, and 21, but no measurements above 70 after 9/30.
	48 Liberty/S. End 9/14 90 Liberty/S. End 9/15 (dup) 53 Liberty/S. End 9/15 80 Liberty/S. End 9/30	There were measurements between 9/15 and 9/30 that were less than 50 S/mm ² , but no measurements after 9/30 above 50 S/mm ²
Asbestos ^a , f/cc	0.1 Fulton/Church 9/13 0.078 Fulton/Church 9/26 0.059 Fulton/Church 9/28	All subsequent measurements after 9/28 were under 0.04 f/cc (through 8/03/02), but 6 samples between 9/15 and 9/19 were between 0.020 and 0.034, and samples on 10/1 and 10/3 were at 0.035 and 0.023, respectively.
	0.035 Vesey ^b 9/16 0.024 Vesey ^b 9/17	Next samples on 10/6-10/9 were between 0.008 and 0.023; no samples taken after 10/9.
	0.035 Fulton ^c 9/15 0.030 Fulton ^c 9/16	One sample on 9/14 was below LOQ; no samples taken after 9/16.
	0.020 Liberty/Church 9/14 0.021 Liberty/Church 9/18	One sample on 9/14 was below LOQ; no samples taken after 9/18.
Lead, µg/m ³	4.3 Barclay/W. Broadway 9/23 2.8 Barclay/W. Broadway 9/27	One sample > 1.0 µg/m ³ on 10/4, otherwise, all samples < 0.6 µg/m ³ ; samples through 2/5/02
	1.9 Church/Dey 9/18 1.7 Church/Dey 9/23	all other < 0.7 µg/m ³ ; samples through 2/5/02.
	5.4 WTC 9/23	next sample at WTC on 10/2 was 1.1, and one other 1.1 on 10/15, but otherwise all samples < 0.8 through 2/5/02. Similar to dioxin, all South End sampling locations had very low findings in initial 9/23 samples.

^a All of these samples taken by and reported by the NYCDEP.^b Actual location was described as, Vesey between Church and Broadway.^c Actual location was described as, Fulton between Church and Broadway.

Section VI. Data on Indoor and Occupational Exposures

This section provides a summary of data that are available, or that are being generated, from Ground Zero sites where rescue, clean-up and other workers may have been present, and on the indoor air environment. The occupational exposure data are related directly only to the workers on Ground Zero who were potentially exposed to contaminants generated during the course of their work. The indoor exposure data relate to residents in buildings off of Ground Zero whose exposure is from contaminated air that may have infiltrated their living or working spaces during or at some point after the disaster. It is emphasized that this section does not provide any human health risk evaluations in the way that ambient air data were evaluated in Section IV, except when summarizing conclusions of the original authors of the data.

VI.a. Data From Ground Zero Relating to Occupational Exposures

The Occupational Safety and Health Agency (OSHA) and the National Institute for Occupational Safety and Health (NIOSH) have generated data sets on air quality at Ground Zero. These data sets are summarized below. Further information on the data and the evaluation of these data can be obtained through the web sites that are identified below.

As part of the evaluations contained in this report, exposures to PCBs and dioxin toxic equivalent (TEQ) concentrations that were measured at Ground Zero were evaluated for on-site workers, which could include workers involved in rescue and clean-up operations. That evaluation is summarized here, with reference to the more detailed evaluations in Sections IV.

VI.a.1. OSHA Data

The OSHA data are posted on their Ground Zero monitoring web-site, at <http://www.osha.gov/nyc-disaster/summary.html>. Further information on these data can be obtained from OSHA, with contacts provided at, <http://www.osha.gov>. No interpretative analysis of the OSHA data, other than an identification of exceedences of benchmarks, is provided on the web site or from other available sources.

A total of 1434 asbestos samples (excluding bulk and blank samples) were taken. From September 19-21, 177 samples were taken in the financial district. Since September 21, sampling focused on the WTC site and those workers working in or immediately next to it. A total of 179 of the samples analyzed exceeded OSHA's PEL of 0.1 f/cc. However, upon further analysis using discriminating counting methods and/or TEM analysis, the number of asbestos fibers found dropped dramatically to below detectable levels or well below 0.1 f/cc.

The web site also summarizes data taken on CO, total dust, respirable silica, several organic compounds including PCBs, PAHs, dioxins, VOCs, and others, freon-22, hydrogen fluoride, phosgene, inorganic acids, oxides of nitrogen/sulfur, metals including lead, mercury, arsenic, and others, ionizing radiation, and noise. The web site indicates very few exceedences of OSHA PELs or other relevant benchmarks.

The results for respirable silica suggested some exposure. Of 1353 silica samples, 94 exceeded the PEL. The highest sample result was approximately twenty-one times the OSHA limit (jack hammering concrete 16 feet below grade). The other elevated exposures were

approximately one to fourteen times the OSHA limit. These exposures occurred during: 1) pre-drilling /slurry wall 2) jack hammering, 3) rubble removal and loading operations near the Winter Garden; 4) during the breaking up of concrete in the pit; 5) while drilling the concrete slurry wall, 6) flagging operations in pit, and 7) while chipping concrete with power tools during demolition activities at 7 WTC's parking garage. Most of the work areas where apparent overexposures to silica occurred were in the rubble pile/pit.

Similar to other results, very few exceedences of organic compounds were identified, including one exceedence for the benzene OSHA Allowable Limit of 1 ppm, and 8 PAH exceedences of OSHA's coal tar pitch volatile PEL of 0.2 mg/m³.

For metals, OSHA took a total of 1331 samples (excluding bulk and blank samples) to monitor worker exposures to dusts, fumes, oxides, and other compounds of metals such as antimony, beryllium, chromium, cobalt, copper, iron, lead, manganese, mercury, molybdenum, nickel, vanadium, zinc, cadmium, magnesium, and arsenic. Results from these samples were generally well below the applicable OSHA limits. However, torch cutting and burning structural steel at the rubble pile resulted in instances of overexposures as follows: copper (17); iron oxide (28); lead (19); zinc oxide (1), antimony (1); and cadmium (3).

There were 236 samples collected for employee noise, and 20 samples exceeded the OSHA PEL of 90 dBA.

VI.a.2. NIOSH Data

The NIOSH sampling occurred between September 18 and October 14, 2001. The focus was on search-and-rescue personnel, heavy equipment operators, and workers cutting metal beams, but other occupations were also sampled. A total of 1174 air samples were taken, including 804 for asbestos. The New York City Department of Health and Mental Hygiene (NCDOHMH) collected most of the asbestos samples, while NIOSH personnel collected all other samples. In addition to air samples, 33 samples of dust, debris, and other materials were taken. NIOSH has reported on results for asbestos, metals, respirable particulate, CO, hydrogen sulfide, inorganic acids, VOCs, elemental carbon, FreonTM-22, and PAH (CDC, 2002).

The bulk samples mostly showed asbestos concentrations at <1% (by mass); 3 of 29 samples had mass concentrations ranging from 1-3%. Analysis of air samples for asbestos by PCM revealed fibers in 358 of 804 samples (45%). Of 25 samples measured by PCM which exceeded the 0.1 f/cc REL, 18 were measured then by TEM, and all had asbestos concentrations less than 0.1 f/cc. Differential analysis by polarized light microscopy of these 25 air samples revealed that most nonasbestos fibers were fibrous glass, gypsum, and cellulose.

Air concentrations of total (36 samples) and respirable (18 samples) particles showed maximum concentrations of 2.3 and 0.3 mg/m³, respectively, which are below the corresponding RELs of 10 and 5 mg/m³ for Portland cement. Respirable crystalline silica was not detected in any of the 18 samples measured for it.

Two instantaneous peak CO measurements exceeded the 1,200 ppm level (at 1239 and 1369 ppm), the level NIOSH considers an immediate danger to life and health. One was from a

torch cutter and the other from a gasoline-powered saw operator. In 99 other samples, concentrations of CO ranged from 0.2 to 242.0 ppm. This high value was from a 32 minute sample, and it exceeded the NIOSH limit of 200 ppm and would have exceeded the PEL of 50 ppm had it been sustained for 2 hours.

CDC (2002) contains descriptions of all other contaminants measured. In general, nearly all samples were below relevant limits. An "Editorial note" at the end of the article (CDC, 2002, p. 455) concludes that, "At the time of the NIOSH sampling, the ambient air did not appear to be contaminated with toxic substances from the building or their contents or with combustion products to an extent that posed an occupational health hazard."

V1.a.3. Occupational Exposure to PCBs and Dioxins

These two classes of compounds were measured at Ground Zero (WTC Building 5 monitor) and for several locations just off-site. It was judged that EPA's 8-hour continuous air monitoring data on these two classes of compounds was adequate to be evaluating worker exposures in Section IV. These evaluations are contained in Sections IV.c (PCBs) and IV.d (dioxins), and are summarized here.

For PCBs, the highest concentration measured was 153 ng/m³, which was measured at Ground Zero. All other measurements were less than 100 ng/m³, with most at ND or within a typical urban range of 1-8 ng/m³. These are much lower than the NIOSH REL at 1000 ng PCB/m³ as an 8-hr time weighted average air concentration (NIOSH, 2002) and the OSHA PEL at 500,000 ng PCB/m³ as an 8-hr time weighted average air concentration (NIOSH, 2002). Using EPA procedures for estimating 95% upper bound cancer risk, an individual exposed to the highest concentration found at 153 ng PCB/m³ for a period of one month is estimated to have an excess lifetime cancer risk of about 2×10^{-8} . EPA regulatory programs, such as the Superfund Program, typically consider individual incremental cancer risk estimates made in this manner (i.e., in the context of a scenario-based risk assessment) in the range of 10^{-4} to 10^{-6} to be of potential significance, depending on the circumstances. On this basis, an incremental cancer risk estimate in the range of 10^{-8} is judged to be insignificant.

For dioxins, potential cancer and non-cancer risk were assessed using methods that are detailed in EPA's Draft Dioxin Reassessment (EPA, 2000). First, a "scenario" is defined, which describes the pathways of exposures, contact rates with dioxin within these pathways, and the concentrations of dioxin in the exposure media. The scenario for the Ground Zero worker was as follows: this individual is exposed 10 hours per day, 5 days per week, in the time period between September 12 until November 30, 2001. The pathway of exposure is via inhalation, and the rate of inhalation for a WTC worker is 1.3 m³/hr. Using data from the Ground Zero monitor, the average concentration during this time was calculated as 60.7 pg TEQ/m³. Exposure during that time, expressed in terms of mass inhaled divided by body weight and time (pg TEQ/kg-day) is converted to a lifetime dose, and when combined with the appropriate dioxin cancer slope, a 95% upper bound estimate of cancer risk was estimated as 3×10^{-6} , which is about 2 orders of magnitude lower (100 times lower) than current US background cancer risk to dioxin-like compounds. This background risk is primarily due to ingestion of foods of animal origin. For non-cancer risk, a newer approach based on calculation of an incremental increase to background body burden was employed. The dose over the three month exposure period was used in a

simple model to predict body burden increase, and it was found that the exposure of the WTC workers suggests that their body burden could rise up to 10% above current average background. These cancer and non-cancer results resulting from dioxin exposure were evaluated as not significant risks over average background risks for this class of compounds.

VI.b. Data on Indoor Environments

EPA Region 2 is currently conducting extensive monitoring and clean-ups of indoor residences. Much of the information on this effort can be seen on the following website: www.epa.gov/wtc. Part of their effort is to also evaluate background conditions, so that measurements can be compared to this background. It is expected that this effort will provide data from which EPA can conduct further health risk assessments. Also, EPA's Region 2 has been collecting data from various public and private monitoring efforts, and have provided a tabular summary of their preliminary compilation for use in this report (table provided by M. Maddaloni, Region 2, to M. Lorber, EPA Washington, August 18, 2002). The Agency for Toxic Substances and Disease Control (ATSDR) has conducted the only systematic study of the residential environment to date, and the results of their efforts are summarized below. Other summaries are provided below only for the systematic efforts that have been conducted or are underway.

VI.b.1. ATSDR Study on Apartments in Lower Manhattan

The New York City Department of Health and Mental Hygiene (NYCDOHMH) and ATSDR have released the Final Report of the Public Health Investigation to Assess Potential Exposures to Airborne and Settled Surface Dust in Residential Areas of Lower Manhattan (NYCDOHMH/ATSDR, 2002). From November 4 through December 11, 2001, environmental samples were collected in and around 30 residential buildings in lower Manhattan. In addition, four buildings above 59th Street were sampled and used as a comparison area for this investigation.

Bulk dust samples were collected both indoors and on outdoor surfaces and analyzed for the presence of asbestos by both the PLM (polarized light microscopy) and TEM methods. PLM can distinguish between fiber types in a bulk sample by their unique appearance and color when viewed under different wavelengths of light. Asbestos was detected in settled indoor dust in 10 out of 57 (18%) residential units sampled, with the positive samples showing a maximum of 1.5% asbestos in dust. By comparison no asbestos was detectable in dust samples collected in the 5 comparison residences. In outdoor dust collected at Lower Manhattan properties, asbestos was detected in 6 of 14 (43%) samples, with a maximum asbestos concentration in dust of 3.4%.

Importantly, airborne fibers were not detected above background levels (stated as 0.003 f/cc -- fibers meeting criteria for optical visibility) in any of the indoor air samples collected at the 57 residences in Lower Manhattan. Some understanding of the protocol design is needed to interpret the air sampling data. All air filter samples were analyzed first using PCM to determine if fibrous materials were present. If the PCM count (which does not distinguish fiber type) exceeded a 0.003 f/cc level identified as background (using the upper Manhattan measurements), then a TEM analysis for asbestos fibers was performed. Airborne asbestos fibers (meeting equivalent criteria for optical visibility) were not detectable in any TEM analyses (generally <

.001 f/cc).

However, since only a minority of the sampling locations (6) had PCM counts above background, and hence were analyzed by TEM; results are not conclusive regarding the potential for low levels of airborne asbestos (i. e., at levels < 0.003 f/cc -- fibers meeting criteria for optical visibility). In this regard it should be noted that the specific residences that had detectable asbestos in indoor dust did not have elevated airborne PCM levels and TEM data were not collected for these residences. Note that when conditions allowed the residential sampling utilized an "aggressive" methodology involving the operation of the vacuum exhaust, used for settled dust sample collection, to stir the air. Additionally, evidence of elevated asbestos levels was not found in air samples collected in common areas of the apartment buildings or in adjoining outdoor areas.

The NYCDOHMH/ATSDR Final report also included data on synthetic vitreous fibers (SVF or fibrous glass) concentrations in indoor and outdoor dust samples at the same residential locations. SVF (PLM analysis) was detected in a larger number of indoor dust samples (26 of 57 or 46%) and at higher concentrations (range 2-35%) than asbestos. In outdoor dust at these properties, SVF was detected in 11 of 14 (79%) of samples (concentration range 15 - 72%). As with asbestos, the study did not provide evidence of airborne SVF above background levels in indoor air samples collected at the residences in lower Manhattan. PCM measurements will detect SVF, and in those locations where air samples had total PCM fibers exceeding background, Scanning Electron Microscopy (SEM) also reexamined filters for SVF. While such fibers were detectable in two samples, all samples were below 0.001 f/cc.

Air and settled surface dust samples were also analyzed for mineral components of concrete (quartz, calcite, and portlandite) and mineral component of building wallboard (gypsum, mica, and halite). The X-ray diffraction analysis (XRD) analysis for crystalline minerals in air and settled surface dust is reported by NYCDOHMH/ATSDR as semiquantitative (labeled with a "J"). Air sampling for minerals detected quartz and other building-related materials in lower Manhattan. The other forms of crystalline silica were not detected in any air samples except for a one-time detection of cristobalite. The estimated concentrations of these minerals in air were low. In some locations, mineral components of concrete (quartz [3-19 $\mu\text{g}/\text{m}^3\text{J}$], calcite [ND-14 $\mu\text{g}/\text{m}^3\text{J}$], and portlandite [ND-95 $\mu\text{g}/\text{m}^3\text{J}$]) and mineral components of building wallboard (gypsum [4-15 $\mu\text{g}/\text{m}^3\text{J}$] and mica [ND-43 $\mu\text{g}/\text{m}^3\text{J}$]) were detected in air samples at higher estimated levels in lower Manhattan residential areas than in samples taken at comparison residential areas above 59th Street (quartz up to 6 $\mu\text{g}/\text{m}^3\text{J}$, calcite up to 6 $\mu\text{g}/\text{m}^3\text{J}$, portlandite up to 30 $\mu\text{g}/\text{m}^3\text{J}$, gypsum up to 6 $\mu\text{g}/\text{m}^3\text{J}$, and mica up to 17 $\mu\text{g}/\text{m}^3\text{J}$). Quartz, calcite, portlandite and gypsum appear to make up a higher percentage of dust in some buildings in lower Manhattan when compared to settled surface dust samples from buildings above 59th Street. Quartz was detected up to an estimated 31%J versus up to 2%J found in the comparison areas above 59th Street. Neither cristobalite nor tridymite was detected in any of the settled surface dust samples. Similarly gypsum was found at a maximum estimated concentration of 30%J in settled surface dust, higher than the 4%J estimated in the comparison areas above 59th Street. Calcite and portlandite had maximum concentrations of 21%J and 8%J respectively. At lower Manhattan locations sampled, quartz was detected in 81% of common areas and 53% of residences. Gypsum was seen in 88% of common areas and 79% of residences. Minerals were

found in all lower Manhattan outdoor settled surface dust samples at estimated values ranging as high as 27%J quartz, 19%J calcite, 5.5%J portlandite, and 27%J gypsum. No visible settled outdoor dust was available in the comparison areas above 59th Street.

The NYCDOHMH/ATSDR investigators caution that the results of the NYCDOHMH/ATSDR investigation in Manhattan cannot be extrapolated to the lower Manhattan dwellings due to the limited number of units sampled and limited ability to address different cleaning methods, distance from ground zero, or other confounding factors.

Some of the key conclusions of the NYCDOHMH/ATSDR final report are:

- Exposure to significant amounts of SVF, mineral components of concrete (quartz, calcite, and portlandite), and mineral components of building wallboard (gypsum) may cause skin rashes, eye irritation, and upper respiratory irritation, all of which have been voiced as concerns by citizens and first responders. These irritant effects will subside once exposure to SVF, mineral components of concrete, and mineral components of building wallboard end. Some people with pre-existing heart or lung disease (e.g., asthma) or a previous history of very high levels of exposures (occupational) to SVF, mineral components of concrete, and mineral components may be more sensitive to the irritant effects of SVF, mineral components of concrete, and mineral components of building wallboard.
- Sometimes mineral components of concrete (calcite and portlandite) and mineral components of building wallboard (gypsum, mica, and halite) were detected in air samples at higher estimated levels in lower Manhattan residential areas than in samples taken at comparison residential areas. These detected mineral levels are orders of magnitude below occupational standards. Although the occupational standards do not account for sensitive individuals or extended periods of exposure, they provide a comparison to an established health guidance value. The levels of minerals seen in airborne dust do not pose potential health hazards even for a continuous year of exposure at the highest levels detected.
- Some settled surface dust could become airborne if disturbed. Therefore, people could potentially inhale the asbestos, SVF, mineral components of concrete (quartz, calcite, and portlandite), and mineral components of building wallboard (gypsum, mica, and halite) found in settled surface dust of some lower Manhattan residences. Because the weight of dust present in the areas sampled was not determined, it is not possible to determine whether any particular residence had an elevated dust loading. Appropriate continued frequent cleaning should minimize exposures.
- Several worst-case assumptions were made in order to assess the potential long-term public health risks of airborne asbestos and quartz. Some of the assumptions were that no cleaning of indoor spaces has occurred or will occur, all fibers found in air were asbestos fibers, and the highest levels detected last fall in air represent long-term air levels. Using these worst-case assumptions, prolonged exposure (decades) to airborne asbestos and quartz *may* increase the long-term, theoretical risk of people developing lung cancer and

other adverse lung health effects (more than 1 additional case in 10,000 people exposed). For individuals who conduct frequent cleaning of their residences, or participate in the EPA cleaning/sampling program (described below), it is unlikely that their exposure would resemble these worst-case conditions. Residents who follow these cleaning recommendations would not be expected to have any significant increased risk of cancer or other long-term health effects due to asbestos or quartz.

Based upon the conclusions of their investigation, NYCDOHMH and ATSDR made the following recommendations (NYCDOHMH/ATSDR, 2002):

- Because more asbestos, synthetic vitreous fibers (e.g., fiberglass), mineral components of concrete (quartz, calcite, and portlandite), and mineral components of building wallboard (gypsum, mica, and halite) were found in settled surface dust in lower Manhattan residential areas when compared to comparison residential areas, the New York City Department of Health and Mental Hygiene and the Agency for Toxic Substances and Disease Registry are recommending that people continue to conduct frequent cleaning with HEPA vacuums and damp cloths/mops to reduce the potential for exposure.
- To ensure that the recommended frequent cleaning is effective and to ensure that the health of the people of New York City is protected, the New York City Department of Health and Mental Hygiene and the Agency for Toxic Substances and Disease Registry are recommending additional monitoring of residential areas in lower Manhattan. In addition, an investigation should be conducted to better define background levels specific to the city of New York for asbestos, synthetic vitreous fibers, mineral components of concrete (quartz, calcite, and portlandite), and mineral components of building wallboard (gypsum, mica, and halite).
- Lower Manhattan residents concerned about possible World Trade Center-related dust in their residential areas can request cleaning and/or testing from the Environmental Protection Agency as part of their current clean-up program (see next section).

VI.b.2. The Multi-Agency Task Force Efforts Led by US EPA

EPA and its federal, state and city partners have begun to clean up residences impacted by the collapse of the World Trade Center. The clean-up covers residential units south and west of Canal, Allen and Pike Streets, river to river. This effort is being coordinated by the multi-agency Task Force on Indoor Air in Lower Manhattan created by the EPA Administrator. Much of the information on this clean up effort can be found on the web at <http://www.epa.gov/wtc>. The clean-up includes:

- upon request, the clean-up of residential units, using certified contractors, with followup testing for asbestos in the indoor air, or; testing-only of asbestos in the indoor air;
- reimbursement for HEPA (High Efficiency Particulate Air) filter vacuums;
- distribution of health and cleanup information;

- establishment of a Web page (<http://www.epa.gov/nyrdust2/dustcleanup/>) and a toll-free hotline (1-877-796-5471 (TTY for the deaf and hard of hearing: 1-800-396-1018)) to take cleanup and testing requests;
- professional cleanups of remaining unoccupied, uncleaned buildings;
- evaluation of effectiveness of dust cleanup techniques already used, and testing to establish what the pre-existing levels of contaminants were for Manhattan residences.

Three studies are underway to help develop this clean up plan:

Indoor Air Assessment: Selecting Contaminants of Potential Concern and Setting Health-Based Benchmarks - The Contaminants of Potential Concern (COPC) Committee of the World Trade Center Indoor Air Task Force is preparing this report to select COPCs and set health-based benchmarks for levels in residences to assist the Pilot Cleaning Effectiveness Initiative and inform the selection of contaminants in the Background Study (these latter two studies are addressed below). Six COPCs have been proposed: lead, PAHs, dioxin, asbestos, fibrous glass and crystalline silica. For each COPC, benchmark screening levels have been established for both indoor air and indoor surfaces, using a three tier approach:

- Tier I - Level above which, after elimination of potential indoor sources (combustion by-products, stored chemicals, etc.), aggressive clean-up action should be taken expeditiously along with follow-up sampling to confirm attainment of Tier III level.
- Tier II - Range where diligent cleaning should continue, after elimination of potential indoor sources (combustion by-products, stored chemicals, etc.), with follow-up sampling to confirm attainment of Tier III level.
- Tier III - Level below which the risk is negligible or consistent with the New York City background level found in the Background Study.

Pilot Cleaning Effectiveness Initiative - EPA is conducting a pilot program in an uncleaned/unoccupied building at 110 Liberty Street to determine the effectiveness of various cleaning methods for removing asbestos and other contaminants of potential concern from residential dwellings. EPA has completed sampling for contaminants in 110 Liberty Street, a still-unoccupied building close to the WTC site, in what is a comprehensive test of the effectiveness of various cleanup techniques. Cleaning procedures to be tested include those that were recommended following the collapse of the WTC as well as others that may have been used in cleaning residential units. Comprehensive sampling has been or will be conducted before, during and after the pilot cleanup.

Background Study - Most if not all of the pollutants associated with the collapse of the World Trade Center were present in New York City's environment prior to September 11. To establish a baseline for the presence of these contaminants in affected residences, EPA will collect and analyze samples to look for some of these pollutants in apartments in parts of Manhattan that were not impacted. The Agency will use the data to determine pre-existing or "background" levels of these pollutants in interior spaces in New York City.

VI.b.3. Ground Zero Elected Officials Task Force Study of Apartments

A "Ground Zero" Elected Officials Task Force convened within days of September 11 to evaluate the environmental safety of apartments that housed an approximate population of 50,000 residents of lower Manhattan who lived within blocks of Ground Zero. A small-scale monitoring study of two residential buildings was conducted by contract (Chatfield and Kominsky, 2001). Surface wipe samples were taken from both exposure residential dwellings characterized as "high" and "low". The "high" location, so named due to the expectation that higher concentrations would be measured, was in an apartment building located on South End Avenue, close to and southwest of Ground Zero. Apartment 10D, on the East side of this building and which had sustained window damage, was selected for sampling. Heavy dust deposits were in the apartment and were sampled for this study. The "low" location was located four blocks north on Warren Street. The apartment building did not appear to sustain any external damage. Apartments on the 2nd and 5th floor were sampled. Dioxins, furans, and PCBs were measured in wipe and bulk dust samples. Inorganic metals including arsenic, cadmium, mercury, lead, and several others, were measured. Asbestos in air and dust was also measured in all sites.

Concentrations of dioxin, PCBs and metals were generally within "background" levels for both the "high" and "low" exposure apartments. However, asbestos readings were elevated in both air and dust, particularly in the "high" apartment. Chrysotile asbestos fiber counts were obtained using TEM analysis and AHERA counting protocols (fibers > .5 μm) and also by PCME (fibers > 5 μm). Looking at the TEM analysis using AHERA protocols for this summary, 7 air samples in the "low" site showed 6 indoor exceedences of the 70 S/mm² AHERA standard at 316, 379, 279, 142, 141, and 162, with the last sample being a rooftop sample showing a reading of 6.5 S/mm². All these samples were obtained at volumes very near the required 1200 liters. The "high" air samples were extremely elevated with asbestos, and most samplers were discontinued before 1200 liters due to high dust on the filter. Counting of structures greater than 0.5 μm was stopped after just one grid opening because of the large number of structures to count. The six measurements equal 10,620; 7,832; 6,277; 6,285; 7,155; and 548 S/mm². The last sample listed here was an exterior sample; it was taken from just outside a sliding window in the apartment.

Asbestos in indoor dust samples was similarly very high. At the "low" apartment, dust was visible on all surfaces and wipe samples were taken with a wet non-woven cloth in accordance with ASTM D6480-99. At the "high" apartment, furniture and surfaces were coated with a thick coat of dust that could be swept up with a brush. A new toothbrush was used to collect samples in this apartment. At the low apartment, surface chrysotile concentrations up to 470,000 S/cm² were observed, of which up to 79,000 fibers/cm² were fibers and bundles longer than 5 μm . In the high apartment, surface chrysotile concentrations of up to 990,000 S/cm² were observed, of which up to 46,000 were fibers and bundles longer than 5 μm .

Outdoor dust samples and the percentage chrysotile by weight included: a sample collected on the roof of an automobile parked on Church St on the North side of the WTC site - 0.67% chrysotile, on top of an apartment house on Warren St - 1.05% chrysotile, and two samples on the southwest side of the WTC site on South End Ave - 2.25% and 2.05% chrysotile.

VI.b.4. New York City Department of Environmental Protection Sampling of Indoor Dust and Air

The New York City Department of Environmental Protection (NYCDEP) has gathered indoor dust and air sampling data from numerous building owners/managers as part of their Asbestos Control Program. Sampling procedures and analytical methods vary from building to building making data summary difficult. For settled dust, bulk samples were generally obtained and analyzed for percent asbestos content. Many buildings report pending results. Available data indicate non-detect or trace amounts of asbestos in most locations. In general, asbestos concentrations were low especially when compared with occupational standards. Most sample analyzed by PCM (NIOSH 7400) were below 0.01 f/cc while most samples analyzed by TEM (AHERA) were below 70 S/mm².

VI.b.5. New York City Board of Education Sampling of Schools

At the request of the Board of Education (BOE), ATC Inc. conducted bulk dust/wipe and indoor air sampling during the time period December 2001 to March 2002 in the following WTC area schools: Stuyvesant HS (345 Chambers St), HS of Economics and Finance (100 Trinity Place), HS for Leadership and Public Service (90 Trinity Place), PS 150 (334 Greenwich St), PS 234 (292 Greenwich St), and PS/IS 189 (201 Warren St). A limited amount of asbestos bulk dust samples were obtained from these schools. No samples taken from inside the schools exceed 1% asbestos. One sample from PS/IS 189 taken from outside the building exceed 1% asbestos and one sample taken from debris on the roof of PS 234 exceeded 1% asbestos. Wipe samples were obtained and analyzed for a host of contaminants including lead, chromium, cyanide, PCBs, dioxin, silica and fibrous glass. With few exceptions, levels were below health-based guidelines/standards. Exceptions included: P S 150 had one lead sample from a window well that exceeded HUD guidelines; HS of Economics and Finance had multiple lead wipes that exceeded HUD guidelines; Stuyvesant HS had lead samples from the 5th and 6th floor that exceeded 40 µg/ft² on 02/06/02, but follow-up sampling the next day were below HUD guidelines.

All six schools were repeatedly sampled (samples from Stuyvesant began on 9/21/01) for asbestos in air by AHERA TEM protocols. All samples were below the AHERA standard of 70 S/mm² with the following exceptions: HS for Leadership and Public Service - (2/23/02) 956 S/mm² found in the 2nd floor auditorium; (2/24/02) 2,379 S/mm² found in the basement gym; (2/26/02) 978 S/mm² found in the basement gym. Two follow-up samples taken from the gym on 2/28/02 were non detect.

All six schools were repeatedly sampled for respirable particulates (PM_{2.5}). Most schools had multiple days that exceeded the 24 hr standard for sensitive subpopulations (40 µg/m³) but few exceeded the 24 hour standard of 65 µg/m³.

Section VII. Comments and Future Studies

Ambient concentrations of monitored substances of concern have generally decreased to background concentrations in the aftermath of the September 11 disaster. Most substances were at these backgrounds during 2002, although some exceptions were noted. Concentrations of benzene and other VOCs at the North Tower site were considerably above typical urban background as recently as early January, and air concentrations of dioxin were considerably elevated above urban background at monitors close to the WTC site through early December. The average daily benzene concentration at the North Tower was also above the OSHA PEL in early January. Concentrations of asbestos above the AHERA standard were detected in February and March.

Although the general trend of decreasing ambient concentrations for the measured pollutants is reassuring, there are limitations in the interpretation of the data. For example, very little data are available for exposures in the few days to a week immediately after September 11, and there is very little information on exposures inside residences or offices where people spend most of their time. Sampling of air and dust within residences has been conducted by ATSDR, and the results of that study are provided. This study showed very little asbestos in the air in apartments near the WTC and in the comparison apartments. However, a small number of apartments near the WTC had asbestos detected in the dust samples while none were detected in the comparison apartments. Air and dust within 2 apartments located near the WTC were sampled on September 18. Very low concentrations of dioxin, PCBs and metals were found. However, asbestos readings were elevated in both air and dust in both apartments. EPA is now conducting extensive indoor air monitoring, and the results will be evaluated in future EPA reports.

Data do exist for reactive VOCs such as formaldehyde, acetaldehyde, or acrolein, all of which are irritants and might have been produced by the fires at WTC, but to date, these data have not been evaluated. Although the press reported the fires to be out at the WTC site on December 20, there are reports that the fires flared up on occasion in January.

From interpretation of photographs taken during the hours following the collapse of the WTC Towers, it is speculated that some people may have been exposed to the extremely high levels of ambient PM and its constituents were likely to be at risk for immediate acute respiratory and other symptoms. Fine particles or metals such as chromium and nickel in the initial dust cloud could have been irritating or sensitized individuals to further response. The cumulative risk from so many different exposures at high concentrations may well have produced effects that cannot be fully discerned by examination of exposure to individual substances. The potential for multiple chemical sensitivities is of potential concern. Also, even though data may suggest that a substance is associated with a particular effect, a quantitative guidance value may not have been developed. Thus, simply comparing ambient concentrations against known health guidance values may overlook some effects.

Further studies of potential health effects resulting from the WTC disaster are being conducted by a variety of agencies and institutions. These should help in evaluating some of the remaining uncertainties regarding exposure and human health impacts resulting from the

collapse of the WTC buildings. Results from some of these studies are not expected for several years, although some results should be available earlier. Studies are being conducted to evaluate exposures and health effects in persons that were around Ground Zero on September 11 and throughout the rescue and cleanup operations. The studies will be extremely important in getting a more complete picture of some of the actual health effects that resulted from exposures. This is particularly important because we have very little data regarding what people were exposed to September 11 as they were leaving the WTC area surrounded by plumes of dust and burning debris.

To better understand actual exposures on September 11, researchers at EPA's ORD laboratories, in cooperation with academic institutions, are working on projects that will use computer models to reconstruct the plume of dust and debris. The goal is to model and predict the levels of contaminants that were present in the air immediately following the collapse of the WTC buildings. Preliminary results from that modeling have been presented in this report. Investigators are using meteorological data and data available from monitoring results later in September to estimate exposures. When this research is complete, it will aid in addressing health effects.

Studies are also being conducted to help us better understand how exposure to contaminants measured and collected in lower Manhattan throughout this period may cause adverse effects in laboratory tests and animal models. As cited previously, ORD NHEERL scientists conducted several studies to examine the chemical and toxicological properties of PM_{2.5} derived from bulk settled WTC dust (EPA, 2002c; McGee et al., 2002). Comparative respiratory toxicology studies showed that a high dose of WTC PM_{2.5} caused mild lung inflammation and significant respiratory tract hyperresponsiveness in mice. Ambient concentrations which could cause comparable doses and effects in people are high but conceivable (425 µg/m³ over 8 hours) in the immediate aftermath of the collapse of the towers (Gavett et al., 2002). Dust and air samples are also being evaluated by other researchers through funding from NYSDOH.

Most importantly, many local, state and federal agencies and academic institutions have already begun or are planning studies that will monitor the health status of various groups of people that were affected by the events of September 11. In total, EPA is aware of more than 120 studies on health effects in populations impacted by the events of September 11. Although it is impossible to address them all, a few general categories are discussed below.

- Health status, including asthma, among students is one of the many health endpoints that will be evaluated at many schools. Respiratory symptoms, including asthma, will be studied in pre-school children and other child populations as well.
- Longitudinal cohort studies on the impacts on pregnant women and birth outcomes are also being conducted.

- Cohort studies looking at multiple health endpoints are beginning to evaluate health effects in populations of workers involved in the rescue and cleanup efforts.
- Studies are under way to investigate many different mental health outcomes including post-traumatic stress disorder, trauma to children, behavioral changes in adolescents, changes in therapy adherence in HIV/AIDS patients, impacts in particular NYC minority populations, and impacts in occupational groups involved in the cleanup and rescue efforts.
- While monitoring air quality is complete at the WTC site, NYSDEC will continue its routine ambient air monitoring at a number of nearby sites and at the sites around New York City, including a number of schools.

It is hoped that when they are completed, these studies will provide a more complete picture of actual health outcomes. This current report can only evaluate exposures based upon available monitoring data and results, and what these exposures could mean to human health. When the health studies are completed, it will be very useful for EPA to go back and reassess how well the evaluations in this report identified, or didn't identify, a human health concern. This retrospective look will help EPA and other health agencies to do a better job assessing health risks and outcomes in the future. In the meantime, EPA will continue to address potential health risks as needed. EPA will provide a more thorough report, looking at additional contaminants and conducting further evaluations as needed.

Section VIII. References

- Albin M, Horstmann V, Jakobsson K, et al. 1996. Survival in cohorts of asbestos cement workers and controls. *Occup Environ Med* 53:87-93.
- ATSDR (2001). Toxicological Profile for Asbestos. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia.
- ATSDR (2000a). Toxicological Profile for Polychlorinated Biphenyls (PCBs). Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia.
- ATSDR (2000b). Toxicological Profile for Toluene. Agency for Toxic Substances and Disease Registry U.S. Department of Health and Human Services, Atlanta, Georgia.
- ATSDR (2000c). Toxicological Profiles for Chromium. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia.
- ATSDR (1999) Toxicological Profile for Ethylbenzene. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia.
- ATSDR (1998). Toxicological Profile for Chloromethane. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia.
- ATSDR (1997a) Toxicological Profile for Benzene. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia.
- ATSDR (1997b) Toxicological Profile for Nickel. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia.
- ATSDR (1994). Toxicological Profile for Acetone. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia.
- ATSDR (1993). Toxicological Profile for 1,3-Butadiene. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, Georgia.
- Bernstein, D. M.; Rahn, K. A. (1979) New York Summer Aerosol Study: trace element concentrations as a function of particle size. In: Kneip, T. J.; Lippmann, M., eds. *The New York Summer Aerosol Study, 1976*. Ann. N. Y. Acad. Sci. 322: 87-97.
- Brown DP, Dement JM, Okun A. (1994) Mortality patterns among female and male chrysotile asbestos textile workers. *J Occup Med* 36:882-888.
- Cahill, T. A.; Cliff, S. S.; Jimenez-Cruz, M.; Shackelford, J. F.; Dunlap, M.; Kelly, P.; Riddle, S.; Selco, J.; Bench, G.; Grant, P.; Ueda, D.; Perry, K. D.; Leifer, R. Z. (2002) Analysis of

aerosols from the World Trade Center collapse site, September 11 to October 31, 2001. In preparation.

Case BW, Dufresne A. (1997) Asbestos, asbestosis, and lung cancer: Observations in Quebec chrysotile workers. *Environ Health Perspect Suppl* 105:1113-1119.

CDC. 2002. Centers for Disease Control and Prevention. Occupational Exposures to Air Contaminants at the World Trade Center Disaster Site - New York, September-October 2001. *MMWR (Morbidity and Mortality Weekly Report)*, Volume 51:453-456.

Chatfield, E.J., J.R. Kominsky. (2001) Summary Report: Characterization of Particulate Found in Apartments After Destruction of the World Trade Center. Requested by "Ground Zero" Elected Officials Task Force comprised of: U.S. Congressman Jerrold Nadler, Manhattan Borough President Virginia Fields, and 7 other New York State and New York City Officials. Available online at, <http://www.eqm.com>.

Coutant, B.; Zhang, X.; Pivetz, T. (2001) Summary statistics and data displays for the speciation minitrends study: final report. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; contract no. 68-D-98-030.

de Klerk NH, Musk AW, Cookson WO, et al. (1993) Radiographic abnormalities and mortality in subjects with exposure to crocidolite. *Br J Ind Med* 50(10):902-906.

Elsom, D. M. (1992) *Atmospheric Pollution: A Global Problem*, Blackwell Publishers, Oxford, U.K. (2nd edition).

Erickson, M.D. (1997). *Analytical Chemistry of PCBs (2nd Edition)*. CRC Press, Boca Raton, FL.

Freijer JI, Cassee FR, Subramaniam R, Asgharian B, Anjilvel S, Miller FJ, van Bree L, Rombout PJA. 1999. Multiple path particle deposition model (MPPDep version 1.11). A model for human and rat airway particle deposition. Bilthoven, The Netherlands: RIVM Publication 650010019. Available through searching: http://www.rivm.nl/index_en.html [accessed 2 July 2002].

Gavett, S. H, N. Haykal-Coates, J.W. Highfill, A.D. Ledbetter, L.C. Chen, M.D. Cohen, J.R. Harkema, J.G. Wagner, and D.L. Costa. (2002) World Trade Center fine particulate matter causes respiratory tract hyperresponsiveness in mice. *Environ. Health Perspect.* (In review - 8/12/02).

Hill, J.S., Patel, D.P., and Turner, J.R. (1999) Performance Characterization of the MiniVol™ PM2.5 Sampler (<http://www.airmetrics.com/products/studies/1.html>)

IRIS (2002). Integrated Risk Information System. Office of Research and Development, National Center for Environmental Assessment, Washington, DC. Accessible on-line at URL: <http://www.epa.gov/iris/index.html>

Kimbrough, R.D., R.A. Squire, R.E. Linder, J.D. Strandberg, R.J. Montali and V.W. Burse. (1975). Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. *J. Natl. Cancer Inst.* 55(6):1453- 1459.

Lanphear BP, Buncher CR. (1992) Latent period for malignant mesothelioma of occupational origin. *J Occup Med* 34:718-721.

Lorber, M. (2002). A pharmacokinetic model for estimating exposure of Americans to dioxin-like compounds in the past, present, and future. *Science of the Total Environment* 288:81-95.

Lioy, P. J.; Weisel, C. P.; Millette, J. R.; Eisenreich, S.; Vallero, D.; Offenberg, J.; Buckley, B.; Turpin, B.; Zhong, M.; Cohen, M. D.; Prophete, C.; Yang, I.; Stiles, R.; Chee, G.; Johnson, W.; Porcja, R.; Alimokhtari, S.; Hale, R. C.; Weschler, C.; Chen, L. C. (2002) Characterization of the dust/smoke aerosol that settled east of the World Trade Center (WTC) in lower Manhattan after the collapse of the WTC 11 September 2001. *Environ. Health Perspect.* 110: 703-714.

Lippmann, M.; Kleinman, M. T.; Bernstein, D. M. (1979) Size-mass distributions of the New York summer aerosol. In: Kneip, T. J.; Lippmann, M., eds. *The New York Summer Aerosol Study, 1976*. Ann. N. Y. Acad. Sci. 322: 29-44.

McGee, J.K., L.C. Chen, M.D. Cohen, G.R. Chee, C.M. Prophete, N. Haykal-Coates, S.J. Wasson, T.L. Conner, D.L. Costa, and S.H. Gavett. (2002). Chemical analysis of World Trade Center fine particulate matter for use in toxicological assessment. *Environ. Health Perspect.* (In review - 8/12/02).

NIOSH (2002). Pocket Guide to Chemical Hazards (NPG). National Institute for Occupational Safety and Health. NPG is available online at <http://www.cdc.gov/niosh/npg/npg.html>.

NIOSH (1994). NIOSH Manual of Analytical Methods. 4th Edition. DHHS (NIOSH) Publication No. 94-113. August, 1994.

Norback, D.H. and R.H. Weltman. (1985) Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. *Environ. Health Perspect.* 60: 97-105.

NYCDOHMH/ATSDR. (2002) New York Department of Health and Mental Hygiene and Agency for Toxic Substances and Disease Registry. Final Technical Report of the Public Health Investigation To Assess Potential Exposures to Airborne and Settled Surface Dust In Residential Areas of Lower Manhattan. Agency for Toxic Substances and Disease Registry, US Department of Health and Human Services, Atlanta, GA.

OSHA (1994). Occupational Exposure to Asbestos. 59 FR 40964, Aug. 10, 1994.

Peto J, Doll R, Hermon C, et al. (1985) Relationship of mortality to measures of environmental asbestos pollution in an asbestos textile factory. *Ann Occup Hyg* 29:305-345.

Prezant, D.J., et al. (2002) Cough and bronchial responsiveness in firefighters at the World Trade Center site, New England J. Medicine, 347, 806-815.

Schaeffer, E., H. Greim and W. Goessner. (1984) Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. Toxicol. Appl. Pharmacol. 75: 278-288.

Stevens, Robert K., Thomas G. Dzubay, Charles W. Lewis, and Robert W. Shaw, Jr. Source apportionment methods applied to the determination of the origin of ambient aerosols that affect visibility in forested areas. Atmospheric Environ. 18(2): 261-272, 1984.

Stull, R.B., (2000) Meteorology for Scientists and Engineers: Second Edition, Brooks/Cole, Pacific Grove, CA, 502 pp.

US EPA (2002a). Environmental Data Trend Report World Trade Center Disaster. Final Update - Trends for Data Collected from 9/11/01 to 4/24/02 from Lower Manhattan. Prepared by, Quality Assurance Technical Support Laboratory, IT Corporation, 2700 Chandler Avenue, Las Vegas, Nevada 89120. May 1, 2002. Contract Number: 68-W-01-010. Prepared for, Office of Emergency and Remedial Response, U.S. Environmental Protection Agency, Washington, D.C. 20460.

US EPA (2002b). Personal communication, Region 2 to National Center for Environmental Assessment, Office of Research and Development, US EPA, in January 2002. Spreadsheet titled, "Background Conc kf 02 12-20-01" displaying background concentrations for several contaminants of interest, and minimal information on references for said concentrations - date(s) and general location measured only.

US EPA (2002c). Toxicological effects of fine particulate matter derived from the destruction of the World Trade Center. United States Environmental Protection Agency, Office of Research and Development, National Health and Environmental Effects Research Laboratory, Research Triangle Park, NC. September, 2002. EPA/600/R-02/028.

US EPA (2000). Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. United States Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment. Review Draft. September, 2000. EPA/600/P-00/001B(a-f). Available at, <http://www.epa.gov/ncea>.

US EPA (1999a). EPA Superfund Technical Support Center Risk Assessment Issue Paper for: Derivation of a Subchronic RfC for Ethylbenzene Draft, 99-011/10-12-99.

US EPA (1999b). EPA Superfund Technical Support Center Risk Assessment Issue Paper for: Deriving a Provisional Subchronic Inhalations RfC for Toluene, Draft. 98-031/07-19-99.

US EPA (1998). EPA Superfund Technical Support Center Risk Assessment Issue Paper for: Derivation of the Inhalations Toxicity and an Evaluation of the Oral Toxicity of Chloromethane Draft, 98-022/6-14-99.

US EPA (1997a) Exposure Factors Handbook. United States Environmental Protection Agency. National Center for Environmental Assessment, Office of Research and Development, US Environmental Protection Agency. 1997. EPA/600/P-95/002F(a-c).

US EPA. (1997b). Region III. Hazard Evaluation Handbook. A Guide to Removal Actions. EPA 903/B-97-006.

US EPA. (1996) Urban soil lead abatement demonstration project. Volume I: EPA integrated report. Research Triangle Park, NC: Office of Research and Development, National Center for Environmental Assessment; report no. EPA/600/P-93/001aF. Available from: NTIS, Springfield, VA; PB96-168356.

US EPA. (1989) Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-p-dioxins and -dibenzofurans (CDDs and CDFs) and 1989 update. United States Environmental Protection Agency, Washington, DC. Risk Assessment Forum. EPA/625/3-89/016.

US EPA. (1987) Asbestos-Containing Materials in Schools; Final Rule and Notice. 40 CFR Part 763. Federal Register, Vol 52 (210): 41826-41905. October 30, 1987.

US EPA. (1986a) Second addendum to air quality for particulate matter and sulfur oxides (1982): assessment of newly available health effects information. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-86-020F. Available from: NTIS, Springfield, VA; PB87-176574.

US EPA. (1986b) Air quality criteria for lead: volume II. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; report no. EPA-600/8-83/028bF. Available from: NTIS, Springfield, VA; PB87-142394.

US EPA (1986c) Airborne asbestos health assessment update. United States Environmental Protection Agency, Office of Health and Environmental Assessment. EPA/600/8-84/003F.

US EPA. (1984) Risk Analysis of TCDD Contaminated Soil. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. EPA-600/8-84-031.

US EPA. (1982) Air quality criteria for particulate matter and sulfur oxides. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-82-029aF-cF. 3v. Available from: NTIS, Springfield, VA; PB84-156777.

USGS. 2002. U. S. Geological Survey Open File Report OFR-01-0429. Environmental Studies of the World Trade Center area after the September 11, 2001 attack. Denver, CO: U. S. Geological Survey. Available: <http://geology.cr.usgs.gov/pub/open-file-reports/ofr-01-0429/> [accessed 6 July 2002].

Van den Berg ML, Birnbaum L, Bosveld ATC, Brunstrom B, Cook P, Feeley M, Giesy JP, Hanberg A, Hasegawa R, Kennedy SW, Kubiak T, Larsen JC, van Leeuwen FXR, Liem AKD, Nolt C, Peterson RE, Poellinger L, Safe S, Schrenk D, Tillitt D, Tysklind M, Younes M, Warn F, Zacharewski T. Toxic Equivalency Factors (TEFs) for PCBs, PCDDs, PCDFs for Humans and Wildlife. *Environmental Health Perspectives*, 1998; 106: 775.

Vette, A.; Landis, M.; Williams, R.; Webb, L.; Ellestad, T.; Vallero, D. (2002) Concentrations and composition of PM at Ground Zero and lower Manhattan after September 11, 2001. In preparation.

Walker Y, Lorber M, Wang V, Gillooly P. 2002. Impacts of dioxin emissions from the Shinkapo Incinerator to the United States Naval Air Facility at Atsugi, Japan. Part 2: Air impacts. *Organohalogen Compounds* 56:481-484.

WHO 1998. Chrysotile asbestos: Environmental Health Criteria. Geneva: Switzerland: World Health Organization.

**APPENDIX A - WORLD TRADE CENTER HEALTH EFFECTS SCREENING
CRITERIA FOR AMBIENT AIR DEVELOPED BY EPA'S REGION 2**

**Provided by: Mark Maddaloni
US EPA, Region 2**

DRAFT--DO NOT QUOTE OR CITE

A-1

October 2002

World Trade Center Health Effects Screening Criteria for Ambient Air

Introduction

Extensive air quality monitoring data have been collected at and around the World Trade Center (WTC) site since 9/11/01. Table 1 (Screening Criteria) is intended to provide health protective screening values for data evaluation. Analysis has been performed on an extensive list of potentially WTC-related contaminants. Many of the chemicals screened have demonstrated a consistently low (i.e., below detection limits or trace amounts) trend. Consequently, the list of contaminants in Table 1 represents those chemicals that, because of their intrinsic toxicity and frequency/magnitude-of-detection, pose the greatest potential hazard from exposure. This selection process (i.e., a toxicity/concentration analysis), although qualitative, reflects the contaminant-of-concern identification process recommended in the Risk Assessment Guidance for Superfund. Table 1 may be expanded as additional data analysis becomes available. Two populations have been identified for assessment: response/demolition (i.e., WTC site) workers; and residents living in Lower Manhattan (e.g., Battery Park City, Tribeca and other residential locations close to Ground Zero). Included in the resident category are all other workers located in Lower Manhattan with the exception of WTC site workers.

Relevant Standards

The following paradigm has been employed to develop screening values. For each of the two identified receptor populations (i.e., site workers and residents), existing standards are utilized where appropriate. Occupational standards (i.e., OSHA PELs) are used for all site workers conducting response/demolition activities covered by OSHA. Monitoring data from demolition areas are compared to OSHA PELs. (For example, the OSHA PEL of 1 ppm for benzene is used to evaluate benzene air samples taken directly from within the plume on the debris pile.) Environmental standards (e.g., NAAQS, AHERA) are utilized to evaluate monitoring data from the site perimeter and beyond where residents or non-WTC site workers may be exposed. (For example, lead air monitoring data from perimeter stations outside of the immediate work zone are evaluated against the NAAQS of 1.5 $\mu\text{g}/\text{m}^3$).

Risk-Based Screening Criteria

In cases where appropriate standards do not exist, risk-based screening criteria have been developed for residential (including the non-WTC site workers) receptors. (In the absence of OSHA standards, it is beyond the scope of EPA's mission to develop "occupational" screening values.) The risk assessment paradigm detailed in EPA's "Hazard Evaluation Handbook: A Guide to Removal Actions" (HEH) was employed for this initiative (except where otherwise noted in the Table 1 footnotes). Screening levels reflect the most current toxicity criteria (Slope Factors and RfCs) on EPA's IRIS database.

For carcinogenic compounds excess lifetime cancer risk was set at E-04 (one-in-ten thousand). The residential exposure scenario in the HEH was modified for carcinogens from the default of 30 years (upper-bound estimate for residency in one dwelling) to 1 year (to reflect an upper bound estimate for the length of time a resident may be potentially exposed to WTC-related

contaminants). In cases where the screening value based on a noncancer endpoint is more stringent, screening values for both cancer and noncancer endpoints are presented. It is also noted that the default 30 year exposure duration (and the 1 year site-specific adjustment) reflects an apportionment between child (20% of total exposure duration) and adult (80 % of total exposure duration) receptors. Because children have comparatively greater (as a function of body weight) respiration rates than adults, the screening values presented in Table 1 are marginally more stringent than values that would otherwise be derived by direct application of IRIS verified Unit Risk values.

For noncarcinogenic compounds, the Hazard Quotient (chronic daily intake/RfC) was set at 10. A Hazard Quotient of 10 is employed in the HEH to account for the fact that chronic toxicity criteria (RfDs/RfCs) are being applied to sub-chronic exposure scenarios that are not expected to exceed 6 months - 1 year in duration. Accordingly, a Hazard Quotient of 10 was utilized for non-carcinogens in Table 1 to reflect a similar (i.e., upper bound of 1 year) exposure duration. It is noted that contaminants (both non-carcinogens and carcinogens, alike) can exhibit acute effects from short-term, high-dose exposures. Because the screening values in Table 1 are based on subchronic exposure (i.e., 1 year), acute effects from exposures that are below the screening levels would be unlikely. Additionally, a review of California EPA's (CAL-EPA) Acute Risk Levels demonstrates that the screening criteria in Table 1 are categorically more stringent than the Cal-EPA's analogous acute levels.

NOTE: Individual sampling results that exceed screening values should not be interpreted to represent the occurrence of an adverse health effect. Rather, such information indicates the need for careful monitoring and the assessment of longer-term data trends for evaluation against appropriate health criteria. That is, most of the screening levels have been developed to account for continuous one year average exposure durations. Because these screening levels assume continuous exposure for an extended duration, the average of the measured concentrations is more appropriate for evaluating risk than an individual measurement. Consequently, miscellaneous individual values above the screening level may not necessarily be indicative of potential for concern.

Table 1
World Trade Center Screening Criteria

Contaminant	Site Worker ⁽¹⁾	Resident ⁽²⁾
<u>Inorganics</u>		
Asbestos ⁽³⁾	.1 f/cc (PCM)	70 S/mm2 (TEM)
Cadmium	5 µg/m ³	.2 µg/m ³ ⁽⁹⁾ 3 µg/m ³ ⁽⁵⁾
Chromium ⁽⁴⁾	100 µg/m ³	.6 µg/m ³ ⁽⁵⁾
Lead	50 µg/m ³	1.5 µg/m ³ ⁽⁷⁾
Manganese	5 mg/m ³	.5 µg/m ³ ⁽⁶⁾

Contaminant	Site Worker ⁽¹⁾	Resident ⁽²⁾
Sulfur Dioxide	5 ppm	.14 ppm ⁽⁷⁾
<u>Particulates</u>		
Total	15,000 µg/m ³	NA
Respirable	5,000 µg/m ³	NA
PM _{2.5}	NA	40 µg/m ³ ⁽⁸⁾ 65 µg/m ³ ⁽⁷⁾
PM ₁₀	NA	150 µg/m ³ ^(7,8)
<u>Semivolatiles</u>		
Dioxin/Furans (TEQ)	NA	.162 ng/m ³ ⁽⁵⁾
PCBs	1,000 µg/m ³	.73 µg/m ³ ⁽⁶⁾ 9 µg/m ³ ⁽⁵⁾
PAHs ⁽¹⁶⁾	NA	6 µg/m ³ ^(5, 17)
<u>Volatiles</u>		
Acetone	1,000 ppm	1.5 ppm ⁽⁶⁾
Benzaldehyde	NA	860 ppm
Benzene	1 ppm	.02 ppm ⁽⁹⁾ .21 ppm ⁽⁵⁾
Benzonitrile	NA	NA
1,3 Butadiene	1 ppm	.01 ppm ^(5, 15)
Chloromethane	100 ppm	.4 ppm ⁽⁶⁾ 2.6 ppm ⁽⁵⁾
1,4 Dioxane	100 ppm	.5 ppm ⁽⁵⁾
Ethanol	1,000 ppm	45 ppm ⁽¹⁰⁾
Ethylbenzene	100 ppm	2.5 ppm ⁽⁶⁾
Freon 22	1,000 ppm ⁽¹⁴⁾	140 ppm
Propylene	LEL ⁽¹³⁾	simple asphyxiant
Styrene	100 ppm	2.3 ppm ⁽⁶⁾
alpha methylstyrene	100 ppm	.1 ppm ⁽⁶⁾

Contaminant	Site Worker ⁽¹⁾	Resident ⁽²⁾
Tetrahydrofuran	200 ppm	.9 ppm ⁽⁵⁾
Toluene	200 ppm	1.1 ppm ⁽⁶⁾
Xylenes	100 ppm	1 ppm ⁽¹¹⁾
<u>Reactive Gases</u>		
Acetaldehyde	200 ppm	.05 ppm ⁽⁶⁾ 1.3 ppm ⁽⁵⁾
Formaldehyde	.75 ppm	.04 ppm ⁽¹²⁾ .35 ppm ⁽⁵⁾
Acrolein	.1 ppm	.0001 ppm ⁽⁶⁾

Units

f/cc = fibers (>5 µm length) per cubic centimeter of air

S/mm2 = structures (>.5 µm length) per square millimeter of filter paper

ppm = parts per million in air

µg/m³ = micrograms of contaminant per cubic meter of air

ng/m³ = nanograms of contaminant per cubic meter of air

NA - Not Applicable

Footnotes:

1. "Site Workers" refers to all workers involved in the response/demolition of the World Trade Center. Listed values are Occupational Safety and Health Administration (OSHA) Permissible Exposure Limits (PELs), Time Weighted Averages (TWA) unless otherwise noted.

2. "Residents" refers to people living in the vicinity of the World Trade Center as well as all other potentially exposed workers not involved in the response/demolition

3. Resident screening value is based on Asbestos Hazard Emergency Response Act (AHERA) methodology which uses transmission electron microscopy (TEM), and because of its basis in "background" (vs a risk basis) includes all asbestos fibers greater than 0.5 microns in length. Worker values are based on phase contrast microscopy (PCM, - which doesn't distinguish asbestos from other fibers) or, for results above the PCM screening value, TEM to derive a PCM equivalence that includes all asbestos fibers greater than 5 microns in length.

4. Screening values for chromium were based on the most toxic form (hexavalent)

5. EPA - Hazard Evaluation Handbook (HEH) (carcinogen) > 1 year of continuous exposure equating to an excess lifetime cancer risk of one-in ten thousand

6. EPA - HEH (noncarcinogen) > Hazard Quotient (HQ) = 10
7. National Ambient Air Quality Standard (NAAQS)
 - Lead is a 3 month average
 - PM_{2.5} is a 24 hour average
 - Sulfur Dioxide is a 24 hour average primary standard
8. Air Quality Index (AQI)
9. Non cancer effects based on CAL-EPA toxicity studies
10. American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV)
11. Agency for Toxic Substances and Disease Registry (ATSDR) Inhalation minimum risk level (MRL) x 10
12. ATSDR acute MRL
13. Lower Explosive Limit (2 - 11 %)
14. National Institute of Occupational Safety and Health (NIOSH)
15. Proposed Reference Concentration (RfC) - HEH (noncancer) > Hazard Quotient (HQ) = 10
16. Based on Benzo(a)pyrene toxicity equivalency factor toxicity equivalency factor (TEF)
17. EPA National Center for Environmental Assessment (NCEA) provisional inhalation Slope Factor (3.1 E 00 mg/kg/day⁻¹)

APPENDIX B - TABLE OF MONITORING LOCATIONS

DRAFT--DO NOT QUOTE OR CITE

B-1

October 2002

Table 1. Overview of monitoring locations and responsible parties (see note at end of table for specific notes).

Site Name	Site Location	Pollutants Measured	Sampling Frequency	Analytical Laboratory
I. EPA's Environmental Response Team Lettered Sites				
Location A	Barclay St & West Broadway	Asbestos	2 - 12 hour samples daily	Contract
		Dioxins (2)/PCBs, PAHs, Metals, Silica	8 hour sample twice per week	
		Particulates, VOCs	24 hr. sample daily; grab (VOC)	EPA/ORD
Location B	Church St and Dey St	Asbestos	2 - 12 hour samples daily	Contract
		Dioxins (2)/PCBs, PAHs, Metals, Silica	8 hour sample twice per week	
Location C	Liberty St and Trinity St	Asbestos	2 - 12 hour samples daily	Contract
		Dioxins (2)/PCBs, PAHs, Metals, Silica	8 hour sample twice per week	
		Particulates, VOCs	24 hr. sample daily; grab (VOC)	EPA/ORD
Location C1 (note: station C and C1 are "alternate" stations; see footnote 1)	Broadway and Liberty St	Asbestos	2 - 12 hour samples daily	Contract
		Dioxins (2)/PCBs, PAHs, Metals, Silica	8 hour sample twice per week	
		Particulates	24 hr. sample every 3rd day	EPA/ORD
Location D	Albany St & Greenwich St	Asbestos	2 - 12 hour samples daily	Contract
		Dioxins (2)/PCBs, PAHs, Metals, Silica	8 hour sample twice per week	
Location E	Liberty St & South End Ave	Asbestos	2 - 12 hour samples daily	Contract
		Dioxins (2)/PCBs, PAHs, Metals, Silica	8 hour sample twice per week	

Table 1. Overview of monitoring locations and responsible parties (cont'd).

Site Name	Site Location	Pollutants Measured	Sampling Frequency	Analytical Laboratory
I. EPA's Environmental Response Team Lettered Sites (cont'd).				
Location F	Vesey St & West St	Asbestos	2 - 12 hour samples daily	Contract
		Dioxins (2)/PCBs, Metals	8 hour sample twice per week	
Location G	Church & Duane St	Asbestos	2 - 12 hour samples daily	Contract
Location H	Chase Manhattan Plaza	Asbestos	2 - 12 hour samples daily	Contract
Location I	Broadway & Wall St	Asbestos	2 - 12 hour samples daily	Contract
Location J	Warren & West St	Asbestos	2 - 12 hour samples daily	Contract
Location K	Albany & West St	Asbestos	2 - 12 hour samples daily	Contract
		Particulates, VOCs	24 hr. sample daily; grab (VOC)	ORD
		Metals, Silica	8 hour sample twice per week	Contract
Location L	North Side of Stuyvesant High	Total particulates	~ 8 hours/day	Onsite Dataram
		Asbestos	2 - 12 hour samples daily	Contract
		Metals, Silica	8 hour sample twice per week	Contract
Location M	Harrison St & West St	Total particulates	~ 8 hours/day	Onsite Dataram
		Asbestos	2 - 12 hour samples daily	Contract
Location N	Pier 25, Southside	Total particulates	~ 8 hours/day	Onsite Dataram
		Asbestos	2 - 12 hour samples daily	Contract
Location P	Albany St & South End Ave	Asbestos	2 - 12 hour samples daily	Contract
		Dioxins (2)/PCBs, PAHs Metals, Silica	8 hour sample twice per week	
Location Q	Barclay St & West St	Asbestos	2 - 12 hour samples daily	Contract
Location R	EPA Taga Bus	Total particulates	~ 8 hours/day	Onsite Dataram
		Dioxins (2)/PCBs, PAHs	8 hour sample twice per week	Contract
		Metals, Silica		

Table 1. Overview of monitoring locations and responsible parties (cont'd).

Site Name	Site Location	Pollutants Measured	Sampling Frequency	Analytical Laboratory
I. EPA's Environmental Response Team Lettered Sites (cont'd).				
Location S	Rector Pl & South End Ave	Asbestos	2 - 12 hour samples daily	Contract
		Dioxins (2)/PCBs, PAHs Metals, Silica	8 hour sample twice per week	
Location T	Pier 6 Heliport	Asbestos	2 - 12 hour samples daily	Contract
Location U	Pier 6, Exit 2	Asbestos	2 - 12 hour samples daily	Contract
Location V	Pier 6, Bus Sign	Asbestos	2 - 12 hour samples daily	Contract
Location W	Wash Tent, West Street & Murray	Asbestos	2 - 12 hour samples daily	Contract
WTC - Building 5 SW	AKA, Location 3A	Dioxins (2)/PCBs, PAHs, Metals, Silica	8 hour sample twice per week	Contract
WTC - Church & Vesey (alternate for WTC; see footnote 1)	AKA, Location 3B	Dioxins (2)/PCBs, PAHs, Metals, Silica	8 hour sample twice per week	Contract
S.I. Landfill Sites	17 Landfill Sites + 3 offsite locations	Asbestos	1 - 12 hour sample daily	Contract
	5 Landfill Sites	Total particulates	12 hour sample daily when temp. & humidity specs are met.	Onsite Dataram
	2 Landfill Sites + 3 Offsite Locations	Metals	1 - 12 hour sample weekly	Contract

Table 1. Overview of monitoring locations and responsible parties (cont'd).

Site Name	Site Location	Pollutants Measured	Sampling Frequency	Analytical Laboratory
II. Extended Monitoring Network- NYSDEC Numbered Air Monitoring Stations				
1	Park Row & Spruce Street, NY, NY	Asbestos	12 hour sample daily	Contract
		Dioxin (1)	72 hour sample every 3 days	Region 7
		VOC's	24 hr. sample every 3rd day	ORD
		Aldehydes	24 hr. sample every 3rd day	NYSDOH
		PM Speciation	24 hr. sample every 3rd day	Contract
		PM Sizing	continuous-by NYSDEC	Climet
		PM10, PM2.5	continuous- by NYSDEC	PM 2.5 TEOM/ PM10 Filter
2	Chambers St.& West St., NY, NY	Asbestos	12 hour sample daily	Contract
		Dioxin (1)	72 hour sample every 3 days	Region 7
		VOC's	24 hr. sample every 3rd day	ORD
		Aldehydes	24 hr. sample every 3rd day	NYSDOH
		PM Speciation	24 hr. sample every 3rd day	Contract
		PM Sizing	continuous-by NYSDEC	Climet
		PM10,PM2.5,PM	continuous- by NYSDEC	PM 2.5 TEOM/ PM10 Filter
3	U.S. Coast Guard, 1 South Street, NY, NY (Battery Pk)	Asbestos	12 hour sample daily	Contract
		PM10, PM2.5	continuous- by NYSDEC	PM 2.5 / PM10 Filter

Table 1. Overview of monitoring locations and responsible parties (cont'd).

Site Name	Site Location	Pollutants Measured	Sampling Frequency	Analytical Laboratory
II. Extended Monitoring Network- NYSDEC Numbered Air Monitoring Stations (cont'd)				
4	Canal Street Post Office, 350 Canal Street, NY NY	Asbestos	12 hour sample daily	Contract
		PM10, PM2.5	24 hour sample daily-by NYSDEC	PM 2.5 / PM10 Filter
5	PS 154, 333 East 35th Street, Bronx, NY 10454	PM2.5	continuous-by NYSDEC	PM 2.5 TEOM
		Asbestos	12 hour sample daily	Contract
6	IS 143, 511 West 182nd Street NY NY 10033	PM2.5	continuous-by NYSDEC	PM 2.5 TEOM
		Asbestos	12 hour sample daily	Contract
7	PS 274, 800 Bushwick Ave, Brooklyn, NY 11221	PM2.5	continuous-by NYSDEC	PM 2.5 TEOM / PM10 Filter
		Asbestos	12 hour sample daily	Contract
8	PS 44, 80 Maple Parkway, Staten Island, NY 10303	PM2.5	continuous-by NYSDEC	PM 2.5 TEOM
		Asbestos	12 hour sample daily	Contract
9	PS 199, 39-20 48th Ave, Long Island City, NY 11104	PM2.5	continuous-by NYSDEC	PM 2.5 TEOM
		Asbestos	12 hour sample daily	Contract

Table 1. Overview of monitoring locations and responsible parties (cont'd).

Site Name	Site Location	Pollutants Measured	Sampling Frequency	Analytical Laboratory
III. Extended Monitoring Network- NJDEP Air Monitoring Stations				
NJ-Shell	West Avenue, Sewaren, NJ	Asbestos	1 - 12 hour sample on Mon. and Thurs.	Contract
NJ-Citgo	Tremly Point Road, Linden, NJ	Asbestos	1 - 12 hour sample on Mon. and Thurs.	Contract
NJ-FMC	Roosevelt Blvd., Cartaret, NJ	Asbestos	1 - 12 hour sample on Mon. and Thurs.	Contract
NJ-Liberty	Liberty State Park, @ WTC Disaster Family Center	Asbestos	1 - 12 hour sample on Mon. and Thurs.	Contract
IV. Extended Monitoring Network- EPA/ORD Numbered Air Monitoring Stations				
14	Albany & West St NY, NY	Dioxin (1)	72 hour sample every 3rd days	Region 7
		PM Speciation	24 hr. sample every 3rd day	Contract
		PM Sizing	continuous-by NYSDEC	Climet
		PM10, PM2.5	continuous-by NYSDEC	PM 2.5 TEOM/ PM10 Filter
		VOCs	24 hr sample every 3rd day	ORD
15	23 Wall Street, NY, NY	PM10, PM2.5	continuous-by NYSDEC	PM 2.5 TEOM/ PM10 Filter
		Aldehydes	24 hr. sample every 3rd day	NYSDOH

Table 1. Overview of monitoring locations and responsible parties (cont'd).

Site Name	Site Location	Pollutants Measured	Sampling Frequency	Analytical Laboratory
IV. Extended Monitoring Network- EPA/ORD Numbered Air Monitoring Stations (cont'd)				
16	290 Broadway NY, NY	PAHs & SVOCs	12 & 23 hour samples	ORD
		Particulates	continuous	ORD

Notes for Table.

Column 1: Site Name

- Though not "lettered", the WTC - Building 5 SW site and the Staten Island Landfill Sites have been run by EPA's Environmental Response Team and are included here.
- The New Jersey Department of Environmental Protection (NJDEP) site names were designated by NJDEP
- Locations C (Liberty & Trinity) and C1 (Liberty and Broadway), and 3 (WTC Building 5) and 3A (Church & Vesey) were "alternate" pairs of stations, meaning that sample dates alternated between the paired sites.
- 290 Broadway was a sample site that measured several contaminants, but only on 1 date, October 10, 2001. Because there was only one date of sampling, this site is not listed above.

Column 2: Site Location:

Column 3: Pollutants Measured:

- Dioxin (1) = 72-hr samples collected by EPA Department of Environmental Science and Assessment (DESA) Region 2 personnel and analyzed by Region 7.
- Dioxin (2) = 8-hr samples collected by EPA Emergency and Remedial Response Division (ERRD) Region 2 personnel and analyzed by a contract laboratory.

Column 4: Analytical Laboratories:

- Contract = Laboratories contracted by EPA to conduct analysis.
- ORD = Human Exposure and Atmospheric Sciences Division (HEAS) in the National Exposure Research Laboratory (NERL), in Research Triangle Park, North Carolina.
- Region 7 = Regional Laboratory Branch, in the Environmental Services Division (ESD) in Region 7, Kansas City, Kansas.

September/mid-October (although some personal sampling results were reported that exceeded PEL levels on widely-scattered days from late October into early 2002 for the WTC 5 Building area at Ground Zero). Nor were there any notable blood lead elevations (maximum values < 20 µg/dl) among more than 300 male fire fighters serving at WTC Ground Zero and sampled by CDC in October 2001 (personal communication, P. Edelman). These data tend to suggest that, although it can not be entirely ruled out, it is unlikely that any pregnant or other women of childbearing age working within the WTC Ground Zero perimeter (e.g., among rescue/recovery personnel or assisting with dispensing of food, beverages or other aid to such personnel) would have experienced sufficient lead exposures to be at high risk for lead intoxication effects on them or any fetuses in-utero during or soon after WTC-related lead exposures.

It should also be noted that limited lead results available for analyses of bulk dust samples taken at locations close to the WTC did not appear to show any notably high lead concentrations. The values for bulk dust samples near the WTC noted in the WTC trends report ranged from 120 to 370 µg/g (ppm) - the latter value for a sample taken at Park Place and West Broadway on September 16. These values are consistent with lead concentrations found by ORD in bulk dust near the WTC (median 142 ppm) or those reported by cooperating academic investigators (Lioy et al., 2002) as ranging from 101 to 625 µg/g in bulk dust collected several blocks east of the WTC within days after September 11. They are also not exceptional in comparison with the 500 - 1000 ppm street dust or residential soil lead concentrations often still found in U.S. urban areas in the 1990s, as earlier stated. However, as Lioy noted, indoor exposures to lead-contaminated WTC-derived dust that penetrated indoors could continue to pose risks to individuals re-occupying buildings not cleaned by effective decontamination procedures.

On the basis of results evaluated to date, there is little indication of any substantial health risks being associated with lead exposures of the general population in lower Manhattan areas around the WTC site. However, evaluation of blood lead levels and pertinent medical records for any pregnant women exposed at Ground Zero or in its immediate vicinity during September or early October could provide useful further data by which to assess any such possible health risks associated with WTC-generated lead emissions.

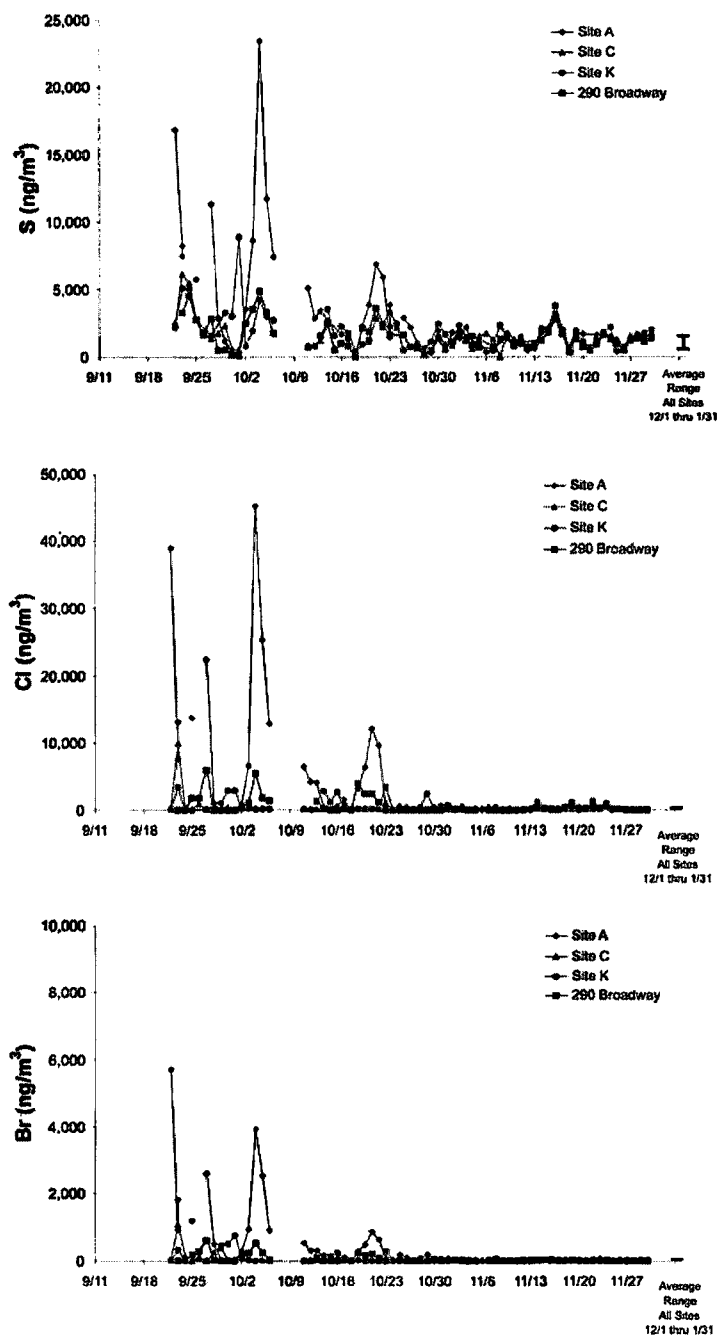


Figure 15. ORD measurements of $\text{PM}_{2.5}$ elemental composition for S, Cl, and Br at Ground Zero perimeter sites and 290 Broadway site.

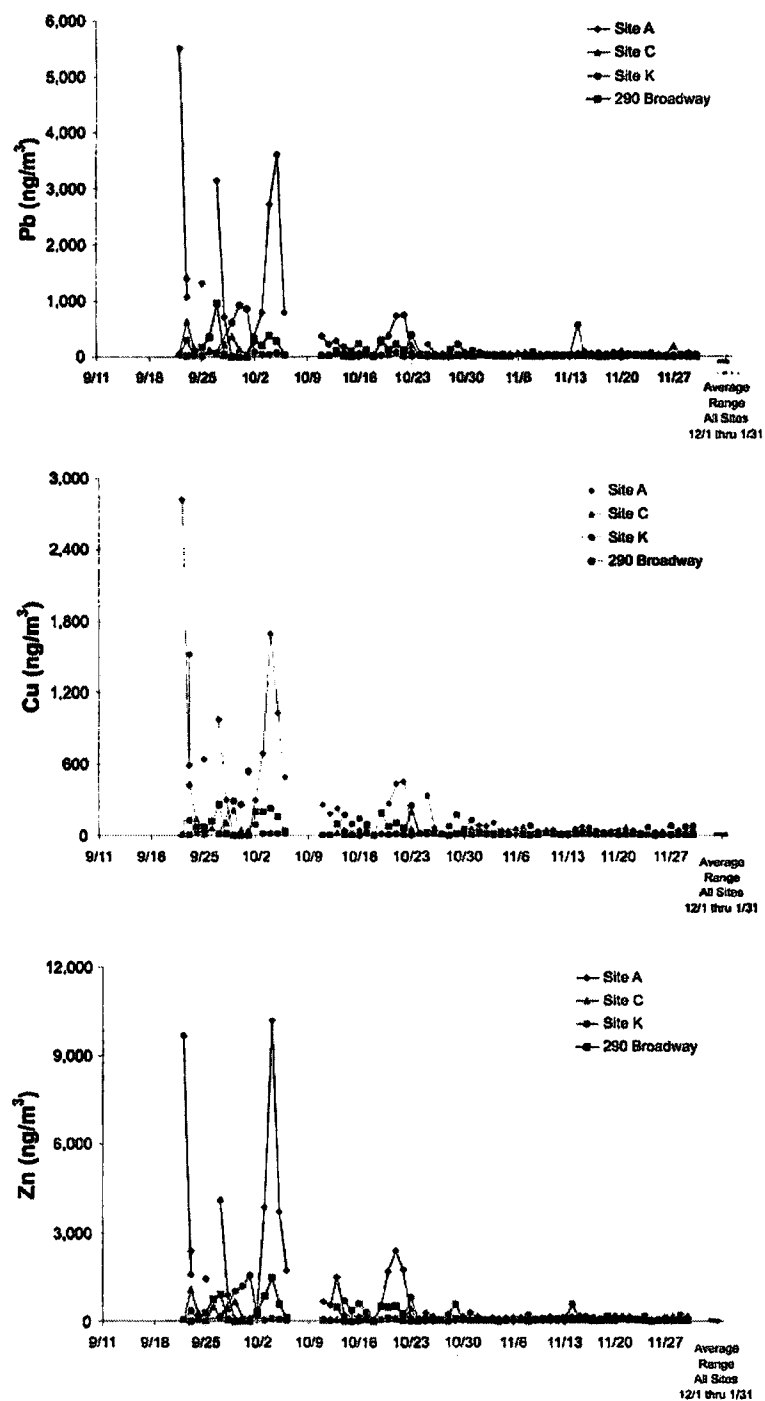


Figure 16. ORD measurements of PM_{2.5} elemental constituents Pb, Cu, and Zn at Ground Zero perimeter sites and 290 Broadway site.

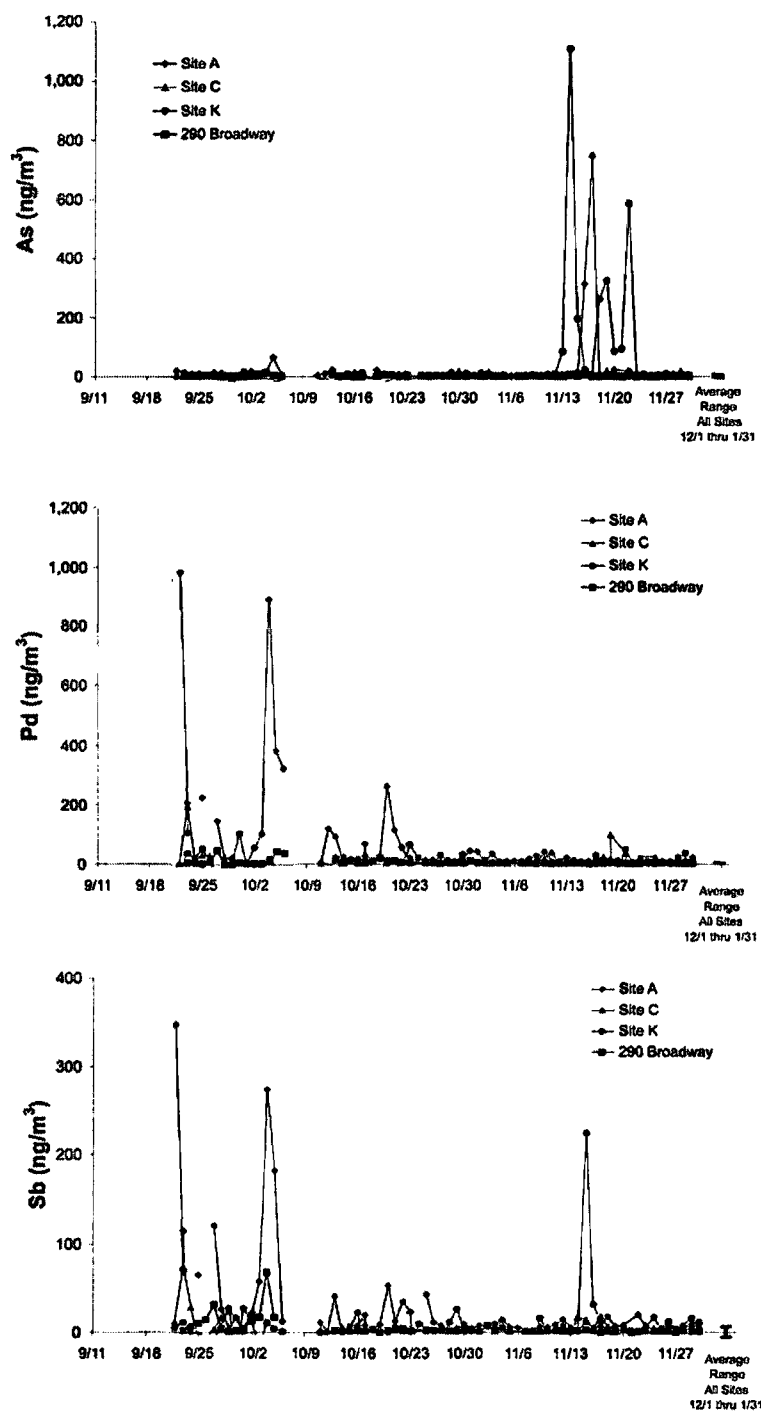


Figure 17. ORD measurements of $\text{PM}_{2.5}$ elemental constituents As, Pd, and Sb at Ground Zero perimeter sites and 290 Broadway site.

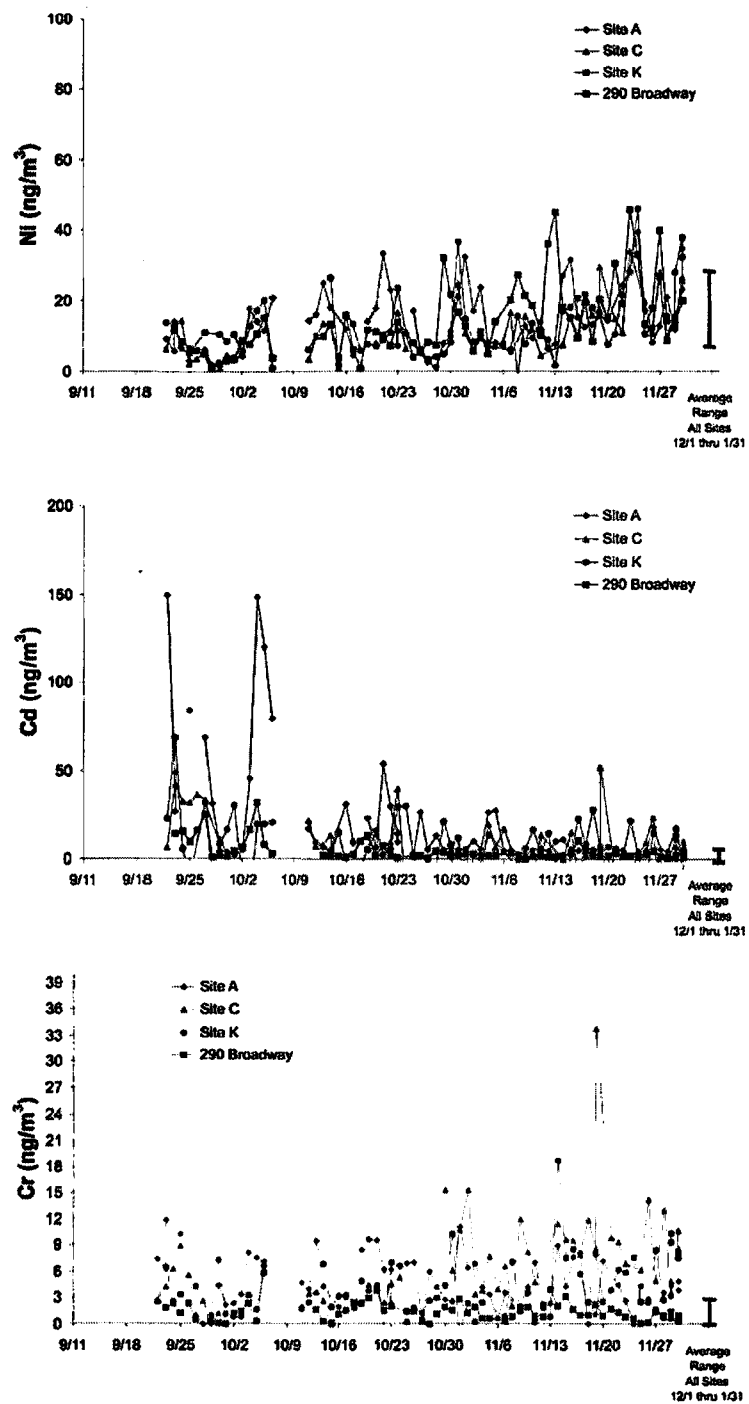


Figure 18. ORD measurements of $\text{PM}_{2.5}$ elemental constituents Ni, Cd, and Cr at Ground Zero perimeter sites and 290 Broadway site.

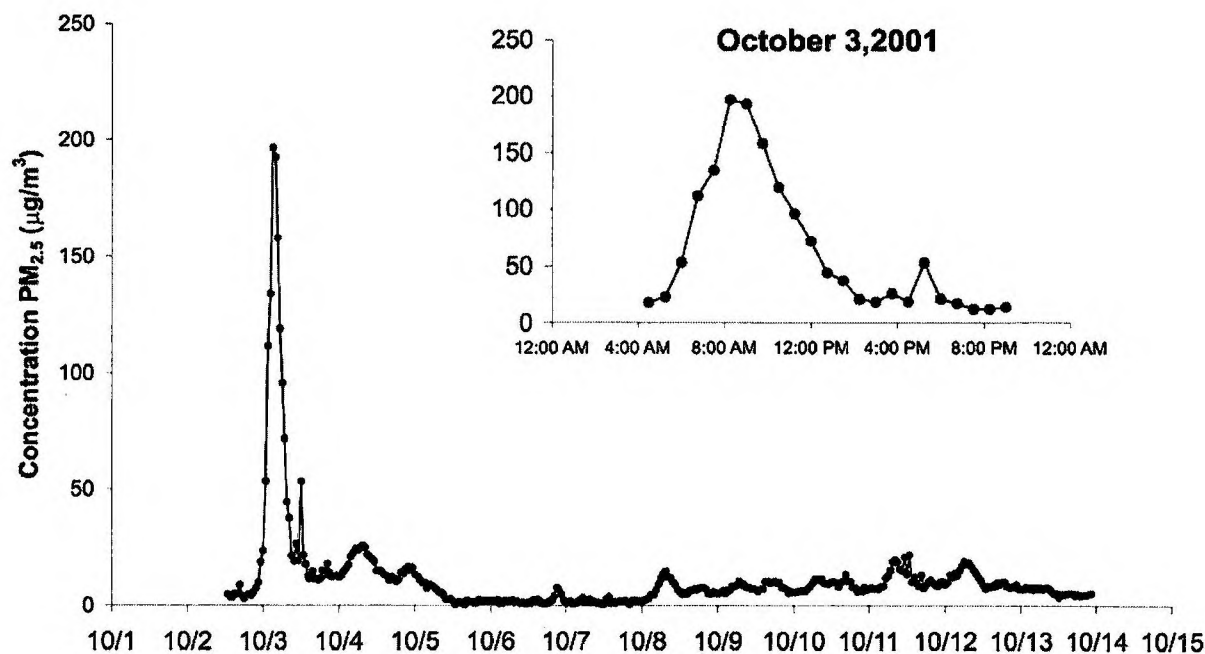


Figure 19. PM_{2.5} concentrations recorded on rooftop of DOE Facility at Varick St., approximately 2.0 miles N/NE of Ground Zero. Note single very high PM_{2.5} excursion mainly restricted to morning hours of October 3 (see inset figure for October 3), consistent with ORD measurements at Location A on the WTC north perimeter and ORD WTC plume plot shown in Figure 12 for October 3.

IV.b. Metals

IV.b.1. Lead

Lead, a silver-grayish soft metal with a relatively low melting point, was still widely used in the 1960s and 1970s (when the WTC was built) in paint and for soldering indoor plumbing joints and electrical wiring systems. It was also used for soldering of computer circuit boards and wiring in a variety of electrical appliances. These uses make it likely that lead would be among the toxic substances of concern at the WTC site. Lead could have been emitted as a combustion product of the ensuing fires or could have been present in reentrained particles stirred up into the air in the course of recovery activities and transport of debris away from the WTC site.

The potential public health concern due to lead exposure most relevant for consideration in relation to the WTC situation is lead intoxication associated with prolonged low-level lead exposures. As discussed in EPA (1986b), such exposure can result in subtle, often subclinical, health effects such as altered calcium metabolism and bone formation/loss, slowed physical growth, and slowed nervous system development of the fetus. Effects on the fetus may be due to exposure of the mother during pregnancy, therefore, women of childbearing age have been identified as an important susceptible population. Other effects of lead intoxication may be slower postnatal growth and neurobehavioral development, IQ decrements and learning deficits, and other neuropsychological effects among young infants and children (another susceptible group).

In 1978, the EPA NAAQS for lead was set at $1.5 \mu\text{g}/\text{m}^3$ (90-day average). This level was set to reduce the risk of occurrence of lead intoxication impacts associated with prolonged low-level exposures of susceptible groups. With the EPA phase-down of lead as an additive in gasoline during the past several decades and the current widespread use of unleaded gasoline in the U.S., ambient air lead concentrations have decreased dramatically. Before the start of the phase-down of lead in gasoline in the late 1970s, air lead levels as high as $2.0 \mu\text{g}/\text{m}^3$ or more were often detected in U.S. urban areas such as NYC. Currently, 24-hour ambient air lead levels below $0.5 \mu\text{g}/\text{m}^3$ are typical of NYC and other U.S. urban areas. NYSDEC Annual Air Quality Reports, for example, indicate (a) arithmetic mean annual-average 24-hour airborne lead levels during 1994 - 1998 of 0.04 to $0.08 \mu\text{g}/\text{m}^3$ for a Manhattan (Madison Ave.) curbside sampling site (maximum daily value = $0.34 \mu\text{g}/\text{m}^3$) and (b) arithmetic mean annual-average values ranging from 0.02 to $0.09 \mu\text{g}/\text{m}^3$ for three Brooklyn/Staten Island sites (maximum daily value = $0.63 \mu\text{g}/\text{m}^3$) during 1992 - 1997.

Before the start of gasoline lead phase-out, lead concentrations in outdoor dust were reported (in studies assessed in EPA, 1986b) to range from 280 to 1500 $\mu\text{g}/\text{g}$ (ppm) in residential areas of NYC and Philadelphia and from 900 to 13,000 $\mu\text{g}/\text{g}$ in street dust or near heavily traveled roadways in several northern U.S. urban areas (NYC; Philadelphia; Washington, DC; Chicago; Detroit). Despite significant decreases in air lead concentrations, soil and street dust lead concentrations in excess of 500 - 1000 $\mu\text{g}/\text{g}$ were still observed into the early 1990s in U.S. urban areas (e.g., Boston, Baltimore, and Cincinnati, as described in EPA, 1996). This may reflect, in part, the residuum from earlier gasoline lead deposition from air or more current contamination from deterioration of lead-based paint from residential or other structures or from

industrial production or waste disposal activities.

The potential for very high short-term lead exposures existed during the initial spread of the dust/smoke cloud from the initial WTC collapse; and the ensuing fires, recovery operations, and debris removal may have also posed some lead exposure risks. Examination of air lead data from ORD WTC perimeter sites (Figure 16) and for additional lower Manhattan sites (Figure 20) reveals that 24-hour lead concentrations within (e.g., at WTC Building 5 SW) or at the WTC Ground Zero perimeter (e.g., at Location A, Barclay and W. Broadway; Location B, Church and Dey) approached or exceeded $1.5 \mu\text{g}/\text{m}^3$ on several days in late September/early October (e.g., September 17, 23, 27 and October 4, 5). There appeared, however, to be rapid fallout (deposition) of the lead from air close to the WTC rather than the lead being transported over longer distances. This interpretation is based on the relatively uniform low air lead values (mostly less than $0.5 \mu\text{g}/\text{m}^3$) seen at the EPA ORD 290 Broadway monitoring site (Figure 16) and at several other locations within a few blocks of the WTC (Figure 20). Consistent with the pattern seen in Figure 16 for EPA/ORD WTC perimeter and 290 Broadway monitoring sites, lead elevations at other lower Manhattan sites outside the WTC work zone generally returned to more typical low background concentrations by mid-October. After October 8, none of the air lead concentrations shown by the WTC Trends Report (EPA, 2002a) for any of the lower Manhattan monitoring sites outside WTC Ground Zero approached or exceeded $1.5 \mu\text{g}/\text{m}^3$; in fact, with very few exceptions, nearly all were below $0.5 \mu\text{g}/\text{m}^3$. Thus, prolonged lead concentrations averaged over 90 days during late September to late November, 2001, at WTC perimeter or other nearby lower Manhattan sites did not exceed the EPA Lead NAAQS (i.e., $1.5 \mu\text{g}/\text{m}^3$, 90-day average). The overall pattern of data, coupled with restrictions of vehicular or pedestrian traffic in lower Manhattan areas close to Ground Zero until very late September/early October, make it doubtful that persons outside the Ground Zero area were exposed sufficiently to airborne lead levels so as to experience any chronic health risks.

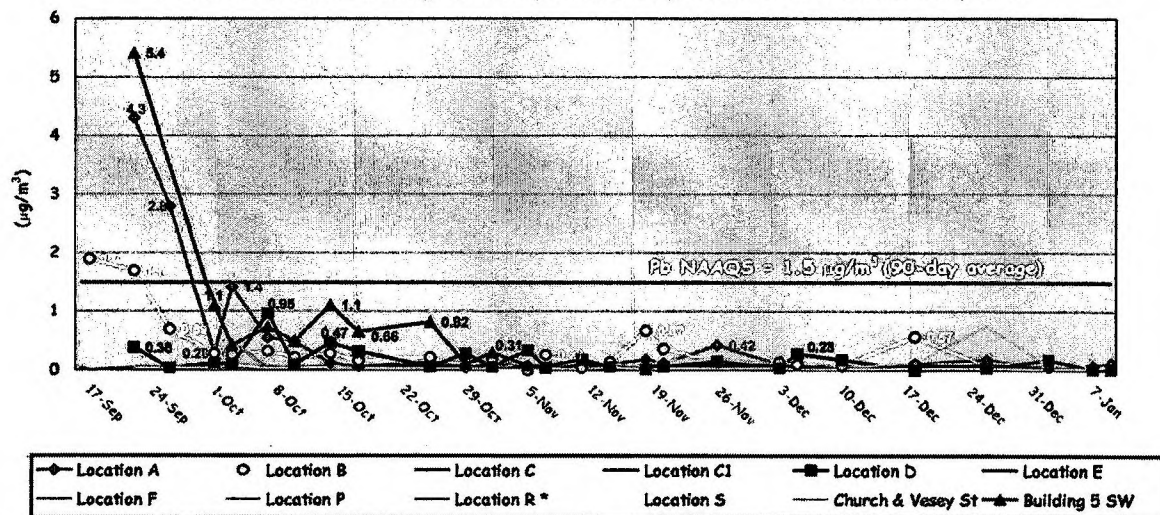
There may, however, exist some basis for potential concern with regard to short-term (hours, days) highly elevated lead exposures of individuals working within the Ground Zero perimeter without appropriate respiratory protection. The highest 24-hour lead level shown in Figure 20 was $5.4 \mu\text{g}/\text{m}^3$ at WTC Building 5 on September 24. It is likely that comparable or higher elevations in ambient air may have occurred within Ground Zero on some days preceding the start of EPA monitoring in late September. This possibility and the above-noted data showing air lead values approaching or exceeding $1.5 \mu\text{g}/\text{m}^3$ on certain days during late September and into October at sites within the WTC work zone or at perimeter sites very close to Ground Zero (within 100 - 200 m) suggest that flare-ups of Ground Zero fires or lead emissions associated with recovery and debris removal operations might have posed risks for individuals within the WTC Ground Zero perimeter. The potential risks would probably be of most concern for pregnant women or other women of childbearing age if any were working within Ground Zero without wearing appropriate protective respirators for extended periods during the first 2 to 4 weeks after September 11. OSHA data (<http://osha.gov/nyc-disaster/map.html>) do indicate that high air lead levels were detected on September 22 ($69.3 \mu\text{g}/\text{m}^3$, averaged over 4.5 hr) and September 23 ($18.1 \mu\text{g}/\text{m}^3$, averaged over 3 hr) by area monitoring near WTC Building 5 well within the Ground Zero work zone. However, none of the OSHA personal sampling data reported for WTC recovery workers (e.g., ironworker, torch-cutter/burner) exceeded the OSHA Lead PEL ($50 \mu\text{g}/\text{m}^3$, 8-hr average) during late



Lead (Pb) Air Monitoring Trends

Inside and Outside WTC Zone

255 Ambient Air 24hr integrated samples from September 2001 to mid-January 2002



*Higher of two (2) sample results, taken per day, is included here.

*Location R has 2 results therefore 255.

Figure 20. Ambient air lead concentrations ($\mu\text{g}/\text{m}^3$) at sites within Ground Zero or in lower Manhattan locations in immediate vicinity of the WTC.

Source: EPA Region 2

IV.b.2. Chromium and Nickel

Chromium and nickel were chosen for evaluation in this assessment because both can be irritating and sensitizing. Chromium and nickel are used in the production of stainless steel and other metal alloys. Chromium, in the hexavalent form, Cr^{+6} , can damage the nose and cause cancer. Similarly, workers who have breathed large amounts of nickel have developed lung and nasal sinus cancers. Total chromium in urban air typically ranges from 0.01 to 0.03 $\mu\text{g}/\text{m}^3$ (ATSDR, 2000c), and nickel concentrations in urban air range from 0.001 to 0.328 $\mu\text{g}/\text{m}^3$ (ATSDR, 1997b).

To evaluate chromium and nickel, the OSHA PELs (chromium, 1 mg/m^3 ; nickel, 1 mg/m^3) were used as a screening benchmark (NIOSH, 2002). For chromium, the ATSDR intermediate inhalation MRL for Cr^{+6} PM (1.0 $\mu\text{g}/\text{m}^3$) was also used (ATSDR, 2000c). For this evaluation, it was assumed that chromium would be released as solid PM, not as a mist, in order to compare measurements with the ATSDR MRL, which is specific to PM concentrations.

Data for evaluating chromium and nickel came mostly from the EPA WTC monitoring database. A total of 21 air samples, collected between September 23 and January 31 at Building 5, were evaluated for chromium and nickel at Ground Zero. None of the samples evaluated exceeded a screening benchmark for either chromium or nickel, nor did any values detected by ORD monitoring on the Ground Zero perimeter (Figure 18) exceed any benchmark values for chromium or nickel. On the basis of the results reported, chromium and nickel releases would not have been expected to cause any adverse health effects within Ground Zero.

Approximately 512 monitoring samples collected at sites surrounding Ground Zero were evaluated for chromium, including 86 samples taken at the Staten Island landfill and 16 samples from personal air monitors worn by NYC fire department personnel. Approximately 637 monitoring samples collected at sites surrounding Ground Zero were evaluated for nickel, including 86 samples taken at the Staten Island landfill. Samples were collected between September 23 and January 31. None of the samples evaluated for either chromium or nickel exceeded a screening benchmark. On the basis of the samples collected, chromium and nickel releases would not have been expected to cause any adverse health effects at sites surrounding Ground Zero.

Like most contaminants, however, elevations in chromium were seen in concentrations measured near Ground Zero, and near September 11 in time. At the Ground Zero monitoring, WTC building 5, chromium was not detected in four samples from September 23 to October 8, but then it was detected at 0.24 and 0.38 $\mu\text{g}/\text{m}^3$ on October 11 and October 15. Further sampling at Ground Zero through February of 2002 showed mostly non-detects (9 samples) and samples near typical background for chromium (4 samples between 0.02 and 0.07 $\mu\text{g}/\text{m}^3$), except for one higher reading at 0.22 $\mu\text{g}/\text{m}^3$ in January, 2002. Chromium sampling at all other sites around Ground Zero showed the same trend: elevations above typical urban background through about mid-October, with measurements then dropping to typical urban background (with a spike in January, which may or may not be due to Ground Zero emissions) through the sampling in February of 2002.

Unlike chromium, nickel was not found elevated above background at any time or location

in sampling. Measurements were mostly non-detects in within Ground Zero and in all locations measuring nickel, with sporadic measurements all less $0.1 \mu\text{g}/\text{m}^3$.

IV.c. Polychlorinated Biphenyls (PCBs)

PCBs are a group of synthetic organic chemicals potentially composed of 209 individual chlorinated biphenyl compounds (known as congeners). PCBs were manufactured as mixtures of individual compounds having 1 to 10 chlorine atoms on the molecule. Being relatively stable compounds, their high boiling points and resistance to breakdown by high temperatures made them useful in a broad array of industrial applications. Furthermore, as PCBs do not conduct electric current, they were useful for commercial purposes as insulating material and electrical dielectric fluid in transformers and capacitors.

In 1971, Monsanto Corporation, the major U.S. producer, voluntarily restricted the sales of PCBs to uses as dielectric fluids in “closed electrical systems.” This restriction was prompted by growing evidence of PCBs’ persistence in the environment, their tendency to bioaccumulate in animal tissues, and their toxic effects, namely as probable human carcinogens. Monsanto ceased PCB manufacture in mid-1977 and shipped the last inventory in October 1977 (Erickson, 1997). Regulations issued by EPA beginning in 1977, principally under the Toxic Substances Control Act (40 CFR 761), have strictly limited the production, import, use, and disposal of PCBs.

Because the WTC was built in the early 1970s, it can be surmised that PCBs may have been present or contained in transformers, capacitors, electrical insulating and cooling applications, fire-resistant coatings to building materials, and electrical fluorescent lighting fixtures. As a consequence of the collapse of the WTC towers, many of these materials were pulverized, ruptured, or burned, which caused PCBs to be released into the surrounding environment. Additionally, PCBs were likely entrained within the smoke plume that emanated from the debris piles at Ground Zero. The primary focus of this section is to evaluate the potential human health risks that may be associated with inhalation exposure to the variable air concentrations of PCBs measured in lower Manhattan in the aftermath of the disaster.

IV.c.1. Air Monitoring for PCBs

PCBs were monitored at 12 different sites around Ground Zero and in other areas of lower Manhattan. Several hundred ambient air samples were collected between September 16, 2001, and April 24, 2002. One-day samples were taken using a high-volume polyurethane foam (PUF) and glass fiber filter (GFF) sampler. The GFF is used to collect and retain PCB- contaminated particles that may be present in the air, whereas the PUF material is used to capture any gaseous form of PCBs. In this monitoring program, only the sum of the PCB congeners present in air was quantified. Figure 21 displays the locations of the PCB air monitoring stations in lower Manhattan.

The primary source of PCB monitoring data used in this analysis was the publicly accessible information posted on EPA’s WTC web site (<http://www.epa.gov/wtc>). This information is current through April 24, 2002. Table 3 presents a summary of the ambient air sampling results for PCBs in and near the WTC disaster site.

To put these measurements into perspective, background levels of total PCBs in air at urban locations in the U.S. are typically in the range of 1 to 8 ng/m³ (ATSDR, 2000a). Slightly elevated air concentrations were found up to 1 month after September 11 only at the Ground

Zero site, WTC Building 5 SW. The highest one-day PCB air measurement of 153 ng/m³ occurred on October 2, 2001. This level is approximately three-fold higher than the next two PCB levels observed at WTC Building 5 on September 16 and October 4: 55.9 and 58.6 ng/m³, respectively. By November 2, PCB levels at this site had further decreased to 18 ng/m³. Measurements from November 6, 2001 to April 24, 2002 showed that total PCB levels in air decreased to below the limits of detection. Barclay and West Broadway registered the next highest one-day PCB air concentration at 77 ng/m³ on October 4. This monitor bordered a restricted zone, but could represent a concentration in an area just above the corner of Barclay and West Broadway that was unrestricted after September 19. This October 4 measurement was about nine times greater than the measurement taken October 2 at this site (8.3 ng/m³), and approximately ten times higher than typical urban background air. By November 2, PCB air levels had decreased to 9.7 ng/m³. Following November 2, total PCB was not detected at Barclay and West Broadway until February 19, 2002, at which time a concentration of approximately 3 ng PCB/m³ was detected. From February 24, 2002, through April 24, 2002, PCB levels dropped below the limit of detection. Because the limits of detection were within the range of typical urban air measurements, it can be concluded that PCBs had dropped to and remained within this range of typical urban air concentrations after November 8.

To summarize, elevations above the typical background range of 1 - 8 ng/m³ were only seen in the initial month after 9/11, and only within Ground Zero and the border sampling location of Barclay and West Broadway. All monitoring sites to the west of Ground Zero showed no elevation above background PCB concentrations at any time in the month following the disaster. By November 8, PCB levels in air were within the range of expected urban background air at all monitoring locations, including Ground Zero.

IV.c.3. Potential Human Health Consequences of Exposure to PCBs in Air

Different approaches are used here to assess potential health effects of exposure to PCBs at or near the WTC site. First, EPA's procedure for estimating cancer risk is used. Then, comparison of air concentrations to benchmarks published by ATSDR, NIOSH, and OSHA are conducted.

EPA currently classifies PCBs as B2 carcinogens; a probable human carcinogen (IRIS, 2002). The basis for this classification stems largely from long-term animal studies supplemented with human studies. A 1996 study found liver tumors in female rats exposed to Aroclors 1260, 1254, 1242, and 1016, and in male rats exposed to 1260. These mixtures contain overlapping groups of congeners that, together, span the range of congeners most often found in environmental mixtures. Earlier studies found high, statistically significant incidences of liver tumors in rats ingesting Aroclor 1260 or Clophen A 60 (Kimbrough et al., 1975; Norback and Weltman, 1985; Schaeffer et al., 1984). Mechanistic studies are beginning to identify several congeners that have dioxin-like activity and may promote tumors by different modes of action.

PCBs are absorbed through ingestion, inhalation, and dermal exposure, after which they are transported similarly through the circulatory system. This pattern provides a reasonable basis for expecting similar internal effects from different routes of environmental exposure. Information on relative absorption rates suggests that differences in toxicity across exposure routes are small. The human studies are being updated; currently available evidence is

inadequate, but suggestive of PCB carcinogenicity.

From the dose-response data derived from animal studies, the EPA has calculated an upper bound cancer slope factor of $1 \times 10^{-4} [\mu\text{g}/\text{m}^3]^{-1}$ associated with continuous lifetime inhalation exposure to PCBs. This slope factor pertains to exposure to total PCBs, which may or may not contain dioxin-like PCBs. For exposure to dioxin-like PCB congeners alone, the slope factor developed for dioxin-like compounds should be applied (EPA, 2000a). This assessment does not consider exposure and risk from dioxin-like PCBs because these congeners were not measured separately. The Toxic Equivalent (TEQ) concentrations discussed in the dioxin section below are specific to dioxin and furan congeners only.

A cancer risk from a less-than-lifetime inhalation exposure to total PCBs is given as:

$$\text{Risk} = \text{LAC} * \text{UR} \quad (2)$$

where LAC is the air concentration averaged over a lifetime, calculated as: $\text{AC} * [\text{ED}/\text{LT}]$, where AC is the average air concentration during the period of exposure ($\mu\text{g}/\text{m}^3$), ED is the exposure duration (days), LT is lifetime (days), typically 70 years, and UR is the unit risk factor, expressed in units of 1/concentration.

In order to conduct a simple screening exercise to evaluate the cancer risk from inhalation of elevated levels of PCBs near the WTC site, a representative air concentration and a time during which exposure to that concentration occurred need to be determined. The areas in which PCB air concentrations were elevated were generally located within the “restricted zone”. Still, even if an individual were exposed to the highest concentration found at $153 \text{ ng PCB}/\text{m}^3$ for a period of 1 month (and all the data suggests that elevations did not exist beyond 1 month), the lifetime cancer risk would be estimated at about 2×10^{-8} (calculated as: $[0.153 \mu\text{g}/\text{m}^3] * [30 \text{ days}/(70 \text{ years} * 365 \text{ d/yr})] * [1 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}]$). EPA regulatory programs, such as the Superfund Program, typically consider individual incremental cancer risk estimates made in this manner (i.e., in the context of a scenario-based risk assessment) in the range of 10^{-4} to 10^{-6} to be of potential significance, depending on the circumstances. On this basis, an incremental cancer risk estimate in the range of 10^{-8} is judged to be insignificant.

ATSDR has published a Toxicological Profile for PCBs (ATSDR, 2000a). This Toxicological Profile is a comprehensive review and summary of existing health effects information relevant to human exposures. From this review, it is concluded that all the measured PCB levels in air in or around the WTC site were well below the levels of significant exposure to PCB mixtures that were found not to cause adverse effects in experimental animals as a consequence of short-term inhalation exposure. These no-observed-adverse-effect-levels (NOAELs) in experimental animals ranged from $1.5 \times 10^6 \text{ ng}/\text{m}^3$ for renal effects to $9 \times 10^3 \text{ ng}/\text{m}^3$ for hepatic effects. These NOAELs are one to six orders of magnitude higher than the highest PCB air levels measured in Lower Manhattan.

Occupational exposure limits provide an additional perspective by which to evaluate potential non-cancer health effects that may be associated with inhalation exposure to PCBs measured in the air at or near the WTC site. NIOSH publishes RELs and OSHA publishes PELs

for occupational exposures to chemical contaminants. The NIOSH REL is 1×10^3 ng/m³ as an 8-hr TWA air concentration (NIOSH, 2002). The REL is associated with long-term or repeated exposures and is protective of effects on the liver and the reproductive system. All PCB air measurements at or near the WTC site have been below the NIOSH REL. The OSHA PEL is 5×10^5 ng/m³ as an 8-hr TWA air concentration (NIOSH, 2002). The PEL is associated with long-term or repeated exposures and is protective of effects on the skin (dermatitis). All PCB air measurements at or near the WTC site have been below the OSHA PEL.

Table 3. Summary of PCB monitoring data between September, 2001, and April, 2002.

Location	Sampling Date	Concentration ng PCB/m ³ air	Sampling Date	Concentration ng PCB/m ³ air
Albany & Greenwich	9/16 - 10/4; 7 samples	ND	11/02-12/11; 10 samples	ND
	10/8	9.2	12/19/01	2.2
	10/11 - 10/26; 4 samples	ND	12/27 - 4/24; 29 samples	ND
	10/30/01	1.8		
Albany & South End	9/16 - 4/24; 53 samples	ND		
Barclay & West Broadway	9/16/01 & 9/23/01	ND	10/15 - 10/30; 4 samples	ND
	9/27	38.7	11/02	
	10/2	8.3	11/8 - 2/14; 23 samples	ND
	10/4	77.0	2/19	3.2
	10/8	ND	2/21 - 4/24; 15 samples	ND
	10/11	25.0		
Church & Dey	9/16	7.0	10/8	4.5
	9/23	4.0	10/11	2.2
	9/27	5.0	10/15 - 10/26; 3 samples	ND
	10/2	17.9	11/02	3.3
	10/4	13.3	11/8 - 4/24; 39 samples	ND
Church & Vesey	11/15 - 11/27; 4 samples	ND	12/11	1.4
	12/4	1.7	12/19 - 4/24; 30 samples	ND
	12/6	ND		
EPA TAGA Lab	9/11 - 4/24; 54 samples	ND		
Liberty & Trinity	9/16	8.1	9/23 - 4/24; 52 samples	ND
Liberty & Broadway	9/23 - 9/28; 3 samples	ND	10/8	16
	10/2	8.2	10/11 - 10/26; 4 samples	ND
	10/4	ND		
Rector & South End	9/16 - 4/11; 51 samples	ND		

Table 3. Summary of PCB monitoring data between September, 2001, and April, 2002 (cont'd).

Location	Sampling Date	Concentration ng PCB/m ³ air	Sampling Date	Concentration ng PCB/m ³ air
Vesey & West	9/16 & 9/27	ND		
Liberty & South End	9/23 - 5/28; 67 samples	ND		
WTC Building 5 SW	9/16	55.9	10/15	16.0
	10/2	153.0	10/18	5.7
	10/4	58.6	10/26	6.8
	10/8	18.1	11/2	18.2
	10/11	28.2	11/6 - 4/24; 38 samples	ND

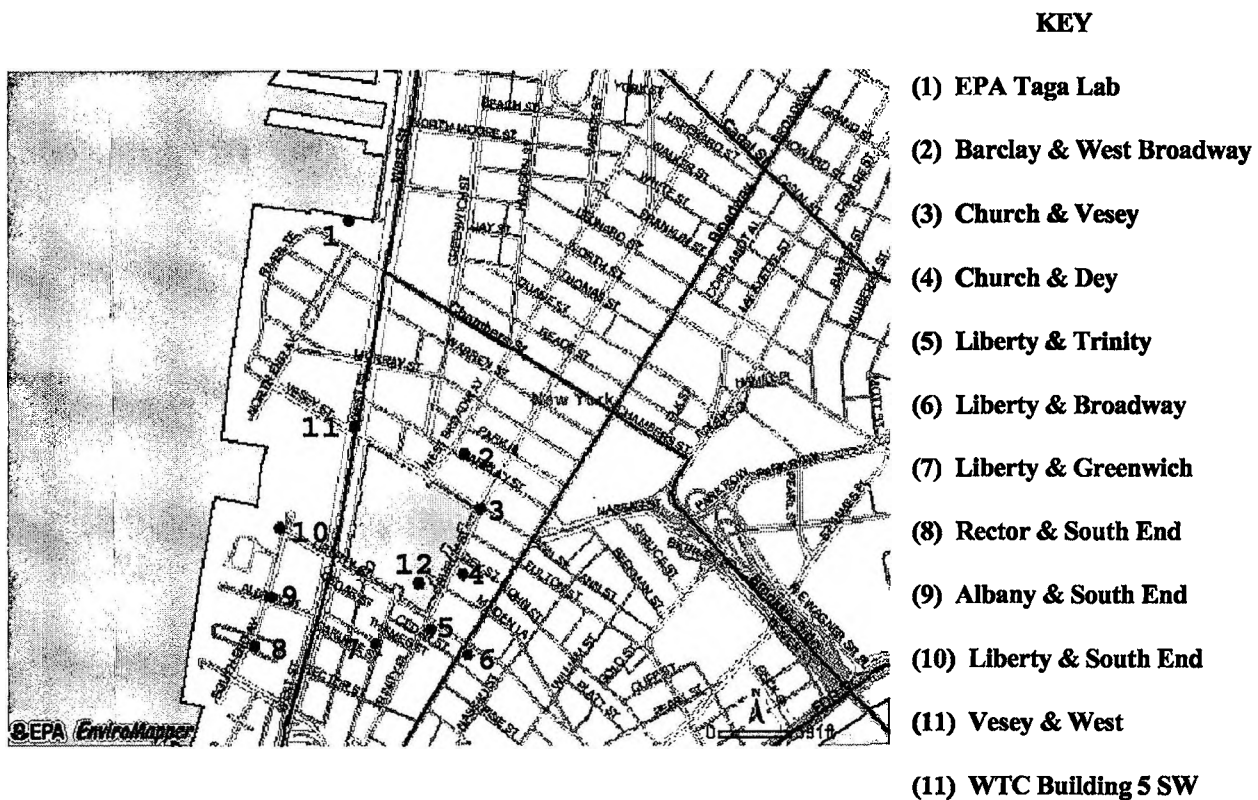


Figure 21. Location of PCB monitoring stations.

IV.d. Dioxin

Dioxin-like compounds (referred to also in discussions below simply as “dioxins”) are formed during combustion, and it is expected that production of these compounds would have resulted from the initial impact and ensuing fires at the WTC. Dioxin-like compounds may be formed in other ways as well, such as in the chlorine bleaching process for paper products or in the manufacturing process for certain chlorinated organic chemicals. However, uncontrolled or improperly controlled combustion appears to be the major source of new emissions (EPA, 2000). A total of 30 compounds are considered to be “dioxin-like”: 7 polychlorinated dibenzo-p-dioxins (abbreviated dioxins), 10 polychlorinated dibenzofurans (furans), and 13 coplanar PCBs. These individual compounds are called “congeners.” Measurements at the WTC included only the 17 polychlorinated dioxin and furan congeners, not the PCB congeners (total PCBs were measured, as discussed above). Because dioxin-like compounds are present at minute quantities, concentrations in this section will be described in terms of picograms per cubic meter (pg/m³).

Concentrations of dioxin-like congeners are expressed on a toxic equivalent, or TEQ basis. A TEQ concentration is calculated as the sum of the toxically equivalent concentrations of each of the 17 congeners. A congener's TEQ concentration is calculated as its concentration (C_i) times its toxicity equivalency factor, or TEF_i. TEF values are equal to 1.0 or less, and relate the toxicity of 16 of the 17 congeners to the most toxic congener, 2,3,7,8-TCDD (the 17th congener naturally assigned a TEF of 1.0). An overall TEQ concentration is, therefore, $\Sigma(\text{TEF}_i * C_i)$. When a congener was not detected in the sample, a value of one-half the detection limit was used for that congener in calculating the TEQ concentration. This is the traditional approach to calculating a TEQ concentration when some of the congeners are not detected and others are; alternate approaches include assigning either 0 or the detection limit to the congeners that are not detected.

The TEQ concentrations on the WTC web site were developed using the “International” set of TEF values (I-TEF; EPA, 1989). In 1998, the World Health Organization proposed a new set of TEF values (WHO-TEF; Van den Berg, 1998). The principal change relevant to quantifying the TEQ concentration of a mixture comprised of the 17 dioxin and furan congeners is that the TEF for the penta dioxin-like congener, 1,2,3,7,8-PCDD, has been increased from 0.5 to 1.0. The other change of less impact to calculating the TEQ concentration is that the TEF values of the two octa congeners, OCDD and OCDF, were reduced from 0.001 to 0.0001. Since the penta congener occurs in most samples, the net effect of this change is to increase the TEQ concentration slightly, in the range of 5-10%, depending on the prevalence of 1,2,3,7,8-PCDD in the sample. For example, on the sample taken from the WTC Building 5 monitor on September 23, when the I-TEQ concentration was 161 pg TEQ/m³, the 1,2,3,7,8-PCDD concentration was 40.8 pg/m³. In the I-TEF scheme, this contributes 20.4 pg/m³, but in the WHO-TEF scheme, this would add 40.8 pg/m³ to the final TEQ concentration. Therefore, the measurement of 161 pg TEQ/m³ would increase to 181 pg TEQ/m³ when switching to the WHO-TEF scheme, an increase of about 12%. An examination of the data shows that other I-TEQ concentrations would also increase, some by less than 5%.

This assessment uses the I-TEQ concentrations presented on the EPA WTC web site. Converting to WHO-TEQ concentrations would make a small and insignificant change to the

calculations presented in this section, would not result in changing any of the principal findings, and would possibly be confusing to those who notice a difference in the concentrations described here compared to those posted on the EPA web site.

Some of the major health risks associated with dioxin exposure include, but are not limited to, cancer and noncancer effects, including reproductive, developmental, and immunologic effects. A complete discussion of the health effects of exposure to dioxin-like compounds can be found in EPA's draft Dioxin Reassessment Document (EPA, 2000). This WTC report draws on procedures outlined in that document for conducting simple screening assessments relating to potential long-term cancer and noncancer effects from exposure to dioxin concentrations measured on and near the WTC site.

IV.d.1. Dioxin Air Monitoring Data

Figure 22 shows the location of 16 air monitoring sites at which dioxin measurements have been taken. Four of the sites had very few samples, so the focus in this section is on the 12 samplers that had a significant number of samples. At all sites, high-volume air samplers were used. Each sampler contained both a GFF and a PUF cartridge. The GFF is used to collect and retain particle-phase dioxins, whereas the PUF material is used to capture gas-phase dioxins. The GFFs and PUFs were sent to laboratories for measurement of the 17 dioxin-like congeners using EPA method SW8290.

Two different groups conducted the dioxin monitoring. Nine of the 12 sites measuring a significant number of dioxin samples were "lettered" sites managed by the EPA's Environmental Response Team (EPA ERT). These 9 sites sampled for 8 hours per sampling event. On the other hand, 3 of the 12 sites were "numbered" sites were established by the New York State Department of Environmental Conservation (NYSDEC), and they monitored for 72 hours per sampling event. For dioxin sampling, these 3 NYSDEC samplers were managed by EPA's Region 2 personnel, who sent the samples to EPA's Region 7 laboratory for analysis. For this reason, these will be referred to as the Region 2/7 monitors/samples.

The amount of time the monitor operates directly affects the amount of air that went through the monitors for dioxin collection: the Region 2/7 sampling captured dioxins contained in about 1000 m³ of air (i.e., about this much air was drawn into the sampler over 72 hours), whereas the EPA ERT sampling captured dioxins in about 7 m³ air. The majority of the EPA ERT samples simply did not contain enough mass of dioxins to be able to detect, much less quantify, the dioxin-like congeners in the sample. When the method cannot detect the congener in the sample, a result of "nondetect" (ND) is reported, and the detection limit (DL) is supplied. The method's detection limit is calculated by dividing the lowest amount of dioxin that it can detect (on the GFF and PUF) by the air that contained that amount, namely the air that flowed through the sampler. The greater the air flow, the greater the divisor for the unchanging detection amount, and the lower the detection limit. Except for the high concentrations measured within and near Ground Zero from September through late November, most of the samples taken by the EPA ERT samplers contained mostly non-detects. Therefore, TEQ concentrations ended up being calculated with all or most congeners set equal to a value of one-half the detection limit.

Calculating TEQ concentrations with most congeners assigned values of one-half detection limit is not an issue when a sufficient quantity of air is drawn into the monitor, and the inability to measure the dioxin in the sample truly does signify a very low amount of dioxin in the sample. For example, congeners in the Region 2/7 samples could be quantified if they were present at concentrations higher than about 0.5 pg/m³ because enough air was drawn into the monitors leaving behind enough dioxin molecules on the monitor's filters. The congeners in the EPA ERT samples could not be quantified unless they were present at about 5-10 pg/m³. When most of the congeners reported a non-detect in one of the Region 2/7 samples, the calculated TEQ concentration was in the range of 0.01 to 0.10 pg TEQ/m³, whereas when most of the congeners were reported as non-detect in the EPA ERT samples, the calculated concentration ranged from 0.5 to 5.0 pg TEQ/m³. As will be discussed below, typical urban air concentrations are in the range of 0.10 to 0.20 pg TEQ/m³, and these measurements come out of studies where, like the Region 2/7 samples, a sufficiently large quantity of air was drawn into the monitors.

Assigning one-half detection limit for non-detects is typical and appropriate for evaluating exposure to airborne contaminants, dioxins or otherwise. In some circumstances, this practice can underestimate the amount of contaminant in the air - i.e., when the actual concentration is just below the detection limit and above the half-detection limit value. However, in this case, it is clear based on comparison with the Region 2/7 samples and from other historical measurements around the United States, that the practice of assigning one-half detection limit to non-detects in the EPA ERT samples resulted in an overestimate of the TEQ concentration when most of the congeners in the sample were non-detects.

For purposes of analysis in this section, a limited set of the dioxin data is used. Table 4 shows reported TEQ concentrations for the WTC Building 5 EPA ERT monitor, the Church & Dey EPA ERT monitor, and the Park Row Region 2/7 monitor. These values were calculated at ND = ½ DL; in parenthesis for the two EPA ERT monitors is the TEQ calculated at ND = 0. The WTC Building 5 monitor had the highest concentrations, and the Church & Dey monitor was in the predominant downwind direction for most monitoring events and had the second highest measurements. The Park Row monitor began operation on October 12 and had numerous measurements through March of 2002. Figure 22 shows the location of these three monitors. The following observations are based on the data in Table 4:

- The WTC and Church & Dey measurements from the first measurement day of September 23 through November 21 show unambiguous elevation, with concentrations ranging from about 10 to 170 pg TEQ/m³.
- In this same time frame and for these two samplers, the influence of having high detection limits is seen in a few samples (WTC sample on 10/4: 176 pg TEQ/m³ at ND = ½ DL versus 140 pg TEQ/m³ at ND = 0; WTC sample on 10/11: 52.4 versus 9.6), but mostly, the congeners were detected and quantified, and TEQs were similar at ND = 0 and ND = ½ DL.
- The 6 Park Row measurements between October 12 and October 29 averaged 5.6 pg TEQ/m³. These measurements are consistent with the mid- to late-October measurements at Church & Dey, which is slightly off-site from Ground Zero, of

10 to 20 pg TEQ/m³. Further, the Church & Dey measurements of 10 - 20 pg TEQ/m³ for mid to late October are consistent with the WTC measurements for that time period of 20 - 50 pg TEQ/m³. In other words, the highest measurements are onsite (WTC); the next highest measurements are slightly offsite (Church & Dey), and slightly lower concentrations are farther offsite (Park Row). This is strong evidence that emissions from the WTC site are the cause for elevated air concentrations within and near the WTC site.

- The Park Row measurements from December 2001 through the last reported measurements of March 18, 2002, are less than 0.11 pg TEQ/m³, and all the 2002 measurements are less than or equal to 0.05 pg TEQ/m³. These values are consistent with those of the other two Region 2/7 samplers (see Figure 22). They are also consistent with typical urban concentrations of dioxin TEQs (see discussion after bullets), which leads one to believe that these concentrations might be representative of typical “background” New York conditions.
- The December through April measurements from the WTC Building 5 and Church & Dey monitors are nearly all non-detects. The reported concentrations average about 1.4 pg TEQ/m³, but this is of limited interpretive value since the detection limits were too high. The actual concentrations could be more like the concentrations of about 0.05 pg TEQ/m³ found at the Park Row sampler, but it is not possible to conclude that since the data are not available.

Other measurements made in the United States and around the world can be used to put these measurements in perspective. As noted earlier, EPA’s draft Dioxin Reassessment compiled urban and rural air monitoring studies and found average ambient concentrations of 0.12 pg TEQ/m³ for urban and 0.017 pg TEQ/m³ for rural settings. Higher concentrations have been identified in the literature, particularly near a known source of dioxin emissions. The highest TEQ concentration reported in the U.S. was > 1.0 pg/m³, downwind of an incinerator in Niagara Falls, NY. Concentrations in the plume of a solid waste incinerator in Columbus, OH, that was known to be emitting large amounts of dioxins were about 0.25 pg TEQ/m³. In this case, the stack was very tall (about 80 meters) and air measurements were taken about 2 kilometers away. Background air concentrations in Columbus were measured to be about 0.05 pg TEQ/m³.

Air concentrations near an incinerator in Japan adjacent to a U.S. Naval Air Base were regularly measured on the base for dioxin-like compounds. Measurements at the nearest downwind monitor, at about 200 meters, averaged 3.5 pg TEQ/m³ for weekly samples over a 15-month period. For five samplers (including the nearest downwind sampler) at various locations on the base up to 800 meters, the average air concentration was 1.6 pg TEQ/m³. An examination of the air concentration data on this base in conjunction with wind rose data, suggested that a background air concentration in this area was less than 0.5 pg TEQ/m³ and that measurements above that were due to the influence of the incinerator (Walker, et al., 2002). Other measurements in cities in Japan have been in the 0.3 to 0.7 pg TEQ/m³ range, and these are the highest that have been reported as typical air concentrations worldwide.

Although none of these literature measurements can be assumed to represent New York levels, they provide some basis for perspective. Certainly, no reports in the literature could be found on similar circumstance where there is, what is essentially, an area source at ground level continually emitting dioxins near to where individuals are exposed. It would be reasonable to conclude that the concentrations to which individuals could potentially be exposed, in the range 10.0 to 170.0 pg TEQ/m³ within and near the WTC site found through the latter part of November, are likely the highest ambient concentrations that have ever been reported.

IV.d.2. Potential Human Health Consequences of Exposure to Dioxins in Air

The exposure of humans to dioxins is predominantly through the food supply; about 95% of exposure is through consumption of animal food products. Inhalation exposure and absorption via the skin are generally minor pathways for the average U.S. citizen (i.e., the background population). EPA has estimated that about 4% of the background human dose of dioxin is due to inhalation, based on an average urban level of 0.12 pg TEQ/m³ and an inhalation rate of 13.3 m³/day. If the concentration of dioxin in the air is increased, the amount of exposure due to inhalation would be increased, but the total amount of dioxins contributed by the inhalation pathway might still be small in comparison to the contribution made by food ingestion.

In the WTC situation, elevated dioxin concentrations in the air can be expected to increase the proportion and extent of human exposure via inhalation and possibly skin contact routes for those people residing or working close to the WTC location. The extent to which these elevated atmospheric levels will translate into increased human doses depends on an individual's pattern of exposure, considering, for example, his or her location in relation to the WTC and downwind areas; the duration and time period of exposure such as workshift and return to residence patterns, movement and activity patterns during the time of elevated atmospheric levels; and whether respiratory protection devices were used. Dioxin-like compounds exist substantially in the atmosphere attached to particles, and these devices would remove a substantial portion of the dioxins through the removal of particulates; if these respirators contained a carbon filter, a substantial portion of the dioxins in vapor phase would also be removed.

This section will use the data shown in Table 4 to conduct cancer and non-cancer assessments of an individual's inhalation exposure to dioxins. The procedures used are described in detail in the draft Dioxin Reassessment (EPA, 2000; available at, <http://www.epa.gov/ncea/dioxin.htm>), with further references supplied below as necessary.

1. Daily Inhalation Doses

Cancer and non-cancer assessments entail the development of a "dose" term, which in this case is the dose received via inhalation. Inhalation dose estimates require assumptions about the hourly rate of inhalation (m³/hr), the number of hours per day a person inhales at the site where he or she could be exposed (which could obviously be less than 24 hours if the individual does not live in the vicinity where air concentrations measurements were taken), the time period during which this exposure occurs, and, of course, the air concentration to which the individual is exposed.

Daily inhalation exposure dose is given by:

$$DD = [IN * HRD * C * ABS]/[BW] \quad (3)$$

where DD is daily dose (pg TEQ/kg-day); IN is the inhalation rate (m³/hr), HRD is the hours/day of inhalation, C is the concentration (pg TEQ/m³), ABS is the fraction of contaminant inhaled which is absorbed (unitless), and BW is the body weight (kg). The draft Dioxin Reassessment assumes an adult body weight of 70 kg and an absorption fraction of 0.8 for TEQ exposures, both from inhalation and food consumption, and these assumptions are used here. The average daily dose (ADD) is calculated simply as the average of the daily doses over the period of exposure.

2. *Exposure Scenarios*

The scenarios which were evaluated here include:

1) WTC worker: This individual was exposed 10 hours per day, 5 days per week, in the time period between September 12 and November 30, 2001. This time frame roughly corresponds to the time when it seemed clear that dioxin air levels were elevated according to the monitoring data. Measurements from the WTC Building 5 monitor represent the concentration to which this individual is exposed. A time-weighted average (TWA) air concentration was derived for this period. The air concentration between September 12 and September 23 (the date of the first measurement at the WTC monitor) is assumed to be equal to the September 23 measurement, and the concentration between November 8 (the last date in November for the WTC monitor) and November 30 was similarly assumed to be equal to the measurement on November 8. These seem to be reasonable assumptions since the September 23 measurement from the WTC monitor was the second highest found at 161 pg TEQ/m³, and the November 8 measurement of 5 pg TEQ/m³ reasonably reflects the downward trend of measurements at the site. Between September 12 and November 30, a TWA air concentration was derived to represent the concentration to which the workers were exposed. A TWA concentration is not the same as the simple average of concentrations measured. To derive the time-weighted concentration, concentrations were assigned to each day between September 12 and November 30. From one measurement to the next, air concentrations were assumed to linearly rise or fall. For example, if the concentration was measured as 100 pg TEQ/m³ on one day and 5 days later it was 50 pg TEQ/m³, the concentrations assigned to the intervening days were 90, 80, 70, and 60 pg TEQ/m³. The TWA concentration is then simply the average of all the concentrations assigned to days when the worker was assumed to be exposed. With these assumptions, the average TEQ concentration during this time was calculated as 60.7 pg/m³. The rate of inhalation for a WTC worker was 1.3 m³/hr. This is equivalent to the rate for a "laborer" as quantified in EPA's Exposure Factors Handbook (EPA, 1997).

2) Office worker: This individual is exposed 10 hours per day, 5 days per week, and the exposure began on September 19, corresponding to the time when individuals were allowed back into office buildings outside of the Ground Zero site itself but in areas initially "restricted" near Ground Zero. The office worker was assumed to be exposed to air concentrations measured by the Park Row monitor. This simplistically assumes that the air concentrations within office buildings near Ground Zero were similar to air concentrations outside of office buildings this close to Ground Zero. The period of exposure was September 19 to November 30, and the same strategy to derive TWA concentrations was used as for the WTC worker. The TEQ air

concentration from September 19 to October 12 was assumed to be 8.4 pg/m^3 , the measurement on October 12 at the Park Row monitor, and the TWA concentration between September 19 and November 30 was 4.8 pg TEQ/m^3 . The rate of inhalation for an office worker was assumed to be the average inhalation rate of $1.0 \text{ m}^3/\text{hr}$, which is defined as “light activity” in EPA’s Exposure Factors Handbook (EPA, 1997).

3) Resident: This individual was exposed 24 hours per day, 7 days per week, and the exposure began on September 19 and ended on November 30. As with the office worker scenario, the assumption here again was that individuals living near Ground Zero, in the vicinity of the Park Row monitor, were more exposed after September 19, when some of the locations outside of Ground Zero opened up to workers and residents. The daily inhalation rate was $0.55 \text{ m}^3/\text{hr}$, an average daily rate, activity unspecified, as developed in EPA’s Exposure Factors Handbook (EPA, 1997). As noted, the air monitor used to characterize the air concentration was the Park Row monitor, which was located in an area not restricted after September 19. The average TEQ concentration during this time was 4.8 pg/m^3 , as noted above.

3. Procedure for Cancer Risk Estimation

These assumptions and the resulting ADDs are shown in Table 5. Cancer risk were estimated simply as:

$$\text{LADD} = \text{ADD} * [\text{ED}/\text{LT}] \quad (4a)$$

$$\text{Risk} = \text{LADD} * \text{SF} \quad (4b)$$

where LADD is the lifetime daily dose (pg TEQ/kg-day), Risk is the upper bound incremental excess lifetime cancer risk that results from the exposure described by LADD, ADD is the average daily dose during the period of exposure (pg TEQ/kg-day), ED is the exposure duration (days), and LT is lifetime (days), typically 70 years, and SF is the upper bound cancer slope factor, expressed in inverse units to LADD, or $[\text{pg TEQ/kg-day}]^{-1}$. The SF of $.000156 [\text{pg/kg-day}]^{-1}$ was developed by EPA in 1984 for 2,3,7,8-TCDD exposures (EPA, 1984). It is applied to TEQ exposures in this cancer risk screening exercise. The draft Dioxin Reassessment (EPA, 2000) proposed an SF of $0.001 [\text{pg/kg-day}]^{-1}$, which applies to dioxin TEQ exposures.

4. Procedure for Non-Cancer Risk Estimation

For noncancer risk, a different approach was taken. The best indicator of exposure for persistent, bioaccumulative, toxic substances such as dioxin is the concentration of the chemical in the organ or tissue of concern. A common metric for dioxin exposure is the “body burden”, which is defined as the concentration of dioxins in the body, typically on a whole-weight basis. Body burden in this screening assessment is expressed on a lipid basis. It is assumed that adults are 25% lipid by weight, so that a lipid-based concentration can easily be converted to a whole-weight-based concentration by multiplying by 0.25.

Dioxins build up and decline over prolonged periods of time, since the overall biological half-life (the time for half the chemical to dissipate by either biological degradation or elimination) of dioxins in the human body is approximately 7 years. The use of the body burden as the measure of dose has implications for short-term exposures, such as those near the WTC

site, where elevated exposure rates limited to a period of days or months contributed to a pool of dioxin already accumulated in the human body over a lifetime. The current estimated body burden of dioxin (including only the 17 dioxin and furan congeners, not the dioxin-like PCB congeners discussed above) in U.S. adults is approximately 18 pg TEQ per gram of body lipid (18 ppt TEQ lipid). This average was derived from data on older as well as younger adults. Because exposures were known to be higher in the past, the body burdens of younger adults will be lower than those of older adults. Another factor contributing to the variability seen in the entire population is dietary pattern; individuals whose diets are higher in animal fat will have higher body burdens.

The effects of dioxin in humans range from biochemical changes at or near background levels to potentially adverse effects of increasing severity as body burdens increase above background levels. The “margin of exposure”, MOE, can be defined as the ratio of body burden where effects are found divided by a body burden at a level of interest. The MOE for dioxin at current average body burdens (i.e., current average body burdens being the level of interest) is considerably less than that typically seen for environmental contaminants of toxicological concern. The potential contribution to health risks from specific dioxin sources or specific exposures, such as exposures from inhalation of air with elevated levels of dioxin, is best evaluated through calculating the incremental contribution of this source to the body burden.

The draft Dioxin Reassessment has assumed that a one-compartment, first-order pharmacokinetic (PK) model can be used to estimate the body burden that results from a specific intake regime. This simple PK model and its application to dioxin TEQs is also described in Lorber (2002). For an exposure of a finite time, the nonsteady-state form of this model to predict an increment in body burden (IBB) from a constant intake dose is given by:

$$IBB = [ADD/(k * LW)] * [1 - e^{-kt}] \quad (5)$$

where IBB is the increment of body burden on a lipid basis (pg/g, or ppt, lipid basis); ADD is the average daily dose (pg TEQ/day; not on a body weight basis), k is the first-order dissipation rate constant (1/day), LW is the weight of body lipids (g; equal to full body weight times 0.25, as described above), and t is the time of exposure (days). Use of Equation (5) with an average ADD over the period of exposure will provide an estimate of body burden at the end of the exposure. This is the time when the incremental body burden will be at its largest. In the scenarios of this assessment, different daily exposures result from different air concentrations as well as differences in exposure - 5-day work week followed by 2 days of non-exposure for the office worker scenario. Equation (5) is applied on a daily time step using Excel® spreadsheet procedures for this simple screening exercise.

A value of 17,500 g for the lipid weight (calculated as: 70 kg * 0.25 lipid fraction * 1000 g/kg), and a k of 0.000267 day⁻¹ (= 0.098 yr⁻¹, corresponding to a 7.1 year half-life) will be used (Lorber, 2002). Results for this exercise include both an incremental body burden estimate, the IBB of Equation (5), calculated at the end of the exposure period, as well as a percent increase over background this represents. This percent increase is calculated as, [IBB/BK] * 100%. The BK is the background, which was assigned a value of 18 ppt TEQ lipid, as described above.

5. Results and Discussion

The exposure assumptions, the cancer risk estimates, and the incremental body burdens, are shown in Table 5. Before commenting on these results, it is important that they be put into perspective for general U.S. population dioxin TEQ exposures. As noted above, the background body burden of dioxin TEQs in adults is 18 ppt lipid. The draft Dioxin Reassessment estimates that the general adult population background TEQ exposure is 65 pg/day, or, expressed on a body-weight basis, 0.93 pg TEQ/kg-day. If this exposure is experienced over a lifetime, then the resulting incremental cancer risk from background TEQ exposure to the general adult population is equal to about 1.4×10^{-4} (assuming the 1984 SF of $0.000156 \text{ [pg/kg-day]}^{-1}$).

Table 5 shows that the TEQ ADD due only to inhalation during the period of exposure is 9 pg/kg-day for the WTC worker, 0.55 pg/kg-day for the office worker, and 0.73 pg/kg-day for the nearby resident. Although the WTC worker's daily exposure is higher than that of the general U.S. population, it is experienced for only a small number of days. Therefore, when averaged over a lifetime, the WTC worker dose calculates to an incremental cancer risk that is 3×10^{-6} , which is about 2 orders of magnitude lower (100 times lower) than the U.S. background cancer risk from dioxin-like compounds (1.4×10^{-4} as calculated above). The office worker and resident experience incremental lifetime cancer risk at about 3×10^{-7} , three orders of magnitude lower (1000 times lower) than background. EPA regulatory programs, such as the Superfund Program, typically consider individual incremental cancer risk estimates made in this manner (i.e., in the context of a scenario-based risk assessment) in the range of 10^{-4} to 10^{-6} to be of potential significance, depending on the circumstances. Exposure to dioxin-like compounds represents a unique circumstance, in that background exposures are already within this range and, in fact, at the upper end of this range. Therefore, although the upper bound incremental cancer risk to the WTC worker is estimated to be within the range of 10^{-4} to 10^{-6} , EPA judges these incremental cancer risks to be of minimal concern because they are 100 times and more lower than typical background exposures to dioxin-like compounds.

For noncancer risk, an increment of body burden, IBB, approach has been used. Table 5 shows that the exposure of the WTC worker suggests that his or her body burden could rise up to 10% above current average background, but that the nearby office worker and the residents have a rise of only 1% or less. EPA judges these incremental body burden increases to be of low significance, given the relatively high background exposures already experienced by the general population.

A key uncertainty remains as to the inhalation exposures that could be experienced by WTC rescue or clean-up workers, or nearby resident and office workers, who were in the area during the time period from about December onward. As discussed earlier, most of the samples had non-detects, but after assuming that the concentrations in the air were one-half the detection limit, all the measurements in 2002 from the EPA ERT samplers, which included the WTC Building 5 monitor, the Church & Dey monitor and many others, ranged from about 0.5 to 5.0 pg TEQ/m³, which is about 5 to 50 times higher than normal background air concentrations. The three Region 2/7 samplers, the Park Row, Chambers St, and the Albany & West samplers, reported concentrations near 0.05 pg TEQ/m³ from around December, 2001, through their last reported measurements in March, 2002. These were further away or generally upwind from the WTC site, so it cannot be assumed that they represent concentrations to which WTC workers and

others were exposed. Because the health risk from dioxin exposure is associated with accumulation of residues in body tissues, continued exposure throughout 2002 to dioxin, which was possibly elevated in the air, could not be evaluated. The risk screening exercises conducted for dioxin were limited to the time period when the concentrations were highest and dioxin was detected.

Table 4. Measured dioxin TEQ air concentrations at the WTC Building 5 monitor, the Church & Dey monitor, and the Park Row monitor (all units = pg TEQ/m³; NR = not reported; all TEQ calculated at ND = ½ DL except values in parenthesis, which are calculated at ND = 0).

Date	WTC - Building 5	Date	Church & Dey	Date	Park Row
9/23	161.0 (161.0)	9/23	139.0 (139.0)	10/12 ^a	8.35
9/27	NR	9/27	50.0 (NR)	10/14 ^a	0.34
10/2	175.0 (170.0)	10/2	59.3 (57.2)	10/15 ^a	4.78
10/4	176.0 (140.0)	10/4	51.9 (50.6)	10/16 ^a	7.55
10/8	32.0 (28.7)	10/8	17.7 (15.5)	10/26	6.51
10/11	52.4 (9.6)	10/11	15.6 (11.8)	10/29	6.34
10/18	NR	10/18	9.6 (8.8)	11/1	3.05
10/26	28.1 (24.9)	10/26	11.4 (10.2)	11/5	1.54
11/2	26.8 (25.4)	11/2	16.1 (15.1)	11/8	0.27
11/6	0.3 (0)	11/6	0.1 (0)	11/12	1.33
11/8	5.6 (4.9)	11/8	7.6 (7.1)	11/15	1.33
11/12	NR	11/12	1.3 (0.6)	11/19	2.50
11/15 ^b	5.4 (1.6)	11/15	3.4 (1.6)	11/22	1.30
11/21 ^b	4.1 (3.1)	11/21	10.0 (8.3)	11/26	0.80
No samples reported from 11/21 to 1/15		11/27	2.5 (NR)	11/29	0.16
		12/4	0.7 (NR)	12/3	0.12
Jan 15 - April 24 n = 31 reported range: 0.4-5.5 average: 1.4 at ND = ½ DL and 0.0 at ND = 0.		12/6	0.2 (NR)	12/6	0.04
		12/11	0.2 (NR)	12/10	0.05
		12/19	0.6 (NR)	12/13	0.04
		12/27	0.3 (NR)	12/24	0.04
		Jan 3 - April 24 n = 29 reported range: 0.2 - 4.1 average: 1.4 at ND = ½ DL and 0.0 at ND = 0.		12/27	0.06
				12/31	0.11
				Jan - Feb n = 17 all samples reported <= 0.05	

^a These Park Row samples were 24-hour samples; all other Park Row samples were 72 hour samples.

^b These two World Trade Center samples were actually taken at the Church & Vesey sampler, which was sometimes used in place of the WTC Building 5 sampler.

Table 5. Human exposure and health risk assessment assumptions and results for dioxin TEQs.

Description	WTC worker	Office Worker	Resident
I. Exposure Assumptions and Results			
Inhalation rate, m ³ /hr	1.3	1.0	0.55
Hours/day exposed	10	10	24
Days/week exposed	5	5	7
Air monitoring data used	WTC site	Park Row	Park Row
Period of exposure, dates/days	Sep 12- Nov 30, 57 working days	Sep 17 - Nov 30, 54 working days	Sep 12 - Nov 30, 79 days
Average TEQ air concentration, pg/m ³	61	4.8	4.8
Body weight, kg	70	70	70
Absorption, fraction	0.8	0.8	0.8
ADD, pg TEQ/kg-day	9.0	0.55	0.73
II. Cancer Risk Estimates			
Exposure Duration, yrs	0.16	0.15	0.22
LADD, pg TEQ/kg-day	2.0*10 ⁻²	1.2*10 ⁻³	2.1*10 ⁻³
Cancer Risk	3.1*10 ⁻⁶	1.9*10 ⁻⁷	3.6*10 ⁻⁷
Percent increase over 1.4*10 ⁻⁴ background risk	2.2 %	<1 %	<1 %
III. Body Burden Increases as a Measure of Potential NonCancer Risk			
Dissipation rate, 1/day	0.000267 ¹	0.000267 ¹	0.000267 ¹
Change in body burden, pg TEQ/g lipid	+1.86	+0.14	+0.20
Percent increase over 18.0 pg TEQ/kg lipid background	10 %	<1 %	1 %

¹This dissipation rate corresponds to a 7.1 year half-life in the body.

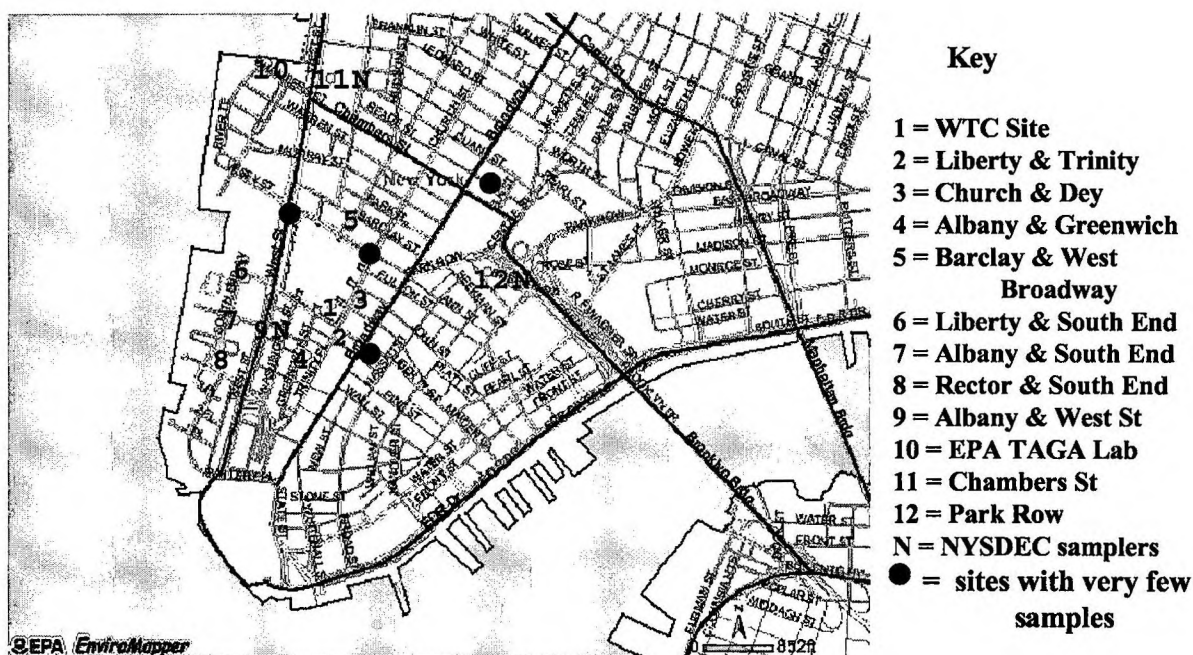


Figure 22. Location of dioxin air monitoring. The locations marked “N” are New York State Department of Environmental Conservation (NYSDEC) samplers maintained by EPA Region 2 with analysis of the samples by Region 7 (Region 2/7), whereas all other samplers are maintained by EPA’s Environmental Response Team (EPA ERT). See text for discussion of the differences in the two sets of data from these two air monitoring teams.

IV.e. Asbestos

Asbestos is a term used to describe a family of hydrated metal silicate minerals. Asbestos exhibits some special properties, such as high tensile strength, the ability to be woven, heat stability, and resistance to attack by acid and alkali. Thus, it was widely used for building fireproofing insulation and other purposes during the 1960s and early 1970s. At the peak of its demand, about 3000 applications or types of products were listed for asbestos. In 1973, EPA prohibited the spraying of asbestos-containing material on buildings and structures for fireproofing and insulation purposes. The use of asbestos has been sharply declining for more than two decades. Sprayed on asbestos was used to fireproof approximately the lower half of one of the WTC towers, and may have been used in other places in the towers as well. One of the reasons that asbestos has been so useful is that it exists in long, thin fibers that can be sprayed, woven or mixed. The extremely light and aerodynamic asbestos fibers also lead to their ability to become and remain airborne. The fibrous nature also contributes to the health effects associated with asbestos exposure.

There are six minerals whose fibrous forms are characterized as asbestos and that are currently regulated. All the six minerals also occur in non-fibrous forms and these forms are not known to cause any health effects. The six regulated asbestos fibers include one from the serpentine family of minerals: chrysotile, and five from the amphibole family: fibrous riebeckite (crocidolite), fibrous grunerite (amosite), actinolite asbestos, anthophyllite asbestos, and tremolite asbestos. Inhalation of these asbestos fibers has been linked to several adverse health effects including primarily fibrosis of the lungs (asbestosis), benign pleural plaques and thickening, lung cancer, and mesothelioma (a cancer of the thin membrane that surrounds the lungs and other internal organs). It also may increase the risk of cancer at other sites, but the evidence is not strong. Over the years the evidence has accumulated that longer thinner "asbestiform" fibers are of more concern for human health. The widely accepted definition of the asbestiform fiber is a particle having a length to diameter (aspect) ratio of $\geq 3:1$ and a length of at least $\geq 5 \mu\text{m}$. The evidence is overwhelming that both the mineral content and the size and shape of the fiber affects the severity of the disease. The respirable fibers are those with the diameter of $\leq 3 \mu\text{m}$. Fibers exceeding the diameter of $3 \mu\text{m}$ are considered to be non-respirable.

Asbestosis, a chronic, degenerative lung disease, has been documented among asbestos workers from a wide variety of industries. The disease is generally expected to be associated only with the higher levels of exposure commonly found in workplace settings (Brown et al., 1994; Case and Dufresne, 1997). Several researchers have found that asbestosis and lung cancer are associated with cumulative exposure to asbestos. Benign pleural plaques and thickening also have been linked to higher cumulative exposure to asbestos (Albin et al., 1996; de Klerk et al., 1993). Both asbestosis and benign pleural plaques result in reduced breathing capacity and mortality. In a review of the epidemiologic evidence for asbestosis exposure-response relationship, the World Health Organization Task Group on Environmental Criteria for Chrysotile Asbestos (WHO, 1998) concluded that "asbestotic changes are common following prolonged exposures of 5 to 20 f/mL." These prolonged exposures corresponded to cumulative exposure of 50 to 200 f/mL for a 10-year exposure period. They also concluded that "the risk at lower exposure levels is not known."

The majority of evidence indicates that lung cancer and mesothelioma are the most important risks associated with exposure to low levels of asbestos over a long period of time. There is ample evidence that all types of asbestos have been found to be associated with lung cancer. Several investigators have reported lung cancer mortality in workers exposed to chrysotile, amosite, crocidolite, anthophyllite, tremolite, and to multiple fiber types. The onset of exposure to time of occurrence of the disease is known as "latency period." As with most carcinogens, asbestos-related cancers have a substantially long latency period. The latency period for lung cancer has been reported to be 10 to 40 years. Most researchers have found that occurrence of lung cancer depends on the cumulative dose as well as other underlying lung cancer risk factors (U.S. EPA, 1986c; Peto et al., 1985). Similarly, several investigators have found that all types of asbestos cause mesothelioma of either pleura or peritoneum in adults who had occupational exposure. This finding also pertains to individuals who had no occupational exposure but who lived with a parent, spouse, or sibling who was an asbestos worker and presumably carried asbestos home on work clothes. Mesothelioma has a latency period of about 30 to 40 years. Lanphear and Buncher (1992) reviewed 1,105 mesothelioma cases in workers occupationally exposed to asbestos. They reported that 99% had a latency period >15 years and calculated a median latent period of 32 years. Further details on the toxicology and epidemiology of asbestos exposure can be found in the recent ATSDR Toxicological Profile for asbestos (ATSDR, 2001).

This next section reviews the air monitoring data for asbestos and discusses how these data relate to human health benchmarks developed for asbestos. The following sections discuss the analytical methods for measuring asbestos in air, the health risk benchmarks that measured air concentrations will be compared to, the background concentration of asbestos that can be compared to the measured values, and then the actual air data.

IV.e.1. Analytical Methods for Asbestos Ambient Air Measurements

The two analytical methods used in analyzing the WTC air samples for asbestos are phase contrast light microscopy (PCM) and transmission electron microscopy (TEM). PCM, although cheaper, is unable to distinguish between asbestos and nonasbestos fibers. It counts all fibrous structures with a minimum diameter of 0.3 μm and has a magnification range of 100 - 400X. Fibrous structures are defined as particles exhibiting a length of > 5 μm and an aspect ratio of length to width of 3:1. PCM cannot resolve internal structure or distinguish the mineralogy. PCM results are reported on a mass-per-volume basis, fiber per cubic centimeter (f/cc or f/cm³) or, equivalently, fiber per milliliter (f/mL). TEM, on the other hand, is more expensive, but it can count the fibrous structures with a diameter of < 0.01 μm , and it can resolve internal structure and distinguish mineralogy. It has a magnification range of 5,000 - 20,000X. TEM results may be reported as concentrations or, for comparison with EPA AHERA standards (see below), as structures per square millimeter (S/mm²) of filter in the ambient air monitor used. Details about the monitoring apparatus appropriate for measurement of asbestos using the TEM method and the appropriate ways to count and interpret the electron microscopy results are supplied in EPA (1987) and NIOSH (1994). Details about the PCM method for workplace measurements can be found in NIOSH (1994) and in OSHA (1994).

TEM data, expressed on a structures per filter unit area basis (as listed on the EPA web site), can be converted to a concentration in air in structures per cubic centimeter. This is

accomplished by multiplying the S/mm^2 term by a conversion factor defined as the area of the filter paper, mm^2 , divided by the volume of air, liters or cubic centimeters, that is drawn into the air monitor. The AHERA Final Rule establishing a $70 S/\text{mm}^2$ standard for asbestos in schools (EPA, 1987; see next section for more details on this benchmark), one of the standards used in this report to evaluate the WTC data, specifies that a volume at least 1199 L (liters; 1.199×10^6 cc) must be drawn into a monitor with a 25 mm filter (this filter size corresponds to an effective area of 385 mm^2) or that a volume of at least 2799 L (2.799×10^6 cc) must be drawn into a monitor with a 37 mm filter (effective area of 855 mm^2) when applying the standard. Therefore, the conversion factors for both filters, to convert S/mm^2 to S/cc , are $0.000321 \text{ mm}^2/\text{cc}$ for the 25 mm filter and $0.000305 \text{ mm}^2/\text{cc}$ for the 37 mm filter. As a reasonable approximation, all results in S/mm^2 can be converted to a volumetric S/cc basis by multiplying by $3 \times 10^{-4} [S/\text{cc}]/[S/\text{mm}^2]$, assuming 1200 L or 2800 L is drawn through an appropriate filter. Using this conversion factor, the AHERA standard of $70 S/\text{mm}^2$ is equivalent to $0.021 S/\text{cc}$.

However, the conversion to S/cc still does not put the data on an equal footing with PCM data expressed on a f/cc basis, because “fibers” are almost always different from “structures”. Structures are bundles of fibers and many more “structures” can be identified by TEM than “fibers” by PCM because TEM can identify much smaller structures. Therefore, a TEM result for a given air sample (expressed on a volume basis, S/cc) will generally be greater than a PCM result for that same air sample (expressed as f/cc). In general there is not a good correlation between PCM and TEM measurements, and the ratios of the fiber counts seen with these two methods will vary according to the types of asbestos involved and the nature of the exposure setting. As noted below in making some rough comparisons, ATSDR has assumed a ratio of 60 of volumetric TEM data to volumetric PCM data; that is, PCM data was multiplied by 60 to convert the data to TEM units by ATSDR (ATSDR, 1999). This was done for the purpose of comparing different data from around the country to make observations about background asbestos concentrations.

As much of the health effects data on asbestos are expressed in terms of PCM f/cc , a useful variant of the TEM technique is to include counts of structures that would be expected to be visible under PCM and meet PCM criteria for counting as fibers. Specifically, structures meeting a minimum diameter of $>0.3 \mu\text{m}$ with length $>5 \mu\text{m}$ are counted as PCM equivalent (“PCME”) fibers.

IV.e.2. Risk Assessment Benchmarks for Evaluation of Asbestos Air Data

The principal benchmark used in this assessment for evaluation of asbestos in air data from the WTC site is the Asbestos Hazard Emergency Response Action (AHERA) standard of $70 S/\text{mm}^2$. This standard is determined by TEM analysis. This standard is described in the Final Rule and Notice for Asbestos-Containing Materials in Schools (40 CFR Part 763, October 30, 1987; cited in this report as EPA, 1987), and that rule also provides details on the monitoring apparatus and the structure counting procedure. This counting procedure includes discussions on the amount of filter area to examine with different volumes of air, and also the requirement to count fibers with an aspect ratio of $\geq 5:1$ (aspect ratio = length to width ratio) and a length $\geq 0.5 \mu\text{m}$. Briefly, this count of $70 S/\text{mm}^2$ is specific to a minimum volume of air requirement (1200 L if the filter size is 25 mm, and 2800 L if the filter size is 37 mm), and with this volume, a reading

of 70 S/mm² was evaluated as being statistically distinguishable from the count that would come from a blank filter. That background count is about one-fourth the standard, or 17.5 S/mm². The AHERA rule specifies that children would be allowed back into a school that has been undergoing asbestos abatement (removal and/or encapsulation) if the TEM readings were consistently below the count of 70 S/mm². This would be evidence that the concentration was similar to background readings. Alternately, an abatement area could be deemed suitable for occupation if samples taken within the area were statistically similar to samples simultaneously taken outdoors in an asbestos-free environment. Details of these procedures are provided in the Final Rule, as cited above.

It should be noted that this standard is not health based, but rather technology based. It is also noted that the technology has improved since 1987, such that current filters often have much less than 17.5 S/mm², sometimes close to 0 S/mm². Therefore, 70 S/mm² would be much higher than blanks and represents more than just a statistical elevation above background. above. Finally it is noted that while the AHERA standard was originally intended as an indoor 'clearance' standard, it is being used to evaluate outdoor exposures in this assessment.

The current OSHA PEL is also used in this assessment to evaluate air concentrations of asbestos measured using the PCM method. The PEL for occupational exposures to asbestos is 0.1 f/cc by PCM averaged over an 8-hour day (OSHA, 1994). This standard is relevant for the comparison of exposure of rescue and other workers at the WTC site with current workplace standards. EPA also collected air samples analyzed for fibers by the PCM method that may be used for this purpose.

IV.e.3. Background Air Concentrations for Asbestos

ATSDR's Toxicological Profile for Asbestos (ATSDR, 1999) provides a summary of background asbestos levels. Because the health effects data regarding inhalation exposure to asbestos are usually expressed in terms of PCM f/cc, ATSDR chose to convert ambient air data reported in units of ng/m³ or TEM f/cc to units of PCM f/cc. ATSDR's summary included these crude assumptions: 1 PCM f/cc is equal to 60 TEM f/cc and also approximately equivalent to a mass concentration of 30,000 ng/m³. (Note, however, that there was not sufficient analysis available for this current report to suggest applying these factors to asbestos measurement data for the WTC data.)

On this basis, the following summaries are excerpted from the profile (specific references supplied in ATSDR, 1999). It should be noted that these "background" data are derived from settings where no identified asbestos materials are present, as well as other settings, such as buildings containing asbestos materials where there may have been some local releases.

- Data from several studies indicate that in urban areas, most ambient air concentrations range from 3×10^{-6} to 3×10^{-4} PCM f/cc, but they may range up to 3×10^{-3} PCM f/cc as a result of local sources. In another investigation, the median concentration in U.S. cities has been estimated to be 7×10^{-5} PCM f/cc.
- A recent analysis of monitoring data for asbestos in ambient air worldwide estimated rural and urban levels at about 1×10^{-5} TEM f/cc (2×10^{-7} PCM f/cc) and 1×10^{-4} TEM f/cc (2×10^{-6}

PCM f/cc), respectively.

- In a review of indoor air monitoring data from a variety of locations, arithmetic mean concentrations ranged from 3×10^{-5} to 7×10^{-3} PCM f/cc. Levels of asbestos in 94 public buildings that contained asbestos ranged from ND to 0.2 TEM f/cc (ND to 3×10^{-3} PCM f/cc), with an arithmetic mean concentration of 0.006 TEM f/cc (10^{-4} PCM f/cc). Analysis of data based on air samples from 198 buildings with asbestos-containing materials (ACM) indicated mean asbestos levels ranging from 4×10^{-5} to 2.43×10^{-3} TEM f/cc (7×10^{-7} to 4×10^{-5} PCM f/cc).
- Asbestos concentrations in 41 schools that contained asbestos ranged from ND to 0.1 TEM f/cc (ND to 2×10^{-3} PCM f/cc), with an arithmetic mean of 0.03 TEM f/cc (5×10^{-4} PCM f/cc). Another study reported average concentrations of airborne asbestos fibers $> 5 \mu\text{m}$ in length of 8×10^{-5} TEM f/cc and 2.2×10^{-4} TEM f/cc in 43 non-school buildings and 73 school buildings, respectively (the 60:1 conversion factors would not apply to these data, since the TEM readings were already on fibers $> 5 \mu\text{m}$ in width, so they are likely to be more directly comparable to PCM results). In another study in 71 U.S. schools, the mean, the 95 percentile, and the maximum asbestos levels were 1.7×10^{-4} , 1.4×10^{-3} , and 2.3×10^{-3} PCM f/cc, respectively.
- A study of 49 buildings in the United States reported mean asbestos fiber levels of 9.9×10^{-4} PCM f/cc in buildings with no ACM, 5.9×10^{-4} PCM f/cc in buildings with ACM in good condition, and 7.3×10^{-4} PCM f/cc in buildings with damaged ACM.

In general, concentrations of asbestos in both indoor and outdoor settings and in both rural and urban settings appears to be less than, and by some studies, sometimes substantially less than, 3×10^{-3} f/cc on a PCM volumetric basis.

IV.e.4. Asbestos Air Monitoring Data at the WTC

Three sources of information were used to evaluate the asbestos air monitoring. One was the EPA WTC database itself. Downloads of this database occurred in May of 2002, and the data evaluated are current as of about mid-April, 2002. The WTC database includes measurements by several federal, state, and local agencies. The second source of data was the Trends Report dated May 16, 2002 (EPA, 2002a). There have been three Trend Reports. Generally, these reports obtain all their data from the EPA WTC database and provide summaries and interpretative analyses. The third source of data is a study commissioned by a "Ground Zero Elected Officials Task Force" to principally sample two apartments on September 18, 2001 (Chatfield and Kominsky, 2001). While the focus of that study was on the indoor environment (it is reviewed in more detail in Section V), two outdoor air samples were also taken.

The May Trends Report contains a summary of the PCM and TEM data collected between September 12, 2001 (the date of the first reported sample), and April 13, 2002, in the lower Manhattan area in the vicinity of Ground Zero. Additional TEM data from the Staten Island Landfill were obtained directly from the Region 2 WTC database. Figures 23 and 24 show the locations of fixed monitors at the Lower Manhattan and Staten Island Landfill areas,

respectively. A few more stations were set up in public schools (Manhattan - PS143, Queens - PS199, Brooklyn - PS274, Bronx - PS154, and Staten Island - PS44), and New Jersey (4 locations). There were over 600 samples taken in these public schools. Nearly all the samples were non-detected, with only one high reading of 93.3 S/mm² in PS199 in Queens on October 12, 2001, and 2 other low readings (< 20 S/mm²). Over 100 samples were reported for the New Jersey locations through early December of 2001. Nearly all readings were non-detect with a few low measurements (< 20 S/mm²).

On the basis of these findings, there does not appear to be any significant concern for a health impact at these Brooklyn, upper Manhattan, and New Jersey locations, and they are not discussed further.

The latest trends report (EPA, 2002a) summarizes the results of 8,870 samples taken from lower Manhattan and measured for TEM (locations in Figure 23). Samples taken during the time period between September 14 (the date of the first asbestos sample taken) and September 30 showed generally the highest concentrations. Table 6 lists the TEM measurements above 70 S/mm², and, as shown, more readings above this level were found for September (11 readings) than for any other month: October (2), November (1), December (1), January (1), February (2), March (3), and April (1).

The Trends Report also presents a directional analysis that supports this general observation in most cases. For this directional analysis, a subset of the sites was selected to represent the north, south, east, west, northeast, northwest, southeast, and southwest quadrants. Two-week maximums were then identified for each of these sites. These two maximums were plotted on 3-d graphs for N-S, E-W, NE-SW, and NW-SE. These graphs are duplicated in Figures 25 - 28 for TEM. The N-S graph in Figure 25, for example, shows that most of the samplers had their highest 2-week maximums during the first 2-week period identified, September 16 to September 30. Some samplers did have additional elevations later in time, such as Albany and Greenwich, which had a high reading of 204 S/mm² in December (from Figure 25 and also listed in Table 6).

The same general observation that early readings were the highest is seen in the NE-SW graph of Figure 28. These two graphs also do not exhibit a predominant wind direction: high measurements of the same magnitude were found on both the N and S sides (Figure 25) and the NE and SW sides (Figure 28). Figures 26 and 27, on W-E and NW-SE, do not show the same predominant elevations in September; concentrations are mostly level throughout time, with occasional elevated readings, such as a reading of 213 S/mm³ in February, 2002, at Church & Dey (from Figure 26 and Table 6). It does appear that during 2002, most readings were at what appears to be background for the area, at nondetect or reported at less than 20 S/mm².

A number of samples at the Staten Islands Landfill recorded higher levels than the AHERA standard. This presumably results from WTC debris being unloaded at this location, which causes the asbestos structures to become airborne. The WTC database listed 5207 measurements in numerous Staten Islands Landfill sites (see Figure 24), and 50 samples were identified as above 70 S/mm². These are listed in Table 6. Unlike the air monitors near Ground Zero, which showed the most elevations in September, the most elevations in the landfills occurred fairly

uniformly in October and November. This was perhaps a time period of most rigorous unloading. Of the 50 readings above 70 S/mm², 36 occurred during these two months. The highest level of 275.56 S/mm² was observed on November 5, 2001.

The Trends Report (EPA, 2002a) summarizes the results of 12,674 ambient samples measured for PCM, of which 8870 were also measured for TEM. Figures 29 - 32 show the directional analysis results for PCM. It is noted that all samples were less than 0.1 f/cc. The trend observed above for TEM, that most high readings were found in the early readings in September, holds as well for PCM. The range of these higher measurements is about 0.04 to 0.08 f/cc, and only 7 measurements in this range are seen in Figures 29 - 32. As noted above, PCM analyses identify the presence of fibers, including fibers of materials other than asbestos. By December, the maximum 2-week readings were all at apparent background for the area. With a few isolated exceptions, levels ranging from non detect to about 0.003 f/cc had been observed from lower Manhattan sampling sites since February, 2002 (EPA, 2002a). This background is consistent with the background measurements in other locations summarized above.

Chatfield and Kominsky (2001) describe the sampling of two apartments one week after September 11, on September 18. In addition to sampling of indoor air and dust (described in Section V), and some outdoor dust, two samples of outdoor air were also sampled. One was at a residential dwelling characterized as “high”, so named due to the expectation that higher concentrations would be measured within it. It was in an apartment building located on 250 South End Avenue, close to and southwest of Ground Zero. Apartment 10D, on the east side of this building and which had sustained window damage, was selected for sampling. Heavy dust deposits were in the apartment. One air sample at this site was taken by positioning the sampler outside a sliding window. The concentration of chrysotile asbestos ($S > 0.5 \mu\text{m}$) was 548 S/mm², which is the highest outdoor air measurement found. However, it is likely this high reading was influenced by the air quality on the inside of the apartment, which showed exceedingly high asbestos concentrations ($>10,000 \text{ S/mm}^3$; see the discussion on this study in Section VI. Data on Occupational and Indoor Exposures), and was likely not representative of outdoor concentrations. The “low” location was located four blocks north on 45 Warren Street. The apartment building did not appear to sustain any external damage. Apartments on the second and fifth floors were sampled. A sample taken on the roof above the fifth floor apartment showed a reading of 6.5 S/mm² chrysotile asbestos.

IV.e.5. Human Health Evaluation of Asbestos Air Measurements

Only 22 of 8870 TEM measurements in lower Manhattan from EPA’s WTC data base exceeded the AHERA standard of 70 S/mm², and one additional sample (of two taken) from an independent study exceeded the standard. The 12 exceedences, which occurred in September, were all at sites bordering Ground Zero: 250 South End Ave., Barclay & West Broadway, Albany & Greenwich, Liberty & South End, Vesey & West, and Albany & West. These sites were still in the restricted zone during September. The same general trend can be seen with the PCM data. Measurements near to or greater than 0.04 f/cc occurred mostly in September (6 of 7 samples during September, with 1 high sample during the first two weeks in October) and at sites bordering Ground Zero: Broadway & Liberty, Rector & South End, Albany & West, West Broadway & Barclay, Wall & Broadway, Albany & Greenwich, and Liberty & South End. It is

reasonable to conclude that general population exposures to ambient levels of asbestos were minimal and potential short- and long-term health impacts were minimal during the early weeks when a small percent of elevated measurements of asbestos were reported.

The potential for exposure appeared to be somewhat greater at the Staten Island Landfill. A total of 50 samples exceeded the AHERA standard of 70 S/mm², with most exceedences occurring during October and November. The average of the 36 exceedences during October and November was 92.6 S/mm². Exceedences in February through April of 2002 were likely due to the continued unloading of WTC debris. Assuming the crude TEM to PCM conversion factor of 1/60 used by ATSDR and the TEM surface area to volume conversion factor of 3×10^{-4} [f/cc]/[S/mm²], then this converts to a PCM-equivalent concentration of 0.0005 f/cc. This is significantly lower than the OSHA PEL of 0.1 f/cc. It is reasonable to conclude that the exposure of workers to asbestos at the Staten Island Landfill was minimal and potential short- and long-term health impacts were minimal during the unloading of debris at the site.

Table 6. Locations and concentrations of asbestos exceeding the AHERA level of 70 S/mm².

Location	Date	Concentration	Date	Conc
I. Landfill Locations				
Location 01 Landfill	Apr 27	125.98		
Location 02 Landfill	Mar 23	170.6		
Location 05 Landfill	Mar 14	78.74		
Location 08 Landfill	Nov 5	112		
Location 09-A Landfill	Oct. 8	71.11	Nov 5	71.11
	Oct 25	128	Nov 11	120.0
	Oct 25	128		
Location 09-B Landfill	Oct 18	96.24	Nov 5	71.11
Location 09-C Landfill	Oct 17	97.78		
Location 10-A Landfill	Nov 7	80		
Location 11 Landfill	Oct 18	96.24	Feb 6	110.24
	Nov 6	80	Feb 7	170.6
	Nov 7	88.89	Feb 15	78.74
	Nov 17	80	Feb 16	78.74
Location 12a Landfill	Oct 8	88	Nov 12	80
	Oct 16	124.44	Nov 13	72
	Oct 18	96.24	Nov 20	71.11
	Oct 20	80	Jan 11	166.23
	Oct 31	115.56	Mar 21	157.48
	Nov 5	275.56	Mar 23	125.98
	Nov 11	195.56	Apr 20	104.99
Location 12b Landfill	Oct 25	115.56	Nov 5	106.67
Location 13 Landfill	Oct 15	80	Nov 13	97.78
	Oct 25	88.89		
Location 14 Landfill	Oct 17	80	Dec 11	104.99
	Oct 26	88.89	Dec 11	104.99
	Nov 7	80	Apr 3	91.86
Location 15 Landfill (mess tent)	Nov 6	90	Nov 18	97.78
Location 16 Landfill (supply tent)	Oct 20	72	Nov 9	80
II. Lower Manhattan Locations				
250 South End Avenue ^a	Sep 18	548		
Location A - Barclay St & West Broadway	Sep 15	128	Sep 15	160
Location B - Church St & Dey St	Feb 11	213.33		
Location C - Liberty St & Trinity St	Feb 5	88		
Location D - Albany St & Greenwich St	Sep 27	97.78	Dec 27	204.44
	Sep 30	88.88		
Location E - Liberty St & South End Ave	Sep 16	90	Sep 30	80
Location F - Vesey St & West St	Sep 27	71.11		

Table 6 Locations and concentrations of asbestos exceeding the AHERA level of 70 S/mm² (cont'd).

Description	Date	Conc	Date	Conc
Location K - Albany & West St	Sep 22	80	Sep 27	177.78
	Sep 23	88.89	Sep 30	71.11
Location L - North Side of Stuyvesant High	Nov 28	124.44		
Location V - Pier 6 Bus Sign	Jan 14	72		
Wash Tent - West St. Between Murray & Vesey	Mar 9	144	Mar 30	96
	Mar 29	96	Apr 2	80
Site 2 - Chambers Street	Oct 9	104.99		
Public School 199 in Queens	Oct 12	93.33		

^a This sample was reported on in Chatfield and Kominsky (2001); all other data was from the EPA WTC data base. See text for more detail.

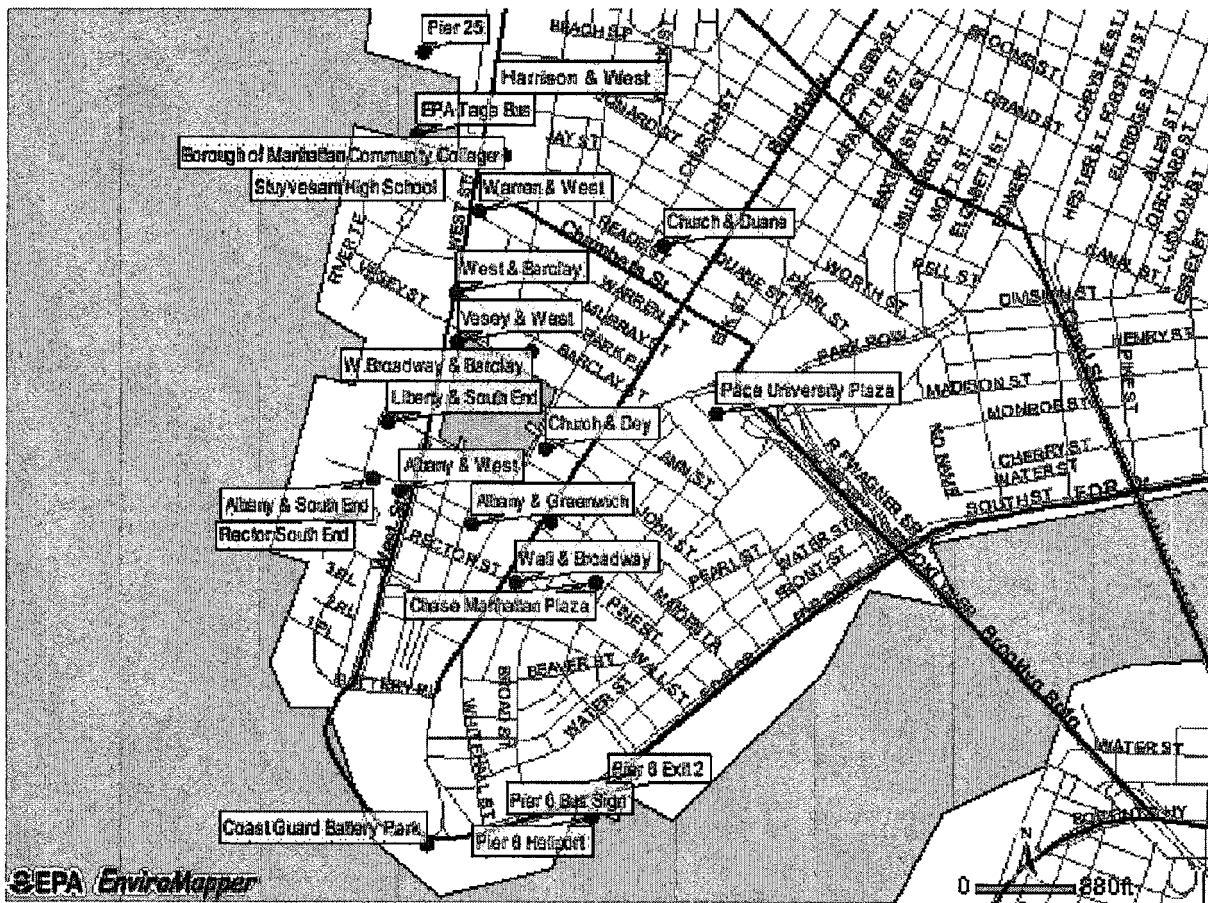


Figure 23. Location of asbestos monitoring stations in Lower Manhattan.

NYC-WTC_000149041

**Asbestos TEM - Weekly Maximums
North - South Axis**

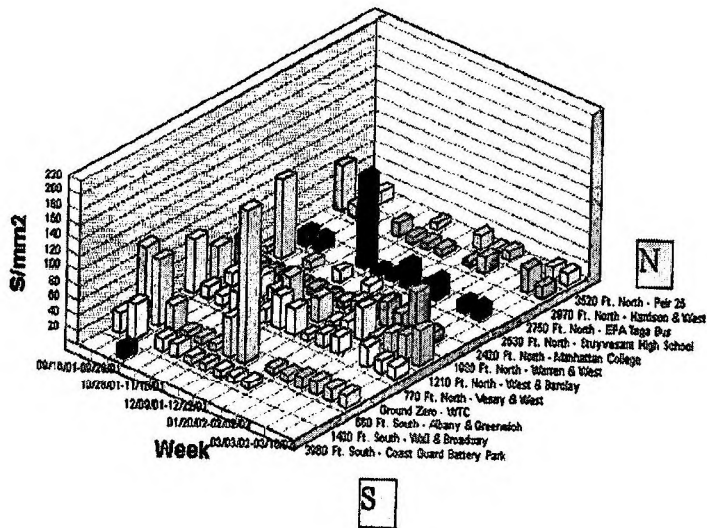


Figure 25. North-South directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

**Asbestos TEM - Weekly Maximums
East-West Axis**

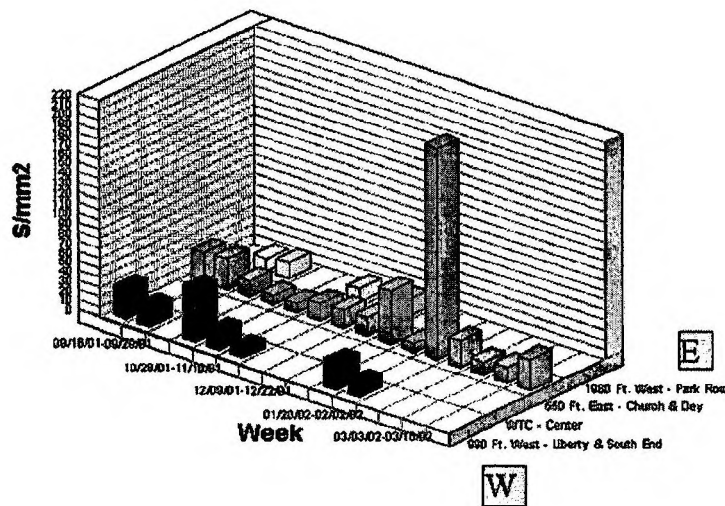


Figure 26. East-West directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a)

**Asbestos TEM - Weekly Maximums
Northwest - Southeast Axis**

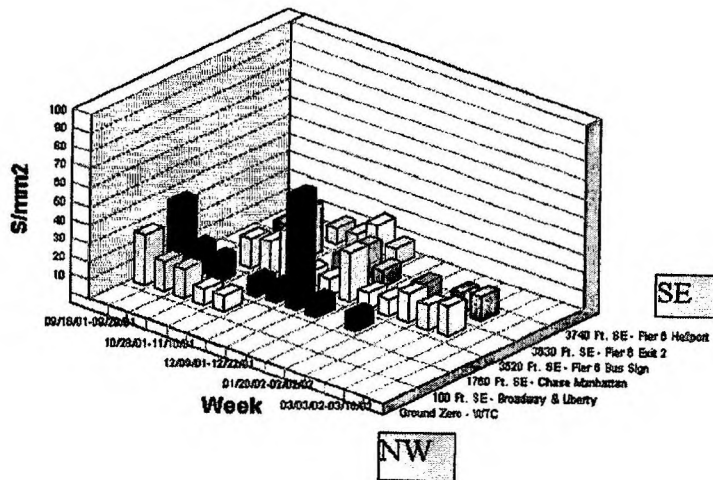


Figure 27. Northwest-Southeast directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

**Asbestos TEM - Weekly Maximums
Northeast-Southwest Axis**

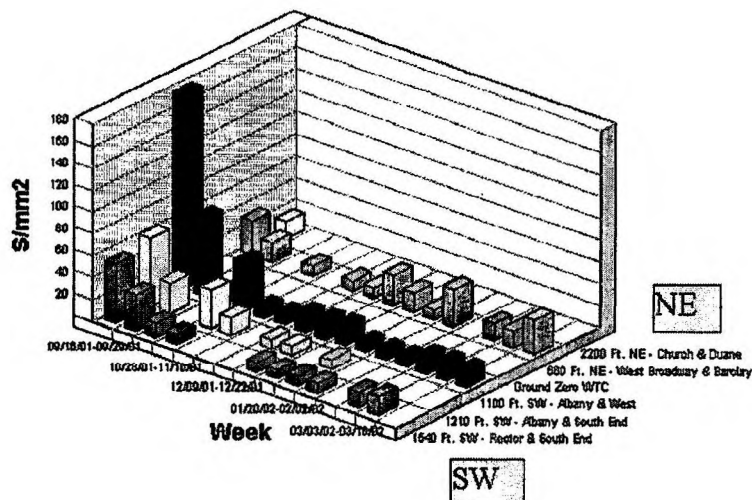


Figure 28. Northeast-Southwest directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

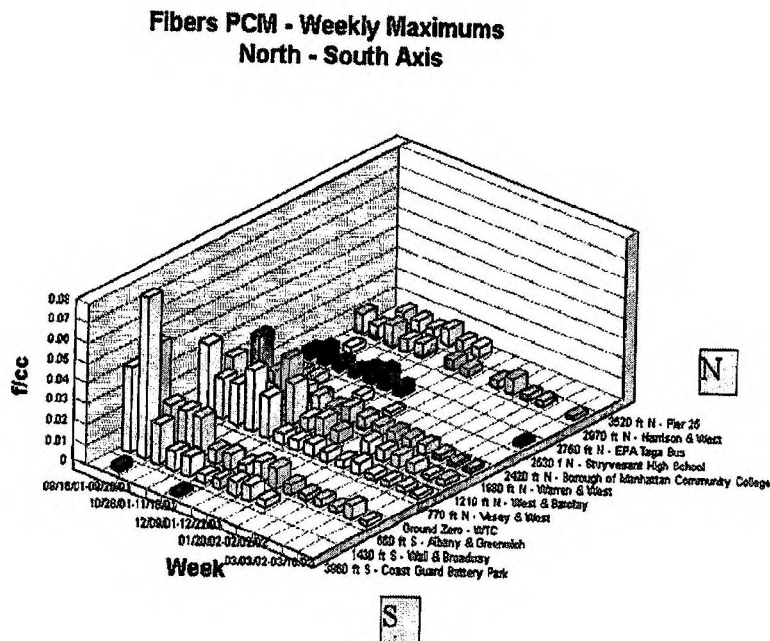


Figure 29. North-South directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

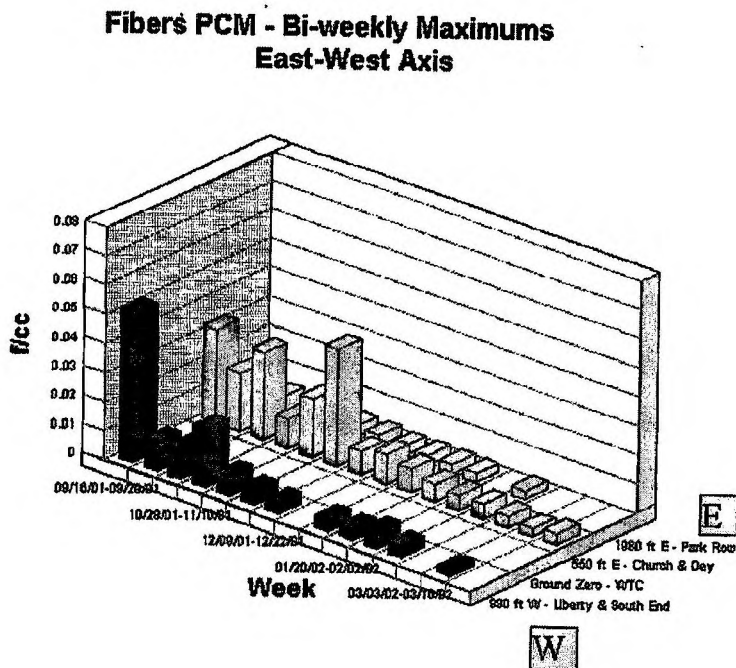


Figure 30. East-West directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a)

**Fibers PCM - Weekly Maximums
Northwest - Southeast Axis**

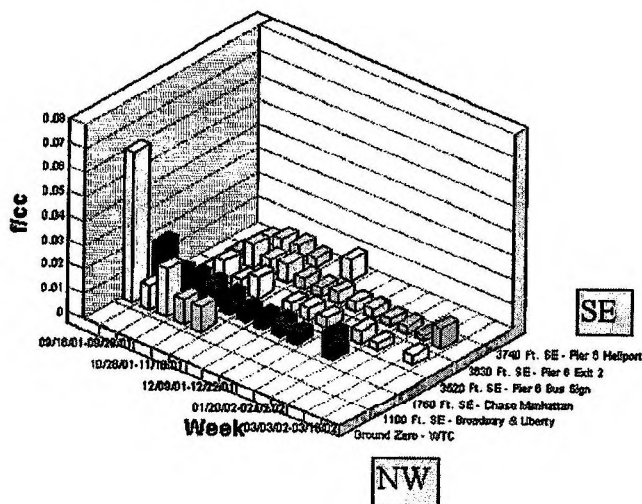


Figure 31. Northwest-Southeast directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

**Fibers PCM - Weekly Maximums
Northeast - Southwest Axis**

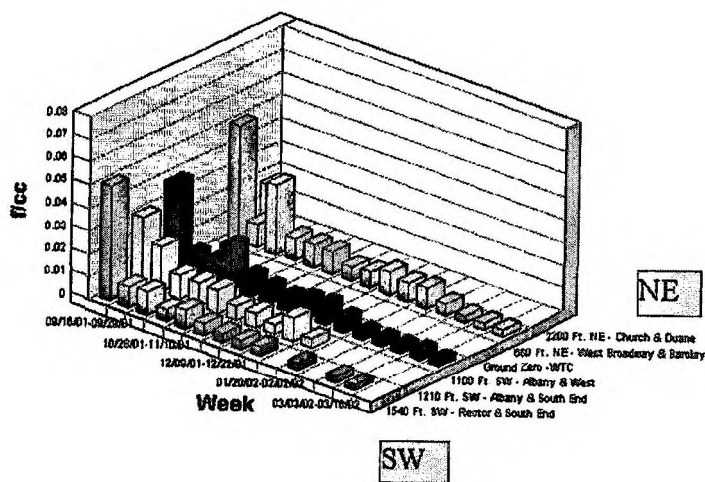


Figure 32. Northeast-Southwest directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

IV.f. Volatile Organic Compounds (VOCs)

VOCs are carbon compounds that exist exclusively in the gaseous phase in the ambient environment and include benzene, toluene, chloromethane, ethylbenzene, acetone, and styrene. VOCs have been associated with a variety of health effects, including immunologic, hematotoxic and neurologic effects; chromosomal damage; and cancer. VOCs are produced as a result of combustion of products containing carbon, such as plastics, wood, paper, carpeting, gasoline, and jet fuel. Thus, they would have been produced as a result of the WTC disaster.

Analysis of all VOCs released at the World Trade Center would require evaluating hundreds of different compounds. The list was narrowed down by emergency response personnel, representatives from EPA headquarters, EPA Region 2, NYSDEC, and NYSDOH. In this assessment, data for 14 VOCs were examined and screened, including acetone, benzene, 1,3 butadiene, chloromethane, 1,4 dioxane, ethanol, ethylbenzene, freon-22, methyl styrene, propylene, styrene, tetrahydrofuran, toluene, and xylenes. These VOCs were chosen on the basis of frequency of detection, concentration, toxicity, and carcinogenicity. Two of these 14 VOCs, freon-22 and methyl styrene, were not systematically measured outside of Ground Zero. Freon-22 was measured only at the WTC Chiller Plant and methyl styrene was only measured at Ground Zero. For this reason, freon-22 and methyl styrene are not evaluated in this report. Additionally, because no screening standards are available for propylene, a health assessment cannot be conducted for this chemical. Therefore, a total of eleven VOCs were evaluated for this report.

To evaluate the VOC exposures, a variety of screening benchmarks were used, including OSHA PELs and STELS, ATSDR acute and intermediate inhalation MRLs, and EPA Superfund Technical Support Center (STSC) provisional subchronic RfCs. If none of these benchmarks were available, the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs) for an 8-hour exposure and the NIOSH RELs were used. The goal of this assessment was to evaluate effects due to short-term exposure, thus screening tools that are based upon lifetime exposures to a chemical (i.e. EPA RfC values or ATSDR chronic MRLs) were not used.

IV.f.1. Evaluation of VOCs at Ground Zero

The majority of the EPA data collected for VOCs were within Ground Zero (North Tower Center, South Tower Center, and Austin Tobin Plaza) and nearby locations. For example, for acetone, benzene, ethylbenzene, and 1,3 butadiene, more than 500 samples were taken in the restricted zone near and within Ground Zero, where authorized personnel were directed to wear respirators. The majority of the measurements taken were grab samples that were usually collected within a 4-minute period. Samples at North Tower Center and South Tower Center were taken in potential hot spot areas such as plumes, areas of fire and combustion, and steam releases. The efforts at these two locations were subjective and were intended to capture potential worst case emissions. Samples at Austin Tobin Plaza were taken at a breathing zone height, but were still grab samples and were not purposively where workers were currently working, but where they might work or where there was visible smoke to contend with. Thus, collected samples at the three sites cannot be considered representative of the general air quality to which workers were exposed. Rather, the principal purpose of the EPA sampling at Ground

Zero was to provide results within four hours to alert the Fire Department of New York (FDNY) and the contractors/union health/safety officers working at Ground Zero about conditions that posed immediate health concern to the workers. This sampling was specifically requested by the FDNY and was conducted on a daily basis until the removal activities were completed at Ground Zero (end of May 2002).

As would be expected given this intention, some of the results (including data for benzene, 1,3-butadiene, and styrene) did show exceedences of OSHA limits. A few samples clearly demonstrated exceptionally high measurements of VOCs produced as a result of the disaster. Some samples were collected on top of, and at times, inside the actual debris pile, clearly demonstrating subjective sample design. Because the VOC sampling at the site is not believed to be representative of actual exposures to personnel, it would not be appropriate, or valid, to use these VOC sampling data to analyze worker exposures.

OSHA collected approximately 700 samples for organic compounds, as described in Chapter VI. Most of these samples were taken using personal air monitors, and the samples were taken over longer periods (one to eight hours) and are representative of a worker's breathing zone exposure. As described in Chapter VI, the OSHA data did not show routine exceedences of screening values (the OSHA standards). For that reason, WTC worker exposures will not be evaluated further. Instead, sampling data that were collected at sites surrounding the WTC site were used to evaluate potential health risks to persons who live or work in surrounding areas.

IV.f.2. Evaluation of VOCs at Sites Surrounding Ground Zero

The EPA WTC monitoring database and data from EPA-ORD monitors were used to evaluate VOC exposures to persons who may live and work in areas surrounding the WTC site. Most of the data in the WTC monitoring database are from EPA sampling, but data from other groups (e.g., NYSDEC, OENHP) are also included. Data from the WTC monitoring database covered approximately 27 locations and the EPA-ORD monitors covered 5 locations.

A map of the VOC sampling locations outside of Ground Zero is shown in Figure 33. Table 7 lists the site name, street address, approximate dates of sampling and the approximate number of samples that were taken. Because the number and dates of sampling varied depending on the VOC, the sample dates and number of samples taken shown in the table are for benzene monitoring only. These numbers are, in general, representative of the dates and frequency of sampling of the other VOCs.

Exceedences of benchmark standards were seen for 6 of the 11 VOC compounds evaluated in this assessment, including acetone, benzene, 1,3-butadiene, chloromethane, ethylbenzene, and toluene. 1,4 Dioxane, ethanol, styrene, tetrahydrofuran, and xylenes showed no exceedences of screening benchmarks at any of the sampling sites and are therefore not considered to be contaminants of concern at sites surrounding Ground Zero. Table 8 lists the sites that showed exceedences of screening benchmarks, the dates of exceedence, and the location of the site in relation to the restricted zone. Tables 9-14 show the exceedences of these 6 VOC compounds.

For 5 of these 6 VOC compounds, data were also available for 24-hour samples taken by

EPA; 24-hour samples were not available for chloromethane. These 24-hour sample results are also provided in Tables 9, 10, 11, 13, and 14 to compare with the exceedences (Table 12 is chloromethane).

Chemical-by-chemical summaries of the 6 VOCs that exceeded benchmarks are presented below.

Acetone: Most people are exposed to acetone through consumer product use, including nail polish remover, particle board, and paint removers. Acetone exposure may also occur as a by-product of exposure to isopropyl alcohol. The typical level of acetone in the air in cities in the United States is about 0.007 ppm (ATSDR, 1994). Acute exposure to acetone at levels above 100 ppm may cause irritation to the nose, throat, lungs, and eyes (ATSDR, 1994).

A total of 264 acetone monitoring results were evaluated at sites surrounding Ground Zero. A summary of the screening benchmarks and exceedences is shown in Table 9. All three exceedences were seen at Greenwich and Liberty. Two of the exceedences were on September 28, where grab samples found concentrations of 29 ppm and 22 ppm. On October 1, a concentration of 20 ppm was detected. The other 12 samples taken at Greenwich and Liberty did not show concentrations above 13 ppm, including the samples taken on September 26 and September 27, as well as October 2, October 3, and February 23.

The monitoring data indicate that the acetone level was found to be elevated above a typical background (0.007 ppm as noted above) in grab samples for 4 days only on the end of September and beginning of October just off the southern portion of Ground Zero. Because this would correspond to an acute exposure, the ATSDR acute MRL screening value of 26 ppm (ATSDR, 1994) is most appropriate. This value was only slightly exceeded in one sample on 1 day (29 ppm). The Greenwich and Liberty sampler location was in the restricted zone at the time of the exceedence and it was unlikely that residents would be present in that area.

Twenty-four hour samples were taken on September 27 and for three days in December. As seen in Table 9, these locations bordered Ground Zero and the day-long concentration was nearly 4 orders of magnitude (10,000 times) lower than the grab samples, and well within the range of the cited typical background of 0.007 ppm. This clearly demonstrates that sampling within a "hot spot" can result in very high concentrations that are representative of only the few minutes during which the grab sample is taken.

Benzene: Benzene is widely used in the production of other chemicals. It is also found in crude oil, gasoline, and cigarette smoke. Excluding occupational exposures, the major sources of benzene exposure are tobacco smoke, automobile service stations, automobile exhaust, and industrial emissions. Urban air concentrations of benzene can vary widely, depending on mobile source pollution. One large urban study (ATSDR, 1997a) detected a median benzene level of 0.013 ppm. Background concentrations reported by EPA Region 2 for benzene are 0.00051 ppm and 0.00053 ppm (annual averages of Brooklyn and Staten Island locations, respectively, over the period 1994 - 98; EPA, 2002b).

Breathing levels of benzene above 100 ppm can cause drowsiness, dizziness, and

unconsciousness. Long-term continued exposures to benzene will depress and may cause damage to the blood-forming system, as seen by a decrease in red and white blood cells, lymphocytes, platelets, and other blood constituents. However, after exposure has ended, red blood cell levels may return to normal. Benzene can also harm the immune system and increase the chances of infection and cancer. Benzene is a known human carcinogen, causing leukemia. Most of what is known about the acute and chronic effects of benzene comes from animal and human studies where decreases in bone marrow function have been measured. The intermediate MRL, 0.004 ppm, was derived from a study in mice, where changes in locomotor activity were seen after exposure to 0.78 ppm benzene for 2 hours a day for 30 days. A 100-fold uncertainty factor was used to derive the intermediate MRL (ATSDR, 1997a).

A total of 332 benzene monitoring results were evaluated at sites surrounding Ground Zero. A summary of the screening benchmarks and exceedences is shown in Table 10. According to sampling notes for other VOCs with the same sample number, all these samples were taken at ground level, and the October 1 measurement was taken at ground level in a plume. Nine measurements taken at Greenwich and Liberty between September 16 and September 26, and on October 2 and October 3, were at levels below 0.02 ppm. From these data, it appears that exceedences did not last more than 5 days. Furthermore, these exceedences all occurred within the restricted zone. At Liberty and Trinity, the exceedence detected was 11 ppm on September 26. According to sampling notes for other VOCs with the same sample number, this measurement was taken at ground level in a plume. Measurements taken on September 16, September 22, September 23, and on October 26 did not show exceedences.

From these data, it appears that the exceedence did not last longer than 32 days and throughout this period was in a restricted zone. Because previous sampling did not show exceedences, it is likely that the measurement was taken deliberately in a plume to represent a worst-case transient exposure.

For the 262 samples recorded in the WTC EPA database for sites surrounding Ground Zero, the minimal detection limit was 0.02 ppm, as compared to the ATSDR intermediate MRL of 0.004 ppm (ATSDR, 1997a). This means that the analytical method could not detect benzene contamination levels below 0.02 ppm. Therefore, one cannot evaluate whether or not the ATSDR intermediate MRL was exceeded because the detection level for the samples was higher than the intermediate MRL. Five samples exceeded the ATSDR acute MRL of 0.05 ppm (ATSDR, 1997a), and these are shown in Table 10. Four samples had values above 0.02 ppm but below the 0.05 ppm level. The rest of the samples in the EPA WTC database were reported to be 0.02 ppm. These values are not reported above. Sample results reported to be below 0.05 ppm (257 sampling results) were not screened against the intermediate MRL.

Results from the 70 EPA-ORD samples show that the OSHA PEL and STEL values for benzene were never exceeded. Since these EPA-ORD samples did have a lower detection than the samples reported on in the WTC EPA database, they could be compared against the ATSDR intermediate MRL of 0.004 ppm. It was found that the ATSDR acute and intermediate MRL values were exceeded a total of 17 times at 4 different sampling sites. At West Broadway and Park Place, 10 samples were taken between November 9, 2001, and January 3, 2002. They showed benzene levels to be below the intermediate MRL. Additionally, exceedences at West

Broadway and Park Place were not found on October 15, October 16, October 17, and October 22 (the highest value was only 0.002 ppm). The range of sampling results (0.002 to 0.026 ppm) shows the temporal variability in the benzene levels. At 290 Broadway from November 26 through January 3, 10 samples were taken. They showed benzene levels to be below the intermediate MRL. At Broadway and Liberty, the sample taken on October 17, the only sample taken after the exceedence on October 8, was below the intermediate MRL at 0.0009 ppm. At Albany and West, 16 samples were taken between October 22 and January 3. They showed the benzene levels to be below the intermediate MRL.

Monitoring results from the EPA-ORD sampling suggest that benzene never exceeded screening benchmarks for more than 45 days. At all the sites there was great temporal variability in the samples. The largest gap between an exceedence of the intermediate MRL and a value below the MRL was only 11 days at West Broadway and Park Place. For 290 Broadway, Broadway and Liberty, and Albany and West, the first samples below screening benchmarks were taken November 26, September 22, and September 23, respectively. If one assumes that exposures began September 11, the worst case exceedences of the intermediate MRL could not have lasted longer than 45, 8, and 18 days, respectively, at 290 Broadway, Broadway and Liberty, and Albany and West. Thus the worst-case, longest possible exposure, would have been 45 days at 290 Broadway.

On the basis of data summarized above, it is concluded that the exceedences measured at Liberty and Trinity, Greenwich and Liberty, W. Broadway and Park Place, Broadway and Liberty and Albany and West were not a public health risk to residents. These sites were in the restricted zone or on the border of the zone, and it is unlikely that residential exposures occurred for extended periods. The samples taken were grab samples (taken within a 4 minute period) and are not representative of the average exposures. Samples taken at Liberty and Trinity and Greenwich and Liberty were purposefully taken at ground level and/or in a plume and are not representative of average breathing zone exposures. The temporal variability of the other samples, as shown by exceedences intermixed between days that were below screening benchmarks, also leads to the conclusion that exceedences of the Intermediate MRL were not sustained for extended periods of time. For exposures lasting less than 14 days, the acute MRL is a more appropriate health screening benchmark, and this value was not exceeded at these sites.

At 290 Broadway, elevated measurements were found as late as October 11 (see Table 10) and exposures could have occurred, as the measurement was taken in a non-restricted zone. In a worst-case scenario, the longest possible exposure would have been 45 days at this site. At this site, measurements were taken on a 16th floor balcony. Whether or not the VOC samples collected at this site are representative of the breathing zone exposures will depend on the meteorology and air mixing at the site. The highest benzene value measured at this site was 0.007 ppm and the intermediate MRL is 0.004 ppm (ATSDR, 1997a). Adverse effects would not be expected at 0.004 ppm, and it is unlikely that adverse effects would occur at 0.007 ppm.

The 24-hour samples are mostly lower than all the grab sample exceedences; only the finding of 0.005 ppm at Church and Dey on September 28 approached the grab samples listed as exceeding the ATSDR Intermediate MRL of 0.004 ppm. This Church and Dey location is on the edge of Ground Zero which was also restricted on September 28. In the northwest direction on

September 28, at the Vesey and West and the EPA Taga Bus locations showed a very low 24-hour concentration, 0.00081 at Vesey and West and ND at EPA Taga Bus. This demonstrates that concentrations were likely higher in the general direction of plume movement, which on September 28 was likely in the east direction.

Of all the VOC data, the benzene data does suggest that sustained concentrations above the typical New York City background could have occurred for about a month after September 11 outside of Ground Zero. As noted above, the background concentration reported by EPA Region 2 for benzene is about 0.0005 ppm. Four of the nine 24-hour measurements taken on September 28 exceeded this background. The grab sample exceedences were substantially higher than this background, and there were several exceedences above the ATSDR Intermediate MRL of 0.004 ppm. Whether or not specific health effects occurred due to exposure to benzene is unknown, but given that the exceedences and elevations above typical background were near Ground Zero and mostly within restricted zones, the data suggests that exposures to the general population were of minimal concern.

1,3-Butadiene: 1,3-Butadiene is found in automobile exhaust, wood smoke, and cigarette smoke, and in the breakdown of other materials. 1,3-Butadiene is almost always found at low levels in urban air samples, but it breaks down very quickly. In sunny weather, the half-life of 1,3-butadiene is only 2 hours. The median concentration of 1,3-butadiene in urban air has been estimated at approximately 0.0003 ppm (ATSDR, 1993).

A total of 304 1,3-butadiene monitoring results were evaluated at sites surrounding Ground Zero. A summary of the available screening benchmarks and exceedences is shown in Table 11. One exceedence of the 1.0 ppm PEL was detected in a ground-level plume on October 1, at 1.5 ppm. On days preceding and following October 1 the levels of 1,3-butadiene measured at this location were below the screening benchmark; in fact, 10 of the 12 samples taken at this location were at 0.002 ppm.

All 24-hour samples were non-detects for 1,3-butadiene.

Chloromethane: Chloromethane is always present in the air at very low levels. Most of the naturally occurring chloromethane comes from chemical reactions that occur in the oceans or that occur when materials such as grass, wood, charcoal, and coal are burned. Reported urban levels of chloromethane have been between 0.00066 and 0.00096 ppm (ATSDR, 1998). The background concentration reported by EPA Region 2 for chloromethane is approximately 0.00029 ppm (the annual average for a Staten Island location for the period 1995-1999; EPA, 2002b).

High-level exposures - above 100 ppm - to chloromethane can cause nervous system damage and adversely affect the liver, kidney, and heart. Lower-level exposures - above 50 ppm - have been shown to cause delayed growth, liver changes, and neurological effects in animals. Data do not exist to determine health effects that would be seen with short-term, very low-level exposures. The EPA-STSC provisional subchronic RfC, the screening benchmark with the lowest acceptable exposure limit, is based on a 2-year animal study that showed neurological effects and liver and kidney damage at 1000 ppm but not at 225 ppm (EPA, 1998).

A total of 257 chloromethane monitoring results were evaluated at sites surrounding Ground Zero. A summary of the available screening benchmarks and exceedences is shown in Table 12. As with all VOCs, all grab sample exceedences were taken in a restricted zone, and in the time frame from late September to early October. It is noted that all of the samples that showed exceedences at Greenwich and Liberty were taken at ground level and that the October 1 measurement was taken at ground level in a plume. Measurements taken at this site September 24 - 27 and on October 2 and 3 were all at levels below 0.02 ppm. From these data, it appears that exceedences did not last more than 4 days. At Liberty and Trinity, the exceedence detected was 0.82 ppm on September 26, and it was noted that this measurement was taken at ground level in a plume. Measurements taken on September 22, September 23, and on October 26 did not show exceedences.

Ethylbenzene: Ethylbenzene occurs naturally in petroleum and coal tar and can be released into the air from burning oil, gas, and coal. The median level of ethylbenzene in city and suburban air is about 0.00062 ppm (ATSDR, 1999).

Breathing high levels of ethylbenzene (above 100 ppm) can cause dizziness, tightness in the chest, and eye and throat irritation. Short-term exposure of laboratory animals to high concentrations of ethylbenzene in air may cause liver and kidney damage, nervous system changes, and blood changes. No data exist to evaluate the short-term effects of low levels of ethylbenzene exposure in humans or animals. The EPA-STSC provisional subchronic RfC for ethylbenzene is adopted directly from the EPA RfC for a lifetime exposure. The EPA-STSC confidence level in this derivation is low. The EPA RfC is based on developmental effects seen in female rats that were exposed to ethylbenzene throughout their pregnancy. Adverse effects in this study were not seen at levels below 100 ppm (EPA, 1999a).

A total of 338 ethylbenzene monitoring results were evaluated at sites surrounding Ground Zero. A summary of the available screening benchmarks and exceedences is shown in Table 13. As with the other VOCs, exceedences occurred in the restricted zone in the latter part of September and early October. A value of 0.4 ppm ethylbenzene was detected at Liberty and Trinity and it was noted that this sample was taken in a ground-level plume. Three samples obtained between September 16 and September 23 and a breathing zone sample taken on October 26 were all below 0.05 ppm.

The 24-hour ethylbenzene samples that were detected were three orders of magnitude (1000 times) lower than these grab sample exceedences; 10 out of 13 samples were non-detected. This demonstrates again the difference between grab samples taken within a plume and the 24-hour average concentration of the VOC.

Toluene: Toluene occurs naturally in crude oil and is added to gasoline. Toluene is also used in making paints, paint thinners, fingernail polish, lacquers, and adhesives. It can also be detected in cigarette smoke. Urban concentrations of toluene have been estimated to be around 0.003 ppm (ATSDR, 2000b). The background concentration reported by EPA Region 2 for toluene is approximately 0.002 ppm (annual average for a Brooklyn location over the period 1994 - 1998).

High-level exposures to toluene (above 100 ppm) may affect the nervous system and kidneys. Headaches, confusion, and sleepiness are also seen after high-level exposures. A short-term exposure study (4 days) in human subjects showed eye and nose irritation and neurological effects at 100 ppm; these effects were not seen at 40 ppm. The EPA-STSC provisional subchronic RfC for toluene is adopted directly from the EPA RfC for a lifetime exposure. The EPA-STSC confidence in this value is medium. This value is derived from a study where workers were exposed to 88 ppm of toluene for 6 years. In this study, adverse neurological effects were seen in the workers (EPA, 1999b).

A total of 335 toluene monitoring results were evaluated at sites surrounding Ground Zero. A summary of the available screening benchmarks and exceedences is shown in Table 14. The available benchmarks that were exceeded were the ATSDR acute MRL of 1 ppm and the EPA-STSC provisional subchronic RfC of 0.25 ppm. At Greenwich and Liberty, measurements taken on September 28 were collected in ground-level grab samples; on October 1, the sample was taken in a ground-level plume. Measurements taken at this site on September 16 through September 27 and on Oct 2 and 3 were all at levels below 0.02 ppm. From these data, it appears that exceedences did not last more than 4 days. At Liberty and Trinity, the September 26 sample was collected in a ground level plume. Four samples obtained between September 16 and September 23 and a breathing zone sample taken October 26 were all below 0.05 ppm.

The 24-hour toluene samples were three orders of magnitude (1000 times) lower than these grab sample exceedences. Similar to ethylbenzene, 1,3-butadiene, and acetone, this difference in toluene concentrations between the grab sample exceedences and the 24-hour samples demonstrates the difference between grab samples taken within a plume and the long-term average concentration of the VOC.

Findings from VOC monitoring: As discussed above, most of the samples that showed exceedences were short duration grab samples that were taken in a plume or at ground level, not in the breathing zone. In fact, the exceedences for benzene, ethylbenzene, chloromethane and toluene measured at Liberty and Trinity on September 26, all came from the same collected grab sample which was taken in a plume. Similarly, all the exceedences measured at Greenwich and Liberty for acetone, 1,3-butadiene, benzene, chloromethane and toluene, came from the same 3 collected grab samples. Because these are 4-minute grab samples, it is not known how long the plume lasted - from minutes to hours to days. These exceedences all occurred in late September and early October; available grab sample data before and after these exceedences are all lower in value. In addition to being grab samples, all exceedences occurred within restricted zones or just on the border of the restricted zones. Finally, all 24-hour samples of four of the VOCs - ethylbenzene, 1,3-butadiene, acetone, and toluene - were lower than the grab samples, by about a factor of 1000. On the basis of the available monitoring data, it is concluded that the exceedences of the screening benchmarks in the restricted zone did not represent a public health risk to persons living or working at sites surrounding Ground Zero for at least these four VOCs.

The data for benzene was not as definitive. When compared with the other VOCs, the 24-hour benzene samples were measured at levels that were closer in magnitude to the grab sample exceedences, within a factor of 10. This would suggest that the grab sample concentrations were

closer to sustained concentrations rather than short-term plume concentrations only. Also, these 24-hour concentrations were near the ATSDR Intermediate MRL of 0.004 ppm and higher than the historical average for New York City of about 0.0005 ppm. The data suggests that the exposures to benzene at levels that approach the MRL were no longer than 45 days. Whether or not specific health effects occurred due to exposure to benzene is unknown, but given that the exceedences and elevations above typical background were near Ground Zero and mostly within restricted zones, the data suggests that exposures to the general population were of minimal concern.



FACT SHEET: RELEASE OF REPORTS RELATED TO THE WORLD TRADE CENTER DISASTER

Exposure and Human Health Evaluation of Airborne Pollution from the
World Trade Center Disaster (DRAFT)

Toxicological Effects of Fine Particulate Matter Derived from the
Destruction of the World Trade Center (FINAL)

ACTIONS:

The U.S. Environmental Protection Agency (EPA) has completed a draft report entitled, "Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster," and is soliciting comments from the public on this draft report during a 60-day comment period. In addition, the EPA has completed a final report, entitled "Toxicological Effects of Fine Particulate Matter Derived from the Destruction of the World Trade Center." Both of these documents are available to the public. A *Federal Register* notice, published December 27, 2002, announced: 1) the public availability of the external review draft and the start of the comment period; and 2) the availability of the final rodent respiratory studies report.

SUMMARY:

EPA has released for public review and comment a draft evaluation of exposure and human health impacts of the September 11, 2001, terrorist attack on the World Trade Center (WTC). The draft evaluation concludes that, with the exception of those exposed immediately following the collapse and perhaps during the next few days, people in the surrounding community are not likely to suffer from serious long or short term health effects. The draft report evaluates the measured outdoor levels of various air pollutants to which the public had been potentially exposed as a result of the collapse of the WTC. These data were evaluated in terms of available health benchmark concentrations and typical background concentrations for New York City or other urban areas. The draft report focuses on: particulate matter (PM), metals (lead, chromium and nickel compounds), polychlorinated biphenyls, dioxin-like compounds, asbestos, and volatile organic compounds.

The draft report includes a discussion of rodent respiratory toxicology studies, conducted by EPA scientists, that found that high doses of WTC dust could cause mild respiratory effects in mice. These studies exposed mice to fallen dust samples collected at or near the WTC site on September 12 and 13, 2001. The report, "Toxicological Effects of Fine Particulate Matter Derived from the Destruction of the World Trade Center," summarizes these studies. The purpose of these studies was to evaluate the toxicity of fine PM on the respiratory tract of mice and to compare well-studied PM reference samples, ranging from essentially inert to quite toxic, to those collected at the WTC site. These studies found that fine particles were mostly dominated by calcium containing compounds derived from WTC building materials and that a high dose of WTC fine PM could cause mild lung inflammation and air flow obstruction in mice. These findings suggest that a similarly high dose in people could cause short-term respiratory effects such as inflammation and cough. These studies have been accepted for publication in the peer-reviewed journal, *Environmental Health Perspectives*, and are expected to be published shortly.

It is important to note that the draft human health evaluation is undergoing public review, as well as external independent expert scientific peer review. These review processes are usual steps that the Agency takes to

insure full and open participation by interested parties. These steps also help the Agency to identify areas where a draft document could be improved to strengthen both clarity and completeness of the draft. Comments from the public and from the expert peer review will be used to improve the draft report before it is finalized.

BACKGROUND:

EPA responded immediately to the September 11, 2001, terrorist attack on the WTC. The Agency, along with many other federal, state and local agencies, was called upon to bring its technical and scientific expertise to the national emergency. EPA, other federal agencies, and New York City and New York State public health and environmental authorities initiated numerous air monitoring activities to better understand the ongoing impact of emissions from that disaster. Many EPA offices and programs quickly became involved with these activities, providing scientific, engineering, public health, and management expertise to help cope with the after effects of the collapse of the WTC. EPA Region 2, which includes the New York City metropolitan area in New York and New Jersey, is the Agency's lead office on these activities, including the important and complicated task of community outreach and communication. As part of these activities, Region 2 requested that ORD conduct a human health evaluation of exposure to air pollutants resulting from the WTC disaster.

DOCUMENT AVAILABILITY:

Draft Human Health Evaluation: The primary method for document availability will be via the Internet at <http://www.epa.gov/ncea/wtc.htm>. A limited number of CDs and paper copies are available from: Technical Information Staff (8623D), U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, 1200 Pennsylvania Avenue, NW, Washington, DC 20460; Telephone: 202-564-3261; Facsimile: 202-565-0050; Email: nceadc.comment@epa.gov.

Final Rodent Respiratory Toxicology Report: The primary method for document availability will be via the Internet at <http://www.epa.gov/nheerl>. A limited number of paper copies will be available from EPA's National Service Center for Environmental Publications (NSCEP) in the near future. To obtain copies, please contact NSCEP by telephone (1-800-490-9198 or 513-489-8190), by facsimile (513-489-8695), or by mail (P.O. Box 42419, Cincinnati, OH 45242-0419). Please provide your name and mailing address and the title and EPA number of the document requested (*Toxicological Effects of Fine Particulate Matter Derived from the Destruction of the World Trade Center*. EPA/600/R-02/028, June 2002).

CONTACTS:

Draft Human Health Evaluation: Linda Tuxen, NCEA Communications, National Center for Environmental Assessment (8601D) Office of Research and Development, U.S. Environmental Protection Agency, Washington, DC; Tel: 202-564-3332; Email: tuxen.linda@epa.gov

Final Rodent Respiratory Toxicology Report: Debbie Janes, Public Affairs, Office of Research and Development (B34303), U.S. Environmental Protection Agency, Research Triangle Park, NC; Tel: 919-541-4577; Email: janes.deborah@epa.gov



U.S. Environmental Protection Agency

EPA Response to September 11

[Recent Additions](#) | [Contact Us](#) |
Search: [EPA Home](#) > Response to 9-11

NEWS of INTEREST [En Español]:

New! Access Your Personal Testing Data On-line
Exposure & Human Health Evaluation of Airborne Pollution from the WTC Disaster
Toxicological Effects of Fine Particulate Matter Derived from the Destruction of the
World Trade Center
New Scheduling and Cancellation Policies Announced
Airborne Asbestos Testing Data Now Available
Residential Cleaning and Testing is Underway
Consumer Warning: Beware Phony Contractors
Work Begins on Confirmation Cleaning Study

ACCESS YOUR PERSONAL TESTING DATA ON-LINE!

Residents who have had their homes tested can now access their testing data on-line. You will need your confirmation number and PIN number to access the data. If you have forgotten your confirmation number, you can call the Cleanup Hotline at **1-877-796-5471**. You will need to give the operator your PIN number (usually the last four digits of your social security number).

SCHEDULING PROBLEMS AND CANCELLATIONS LEAD TO NEW POLICIES FOR LOWER MANHATTAN DUST CLEANUP

EPA has established firm new policies to deal with ongoing problems in scheduling the cleaning and/or testing of residences in lower Manhattan.

People who cancel appointments with less than 24 hours notice or people who do not provide access at the appointed time will be removed from the assistance program. These cancellations without notice waste resources; the crews are not available to assist people who are waiting to be scheduled and contractors are paid a penalty when scheduled crews cannot work. If you have an appointment and need to change it, you must give your monitoring contractor at least 24 hours notice – they are available seven days a week.

Scheduling contractors have also had difficulty reaching people who have requested assistance and, in some instances, scheduling an appointment once the resident has been reached. The new policies spell out the limits to which schedulers will go before the requestor is dropped from the assistance program.

Emergency situations will be handled on a case-by-case basis. EPA is committed to helping those who truly want and need assistance. We are implementing the policy to help support those residents with the most efficient and effective use of program funds and staff as possible.

Policy . . .

RESIDENTIAL CLEANING AND TESTING IS UNDERWAY

Lower Manhattan Dust Cleanup Program Registration Period Ended

Registration for Indoor Air Dust Cleanup Services in Lower Manhattan ended on Saturday, December 28. To update or revise an earlier request, please contact the Dust Cleanup Services Hotline.

Dust Cleanup Toll-Free Hotline
1-877-796-5471
 (TTY for the deaf and hard of hearing: 1-800-396-1018)

WTC Email Notification Service

Sign up to receive email about scheduling updates, news releases, and more

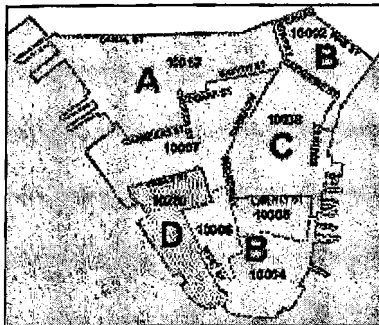
Phone Numbers

EPA Dust Cleanup Toll-Free Hotline
 1-877-796-5471
 (TTY: 1-800-396-1018)
FEMA Assistance Toll-free Hotline
 1-800-462-9029
 (TTY: 1-800-462-7585)
NYS Individual and Family Grant Program
 1-866-346-0348
NYCDEP Help Line
 718-DEP-HELP (337-4357)
NYSERDA ("Keep Cool" AC Help)
 1-877-NYSMART (1-877-697-6278).
Mental Health Association of New York City
 1-800-Lifenet (1-800-543-3638)

EPA Employees: Personally Involved



It is sometimes easy to forget that EPA Region 2 employees, working virtually in the shadow of the World Trade Center, were as personally affected by the collapse of the towers



(September 16, 2002) The work of cleaning and testing residences in lower Manhattan is underway. Eight contracts have been awarded for the downtown World Trade Center residential dust cleanup program. Contractors will have identification and will be able to give the resident the confirmation number that was provided during sign up. Two contractors, one project monitoring contractor and one cleanup contractor, will work in each of four quadrants of the eligible cleanup area – south of Canal, Pike and Allen Streets, river to river.

News Release . . .

as anyone in lower Manhattan.
Story . . .

Contractor Information

Asbestos removal contractors seeking information about the project should write to:

NYCDEP
Asbestos
Department
59-17 Junction Blvd.
Flushing, NY 11373

Please Note: Test results will be mailed in four to six weeks. Please be patient and refrain from calling.

See Related Fact Sheets:

Cleaning & Testing
Preparing for Cleaning & Testing
Carpets, Upholstered Furniture and Other Fabrics
Common Area and HVAC Fact Sheet
Modified-Aggressive and Aggressive Sampling
Testing Criteria and Limits

[EPA Home](#) | [Privacy and Security Notice](#) | [Contact Us](#)

This page was generated on Monday, March 10, 2003

View the graphical version of this page at: <http://www.epa.gov/wtc/>



U.S. Environmental Protection Agency

Federal Register Environmental Documents

[Recent Additions](#) | [Contact Us](#) | [Print Version](#) Search:

[EPA Home](#) > [Federal Register](#) > [FR Years](#) > [FR Months](#) > [FR Days](#) > [FR Daily](#) > Draft Exposure and Human Health Evaluation of Airborne Pollution From the World Trade Center Disaster and Final Toxicological Effects of Fine Particle Matter Derived From the Destruction of the World Trade Center

Draft Exposure and Human Health Evaluation of Airborne Pollution From the World Trade Center Disaster and Final Toxicological Effects of Fine Particle Matter Derived From the Destruction of the World Trade Center

[FR Home](#)

[About the Site](#)

[FR Listserv](#)

[FR Search](#)

[Contact Us](#)

[Selected Electronic Dockets](#)

[Regulatory Agenda](#)

[Executive Orders](#)

[Current Laws and Regulations](#)

[Federal Register: December 27, 2002 (Volume 67, Number 249)]

[Notices]

[Page 79089-79090]

From the Federal Register Online via GPO Access [wais.access.gpo.gov]

[DOCID:fr27de02-90]

ENVIRONMENTAL PROTECTION AGENCY
[FRL-7429-8]

Draft Exposure and Human Health Evaluation of Airborne Pollution From the World Trade Center Disaster and Final Toxicological Effects of Fine Particle Matter Derived From the Destruction of the World Trade Center

AGENCY: Environmental Protection Agency.

ACTION: Notice of availability of a draft document for public review and comment, and notice of availability of a final document.

SUMMARY: The U.S. Environmental Protection Agency's (EPA) Office of Research and Development (ORD) is announcing a 60-day public comment period for the external review draft (ERD) entitled, "Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster (EPA/600/P-02/002A, October 2002)." This draft document was prepared by ORD's National Center for Environmental Assessment (NCEA). ORD also is announcing the public availability of a final report entitled, "Toxicological Effects of Fine Particle Matter Derived from the Destruction of the World Trade Center (EPA/600/R-02/028, June 2002)," hereafter, rodent respiratory toxicological report. This final report was prepared by ORD's National Health and Environmental Effects Research Laboratory (NHEERL)..

DATES: The 60-day public comment period on the ERD begins December 27, 2002, and ends February 25, 2003. Technical comments should be in writing and must be postmarked by February 25, 2003. The final rodent respiratory toxicological report is available today.

ADDRESSES: The primary distribution method for the ERD will be via ORD's web site at <http://www.epa.gov/ncea/wtc.htm>. This draft report, in PDF format, can be viewed and downloaded from the Internet for review and comment. In addition, a limited number of CD-ROM and paper copies of the ERD are available by contacting the Technical Information

Staff, NCEA-W (8623D), U.S. Environmental Protection Agency, Washington, DC 20460; telephone: 202-564-3261; facsimile: 202-565-0050; email: nceadc.comment@epa.gov. Please provide your name, mailing address, and the title and EPA number of the requested publication.

The rodent respiratory toxicological report is also available via ORD's web site at <http://www.epa.gov/nheerl>. A limited number of paper copies are available from EPA's National Service Center for Environmental Publications (NSCEP). To obtain copies, please contact NSCEP by telephone: 1-800-490-9198 or 513-489-8190; facsimile: 513-489-8695, by mail: P.O. Box 42419, Cincinnati, OH 45242-0419. Please provide your name and mailing address and the title and EPA number of the document requested.

COMMENT SUBMISSION: Comments on the ERD may be mailed to the Technical Information Staff, NCEA-W (8623D), U.S. Environmental Protection Agency, Washington, DC 20460; telephone: 202-564-3261; facsimile: 202-565-0050. Comments should be in writing. Please submit one unbound original with pages numbered consecutively, and three copies of the comments. For attachments, provide an index, number pages consecutively with the comments, and submit an unbound original and three copies. Electronic comments may be emailed to: nceadc.comment@epa.gov.

Please note that all technical comments received in response to this notice will be placed in a public record. For that reason, commentors should not submit personal information (such as medical data or home address), Confidential Business Information, or information protected by copyright. Due to limited resources, acknowledgments will not be sent.

FOR FURTHER INFORMATION CONTACT: For information on the public comment period, contact the Technical Information Staff of the National Center for Environmental Assessment-Washington by telephone: 202-564-3261; facsimile: 202-565-0050; email: nceadc.comment@epa.gov.

SUPPLEMENTARY INFORMATION: Immediately following the September 11, 2001, terrorist attack on New York City's World Trade Center, many federal agencies, including the EPA, were called upon to focus their technical and scientific expertise on the national emergency issues. EPA, other federal agencies, New York City, and New York State public health and environmental authorities focused on numerous air monitoring activities to better understand the ongoing human health impact of the disaster. Many EPA offices and programs quickly became involved with these activities, providing scientific, engineering, public health, and management expertise to help cope with the aftereffects of the collapse of the World Trade Center.

As part of these activities, a human health evaluation of exposure to air

[[Page 79090]]

pollutants resulting from the World Trade Center disaster was initiated. The primary purpose and scope of this draft report were to evaluate the environmental levels of various air pollutants to which the public could potentially be exposed as a result of the collapse of the towers. The draft report evaluates the measured outdoor levels of various air pollutants to which the public potentially had been exposed. These data were evaluated in terms of available health benchmark concentrations and typical background concentrations for New York City or other urban areas. The draft evaluation concludes that, with the exception of those exposed immediately following the collapse and perhaps during the next few days, people in the surrounding community are not likely to suffer from serious long- or short-term health effects.

While the primary focus of EPA's draft evaluation is on outdoor levels of various air pollutants to which the public could potentially

be exposed as a result of the collapse of the towers, some information on indoor and occupational exposures is summarized in EPA's draft report. The incursion of dust and other contaminants into residences and buildings is being addressed via a number of other studies initiated in conjunction with the plans by EPA and its federal, state, and city partners to clean up residences impacted by the collapse of the World Trade Center.

The draft report also includes a discussion of rodent respiratory toxicology studies, conducted by EPA scientists, that exposed mice to fallen dust samples collected at or near Ground Zero on September 12 and 13, 2001. The purpose of these studies was to evaluate the toxicity of fine particulate matter dust on the respiratory tract of mice and to compare well-studied particulate matter reference samples, ranging from essentially inert to quite toxic, to those collected at the World Trade Center site. These studies found that fine particles were dominated by calcium containing compounds derived from World Trade Center building materials, and that a high exposure to World Trade Center fine particulate matter could cause mild lung inflammation and airflow obstruction in mice. These findings suggest that a similarly high exposure in people could cause short-term respiratory effects such as inflammation and cough.

Further, it is important to note that while this ERD is undergoing public review and comment, a process of external independent expert scientific peer review also is underway. These review processes are the usual steps that EPA takes to ensure full and open participation by interested parties. These steps also help EPA identify areas where a draft document could be improved to strengthen both clarity and completeness of the draft. Comments from the public and from the expert peer reviewers will be used to improve the draft report before it is finalized.

Finally, EPA scientists, in collaboration with other federal and state environmental health professionals, as well as colleagues in academia and medical institutions, will continue to analyze available data on human exposures to environmental contaminants resulting from the World Trade Center disaster. This continuing work will help us to better understand the potential human health impacts.

Dated: December 20, 2002.

Paul Gilman,

Assistant Administrator, Office of Research and Development.

[FR Doc. 02-32600 Filed 12-26-02; 8:45 am]

BILLING CODE 6560-50-P

[EPA Home](#) | [Privacy and Security Notice](#) | [Contact Us](#)

Last updated on Friday, December 27th, 2002

URL: <http://www.epa.gov/EPA-TOX/2002/December/Day-27/t32600.htm>



U.S. Environmental Protection Agency

Federal Register Environmental Documents

[Recent Additions](#) | [Contact Us](#) | [Print Version](#) Search: [GO](#)

[EPA Home](#) > [Federal Register](#) > [FR Years](#) > [FR Months](#) > [FR Days](#) > [FR Daily](#) > Voluntary Data Call-In; World Trade Center Disaster Exposure and Human Health Information

Voluntary Data Call-In; World Trade Center Disaster Exposure and Human Health Information

[FR Home](#)

[About the Site](#)

[FR Listserv](#)

[FR Search](#)

[Contact Us](#)

[Selected Electronic Dockets](#)

[Regulatory Agenda](#)

[Executive Orders](#)

[Current Laws and Regulations](#)

[Federal Register: December 27, 2002 (Volume 67, Number 249)]

[Notices]

[Page 79079]

From the Federal Register Online via GPO Access [wais.access.gpo.gov]

[DOCID:fr27de02-82]

ENVIRONMENTAL PROTECTION AGENCY

[FRL-7429-9]

Voluntary Data Call-In; World Trade Center Disaster Exposure and Human Health Information

AGENCY: Environmental Protection Agency.

ACTION: Notice; request for voluntary submission of human health data, exposure data, or other similarly technical information or reports related to the after effects of the collapse of the World Trade Center.

SUMMARY: The U.S. Environmental Protection Agency's (EPA) Office of Research and Development (ORD) is requesting the voluntary submission of data and other information or reports on human health effects or exposures that may have been generated by academia, hospitals, public or private institutions, businesses and corporations, or any other public or private sector entity, following the collapse of the World Trade Center on September 11, 2001.

Please note that this request for information is a voluntary data call-in. No one is obligated by this notice to submit information and there are no penalties for not submitting information. Submitted data and information will be analyzed by the EPA to help the Agency better assess the potential human health impacts of exposure to the environmental contaminants in both the outdoor and indoor air and in the settled dust in businesses and residences.

DATES: Please submit this information by March 1, 2003. While EPA is interested in receiving all technical information and data that may have been developed, the information received by March 1, 2003 will be the most useful to the Agency as it revises its draft report.

ADDRESSES: Submissions of reports, data, or other technical information should be mailed to the Technical Information Staff (8623D), U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, 1200 Pennsylvania Avenue NW, Washington, DC 20460; Telephone: 202-564-3261; Facsimile: 202-565-0050. If an overnight delivery service is used, information submissions should be delivered to: Technical Information Staff, National Center for Environmental Assessment, Suite 500, 808 17th Street NW,

Washington, DC 20006. Electronic submissions may be e-mailed to:
nceadc.comment@epa.gov.

Please note that all submissions received in response to this notice will be considered public information. For that reason, please do not submit any uncoded personal information (such as medical data), Confidential Business Information, or information protected by copyright. Due to limited resources, acknowledgments will not be sent.

FOR FURTHER INFORMATION CONTACT: For general information on this notice contact: Joanna Foellmer, National Center for Environmental Assessment, Telephone: 202-564-3208; E-mail: foellmer.joanna@epa.gov. For technical questions regarding this notice, contact: Matthew Lorber, National Center for Environmental Assessment, Telephone: 202-564-3243; E-mail: lorber.matthew@epa.gov.

SUPPLEMENTARY INFORMATION: In a companion Federal Register notice published today, EPA is announcing a 60-day public comment period on its external review draft titled, "Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster (EPA/600/P-02/002A, October 2002)." During the development of this draft report, EPA became aware that potentially significant data may have been developed by public and private sector institutions that could be pertinent to this report. These data, which to date have not been available to EPA, could help the Agency to better understand the potential human health impacts of exposures to environmental contaminants resulting from the World Trade Center disaster. Therefore, EPA encourages and appreciates the voluntary submission of these data, which will be considered in the development of the Agency's final report on potential human health impacts from exposure to the environmental contaminants resulting from the World Trade Center collapse.

Dated: December 20, 2002.
 Paul Gilman,
 Assistant Administrator, Office of Research and Development.
 [FR Doc. 02-32601 Filed 12-26-02; 8:45 am]
 BILLING CODE 6560-50-P

[EPA Home](#) | [Privacy and Security Notice](#) | [Contact Us](#)

Last updated on Friday, December 27th, 2002
 URL: <http://www.epa.gov/fedrgstr/EPA-AIR/2002/December/Day-27/a32601.htm>



U.S. Environmental Protection Agency

Federal Register Environmental Documents

[Recent Additions](#) | [Contact Us](#) | [Print Version](#) Search: [GO](#)

[EPA Home](#) > [Federal Register](#) > [FR Years](#) > [FR Months](#) > [FR Days](#) > [FR Daily](#) > Draft Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster

[FR Home](#)
[About the Site](#)
[FR Listserv](#)
[FR Search](#)
[Contact Us](#)

Draft Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster

[Federal Register: March 6, 2003 (Volume 68, Number 44)]

[Notices]

[Page 10723-10724]

From the Federal Register Online via GPO Access [[waiss.access.gpo.gov](http://www.access.gpo.gov)]

[DOCID:fr06mr03-59]

[Selected Electronic Dockets](#)
[Regulatory Agenda](#)
[Executive Orders](#)
[Current Laws and Regulations](#)

ENVIRONMENTAL PROTECTION AGENCY
[FRL-7459-3]

Draft Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster

AGENCY: Environmental Protection Agency.

ACTION: Notice of extension of public comment period.

SUMMARY: The U.S. Environmental Protection Agency's (EPA) Office of Research and Development (ORD) is announcing the extension of the public comment period for the external review draft (ERD) document, Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster (EPA/600/P-02/002A, October 2002). This draft document was prepared by ORD's National Center for Environmental Assessment (NCEA) within the Office of Research and Development.

DATES: In the December 27, 2002, Federal Register (67 FR 79089), EPA announced a limited comment period through February 25, 2003. The Agency is now extending the public comment period to April 7, 2003. Technical comments should be in writing and must be postmarked by April 7, 2003.

ADDRESSES: The primary distribution method for the ERD will be via ORD's Web site at <http://www.epa.gov/ncea/wtc.htm>. This draft report, in PDF format, can be viewed and downloaded from the Internet for review and comment. In addition, a limited number of CD-ROM and paper copies of the ERD are available by contacting the Technical Information Staff, NCEA-W (8623D), U.S. Environmental Protection Agency, Washington, DC 20460; telephone: (202) 564-3261; facsimile: (202) 565-0050; e-mail: nceadc.comment@epa.gov. Please provide your name and mailing address, and the title and EPA number of the requested publication.

Comment Submission: Comments on the ERD may be mailed to the Technical Information Staff, NCEA-W (8623D), U.S. Environmental Protection Agency, Washington, DC 20460; telephone: (202) 564-3261; facsimile: (202) 565-0050. Comments should be in writing. Please submit one unbound original with pages numbered consecutively, and three

copies of the comments. For attachments, provide an index, number pages consecutively with the comments, and submit an unbound original and three copies. Electronic comments may be e-mailed to: nceadc.comment@epa.gov.

Please note that all technical comments received in response to this notice will be placed in a public record. For that reason, commentors should not submit personal information (such as medical data or home address), Confidential Business Information, or information protected by copyright. Due to limited resources, acknowledgments will not be sent.

FOR FURTHER INFORMATION CONTACT: For information on the public comment period, contact the Technical Information Staff of the National Center for Environmental Assessment-Washington, telephone: (202) 564-3261; facsimile: (202) 565-0050; e-mail: nceadc.comment@epa.gov.

SUPPLEMENTARY INFORMATION: Immediately following the September 11, 2001, terrorist attack on New York City's World Trade Center, many federal agencies, including the EPA, were called upon to focus their technical and scientific expertise on the national emergency issues. EPA, other federal agencies, New York City, and New York State public health and environmental authorities focused on numerous air monitoring activities to better understand the ongoing human health impact of the disaster. Many EPA offices and programs quickly became involved with these activities, providing scientific, engineering, public health, and management expertise to help cope with the aftereffects of the collapse of the World Trade Center.

As part of these activities, a human health evaluation of exposure to air pollutants resulting from the World Trade Center disaster was initiated. The primary purpose and scope of this draft report were to evaluate the environmental levels of various air pollutants to which the public could potentially be exposed as a result of the collapse of the towers. The draft report evaluates the measured outdoor levels of various air pollutants to which the public potentially had been exposed. These data were evaluated in terms of available health benchmarks and typical background concentrations for New York City or other urban areas. The draft evaluation concludes that, with the exception of those exposed immediately following the collapse and perhaps during the next few days, people in the surrounding community are not likely to suffer from serious long-or short-term health effects.

While the primary focus of EPA's draft evaluation is on outdoor levels of various air pollutants to which the public could potentially be exposed as a result of the collapse of the towers, some information on indoor and occupational exposures is summarized. The incursion of dust and other contaminants into residences and buildings is being addressed via a number of other studies initiated in conjunction with the plans by EPA and its federal, state, and city partners to clean up residences impacted by the collapse of the World Trade Center.

The draft report also includes a discussion of rodent respiratory toxicology studies, conducted by EPA scientists, that exposed mice to fallen dust samples collected at or near Ground Zero on September 12 and 13, 2001. The purpose of these studies was to evaluate the toxicity of fine particulate matter dust on the respiratory tract of mice and to compare well-studied particulate matter reference samples, ranging from essentially inert to quite toxic, to those collected at the World Trade Center site. These studies found that fine particles were dominated by calcium containing compounds derived from World Trade Center building materials, and that a high exposure to World Trade Center fine particulate matter could cause mild lung inflammation and airflow obstruction in mice. These findings suggest that a similarly high exposure in people could cause short-term respiratory effects such as inflammation and cough.

Further, it is important to note that while this ERD is undergoing

public review and comment, a process of external independent expert scientific peer review also is underway. These review processes are the usual steps that EPA takes to ensure full and open participation by interested parties. These steps also help EPA identify areas where a draft document could be improved to strengthen both clarity and completeness of the draft. Comments from the public and from the expert peer reviewers will be used to improve the draft report before it is finalized.

Finally, EPA scientists, in collaboration with other Federal and State environmental health

[[Page 10724]]

professionals, as well as colleagues in academia and medical institutions, will continue to analyze available data on human exposures to environmental contaminants resulting from the World Trade Center disaster. This continuing work will help us to better understand the potential human health impacts.

Dated: February 27, 2003.

Art Payne,

Acting Director, National Center for Environmental Assessment.

[FR Doc. 03-5322 Filed 3-5-03; 8:45 am]

BILLING CODE 6560-50-P

[EPA Home](#) | [Privacy and Security Notice](#) | [Contact Us](#)

Last updated on Thursday, March 6th, 2003

URL: <http://www.epa.gov/fedrgstr/EPA-AIR/2003/March/Day-06/a5322.htm>



U.S. Environmental Protection Agency National Center for Environmental Assessment

[Recent Additions](#) | [Contact Us](#)

Search:

GO[EPA Home](#) > [Research & Development](#) > [NCEA Home](#) > [EIMS Record Results](#)

EXPOSURE AND HUMAN HEALTH EVALUATION OF AIRBORNE POLLUTION FROM THE WORLD TRADE CENTER DISASTER (EXTERNAL REVIEW DRAFT)

Following the collapse of the World Trade Center towers on September 11, 2001, New York State and Federal agencies initiated numerous air monitoring activities to better understand the ongoing impact of emissions from the disaster. This report focuses on these air measurement data, evaluating them in terms of what is typical for New York City or general urban background and interpreting it with regard to the potential for human health consequences. The report does not evaluate exposures possibly faced by rescue or clean-up workers and briefly discusses past and current indoor monitoring efforts.

The analysis in this report supports three general findings: 1) Persons exposed to the extremely high levels of ambient particulate matter and its components during the collapse of the World Trade Center towers and for several hours afterwards were likely to be at risk for immediate acute (and possibly chronic) respiratory and other types (e.g., cardiovascular) of symptoms. 2) The first measurements of some of the contaminants were on September 14, while other contaminants were not measured until September 23. Available data suggest that the concentrations within and near Ground Zero were likely to be highest in the few days following September 11. Because there are only limited data on these critical few days, exposures and potential health impacts cannot be evaluated with certainty for this time period. 3) Except for exposures on September 11 and possibly during the next few days, persons in the surrounding area were unlikely to suffer short-term or long-term adverse health effects caused by exposure to elevations in ambient air concentrations of the contaminants evaluated in this report. These elevated concentrations were measured mostly within and near Ground Zero, and they lasted for one to three months after September 11. The monitoring data indicate that air concentrations decreased to background levels that are characteristic of pre-September 11 levels in the New York City metropolitan area by around January or February of 2002.

NOTICE: The 60-day comment period, which ended on February 25, has been extended. See Federal Register notice link below on a 30-day extension to this comment period.

Citation:

2002. EXPOSURE AND HUMAN HEALTH EVALUATION OF AIRBORNE POLLUTION FROM THE WORLD TRADE CENTER DISASTER (EXTERNAL REVIEW DRAFT). . 01 Jan 2002..

Contact Information:

MATTHEW LORBER

by phone at: 202-564-3243

by fax at: 202 565-0078

or by email at: lorber.matthew@epa.gov

Downloads:

- ➔ [Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster \(4.8 MB\)](#)
- ➔ [Toxicological Effects of Fine Particulate Matter Derived from the Destruction of the World Trade Center](#)
- ➔ [Fact Sheet: Release of Reports Related to the World Trade Center Disaster](#)
- ➔ [EPA Response to September 11\(EPA's main web page on 9/11 activities\)](#)
- ➔ [Federal Register Notice - Availability of Draft Document and Request for Public Comment](#)
- ➔ [Federal Register Notice - Voluntary Data Call-In](#)
- ➔ [Federal Register Notice - Comment Period Extension](#)

[View Additional Details for this Record](#)

[ORD Home](#) | [Search EPA](#) | [Search NCEA](#) | [Search EIMS](#) | [Contacts](#) | [Help](#)

[EPA Home](#) | [Privacy and Security Notice](#) | [Contact Us](#)

Last Updated on Wednesday, November 27, 2002
URL: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm>

DRAFT
DO NOT CITE OR QUOTE

NCEA - W - 1395
EPA/600/P-2/002A
October 2002
External Review Draft
<http://www.epa.gov/ncea>

Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster

National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
October, 2002

DISCLAIMER

This document is a draft. It has not been formally released by the U.S. Environmental Protection Agency and should not at this stage be construed to represent Agency policy. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Table of Contents

	<u>Page</u>
Executive Summary	
I. Overall Purpose and Scope of Assessment	
II. Exposure Assessment and Risk Characterization Approach	
III. Monitoring Data	
IV. Evaluation	
IV.a. <i>Particulate Matter</i>	
IV.b. <i>Metals</i>	
IV.c. <i>Polychlorinated Biphenyls</i>	
IV.d. <i>Dioxins</i>	
IV.e. <i>Asbestos</i>	
IV.f. <i>Volatile Organic Compounds</i>	
V. Comment on the First Several Days After September 11	
VI. Data on Occupational and Indoor Exposures	
VI.a. <i>Occupational Exposures</i>	
VI.b. <i>Indoor Exposures</i>	
VII. Overall Comments and Future Studies	
VIII. References	
Appendix A. World Trade Center Health Effects Screening Criteria for Ambient Air Developed by EPA's Region 2	
Appendix B. Table of Monitoring Locations	

List of Tables

Table 1. Inhalation health risk screening benchmarks used in this assessment.

Table 2. Concentrations (maximum and Sept-Oct average) of PM_{2.5} and Component Elements Measured at the EPA/ORD World Trade Center Perimeter Sites and at Four Other Sites in the Northeast U.S. (PM_{2.5} concentrations in µg/m³; all other components in ng/m³).

Table 3. Summary of PCB monitoring data between September, 2001, and April, 2002.

Table 4. Measured dioxin TEQ air concentrations at the WTC Building 5 monitor, the Church & Dey monitor, and the Park Row monitor (all units = pg TEQ/m³; NR = not reported; all TEQ calculated at ND = ½ DL except values in parenthesis, which are calculated at ND = 0).

Table 5. Human exposure and health risk assessment assumptions and results for dioxin TEQs.

Table 6. Locations and concentrations of asbestos exceeding the AHERA level of 70 S/mm².

Table 7. VOC sampling locations outside of Ground Zero.

Table 8. Locations that showed exceedences of screening benchmarks for VOCs and restrictions to access.

Table 9. Acetone grab sample exceedences and 24-hour sample monitoring summary.

Table 10. Benzene grab sample exceedences and 24-hour sample monitoring summary.

Table 11. 1,3-Butadiene grab sample exceedences and 24-hour sample monitoring summary.

Table 12. Chloromethane grab sample exceedences monitoring summary

Table 13. Ethylbenzene grab sample exceedences and 24-hour sample monitoring summary.

Table 14. Toluene grab sample exceedences and 24-hour sample monitoring summary.

List of Figures

Figure 1. Figure showing the shrinkages of the restricted zones in the vicinity of Ground Zero over time. For example, the area below Canal Street was no longer prohibited after 9/14, and similarly, the shaded area beneath Chambers Street became available only after 9/27. [Figure extracted from map supplied by City of New York, Emergency Mapping Center].

Figure 2. Particulate matter monitoring sites, including ORD surface sites (A, C, K) on the WTC perimeter, the ORD site at 290 Broadway, and NYSDEC sites located elsewhere.

Figure 3. Spread of dense dust/smoke cloud over all of lower Manhattan and drifting to the E/SE immediately after the September 11, 2001, collapse of the World Trade Center buildings.

Figure 4. World Trade Center plume from intense fires ($>1000^{\circ}\text{F}$) during days following September 11, 2001, with high concentrations of both newly formed fine particles from combustion and reentrained coarse particles likely being transported upward by convection processes and being dispersed in the WTC plume over varying NY City areas, depending on prevailing wind directions and speeds.

Figure 5. ORD-modeled WTC plume dispersion on September 11, 2001, at 12 noon. The values indicated by red numerals are hourly $\text{PM}_{2.5}$ concentrations (in $\mu\text{g}/\text{m}^3$) measured at pre-existing NJ and NY State-operated PM monitoring stations in northern New Jersey and New York City. Red, orange, and yellow shading represent most likely areas of plume dispersion (red = estimated dilution to 100^{th} to 500^{th} and dark blue = dilution to $<\text{one millionth}$ of pollutant concentration at WTC source).

Figure 6. Satellite photograph of the WTC plume lofting from Ground Zero at 11:43 am EDT on September 12, 2001. Note the very concentrated vertical convection of dust/smoke particles upwards and the flow in a well-defined plume towards the S/SE. (Source: Mandatory Credit: "spaceimaging.com").

Figure 7. Increased hourly $\text{PM}_{2.5}$ concentrations measured on September 12 and/or 13 at PS 64, PS 199, Maspeth Library, and PS 274 to the E/SE of WTC, reflecting dispersal of newly formed fine particles from WTC fires and/or fine particles reentrained from the settled dust from initial collapse of the WTC buildings. ($\text{PM}_{2.5}$ data provided courtesy of NYSDEC).

Figure 8. ORD-modeled WTC plume dispersion for September 13, 2001 at 9:00 am. Note the increased hourly $\text{PM}_{2.5}$ concentrations depicted in red for PS 64 ($166 \mu\text{g}/\text{m}^3$) and PS 199 ($100 \mu\text{g}/\text{m}^3$) NYSDEC monitoring stations, consistent with the E/NE direction of the modeled plume dispersion and likely touchdown of the plume at those and intervening sites but not at sites further E, SE, or to the N.

Figure 9. ORD-modeled WTC plume dispersion on September 14, 2001 at 12 noon, indicative of plume flow mainly out over New York Harbor.

Figure 10. Satellite photograph of WTC plume lofting from Ground Zero at 11:54 am EDT on September 15, 2001, and dispersion to the S/SW out over the New York Harbor. (Source: Mandatory Credit: "spaceimaging.com").

Figure 11. *Panel A (top):* Daily PM_{2.5} concentrations monitored by EPA/ORD at sites A, C, and K around Ground Zero perimeter and at 290 Broadway 6 blocks northeast of Ground Zero. *Panel B (bottom):* PM_{2.5} concentrations observed at several extended monitoring network sites in lower Manhattan within 3 to 10 blocks of WTC Ground Zero.

Figure 12. ORD-modeled WTC plume dispersion on October 4, 2001 at 3:00–4:00 a.m. Note the general regional elevation of hourly PM_{2.5} levels (in µg/m³) indicated by red numerals for monitoring sites scattered across both northern New Jersey and NYC areas, even outside modeled areas of likely greatest plume intensity indicated by red shading.

Figure 13. Daily PM_{2.5} concentrations recorded at NYSDEC PS 64 monitoring site after September 11, 2001 (9/11/01 to 10/27/01) compared to historic record of 24-hr PM_{2.5} values at the same site during prior 2 years (2/23/00 to 9/01/01). Note exceedence of 40 µg/m³ AQI Level of Concern on September 13 and likely again on October 4; red portion of bar indicates 24-hr average if three high hourly values (> 100 µg/m³) being evaluated for data quality are included in 24-hr average calculation.

Figure 14. ORD measurement of PM_{2.5} elemental constituents Ca, Si, and K at Ground Zero perimeter sites and 290 Broadway site.

Figure 15. ORD measurements of PM_{2.5} elemental composition for S, Cl, and Br at Ground Zero perimeter sites and 290 Broadway site.

Figure 16. ORD measurements of PM_{2.5} elemental constituents Pb, Cu, and Zn at Ground Zero perimeter sites and 290 Broadway site.

Figure 17. ORD measurements of PM_{2.5} elemental constituents As, Pd, and Sb at Ground Zero perimeter sites and 290 Broadway site.

Figure 18. ORD measurements of PM_{2.5} elemental constituents Ni, Cd, and Cr at Ground Zero perimeter sites and 290 Broadway site.

Figure 19. PM_{2.5} concentrations recorded on rooftop of DOE Facility at Varick St., approximately 2.0 miles N/NE of Ground Zero. Note single very high PM_{2.5} excursion mainly restricted to morning hours of October 3 (see inset figure for October 3), consistent with ORD measurements at location A on the WTC north perimeter and Ground Zero plume plot shown in Figure 2 for October 3.

Figure 20. Ambient air lead concentrations (µg/m³) at sites within Ground Zero or in lower Manhattan locations in immediate vicinity of the WTC.

Figure 21. Location of PCB monitoring stations.

Figure 22. Location of dioxin air monitoring. The locations marked “N” are New York State Department of Environmental Conservation (NYSDEC) samplers maintained by EPA Region 2 with analysis of the samples by Region 7 (Region 2/7), whereas all other samplers are maintained by EPA’s Environmental Response Team (EPA ERT). See text for discussion of the differences in the two sets of data from these two air monitoring teams.

Figure 23. Location of asbestos monitoring stations in Lower Manhattan.

Figure 24. Location of asbestos monitoring stations in Staten Island and nearby locations in New Jersey (note: sampling sites in the Staten Island Landfill identified only by number).

Figure 25. North-South directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

Figure 26. East-West directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

Figure 27. Northwest-Southeast directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

Figure 28. Northeast-Southwest directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

Figure 29. North-South directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

Figure 30. East-West directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

Figure 31. Northwest-Southeast directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

Figure 32. Northeast-Southwest directional analysis for asbestos TEM weekly maximums (taken from EPA, 2002a).

Figure 33. Location of VOC monitoring stations outside Ground Zero.

List of Acronyms and Abbreviations

ACGIH	American Conference of Governmental Industrial Hygienists
ACM	asbestos-containing materials
ADD	Average Daily Dose
AHERA	Asbestos Hazard Emergency Response Act
AIRS	Aerometric Information Retrieval System
AQI	Air Quality Index
As	arsenic
ATSDR	Agency for Toxic Substances and Disease Registry
Br	bromine
Ca	calcium
cc	cubic centimeters
Cd	cadmium
CDC	Centers for Disease Control and Prevention
Cl	chlorine
CO	carbon monoxide
Cr	chromium
Cu	copper
DL	detection limit
DOE	Department of Energy
EOHSI	Environmental and Occupational Health and Safety Institute
EPA	Environmental Protection Agency
EPA ERT	Environmental Protection Agency Environmental Response Team
EPA-STSC	Environmental Protection Agency Superfund Toxics Support Center
ESD	Environmental Services Division, of EPA's Region 7 Office in Kansas City
f	fibers of asbestos
FDNY	Fire Department of New York
FEMA	Federal Emergency Management Agency
GFF	glass fiber filter
HEAS	Human Exposure and Atmospheric Sciences Division, in EPA's NERL
IBB	increment in body burden
K	potassium
kg	kilogram
LADD	lifetime average daily dose
LOC	level of concern
LW	lipid weight
MCL	Maximum Contaminant Level
µg	microgram
µm	micrometer
MOE	Margin of Exposure
MRL	Minimum Risk Level
NAAQS	National Ambient Air Quality Standard
NCEA	National Center for Environmental Assessment, of EPA's Office of Research and Development

ND	Non-detect
NERL	National Exposure Research Lab of EPA's Office of Research and Development
ng	nanogram
NHEERL	National Health and Environmental Exposure and Risk Laboratory, of EPA's Office of Research and Development
NIEHS	National Institute of Environmental Health and Safety
NIOSH	National Institute for Occupational Safety and Health
NO ₂	nitrogen dioxide
NOAEL	no-observed-adverse-effect level
NY	New York
NYC	New York City
NYCDEP	New York City Department of Environmental Protection
NYCDOHMH	New York City Department of Health and Mental Hygiene
NYSDOH	New York State Department of Health
NYSDEC	New York State Department of Environmental Conservation
ORD	EPA's Office of Research and Development
OSHA	Occupational Safety and Health Administration
PAH	polycyclic aromatic hydrocarbons
Pb	lead
PCB	polychlorinated biphenyl compounds
PCM	phase contrast light microscopy
Pd	palladium
PEL	Permissible Exposure Limit
pg	picogram
PM, PM _{2.5} , PM ₁₀	Particulate matter, and PM at less than 2.5 µm and less than 10 µm diameter
ppb	part per billion
ppm	part per million
PS	public school
PUF	polyurethane foam plug for air monitoring
REL	Recommended Exposure Levels
RfC	Reference Concentration
S	sulfur, or structures of asbestos
Sb	antimony
SF	cancer slope factor
Si	silica
SO ₂	sulfur dioxide
SRIXE	synchrotron radiation-induced X-ray emission
STEL	Short Term Exposure Level
STSC	Superfund Technical Support Center
TEF	toxicity equivalency factor
TEM	transmission electronic microscopy
TEOM	tapered element oscillating microbalance
TEQ	toxic equivalent concentration
TLV	Threshold Limit Value

TWA	time-weighted average
UR	unit risk, for estimating cancer risk due to inhalation
USGS	United States Geological Survey
VAPS	versatile air pollutant sampler
VOC	Volatile organic compound
WTC	World Trade Center
XRF	x-ray flourescent
Zn	zinc

FOREWORD

The National Center for Environmental Assessment (NCEA), a major component of the Office of Research and Development (ORD), is EPA's national resource center for human health and ecological risk assessment. NCEA conducts risk assessments, carries out research to improve the state-of-the-science of risk assessment, and provides guidance and support to risk assessors.

Following the collapse of the World Trade Center towers on September 11, 2001, New York State and Federal agencies initiated numerous air monitoring activities to better understand the ongoing impact of emissions from the disaster. This report focuses on these air measurement data, evaluating them in terms of what is typical for New York City or general urban background and interpreting it with regard to the potential for human health consequences. The report does not evaluate exposures possibly faced by rescue or clean-up workers and briefly discusses past and current indoor monitoring efforts.

The analysis in this report supports three general findings: 1) Persons exposed to the extremely high levels of ambient particulate matter and its components during the collapse of the World Trade Center towers and for several hours afterwards were likely to be at risk for immediate acute (and possibly chronic) respiratory and other types (e.g., cardiovascular) of symptoms. 2) The first measurements of some of the contaminants were on September 14, while other contaminants were not measured until September 23. Available data suggest that the concentrations within and near Ground Zero were likely to be highest in the few days following September 11. Because there are only limited data on these critical few days, exposures and potential health impacts cannot be evaluated with certainty for this time period. 3) Except for exposures on September 11 and possibly during the next few days, persons in the surrounding community were unlikely to suffer short-term or long-term adverse health effects caused by exposure to elevations in ambient air concentrations of the contaminants evaluated in this report. These elevated concentrations were measured mostly within and near Ground Zero, and they lasted for one to three months after September 11. The monitoring data indicate that air concentrations decreased to background levels that are characteristic of pre-September 11 levels in the New York City metropolitan area by around January or February of 2002.

Ultimately, it will be difficult to ascertain with certainty what effects resulted when people were surrounded by initial clouds of dust, or were subsequently exposed to the elevated concentrations that are discussed in this report. Epidemiologic studies of the exposed populations that are being conducted by various agencies and institutions should provide a more scientifically robust evaluation for future evaluations of health effects.

George W. Alapas
Acting Director
National Center for Environmental Assessment

AUTHORS, CONTRIBUTORS, AND REVIEWERS

The National Center for Environmental Assessment (NCEA), Office of Research and Development, was responsible for preparing this document. Matthew Lorber and Herman Gibb, of NCEA, provided overall direction and coordination of the production effort.

AUTHORS

Executive Summary	Herman Gibb Matthew Lorber Linda Tuxen
Section I. Overall Purpose and Scope of Assessment	Herman Gibb Matthew Lorber John Schaum
Section II. Exposure Assessment and Risk Characterization Approach	Matthew Lorber Herman Gibb Nancy B. Beck
Section III. Environmental Monitoring	Herman Gibb Matthew Lorber
Section IV. Evaluation	
IV.a. Particulate Matter	Lester Grant Joseph Pinto
IV.b.1. Lead	Lester Grant Joseph Pinto
IV.b.2. Chromium and Nickel	Nancy B. Beck
IV.c. Polychlorinated Biphenyls	David Cleverly
IV.d. Dioxins	Matthew Lorber
IV.e. Asbestos	Matthew Lorber Aparna Koppikar Paul White
IV.f. Volatile Organic Compounds	Nancy B. Beck Matthew Lorber
V. Comment on the First Several Days After September 11	Matthew Lorber

VI. Data on Indoor and Occupational Exposures

Matthew Lorber
John Schaum

VII. Comments and Future Studies

Herman Gibb
Matthew Lorber
Nancy B. Beck

VIII. References

CONTRIBUTORS

The following individuals have also provided valuable support in the review and comment of this document, as well as supplying text and other materials for individual sections:

Stephen H. Gavett, NHEERL/ORD/US EPA
Sven Rodenbeck, ATSDR
Audrey Galizia, Region 2, US EPA
Barbara Finazzo, Region 2, US EPA
Mark Maddaloni, Region 2, US EPA
Marcus Kantz, Region 2, US EPA
Robert Kelly, Region 2, US EPA

REVIEWERS

Philip J. Landrigan, M.D., M.Sc.
Ethel H. Wise Professor Community Medicine
Chairman, Department of Community and Preventive Medicine
Mount Sinai School of Medicine
One Gustave L. Levy Place
Box 1057
New York, New York 10029-6574

Paul J. Liroy, Ph.D
Associate Director
Environmental and Occupational Health Sciences Institute of New Jersey
UMDNJ-Robert Wood Johnson Medical School, and Rutgers University
170 Frelinghuysen Road
Piscataway, NJ 08854

George D. Thurston, Ph.D
Associate Professor
NYU School of Medicine
National Institute of Environmental Medicine
57 Old Forge Rd.
Tuxedo, NY 10987

Executive Summary

In the days following the September 11, 2001, terrorist attack on New York City's World Trade Center (WTC) towers, many federal agencies, including the United States Environmental Protection Agency (EPA), were called upon to bring their technical and scientific expertise to the national emergency. EPA, other federal agencies, and New York City and New York State public health and environmental authorities initiated numerous air monitoring activities to better understand the ongoing impact of emissions from that disaster. These efforts generated an immense amount of data. Many EPA offices and programs quickly became involved with these activities, providing scientific, engineering, public health, and management expertise to help cope with the after effects of the collapse of the WTC towers. EPA Region 2, which includes the New York City metropolitan area in New York and New Jersey, is the Agency's lead office on these activities, including the important and complicated task of community outreach and communication. As part of these activities, Region 2 requested that EPA's Office of Research and Development (ORD) conduct a human health evaluation of exposure to air pollutants resulting from the WTC disaster.

The evaluation in this report relies primarily on the analyses of ambient air samples from monitors located at the perimeter of the WTC Ground Zero and at various other sites in lower Manhattan and surrounding areas. It is an assessment of the inhalation exposure and potential human health risk incurred by the general population residing and working in the vicinity of the WTC. Numerous other efforts have been conducted or are ongoing that address other aspects of exposure and potential risk associated with the collapse of the WTC towers, including:

1) Ground Zero worker exposures: This report reviews some of the data collected by the Occupational Safety and Health Administration (OSHA) and the National Institute of Occupational Safety and Health (NIOSH) that address the exposures faced by fireman and rescue workers, but does not explicitly evaluate these exposures.

2) Indoor exposures: Similarly, this report reviews some of the data collected on indoor air and dust, particularly a recently completed study by the New York City Department of Health and Mental Hygiene (NYCDOHMH) and the Agency for Toxic Substances and Disease Control (ATSDR). It also provides an overview of the ongoing efforts by EPA Region 2 to clean apartments and evaluate the quality of indoor air and dust. Future reports by EPA Region 2 will detail these efforts and monitoring results.

3) Epidemiology studies: Chapter 7 of this report provides an overview of the types of studies that are ongoing which will evaluate health impacts experienced by workers and others known to be in the vicinity of WTC in the days and weeks following September 11, 2001. These studies are being conducted and sponsored by the National Institute of Environmental Health Studies (NIEHS), and others.

The ambient air monitoring activities described in this report were undertaken by federal, state and local agencies which have made their analytical results available to EPA for analysis. Most of the monitors were placed following the disaster, with the intent of characterizing outdoor levels of WTC-generated air pollutants at locations surrounding the WTC site at

different distances. Some monitors for particulate matter (PM), operated by New York State, existed prior to the disaster.

This report focuses on: *PM, metals (lead, chromium and nickel compounds), polychlorinated biphenyls (PCBs), dioxin-like compounds, asbestos, and volatile organic compounds (VOCs)*. These substances are included because monitoring indicated that they correlated with the disaster site in both time and space, and because they pose a potential concern for health impacts. PM was generated by the collapse of the WTC buildings, the recovery and demolition operations, and the lingering fire. Lead and asbestos were believed to be components of the WTC building materials. PCBs were used as dielectric fluid in transformers and capacitors. Dioxin and VOCs are produced as a result of combustion and volatilization from fuels. The assessment is limited to an evaluation mainly of the inhalation of airborne contaminants, although dust ingestion and dermal contact may also have led to exposures within and near Ground Zero.

Elevated concentrations of these contaminants were found within and near Ground Zero for a short period of time after September 11. "Elevated" is used in this discussion to denote concentrations of a contaminant that were significantly higher, by a factor of 10 or more and often by factors of 100 or 1000, compared to other measurements of the contaminant taken in the WTC monitoring program or compared to concentrations that are typically found in New York City or in general United States urban settings. Many of these elevated measurements were identified as having occurred in "restricted zones," that is, zones where access was limited to emergency management and rescue personnel and to other credentialed people. In general, the monitoring data, even within Ground Zero, indicate that ambient air levels for all of these substances decreased to background ambient concentrations that are characteristic of pre-September 11 levels in the New York City metropolitan area by around January or February of 2002.

The analysis in this report finds that:

- ***Persons exposed to the extremely high levels of ambient particulate matter and its components during the collapse of the World Trade Center towers and for several hours afterwards were at risk for immediate acute (and possibly chronic) respiratory and other types (e.g., cardiovascular) of symptoms.***
- ***The first measurements of some of the contaminants were on September 14, while other contaminants were not measured until September 23. Available data suggests that the concentrations within and near Ground Zero were likely to be highest in the few days following September 11. Because there are only limited data on these critical few days, exposures and potential health impacts cannot be evaluated with certainty for this time period.***
- ***Except for exposures on September 11 and possibly during the next few days, persons in the surrounding community were unlikely to suffer short-term or long-term adverse health effects caused by exposure to elevations in ambient air concentrations of the contaminants evaluated in this report. These elevated concentrations were***

measured mostly within and very near Ground Zero, and they lasted for 1 to 3 months after September 11.

While the conclusions reached in this report represent the current scientific understanding of the toxicity that these contaminants pose to people, combined with EPA's evaluation of exposure to these contaminants based on available air monitoring data, it cannot be stated with certainty what effects resulted when people were engulfed in the initial cloud of dust or were subsequently exposed to the elevated concentrations that were found. Epidemiologic studies of the exposed populations that are currently being conducted by various agencies and institutions should provide a more scientifically robust evaluation for future evaluations of health effects.

The risk evaluation approach taken in most instances was to compare the measured air levels at locations near Ground Zero to established health benchmarks for inhalation exposure and to typical urban background levels. OSHA Permissible Exposure Levels (PELs), NIOSH Recommended Exposure Levels (RELs), and Agency for Toxic Substances and Disease Registry (ATSDR) Minimum Risk Levels (MRLs) were among the benchmarks included in this evaluation. Where available, benchmarks established to protect against acute and subchronic exposures were used. Benchmarks that are intended to protect against exposures lasting over one year or throughout a lifetime, like the EPA Reference Concentration (RfC), were only used if other more appropriate benchmark values were not available.

A simple comparison of air measurements to health benchmarks or to typical background levels can be thought of as a "screening" exercise; the purpose of the exercise is to identify possible problems. If the majority of samples are much less than a benchmark, in most cases it would be appropriate to conclude that a health impact is unlikely. Similarly, if most air measurements are similar to typical background levels, then it can be concluded that emissions from the WTC are not impacting air or influencing exposure and health. On the other hand, if most samples exceed a benchmark, then it may be appropriate to consider the possibility that a health impact may have occurred, or could occur, depending on the circumstances.

Efforts will continue at EPA to further characterize exposures and health impacts that resulted from the collapse of the WTC Towers, and to build on the risk evaluation presented in this report. Some additional future considerations could include: evaluating other contaminants that were measured, evaluating the indoor environment in more depth, evaluating other pathways of exposure and other exposure media such as dermal contact to contaminated dust, investigating the combined effects of exposure to more than one contaminant, conducting further toxicity testing with laboratory animals, and considering results from ongoing epidemiological studies.

Summaries of the findings for each contaminant/class of contaminants are presented below:

Particulate Matter. *People caught in the initial dust/smoke cloud that encompassed lower Manhattan after the collapse of the WTC buildings on September 11 were briefly exposed (4 - 8 hours) to quite high levels (in the milligrams per cubic meter, mg/m³, range) of airborne particulate matter (PM). Also, during the first several days after the disaster, PM levels in the air at the WTC perimeter exceeded EPA's daily PM_{2.5} NAAQS (65 µg/m³, 24-hr); and PM_{2.5}*

concentrations at some other nearby lower Manhattan sites exceeded EPA's 40 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) 24-hour Air Quality Index (AQI) level of concern for susceptible subgroups. The high PM concentrations recorded very near WTC Ground Zero during late September and early October may imply increased chronic health risks for the most highly exposed individuals (e.g., persons spending extended periods of time within the WTC work zone without wearing protective respirators). By mid- to late October, PM values across lower Manhattan had largely returned to levels typical of New York City and other US urban areas, with only a few WTC or nearby sites occasionally approaching or exceeding the AQI level of concern.

Many individuals were exposed for a few hours to very high PM concentrations in the initial dust cloud that spread over lower Manhattan on September 11. Also, high levels of airborne particles were detected during the first several days after September 11 at a few already existing PM monitoring sites scattered across the New York City area. Hourly or daily fine particle (less than $2.5\ \mu\text{m}$ in diameter, $\text{PM}_{2.5}$) values at some new sites set up by EPA or New York State exceeded $40\ \mu\text{g}/\text{m}^3$ (though the AQI applies only to the daily averages).

$\text{PM}_{2.5}$ measurements from newly established monitoring sites around the WTC perimeter varied widely, depending on wind direction. Daily average (24-hr) $\text{PM}_{2.5}$ concentrations on some days exceeded $200\ \mu\text{g}/\text{m}^3$ at one WTC perimeter site or another during late September and early October. However, $\text{PM}_{2.5}$ concentrations decreased rapidly with distance from the WTC, with few $\text{PM}_{2.5}$ values exceeding the $40\ \mu\text{g}/\text{m}^3$ AQI at monitoring locations ranging from 3 to 10 blocks away from the WTC. During the entire period following September 11, $\text{PM}_{2.5}$ values recorded at lower Manhattan sites away from the WTC perimeter were not markedly different than during periods before or since, as the New York metropolitan area routinely experiences $\text{PM}_{2.5}$ values near and above the AQI.

Concentrations of various elements (e.g., calcium, sulfur, silicon, lead, and other metals) in WTC $\text{PM}_{2.5}$ particles also were enriched above typical background levels on an episodic basis at sites mainly on or near the WTC perimeter, including on some days extending into late October and into November.

The issue of alkalinity of WTC dust and its potential as a possible health concern for exposed individuals is raised by observations by the United States Geological Survey (USGS) and academic researchers of high pH (> 11.0) of aqueous solutions of settled WTC dust not leached by rainfall. After late September, indoor exposures to such dust probably warrant more concern than outdoor exposures for possible acute irritative effects or more chronic health effects, not only because of the basic nature of some constituent particles but also because of other unusual features, such as slender microscopic glass fibers with toxic materials attached to them or very fine particles composed of unusual combinations of silica coalesced with lead or other toxic materials.

Metals

Lead. *Persons caught in the initial WTC-related dust cloud experienced brief exposures to high levels of lead (Pb), based on analyses of deposited dust samples. In late September 2001, air lead concentrations at the WTC perimeter sites reached levels above $1.5 \mu\text{g}/\text{m}^3$ on some days. However, the air lead levels averaged over 90 days (late September - late November) did not exceed the EPA National Ambient Air Quality Standard (NAAQS) of $1.5 \mu\text{g}/\text{m}^3$ averaged over a 90-day period. After mid-October, air lead at all sites in lower Manhattan outside WTC Ground Zero dropped to levels more comparable with background concentrations typical of NYC and other northeastern United States urban areas. On the basis of ambient air and dust data, there is little indication of any substantial health risks being associated with lead exposures of the general population in lower Manhattan areas around the WTC site.*

On several days in late September 2001, 24-hour Pb concentrations at the WTC Ground Zero perimeter sites exceeded $1.5 \mu\text{g}/\text{m}^3$. This level was again approached or exceeded at one or another WTC perimeter site during early October (10/3 -10/5). However, airborne Pb rapidly decreased with distance from the WTC perimeter sites, with Pb concentrations on the same days when Pb levels were elevated at Ground Zero being substantially lower at several locations within 3-10 blocks from the WTC (i.e., mostly within the 0.11 to $0.63 \mu\text{g}/\text{m}^3$ range of 24-hour Pb levels observed at some Manhattan and Brooklyn sites during the 1990's). After mid-October, 2001 Pb readings for all WTC perimeter sites and other lower Manhattan sites remained below $1.5 \mu\text{g}/\text{m}^3$, with few even exceeding $0.5 \mu\text{g}/\text{m}^3$.

Lead concentrations in bulk dust samples taken close to the WTC within days after September 11 ranged up to $625 \mu\text{g}/\text{g}$ (ppm). This level is well below street dust Pb levels on or near heavily traveled roadways prior to phase-down of Pb in gasoline in the late 1970's, which often were well in excess of 1000 - 2000 ppm, and it compares well with the 500 - 1000 ppm street dust or soil lead levels found in northeastern or midwestern U.S. urban areas well into the 1990s.

In general, the observed ambient air lead levels did not appear to pose increased health risks for the general public. However, susceptible persons (especially any pregnant women) who may have experienced extended exposures to elevated Pb levels within WTC Ground Zero work areas while not wearing appropriate respiratory protective gear or who were exposed to indoor WTC-derived dusts with high Pb loadings could possibly be at increased risk for chronic health effects. Evaluation of blood lead levels and pertinent medical records for any pregnant women exposed at Ground Zero or in its immediate vicinity during late September/early October could provide useful further data by which to assess any such possible health risks associated with WTC-generated lead emissions.

Chromium. *Samples evaluated for total chromium at Ground Zero and at sites surrounding Ground Zero never exceeded the OSHA PEL of $1 \text{ mg}/\text{m}^3$ or the ATSDR Intermediate Minimum Risk Level (MRL) for chromium VI particulates of $1.0 \mu\text{g}/\text{m}^3$. On the basis of the samples evaluated, exposures to chromium were not likely to cause any adverse health effects.*

All 21 Ground Zero air samples evaluated for total chromium were collected at Building 5 between September 23 and January 31. Additionally, approximately 512 air samples collected at sites surrounding Ground Zero were evaluated for chromium, including 86 samples collected at landfills and 16 samples from personal air monitors worn by New York City fire department personnel. No exceedences of the OSHA PEL or the ATSDR Intermediate MRL for chromium VI particulates were detected. However, it was noted that concentrations in the range of 0.20 and 0.40 $\mu\text{g}/\text{m}^3$ were measured for about a month month after September 11, to then drop to more typical urban backgrounds less than 0.10 $\mu\text{g}/\text{m}^3$.

Nickel. *Nickel samples evaluated at Ground Zero and at sites surrounding Ground Zero never exceeded the OSHA PEL of 1 mg/m³. On the basis of samples evaluated, exposures to nickel were not likely to cause any adverse health effects.*

All 21 Ground Zero air samples evaluated for nickel were collected at Building 5 between September 23 and January 31. Additionally, approximately 637 air samples collected at sites surrounding Ground Zero were evaluated for nickel, including 86 samples collected at landfills. No exceedences of the OSHA PEL were detected. Furthermore, and unlike chromium and other contaminants, all measurements from September 11 on at all sites were at background levels.

Polychlorinated Biphenyls. *Of the several hundred polychlorinated biphenyl (PCB) air measurements available, only one sample was elevated above 100 nanograms total PCB per cubic meter (ng/m³), at 153 ng total PCB/m³, and only three samples were above 50 ng total PCB/m³. This compares to typical urban background PCB concentrations in the range of 1 - 8 ng total PCB/m³. After a month, nearly all readings were in the range of typical urban PCB concentrations or were not detected. There were no exceedences of any short-term occupational health benchmark, including the NIOSH REL of 1*10³ ng/m³ or the OSHA PEL of 5*10³ ng/m³. It is concluded that exposures were of minimal concern for cancer risk.*

Several hundred PCB air measurements were obtained at a total of 12 locations in the vicinity of Ground Zero from September 16, 2001, through April 24, 2002. The highest PCB air concentration measured was 153 ng PCB/m³, and this occurred on October 2, 2001, at the Ground Zero site, WTC Building 5 SW. Typical urban air concentrations of PCBs are in the range of 1 - 8 ng/m³. The source of these elevated PCB air measurements is speculated to be the smoke emanating from the smoldering fires at Ground Zero. PCBs were entrained in the smoke as a consequence of PCB-containing materials in the WTC buildings. After November 3, 2001, all of the PCB monitoring sites showed results consistent with PCB levels in air that are typical of urban areas of the U.S. A simple screening exercise showed that an incremental lifetime cancer risk due to exposure to short-term elevation of PCBs would be in the range of 10⁻⁸ or lower, which is judged to be of minimal concern. With respect to non-cancer effects, all PCB air measurements are several orders of magnitude below No Observed Effect Levels (NOELs) in experimental animal studies. In addition, levels of PCBs observed near or at the WTC site are below the NIOSH REL of 1*10³ ng/m³ (NIOSH, 2002), and several

orders of magnitude below the OSHA PEL of 5×10^5 ng/m³. The NIOSH REL is an 8-hr time-weighted average air concentration. It is associated with long-term or repeated exposures, and is protective of effects on the liver and the reproductive system. The OSHA PEL is also an 8-hr time-weighted average air concentration. It is associated with long-term or repeated exposures and is protective of effects on the skin (dermatitis).

Dioxins. *Monitoring data indicate that dioxin toxic equivalent (TEQ) levels in air near the WTC were up to three orders of magnitude higher (1000 times higher) than is typical for urban areas in the United States. Typical levels for urban areas are 0.1 to 0.2 picograms of TEQ per cubic meter (pg TEQ/m³), while levels found in Ground Zero and near Ground Zero, starting September 23 (the date of the first sample taken) and continuing through late November ranged from 10 to over 150 pg TEQ/m³. Concentrations measured several blocks from Ground Zero were still elevated above typical urban background, but considerably lower than sites in or near ground zero, ranging from 1 to 10 pg TEQ/m³ during this same time period. Everywhere these elevations dropped rapidly, and the data suggest that by December 2001, levels decreased to background levels. These levels need to be considered in the context of total exposure to dioxin, 95% of which is attributed to dietary intake in normal background settings. Therefore, although inhalation exposure to dioxin at these elevated air concentrations is significantly higher than typical inhalation exposure to dioxin, an individual's overall exposure to dioxin may not be impacted significantly. An exposure and risk screening exercise was conducted with available monitoring data, and the results suggest that these elevations did not result in a significant elevation in cancer or non-cancer risk over the background risk for exposure to dioxin-like compounds.*

The monitoring data indicate that, through late November, dioxin TEQ levels in air near the WTC were distinctly elevated compared with typical levels in urban air. An exposure and risk screening exercise based on these high concentrations suggest a temporary elevation in exposures for Ground Zero workers but very minimal impact for nearby residents and office workers. It is concluded that these potential exposures during 2001 do not constitute a public health concern. Dietary intake of dioxins is much higher than inhalation intake, and thus, the ambient concentrations of dioxin within and near Ground Zero, although considerably elevated above typical urban air concentrations of dioxin, are not as significant as suggested by the orders of magnitude in elevation indicated above.

However, much of the data obtained from within and near the WTC site are of limited interpretive value due to high detection limits. When dioxin-like compounds were not detected in an air sample, the TEQ concentration was determined by assuming that each dioxin-like compound was present in the air at one-half the detection limit for that compound. This is typical for calculating dioxin TEQ concentrations, and for other contaminants as well. Because dioxin-like compound concentrations were considerably elevated in the ambient air from September through late November 2001 within and near Ground Zero, these concentrations were able to be measured, despite high detection limits. Concentrations ranged from 10 to 150 pg TEQ/m³ during this time, which was between 100 and over 1500 times higher than typically found in urban air.

The reported TEQ concentrations that were compromised due to high detection limits ranged in value from 0.5 to 5.0 pg TEQ/m³, which is about 5 to 50 times higher than normal urban background air concentrations. These measurements came from 9 specific air samplers which were located within Ground Zero (1 sampler) and near the WTC site (8 samplers). In general, the dioxin method's detection limit is calculated by dividing the lowest mass of dioxin that the method can detect by the air that contained that amount, namely the air that flowed through the sampler. Thus, as more air is drawn through the sampler, the detection limit is lowered. These 9 air samplers operated for 8-hour periods, and drew in about 7 m³ of air. They began operation on September 23, 2001.

Three other air samplers were operated for 72-hour periods, and drew in about 1000 m³ of air. These monitors were located several blocks from the perimeter of Ground Zero, and began operation on October 12. Because much more air was drawn into these samplers, the detection limits obtained for the dioxin-like compounds were lower, and TEQ concentrations could be routinely quantified as levels less than 1.0 pg TEQ/m³. The first sampling events in October through November in these three monitors resulted in concentrations that were still elevated above typical background TEQ concentrations, at between 1 and 10 pg TEQ/m³. Starting at the beginning of December, 2001, and continuing through the termination of sampling in May of 2002, the measurements in these samplers decreased to levels that were mostly less than 0.10 pg TEQ/m³. More detail on these two sets of monitors (the 9 monitors sampling for 8 hours, and the 3 monitors sampling for 72 hours) is provided in Section IV.d below.

All the reported TEQ measurements from these 9 monitors for 2002, and the measurements from them in upwind conditions during 2001 (i.e., when the plume was moving in a direction opposite the monitors), were in this one-half detection limit range of 0.5 to 5.0 pg TEQ/m³. Thus, the ability to assess exposure during the early months of 2002, where there may have been elevations near Ground Zero as a result of cleanup activities, was compromised. Because the health risk from dioxin exposure is associated with accumulation of residues in body tissues, continued dioxin TEQ exposure within and near Ground Zero throughout 2002 could not be evaluated. The risk screening exercises conducted for dioxin were limited to the time period when the concentrations were highest and dioxin was detected. This issue is described in more detail in Section IV.d.

Asbestos: *The large majority of outside air measurements of asbestos were below established benchmarks and within the range of typical background levels. However, and similar to other contaminants, the few exceedences that were measured occurred near September 11 in time and close in proximity to the WTC. Limited available evidence suggest the incursion of asbestos to the indoor environment. A small study which sampled the indoor environment of two apartments on September 18 showed very high indoor levels of asbestos. A larger and more systematic study which sampled in November and December of 2001 suggested that indoor levels of asbestos in dust were slightly higher near the WTC as compared to indoor levels in dust further away. Current efforts by the EPA focus on the measurement and clean-up of residential apartments near the WTC.*

A total of 12,676 ambient samples in lower Manhattan were measured by phase contrast light microscopy (PCM, used to identify structures greater than 5 μm in length), and 8,872 of these were also measured by transmission electronic microscopy (TEM; used to identify structures less than 0.5 μm in length). Only 23 samples were found to exceed the Asbestos Hazard Emergency Response Act (AHERA, which uses the TEM measurement technique) abatement standard of 70 structures per square millimeter (S/mm²), and there were no exceedences of the OSHA PEL (which uses the PCM measurement technique) of 0.1 fiber per cubic centimeter (f/cc). Most of the exceedences of the AHERA standard occurred during September 2001 adjacent to Ground Zero in "restricted zones". The highest concentrations of PCM-measured fibers in ambient air were recorded during the 30 days following September 11, 2001, at sites in the vicinity of the WTC. Concentrations during this time were in the range of 0.04 to 0.08 f/cc. There has been a steady decline in the asbestos levels through the first few months of 2002, to correspond to a steady background state of ND to <20 S/mm² as measured by TEM and in the range of 0.003 f/cc as measured by PCM, a level which is typical for urban background.

A slightly higher occurrence rate of AHERA exceedences occurred within the exclusion zone at the Staten Island Landfill; 51 out of 5,207 samples taken. Most of the exceedences occurred during October and November 2001, corresponding to the time that most of the debris was being unloaded. Only one sample in Queens was above 70 S/mm², whereas no exceedences were observed in the other boroughs of NYC or in New Jersey.

The highest measurements of asbestos available for evaluation in this report were taken within two apartments sampled on September 18, 2001. One apartment was highly affected by the collapse of the WTC towers with completely shattered windows and dust piled throughout the apartment. The other was in a building that had little exterior damage, but had visible dust on surfaces within the building and in the apartment sampled. In the severely damaged apartment, five air measurements of asbestos ranged from 6277 to 10,620 S/mm² using the AHERA protocol. One sample taken just outside on a window ledge of this apartment measured 548 S/mm². However, it is likely this high reading was influenced by the air quality on the inside of the apartment, which showed exceedingly high asbestos concentrations, and was likely not representative of outdoor concentrations. The six indoor samples in the less impacted apartment exceeded the 70 S/mm² AHERA standard at levels ranging from 141 to 379 S/mm². A rooftop sample at this location was low at 6.5 S/mm².

A systematic study of residential apartments by the NYCDOHMH and the ATSDR showed very little impact to residential apartments compared with this September 18 study, but still a difference between apartments in lower Manhattan and comparison apartments. From November 4 through December 11, 2001, environmental samples were collected in and around 30 residential buildings in lower Manhattan. In addition, four buildings above 59th Street were sampled and used as a comparison area for this investigation. Importantly, asbestos was not detected above background levels in air samples in all apartments sampled (with background defined as 0.003 f/cc, a level which is typical for urban background). Bulk dust samples were collected both indoors and on

outdoor surfaces and analyzed for the presence of asbestos by both PLM (polarized light microscopy) and TEM. Asbestos was detected in settled indoor dust in 10 of 57 (16%) lower Manhattan residential units sampled, with the positive samples showing a maximum of 1.5% asbestos in dust. By comparison, no asbestos was detectable in dust samples collected in the 5 comparison residences. In outdoor dust collected at lower Manhattan properties, asbestos was detected in 6 of 14 (43%) samples, with a maximum asbestos concentration in dust of 3.4%.

Volatile Organic Compounds. *The Ground Zero samples of volatile organic compounds (VOCs) were generally not taken in the breathing zone of workers and were not representative of the general air quality at the site. Most of the data were collected within plumes from fires and smoldering rubble to alert the Fire Department of New York (FDNY) and the contractors/union health/safety officers working at Ground Zero about conditions that posed immediate health concern to the workers and, as such, were more representative of emissions rather than exposures. For this reason, an analysis of Ground Zero worker exposures for VOCs was not conducted. However, eleven VOCs were evaluated at sites surrounding Ground Zero. No exceedences of screening benchmarks were seen for 1,4-dioxane, ethanol, styrene, tetrahydrofuran, and xylenes. Exceedences of screening benchmarks were seen for acetone, benzene, 1,3-butadiene, chloromethane, ethylbenzene, and toluene. Except for benzene, exceedences for these chemicals occurred in restricted zones. Also, the exceedences were all grab samples. Twenty-four hour samples of benzene, 1,3-butadiene, ethylbenzene, and toluene all were about three orders of magnitude (1000 times) lower than the grab samples, demonstrating the difference between 4-minute grab samples taken within plumes and day-long averages. The exceedences for benzene were more frequent, some were further from Ground Zero than the other VOCs, and the 24-hour samples were lower but within a factor of 10 of the grab sample exceedences. This suggests that elevated concentrations of benzene (above typical background by about a factor of 10) may have been sustained for a month or more after September 11.*

On the basis of available monitoring data, it is concluded that the exceedences of the screening benchmarks in restricted zones for acetone, 1,3-butadiene, chloromethane, ethylbenzene, and toluene do not represent a public health risk to persons living or working at sites surrounding Ground Zero.

The data for benzene were not as definitive. Because the 24-hour samples were measured at levels that were closer in magnitude to the grab sample exceedences than the other VOCs, within a factor of 10, this would suggest that the grab sample concentrations were closer to sustained concentrations rather than short-term plume concentrations only. Also, these 24-hour concentrations were near the ATSDR Intermediate MRL of 0.004 ppm and higher than the historical average for New York City of about 0.0005 ppm. The data suggest that the possible exposures to benzene at levels that approach the MRL did not last longer than 45 days. Whether or not specific health effects occurred due to exposure to benzene is unknown, but given that the exceedences and elevations above typical background were near Ground Zero and mostly within restricted zones, the data suggests that the exposures to the general population were of minimal concern.

Air Concentrations During the First Several Days After September 11: An event such as September 11 demonstrates that the greatest environmental impacts occur in the first 24 to 48 hours and in areas close to the site. Difficulties associated with site access and security, power supply sources, equipment availability and analytical capacity hindered efforts by EPA and the New York State Department of Environmental Conservation (NYSDEC) to put air monitors in place immediately after the attack. While dust samples were collected for analysis on September 11, the first air samples of some of the critical contaminants were not taken until September 14, such as asbestos, while other contaminants were not sampled until September 23, such as dioxin. Rapid initiation of monitoring will allow the measurement of air concentrations that can be very important for evaluation of inhalation exposures and potential short and long-term human health impacts. An examination of the concentrations measured during September show that the highest concentrations were the ones taken closest in time to September 11, closest in proximity to Ground Zero, and in the downwind direction. For example, five measurements of dioxin TEQs were over 100 pg TEQ/m³ (all others were under 100 pg TEQ/m³), and these were the first measurements in the three nearest downwind monitors: the first 3 measurements in the WTC Building 5 monitor on September 23 (160 pg TEQ/m³), October 2 (170 pg TEQ/m³), and October 4 (170 pg TEQ/m³), the first measurement at the Church and Dey monitor on September 23 (130 pg TEQ/m³), and the first measurement at the Liberty and Broadway monitor on September 23 (100 pg TEQ/m³). While the highest dioxin measurements were found on September within and to the east of Ground Zero, three samplers to the west of Ground Zero showed background levels of dioxins on September 23. Similar trends were seen for other contaminants. It is reasonable to conclude that air concentrations within and very near Ground Zero would have been at least at these high levels and probably higher during the first several days after September 11. These areas were in restricted zones, which minimized overall exposures, and exposures were further minimized for individuals who used protective gear and clothing.

Occupational and Indoor Exposures: Extensive data sets are available from OSHA and NIOSH on occupational exposures on the Ground Zero site. Many of these samples were personal air monitors, and as such, are the most appropriate types of samples for evaluating inhalation exposures of workers. The contaminants evaluated in this report and many more are included in these data sets. The vast majority of samples in both data sets were below occupational standards including OSHA PELs and NIOSH RELs. The ATSDR has completed a study of residential apartments (NYCDOHMH/ATSDR, 2002). Testing occurred between November 4 and December 11, and included 57 apartments in lower Manhattan as well as 5 comparison apartments. In all tested apartments (lower Manhattan and comparison), airborne fibers were not detected above background levels in any of the indoor air samples. However, bulk dust samples showed asbestos in 16% of the apartments in Lower Manhattan, and none in the comparison apartments. Also, synthetic vitreous fibers (SVF or fibrous glass) were found in both indoor and outdoor samples in Lower Manhattan. Another study sampling indoor air and dust on September 18 in 2 locations very near Ground Zero found significantly high concentrations of asbestos in both air and dust, but low, background, concentrations of dioxin, PCBs and metals.

Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster

Section I. Overall Purpose and Scope of Assessment

The purpose of this document is to provide a preliminary assessment of the potential human health impacts associated with exposures to emissions caused by the September 11, 2001, collapse of the World Trade Center (WTC) towers. This assessment focuses on a disaster that has already occurred, and it became a challenge to evaluate the seriousness of health impacts that may have resulted (or may still result) from past exposures to contaminants. This situation presents problems that are different from those faced in analyses to support the proactive establishment of environmental standards, to determine emission limits from air sources, and in similar regulatory venues where risk assessment procedures are used. In such circumstances, risk managers can make an active choice regarding the level of protection that is to be achieved and how uncertainties will be weighed in that process. In addressing exposures resulting from the WTC attack, those options are not available. Accordingly, this report attempts to take a practical, integrative approach to evaluating and conveying the potential impacts and their seriousness from a public health perspective.

Six contaminants/contaminant classes were evaluated in this assessment. These included: particulate matter (PM), metals (lead, chromium, and nickel), polychlorinated biphenyls (PCBs), dioxins, volatile organic compounds (VOCs; benzene and several others), and asbestos. Although hundreds of different substances have been measured in various media, these substances were selected for evaluation because monitoring indicates that they correlate with the disaster site in both time and space, and because they pose a potential concern for health impacts. PM was generated by the collapse of the WTC buildings, the recovery and demolition operations, and the lingering fire. Lead and asbestos were believed to be components of the WTC building materials. PCBs were likely used as dielectric fluid in transformers and capacitors. Dioxin and VOCs are produced as a result of combustion and volatilization from fuels. A screening of all substances was not possible in the time available. Instead, a judgement was made that the above listed chemicals might pose the greatest health concerns. Other contaminants may be evaluated in later reports.

Potentially exposed populations could include anyone who lives or works in the vicinity of WTC, such as cleanup workers, office workers, merchants, or residents. Available data were not always sufficient to evaluate the potential impacts to all populations from all contaminants. For example, it was decided that monitoring data for VOCs at Ground Zero could not be used to evaluate VOC exposure to Ground Zero workers. The reasons for this are twofold. First, most of the EPA data for VOCs at Ground Zero came from simple grab samples taken within plumes and within rubble piles. The principal purpose for this sampling strategy was to understand the source emissions of VOCs from the WTC rubble and fires, and to alert the Fire Department of New York (FDNY) and the contractors/union health/safety officers working at Ground Zero about conditions that posed immediate health concern to the workers. As such, these data were deemed not appropriate for evaluating human exposure and potential health impacts. Second, analyses of exposure of Ground Zero workers to VOCs were conducted by the Occupational

Safety and Health Administration (OSHA) and the National Institute for Occupation Safety and Health (NIOSH), who employed personal air monitors for their analysis. These monitors are much more appropriate for human exposure assessment. The OSHA data, as posted on their website (<http://www.osha.gov>), are summarized in Section VI. The NIOSH data are also summarized there, with a reference provided for further information (CDC, 2002).

On the other hand, an evaluation of Ground Zero worker exposure was conducted for dioxin-like compounds. Unlike the case with VOCs, the dioxin monitors were stationary high-volume monitors operating for 8-hour periods. High concentrations captured by these monitors in the few months after September 11 are representative of air quality to which unprotected workers were potentially exposed (i.e, those not wearing respirators). Exposures to asbestos at the Staten Island Landfill were also evaluated using ambient air data. Although workers were not specifically identified as the exposed population, conservative assumptions in an exposure and risk assessment suggest that even continuous exposure for the limited time period when asbestos at the Landfill may have been elevated would not have resulted in exposures that suggest a potential human health risk.

Exposures that were specific to the indoor environment were also not explicitly addressed in this assessment. Section VI describes completed and ongoing studies which have measured levels of contaminants, mostly asbestos, in indoor environments. A major study completed by the Agency for Toxic Substances and Disease Registry (ATSDR) is summarized in that section. Also, EPA Region 2 is currently conducting indoor measurements during the clean-up of residences in lower Manhattan. Clearly, this will reduce future indoor exposures. It is expected that this EPA effort, and future compilations of all available indoor data, will allow for a more complete evaluation of impacts on residents and office workers to contaminants that were present as a result of the collapse of the WTC Towers.

This assessment focuses on the inhalation pathway. Exposure can potentially occur via inhalation, dust ingestion and dermal contact with contaminated dust on surfaces. Dermal contact and dust ingestion were not assessed due to a lack of appropriate data and reliable methods. For residents, contacts with contaminated dust will occur mostly indoors where people spend the majority of their time. The health assessment conducted in this study assumed that ambient air measurements were representative of long- and short-term exposures. In some cases, this could be misleading or inappropriate, particularly if indoor concentrations are higher than outdoor concentrations. It is emphasized that the evaluations in this document focus on ambient, outdoor measurements.

Because of the difficulties in setting up an effective monitoring program in such circumstances, very little data for most chemicals were collected prior to September 18, 2001. As a result, exposures occurring on September 11 and during the week after are poorly characterized. It can reasonably be assumed that the concentrations within and near Ground Zero would have been at least as high and more likely higher than the first measurements taken near September 18. Section V reviews this hypothesis and demonstrates that, for many critical contaminants, the highest concentrations were the first ones taken. The individual contaminant assessments address this lack of data in different ways. For dioxin, it was assumed that the first concentrations measured on September 23 were representative of the period between September

11 and that first measurement. For some chemical classes, like VOCs, an exposure and health evaluation for this time period cannot be conducted because the data do not exist. This could be critical for benzene, for example, which could have been present at very high levels near September 11 due to volatilization from gasoline and aviation fuel. EPA-ORD researchers are working to use modeling and available data and meteorological information to reconstruct probable exposures for some contaminants such as PM. Some preliminary results of plume dispersion are included in this report. When this and other work on immediate post-September 11 issues has been completed, further health evaluations of this critical window may need to be conducted.

Similarly, data regarding contaminant levels prior to September 11, or levels that might be considered background and typical for New York City (NYC), do not exist for all compounds because such measurements have not been performed routinely in all urban areas. Where information specific to NYC is available, such data are discussed. Otherwise, general urban or general background concentrations are identified and used to place the post September 11 monitoring results in perspective.

This assessment evaluates the potential for health impacts from the exposures that have occurred from September through about April of 2002. Most monitoring was discontinued in July of 2002, but data only through April was available at the time this evaluation was conducted. The air concentration data shows that, by April 2002, initially high levels decreased to background levels, with most reaching background by December 2001. However, the data collected between April and July, 2002, will be examined in the coming year to see if there were any elevations which could change the findings presented in this report.

As more data become available, further assessments may be conducted in order to evaluate more chemicals, more pathways, the indoor environment, the period between April and July, 2002, and other identified data gaps. Local and Regional needs, peer review and public comments on this assessment, and professional judgment will be used to determine the direction of future assessments.

Section II. Exposure Assessment and Risk Characterization Approach

Exposure assessments and risk characterizations for all contaminants focus on the inhalation pathway. For most chemicals, potential health risks are evaluated by comparing the measured air levels at locations near Ground Zero to established benchmarks for inhalation exposure and to typical urban background levels. Occupational Safety and Health Administration (OSHA) Permissible Exposure Limits (PELs), National Institute for Occupational Safety and Health (NIOSH) Recommended Exposure Levels (RELs), and Agency for Toxic Substances and Disease Registry (ATSDR) Minimal Risk Levels (MRLs) are among the benchmarks included in this evaluation. Where available, benchmarks established to protect against acute and subchronic exposures are used. Benchmarks that are intended to protect against exposures lasting more than one year or throughout a lifetime, such as EPA's Reference Concentrations (RfCs), were only used if other more appropriate benchmark values were not available. Table 1 provides a summary of the inhalation benchmarks used in this analysis.

EPA's Region 2, in consultation with federal health agencies, also used benchmark values to compare with measured air concentrations. These are described and listed in Appendix A, and are also cited on EPA's WTC web site: <http://www.epa.gov/wtc>. Region 2 used existing standards where appropriate, and for other contaminants, developed unique standards specifically for the purpose of evaluating the air measurement data from the WTC site. Some of the existing standards which they used included occupational standards such as OSHA PELs, which were used for all site workers conducting response/demolition activities covered by OSHA, and environmental standards such as National Ambient Air Quality Standards (NAAQS: e.g., for lead) and the Asbestos Hazard Emergency Response Act (AHERA) level of concern for asbestos to evaluate monitoring data from the site perimeter and beyond where residents or non-WTC site workers may have been exposed. In cases where appropriate standards did not exist, the Region developed risk-based screening criteria. The risk assessment paradigm detailed in EPA's "Hazard Evaluation Handbook: A Guide to Removal Actions" (HEH; EPA, 1997b) was employed in the development of these risk-based criteria. Screening levels that the Region developed reflect the most current toxicity criteria (Slope Factors and RfCs) on EPA's IRIS database (<http://www.epa.gov/iris>). The Region developed benchmark values for cancer and non-cancer effects. Details of the Region's derivation procedure are provided in Appendix A.

Some of the benchmarks used by the Region were also used as benchmarks in this report, such as the AHERA standard, while others were not. The unique benchmarks developed by Region 2 for evaluating WTC air measurements were not used in this report. The Region needed to develop and utilize those benchmarks in order to provide daily data reports that were readily understandable. Only existing benchmarks were used in this report, and in some cases, cancer or non-cancer screening exercises that went beyond a simple comparison to a benchmark were employed (e.g., dioxin-like compounds, see discussion below). Efforts are underway within EPA to develop benchmarks and similar standards to evaluate the impacts from a short-term inhalation exposure such as that experienced by some individuals at the WTC site. One such effort entails the development of Acute Exposure Guideline Levels. Final AEGLs are available for some contaminants, such as vinyl chloride, methylene chloride, methyltrichlorosilane, and others, but finalized (or draft) AEGLs are not currently available for the contaminants evaluated in this report.

A simple comparison of an air measurement and a health benchmark can be thought of as a “screening” exercise; the risk assessor is screening for possible problems. If the majority of samples are much less than a benchmark, then in most cases it would be appropriate to conclude that a health impact is unlikely. On the other hand, if most samples exceed the benchmark, then it may be appropriate to consider the possibility that a health impact may have occurred, or could occur, depending on the circumstances.

For dioxin toxic equivalent (TEQ) exposures, the air monitoring data are additionally used to conduct an assessment on cancer and noncancer risk. This involved defining the exposure scenario in greater detail, quantifying exposure within these scenarios for purposes of cancer risk estimation, and modeling the change in body burden over the exposure period for non-cancer assessment. Simple cancer screening exercises are also conducted for PCBs and asbestos. Insufficient information about the other chemicals precluded similar modeling. Where the data allowed, the best possible screening approach appropriate to each chemical class is used to evaluate potential health consequence from measured air concentrations.

In order to characterize exposure and risks, it is necessary to characterize the duration of exposure. Immediately following the collapse of the WTC towers, the NYC Mayor’s Office of Emergency Management restricted access to the WTC and surrounding sites. From September 11 through 14, this restricted zone included lower Manhattan south of 14th Street. Figure 1 shows the zones restricted after September 14. The areas in Figure 1 that are identified by a date show when those areas became accessible to the general public. Further details and additional maps can be found at, http://www.nyc.gov/html/oem/html/other/restricted_zones/frozen_zone_history_pdf_page.html. When a zone was restricted, all pedestrian and vehicular traffic was limited to emergency management and rescue personnel and other credentialed people. Residents were not allowed to occupy homes located in the restricted zones. Although some people in certain areas might have come and gone quickly (for example, to collect pets), no one was living or spending a significant amount of time in these areas unless they were part of the rescue, recovery and cleanup operations. As of mid-May, 2002, there were 15 residential buildings in the restricted zone.

As described below, the collapse of the WTC towers resulted in exceedences of screening benchmarks. However, most of these exceedences were in restricted zones and persons who live and routinely work in these areas were unlikely to have been exposed. Regardless of the level of contamination that may be present, if there is no exposure there is no human health risk. In discussions below, the timing and location of all exceedences are identified, and it is noted whether the location was in a restricted zone at the time of exceedence. These restricted zones also influenced the development of “exposure scenarios” that were used in some of the contaminant-specific evaluations.

Table 1. Inhalation health risk screening benchmarks used in this assessment.

Agency	Screening Benchmark
I. Short-Term Exposures	
OSHA	Permissible Exposure Limit (PEL) The maximum allowable exposure to a concentration of a substance in the air. PELs are set to protect workers and are based upon an 8-hr time-weighted average exposure. PELs are enforceable standards.
OSHA	Short Term Exposure Limit (STEL) A 15-minute time-weighted average that should not be exceeded at any time during a workday. STELs are enforceable standards.
ATSDR	Acute inhalation Minimal Risk Level (MRL) An acute MRL protects against exposures that may last 1 - 14 days. An MRL is defined as an amount of chemical that gets into the body (i.e., dose) below which health effects are not expected.
ATSDR	Intermediate inhalation Minimal Risk Level (MRL) An intermediate MRL protects against exposures that may last 15 - 364 days.
EPA-STSC	Provisional Subchronic Reference Concentration (RfC) The STSC subchronic RfC is the exposure level that is likely to protect humans from adverse health effects when exposed over a period not exceeding 10% of their lifetime (usually assumed to be 7 years). These values are only provisional guidance.
ACGIH	Threshold limit value (TLV) A recommended exposure limit based on an 8-hour workday and a 40-hour work week.
NIOSH	Recommended Exposure Level (REL) The maximum recommended exposure limit determined to protect workers.
EPA	Asbestos Hazard Emergency Response Act (AHERA) level of concern This standard was developed to be applied to asbestos in schools. School children were not to be allowed back into an "abatement" area (an area where activities were undertaken to reduce air concentrations) until several consecutive readings were less than the AHERA standard.

Table 1 (cont'd).

Agency	Screening Benchmark	
EPA	Air Quality Index (AQI)	The AQI provides reference points by which (a) to judge increasing levels of concern for potential health effects associated with acute exposures to air pollutants (e.g., particulate matter [PM]) for which short-term National Ambient Air Quality Standards (NAAQS) have been set and (b) to help guide the public and local government officials on actions to minimize unhealthy exposures.
EPA	National Ambient Air Quality Standard (NAAQS)	National air standards set by EPA (under the Clean Air Act) to protect against effects on public health and welfare of major urban air pollutants, e.g., PM. Short-term (24-hr) NAAQS for PM are more relevant here than are long-term (annual average) PM NAAQS.
II. Long-Term Exposures		
EPA	Reference Concentration (RFC)	An estimate of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of harmful effects during a lifetime.
EPA	Cancer Slope Factor (SF)	An upper-bound 95% confidence limit on the increased cancer risk from a lifetime exposure to an agent. The SF is used in conjunction with a dose term, such as the amount of a chemical inhaled, to conservatively estimate the potential for incurring cancer within a lifetime as a result of that exposure.
EPA	Unit Risk (UR)	The UR factor is used to estimate the upper-bound 95% confidence limit on the increased cancer risk from a lifetime of inhalation to a contaminant. It is derived starting with the SF for a contaminant and then assuming a lifetime of exposure (24 hr/day, 70 yrs). When using the UR, a concentration corresponding to a lifetime average concentration should be used.
EPA	Maximum Contaminant Level (MCL)	The MCL is the highest level of a contaminant that is allowed in drinking water. It is an enforceable standard that is set considering health effects and assuming the best available treatment technology, while taking cost into consideration.

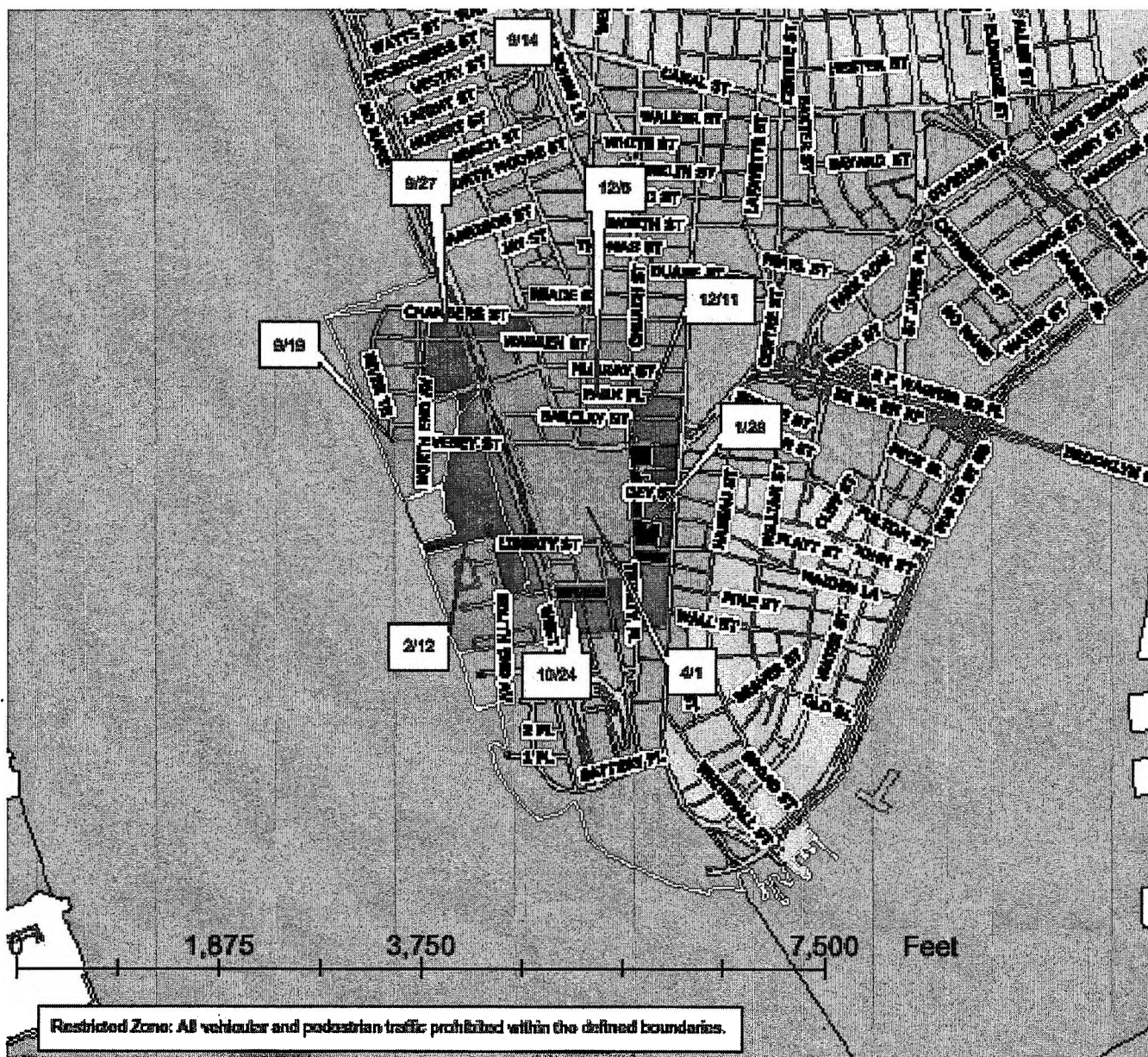


Figure 1. Figure showing the shrinkages of the restricted zones in the vicinity of Ground Zero over time. For example, the area below Canal Street was no longer prohibited after 9/14, and similarly, the shaded area beneath Chambers Street became available only after 9/27. [Figure extracted from map supplied by City of New York, Emergency Mapping Center]

Section III. Environmental Monitoring

Within a month after the disaster, EPA's Office of Environmental Information (OEI) began compiling the monitoring information generated by various agencies and providing that information directly to the public on the EPA web site: <http://www.epa.gov/wtc>. Environmental monitoring data were made available on a daily basis. EPA also developed three "trend reports," dated November 20, 2001; January 24, 2002; and May 1, 2002. Most of these data were made available to the EPA's National Center for Environmental Assessment (NCEA) in an electronic form for purposes of this report. These three principal sources - the web site, the trend reports, and the electronic data base - represent compilations/interpretations of much of the same data.

The sources of data that have been relied upon in the development of this report are further described below.

- The "EPA WTC monitoring database" In the aftermath of the WTC disaster, many organizations and agencies conducted sampling and monitoring activities to assess environmental impacts. The New York City Department of Health and Mental Hygiene (NYCDOHMH) initially requested that these monitoring organizations forward their results to them so they could be aggregated and made available for internal use by federal, state and local decision-makers. On September 25, 2001, NYC asked EPA to assist in the management of these data by developing a database capable of tracking and reporting on the environmental data. The Agency delivered on September 28, 2001, the EPA WTC Multi-Agency Database that houses data from thirteen federal, state and private organizations (including EPA, the New York State Department of Environmental Conservation (NYSDEC), and the New York City Department of Environmental Protection (NYCDEP) who conducted environmental monitoring after the September 11 disaster. Roughly 95% of all data in the database are from either EPA or the NYSDEC. This database has been maintained by EPA's Office of Environmental Information and provided a clearinghouse and comprehensive site for use by all government agencies responding to the disaster. The database was provided to the authors of this report in May of 2002, and at the time, was current as of mid-April, 2002.
- Environmental Data Trend Report World Trade Center Disaster (EPA, 2002a). These reports have been developed by IT Corporation under contract to EPA's Office of Solid Waste and Emergency Response. IT Corporation has completed reports dated November 20, 2001; January 24, 2002; and May 1, 2002. This latest trends report is current to April 24, 2002. These trend reports focus solely on monitoring data and are summaries primarily of the data included in the EPA WTC monitoring database.
- EPA web site. Most of the data in the EPA WTC monitoring database have been summarized for the general public on the EPA web site dedicated to information on the WTC site (<http://www.epa.gov/wtc>).

- Historical concentrations of metals, VOCs, PM, dioxin, PCBs, carbon monoxide (CO), nitrogen dioxide (NO₂), and sulfur dioxide (SO₂) for the NYC area provided by EPA Region 2 and found otherwise in the open literature.
- Air monitoring data for VOCs and PM that were not part of the EPA WTC monitoring data base were provided by the ORD National Exposure Research Laboratory (NERL) staff.
- Reports and data developed by non-EPA Agencies involved in sampling at the WTC site, who used the data for their individual purposes. These data sets are described when appropriate and include, for example, the Occupational Safety and Health Administration (OSHA) Sampling Results Summary posted on their web site (<http://www.osha.gov>) on 8/15/02, the National Institute for Occupational Safety and Health (NIOSH) data base (CDC, 2002), and the Agency for Toxic Substances and Disease Registry (ATSDR) study of indoor residences (NYCDOHMH/ATSDR. 2002).

Appendix B shows a summary of the monitoring stations that provided air monitoring data to the EPA WTC monitoring database that is used in this assessment. Prior to the terrorist attack on September 11, air monitoring stations for PM already existed at a number of locations in NYC, including lower Manhattan, upper Manhattan, Brooklyn/Queens, the Bronx, and Staten Island, as well as New Jersey. Following the disaster, EPA's Environmental Response Team set up monitoring sites at various locations near the WTC. The strategy employed in positioning the various "alphabet" monitoring sites was to place the monitors at varying distances surrounding, but near, Ground Zero. These alphabet monitoring sites are listed first in Appendix B. Pollutants that have been measured at these locations include polycyclic aromatic hydrocarbons (PAHs), dioxins, asbestos, PCBs, metals, silica, and PM. The second major set of monitoring stations listed in Appendix B were established by NYSDEC. These sites have been designated by numbers to contrast with the alphabet sites. Generally, these sites were further from Ground Zero. Pollutants monitored at these sites included particulates, VOCs, dioxins, asbestos, aldehydes, and particle size fractions. In addition, EPA's National Exposure Research Laboratory (NERL) set up monitoring at three of the alphabet sites (A, C, and K) and at site 16 where monitoring generally for particulates (and metals), VOCs and particle size fractions was conducted. Finally, the New Jersey Department of Environmental Protection assisted in the monitoring of asbestos at nearby locations in New Jersey.

Laboratories contracted by EPA have conducted most of the pollutant analyses, but EPA laboratories have done some of the analyses, including some of the dioxin and particulate analyses. The EPA laboratories that have participated in this effort include NERL's Human Exposure and Atmospheric Sciences Division (HEAS) in Research Triangle Park, NC, and the Region 7 Environmental Services Division in Kansas City, KS.

Information on the monitoring procedures and the data includes several other details, such as dates of monitoring, different monitoring and analytical procedures, and other issues that are specific to the pollutants. These pollutant-specific issues are discussed in each of the pollutant sections. Also, each section includes a map showing only those sites where monitoring

was conducted for the specific pollutant.

Meteorological stations were also established following the attack. Six stations were set up by the U.S. Department of Interior, and one station was set up by EPA. These meteorological data, in conjunction with monitoring data, are being used by EPA researchers to model and recreate the potential exposures that might have occurred immediately after the collapse of the WTC buildings. Some important preliminary results of the modeling efforts are presented in this report. The overall modeling analysis, when complete, will be presented in future EPA reports and assessments.

In addition to air samples, bulk dust, water, river sediments, and drinking water samples were collected from sites associated with the WTC and surrounding areas to determine the degree to which the disaster may have caused contamination of these media. Sampling and analyses were conducted by various agencies, including but not limited to, EPA Region 2, the United States Geological Survey (USGS), NYSDEC, and the New York State Department of Health (NYSDOH). Between September 11, 2001, and January 14, 2002, more than 150,000 sampling results were reported for the WTC and other New York and New Jersey sites. Results were reported for over 500 substances (statistics from the second of three trend reports; the third is cited as EPA, 2002a).

Although the data generated since September 11 included results for air, bulk dust, water, river sediments, and drinking water, the focus of this evaluation is on the air sampling, with some discussion of the bulk dust as it relates to asbestos and other contaminants. It should be noted that none of the drinking water samples were found to have concentrations that exceeded any of EPA's Maximum Contaminant Levels (MCLs) for drinking water.

Section IV. Evaluation

A uniform approach to characterizing the impacts of all pollutants is not possible because the available background data and screening benchmarks vary between compounds. Similarly, the relevance of these data for the circumstances of exposure following the WTC disaster also varies with the type of contaminant being evaluated. When benchmarks are available for the circumstances at hand they provide a quantifiable approach for assessing the impact of human exposure. Such benchmarks are used and described, as appropriate.

Background or pre-existing levels of environmental contaminants provide an important reference point for describing the environmental impact of the destruction of the WTC. If the monitored levels for a pollutant after the disaster did not appreciably exceed values commonly found in New York or other urban settings, concerns about elevated health risks will be reduced. On the other hand, when contaminant levels are in excess of background, a careful evaluation of the potential health impacts is warranted. Exceedence of background levels does not itself necessarily imply that potential health risks exist. Also, for some pollutants, background measurements might at times show substantial levels and have associated health risks. Thus, comparisons with background levels need to be interpreted in light of the health risk data for the pollutants.

As discussed below, air concentrations and subsequent exposures after September 11 were generally elevated for each pollutant over a limited span of time. Measured concentrations for most air pollutants were reduced by the beginning of 2002. Many of the environmental health criteria have been developed to address “chronic” exposures to contaminants that are present in the environment for years or decades. Exposure levels that exceed a chronic health reference level for a limited period would generally be less likely to result in adverse health effects. Where available, this report has tried to use acute and sub-chronic screening standards. When chronic screening standards are used, the relevance of the comparison is discussed.

In this report, reference is made to OSHA health criteria for the protection of worker’s health. These criteria are valuable for the analysis of WTC data for several reasons. Criteria exist for many compounds, not all of which have established environmental health criteria, and they address risks due to less than lifetime exposures. However, there are limitations in applying occupational criteria to evaluations of environmental health risks. Occupational criteria serve to protect relatively healthy worker populations that would typically be less diverse than the general population (in terms of age and health status). Occupational criteria also apply for workday periods, with exposures ceasing during nonworking hours; environmental exposures are typically more continuous. Finally, risk levels that have been accepted in occupational settings may exceed those accepted for long-term environmental exposures to the general population. For these reasons, EPA has not generally used occupational standards as a basis for environmental health criteria.

It is important to reiterate that very limited data are available on the levels of exposure that occurred to individuals due to direct contact with the plume of smoke and dust generated by the WTC collapse on September 11. Such exposures would have been of limited duration, but levels of contaminants may have been significantly higher than those measured in monitoring

programs that have tracked exposures in New York since that time. This is an issue that warrants further examination. Also, the ambient monitoring did not begin immediately on September 12. The earliest reported data are from asbestos monitoring, which began on September 14. The dioxin monitoring did not begin until September 23. It is highly likely that air concentrations within and near Ground Zero were highest during these first several days, but monitoring is unavailable to confirm that hypothesis. This issue is addressed in more detail in Section V, which observes that the highest concentrations were among the first ones measured. Many monitoring programs and studies are currently under way to help us understand health outcomes that have resulted from acute exposures on September 11 and the next several days. These studies are discussed in the final section of this report.

This report should be viewed as the first phase of an ongoing analysis, and the conclusions and findings cited below should not be considered the final EPA judgement. At this point, the available data and analyses are still too preliminary to support reliable quantitative predictions of potential human health risks. Although a complete quantitative evaluation of the health impacts of the disaster may never be possible, future EPA analyses will attempt to develop more quantitative estimates of risks and health impacts using additional exposure-related data and possibly, the results of epidemiological studies that are currently underway. An overview of epidemiological studies that are underway is provided in Section VII.

Following are assessments on the individual pollutants.

IV.a. Particulate Matter

Airborne particulate matter (PM) is a complex mixture of inorganic and organic substances transported in air as solid particles or liquid droplets. PM in ambient (outdoor) air can typically be divided by size into two groups: fine particles (less than $\sim 2.5 \mu\text{m}$ diameter) and larger, or coarse, particles (ranging from $\sim 2.5 \mu\text{m}$ to $50 \mu\text{m}$ or more). Fine PM includes primary particles formed by combustion (including condensed metal and organic vapors), as well as secondary aerosols (formed by gas-to-particle conversions). Coarse PM consists mainly of earth crustal materials formed by natural erosion processes or by human activities such as driving on paved or unpaved roads, agriculture and mining operations, industry, and construction/demolition.

PM exposures and associated potential human health risks are addressed in this preliminary analysis because the collapse of WTC buildings resulted in vast quantities of structural materials and building contents being crushed and pulverized into airborne particles in a wide range of sizes, including both fine and coarse particles. These particles, along with particles and gases emitted from burning jet fuel, aircraft parts, and building debris, formed the immense dust/smoke cloud that rapidly spread across the NYC area and dispersed over hundreds of square miles. Thus, PM air monitoring was essential to help characterize potential health effects resulting from human exposure to the initial dust/smoke cloud, particles produced by the ensuing fires, and reentrained particles stirred up into the air during recovery activities and transport of debris away from the WTC site.

Airborne PM exposures are of concern for human health because they can be associated with a wide variety of adverse human health effects. Some health impacts could include

respiratory effects (such as lung inflammation and exacerbation of asthma) and cardiovascular effects (including exacerbation of preexisting chronic heart disease). As with exposures to other environmental contaminants, potential health impacts depend on PM concentrations and duration of exposure, as well as the size of the particles inhaled and many other factors (including the age and health status of exposed individuals). Depending on age and health status, some groups (such as infants and children, the elderly, and/or individuals with preexisting cardiovascular or respiratory diseases) may be considered sensitive or susceptible populations to possible effects of PM exposure.

Particulate matter is one of six common, widespread air pollutants (the others are ozone, carbon monoxide, nitrogen oxides, sulfur dioxide, and lead) for which EPA has set National Ambient Air Quality Standards (NAAQS). A NAAQS is an air quality standard, set under the Clean Air Act, that is designed to protect public health. In 1987, EPA set PM₁₀ NAAQS (150 µg/m³, 24-hour average, and 50 µg/m³, annual average, averaged over 3 years) to protect against health risks associated with inhalable particles (mainly those < 10 µm diameter) that can deposit in lower (thoracic) portions of the human respiratory tract. These health-related PM₁₀ particles include both fine particles < 2.5 µm diameter (PM_{2.5}) and a subset of coarse particles larger than 2.5 µm but less than < 10 µm diameter (PM_{10-2.5}). After reviewing the scientific bases for PM NAAQS in 1996, EPA concluded that fine and coarse components of PM₁₀ particles should be treated as separate classes of pollutants. Thus, EPA moved in 1997 to set an annual PM_{2.5} NAAQS (15 µg/m³, annual average, averaged over 3 years) to protect against both short- and long-term exposures and a supplemental 24-hour average PM_{2.5} NAAQS (set at 65 µg/m³) to protect against unusually high peak levels to decrease health risks associated with fine particle exposures. The PM₁₀ NAAQS were retained to address risks related to coarse particles. As more scientific evidence becomes available, consideration may be given to setting PM standards for shorter averaging periods (< 24 hr).

Also, to provide real-time, day-to-day information to State and local health officials and the public, EPA established an Air Quality Index (AQI) level of concern (LOC) for daily PM_{2.5} ambient concentrations at 40 µg/m³. The AQI is meant to provide reference points for judging levels of potential health concern and to guide actions by citizens or government officials to protect the health of the public, including susceptible groups. Thus, in order to minimize risk of potential health effects among highly susceptible individuals (e.g., the elderly over 65 yr old or individuals with preexisting chronic cardiovascular or respiratory disease), actions should be taken to reduce or avoid exposures of such persons to 24-hour PM_{2.5} concentrations above 40 µg/m³.

IV.a.1. Air Quality Monitoring of Ambient Particulate Matter Mass/Composition

At the time of the September 11, 2001, WTC attack, there were no monitoring sites measuring ambient air PM_{2.5} or PM₁₀ concentrations in the immediate vicinity of the WTC or surrounding neighborhoods in lower Manhattan, except for PM instruments operating at the Canal Street Post Office about a half mile north of the WTC. However, there were numerous New York State-operated PM sampling sites monitoring ambient air PM concentrations at various other locations throughout the five boroughs of New York City (NYC), with most measuring PM_{2.5} levels by Tapered Element Oscillating Microbalance (TEOM) monitors.

During the days following the September 11 events, substantial efforts were made to quickly augment existing PM monitoring capabilities by the addition of PM sampling sites immediately around the WTC Ground Zero work zone and in surrounding lower Manhattan neighborhoods. Of particular note, staff from EPA's National Exposure Research Laboratory (NERL) worked with EPA Region 2 colleagues to set up PM samplers at three surface sites triangulating the WTC Ground Zero work zone perimeter, as shown in Figure 2 (lettered sites A, C, and K). Site A was located at Barclay and W. Broadway just north of Ground Zero, Site C at Liberty and Trinity to the southeast of Ground Zero, and Site K at Albany and West to the southwest of Ground Zero. In addition to these surface sites, ORD PM sampling was initiated at the 16th floor level in EPA Region 2 facilities in the Federal Building at 290 Broadway about six or seven blocks northeast of the WTC. These sites are noted in Table 1 of Appendix B, as EPA Response Team Lettered Sites at Locations A, C, and K and Site #16 under EPA/ORD Numbered Air Monitoring Stations. By October 2, the U.S. EPA Region 2 and EPA OAQPS, along with New York State Department of Environmental Conservation (NYSDEC) personnel, set up PM instruments at Chambers Street, Park Row, and the Coast Guard Building at the Battery, which along with the existing monitors at Canal St., surrounded Ground Zero a few blocks farther out than the ORD sites.

Measurements of ambient air PM_{2.5} elemental composition were made by ORD staff (Vette et al., 2002) at the three ORD surface sites (A, C, and K), using saturation samplers (SS), and on the 16th floor of the 290 Broadway Federal Building, using a modified dichotomous versatile air pollutant sampler (VAPS). The VAPS also collected PM_{10-2.5} samples. Only the battery-powered saturation samplers could be operated within the WTC perimeter because power outages precluded operation of conventional samplers, such as EPA Federal Reference Method (FRM) samplers in use at preexisting NY State-operated PM monitoring sites. Although the SS has been shown (Hill, et al., 1999; also see <http://www.airmetrics.com/products/studies/1.html>) to achieve accuracy within 6-10% compared to PM_{2.5} FRM measurements at concentrations typically found in ambient air, uncertainty in measured PM_{2.5} concentrations increase as ambient concentrations increase, especially above 100 µg/m³ (24-h average). Indeed, even the FRM is subject to increasing uncertainties at high PM_{2.5} levels. However, the general characterization of air quality used for EPA's Air Quality Index (AQI) should be largely unaffected. The use of these samplers (SS) allowed for x-ray fluorescent (XRF) analyses of the elemental composition of aerosol samplers. Such XRF analyses were performed on filters collected from September 21, 2001, to January 31, 2002. These samples were collected on an almost daily basis for sampling periods of about 22 hours per day. In addition to the VAPS sampler, high time-resolution measurements of light scattering by particles (which is related approximately to the concentration of fine PM) and light extinction (which is related to the concentrations of black carbon and to complex organic compounds such as PAHs) were also made. The main focus of discussion here with regard to EPA/ORD results is on findings from filter-derived PM mass and elemental composition measurements.

The ORD PM monitoring sites triangulating the WTC Ground Zero perimeter were placed so as to allow: (a) continuous tracking of PM_{2.5} emissions from Ground Zero, useful for detecting any extraordinarily high-level PM_{2.5} exposures to rescue/recovery workers and others operating within or immediately around Ground Zero; and (b) the determination of the physical/chemical composition of WTC-generated PM emissions from the smoldering fires or

recovery operations, which could serve as a “WTC signature” for tracking the movement of the WTC plume and its potential impacts on ground-level air quality in the NYC area. Along with the addition of several more New York State-operated PM sampling sites in lower Manhattan and elsewhere as part of an extended air monitoring network set up by NYSDEC, the ORD 290 Broadway site helped to ensure reasonably good coverage (within feasibility constraints) of lower Manhattan neighborhoods surrounding the WTC Ground Zero work zone. In addition to the ORD monitoring sites (A, C, K, and 290 Broadway), Figure 2 shows the location of New York State-operated PM_{2.5} and PM₁₀ sampling sites.

This expanded monitoring coverage greatly enhanced federal/state government capabilities for tracking trajectories of WTC-generated plumes, areas potentially affected by the plumes, and possible WTC-related PM exposures in the NYC area. For example, notable PM increases at ORD Site A (on the WTC Ground Zero north perimeter), coupled with PM elevations at the NYSDEC site at Chambers St. and West St. about 5 blocks north of the WTC (labeled BMCC for Borough of Manhattan Community College in Figure 2) and/or at the Canal St. Post Office would be indicative of a northerly trajectory of the WTC plume and potential surface-level exposures of population groups in the vicinity of those monitoring sites and intermediate locations. Alternatively, marked increases PM levels at Site A, coupled with PM elevations at 290 Broadway and/or PS 64 (~ 2.5 km northeast of the WTC), would be consistent with a northeasterly WTC plume trajectory. Also, any increases in PM levels at Site C, coupled with elevations at Park Row and/or preexisting PM sites at PS 199, the Maspeth Library in Queens, or PS 274 in Brooklyn would be consistent with an easterly WTC plume trajectory; whereas PM increases at ORD Sites C or K, along with PM elevations at Battery Park, would indicate a southerly plume movement. PM increases at multiple ORD sites proximal to the WTC perimeter and in several neighborhoods in various directions from the WTC could also conceivably occur under meteorological conditions involving low wind speeds and thermal stability conditions, producing low mixing layer heights and a more well-defined WTC plume traversing across lower Manhattan or other NYC areas. On the other hand, if PM concentrations at other sites across lower Manhattan or other areas in NYC tend to vary with each other (i.e., go up and down together) and are similar in values, this would imply that they are responding more to urban or regional background sources rather than to emissions from the WTC sites.

To aid in assessing the potential effects of WTC-related air pollutants on air quality in NYC areas, ORD has also embarked on the modeling of the dispersal of WTC Ground Zero-generated plumes, based on prevailing meteorological conditions (e.g., wind speed and direction). This includes: (a) initially, classical Gaussian plume modeling, providing regional-scale hour-by-hour plots that roughly delineate the likely spread and direction of the WTC plume, and (b) more recent initiation of detailed local-scale computational fluid dynamic modeling which is expected to provide improved, detailed estimation of the dispersal of emissions from Ground Zero in the street canyons of lower Manhattan. Some preliminary results of the regional-scale plume modeling are discussed below and compared with PM monitoring results for certain days of particular interest. It is important to note that the preliminary results of the regional-scale modeling primarily allow the estimation of the likely direction and width of the WTC plume at particular times, but they do not alone enable one to conclude if or where the plume may have touched down and resulted in surface-level increases in pollutant levels. They alone also do not allow one to conclude what the PM concentrations would have been, only what

the estimated dilution would have been relative to WTC Ground Zero concentrations. During periods when emissions from Ground Zero were low, any increases in concentrations within the plume would also be low. Other inputs (e.g., evaluation of surface-level PM measurements at various sites and/or photos of the WTC plume) are also needed to aid in characterizing likely occurrences of plumes affecting the surface and/or possible human exposures.

In addition to the above expanded EPA and New York State government PM monitoring and modeling efforts, numerous other investigators (e.g., some from other Federal government agencies, academia, and commercial firms or sponsored by nongovernmental organizations) collected data aimed at in estimating likely PM exposures associated with the WTC attack and ensuing fires and recovery operations. Limited published reports from such studies are becoming available, and a few salient points from these reports are alluded to here. More thorough discussion of these and other reports that become available in the coming months will be included in any subsequent, fuller EPA/ORD evaluation.

One important set of newly available findings are those reported by Liroy et al. (2002), based on work done cooperatively with EPA/ORD and also partly funded by NIEHS. Direct air measurements of the composition of WTC-generated airborne particles by EPA/ORD did not begin until September 21, 2001. Before that date, only bulk samples of settled dust were available for chemical analyses. Liroy et al. (2002) measured the mass of particles in several size ranges in the settled dust and analyzed their composition. Also, detailed chemical analyses of fine fraction particles ($< 2.5 \mu\text{m}$ diameter) from settled WTC dust has recently been reported (McGee et al., 2002). Although the bulk samples do not provide direct data on ambient air PM concentrations, they do provide strong clues as to the likely composition of airborne PM in lower Manhattan and the size distribution of particles in the suspended dust immediately after the collapse of WTC buildings. Small amounts of WTC-derived settled dust were also provided by New York University investigators for laboratory toxicity testing by EPA scientists (EPA, 2002c; Gavett et al., 2002) in EPA/ORD's National Health and Environmental Effects Research Laboratory (NHEERL).

Also, in a cooperative U.S. Department of Energy (DOE)/University of California study, measurements of the size distribution of particles in 10 channels from 0.09 to 12 μm were made on the roof of the Federal Building on Varick Street, using the rotating drum impactor developed at the University of California, Davis (Cahill et al., 2002). This Varick Street DOE site is about 2.0 km north of the WTC (see Figure 2). Sampling began October 2, 2001 and continued into December 2001. Elemental analyses of samples collected approximately every 3 hours were done by synchrotron radiation-induced X-ray emission (SRIXE).

IV.a.2. Particulate Matter Air Monitoring/Modeling Results

1. Particulate Matter Mass Measurements and Plume Trajectory Plots

The collapse of the WTC buildings and the associated fires resulted in the initial dispersion of large quantities of various-size particles in the massive dust/smoke cloud that enveloped lower Manhattan. It is clear from Figures 3 and 4 (and other photographs) that the densest portion of the dust/smoke cloud initially spread in all directions and impacted most of lower Manhattan, especially below Chambers Street. On the basis of the size classifications (by

gravimetric and aerodynamic methods) of particles settled in dust collected September 16-17 at weather-protected sites just east of the WTC, Liou et al. (2002) reported the largest mass concentrations in the settled dust to be due mostly to particles > 53 µm diameter (~ 51-64% of total mass) and 10-53 µm (~ 35-45% of total mass), followed by lesser percentages for 2.5-10 µm (0.3-0.4% of total mass), and < 2.5 µm (~ 0.9-1.3% of total mass) particles. Given the tendency of large coarse particles to settle out of the atmosphere closer to their emission source(s) than smaller fine particles, it is likely that higher percentages of small coarse particles (> 2.5 but < 10 µm) and fine particles (< 2.5 µm) were more widely dispersed in the plume of dust and smoke that spread primarily to the southeast over Brooklyn and to the south over New York Harbor during the first 18 to 24 hours after the collapse of the WTC buildings on September 11.

Figure 5 shows the results of initial plume dispersion modeling for September 11. The predominant wind direction was to the south-southeast; and this direction continued well into the next day (September 12). As seen in Figure 5, the predominant direction of the modeled WTC plume flow is to the southeast, based on wind directions and speeds indicated by black arrows in this figure. This is consistent with photo images, such as the one shown in Figure 6.

Although no direct measurements of PM concentrations are available for nearby lower Manhattan areas during the collapse of the WTC, some rough estimates can nevertheless be made of what concentrations may have been reached. The dust cloud was optically dense, as can be seen from the airborne images. Under such conditions, sunlight does not reach the surface, and visibilities are greatly restricted. Conditions such as these have been encountered in dust storms and in the London smog episodes of 1952 and 1962 (Elsom, 1992). During such conditions, PM concentrations could have been several milligrams per cubic meter (mg/m³), i.e., thousands of micrograms per cubic meter (µg/m³).

Particles smaller than 2.5 micrometers (fine particles) limit visibility much more effectively than larger (coarse) particles and under conditions usually found in the eastern United States, ambient air concentrations of fine particles are typically higher than those of coarse particles. As a result, under these conditions, visibility reductions are caused mainly by fine particles. There are a number of simple formulas that relate visibility to the concentration of PM_{2.5}, such as one from Stevens, et al. (1984):

$$0.5 (\text{km} \cdot \text{mg}/\text{m}^3) = \text{Vis (km)} * C (\text{mg}/\text{m}^3) \quad (1)$$

where V is the visibility range (km) and C is the concentration of PM_{2.5} (mg/m³). During the collapse of the WTC towers, visibilities were reduced to less than 100 m (about 1 city block) on many streets. If we assume that visibility on streets in lower Manhattan affected by the dust cloud (see Figure 3) was controlled by fine particles, then application of the above formula indicates that PM_{2.5} concentrations could have been about 5 mg/m³ (5,000 µg/m³). However, the collapse of the World Trade Center towers mainly produced coarse particles (Liou et al., 2002), that, as mentioned above, are less effective than fine particles in controlling visibility. Thus, the values given above represent lower limits on the abundance of total PM. It should also be noted that the above estimate of visibility is based on the loss of contrast between light and dark objects. In many streets, sunlight was blocked and, hence, total PM concentrations could have

been much higher than the lower limit given above, perhaps even approaching concentrations of condensed water vapor that are observed in dense fogs (i.e., thousands of $\mu\text{g}/\text{m}^3$).

Thus, individuals engulfed in the initial dust/smoke cloud may have been exposed for several hours to concentrations of both fine and coarse inhalable particles anywhere in the range from milligrams per cubic meter ($> 1,000 \mu\text{g}/\text{m}^3$) to perhaps hundreds of milligrams per cubic meter ($> 100,000 \mu\text{g}/\text{m}^3$). However, it does not appear that people outside the lower Manhattan area (except possibly very briefly for those on Governor's Island or in Brooklyn Heights) experienced such extreme PM exposures. This estimation is based, for example, on hourly $\text{PM}_{2.5}$ levels observed at several NYSDEC monitoring sites (Figure 7) during September 11 to 13. Of most note, as seen in Figure 7, $\text{PM}_{2.5}$ concentrations at NYC sites generally remained under $25 \mu\text{g}/\text{m}^3$ during most of September 11 and 12. However, hourly $\text{PM}_{2.5}$ concentrations did increase to the $50\text{--}100 \mu\text{g}/\text{m}^3$ range for a few hours on September 12 and 13 at PS 64, which is located about 2.5 km northeast of Ground Zero, and at PS 199, which is located in Queens several miles east-northeast of Ground Zero. These $\text{PM}_{2.5}$ increases most likely reflected east-northeast dispersal of not only windblown fine PM from settled dust but also probably of newly formed fine PM generated by the intense fires ($> 1000^\circ\text{F}$) at WTC Ground Zero. This would be consistent with dispersal to the east-northeast of the WTC plume, as indicated by the ORD modeled trajectory plotted for September 13 (shown in Figure 8). The increased hourly $\text{PM}_{2.5}$ levels at PS 64 and PS 199 (reaching 166 and $100 \mu\text{g}/\text{m}^3$ at 9 a.m. September 13) indicate that the WTC plume likely briefly fumigated the surface for a few hours at those and/or intervening locations during the morning of September 13.

Changes in wind direction later in the day on September 13 resulted in rotation of the WTC plume back to a flow predominantly to the south-southwest (mainly over New York Harbor) through September 14 and 15, as indicated in Figures 9 and 10. Note the very low $\text{PM}_{2.5}$ hourly values (almost all $< 6 \mu\text{g}/\text{m}^3$) at NYSDEC monitoring sites throughout the NYC area following rain associated with a frontal passage and, also, likely reflecting in part decreased vehicular traffic in the aftermath of September 11 events.

During the next several days, ORD plume dispersion modeling indicates that the plume rotated in such a manner as to result in transport in varying directions, including sometimes to the north-northwest (over northern New Jersey and NYC areas), but there was little indication of the plume fumigating the surface, based on surface PM measurements at preexisting PM monitoring sites. During the rest of September and on into October, 24-hour $\text{PM}_{2.5}$ values at preexisting NYSDEC monitoring sites did not exceed the daily $\text{PM}_{2.5}$ NAAQS ($65 \mu\text{g}/\text{m}^3$, 24-h). In fact, daily $\text{PM}_{2.5}$ values from most pre-existing fixed sites throughout the NYC area did not show marked elevations in comparison to historical $\text{PM}_{2.5}$ levels for NYC areas, with the occurrence through mid- to late October (from time to time or from site to site) of a few 24-hour $\text{PM}_{2.5}$ values approaching the 24-hour AQI LOC ($40 \mu\text{g}/\text{m}^3$) not being notably out of line with past frequency of such excursions in NYC.

Starting September 21, the EPA/ORD WTC perimeter monitoring sites at Sites A, C, and K (within 100-200 meters of Ground Zero) allowed tracking of WTC-related ambient PM emissions in the immediate WTC vicinity. The ORD monitoring data, shown in Figure 11 (top) showed widely varying PM concentrations across the WTC perimeter sites from day to day, with

high hourly or daily $PM_{2.5}$ levels being seen at one or another perimeter site downwind from Ground Zero on given days. As seen in Figure 11, exceedances of the 24-hour $PM_{2.5}$ NAAQS level ($65 \mu\text{g}/\text{m}^3$) occurred at some Ground Zero perimeter sites during late September and into October, but on only a few occasions thereafter. A general downward trend in daily PM concentrations, as well as a decreasing range of 24-hour variations, were seen for $PM_{2.5}$ concentrations at these WTC perimeter sites from early October, 2001, onward. The range of 24-hour values among these ORD sites generally remained below the AQI LOC of $40 \mu\text{g}/\text{m}^3$ during December, 2001, and January, 2002 (as depicted by black bar in lower right of Figure 11 top panel). In contrast to the results seen for the ORD/WTC perimeter sites, distinctly lower $PM_{2.5}$ concentrations were observed at the 290 Broadway site about six blocks northeast of Ground Zero. The 24-h $PM_{2.5}$ concentrations recorded there by VAPS sampling exceeded $65 \mu\text{g}/\text{m}^3$ (daily $PM_{2.5}$ NAAQS level) only once, on October 4; and the 24-h AQI LOC ($40 \mu\text{g}/\text{m}^3$) for highly susceptible persons was approached or exceeded at the 290 Broadway site on only a few occasions (e.g., Sept. 27; Oct. 3, 4, 5; Oct 20; Nov. 15-16). These daily values often reflect high hourly values occurring overnight mainly during early morning hours before 7 or 8 a.m. The overall pattern of results from ORD perimeter monitoring stations near the WTC, coupled with distinctly lower $PM_{2.5}$ concentrations monitored by ORD on the 16th floor at 290 Broadway (about 6 blocks northeast of the WTC) suggest occasional short-term increments in fine PM values at WTC Ground Zero and, at times, along the WTC fire plume path (with areas impacted shifting with prevailing winds).

For example, when low wind speeds and mixing layers associated with a high pressure system over New York City area occurred during October 3-5, generally increased region-wide $PM_{2.5}$ levels were observed across much of northern New Jersey and New York City, with some additional PM increments being superimposed at a few monitoring sites within modeled WTC plume dispersion areas. During such weather conditions, plumes tend to be more well-defined than if there were turbulence and are more identifiable for longer distances (see Stull 2000, for example). In particular, 24-hour $PM_{2.5}$ at WTC Site A (on north perimeter of Ground Zero) reached $400 \mu\text{g}/\text{m}^3$ on October 3-4, but 24-hour $PM_{2.5}$ values dropped off to $90 \mu\text{g}/\text{m}^3$ at the 290 Broadway site several blocks northeast of WTC and to $53 \mu\text{g}/\text{m}^3$ at PS 64 about 1.5 km further to the northeast of WTC Ground Zero and only reached $60 \mu\text{g}/\text{m}^3$ at Site K (on southwest perimeter of Ground Zero). This was consistent with prevailing winds (to the northeast) and the modeled plume dispersion depicted in Figure 12 for October 4.

Daily average $PM_{2.5}$ data obtained at additional sites in lower Manhattan (Chambers St., Park Row, and the U.S. Coast Guard Station at Battery Park) are shown in the lower half of Figure 11. These sites are located from 3 to 10 blocks to the north, east, and south from the WTC (see Figure 2). It can readily be seen in Figure 11 that concentrations of $PM_{2.5}$ were much lower than found at the WTC perimeter, indicating a very rapid decline with distance from Ground Zero. $PM_{2.5}$ concentrations at these three sites can also be seen to go up and down together. Correlation coefficients between pairs of these sites are all > 0.9 ; and the concentrations are all very similar, suggesting that these sites were responding mainly to variations in urban and regional background sources rather than to WTC emissions. None of the daily $PM_{2.5}$ values exceeded the $65 \mu\text{g}/\text{m}^3$ $PM_{2.5}$ 24-hr NAAQS. Only a few daily $PM_{2.5}$ values, as seen in Figure 11 and listed in the WTC Environmental Data Trend Report (EPA, 2002a) for the NYSDEC lower Manhattan sites to the north, east, and south of the WTC even approached

the 40 $\mu\text{g}/\text{m}^3$ AQI LOC value; many of the 24-hour $\text{PM}_{2.5}$ levels for such sites were below 20 $\mu\text{g}/\text{m}^3$. PM_{10} values (not graphically depicted here) observed at the same NYSDEC lower Manhattan locations around the WTC were also consistently below the daily $\text{PM}_{2.5}$ NAAQS (150 $\mu\text{g}/\text{m}^3$, 24-h) and showed a general decreasing trend from October 1 to November 30 and beyond, with 24-hour values decreasing from ~ 50 -90 $\mu\text{g}/\text{m}^3$ at some sites in early October to generally less than 50 $\mu\text{g}/\text{m}^3$ in November (except for ~ 114 -135 $\mu\text{g}/\text{m}^3$ on October 25-26 for Park Row and 55-75 $\mu\text{g}/\text{m}^3$ on November 15-16, 2001 for all the sites).

Overall, then, the $\text{PM}_{2.5}$ data appear to support the following conclusions:

- (1) Notable $\text{PM}_{2.5}$ elevations occurred in the immediate vicinity of the World Trade Center Ground Zero during late September/early October, with concentrations at ORD WTC perimeter sites on some days exceeding the 24-hr $\text{PM}_{2.5}$ NAAQS. However, PM concentrations at the WTC perimeter sites fell to typical background levels by late November/early December, 2001.
- (2) Such high $\text{PM}_{2.5}$ elevations were not observed at other lower Manhattan monitoring sites within 3 to 10 blocks of WTC Ground Zero. On only a few sporadic occasions did daily $\text{PM}_{2.5}$ concentrations approach or exceed the AQI LOC (40 $\mu\text{g}/\text{m}^3$) at one or two sites (e.g., 290 Broadway or PS 64) along the WTC plume path in addition to elevations seen at WTC perimeter sites. The frequency of such excursions were not out of line with historical frequency of $\text{PM}_{2.5}$ values approaching or exceeding 40 $\mu\text{g}/\text{m}^3$ either before the Sept. 11 WTC attack or since the WTC fires ended. See Figure 13, for example, where post September 11 $\text{PM}_{2.5}$ 24-h values for PS 64 are compared to historic levels seen at PS 64 during the previous two years.
- (3) No notable elevations in $\text{PM}_{2.5}$ concentrations were seen at NYSDEC lower Manhattan sites located 3-10 blocks to the north, east, and south from the WTC, with no $\text{PM}_{2.5}$ values exceeding either the $\text{PM}_{2.5}$ daily NAAQS or the AQI LOC from the start-up of PM monitoring on October 1 onward.

2. Measurements of Particle Composition

Analyses of bulk samples of dust produced by the collapse of the WTC towers were performed by EOHSI at Rutgers University (Liroy et al., 2002). Two bulk dust samples were collected on September 16 and another on September 17. The samples were collected at weather-protected sites located less than 1 km to the east of the WTC. The particle samples were separated according to size by aerodynamic and gravimetric methods. As noted earlier, results of the aerodynamically separated samples indicate that only a very small fraction (about 1%) of PM was in the $\text{PM}_{2.5}$ size range and less than 0.5% was in the $\text{PM}_{10-2.5}$ size range. The overwhelming fraction of the mass of PM was found in settled dust particles larger than 10 μm . Most of the mass consisted of pulverized building and construction materials such as cement and glass fibers and office building materials such as cellulose. High concentrations of inorganic constituents such as silica, calcium, and sulfate components of building material and metals such as lead and zinc, were found. Also, total polycyclic aromatic hydrocarbons (PAHs), which are products of incomplete combustion, constituted more than 0.1% of the total mass of dust. The fraction of adsorbed PAHs in each size fraction is expected to be roughly related to the relative surface area in each size fraction. According to this criterion, smaller particles would have contained proportionately greater concentrations of PAHs than indicated by their relative

mass.

Liroy et al. (2002) provides complete descriptions of numerous other specific compounds that were found in dust particles that settled outdoors. They also noted that penetration of substantial quantities of WTC-derived dust into indoor office or residential spaces likely notably increased the potential for indoor exposures (via ingestion or by inhalation of re-entrained particles) to high levels of constituent elements and compounds.

More detailed chemical analyses have been performed on aerodynamically size-separated $PM_{2.5}$ derived from bulk dust samples collected on September 12 and 13 from several locations within 0.5 miles of Ground Zero (EPA, 2002c; McGee et al., 2002). These analyses showed that calcium sulfate (gypsum) and calcium carbonate (calcite) were major components of the fine fraction, indicating that very finely crushed building materials were still dominant components even in this size range. Fine particles more easily penetrate into offices and residential spaces and thereby contribute to indoor exposures more readily than coarse particles.

Data for EPA/ORD measurements of PM elemental composition of fine particles ($PM_{2.5}$) for samples collected starting September 21 are graphically depicted in Figures 14 to 18. The elements are grouped in each figure roughly according to their relative abundance in the subject air samples, those in Figures 14 to 16 being among the most abundant and those in Figures 17 to 18 being distinctly less abundant. Most of the elements shown in the figures were well correlated ($r > 0.85$) with each other at individual sites, with several being much more highly correlated ($r > 0.95$) with each other and with $PM_{2.5}$ throughout the sampling campaign. The very high correlations suggest a common source origin for these elements, that is, WTC fires. The composition of the emissions from Ground Zero combustion sources changed with time as evidenced by initial peaks in several elements (e.g., calcium, potassium, sulfur, chlorine, bromine, lead, copper, and zinc) during late September and early October (Figures 14 to 16), followed by later peaks in the concentration of chromium, arsenic and antimony during mid- or late November (Figures 17 and 18). The elements for which data are depicted in Figures 14 to 18 were selected for illustration from a larger set measured by ORD, based on evident elevations of their concentrations over typical background levels at some point during the sampling campaign.

Consistent with Liroy et al.'s finding of highly enriched calcium in both fine and coarse fractions of settled dust near the WTC, markedly increased levels of calcium continued to be seen in airborne fine particles (Figure 14) at ORD's Ground Zero perimeter sites off and on throughout September and October and much of November, decreasing to low background levels by late November. Elevated levels of calcium in the fine fraction are indicative of highly pulverized building materials (e.g., wallboard) from the WTC site. However, except for a few occasions (e.g., on October 6), airborne fine PM calcium levels were not markedly elevated above background levels at the EPA 290 Broadway site a few blocks northeast of Ground Zero, whereas calcium in the $PM_{10-2.5}$ coarse fraction (not graphically depicted here) did show rather frequent elevations at the 290 Broadway site but decreased to low background levels by the end of November.

Fine PM silicon elevations were only evident briefly during October 3-5 at Location A

and at the 290 Broadway site, in contrast with coarse ($PM_{10-2.5}$) silicon elevations (generally in the 1000-3000 ng/m^3 range) seen at 290 Broadway on a number of days well into late November. The coarse fraction calcium and silicon enrichments most likely reflect (a) windblown re-entrainment of calcium and silicon-contaminated dust remaining on rooftops or window ledges, for example, after hazardous material cleanup of WTC-derived dust in lower Manhattan during the two weeks after September 11, and/or (b) calcium and silicon particle re-entrainment into ambient air associated with WTC recovery operations and transport of debris away from Ground Zero.

Data for concentrations of elemental carbon and total organic compounds in the aerosol phase based on analyses of paired filter samples are not yet available. However, ORD nephelometer results and the results of analyses of bulk dust composition mentioned below suggest that the WTC emissions contained substantial quantities of carbon produced by incomplete combustion. Surprisingly low total carbon levels (1.5 to 8.5%) were found in aerodynamically size-separated $PM_{2.5}$ samples from the bulk dust samples collected on September 12 and 13 (McGee et al., 2002). These results indicate that crushed building materials were the dominant sources of fine PM immediately after the collapse of the towers, whereas combustion from ongoing fires was a relatively more important source of $PM_{2.5}$ in later emissions from the WTC disaster site. Potassium enrichments were also especially notable for ORD Site A measurements into early October (consistent with combustion of organic materials such as wooden furniture, paper, etc.); but much lower potassium concentrations occurred at 290 Broadway, and air levels of potassium at all ORD sites returned to very low background levels by late November.

Elevations of sulfur, chlorine, and bromine (shown in Figure 15) were clearly evident at ORD Ground Zero perimeter sites and sometimes at 290 Broadway during late September and decreasingly so into October, again consistent with the notable enrichments seen by Liroy et al. in WTC dust particles. The sulfur was likely in oxidized form, some perhaps having been converted from primary emissions of SO_2 into secondary sulfate particles, consistent again with both reports of elevated sulfate levels in settled WTC dust particles (Liroy et al., 2002; McGee et al., 2002) or airborne particles (including very fine fraction particles) collected at the DOE Varick St. site on October 3 (Cahill et al., 2002). Also consistent with the Liroy et al. findings of chlorine and bromine enrichments in WTC settled dust are ORD measurements of unusually elevated chlorine and bromine at Ground Zero perimeter sites. The WTC sources of these halides are not clear, but chlorine from burning plastics is not unlikely. The specific enrichment in fine particles of chlorine (versus more typical sodium chloride present as coarse particles) and no notable sodium enrichment rule out attribution of the chlorine levels simply to airborne sea salt influxes into the WTC fire site.

As seen in Figures 16-18, lead and certain other metals (copper, zinc, antimony, palladium, and cadmium) were notably elevated on some days in late September and into early October at EPA/ORD Site A, as compared with concentrations at Sites C and K, but the concentrations of these metals had generally decreased to background levels by mid-October. The late September/early October elevations at Site A on the WTC north perimeter indicate that the WTC fires were likely a common source of emissions of these metals, because the winds were mainly from the southwest at this time. The detection of elevated levels of arsenic and

antimony in mid-November at ORD Site K on the southwest perimeter of Ground Zero (but not at Sites A or C or at 290 Broadway to the north, northeast, or southeast of the WTC) suggests both a different source of WTC fire emissions and a likely climatic shift to winds flowing mainly from the north/northeast to the south/southwest. The chromium elevations seen around November 20, mainly at Site C, suggest possibly yet another later shift in the composition of Ground Zero sources of WTC-generated airborne particle emissions. Hence, the “WTC signature” appears to have varied over time in terms of its specific elemental composition.

In contrast to the above patterns of element levels which indicate that they may have originated from the WTC fires, the gradually increasing concentrations of nickel up to a range sustained during December and January (after the WTC fires were out) seem to argue against any notable airborne fine-particle nickel emissions from the WTC fires subsequent to the collapse of the WTC buildings on September 11. Still, enrichments in samples of settled dust from sites east of the WTC (Liroy et al., 2002) are likely indicative of nickel having been among the metals present in high concentrations of airborne particles in the initial dust/smoke that enveloped lower Manhattan on September 11. This raises the possibility of (a) any remaining nickel-containing dust being re-entrained into outdoor air during later rescue/recovery operations and/or (b) continued elevations of nickel concentrations in WTC-derived indoor dust and re-entrained indoor air particles.

3. High Temporal Resolution Analyses

The ORD PM_{2.5} and associated element measurements discussed above were obtained over sampling periods of close to 24 hours. Examination of additional data is necessary to determine more precisely the duration and nature of enhanced concentrations of PM constituents and to help understand possible public health impacts of such excursions. For example, of much interest are PM elemental composition data for the period October 3-5, when a high pressure system settled over New York during the early morning hours of October 3 and 4. Concentrations of sulfur were close to six times higher at the downwind ORD WTC site (Site A) than at the upwind site (Site K) on October 4. Concentrations of a number of other elements were also much higher by large enrichment factors at Site A as compared to Site K, as indicated by the enrichment factors (noted in parentheses) for the following elements: silicon (41X), chlorine (500X), potassium (47X), calcium (5X), bromine (350X), copper (130X), zinc (110X), palladium (> 100X), cadmium (>100X), antimony (>100X), and lead (66X). Overall, the above results are most consistent with brief, episodic increases from WTC Ground Zero during early morning hours on October 3 and 4 leading to elevated concentrations of PM and its constituent elements at WTC perimeter sites and one or another sites in lower Manhattan located downwind of the WTC on those days (i.e., to the north and/or northeast). These enhanced concentrations were superimposed on generally higher concentrations of PM and its constituents found upwind of the WTC on those days. The higher concentrations found at the upwind sites were associated with a high pressure system that settled over the New York Metropolitan Area during that period. In any case, the results suggest strongly that the WTC site was the dominant source of these elements on October 3-4.

University of California, Davis measurements (Cahill et al., 2002) on the roof of the DOE building indicate a sharp increase in concentrations of PM_{2.5}, mainly in the size range between 0.34 to 0.56 μm on the morning of October 3, as shown in Figure 19. This very brief excursion

only lasted a few hours. Concentrations of sulfur and silicon at this time were notably elevated at the DOE site as compared to days immediately before and after. Also, ORD data collected by both the nephelometer and the aetholometer sited on the Federal Building at 290 Broadway indicated substantial elevations of PM and light-absorbing components within the space of a few hours early in the morning of October 4. Hourly PM_{2.5} levels at PS 64 were also elevated (some in excess of 100 µg/m³) during or shortly after the same hours on October 4, suggesting brief surface fumigation by the WTC plume to the north-northeast of Ground Zero.

It has been suggested that the high concentrations of sulfur observed at the DOE building on Varick Street were related to transport from power plants either in the Northeast or in the Ohio Valley. During this early October period, there were most probably some increases in the concentration of sulfur associated with regional scale transport of sulfates derived from distant sources. However, the extremely high abundance of sulfur at Site A on the Ground Zero perimeter observed by ORD and the ratio of sulfur observed at Site A compared with that observed at Site K suggests that Ground Zero was a large source of sulfur on October 3 and could have also contributed to sulfur readings at the DOE rooftop site.

The peak elevations of airborne fine PM silicon limited to October 3-4 at Site A and at 290 Broadway are notable, suggesting high temperature volatilization of silicon from glass and/or cement by intense WTC fires on those dates and transport of the WTC plume in a north to northeasterly direction. This is consistent both with the plume trajectory plotted in Figure 12 and the marked increases in silicon levels reported by Cahill et al. (2002) at the DOE Varick Street site, to the north-northeast of the WTC, including unusual measured elevations of silicon in very fine (0.09-0.50 µm) and, possibly, inferred ultrafine (< 0.01 µm) PM size ranges (silicon otherwise typically being mainly associated with coarse fraction particles > 2.5 µm). There were also marked increases in ORD-observed concentrations of various metals on October 3-4, reinforcing the conclusion that the DOE Varick Street data for October 3 reflected emissions from intense Ground Zero fires. The very brief increase in PM_{2.5} values and high levels of silicon at the DOE site on October 3 apparently did not occur again at that site, based on preliminary EPA evaluation of the Cahill et al. raw data provided by DOE (Figure 19). Interestingly, Liroy et al. (2002) reported lead and other substances as being unusually congealed together with silicon in particles from the WTC settled dust, likely due to the vaporization of silicon, lead, and/or other metals by intense heat, followed by their condensation and coagulation into particles with unusual composition.

The above high-resolution measurements suggest that WTC emissions in late September and early October varied greatly at times over 24-hour periods and that some of the more notable emissions probably occurred in discrete events which resulted in air pollutant elevations that lasted only a few hours (mainly overnight during cooler early morning hours before temperature increases after sunrise). Such events were likely related to activities of rescue and recovery operations at the WTC, such as removal of large pieces of debris perhaps resulting in increased oxygen flow and brief flareups of fires within the WTC rubble pile. Further high temporal resolution analyses, including more detailed local-scale WTC plume plots, will be needed to better understand the specific lower Manhattan areas impacted by short-term WTC emission events and the implications of these sporadic events for potential human exposures and health impacts.

IV.a.3. Evaluation of Potential Particulate Matter Human Exposures and Health Impacts

Because no direct measurements were obtained for airborne particle concentrations present in the dense dust/smoke cloud that enveloped lower Manhattan for up to about 4 hours after the collapse of the WTC buildings on September 11, estimates of likely exposures to airborne PM for individuals caught in the initial dust/smoke cloud can only be deduced from indirect evidence and are subject to great uncertainty. Nevertheless, several tentative conclusions appear to be warranted on the basis of available inputs thus far.

First, it is likely that many persons caught outdoors (or some even indoors) in the initial dust/smoke cloud were exposed for several hours to extremely high levels of airborne particles. This exposure probably included inhalation of PM concentrations in the milligrams per cubic meter range, well in excess of 1 to 2 mg/m³ (1000-2000 µg/m³), for *both* fine (PM < 2.5 µm diameter) and coarse (PM > 2.5 µm) inhalable particles. Although there were no measurements available during this critical time period, an examination of available photographs in combination with an empirical relationship based on visibility suggests that concentrations could very easily have been this high and likely were even much higher (e.g., possibly > 5 mg/m³). Such a finding is also supported by analyses of bulk dust samples conducted by Liroy et al. (2002).

The coarse inhalable particles (PM > 2.5 µm) likely included substantial quantities of particles in the PM_{10-2.5} range, which are capable of reaching lower respiratory tract (thoracic) regions of the lung, even though such coarse particles made up less than 0.5% of the particles found in WTC-derived settled dust by Liroy et al. (2002). Individuals who inhaled such high concentrations of WTC dust particles, even for a few hours, would logically be expected to be at potential risk for immediate acute respiratory and other symptoms and/or, possibly, more chronic health impacts associated with lung deposition of notable quantities of constituent PM materials (e.g., calcium, silicon, potassium, lead, other metals).

Persons exposed to the very high PM levels in the initial dust cloud and who continued to work at Ground Zero or returned to work there within a few days without wearing adequate protective respiratory gear might be at especially increased risk for potential acute or chronic health effects, depending on the extent of any ensuing exposures to high PM levels on or immediately around the Ground Zero rubble pile. The latter could include additional exposures to coarse PM constituents (e.g., calcium or silicon) present in re-entrained dust particles from the initial WTC building collapse and/or exposures to newly formed fine particle constituents (e.g., metals), as well as to organic constituents (e.g., PAHs present in both size ranges) emitted from the WTC fires.

Evaluation of potential health impacts associated with the above types of PM exposures should be further facilitated by disease registry efforts and retrospective epidemiologic analyses of physician/emergency department visits and hospital admission records being sponsored by the Centers for Disease Control (CDC), the Agency for Toxic Substances and Disease Registry (ATSDR), the National Institute of Environmental Health Sciences (NIEHS), and other federal, state, and NYC agencies and that are now under way. Recent reports (Prezant et al., 2002)

indicate that large percentages of firemen caught in the initial WTC dust cloud and others who worked at Ground Zero during the first 2 to 7 days post September 11 experienced respiratory (e.g., “WTC cough” and/or bronchial hyperactivity) or other symptoms that still continue to persist for some individuals several months after cessation of exposures at WTC Ground Zero.

During the week following September 11, the plume from initial high-intensity WTC fires appears to have been largely convected upwards and dispersed mainly to the south-southeast or south-southwest without much evident ground level contact, except perhaps for a few hours on the mornings of September 12 and 13, when it flowed to the east-southeast of the WTC. This resulted in briefly increased hourly $PM_{2.5}$ levels at sites in lower Manhattan ($166 \mu\text{g}/\text{m}^3$ at PS 64 on 9/13) and in Queens ($100 \mu\text{g}/\text{m}^3$ at PS 199 on 9/13). Probably few people were exposed around the PS 64 site, given the restrictions in effect on motor vehicular or pedestrian traffic below 14th Street until September 14, but some may have been briefly exposed in the vicinity of the PS 199 site and/or at locations between the two sites. Although it is doubtful that the brief, several-hour $PM_{2.5}$ excursion on the morning of September 13 resulted in harmful PM exposures, retrospective examination of physician and emergency department visits and/or hospital records in the affected areas may help to verify this.

After September 21, EPA/ORD monitoring indicated initially high levels of WTC-derived airborne particles (especially at certain Ground Zero perimeter sites) during late September and early October, but occurrences of PM excursions decreased over time through late October and into November. The rate of decrease in concentrations was not uniform throughout the monitoring period; rather, there were episodes of high PM levels spaced between periods of much lower concentrations. The frequency of the episodes was highest during the first month following the collapse of the WTC buildings and then declined afterwards. For example, notable PM emission episodes occurred during the first month of sampling, as shown in Figure 11. $PM_{2.5}$ concentrations varied over a wide range (sometimes exceeding the relevant AQI $40 \mu\text{g}/\text{m}^3$ action level) during late September and early October at EPA/ORD WTC perimeter Site A. The concentrations of a number of elements measured at this site also showed large day-to-day variability, as shown in Figures 14-18. On a number of days in late September and early October, concentrations of several elements were many times higher than the more typical background levels recorded for December-January, after the WTC fires had largely or entirely burned out.

On the basis of overall air quality results summarized above, it appears that 24-hour PM_{10} and $PM_{2.5}$ values throughout most all of the NYC metropolitan area generally remained at or returned rather quickly to historical background levels and WTC PM emissions posed no increased health risks beyond those due to usual PM levels for most areas of NYC. On the other hand, high $PM_{2.5}$ concentrations recorded on the perimeter of Ground Zero during late September and early October may imply increased health risks for the most highly exposed individuals (that is, persons who spent extended periods of time within the WTC Ground Zero work zone without wearing protective respirators). Specifically, acute exposures to irritating materials present in either the $PM_{2.5}$ or coarse particle components of PM_{10} , especially during any high hourly peak excursions, may have contributed to acute or continuing respiratory symptoms reported by some workers and/or residents in lower Manhattan areas in the immediate WTC vicinity. It is much less likely that any markedly increased health risks were posed by ambient air PM exposures

elsewhere in the lower Manhattan neighborhoods surrounding the WTC, although more thorough analysis and modeling of potential PM exposures and correlation with health records is needed to evaluate this issue more fully.

It may be useful to place the above potential airborne PM exposures in perspective by comparing them to (a) exposures that occurred during some past notable PM air pollution episodes and (b) more recent historical data recorded for New York City areas. As discussed in U.S. EPA (1982, 1986a) and Elsom (1992), a number of past severe air pollution episodes involved extended periods of exposure of urban populations to high concentrations of airborne PM and associated air pollutants such as SO₂. In contrast to relatively brief periods (< 8 hours) of September 11 inhalation exposures on September 11, 2001 to concentrations in the range of, and maybe in excess of, 1000-2000 µg/m³ of WTC-derived airborne coarse and fine PM, a number of past air pollution episodes in U.S. cities (e.g., Donora, PA in 1948; NYC in 1953 and 1962/63) and internationally (e.g., Neuse Valley, Belgium, 1930; London, UK in 1952, 1957, 1963) involved exposures to very high levels of PM that lasted for at least several days. Probably the most famous such episode occurred in London in December 1952, when millions of Londoners were exposed to daily PM levels (measured as British Smoke, which included high percentages of fine particles) of 1000-4000 µg/m³ (sometimes reaching hourly peaks of 6000 µg/m³ or more) on 3 to 5 consecutive days in the presence of 1000-4000 µg/m³ of SO₂.

In NYC, PM_{2.5} values recorded on some days at the Ground Zero perimeter and occasionally elsewhere in lower Manhattan during late September and early October clearly exceeded the more usual background levels of fine PM seen in NYC since implementation of the PM NAAQS in the 1970s began to substantially reduce ambient PM concentrations in U.S. urban areas. For example, some ORD WTC perimeter site 24-hour PM_{2.5} measurements of more than 100 µg/m³ likely exceeded most - but not necessarily all - values recorded at the New York University Medical Center in an aerosol sampling study conducted in August 1976 (Lippman, et al. 1979), as per the mean value shown for PM_{2.0} samples in Table 2. However, the 24-hour PM_{2.5} concentrations (predominantly below 30-40 µg/m³) usually seen during most of the rest of October and into November at ORD WTC perimeter sites were notably lower than the 1976 values; and the PM_{2.5} levels reached by December and January generally compare favorably with PM_{2.5} values obtained at a monitoring site at the Bronx Botanical Gardens during February-June 2000. This Bronx site can be considered to be a relatively "clean" urban background site largely free of the effects of strong local sources. Maximum PM_{2.5} values ranged from 35.4 to 43.3 µg/m³ from three inter-compared collocated samplers at that Bronx site and average 24-hour PM_{2.5} values ranged from 12.5 to 15.6 µg/m³, analogous to the mean PM_{2.5} concentrations shown in Table 2 for Boston and Philadelphia during February-June, 2000. Also, PM₁₀ values for lower Manhattan sites, which were mainly in the range of 50-90 µg/m³ during October and mostly below 50 µg/m³ in November, were not markedly different from historical values observed in NYC. For example, the fourth highest and the maximum 24-hour PM₁₀ values reported in the EPA Aerometric Information Retrieval System (AIRS) database for the five NYC boroughs during 1996 to 2001 ranged from 40-89 µg/m³ and from 51-121 µg/m³, respectively.

During September-October, 2001, concentrations of many elements, including heavy metals, at Ground Zero perimeter sites were at times much greater than those observed at several sites in the Northeast in February-June 2000. As part of a pilot study for EPA's PM_{2.5} speciation

network, concentrations of PM_{2.5} and a number of key elements were measured from February to June, 2000, in NYC (at the Bronx Botanical Gardens), Boston, and Philadelphia, at sites that were likely characteristic of urban backgrounds. As shown in Table 2, the average concentrations of PM_{2.5} and the individual elements measured at the three sites varied relatively little in contrast to those measured near the WTC site. The highest measurements of PM_{2.5} and heavy metals are also shown in Table 2 for comparison. During the first weeks after September 11, PM_{2.5} levels and concentrations of several elements often were many times higher than those obtained at the northeastern sites listed in Table 2. As also seen in Table 2, concentrations recorded at the New York University Medical Center in 1976 (Bernstein and Rahn, 1979) were higher than those at the other Northeastern sites, but they were still distinctly lower than the measurements made soon after September 11.

During December and January, the concentrations of most of the elements measured by ORD decreased to levels similar to those measured at the northeastern sites, suggesting that the WTC was no longer a significant source of these elements. However, it should be noted that even the markedly elevated element concentrations over typical background values noted mainly in September and October did not exceed applicable OSHA PEL (8-hour time-weighted average) values or other more broadly applicable health benchmark values, suggesting a generally low health risk for those working at Ground Zero or others present in lower Manhattan neighborhoods around the WTC.

Alkalinity of the dust from the WTC disaster may have been a possible health concern for exposed individuals. Some reported symptoms (eye, nose, and throat irritation; nose bleeds; cough) may have been due to exposure to unusually elevated quantities of certain crustal materials derived from pulverized concrete, wallboard, and other WTC structural components present in airborne particles and settled dust in neighborhoods near the WTC. The United States Geological Survey (USGS, 2002), Liroy et al. (2002), and McGee et al. (2002) all reported testing aqueous solutions of WTC settled dust and finding an initially high alkalinity (generally $\geq$ pH 10.0), which decreased to pH 8-9 for outdoor samples taken after rainfall (as reported by USGS). Because much of the outdoor settled dust was removed by hazardous material cleanup procedures or was washed away by rainfall during late September, outdoor exposures to highly basic PM components would seem to be of much less potential health concern beyond late September when restricted zone shrinkages allowed more people and traffic in neighborhoods immediately surrounding the WTC work zone. However, USGS noted higher alkalinity for dusts sampled indoors, raising the possibility of greater risk of acute irritation symptoms being associated with indoor exposures to WTC dusts than with outdoor dusts leached by rainfall.

Liroy et al. (2002) more broadly highlighted indoor exposures to WTC-derived dust PM as posing potential increased health risks. Individuals visiting, residing, or working in buildings not adequately cleaned before reoccupation could have been subjected to repeated, long-duration exposure to many of the components from the original WTC collapse found by Liroy et al. in settled dust to the east of the WTC. Liroy et al. noted that long, narrow glass fibers in the WTC-derived dust had various potentially toxic materials attached to them and could contribute to acute short-term irritative effects and possibly to more chronic health risks.

Also of potential concern would be any extended indoor air exposures to finely pulverized

building materials (e.g., calcium, silicon, iron, and sulfate) in PM particles, to PM of either fine or coarse size containing marked elevations of certain metals, or to fine PM containing usual combinations of silicon coagulated with metals or other toxic materials. Liou et al. directed notable attention to indoor dust loadings of lead as posing potential chronic health risks. The possible contributions of certain other metals (e.g., nickel, chromium) found in settled dusts or airborne PM to irritative symptoms also need further evaluation. The discussions below for lead, nickel, and chromium contain more information on the possible bases for concern with these particular metals. The issue of potentially greater toxicity being associated with unusually increased quantities of very fine or ultrafine particles present in airborne PM also needs to be evaluated further.

Some newly available findings from the laboratory toxicity studies of WTC-derived dusts may offer insights into potential health responses associated with exposures on September 11 to WTC-derived materials in the initial WTC building collapse dust cloud and later exposures to WTC-particles deposited indoors. ORD NHEERL scientists analyzed chemical and toxicological properties of PM_{2.5} derived from the collapsed WTC buildings (EPA, 2002c; McGee et al., 2002; Gavett et al., 2002). Deposited dust samples were collected from sites within a half-mile of Ground Zero on September 11 and 12 and size-separated to collect the PM_{2.5} fraction. Gavett et al. (2002) evaluated responses of young adult female mice to bolus doses of WTC-derived PM_{2.5} dusts administered by intratracheal instillation directly into the lungs and to 5-hour inhalation exposures to WTC PM_{2.5}. On the basis of the overall pattern of results obtained, Gavett et al. observed both a small degree of pulmonary inflammation in response to WTC dust, which was distinctly less compared with exposure to residual oil fly ash (ROFA), and some notable increases in airway hyperresponsiveness to methacholine, a nonspecific bronchoconstricting agent, greater than that observed in mice exposed to ROFA or other PM samples. These effects may be interpreted as being consistent with reports of airway hyperresponsiveness and irritant responses in people exposed to high concentrations of WTC-derived dusts. Whereas the mild pulmonary inflammation in mice diminished from 1 to 3 days after exposure, the airway hyperresponsiveness appeared to persist longer.

These results suggest possible limited, short-term lung inflammation effects from exposures to high concentrations of WTC dust (as may have occurred mainly on September 11) or possible long-term airway hyperresponsiveness that might portend more prolonged sensitivities and irritative symptoms for persons experiencing extended high-level exposures to WTC-derived dusts indoors. Gavett et al. (2002) estimated the human exposure equivalent of the doses that led to these responses in mice, and calculated an 8-hour exposure at moderate activity level to a concentration of 425 µg/m³ using a multiple-path particle deposition model (Ferijer et al., 1999). Individuals who are especially sensitive to inhalation of dusts, such as asthmatics, might experience these effects at lower concentrations over more prolonged periods of exposure. As mentioned previously, it is likely that persons caught in the initial dust/smoke cloud could have been exposed to PM_{2.5} concentrations in excess of 1000 µg/m³. However, a dose equivalent to a human exposure of 130 µg/m³ over 8 hours caused no significant effects in the mice, suggesting that most healthy people would not be expected to respond to this moderately high exposure level with any adverse respiratory responses (EPA, 2002c; Gavett et al., 2002).

These studies did not address the effects of the coarse fraction of WTC PM on responses in mice, which may be very important, considering the upper airways responses and ocular irritant effects that have been reported.

Table 2. Concentrations (maximum and Sept-Oct average) of PM_{2.5} and Component Elements Measured at the EPA/ORD World Trade Center Perimeter Sites and at Four Other Sites in the Northeast U.S. (PM_{2.5} concentrations in µg/m³; all other components in ng/m³).

	WTC Max. (Site, Date)	WTC Average Sept-Oct ¹			NYU Med. Ctr. ²	Bronx, NY ³	Boston, MA ³	Phila, PA ³
		A	C	K	Aug '76	Feb-June '00	Feb-June '00	Feb-June '00
PM_{2.5}	400 (A, 10/4)	85	34	50	81.7 ⁴	12.5	10.7	14.7
Na	870 (A, 10/4)	273	157	169	570	72	178	63
Mg	490 (K, 11/13)	101	67	79	103	4.8	16	7.7
Al	670 (A, 10/20)	198	74	113	187	9.2	25	18
Si	20000 (A, 10/4)	943	224	333		75	92	118
S	23000 (A, 10/3)	4796	1808	2524	6820	1200	933	1500
Cl	45000 (A, 10/4)	7247	540	845	119	9.8	68	7.7
K	5600 (A, 9/22)	988	147	260	194	38	38	60
Ca	4900 (A, 10/11)	1304	345	749		38	50	57
Cr	34 (C, 11/19)	5	4	3	28	0.3	0.5	1.1
Fe	9400 (K, 12/12)	1745	904	975	400	91	76	103
Co	24 (A, 10/3)	4	bdl	bdl	3.2	0.4	0.4	
Ni	50 (K, 12/11)	11	9	11	18	12	2.8	4.4
Cu	2800 (A, 9/22)	435	59	92		2.8	2.2	4.5
Zn	10000 (A, 10/4)	1526	164	307	224	21	9.7	16
As	1100 (K, 11/14)	9	bdl	bdl	3	1.1	0.9	1
Br	5700 (A, 9/20)	800	82	124	133	2.5	2.5	3.4
Pd	900 (A, 10/4)	132	2	7				
Cd	150 (A, 9/22 & 10/4)	33	9	9	7.7	1.7	1.7	1.8
Sb	350 (A, 9/22)	50	2	1	11	3.6	2.4	3.5
Pb	5500 (A, 9/22)	791	83	167	1170	4.2	3.4	5.6

¹A time series of PM_{2.5} measurements for Site A (as well as Sites C, K, and 290 Broadway) can be seen in Figure 11. Time series measurements for the

elements in $PM_{2.5}$ from the same sites can be seen in Figures 14-18. Measurements in these tables are the high measurements seen in these figures, and the dates of the measurements are shown in parenthesis. Nearly all elevated measurements were seen in Site A, and in late September, early October. bdl = below detection limit and n.a. = data not available. ²NY Summer Aerosol Study, 1976. ³Coutant et al., 2001. ⁴ $PM_{2.0} = 0.9 * PM_{2.5}$ (estimated).

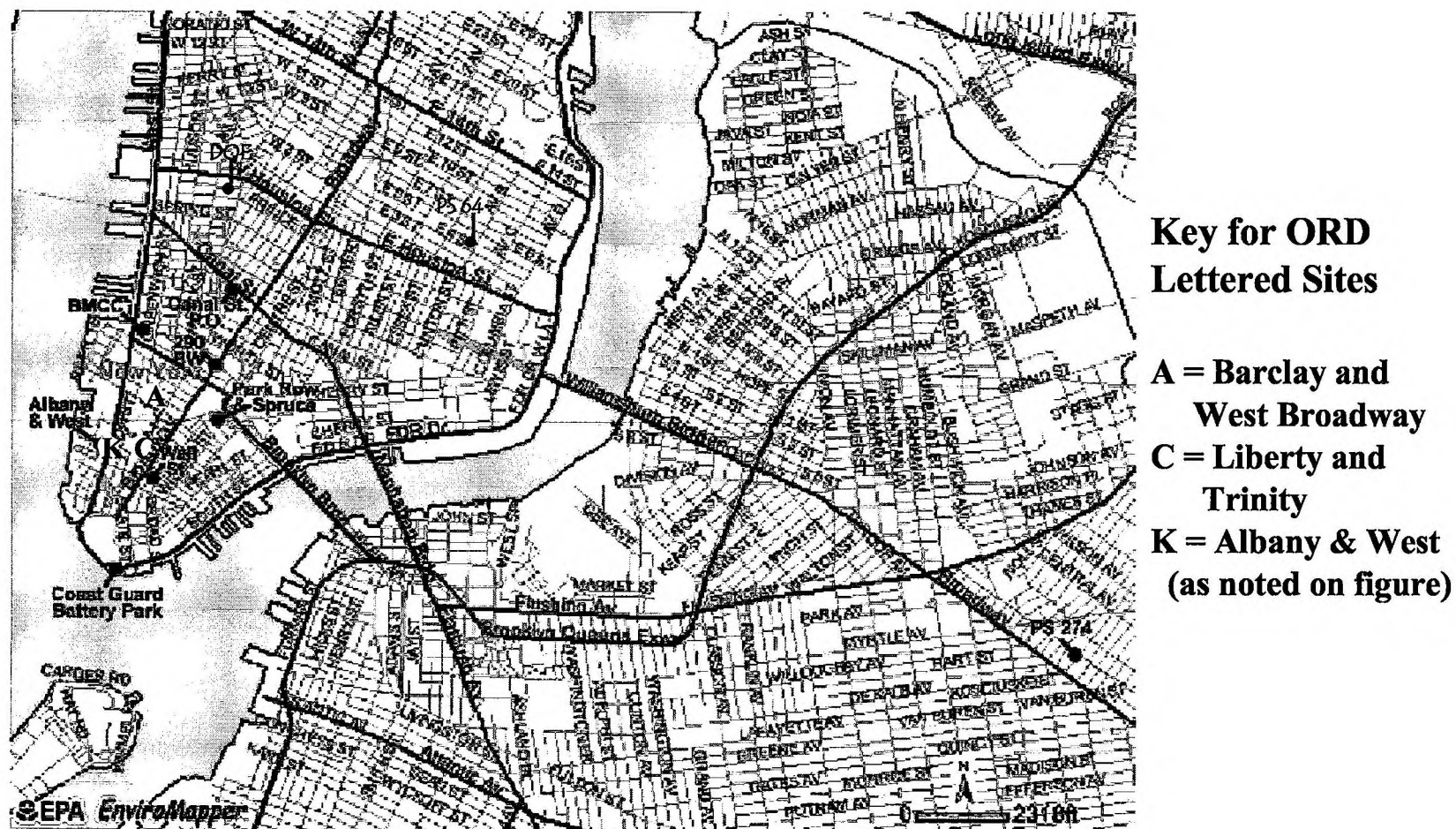


Figure 2. Particulate matter monitoring sites, including ORD surface sites (A, C, K) on the WTC perimeter, the ORD site at 290 Broadway, and NYSDEC sites located elsewhere..



Figure 3. Spread of dense dust/smoke cloud over all of lower Manhattan and drifting to the E/SE immediately after the September 11, 2001, collapse of the World Trade Center buildings.

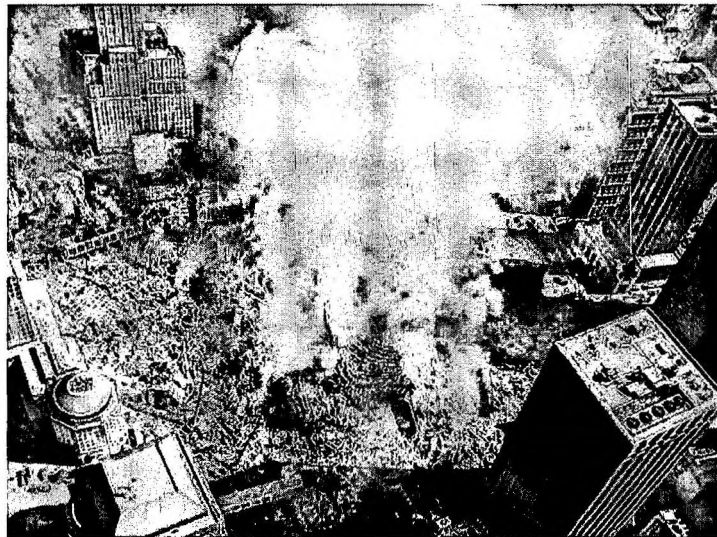


Figure 4. World Trade Center (WTC) plume from intense fires ($>1000^{\circ}\text{F}$) during days following September 11, 2001, with high concentrations of both newly formed fine particles from combustion and reentrained coarse particles likely being transported upward by convection processes and being dispersed in the WTC plume over varying NY City areas, depending on prevailing wind directions and speeds.

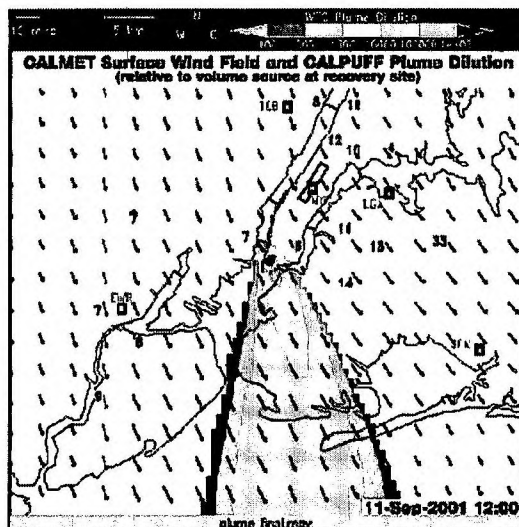


Figure 5. ORD-modeled WTC Plume Dispersion on September 11, 2001 at 12 noon. The values indicated by red numerals are hourly $PM_{2.5}$ concentrations (in $\mu g/m^3$) measured at pre-existing NJ and NY State-operated PM monitoring stations in northern New Jersey and New York City. Red, orange, and yellow shading represent most likely areas of plume dispersion (red = estimated dilution to 100th to 500th and dark blue = dilution to < one millionth of pollutant concentration at WTC source).



Figure 6. Satellite photograph of the WTC plume lofting from Ground Zero at 11:43 a.m. EDT on September 12, 2001. Note the very concentrated vertical convection of dust/smoke particles upwards and the flow in a well-defined plume towards the S/SE. (Source: Mandatory credit: “spaceimaging.com”).

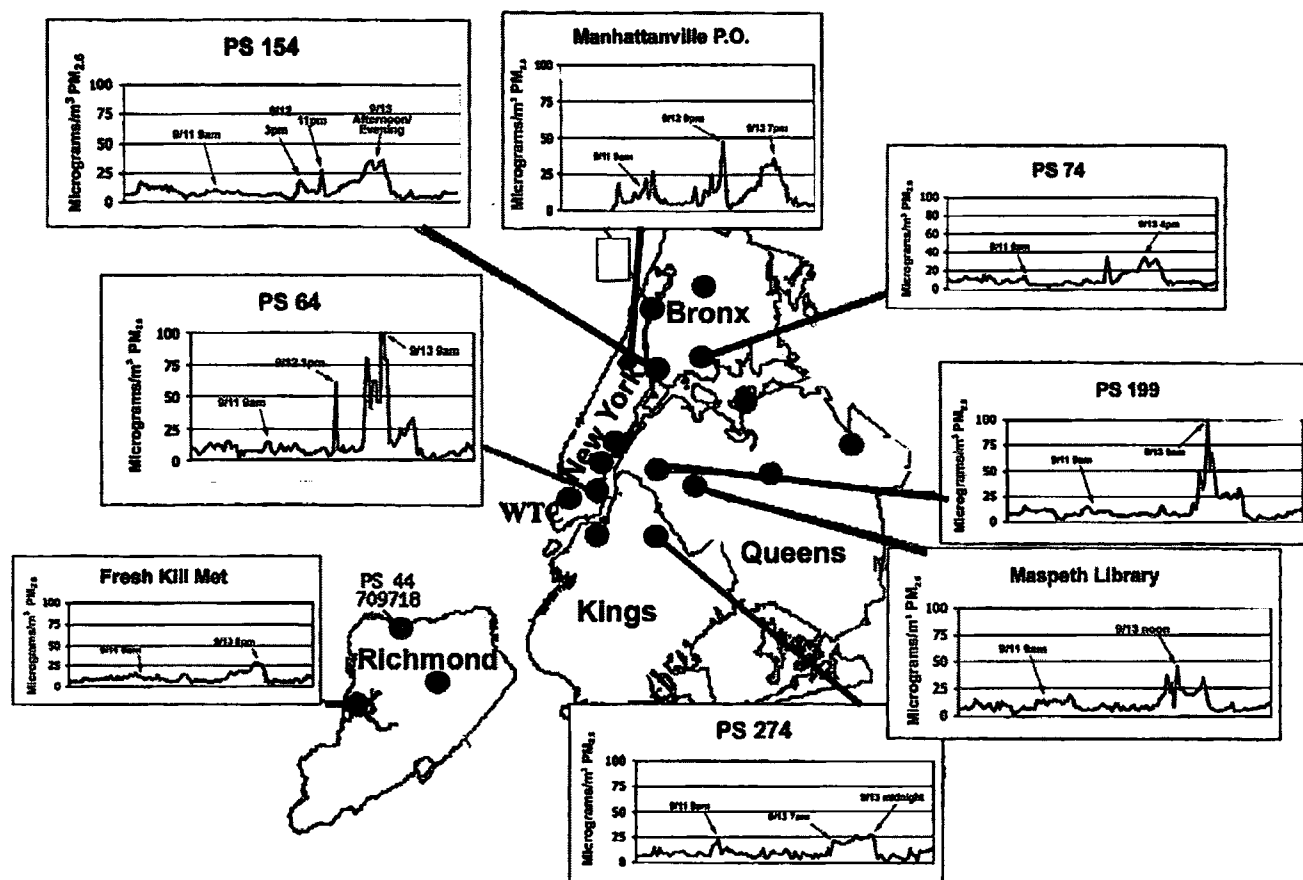


Figure 7. Increased hourly PM_{2.5} concentrations measured on September 12 and/or 13 at PS 64, PS 199, Maspeth Library, and PS 274 to the E/SE of WTC, reflecting dispersal of newly formed fine particles from WTC fires and/or fine particles reentrained from the settled dust from initial collapse of WTC buildings. (PM_{2.5} data provided courtesy of NYSDEC).

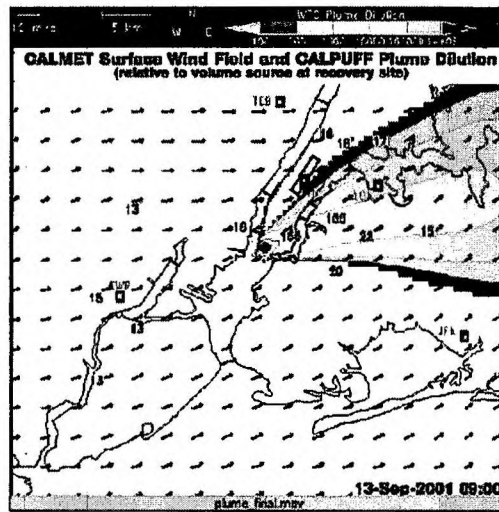


Figure 8. ORD-modeled WTC plume dispersion for September 13, 2001 at 9:00 a.m. Note the increased hourly $PM_{2.5}$ concentrations depicted in red for PS 64 ($166 \mu g/m^3$) and PS 199 ($100 \mu g/m^3$) NYSDEC monitoring stations, consistent with the E/NE direction of the modeled plume dispersion and likely touchdown of the plume at those and intervening sites but not at sites further E, SE, or to the N.

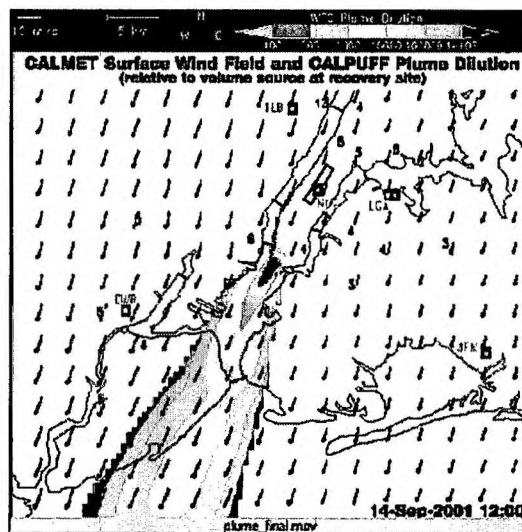


Figure 9. ORD-modeled WTC plume dispersion on September 14, 2001 at 12 noon, indicative of plume flow mainly out over New York Harbor.



Figure 10. Satellite photograph of WTC plume lofting from GZ at 11:54 a.m. EDT on Sept. 15, 2001, and dispersing to the S/SW out over the New York Harbor. (Source: Mandatory credit: “spaceimaging.com”).

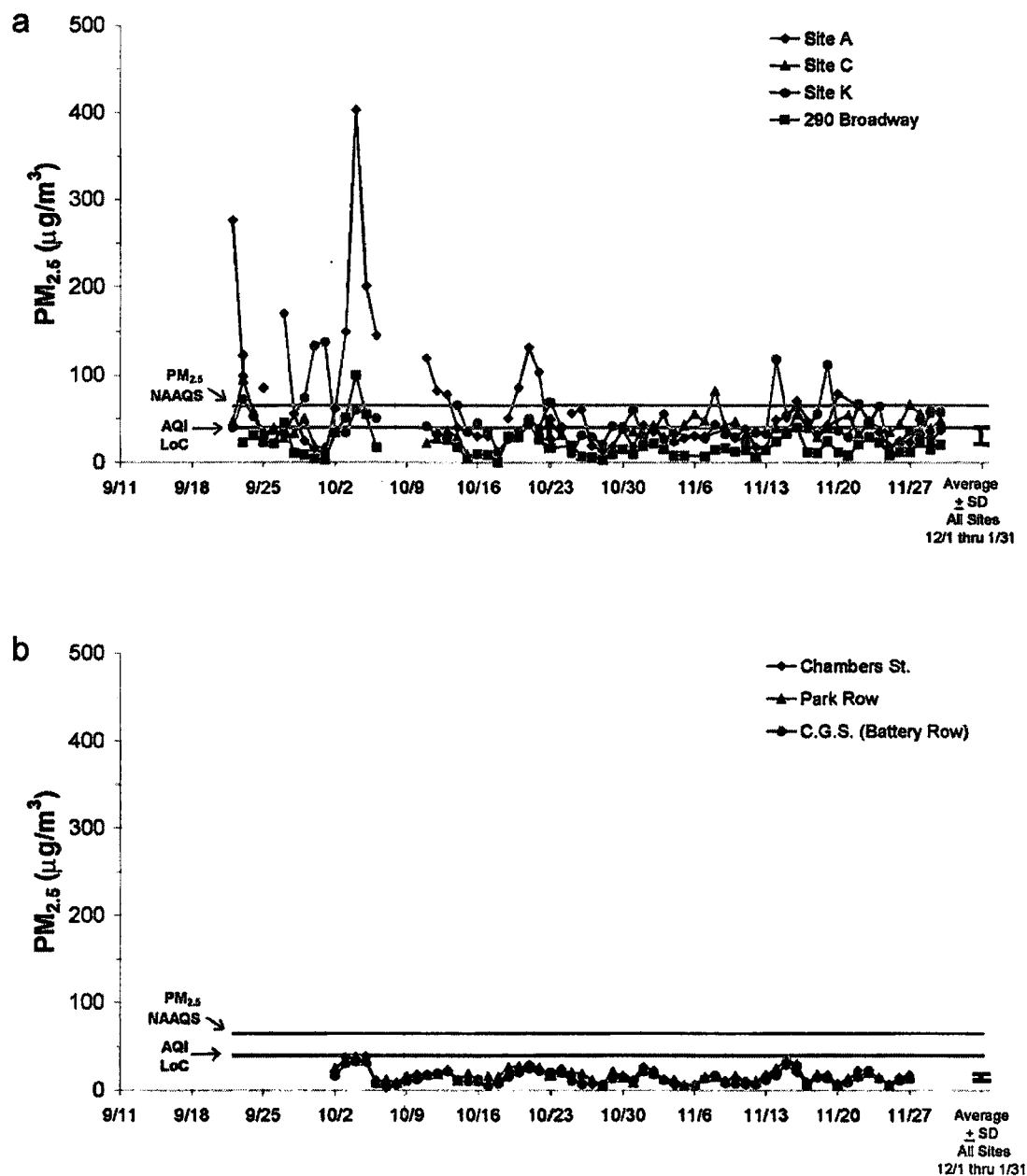


Figure 11. *Panel A (top):* Daily PM_{2.5} concentrations monitored by EPA/ORD at sites A, C, and K around Ground Zero perimeter and at 290 Broadway 6 blocks northeast of Ground Zero. *Panel B (bottom):* PM_{2.5} concentrations observed at several extended monitoring network sites in lower Manhattan within 3 to 10 blocks of WTC Ground Zero.

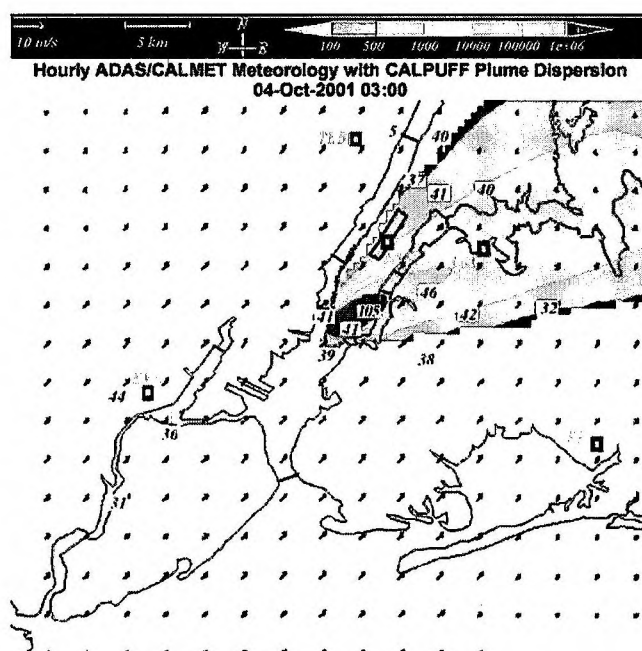


Figure 12. ORD-modeled WTC plume dispersion on October 4, 2001 at 3:00-4:00 a.m. Note the general regional elevation of hourly $PM_{2.5}$ levels (in $\mu g/m^3$) indicated by red numerals for monitoring sites scattered across both northern New Jersey and NYC areas, even outside modeled areas of likely greatest plume intensity indicated by red shading.

PM_{2.5} Since September 11

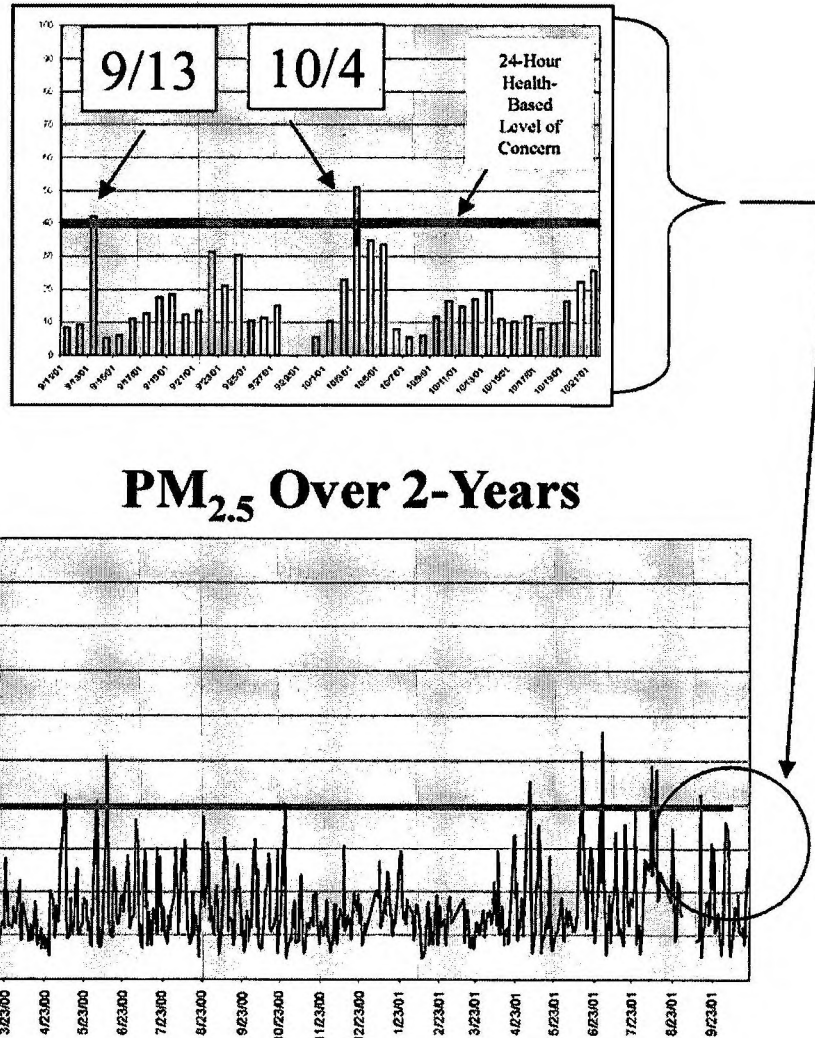


Figure 13. Daily PM_{2.5} concentrations recorded at NYSDEC PS 64 monitoring site after September 11, 2001 (9/11/01 to 10/27/01) compared to historic record of 24-hr PM_{2.5} values at the same site during prior 2 years (2/23/00 to 9/01/01). Note exceedence of 40 µg/m³ AQI Level of Concern on September 13 and likely again on October 4; red portion of bar indicates 24-hr average if three high hourly values (> 100 µg/m³) being evaluated for data quality are included in 24-hr average calculation.

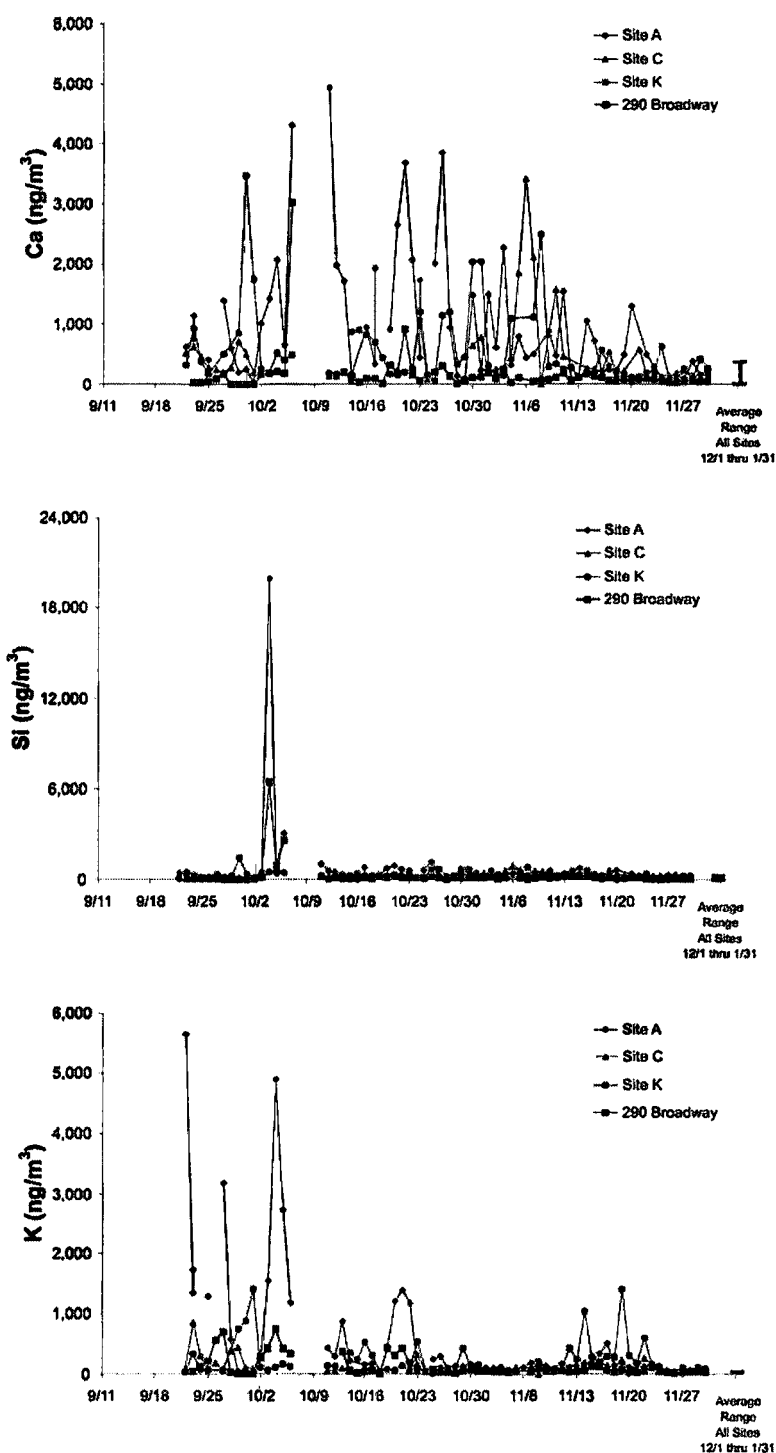


Figure 14. ORD measurement of $\text{PM}_{2.5}$ elemental constituents Ca, Si, and K at Ground Zero perimeter sites and 290 Broadway site.



Toxicological Effects of Fine Particulate Matter Derived from the Destruction of the World Trade Center

EPA/600/R-02/028
December 2002

Toxicological Effects of Fine Particulate Matter Derived from the Destruction of the World Trade Center

National Health and Environmental Effects Research Laboratory
Office of Research and Development
U.S. Environmental Protection Agency
Research Triangle Park, North Carolina 27711

Notice

This report has been reviewed and approved for release by the National Health and Environmental Effects Research Laboratory of the US Environmental Protection Agency. Approval does not signify that the contents necessarily reflect the views and policies of the Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use. This report has been audited for quality assurance purposes and a Quality Assurance statement is included. Supporting documentation and raw data are available from Dr. Stephen H. Gavett, National Health and Environmental Effects Research Laboratory (MD-82), U.S. Environmental Protection Agency, Research Triangle Park, NC 27711 (telephone 1-919-541-2555, e-mail gavett.stephen@epa.gov).

Cover photographs courtesy of the Federal Emergency Management Agency

Contents

Authors, Contributors, and Reviewers	v
Executive Summary	vi
I. Introduction	1
II. Materials and Methods	3
A. WTC PM Sample Collection and Size Fractionation	3
B. Extraction of PM from Teflon Filters	4
C. Control PM Samples Used in WTC2001 Study	5
D. Physical and chemical analysis of solid (bulk and filter) samples.	6
1. Scanning electron microscopy / energy-dispersive x-ray (SEM/EDX) analysis.	6
2. X-ray diffraction (XRD) analysis.	6
3. X-ray fluorescence (XRF) analysis.	7
4. Carbon fraction analysis.	7
E. Chemical analysis of liquid extracts of bulk and filter samples.	7
1. pH.	7
2. Endotoxin.	7
3. Inductively coupled plasma - atomic emission spectrometry (ICP-AES) and - mass spectrometry (ICP-MS).	7
4. Ion chromatography (IC) of deionized water extracts.	8
F. Experimental Animals and Weight Randomization.	8
G. Toxicological Endpoints: Experimental Design.	8
1. Experiment A.	9
2. Experiment B.	9
3. Experiment C.	9
H. Oropharyngeal Aspiration of PM Samples.	9
I. Nose-Only Inhalation Exposure.	10
J. Respiratory Responses Assessed by Whole Body Plethysmography.	10
1. Immediate Airway Responses to PM _{2.5} Exposure.	10
2. Airway Responsiveness to Methacholine Aerosol.	11
K. Diffusing Capacity of the Lung for Carbon Monoxide.	11
L. Bronchoalveolar Lavage (BAL).	11
M. Histopathology	12
1. Lung histopathology.	12
2. Nasal histopathology.	12
N. Statistical Analysis	12

III. Results	14
A. Chemical analysis of solid samples and liquid extracts.	14
1. Endotoxin and pH levels.	14
2. Elemental and Ion Analysis.	14
3. Carbon analysis.	16
4. Compound analysis by XRD.	16
5. SEM/EDX analysis.	17
6. Summary.	18
B. Experiment A: Dose-Response Relationships of WTC PM _{2.5}	19
1. Body weights and immediate airway responses.	19
2. DLCO.	20
3. BAL parameters.	20
4. Responsiveness to methacholine aerosol.	22
5. Lung histopathology.	22
6. Summary.	25
C. Experiment B: Effects of Nose-Only Inhalation Exposure	25
1. Exposure results.	25
2. Body weights.	26
3. Immediate airway responses to nose-only exposure.	26
4. DLCO measurements.	27
5. Responsiveness to methacholine aerosol.	27
6. BAL parameters.	29
7. Nasal histopathology.	30
8. Lung histopathology.	31
9. Summary.	31
D. Experiment C: Effect of Geographical Location of WTC PM Samples on Responses .	31
1. Sub-experiments and body weights.	31
2. Responsiveness to methacholine aerosol.	32
3. BAL cells.	35
4. BAL proteins and enzymes.	38
5. Lung histopathology.	38
6. Summary.	41
IV. Discussion	44
V. Quality Assurance Statement	48
VI. References	50

Authors, Contributors, and Reviewers

Authors

Stephen H. Gavett, Najwa Haykal-Coates, John K. McGee, Jerry W. Highfill, Allen D. Ledbetter, and Daniel L. Costa — National Health and Environmental Effects Research Laboratory (MD-82), U.S. Environmental Protection Agency, Research Triangle Park, NC 27711.

Contributors

John J. Vandenberg, Thomas J. Hughes, Brenda T. Culpepper, M. Ian Gilmour, Judy H. Richards, Paul A. Evansky, Dock Terrell, James R. Lehmann, Elizabeth H. Boykin, Mette J. Schladweiler, and Hassell G. Hilliard — National Health and Environmental Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, NC.

Lung Chi Chen, Mitchell D. Cohen, Glenn R. Chee, Colette M. Prophete, and Jessica Duffy — New York University, Tuxedo, NY (supported by NIEHS Center grant ES00260 and EPA PM Center grant R827351).

Glen E. Marrs and Staff — Experimental Pathology Laboratories, Research Triangle Park, NC.

Jack R. Harkema and James G. Wagner — Michigan State University, East Lansing, MI.

Shirley J. Wasson — National Risk Management Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, NC.

Teri L. Conner — National Exposure Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, NC.

Annette S. King and A. Glenn Ross — NCCBA / Senior Environmental Employment Program, Research Triangle Park, NC.

Dennis D. Williams and William D. Ellenson — ManTech Environmental, Research Triangle Park, NC.

Robert A. Cary and David F. Smith — Sunset Laboratory, Hillsborough, NC.

Reviewers

John B. Morris — University of Connecticut, Storrs, CT.

Michelle M. Schaper — Mine Safety and Health Administration, Pittsburgh, PA.

Michael C. Madden — National Health and Environmental Effects Research Laboratory, U.S. Environmental Protection Agency, Chapel Hill, NC.

Executive Summary

The goal of the experiments described in this report was to evaluate the toxicity of fine particulate matter (PM) derived from the destruction of the World Trade Center (WTC) on the respiratory tract of mice, and thereby contribute to the short-term health risk assessment of WTC PM being conducted by the Environmental Protection Agency. The adopted approach allowed a comparison of the intrinsic acute toxicity of fine WTC PM in the respiratory tract to well-studied PM reference samples that range in toxicity from essentially inert to quite toxic. The fundamental question was whether fine WTC PM was uniquely highly toxic.

This toxicological research complements efforts by EPA and other organizations to assess the extent and level of worker and public exposures to PM derived from the WTC disaster and recovery efforts. This research is informative, but it is of limited scope, with a focus on the toxicological effects of the fine fraction of WTC dust from a single exposure. A more complete characterization of potential health effects would include consideration of other size fractions, repeated exposures, additional doses and endpoints, and responses in species or strains of differing sensitivity. It was not possible to assess these other considerations in the present study.

Fallen dust samples were collected on September 12 and 13 from various sites around Ground Zero, and the fine PM fraction (< 2.5 microns in diameter; $PM_{2.5}$) was isolated on filters. $PM_{2.5}$ was extracted from the filters and extensively analyzed by several chemical and physical techniques. A dose-response study in mice was conducted comparing aspirated WTC $PM_{2.5}$ (pooled from 7 different locations near the WTC site) with low and high toxicity $PM_{2.5}$ control samples (Mt. St. Helens and residual oil fly ash (ROFA), respectively). An acute nose-only inhalation exposure study was conducted on one WTC $PM_{2.5}$ sample, since upper airway irritation has been a primary complaint of those living and working in the WTC area. Finally, a short-term time course study was conducted comparing aspirated samples from the 7 different locations with each other and with a standard $PM_{2.5}$ sample (NIST 1649a, an ambient air PM sample collected in Washington, DC).

Fine size-fractionated WTC $PM_{2.5}$ was composed primarily of calcium-based compounds such as calcium sulfate (gypsum) and calcium carbonate (calcite, the main component of limestone). These and other compounds and elements found in the WTC $PM_{2.5}$ samples are indicative of crushed building materials such as cement, concrete aggregate, ceiling tiles, and wallboard. Levels of carbon were relatively low, suggesting that combustion-derived particles did not form a significant fraction of these samples recovered in the immediate aftermath of the destruction of the towers. Gypsum and calcite are known to cause irritation of the mucus membranes of the eyes and respiratory tract.

Samples of WTC $PM_{2.5}$ induced mild to moderate degrees of inflammation when administered at a relatively high dose (100 μg) directly into the airways of mice. The pulmonary inflammatory response was not as great as that caused by the reference $PM_{2.5}$ samples (toxic ROFA and ambient air NIST 1649a). However, this same dose of WTC $PM_{2.5}$ caused airway hyperresponsiveness (a

greater sensitivity to agents which constrict breathing passages) comparable to NIST 1649a and to a greater degree than ROFA. Doses of 10 and 32 μg administered directly into the airways, or inhalation at 10 mg/m^3 , did not induce significant inflammation or hyperresponsiveness. The significant degree of airway hyperresponsiveness induced by the high dose of WTC $\text{PM}_{2.5}$ implies that components of the dust can promote mechanisms of airway obstruction.

The results from these studies indicate that a high dose of WTC dust as $\text{PM}_{2.5}$ would be necessary to elicit effects in healthy people. Hypothetical calculations are presented indicating that a healthy worker at Ground Zero would have to inhale about 425 $\mu\text{g}/\text{m}^3$ WTC $\text{PM}_{2.5}$ for 8 hours to achieve the same dose per tracheobronchial surface area as occurred with the high dose of WTC $\text{PM}_{2.5}$ used in the mouse studies. These high concentrations are conceivable in the aftermath of the collapse of the towers when rescue and salvage efforts were in effect. Therefore a healthy worker without respiratory protection could have inhaled enough WTC $\text{PM}_{2.5}$ to cause pulmonary inflammation, airway hyperresponsiveness, and manifestations of sensory irritation such as cough. Species differences in responses to inhalation of WTC $\text{PM}_{2.5}$ are unknown and were not considered in these calculations. Individuals who are especially sensitive to inhalation of dusts, such as asthmatics, may experience these effects at lower doses of inhaled WTC $\text{PM}_{2.5}$. These studies suggest that most healthy people would not respond to a single exposure to moderately high WTC $\text{PM}_{2.5}$ levels (about 130 $\mu\text{g}/\text{m}^3$ or less for 8 hours) with any adverse respiratory responses. However, it should be emphasized that the effects of chronic (long-term) or repeated exposures to lower levels of WTC $\text{PM}_{2.5}$, or the persistence of any respiratory effects are unknown and were not components of this study. Although only fine $\text{PM}_{2.5}$ was tested in these experiments, its composition was similar to coarser PM, suggesting that biological responses to both size fractions within the respiratory system may be similar. The results of these studies will need to be placed within the context of an overall risk assessment for exposures to pollutants generated by the World Trade Center disaster.

I. Introduction

The World Trade Center (WTC) disaster sparked enormous concern about the quality of the environment in the surrounding neighborhoods. One of the immediate concerns was the effect of dust from the collapse and burning of the towers on breathing, especially in more susceptible individuals. Dust infiltrated indoors into homes and apartments, in many cases up to several inches in depth. Fires at the WTC site continued for several months before finally being extinguished, and emitted significant quantities of particulate matter (PM). Recovery and reconstruction efforts have also contributed to emissions of fine (< 2.5 microns; $PM_{2.5}$), coarse (> 2.5 and < 10 microns; $PM_{2.5-10}$), and larger (> 10 microns) size PM fractions. The dust particles from the WTC site appear to be quite alkaline in nature, probably due to partial dissolution of concrete, gypsum, and glass fiber particles (USGS, 2002). As people are trying to move back, decisions must be made about cleaning procedures since potential exposure issues are associated with redispersal and residual dust.

Those moving back to their homes as well as those who work in the area have reported throat irritation, cough, and other indications of mucous tissue sensory irritation (New York Times, 2001; Washington Post, 2002). Nose and throat irritation may be caused by particles which deposit in the nasal passages and upper airways and stimulate sensory nerve reflexes (Costa and Schelegle, 1999). Airborne dust may elicit inflammation, mucus production, coughing, and sneezing in an effort to clear the lung of particles (Raabe, 1999). However, inflammation, mucus production, and airway hyperresponsiveness may all contribute to airway obstruction. Since asthma is characterized by all of these cardinal features (Sears, 1997), it is logical to suspect that asthmatic individuals may be more sensitive to agents which further promote airway obstruction.

The National Exposure Research Laboratory (NERL, USEPA), in coordination with Region 2 of the U.S. Environmental Protection Agency (USEPA or EPA) and the New York Department of Environmental Protection (NYDEP), has been monitoring ambient pollutants

including volatile organic compounds (VOCs), dioxins, and PM in an effort to ascertain exposures. In addition, New York University (NYU) and Rutgers University have collected bulk samples of ash and dust in the immediate aftermath of the disaster. The National Health and Environmental Effects Research Laboratory (NHEERL, USEPA) has collaborated with these organizations to study health effects of PM from the immediate vicinity of the WTC site.

The primary goal of the present study was to evaluate the potential health effects of PM in people working or living in the vicinity of the WTC and downwind of fires and dispersed building materials immediately after the WTC collapse. Toxicologic assessment of entrained (settled) dusts and combustion-derived PM dispersed in the areas surrounding the WTC will provide basic hazard identification information from which a broad health assessment may be derived. These findings would provide objective information to EPA, New York State, and local authorities to communicate to the public about collateral public health concerns.

In order to begin assessment of the toxicity of dust derived from the destruction of the WTC towers, scientists from NYU (led by Drs. Lung Chi Chen and Mitchell Cohen) went to the area around "Ground Zero" on September 12 and September 13, 2001. They collected bulk samples of settled dust from several sites in the immediate vicinity (< 0.5 miles). Back in their laboratories at NYU, they utilized a procedure to size-fractionate the dust to obtain both fine and coarse PM fractions which can be readily inhaled and deposit in the respiratory tract, and are therefore relevant for study of toxicological effects. On October 2, 2001, Dr. Chen contacted Dr. Daniel L. Costa of the U.S. EPA NHEERL in order to collaborate on investigations of the toxicity of these size-fractionated WTC PM samples.

The approach of the present study (code name WTC2001) was to compare the toxicity of samples of size-fractionated WTC $PM_{2.5}$ with previously tested PM samples in mice. Mice offer a number of advantages for toxicity studies: 1) less sample is needed to assess toxicity;

2) the biology of the mouse has been intensively studied in the scientific literature; 3) a wide array of mouse-specific analytical reagents is available; and 4) we have extensive experience in assessing physiological responses, inflammation, and respiratory tract injury in mice exposed to other samples of air pollutants. The WTC PM_{2.5} samples were thoroughly characterized by a number of chemical and physical techniques in order to compare the composition of the samples with other reference samples. A dose-response study in mice was conducted comparing aspirated WTC PM_{2.5} (pooled from 7 different locations near the WTC site) with low and high toxicity PM_{2.5} control samples. An acute inhalation exposure study was conducted on one WTC PM_{2.5} sample, since upper airways irritation is a primary complaint of those living and working in the WTC area. Finally, a short-term time course study was conducted comparing aspirated samples from 7 different locations with each other and with a standard PM_{2.5} sample.

Several methods were common to all three of these experiments to determine the toxicological effects of WTC PM_{2.5}. The ability of these PM_{2.5} samples to affect respiratory tract responsiveness to aerosolized methacholine was determined. Since this chemical triggers airway narrowing, the test is appropriate to determine sensitivity to agents which induce airway obstruction. Bronchoalveolar lavage is a common standard technique which quantifies numbers of inflammatory cells and levels of proteins and enzymes indicative of lung injury. Lung pathological effects were assessed in a semi-quantitative fashion in all studies, and pathological effects in the nasal region were determined in the inhalation study. Comparison of the toxicological effects of dust derived from the destruction of the WTC with PM_{2.5} samples which have been extensively characterized in the literature will be clearly beneficial and relevant to the overall assessment of health consequences of environmental pollutants related to this disaster.

II. Materials and Methods

A. WTC PM Sample Collection and Size Fractionation

On 9/12/2001 and 9/13/2001, scientists from New York University went to the WTC area to collect bulk samples of fallen dust. Using a paper scoop, bulk samples were taken from various outdoor locations (e.g. car hood, window ledge, park bench) as well as one indoor location, all of which appeared undisturbed since the collapse of the towers, as judged by the presence of a smooth uniform layer of dust and the absence of indicators of recent human activity. Thirteen samples were collected and labeled with numbers (1 - 13) on 9/12/2001, and six samples were collected and labeled with letters (A - F) on 9/13/2001. Samples were stored in 75 ml or 250 ml polystyrene flasks at room temperature. All samples were collected before rain fell on 9/14/2001, which certainly altered chemical and physical characteristics of the dust. Samples were taken back to NYU for processing to isolate different size fractions.

Bulk samples of dust were sieved with a 53 μ mesh screen (USA Standard Testing Sieves, Fisher Scientific, Pittsburgh, PA) on a shaker (Portable Sieve Shaker, Tyler Industrial Products, Mentor, OH). The sieved material (PM_{53}) was aerosolized through a 10 μ cut inlet to remove particles in the 10 - 53 μ range and isolate the PM_{10} fraction. The PM_{10} fraction then passed through a 2.5 μ cyclone (made in house) to remove the $PM_{2.5-10}$ (coarse) fraction and isolate the $PM_{2.5}$ (fine) fraction. The $PM_{2.5}$ fraction was collected on Teflon filters (Pall Gelman Sciences, Port Washington, NY - Zefluor Supported PTFE, 2 micron pore size, 47 mm, part # P5PJ047). While fractionating the PM samples, the filters became loaded and slowed airflow. Consequently, loaded filters were replaced with fresh filters periodically, and about 10 - 40 filters were used to completely size-fractionate each WTC sample. Analysis of the weights found in the 4 size fractions showed that roughly half of the sample was in the PM_{53} sieved fraction. Of the PM_{53} fraction, about 80-89% was in the 10 - 53 μ size range, which is too large to use in respiratory toxicology studies since only 45% of 10 μ

particles are even inhalable in small laboratory animals (Menache et al., 1995), and deposition of particles greater than 5 μ m is minimal (Raabe et al., 1988). The amount of the 2.5 - 10 μ fraction was very small (0.04 - 1.14 % of the PM_{53} fraction, except 3.23% in sample 13) and was therefore not feasible to study. The $PM_{2.5}$ fraction, however, was present in large enough amounts (2.29 - 4.06 % of PM_{53} fraction) to study for potential respiratory health effects, and is toxicologically relevant since it is associated with epidemiological findings of health effects in humans (Dockery et al., 1993). [The sum of the size fraction percentages does not total 100% of the original PM_{53} fraction because of loss of sample during fractionation steps.] After examination of the available inventory of filters and the locations where the samples

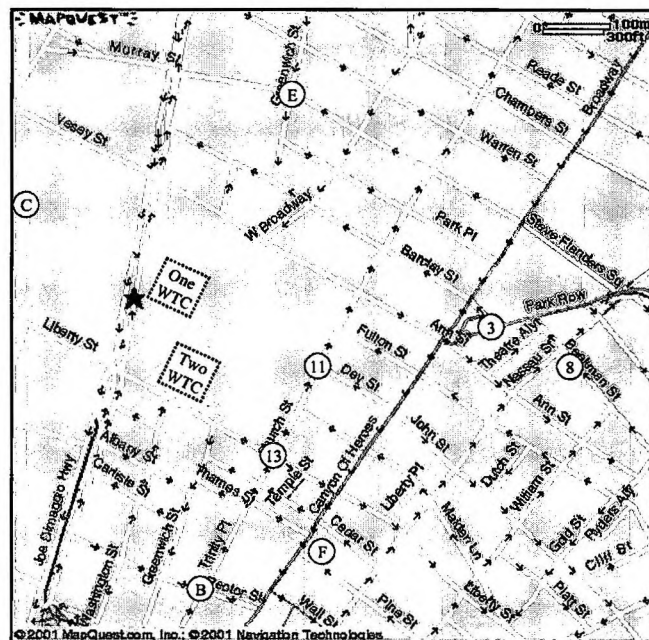


Figure 1. WTC dust samples were collected by New York University (NYU) scientists from 13 sites on 9/12/2001 (numbers) and from 6 sites on 9/13/2001 (letters). Collection sites are shown only for samples used in the WTC2001 study. Map provided by MapQuest.com, Inc.



Figure 2. WTC bulk dust samples were size-fractionated by NYU. Filters containing the $PM_{2.5}$ fraction were received at the U.S. EPA in Research Triangle Park, NC on 10/26/2001, and were inspected and photographed 10/29/2001.

were collected, filters containing the $PM_{2.5}$ fraction were selected from seven locations (sites 8, 11, 13, B, C, E, and F) around Ground Zero, in order to assess toxicity of samples from different geographical locations as well as overall toxicity of a pooled sample from these locations (Figure 1). The locations were selected to represent a distribution surrounding the WTC site, with more collection sites in the east reflecting the predominant winds in that direction.

Fourteen Teflon filters containing the $PM_{2.5}$ fraction from the 7 different sites collected around the World Trade Center on 9/12/01 and 9/13/01 were shipped by overnight express to EPA and received on October 26, 2001, and these were inspected and photographed on October 29, 2001 (Figure 2). [Throughout the WTC2001 study, sample transfers were accompanied by signed chain-of-custody letters]. A total quantity of about 50 mg from each site, collected on 1 to 3 filters per site, was provided. The weight of $PM_{2.5}$ on the filters was determined by NYU, and was separately determined at EPA after overnight dessication using a Cahn electrobalance. The description of the locations of the 7 samples and the total weight of $PM_{2.5}$ on the filters from each site is provided in Table 1. $PM_{2.5}$ could not be efficiently scraped off of one filter, so it was necessary to isolate the $PM_{2.5}$ using an aqueous extraction procedure (see below).

Throat irritation, cough, nosebleeds, and other mucous tissue/sensory irritation were reported by residents and workers in the WTC area (Washington Post, 2002). Oropharyngeal aspiration of PM bypasses the nose and therefore potentially relevant effects may go undetected. Consequently, it was decided that an inhalation exposure study should be conducted which might reveal important

information about the toxicity and mode of action of WTC $PM_{2.5}$. Since there was not enough $PM_{2.5}$ or $PM_{2.5-10}$ sample available to conduct an inhalation exposure study (> 2 g necessary), it was decided to use a PM_{53} sample (sieved but not further fractionated) which was available in large enough quantities to run through the inhalation exposure system. The EPA inhalation exposure system has a $2.5 \mu m$ cut-point cyclone to remove larger particles (Ledbetter et al., 1998), and therefore measurement of the PM concentration in the exposure zone of the chamber represents exposure to $PM_{2.5}$. A sample of PM_{53} from location #3 (figure 1), 0.3 miles east of Ground Zero (in the predominant wind direction), was available in large enough quantities for the nose-only inhalation exposure study. This sample was sent by overnight express from NYU and received on November 21, 2001.

B. Extraction of PM from Teflon Filters

Filters were extracted using a modification of a method by Biran and coworkers (1996). Each filter was handled with clean sterilized stainless steel forceps. Filters from each of the 7 individual collection sites (1 - 3 filters per site) were extracted into a single volume of distilled water (Gibco BRL ultrapure 10977-015, lot 1063705) in the ratio of 0.5 ml water per mg sample (2 mg PM / ml water; range 24.96 - 27.14 ml). This volume of water was pipetted into a 100 ml sterile plastic specimen cup containing a 3 mm thick Teflon ring at the bottom of the cup designed to support the filter. Filters were wetted with 200 μl of 70% ethanol on the particle side. The liquid was gently spread on the filter with the pipet tip, taking care not to scrape the filter. The filter was then placed on top of the 3 mm thick Teflon ring in the specimen cup with the particle side down, and a 6 mm thick Teflon ring was placed on top of the filter. The cup with the filter was secured to an orbital shaker (Titer Plate Shaker, Lab-Line Instruments, Melrose Park, IL). A cleaned sonicator probe (18 mm diameter, Sonic 300 Dismembrator, Artek Systems Corp., Farmingdale, NY) was rinsed with 1% Triton X-100 (Sigma Chemical Co., St. Louis MO; T8787), and then ultrapure distilled water (Gibco BRL ultrapure 10977-015, lot 1063705) before and after each extraction. The probe was then lowered into the water in the specimen cup to a level just above the filter. Ice was placed around the specimen cup to prevent rising temperatures during sonication, and the temperature of the water was measured before and after sonication. The shaker was turned to the lowest speed at which it would run continuously (setting = 2). The sonicator power was set to 30, and the filter was sonicated for 30 minutes while rotating on the orbital shaker. After sonication, the filter

Table 1. Description of Samples Used in WTC2001 Study

WTC Site Samples						
Experiment	Sample Code	Collection Date	Location, Description	Size Fraction	Total Weight on Filters	Extracted Wt., % Extracted
A, C	WTC 8	9/12/2001	Beekman Street - filters # 9, #14 0.4 miles E of Ground 0 center	PM _{2.5}	53.316 mg	46.70 mg 87.6%
A, C	WTC 11	9/12/2001	55 Church Street - filters #13, #14, #15 In front of Millenium Hilton Hotel 0.1 miles E of Ground 0 center	PM _{2.5}	50.097 mg	29.79 mg 59.5%
A, C	WTC 13	9/12/2001	Church & Liberty St. - filters #4, #5 0.1 miles SE of Ground 0 center	PM _{2.5}	51.006 mg	46.29 mg 90.8%
A, C	WTC B	9/13/2001	Trinity & Rector - filter #4 From a car hood and windshield 0.25 miles S of Ground 0 center	PM _{2.5}	52.969 mg	42.31 mg 79.9%
A, C	WTC C	9/13/2001	Winter Garden Park - filters #4, #7 From a park bench facing the Hudson 0.2 miles WNW of Ground 0 center	PM _{2.5}	54.285 mg	47.67 mg 87.8%
A, C	WTC E	9/13/2001	Murray & Greenwich - filters #5, #6 From a window ledge 0.25 miles NNE of Ground 0 center	PM _{2.5}	49.919 mg	45.13 mg 90.4%
A, C	WTC F	9/13/2001	Inside 120 Broadway - filters #2, #12 From a marble staircase with no footprints 0.25 miles SE of Ground 0 center	PM _{2.5}	53.600 mg	38.73 mg 72.3%
B	WTC 3	9/12/2001	23 Park Row - Ground sample in front of J&R Electronics (across City Hall Park) 0.3 miles E of Ground 0 center	PM _{<53}	21.521 g sieved material	sieved - not further fractionated
Control PM Samples						
Experiment	Sample Code	Collection Date	Description	Size Fraction	Weight Available	Extracted Wt., % Extracted
C	NIST	1976-1977	NIST Standard Reference Material 1649a (Urban Dust collected in Washington DC)	PM _{2.5}	47.984 mg	39.33 mg 82.0%
A	MSH	1980	Mt. St. Helens ash, Washington State, from Graham et al., 1985	PM _{2.5}	> 10 g	previously size- separated
A	ROFA	1994	Residual oil fly ash, ROFA Sample 3 from Kodavanti et al., 1998	MMAD: 2.665	> 2 g	milled - not extracted

was gently removed with forceps and excess liquid was drained from the filter into the cup. Filters were placed back in their petri dishes, allowed to dry, and were dessicated before reweighing to determine quantity extracted (i.e. removed) from the filters (Table 1). The suspension of PM was thoroughly mixed, the pH was determined, and 10 ml was pipetted from each of the 7 samples into a single sterilized 150 ml Erlenmeyer flask on ice to make a pooled sample (WTCX). The pH of the pooled sample was also determined. Of the remaining amount from each individual sample, 1 ml was taken for endotoxin analysis, and the remainder was pipetted into sterile 15 ml polystyrene tubes.

The flask containing the pooled sample was covered with parafilm, and the pooled and individual site samples

were frozen at -80 °C prior to lyophilization. Holes were poked in the parafilm of the pooled sample, while the caps on the 15 ml individual site sample tubes were loosened. Samples were lyophilized for 2 days at -55 °C and 140 mtorr (Virtis Company, Gardiner, NY). After lyophilization, samples were stored at 4 °C until resuspension in sterile saline on the day of use in oropharyngeal aspiration.

C. Control PM Samples Used in WTC2001 Study

In order to assess toxicity of WTC PM_{2.5}, pooled and individual site samples were compared with three other well-characterized PM_{2.5} samples. Standard Reference Materials (SRM) are extensively characterized samples available from the National Institute of Standards and

Technology (NIST, Gaithersburg, MD). SRM 1649a is an urban particulate matter sample which was collected in the Washington DC area in 1976-1977 over a 12 month period and represents a time-integrated sample (NIST, 2001). This material was selected in order to compare toxicity of WTC PM_{2.5} with other typical urban air PM_{2.5} (albeit from an earlier era when leaded gasoline was still in use). Since this material was collected as a total suspended particulate (TSP) sample with a large amount of coarse non-respirable PM, it was necessary to size-fractionate it in order to compare it with the WTC PM_{2.5} samples. Vials of NIST 1649a were purchased and then sent to NYU for size-fractionation using the same procedures as outlined above. The PM_{2.5} fraction was sent back to EPA, and NIST 1649a was extracted from Teflon filters as described above.

The toxicity of WTC PM_{2.5} was also compared to that of a PM_{2.5} fraction of ash from Mt. St. Helens (MSH) in Washington state (Graham et al., 1985). Approximately half of MSH is crystalline in nature, primarily plagioclase, a series of compounds beginning with NaAlSi₃O₈ and ending with CaAl₂Si₂O₈ which show continuous solid solution from albite to anorthite, with CaAl replacing NaSi as the series progresses. The remaining portion of MSH is amorphous (glass), while there are minor amounts of cristobalite (3%) and quartz (< 1%). The PM_{2.5} fraction of MSH has low toxicity in rats (Raub et al., 1985) and mice (Hatch et al., 1984). Since the MSH sample had already been size-fractionated (Graham et al., 1985), it was not necessary for NYU to further size-fractionate it with their system.

Residual oil fly ash (ROFA) is a fugitive fine PM sample with a high content of bioavailable transition metals including vanadium, nickel, and iron. Numerous studies by investigators at EPA and other institutions have demonstrated that these metals are associated with lung injury in both healthy animals and animal models of cardiopulmonary injury (Dreher et al., 1997; Gavett et al., 1999; Kodavanti et al., 1998; Watkinson et al., 1998). For the WTC2001 study, we chose a sample of ROFA from a boiler system which is toxic yet not as soluble in water as previous samples of ROFA (ROFA sample #3 from Kodavanti et al., 1998), and is therefore more comparable to WTC PM samples which are not extremely water-soluble. The ROFA samples in the study by Kodavanti (1998) were reduced in size by placing each sample with a stainless-steel ball in a stainless-steel cup and shaking vigorously in a ball mill shaker for 30 - 60 minutes, and then passing the sample through a 100 µ mesh nylon screen. ROFA sample #3 has a mass median aerodynamic diameter (MMAD) of 2.665 µ. Although it was slightly larger in size compared with the other samples used in the

WTC2001 study, it was decided that further size fractionation at NYU was not necessary. Control PM samples were stored at room temperature in polystyrene or polypropylene tubes shielded from light. See Table 1 for the summary descriptions of control PM samples.

Samples of WTC PM, NIST, MSH, and ROFA were characterized by scanning electron microscopy / energy dispersive X-ray (SEM/EDX), X-ray diffraction (XRF), X-ray fluorescence (XRD), carbon fraction analysis, pH and endotoxin analysis, inductively coupled plasma-mass spectrometry / atomic emission spectrometry (ICP-MS / ICP-AES), and ion chromatography (IC).

D. Physical and chemical analysis of solid (bulk and filter) samples.

1. Scanning electron microscopy / energy-dispersive x-ray (SEM/EDX) analysis. SEM/EDX was used to obtain physical and chemical characteristics of particles and fibers found in bulk WTC2001 and control dust PM_{2.5} samples, and on polycarbonate filters taken during an inhalation exposure using the WTC3 sample. The Personal SEM® (PSEM) (formerly R. J. Lee Instruments, Ltd., now Aspex Instruments, Trafford, PA) was used to conduct the manual, single-particle analyses. The PSEM is a digital SEM/EDX system equipped with secondary and backscattered electron detectors for imaging, and a thin-window EDX detector enabling X-ray detection of carbon and heavier elements. For bulk samples, a small amount was applied to an adhesive carbon tab affixed to an aluminum SEM stub. For filter samples, small pieces (less than 1 cm²) were affixed to aluminum SEM stubs using a carbonaceous suspension. Images were created using the backscattered electron mode, which enhances the contrast of metals and other heavy elements with the background carbonaceous medium compared with lighter element particles. Photomicrographs of the individual features provide particle morphology and approximate physical size; an x-ray spectrum displayed below the image provides information on the elemental composition of the feature. SEM/EDX analysis was performed by the National Exposure Research Laboratory, Research Triangle Park, NC. Since only 15-30 images were examined from each sample, the results should not be interpreted as quantitative or comprehensive (Mamane et al., 2001). Rather, these qualitative results were primarily used to determine consistency with other analytical techniques described below.

2. X-ray diffraction (XRD) analysis. XRD was used to determine qualitatively whether any crystalline compounds were present in sufficient quantity to be identified in the WTC3 sample used in the inhalation

exposure study. The bulk solid PM_{2.5} sample was side-drifted into an aluminum holder and mounted into the Siemens D-500 Diffractometer (Bruker Analytical X-Ray Systems, Madison, WI). The generator, set at 45 kilovolts (kV) and 40 milliamperes (mA), generated x-rays from a copper-target x-ray tube. The filament current was 3.46 mA. Intensities were collected by a lithium-drifted silicon detector fitted with a monochromator riding on a goniometer in the coupled $\theta/2\theta$ mode. Peaks were collected in the range $2\theta = 5$ to 85 degrees. Collection software used was Materials Data, Inc. (MDI, Livermore, CA) Datascan, Version 3.2. Evaluation software was MDI Jade 5 using the pattern library Powder Diffraction File (PDF), release 2000 (International Centre for Diffraction Data). XRD analysis was performed by the National Risk Management Research Laboratory, Research Triangle Park, NC.

3. X-ray fluorescence (XRF) analysis. Five polycarbonate filters (Isopore 0.8um #ATTP04700, Millipore Corporation, Bedford, MA) loaded with the WTC3 PM_{2.5} sample used in the inhalation exposure study, along with five lot-matched blank filters, were loaded into liquid-type polyethylene sample cups, placed in a stainless steel sample holder, and analyzed. No film was used to cover blanks or samples in the analysis. X-ray intensities were collected with the Philips PW2404 XRF (Philips Analytical, Inc., Natick, MA), and the loaded particulate was analyzed using the "standardless" software, UniQuant 4, after subtraction of the counts due to the blank filter system (includes filter, polyethylene liquid sample cup and stainless steel sample holder). The intensities were averaged in each channel needed for background subtraction. The blank filter analysis showed slightly elevated counts due to Fe, Cr, Cu, Ca, Cl, S, and Si. The constituents of the dust were evaluated as oxides, but are reported quantitatively as elements with the oxygen stripped. XRF analysis was performed by the National Risk Management Research Laboratory, Research Triangle Park, NC.

4. Carbon fraction analysis. Carbon fraction analysis was used to speciate the carbon content of samples into organic, elemental, and carbonate carbon. Analysis was performed on bulk WTC2001 and control dust PM_{2.5} samples, and on quartz filters taken during an inhalation exposure using WTC3 PM_{2.5}. The thermo-optical method, based upon sequential pyrolytic vaporization and detection of the three carbon fractions (Birch and Cary, 1996; Sunset, 2002), was performed by Sunset Laboratory, Forest Grove, OR (bulk samples), and Hillsborough, NC (filter samples).

E. Chemical analysis of liquid extracts of bulk and filter samples.

1. pH. The pH of samples isolated by aqueous extraction was determined immediately after the extraction procedure with an audited calibrated Corning 440 pH meter (audited by Research Triangle Institute, Research Triangle Park, NC).

2. Endotoxin. Aliquots of samples isolated by aqueous extraction were frozen on dry ice and sent by overnight delivery to Associates of Cape Cod, Inc. (Falmouth, MA) for analysis of endotoxin content using the Limulus Amebocyte Lysate (LAL) gel-clot method. LAL-reagent water (lot # 308-331) was used to reconstitute or dilute Pyrotell lysate, endotoxin, and samples, and served as the negative control. Samples were titrated using a twofold dilution scheme against control standard endotoxin (CSE; lot #85, Escherichia coli O113, 5 EU/ng). Preliminary inhibition tests (positive product controls) were performed on the undiluted samples spiked with CSE equivalent to twice the sensitivity (λ ; 0.03 EU/ml). The error of the gel-clot method is $\pm$ one twofold dilution.

3. Inductively coupled plasma - atomic emission spectrometry (ICP-AES) and - mass spectrometry (ICP-MS). WTC2001 PM_{2.5} samples, control dust PM_{2.5} samples, and polycarbonate filters taken during an inhalation exposure using WTC3 PM_{2.5} were extracted with deionized (d.i.) water or 1M HCl, and analyzed for their elemental content. The two extraction liquids are used to estimate easily bioavailable and total bioavailable metal content, respectively. While this speciation scheme is a rough approximation of bioavailability, it has proved useful in characterizing inhalation toxicology endpoints for various source and ambient particulates (Costa and Dreher, 1997; Kodavanti et al., 1998). Milligram-sized aliquots of bulk samples were extracted with 1.6 ml of either liquid. Polycarbonate filters were extracted with 13 ml of either liquid. High-speed centrifugation was used to separate the liquid and solids (17000 x g for 1.6 ml samples, 51000 x g for 13 ml samples). After dilution, extraction solutions were analyzed quantitatively using ICP-AES (Model P40, PerkinElmer Instruments, Shelton, CT) operated closely following EPA Method 200.7 (EPA, 2002a), and ICP-MS (ELAN 6000, PerkinElmer Instruments, Shelton, CT) operated closely following EPA Method 6020 (EPA, 2002b). Blank Gelman Teflo and Millipore Isopore filters (used in the inhalation study) were run through the extraction procedure. Filter blanks levels for all elements were negligible compared to the levels in the PM samples. Gelman Zefluor and Teflo filters and Millipore Isopore filters are of known, similar

low background levels. These filters are all produced for air particulate sampling and are commonly used for chemical analysis since their background chemical levels are negligible relative to the mass of samples amounts in this study. ICP-AES and ICP-MS analyses were performed by the National Health and Environmental Effects Laboratory, Research Triangle Park, NC.

4. Ion chromatography (IC) of deionized water extracts. Deionized water extracts from the ICP sample prep as described above were analyzed quantitatively for anion and cation content using IC (DX-500, Dionex, Sunnyvale, CA). The AS14 column was used for anion analysis and the CS12 column was used for cation analysis. IC analysis was performed by ManTech Environmental, an onsite contractor for the National Exposure Research Laboratory, Research Triangle Park, NC.

F. Experimental Animals and Weight Randomization.

Young adult (7 week old) female CD-1 mice (an outbred strain) were obtained from Charles River Breeding Laboratory (Crl:CD-1® (ICR) BR) in Raleigh, NC or Portage, MI (the latter used in Experiment A5 only). An outbred strain was chosen because results from any specific inbred strain might be applicable only to that strain. CD-1 mice were selected since researchers in the Experimental Toxicology Division of the U.S. EPA have extensive experience with this strain, while females were chosen for convenience so that they could be housed together in groups corresponding to treatment. The health screening report of mice from the colony accompanied each shipment of animals and was evaluated to determine if there were pathogens detected in the colony which could potentially affect responses. In all shipments, no pathogens were detected which could affect respiratory responses. Mice were housed in plastic cages on beta-chip bedding in groups of 4 per cage in room JJ-4 of the animal colony of the Environmental Research Center, Research Triangle Park, NC. Food (Prolab RMH 3000) and water were provide *ad libitum* and cages were changed at least twice a week. Mice were maintained on a 12 hr light/dark cycle at approximately 22 °C and 50% relative humidity in our AAALAC-approved facility, and held for a minimum of 5 days before treatment. Monthly sentinel screens were negative for sendai, mouse hepatitis virus, mycoplasma pulmonis, CARbacillus, parvovirus, endo- and ecto-parasites, and pinworms. Protocols used in this study were reviewed and approved by the EPA Institutional Animal Care and Use Committee (Laboratory Animal Project Review number 02-03-003 with amendments), and were conducted using national guidelines for the care and

protection of animals.

In all experiments, mice were randomly assigned to exposure groups based on weights. The weight randomization program (RandomVB) was developed in-house, validated, and documented in operating procedure OP-NHEERL-H/ETD/IEG/97/18/01 (Animal randomization using a personal computer). The program takes all animal weights and ranks them from lowest to highest. A group mean and standard deviation is calculated for all animals. The number of animals per group and the number of groups is entered. The numbers of animals available at 1, 2, or 3 standard deviations (SD) are calculated. The user then selects the lowest SD which contains the required number of animals for the study. All outliers are eliminated. Additional animals are then eliminated to fit into the required number for the study. Animals are then randomized by weight into the required groups. All animals are accounted for and reasons why they were not selected are displayed. The weight randomization program was used to identify the groups of 4 mice which are housed together in a single plastic cage. Within each cage, mice were individually identified by 1 to 4 marks applied to the base of the tail with a Sharpie permanent ink marker (Sanford, Bellwood IL). Different experimental groups were identified with different colors (e.g. saline control mice - green marks, etc.). In addition the cage cards were marked to identify the experimental group. Marks remained evident for at least two days which was long enough to identify mice at 24 hr termination points. In cases where mice were killed more than 2 days after the initial marking, the tails were remarked where necessary because of excessive fading. Mice were weighed at the time of randomization, again immediately before exposure if randomization occurred before the day of exposure, and whenever one group of mice was killed.

G. Toxicological Endpoints: Experimental Design.

The toxicity of WTC PM_{2.5} samples was assessed in three separate experiments, designated Experiments A, B, and C. Experiment A was designed to study the dose-response characteristics of the pooled sample of WTC PM (WTCX) in comparison with ROFA (toxic control), MSH (low toxicity control), and saline vehicle control. Experiment B was designed to study the responses associated with nose-only inhalation exposure of WTC3 PM_{2.5} in comparison with the responses of mice exposed to air only. Experiment C was designed to compare toxic responses of WTC PM_{2.5} from individual sites with each other and with NIST 1649a.

In all experiments, a group size of 8 was selected

based on scientific judgement and experience with the typical variability of collected data (except the first part of experiment A, where $n = 12$; see Results - Experiment A - BAL parameters for explanation). Endpoints were analyzed in a total of 388 mice in all 3 experiments. In general, the endpoints were chosen to assess pulmonary function impairment, lung injury and inflammation, and pathological manifestations of respiratory tract injury. Experiments utilizing oropharyngeal aspiration (A and C) were emphasized over inhalation experiments (B) for several reasons: 1) the quantities of samples available for study were generally limited, and aspiration requires much less material (10-100 mg) than inhalation (10 g preferred); 2) aspiration delivers a precise quantity of PM to the lung at a specific time point, while the inhaled dose is more difficult to predict or quantify; 3) inhalation exposure studies are labor intensive and therefore fewer comparative analyses of the WTC PM_{2.5} could be accomplished in the available time frame compared with studies utilizing oropharyngeal aspiration, and 4) oropharyngeal aspiration (equivalent to intratracheal instillation) is specifically recommended in evaluation of panels of test materials for their relative potential to produce toxicity (Driscoll et al., 2000).

1. Experiment A. In 5 sub-experiments, groups of female CD-1 mice were exposed to pooled WTC PM_{2.5} sample X (10, 31.6, or 100 μg), MSH (100 μg), ROFA (10 or 100 μg), or saline vehicle control by oropharyngeal aspiration on day zero. The dose of 31.6 μg represents the half-log difference between 10 and 100 μg (i.e. $10^{1.5} = 31.6$). The high dose of 100 μg was selected based on our experience that at this dose nearly all PM samples will induce at least a mild inflammatory or physiological response; any sample that does not induce any response at all at this dose can be judged to possess low toxicity. Doses higher than 100 μg in the mouse may be of questionable relevance due to the potential for artifactual local inflammatory responses in response to bolus administration (Driscoll et al., 2000). Four mice per sample group were tested within each sub-experiment ($n = 28$ per sub-experiment; total experiment A: $n = 140$).

In sub-experiments A1, A2, and A5 (total $n = 12$ mice per sample group), airway responses to aspiration of the PM samples was assessed by comparison of breathing parameters just before and after aspiration (see method below). On day 1, diffusing capacity of the lung for carbon monoxide (DLCO) was assessed, and mice were then killed and bronchoalveolar lavage (BAL) fluid cells, proteins, and enzymes were recovered and quantified to assess lung injury and inflammation.

In sub-experiments A3 and A4 ($n = 8$ mice per sample

group), airway responsiveness to methacholine (Mch) aerosol was determined on day 1. Mice were then killed, and lungs were removed and fixed for histopathological assessment. Airway hyperresponsiveness to nonspecific bronchoconstrictive agents such as Mch is a primary feature of asthma (Sears, 1997) as well as reactive airways dysfunction syndrome (RADS) which develops after high-level occupational exposure to irritant gases, fumes, or smoke (Gautrin et al., 1999). Induction of this condition by PM in nonallergic normal mice can be considered as a marker of respiratory tract injury.

2. Experiment B. Two groups of female CD-1 mice were exposed in nose-only inhalation exposure tubes one time to a PM_{2.5} sample (WTC3) or air only for 5 hr ($n = 48$ per exposure group; total experiment B: $n = 96$). This WTC3 sample was derived from a sieved but not previously fractionated PM_{2.5} sample of WTC3 by aerodynamic size-separation during exposure. Although some irritant responses are transitory and therefore would be best measured during exposure (Costa and Schelegle, 1999), we do not currently possess the recently developed technology (e.g. Buxco Electronics, Sharon, CT or CH Technologies, Westwood, NJ) which allows some respiratory parameters to be measured during nose-only exposures. Therefore, breathing parameters were compared just before and after inhalation exposure ($n = 12$ per group). On days 1, 3, and 6 after the exposure, 16 mice from each group were assessed for DLCO and BAL parameters ($n = 8$) or responsiveness to Mch aerosol and lung and nasal histopathology ($n = 8$).

3. Experiment C. In 2 sub-experiments, groups of female CD-1 mice were exposed by oropharyngeal aspiration to 100 μg of PM from one of 7 individual WTC sample sites, to 100 μg of NIST 1649a (referred to as NIST hereafter), or to saline vehicle only. In sub-experiment C1, mice were exposed to WTC8, WTC13, WTCF, NIST, or saline. In sub-experiment C2, mice were exposed to WTC11, WTCB, WTCC, WTCE, or saline. On days 1 and 3, mice were assessed for responsiveness to Mch aerosol, BAL parameters, and lung histopathology ($n = 8$ per group per time point, except saline sub-experiment C2: $n = 4$ per time point; total sub-experiment C1: $n = 80$, total sub-experiment C2: $n = 72$, total experiment C: $n = 152$).

H. Oropharyngeal Aspiration of PM Samples.

A Sartorius model AC211S analytical balance (Edgewood, NY; audited by Research Triangle Institute, Research Triangle Park, NC) was used to weigh PM samples for oropharyngeal aspiration. The operation of the balance was tested by weighing calibrated weights

before and after weighing samples each day (Class U calibrated weights, Denver Instrument Company, Arvada, CO). PM samples were allowed to come to room temperature from 4 °C before weighing. Sterile 2.5 ml glass vials or 5 ml polystyrene snap cap vials were used to weigh and resuspend PM samples. Vials weights were tared, and a sterilized stainless steel spatula was used to transfer sample to the weighing vial, and the sample was weighed. The sample was then resuspended with sterile saline (Sigma S-8776 single use vials, lot 128H2310) using a calibrated Rainin Pipetman at a concentration of 2 mg/ml. All mice aspirated a volume of 50 µl. Samples were vortexed and used straight (100 µg dose) or diluted as necessary (to 0.632 mg/ml or 0.2 mg/ml for 31.6 µg or 10 µg doses, respectively). All samples were sonicated for 2-4 minutes at 22 °C (Branson model 3210R-DTH, Danbury, CT) prior to oropharyngeal aspiration.

Mice randomized into different exposure groups were anesthetized in a 2.7 L plexiglass chamber by passing house air through an aerator containing methoxyflurane (Metofane; Mallinckrodt, Mundelein, IL). The vapor induced rapid anesthesia, at which time the mouse was taken out of the chamber and placed on an aspiration platform. The tongue was gently pulled back and held with a forceps, and 50 µl of PM suspension or saline alone was pipetted in the back of oropharyngeal region using a 200 µl tip. The tongue was held until the animal was forced to aspirate the sample, and placed back in its cage. Mice recovered within 5 or 10 minutes of this procedure. This technique is equivalent to intratracheal instillation in deposition efficiency (Foster et al., 2001), and several publications describe experiments in which it was successfully used (e.g. Dreher et al., 1997, Gavett et al., 1999, Kodavanti et al., 1998).

I. Nose-Only Inhalation Exposure.

In order to assess the effects of WTC PM_{2.5} on upper respiratory tract responses, mice were exposed to WTC3 or air only in two separate nose-only inhalation exposure chambers. The exposures were conducted for 5 hours in 52-port nose-only flow-by inhalation chambers (Lab Products) on November 27, 2001. The exposure time was based on practical considerations of the tasks involved on the exposure day. The WTC3 sample was a tan powder received in a plastic jar, and was desiccated at room temperature prior to use. Preliminary exposures were conducted on several days prior to exposure to set exposure parameters, which indicated that an aerosol concentration of 10-15 mg/m³ could be achieved. The control chamber and the WTC3 chamber had similar flow rates (~ 12 L/min) and received air from the same source.

The aerosol was generated using a unique exposure system which conserves sample by using a carpenter's chalk line to pick up particles from a small Tygon tube dust reservoir (illustrated in Ledbetter et al., 1998). The dust is carried out through an orifice and blown off the string in a discharge head with a high velocity air jet. The particles are carried through a particle charge neutralizer and 2.5 µ cut-point cyclone to remove particles larger than PM_{2.5}, and finally enter the inlet of the nose-only chamber.

Nose-only exposure tubes were constructed from 50 ml polypropylene centrifuge tubes with the bottom end removed. Mice were randomized into exposure groups as described above, and 49 in each group were placed in exposure tubes (1 extra per group in case any mice died during the exposure). Mice were not acclimated to the tubes prior to exposure, since stress may be an important component of the response to WTC PM. In order to measure immediate airway responses to air or WTC3 sample and also to handle the large number of mice, the control air exposure was begun 1 hour before the WTC3 exposure.

Dust concentration was determined gravimetrically on 5 Teflon filters (45 mm diameter with 1 µ pore size) taken at a sample flow rate of approximately 0.24 L/min. The filters were weighed just prior to and after sampling using a Cahn C-30 balance housed in a controlled temperature and humidity enclosure. Real-time PM concentration was achieved with an aerosol monitor (Dust Track, TSI Inc., St. Paul, MN) on the chamber exhaust. The particle size was determined gravimetrically using a Mercer Cascade Impactor (Intox Products, Albuquerque, NM). On November 29, 2001, the exposure system was restarted and 7 Teflon and 3 polycarbonate filters were taken for chemical analysis. On December 17, 2001, 18 polycarbonate filters (9 WTC3 and 9 blanks) were collected for chemical analysis and 3 quartz filters were collected for carbon analysis.

J. Respiratory Responses Assessed by Whole Body Plethysmography.

1. Immediate Airway Responses to PM_{2.5} Exposure.

Exposure to PM_{2.5} by oropharyngeal aspiration or by inhalation may result in immediate changes in breathing parameters. Breathing parameters in unanesthetized unrestrained mice were assessed in a 12-chamber whole body plethysmograph system (Buxco Electronics, Sharon, CT). The animal chambers have a pneumotach on the roof to measure pressure (which is proportional to air flow) relative to the pressure in a reference chamber vented to the atmosphere. The breath by breath signals are taken by the program software to compute respiratory rate

(frequency, f , breaths / min) and other parameters including enhanced pause (PenH). Although PenH is at best an indirect measure of flow resistance, it does correlate well with lung resistance and reflects changes occurring during bronchoconstriction (Hamelmann et al., 1997), although other responses such as mucus production may increase PenH. The convenience of rapidly measuring respiratory parameters in twelve mice at once was a major consideration in utilizing this technique rather than the double plethysmograph or tracheotomized ventilated methods which allow direct measures of airways resistance and compliance, but are time and labor intensive. A protocol was written to record and average baseline measurements of mice in calibrated chambers for 10 min, pause for oropharyngeal aspiration (or stop during inhalation exposure), and then resume recording measurements for one hour. The time between oropharyngeal aspiration and monitoring of responses was approximately 6 minutes, while about 20 minutes was needed after inhalation exposure to remove mice from exposure tubes, weigh them, and transport them to the plethysmograph chambers. PenH was automatically calculated by the software (and confirmed by examination of random data) using expiration time (T_e), relaxation time (RT), and peak expiratory and inspiratory flows (PEF, PIF) according to the following expression: $\text{PenH} = [(T_e - \text{RT})/\text{RT}] \times [\text{PEF}/\text{PIF}]$. Examination of the data after exposure showed that utilization of the first 10 or 15 minutes of the data was not more sensitive in detecting changes in respiratory parameters than the entire hour of post-exposure monitoring, and therefore responses over the whole post-exposure hour were utilized and averaged. The percent change in f and PenH after exposure to PM was expressed as $[(\text{Post-value} - \text{Pre-value}) / \text{Pre-value}] \times 100\%$.

2. Airway Responsiveness to Methacholine Aerosol.

Airway responsiveness to increasing concentrations of aerosolized methacholine (Mch) was measured in mice in calibrated chambers. After measurement of baseline PenH for 5 minutes, saline or Mch in increasing concentrations (4, 8, 16, 32, and 64 mg/ml) was nebulized through an inlet of the chamber for 1 min. The aerosol drier was automatically turned on immediately after the aerosolization period for 2 min. Measurements of PenH and other parameters were continued for an additional 1, 2, 3, 4, 8, and 12 minutes after saline or increasing doses of Mch, for a total time of 4, 5, 6, 7, 11, and 15 minutes (0, 4, 8, 16, 32, 64 mg/ml Mch, respectively). One minute pause periods between aerosolizations allowed time to change solutions for nebulization. After subtracting baseline values from responses to saline or Mch, the area

under the curve (PenH AUC; PenH - sec) for these recording intervals was calculated using the trapezoid method.

K. Diffusing Capacity of the Lung for Carbon Monoxide.

The ability of the lungs to allow diffusion of gases (O_2 , CO_2) across the alveolar-capillary barrier is dependent on physical properties of the gases and the alveolar-capillary membrane, and may be limited by perfusion or diffusion (Levitzky, 1995). Diffusion limitation may be caused by thickening of the alveolar-capillary barrier (e.g. by interstitial or alveolar edema). Diffusion of carbon monoxide (CO) is limited only by its diffusivity in the barrier and by the surface area and thickness of the barrier. The diffusing capacity of the lung for CO (DLCO) is therefore a useful test of the integrity of the alveolar-capillary membrane (Levitzky, 1995).

To determine DLCO rapidly and increase sensitivity from individual mice, 4 mice were placed together in a single 7.8 L bell jar associated with a Pharmacokinetic Uptake System (consisting of an oxygen monitor, flow meter, pump, pressure gauge and transducer, mass flow controller, and computerized data collection and control system). Approximately 6.6 ml of research grade CO (99.99%) was injected into the system. The initial concentration of CO in the chamber was approximately 700 ± 10 ppm. CO concentrations were taken every 15 seconds (Bendix Model 8501-SCA CO Analyzer), and continued for approximately 10 minutes. Temperature, humidity, airflow, pressure, and oxygen were monitored during the test. The DLCO is expressed as the slope of the fitted line of [CO] vs. time (ppm/min).

L. Bronchoalveolar Lavage (BAL).

Mice were anesthetized with urethane (1.5 g/kg i.p.) and killed by exsanguination via severing the renal artery. The trachea and lungs were exposed and a 20 g catheter was sutured into the trachea. Mice were lavaged with two aliquots of Ca^{2+} , Mg^{2+} , and phenol red-free Hanks' balanced salt solution (HBSS; 35 ml/kg, Life Technologies, Bethesda, MD). Approximately 85% of the total instilled volume was recovered in all treatment groups. The BAL fluid was maintained on ice and centrifuged at $360 \times g$ for 10 minutes at 4°C . Supernatants were transferred to a separate tube in order to prepare aliquots for biochemical analyses. BAL cells were resuspended in 1 ml of HBSS and counted (Coulter Z1, Hialeah, FL). Cytospin preparations of BAL cells were made for each sample and stained with Wright's Giemsa using an automated slide stainer (Hematek 2000,

Elkhart, IN). Cell differentials were performed by one person (SHG) counting 500 cells per slide. After lavage, the lungs were removed and stored at -80°C for future assays (to be determined).

Assays for total protein, albumin, lactate dehydrogenase (LDH) and N-acetyl- β -D-glucosaminidase (NAG) are routine measures of lung injury (Henderson et al., 1985) and were carried out on an aliquot of BAL supernatant as previously described using a Cobas Fara II centrifugal spectrophotometer (Gavett et al., 1997). Four other BAL supernatant aliquots were prepared from each sample; one of these was supplemented with 10% fetal bovine serum to prevent loss of cytokines and other proteins in low protein concentration fluids, and the 4 aliquots from each sample were stored at -80°C . These samples are available for analysis of cytokines and other proteins (to be determined).

M. Histopathology

1. Lung histopathology. In experiments A and B, mice which were tested for Mch responsiveness were subsequently assessed for lung histopathology, while in experiment C, all mice were tested for Mch responsiveness and were lavaged before assessment of lung histopathology. Mice were anesthetized with urethane and killed as described above for BAL. Lungs were removed and fixed by tracheal perfusion in a fume hood with ice cold 4% paraformaldehyde at 25 cm pressure for 15 minutes. The trachea was then tied off and placed in a vial of 4% paraformaldehyde at 4°C . After 24 hours, the lungs were drained and placed in phosphate buffered saline at 4°C .

The lungs were transferred to Experimental Pathology Laboratories (Research Triangle Park, NC), where fixed lungs were processed to paraffin blocks, sectioned at an approximate thickness of $5\ \mu$, placed on glass slides and stained with hematoxylin and eosin (H&E). Longitudinal coronal sections were cut on a lateral plane to include mainstem bronchi for viewing a maximal amount of lung area. Two additional unstained lung sections were prepared for future use. Histopathologic observations for individual animals in each experiment were tabulated, and the degree of severity of inflammatory changes and the presence of PM-related pigment were graded on a scale of one to five (1 = minimal, 2 = slight/mild, 3 = moderate, 4 = moderately severe, 5 = severe/high). The pathologist knew which animals comprised a group, which group was the saline or air-exposed control group, the day after treatment, and the doses given to the experimental groups, but did not know the identities of the individual PM samples other than by a unique number or letter.

2. Nasal histopathology. Dr. James Wagner of Michigan State University (MSU) instructed EPA personnel in this procedure, utilized on mice from the nose-only inhalation exposure (experiment B). Immediately after death, the head of each animal was removed from the carcass and both nasal passages were fixed by slowly flushing retrograde through the nasopharynx with 1-2 ml 4% paraformaldehyde. The nasal cavity was then immersed in a large volume of the fixative for at least 24h until further processing. The fixed nasal cavities were placed in 0.1 M PBS (pH 7.2, 4°C) and shipped overnight to Dr. Wagner at MSU. Nasal cavities were decalcified in a 13% solution of formic acid for 5 days, and then rinsed in distilled water for 1h. After decalcification, three transverse tissue blocks of the nasal cavity, cut perpendicular to the hard palate, were selected for light microscopic analysis. The first tissue block was sectioned from the proximal aspect of the nasal cavity immediately posterior to the upper incisor tooth (T1). The second transverse tissue block was taken at the level of the incisive papilla (T2) and the third and most distal tissue block was taken at the level of the second palatal ridge (T3). The tissue blocks were embedded in paraffin, and $6\ \mu\text{m}$ -thick sections were cut from the anterior surface of each block. Sections were histochemically stained with hematoxylin and eosin for morphologic identification of nasal tissues. Nasal tissues (three sections/mouse) from a total of 48 mice tested for Mch responsiveness (8 mice/exposure group/time point) were microscopically examined by Dr. Jack Harkema (MSU). Nasal lesions were graded on the following scale: 1 = minimal, 2 = mild, 3 = moderate, and 4 = marked inflammation.

N. Statistical Analysis

All statistical analysis were done using SAS procedures (Cary, NC). There were generally three types of responses collected: 1) PenH responses recorded repeatedly for each animal as area under the curve (AUC) for various concentration exposures to Mch; 2) responses to DLCO were analyzed from a single response from 4 animals; and 3) individual measurements measured once for each animal as a univariate variable. Experimental designs varied with each experiment and each part of an experiment. Statistical designs used were replicated completely random designs for experiment A. Crossed-designs were used for experiment B and C involving treatments (TRT) and days (DAY). Randomized block designs were used for DLCO experiments.

When initial multivariate repeated measures analysis of variance (MANOVA) test showed significant interactions between dose of Mch and TRT or DAY in the

airway responsiveness studies, univariate linear regression was used in all subsequent tests. The models used in these regression studies were analysis of covariance (COV) with tests for parallelism for each TRT and DAY combination. In experiment A, natural logarithms were used for both Mch concentration (C) and PenH AUC responses. The linear regression $\text{Log (PenH AUC)} = a_1 + b(\text{Log C})$ reduces to a power function of the form $\text{PenH} = a_2 \cdot C^b$. No logarithms were used in Experiments B and C due to several negative values resulting after baseline adjustments. Techniques similar to ordinary stepwise regression were used in COV analyses. Overall test of parallelism of regression lines was done first. Subgroups of the TRT and DAY combinations were determined to get subgroups exhibiting a common slope. Within a subgroup with a single slope, subsequent tests were done to determine if the means were different (using individual contrast tests).

Body weight was determined repeatedly for animals. Due to animals being removed and killed, numbers of mice in each group were different on different days after exposure. For days when few animals weights were determined, univariate analysis of variance (ANOVA) was used to test for TRT effects. For those days where most of the weight data occurred, MANOVA techniques were used for statistical tests.

For each TRT and DAY combinations with a univariate response, a determination was made if the variances could be considered homogeneous. If the

variance ratios were greater than 10-fold then all of the responses were ranked from smallest to largest across all TRT and DAY combinations. Then ranks replaced the original responses for the univariate ANOVA. Sometimes the variances of the ranks for TRT and DAY combinations still indicated heterogeneity. Then additional judgment was used to help insure that this heterogeneity of variance did not affect the overall conclusions. In experiment A, a replicate was called DAY. When replication was shown to have no significant contribution in the ANOVA results, DAY was not included in subsequent ANOVAs. For responses with many "zero" values, the residuals from the ANOVA were plotted and analyzed by univariate techniques to determine if the residuals generally met the assumptions required for ANOVA. When interactions between TRT and DAY occurred, these were pointed out and in some cases further ANOVA were done for each DAY. When ranks were used for the response, the ranks were regenerated for each day separately. When TRT was significant, follow-up comparisons of means were done using Tukey's multiple comparison tests.

The statistical tests examined only whether groups were significantly different from each other. In the reporting of the results, for the sake of brevity, groups are sometimes referred to as having significantly greater values than other groups. These statements should be read as groups are significantly different from each other, and the mean of one group is greater than the mean of another.

III. Results

A. Chemical analysis of solid samples and liquid extracts.

1. Endotoxin and pH levels. The pH of water-extracted WTC PM_{2.5} ranged from 8.88 in WTCE to 10.00 in WTC8 (Table 2). The alkaline pH is consistent with previous reports of WTC PM (USGS, 2002) and probably results from the building materials comprising much of the dust (see below). The pH of lyophilized WTC PM_{2.5} reconstituted in unbuffered saline was very close to neutral, while MSH was very slightly acidic and ROFA was moderately acidic (average 3.74 at 2 mg/ml). It is not known why the pH of WTC PM_{2.5} should be close to neutral after reconstitution in saline; perhaps the salt neutralizes a basic component of the extract. Endotoxin levels in WTC PM_{2.5} samples were minimal in comparison with other urban PM samples such as NIST 1649a, which was also low (Table 2). Several thousand times this level of endotoxin caused an acute neutrophilic response in the lungs of CD-1 mice (Dhingra et al., 2001). The level of endotoxin in the samples used in this study would not be anticipated to contribute directly to any inflammatory response in the lungs.

2. Elemental and Ion Analysis. The ICP data showed that water-soluble calcium and sulfate content amounted to 56-63% of the WTC PM_{2.5} samples (Table 3). In general, the elemental and ion compositions were consistent among the different samples tested. ICP data for the 1M HCl-soluble extracts of WTC PM_{2.5} showed an additional 1-2 weight percent calcium content. This increase may be attributed to calcite or other water-insoluble calcium salts which are soluble in 1M HCl (see below for data on compound analysis). There was no evidence of stainless steel contamination from the forceps used to handle the WTC PM filters or from the stainless steel balls used to size-fractionate the ROFA sample.

The ICP results for the aerosolized PM_{2.5} cut fraction of WTC3 generally agree well with those determined by XRF (Table 3). Calcium content of acid-extracted WTC3 was somewhat lower by ICP (20-22%) than calcium content of solid WTC3 by XRF (26.6%). This may reflect an incomplete extraction in the one hour timeframe of

sample preparation method for ICP, or the presence of other insoluble forms of calcium in the WTC3. The XRF values are higher for most other elements, which reflects the incomplete dissolution of the WTC3 matrix by the weak (water) and moderate (1M HCl) extraction liquids. Elements such as magnesium and zinc, which exist in compounds more amenable to acid dissolution, agree more closely (Weast, 1985; Budavari, 1996). Elements such as aluminum, iron, and titanium, which are in the form of

Table 2. Endotoxin and pH Levels of PM Samples after Water Extraction and Resuspension in Saline.

Sample Code ^a	pH in water	Endotoxin ^d EU/ml	Inhibition	pH in Saline ^e
Water ^b	5.28			
WTC 8-100	10.00	0.50	none	
WTC 11-100	9.16	0.25	none	
WTC 13-100	9.47	0.50	none	
WTC B-100	9.54	0.25	none	
WTC C-100	9.32	0.50	none	
WTC E-100	8.88	0.25	none	
WTC F-100	9.55	0.50	none	
NIST-100 ^c	4.20	25	none	
Saline				6.67
WTCX-10				7.38
WTCX-31.6				7.38
WTCX-100	9.35			7.36
MSH-100				6.61
ROFA-100				3.74

^a WTCX indicates pooled sample of WTC8, WTC11, WTC13, WTCB, WTCC, WTCE, and WTCF. "-100" indicates 100 µg/50 µl dose = 2 mg/ml. "-31.6" indicates 31.6 µg/50 µl dose = 0.632 mg/ml. "-10" indicates 10 µg/50 µl dose = 0.2 mg/ml. Water-extracted PM samples were lyophilized and resuspended in sterile saline.

^b Water used to extract filters.

^c NIST Standard Reference Material 1649a (Washington DC TSP PM).

^d Endotoxin levels measured as endotoxin units (EU) per ml water extract. Samples were tested for inhibition of the endotoxin assay (none was detected).

^e Average of 3-4 measurements.

Table 3. Elemental and Ion Analysis of WTC2001 Samples ^a

Sample: Diluent: No. Analyses:	WTC 3			WTCX (pooled)		WTC B	MSH		ROFA		NIST
	DI H ₂ O	1M HCl	none	DI H ₂ O	1M HCl	DI H ₂ O	DI H ₂ O	1M HCl	DI H ₂ O	1M HCl	DI H ₂ O
	4	4	5	2	1	1	2	1	2	1	1
Analyte											
SO ₄ ²⁻	<u>376488.6</u>	<u>344439.8</u>	375300	<u>439120.3</u>	<u>379429.4</u>	<u>432570.7</u>	<u>955.2</u>	<u>973.4</u>	<u>274962.0</u>	<u>242277.8</u>	<u>86725.3</u>
Ca	<u>184904.4</u>	<u>218019.2</u>	265600	<u>187493.7</u>	<u>196745.9</u>	<u>183794.1</u>	<u>351.5</u>	<u>1970.8</u>	<u>18590.8</u>	<u>19663.0</u>	<u>12649.5</u>
Si			30000								
Al	<u>1346.3</u>	<u>4072.2</u>	9930	<u>555.7 (*)</u>	<u>1476.8</u>	<u>537.1 (*)</u>	<u>48.4 (*)</u>	<u>1260.4</u>	<u>6739.8</u>	<u>8604.3</u>	<u>1049.1</u>
Mg	<u>1112.9</u>	<u>5414.1</u>	6550	<u>651.5</u>	<u>2257.6</u>	<u>354.1</u>	<u>37.3</u>	<u>1250.2</u>	<u>24895.8</u>	<u>24655.2</u>	<u>1140.7</u>
Fe	<u>150.0 (*)</u>	<u>2450.4</u>	6290	<u>6.7 (*)</u>	<u>1098.5</u>	<u>6.0 (*)</u>	<u>-0.1 (*)</u>	<u>1833.4</u>	<u>763.0</u>	<u>19512.4</u>	<u>407.5</u>
Cl ⁻	2851.0		3330	2699.0			1103.0		302.0		1164.0
K			2690								
Zn	22.7	1413.1	1760	13.4	410.1	4.1	-0.0 (*)	3.7	<u>6555.9</u>	<u>5932.3</u>	503.9
Ti	12.1	180.9	1450	5.2	41.6	4.0	1.3	17.6	2.6	137.5	1.8
Na ⁺	1290.0		725	1139.0			721.0		44179.0		2153.0
NO ₃ ⁻	938.0			6496.0			0.0		0.0		7390.0
PO ₄ ³⁻	0.0		779	0.0			0.0		0.0		3317.0
F ⁻	406.0			799.0			0.0		352.0		466.0
Pb	2.2	141.3		0.7	33.4	0.4	0.0 (*)	0.5	17.9	789.6	1378.7
Mn	4.5	107.9		1.8	24.0	0.7	3.8	38.4	365.0	458.6	77.2
Cu	5.5	76.0		6.4	21.8	4.7	0.6	6.8	572.8	628.4	83.6
Ba	25.6	75.6		8.7	31.0	7.2	0.1 (*)	5.0	3.0	57.5	11.2
Sb	17.7	43.8		9.4	17.4	7.0	0.0 (*)	-0.0 (*)	1.5	146.5	3.5
NO ₂ ⁻	38.0			0.0			0.0		0.0		0.0
Mo	1.0	33.7		1.8	3.2	4.9	0.1	0.1	0.0 (*)	339.3	1.8
Ni	2.6	19.7		4.1	3.1	1.8	0.0	0.9	<u>17027.7</u>	<u>16988.2</u>	32.2
Sn	0.8	12.1		1.3	3.1	0.1 (*)	0.0 (*)	0.0 (*)	0.0 (*)	51.0	0.1 (*)
Cr	10.0			1.4 (*)		1.2 (*)	0.1 (*)		6.1		6.1
Cd	0.3	7.2		0.7	1.9	0.3	0.0	0.0	8.9	11.9	19.9
Be	-0.0 (*)	2.7 (*)		0.3 (*)	0.2 (*)	-0.1 (*)	0.0 (*)	0.0 (*)	1.4 (*)	1.7 (*)	0.1 (*)
Co	0.2 (*)	1.6		0.8	0.9	0.1 (*)	0.0 (*)	0.4 (*)	495.0	510.4	2.1
As	1.3	-10.4 (*)		1.1	-0.4 (*)	0.3	0.1	0.2	1.7	106.4	13.5
Tl	0.1 (*)	-25.5 (*)		0.5	-1.2 (*)	0.1 (*)	0.0 (*)	-0.1 (*)	0.0 (*)	-0.2 (*)	0.3
V									<u>1748.6</u>	<u>35693.6</u>	
NH ₄ ⁺	0.0			0.0			0.0		0.0		25355.0
Total µg/g:	569632	576475	704404	639019	581598	617299	3223	7362	397593	376566	143953
% Total Mass:	57.0	57.6	70.4	63.9	58.2	61.7	0.3	0.7	39.8	37.7	14.4

^a Results shown are average values for number of analyses indicated, expressed as µg soluble analyte / g solid sample extracted, for deionized (DI) water and 1M HCl extractions. Analyte concentrations were determined by ICP-MS, except underlined values which were determined by ICP-AES, bold values which were determined by ion chromatography, and column labelled none, where solid sample was analyzed by XRF (indicated by heavy solid-line box). (*) Value below detection limit. Analytes are arranged in order of decreasing content in WTC 3 sample, by whatever analysis provided highest content.

complex oxides usually in combination with silicon, are much less soluble under the acid extraction conditions used in this study, and do not agree as well (Weast, 1985; Budavari, 1996). The weight-percent ratios of silicon, aluminum, magnesium, and iron are in the proportion of those found in portland cement, a major component of

concrete (NIST, 2002; McKetta, 1978).

Comparison of the water-soluble transition and heavy metal content of the WTC PM samples with the control dusts shows the overall metal level trend as Mt. St. Helens < WTC2001 < NIST < ROFA. ROFA has high levels of water-soluble transition metals including vanadium,

Table 4. Carbon fraction analysis of PM Samples in WTC2001 Study ^a

Sample:	WTC3	WTCX (pooled)	WTC B	MSH	ROFA	NIST SRM 1649a
% Carbon Fraction:						
Organic	6.88	0.93	2.11	0.06	1.31	10.82
Elemental	0.31	0.00	0.01	0.07	13.63	15.10
Carbonate	1.39	0.60	0.35	0.00	1.32	0.00
Total	8.58	1.53	2.47	0.13	16.26	25.92

^a Organic, elemental, and carbonate carbon fractions were analyzed as described in text. Results are expressed as percent of total mass of sample.

nickel, and iron which are important in its toxicity (Kodavanti et al., 1998). The 1M HCl-soluble metal content trend is Mt. St. Helens < WTC2001 < ROFA (not enough NIST sample was available to run the test).

3. Carbon analysis. The WTC2001 samples had low total carbon content, in the range of 1.5-8.5% (Table 4), in comparison with control samples such as NIST (26%) and ROFA (16%). MSH had almost no carbon, as expected from this crustal PM sample. The WTC3 sample used in the inhalation study had about 4 times as much carbon as

Table 5. XRD Analysis of Compounds Present in WTC 3 Sample ^a

ICDD Number	Formula	Mineral Name	Relative Amount
05-0586	CaCO ₃	Calcite	Major
33-0311	CaSO ₄ ·2H ₂ O	Gypsum	Major
41-0224	CaSO ₄ ·0.5H ₂ O	Bassanite	Minor
46-1045	SiO ₂	Quartz	Minor

^a Analysis showed about half crystalline materials (50.6% above background), and the remainder was amorphous. After smoothing and subtracting background, evaluation software (MDI Jade 5) was used to match patterns with library available from International Centre for Diffraction Data Powder Diffraction File, release 2000.

the other two WTC samples. This result may be due to differences in the method by which the samples were isolated (physical separation vs. aqueous extraction and lyophilization) or may simply be due to variability in carbon content of samples from different locations. Despite the variation in total carbon content of WTC PM samples, the ratios of elemental, organic, and carbonate carbon were similar. Elements not listed in Table 3 or 4 (~30% of total mass) are likely O and H from adsorbed water and O, H, and N from organic or inorganic compounds.

4. Compound analysis by XRD. XRD analysis of WTC3 PM_{2.5} (before size segregation by the inhalation exposure system) showed a complex pattern containing 25 peaks, indicating the presence of several crystalline materials. The peak area above the background curve was 50.6%. The 49.4% below the curve indicated that WTC3 consisted of about half amorphous materials. Four patterns were identified as being consistent with peaks identified in the dust. Figure 3 shows the XRD spectra of WTC3 and those of the matched compounds. Two compounds were identified as major constituents (calcium carbonate (calcite) and calcium sulfate dihydrate (gypsum)), and two were identified as minor constituents (bassanite and quartz, Table 5). The XRD data are

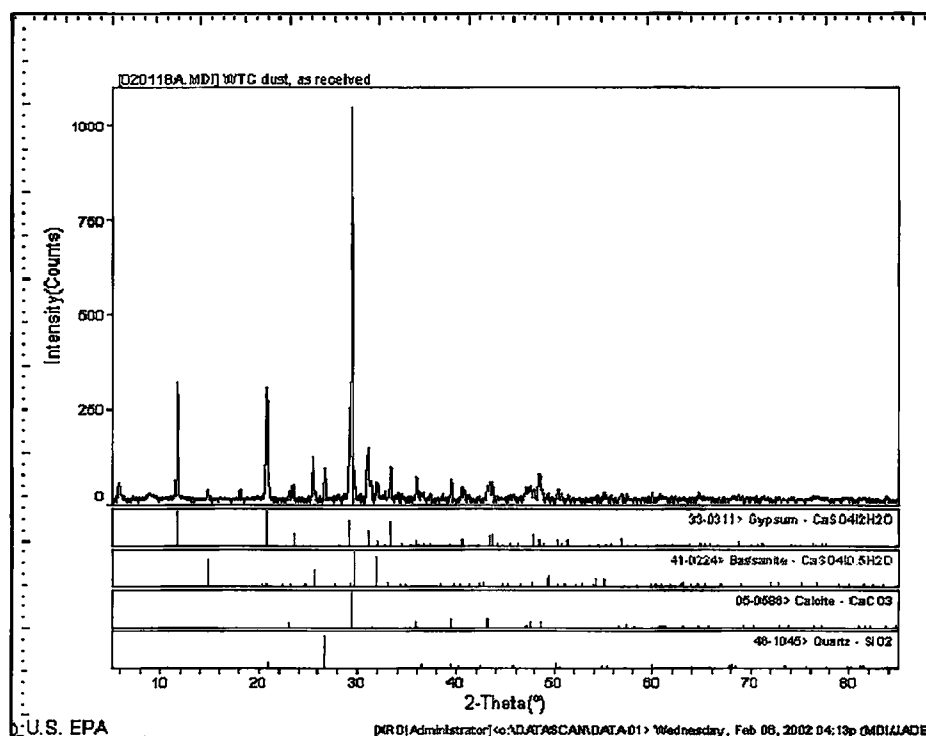


Figure 3. X-ray diffraction (XRD) analysis of WTC 3 sample (PM_{2.5}) used in nose-only inhalation exposure study (Experiment B). Peaks were collected in the range, 2θ = 5 – 85°. Collection software used was Materials Data, Inc. Datascan, version 3.2

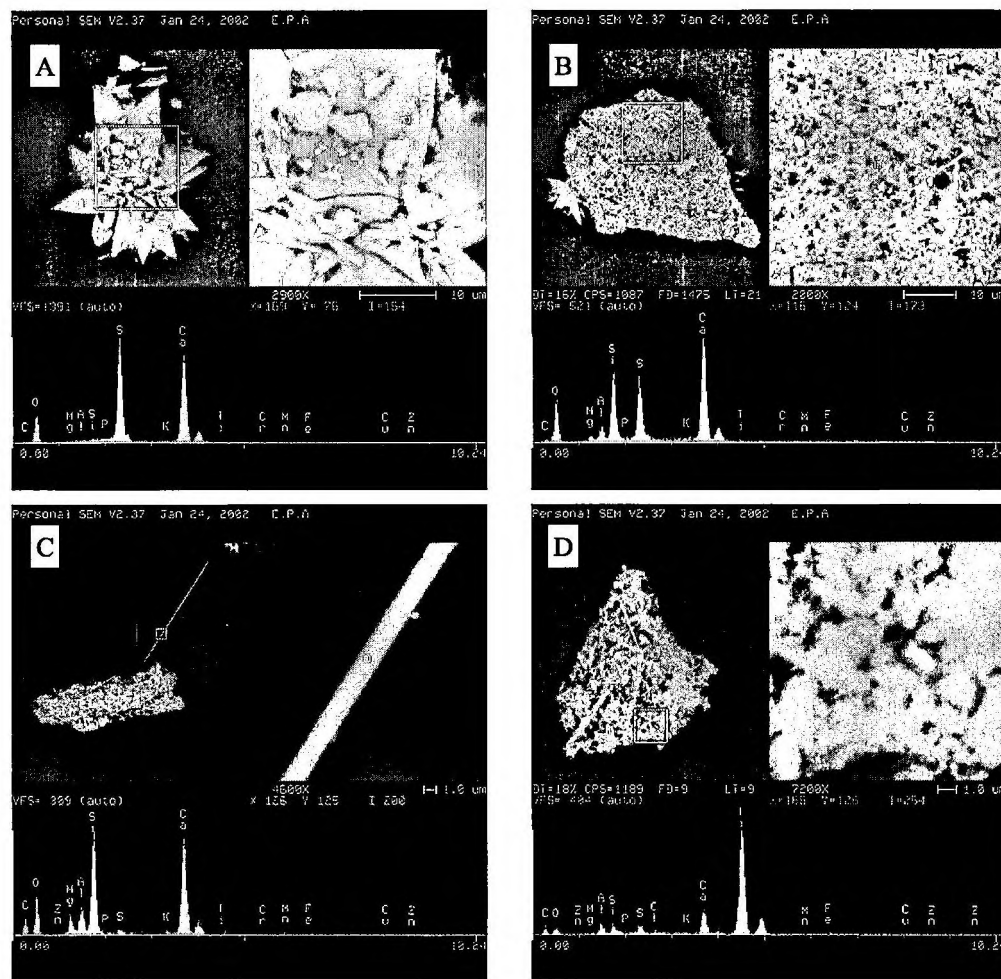


Figure 4. SEM/EDX results from water-extracted lyophilized WTC PM samples. The upper-left quadrant of each photomicrograph shows a field of view with the particle of interest within the smaller square in that field. The upper-right quadrant shows a zoomed-in view of the feature (the area from within the square in the upper-left quadrant), and the lower half shows the elemental spectrum acquired with the electron beam centered on the small (barely visible) square in the zoomed-in view. A. Example of Ca-S crystal which dominated the samples. B. Example of fine particle aggregate which was prominent in the samples. C. Example of fiber found in the samples. D. Example of metallic particle within fine particle aggregate.

consistent with the ICP data which show water-soluble calcium and sulfate in the same proportions as gypsum. Gypsum is completely water-soluble at the solid/liquid ratio of the extraction conditions used in the ICP analysis, while calcite is not water-soluble. The sample of MSH was also analyzed by XRD and the results were consistent with those previously reported (Graham et al., 1985; data not shown).

5. SEM/EDX analysis. Water-extracted and lyophilized WTC PM samples were dominated by snowflake-like crystals composed of calcium and sulfur (Figure 4A). Aggregates of fine particles composed of various combinations of Mg, Al, Si, S, and Ca were also

prominent (Figure 4B). Fibers approximately 1 μm in diameter were found in most of the samples and had a composition similar to the fine particle aggregates (Figure 4C). Metallic particles (mostly Ti and Fe, though Zn, Pb, Ba, and Cu were also found) were found typically as inclusions in the large fine particle aggregates (Figure 4D). The crystals and aggregates were likely not original to the bulk sample but were formed as a result of the aqueous extraction process.

SEM/EDX analysis of the aerosolized $\text{PM}_{2.5}$ cut fraction of WTC3 showed the same overall chemistry as the extracted and lyophilized WTC PM samples: the majority of particles were composed of Ca or Ca-S, some

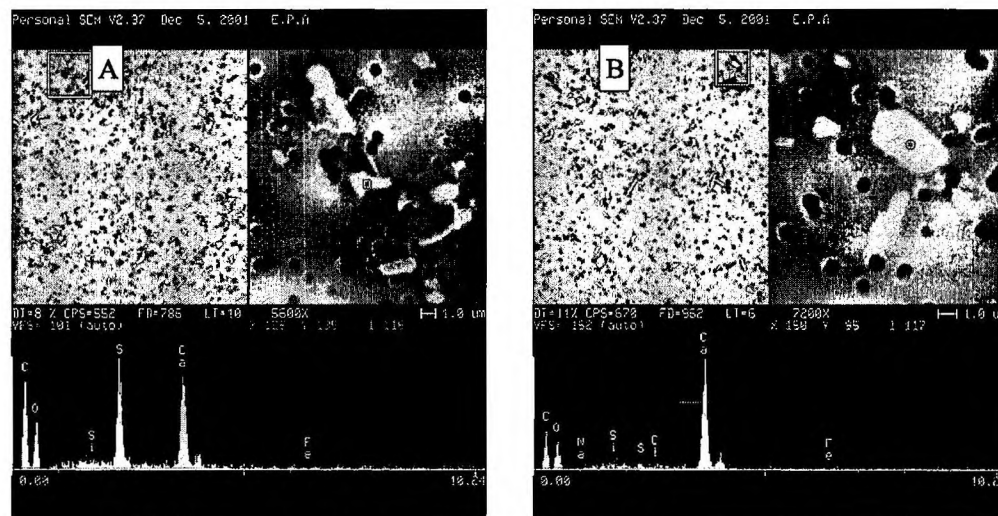


Figure 5. Particle types found in the WTC3 sample used in the nose-only inhalation exposure (Experiment B). A. Example of Ca-S particle which was prominent in the sample. B. Example of Ca particle which was prominent in the sample.

also containing Si. Some representative particles are shown in Figures 5A and 5B. In contrast to the crystals and aggregates of the bulk solid samples as described above and shown in Figure 4, the particles of WTC3 are small, typically about 1 μm , with rough, irregular features. The different form of the Ca-based particles in WTC3 reflects the dry size segregation of the inhalation exposure system. Particles with other compositions were found with far less frequency. These included particles composed of Fe, C, and Sb-Zn (one example found). One

or more possible asbestos fibers (Mg-Si composition) were also found, however polarized light microscopy rather than SEM/EDX is the preferred method for identifying asbestos. SEM/EDX analysis was also performed on MSH, NIST 1649a, and ROFA and showed results typically found in previous analyses (data not shown).

6. Summary. WTC PM samples consist primarily of construction materials from the fallen-down WTC buildings. The bulk of the WTC PM samples are calcium-based compounds, specifically calcium sulfate (gypsum)

Table 6. Experiment A: Body Weights and Immediate Airway Responses. ^a

Group	B.Wt. d 0 B.Wt. d 1		Breathing Frequency (min^{-1})			PenH (unitless)		
	g	g	Pre-	Post-	% increase	Pre-	Post-	% increase
Saline	25.8	24.9	492.3	348.1	-29.7	0.73	0.96	23.9
	0.4	0.4	11.5	25.6	4.2	0.08	0.17	8.3
MSH-100	25.5	25.0	474.0	320.6	-31.7	0.92	1.25	40.5
	0.4	0.3	13.5	23.0	5.2	0.13	0.18	14.8
ROFA-10	25.6	24.3	492.0	343.5	-29.7	0.74	1.05	42.3
	0.4	0.6	14.3	18.6	4.1	0.11	0.17	13.5
ROFA-100	25.2	25.2	461.0	307.0	-33.4	0.88	1.51	76.4
	0.5	0.4	11.9	18.7	3.7	0.10	0.20	18.6
WTCX-10	25.2	24.3	467.2	322.4	-31.1	0.85	1.14	52.4
	0.6	0.5	14.7	22.5	4.1	0.15	0.18	21.8
WTCX-31.6	25.3	24.7	476.8	348.5	-26.3	0.86	1.14	26.8
	0.5	0.5	15.5	18.9	4.3	0.18	0.34	15.6
WTCX-100	25.6	25.1	486.8	325.3	-33.1	0.79	1.11	40.7
	0.6	0.4	14.1	26.6	5.1	0.08	0.16	11.8

^a Values shown are means (in bold) and SEM immediately below means ($n=12$ per group). Body weight (B. Wt.) was measured in the morning. Respiratory parameters were measured immediately before (Pre-) and after (Post-) oropharyngeal aspiration of dust samples or saline on day 0. Values within solid-line boxes indicate significantly greater values in ROFA-100 mice vs. Saline mice ($P < 0.05$).

and calcium carbonate (calcite). Together these salts compose about two-thirds of WTC PM_{2.5} on a weight percent basis. Given the prevalent use of gypsum in ceiling tiles and wallboard, and the ease with which these building materials can be crumbled into dust, the high gypsum content is reasonable. Elemental analysis indicates that the other main components of WTC PM are construction materials such as cement and concrete aggregate. The elemental composition of WTC PM_{2.5} was consistent with that of sieved unfractionated WTC PM_{2.5} (as WTC3, Table 5). Carbon and metal content of the WTC samples were low, as expected from crustal-derived building materials (McKetta, 1978). A more complete chemical and physical analysis of dust samples has recently been reported by the U.S. Geological Survey (USGS, 2002). In that study, dust samples were collected from undisturbed locations within a 1 km radius of the WTC site on September 17 and 18, 2001 (after the rain of September 14, 2001). The present report generally agrees with the findings of the USGS study, including the alkaline nature of the WTC PM extracts.

B. Experiment A: Dose-Response Relationships of WTC PM_{2.5}

1. Body weights and immediate airway responses.

Table 7. Experiment A: Diffusing Capacity of the Lung for Carbon Monoxide (DLCO) ^a

Sub-experiment:	A1	A2	A5		
Date:	11/6/01	11/8/01	12/6/01	Mean	SEM
Treatment Group				n = 3	
Saline	-3.474	-3.610	-4.510	-3.865	0.325
MSH-100	-3.734	-3.944	-4.338	-4.005	0.177
ROFA-10	-3.904	-3.597	-3.928	-3.810	0.107
ROFA-100	-4.015	-3.089	-4.293	-3.799	0.364
WTCX-10	-2.759	-3.811	-4.301	-3.624	0.455
WTCX-31.6	-4.051	-3.338	-4.014	-3.801	0.232
WTCX-100	-3.551	-4.433	-4.299	-4.094	0.275

^a Diffusing capacity of the lung for carbon monoxide was determined one day after exposure on four mice from each treatment group, placed together in a single bell jar, in order to rapidly assess DLCO and reduce individual variability. Values shown are slopes of chamber [CO] vs. time (ppm/min), after subtraction of value from empty chamber. No significant differences among any treatment groups were detected.

Mice were exposed by oropharyngeal aspiration with PM_{2.5} samples of pooled WTC sample X (10, 31.6, or 100 µg), MSH (100 µg), ROFA (10 or 100 µg), or saline on day zero. In sub-experiments A1, A2, and A5, immediate airway responses were determined on day 0, and DLCO

Table 8. Experiment A: BAL Parameters (Day 1). ^a

Group	BAL Cell Number (x 10 ⁴)				Protein	LDH	Albumin	NAG
	Mac	Eos	Neut	Lym	µg/ml	U/L	µg/ml	U/L
Saline	18.80	0.02	0.23	0.10	155.2	29.8	21.8	2.2
	4.05	0.02	0.16	0.02	6.8	2.1	1.2	0.4
MSH-100	27.78	0.07	0.78	0.18	168.8	27.8	22.3	2.0
	5.96	0.03	0.21	0.05	8.8	1.7	1.2	0.4
ROFA-10	27.36	0.00	0.21	0.18	157.9	32.3	20.8	3.1
	2.70	0.00	0.05	0.07	5.3	1.2	0.9	0.4
ROFA-100	28.69	0.16	13.18	0.42	279.5	93.2	39.2	7.9
	3.98	0.06	2.44	0.09	16.8	10.3	2.8	1.2
WTCX-10	22.28	0.00	0.09	0.14	153.7	30.4	20.8	1.9
	4.03	0.00	0.02	0.04	4.3	1.8	0.8	0.3
WTCX-31.6	31.36	0.01	0.37	0.21	160.2	33.6	21.7	1.8
	7.73	0.01	0.14	0.03	6.3	1.6	1.3	0.2
WTCX-100	20.48	0.06	1.43	0.23	161.4	33.7	21.3	2.3
	1.73	0.03	0.24	0.04	4.8	2.1	1.0	0.3

^a Values shown are means (in bold) and SEM immediately below means (n=12 per group). Bronchoalveolar lavage (BAL) cell numbers and proteins were recovered 1 day after exposure. Cell types shown are macrophages and monocytes (Mac), eosinophils (Eos), neutrophils (Neut), and lymphocytes (Lym). Total protein, lactate dehydrogenase (LDH), albumin, and N-acetyl-β-D-glucosaminidase (NAG) were measured in BAL fluid supernatant. Values within solid-line boxes indicate significantly greater values in ROFA-100 mice vs. Saline mice (*P* < 0.05). Values with dashed-line boxes indicate significantly greater values (*P* < 0.05) compared with Saline mice (excluding ROFA-100 data which generally had much larger variances than other groups).

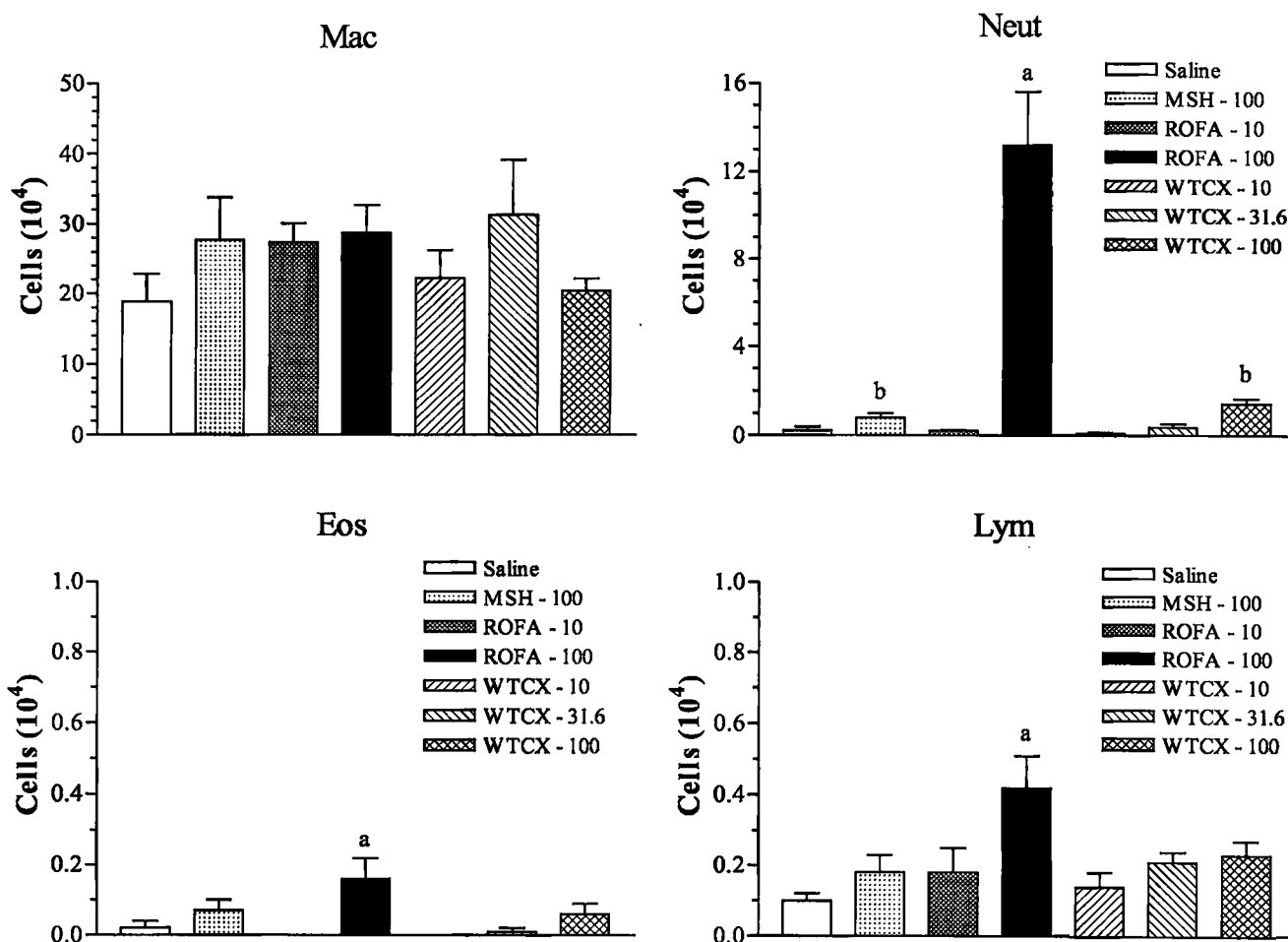


Figure 6. Experiment A. Bronchoalveolar lavage cell numbers recovered from mice one day after exposure by intratracheal instillation to PM samples in saline or saline vehicle alone. Values shown are means and SEM (n=12 per group). Cell types shown are macrophages and monocytes (Mac), eosinophils (Eos), neutrophils (Neut), and lymphocytes (Lym). ^a $P < 0.05$ vs. Saline group. ^b $P < 0.05$ vs. Saline group (comparison of rank values) after exclusion of ROFA-100 data which had much larger variances than other groups.

and BAL parameters were determined on day 1. There were no significant differences in body weights of the seven groups on day 0 or day 1 (Table 6). Ventilatory parameters were assessed in mice immediately before and after exposure. There were no differences among groups in breathing frequency, but mice exposed to the 100 μ g dose of ROFA (ROFA-100) had a significant increase in PenH immediately after exposure in comparison with saline control mice (Table 6). There were no significant changes in immediate responses in mice exposed to any dose of WTCX.

2. DLCO. The diffusing capacity of the lung for carbon monoxide was determined 24 hr after oropharyngeal aspiration on groups of 4 mice from the same exposure group together in the testing chamber. There were no significant differences in DLCO among any

of the groups of mice, which would be indicated by a slower uptake of CO and a reduced slope (Table 7). These data indicate that none of the PM samples caused injury severe enough to significantly reduce gas exchange at the alveolar-capillary barrier.

3. BAL parameters. Bronchoalveolar lavage parameters were determined immediately after testing for DLCO. Originally, we planned to do just two sub-experiments for this part of Experiment A. However, we noted that there was significant variation in the total cell numbers recovered from the 4 saline control mice in each of sub-experiments A1 and A2 (average of 7×10^4 vs. 33×10^4 , respectively). There was no evidence of any infection (in both cases 99% of BAL cells recovered from control mice were alveolar macrophages (AMs)), mice came from the same shipment in the same week, and no

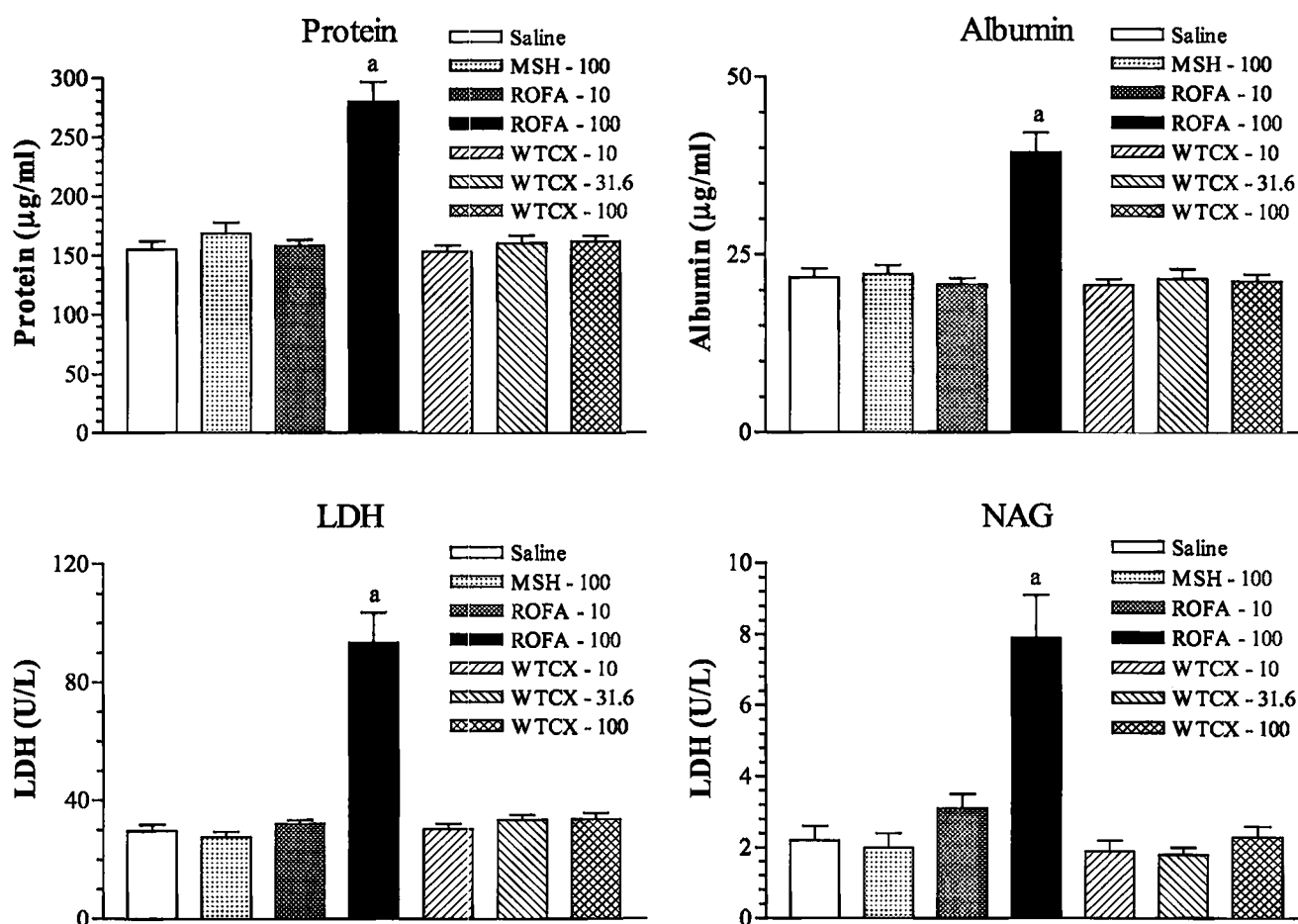


Figure 7. Experiment A. Values for total protein, lactate dehydrogenase (LDH), albumin, and N-acetyl-b-D-glucosaminidase (NAG) were measured in bronchoalveolar lavage fluid supernatants recovered from mice one day after exposure by intratracheal instillation to PM samples in saline or saline vehicle alone. Values shown are means and SEM (n=12 per group). ^a *P* < 0.001 vs. Saline group.

other reason could be deduced for the difference. Consequently, we performed a third sub-experiment to examine these endpoints and increase the number of mice per group (sub-experiment A5). The average total cell number recovered from saline control mice in A5 was 18×10^4 (97% AMs) - about in the middle between A1 and A2. All data shown is combined from the 3 sub-experiments. Due to the high variance of data in the ROFA-100 group, we judged that it was necessary to compare ROFA-100 data alone vs. saline control data. Other comparisons were made between the saline control group and the other groups after excluding ROFA-100 data. Significant increases in neutrophils, eosinophils, and lymphocytes were found in ROFA-100 mice compared with saline control mice (Table 8). Neutrophils comprised 31% of total BAL cells in ROFA-100 mice (Figure 6). After excluding the ROFA-100 data, significant differences in neutrophil numbers were found between the

saline control group and both the MSH-100 group and the WTCX-100 group (Table 8, Figure 6; *P* < 0.05). Neutrophils comprised about 7% of total BAL cells in the WTCX-100 group, but only about 1% or less in the WTCX-31.6 and WTCX-10 groups.

Levels of proteins and enzymes were measured in the BAL supernatant to assess lung damage. Both total protein and albumin are increased after damage to the alveolar epithelial barrier (Henderson et al., 1985). Lactate dehydrogenase (LDH) is a cytoplasmic enzyme which is released by dead or dying cells, while N-acetyl-β-D-glucosaminidase (NAG) is indicative of lysosomal enzyme release (Henderson et al., 1985). All of these parameters were significantly increased in the ROFA-100 group in comparison to saline control mice (Table 8). Total protein and albumin were both increased about 80% compared to saline, while LDH was increased 3-fold and NAG almost 4-fold (Figure 7). No significant changes in BAL proteins

Table 9. Experiment A: Body Weights, Baseline PenH, and Responsiveness to Methacholine Aerosol

Group	B.Wt. d 0 (g)	B.Wt. d 1 (g)	Baseline PenH	Dose Mch (mg/ml) and PenH AUC (PenH - sec)					
				0	4	8	16	32	64
Saline	24.78	23.30	0.97	0.5	39.1	54.0	142.3	309.0	1249.9
	0.42	0.65	0.13	0.5	8.4	8.9	28.5	30.6	360.7
MSH-100	24.82	23.80	1.10	2.4	36.4	99.2	177.9	437.9	1173.3
	0.51	0.45	0.12	0.3	5.5	22.5	23.7	54.3	358.3
ROFA-10	24.99	24.55	0.81	15.5	42.4	114.6	205.5	432.4	1182.5
	0.43	0.45	0.10	3.2	8.9	11.3	56.1	88.5	294.2
ROFA-100	24.86	24.60	0.91	6.1	36.7	118.2	242.1	642.2	2190.9
	0.44	0.45	0.11	6.3	4.1	34.1	37.2	94.0	706.4
WTCX-10	24.96	24.44	0.92	18.5	43.9	88.3	169.1	281.1	968.4
	0.38	0.28	0.11	6.4	11.6	16.2	52.3	35.5	129.9
WTCX-31.6	24.56	23.73	0.85	15.3	38.3	55.2	114.4	249.2	923.3
	0.46	0.51	0.11	3.4	4.8	8.8	7.0	39.8	174.2
WTCX-100	24.68	24.14	1.04	9.5	67.4	208.3	397.3	2265.0	4009.1
	0.44	0.42	0.12	8.2	11.3	47.6	61.4	260.7	580.1

* Values shown are means (in bold) and SEM immediately below means (n=8 per group). Body weight (B. Wt.) was measured in the morning (no significant differences were found). No significant differences were found in baseline PenH (enhanced pause; unitless) on day 1. Methacholine aerosol (Mch) was then administered (see Methods for details) at the indicated doses, and the airway response was calculated as the area under the curve (AUC) of the PenH response over time in seconds. See Figure 8 for description of statistical analysis of PenH AUC data.

and enzymes were found in any of the other PM exposure groups relative to saline controls. In general, the inflammatory response in the WTCX-100 group can be considered to be quite mild considering the fairly high dose.

4. Responsiveness to methacholine aerosol. In sub-experiments A3 and A4, the same groups of mice were exposed and the same time points were examined as described for sub-experiments A1, A2, and A5, but different endpoints were examined. There were no differences in body weights on day 0 or day 1 among the 7 groups of mice (Table 9). On day 1, there was no difference in baseline PenH values (immediately before Mch aerosol) among the 7 groups. Responsiveness to increasing concentrations of Mch aerosol was assessed and quantified by integrating the area under the PenH - time curve (PenH AUC; Table 9). In order to assess overall responsiveness and account for variability, power function equations were fit to the PenH AUC vs. [Mch] data for each group (Figure 8). The analysis showed that the Saline, MSH, ROFA-10, WTCX-10, and WTCX-31.6 groups could all be modeled with a common power function exponent (1.157). It is important to note that once the lines were determined to come from groups with a common exponent, the lines for these 5 groups were fit simultaneously, resulting in fitted equations that did not fit

as well as an individual line would fit the group-specific data. The responses to saline or individual doses of Mch aerosol are not as important as the fitted line describing the groups. Among these 5 groups, ROFA-10 mice had a small but significant increase in the coefficient of the equation vs. the Saline group ($P = 0.03$). The ROFA-100 and WTCX-100 groups could be modeled with a power function with a significantly different exponent (1.471; $P = 0.001$) vs. the common exponent of the other 5 groups, indicating that these 2 groups are hyperresponsive compared with the other 5 groups. In addition, the coefficient for the WTCX-100 group was significantly different from and greater than that of the ROFA-100 group ($P = 0.0001$), showing that mice exposed to the 100 μg dose of WTCX were more reactive to Mch than the ROFA-100 group.

5. Lung histopathology. Following tests for airway responsiveness to Mch aerosol, mice from sub-experiments A3 and A4 were killed and assessed for pathological changes in the lungs. No remarkable findings were observed in the lungs of the saline control group (Table 10). In both the MSH-100 and ROFA-100 groups, focal subacute bronchiolar inflammation was found at similar incidences and average severity, which was minimal (average score: MSH-100 = 0.8; ROFA-100 = 1.0). The ROFA-10 group had a lower average severity

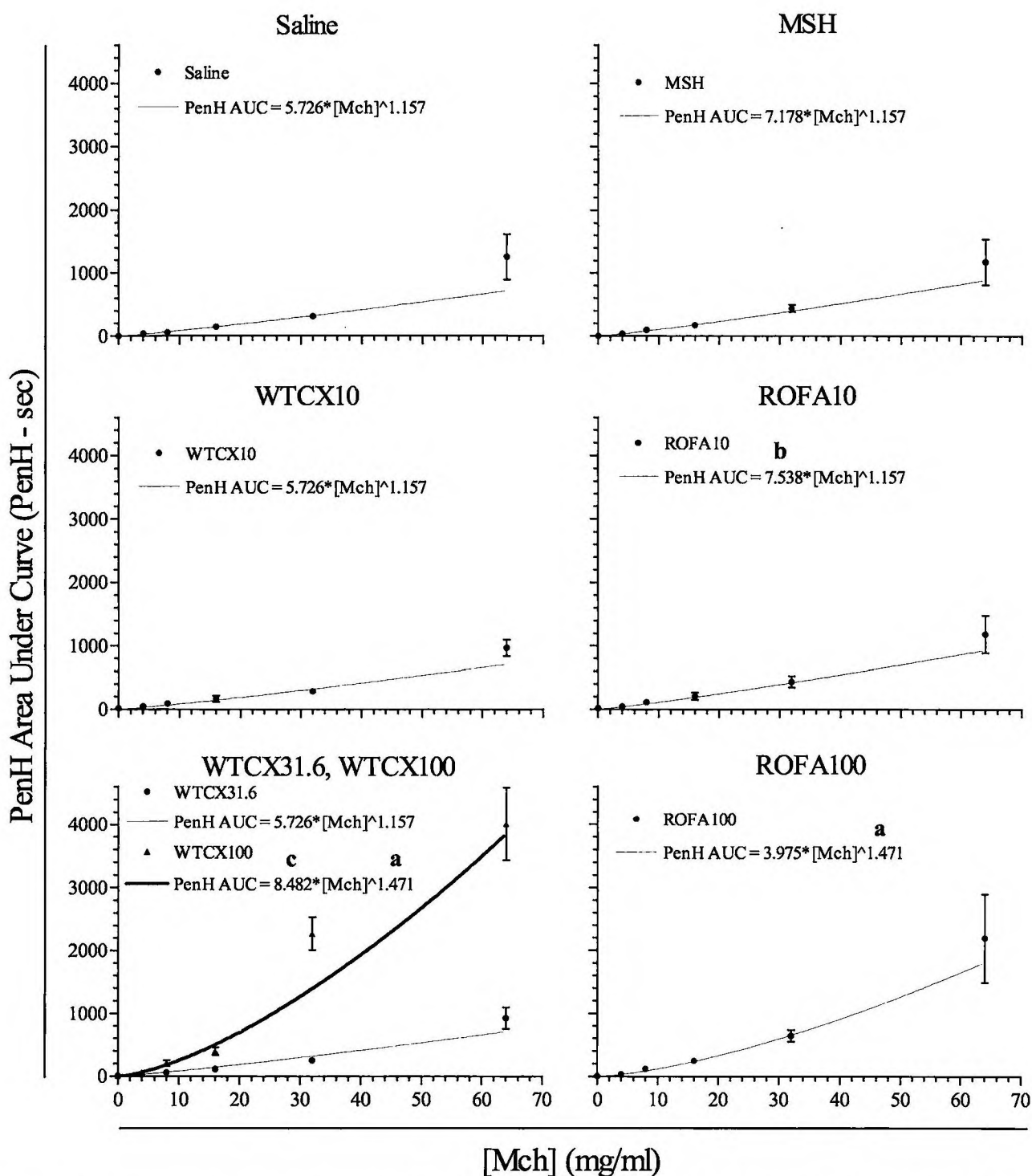


Figure 8. Experiment A: Airway responsiveness to methacholine aerosol challenge in mice exposed to PM samples or saline vehicle and tested one day later (n = 8/group; data shown are mean + SEM). Power function equations were fit to the data. Saline, MSH, ROFA10, WTCX10, and WTCX31.6 equation exponents were not significantly different. ^a Significantly different exponent vs. common Saline, MSH, ROFA10, WTCX10, and WTCX31.6 exponent ($P = 0.001$). ^b Significantly different coefficient vs. Saline coefficient ($P = 0.03$). ^c Significantly different coefficient vs. ROFA100 coefficient ($P = 0.0001$).

Table 10. Experiment A: Summary of Treatment-Related Histopathologic Findings in Mice One Day after Intratracheal Instillation of Particulate Matter Samples ^a

Treatment Group	Bronchiole, Inflammation, Subacute, Focal		Bronchiole, Pigment, Free, Focal		Bronchiole, Pigment, Macrophage, Focal		Peribronchiolar, Inflammation, Acute, Focal	
	Incidence	Severity	Incidence	Severity	Incidence	Severity	Incidence	Severity
Saline	0/8	0.0	0/8	0.0	0/8	0.0	0/8	0.0
MSH-100	6/8	0.8	0/8	0.0	2/8	0.3	0/8	0.0
ROFA-10	2/8	0.3	6/8	0.8	0/8	0.0	1/8	0.1
ROFA-100	6/8	1.0	8/8	1.5	0/8	0.0	0/8	0.0
WTCX-10	1/8	0.1	0/8	0.0	0/8	0.0	0/8	0.0
WTCX-31.6	0/8	0.0	1/8	0.1	0/8	0.0	0/8	0.0
WTCX-100	0/8	0.0	0/8	0.0	0/8	0.0	0/8	0.0

^a Incidence denotes number of mice in group with finding / total number of mice examined. Average severity score for the group is shown based on the following scoring system: 0 = not present, 1 = minimal, 2 = slight/mild, 3 = moderate, 4 = moderately severe, 5 = severe/high.

score (0.3) than the MSH-100 and ROFA-100 groups, and also had one mouse with minimal focal acute peribronchiolar inflammation. Although 1 mouse in the

WTC-X group had a finding of minimal focal subacute bronchiolar inflammation, for an average group score of 0.1, this lesion was not found in any of the mice in the

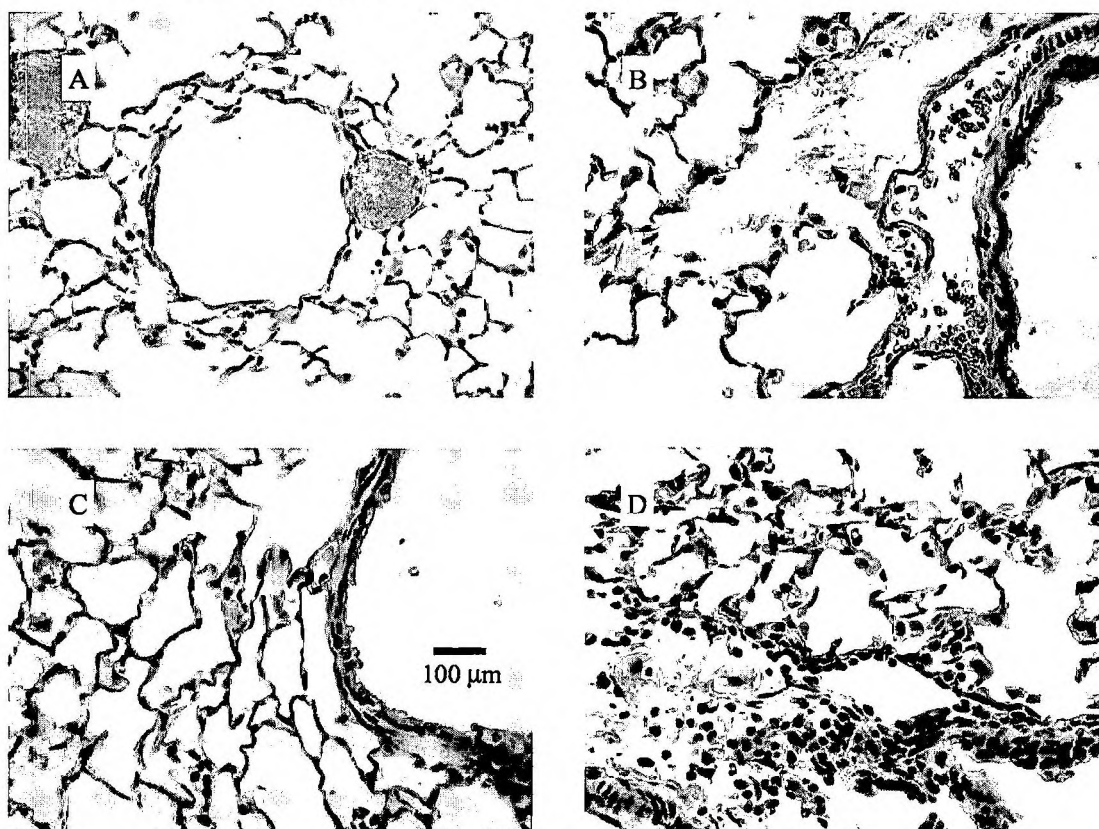


Figure 9. Experiment A: Representative micrographs of lesions occurring in lungs of mice one day after intratracheal instillation of PM samples or saline vehicle (all panels same magnification: bar length = 100 µm). A. Saline-instilled control mouse (#69) with no remarkable findings. B. Mouse #84 instilled with 100 µg MSH showing minimal degree of focal subacute bronchiolar inflammation. C. Mouse #57 instilled with 100 µg pooled WTCX sample with no remarkable findings. D. Mouse #73 instilled with 100 µg ROFA showing slight/mild degree of focal subacute bronchiolar inflammation.

WTCX-31.6 or WTCX-100 groups (Figure 9), suggesting that the lesion in the one WTCX-10 mouse was not treatment-related. Free bronchiolar pigment (presumably corresponding to PM) was identified in all ROFA-100 mice at an average severity of 1.5 (Table 10), and in 6 of 8 mice in the ROFA-10 group at an average severity of 0.8. One mouse in the WTCX-31.6 group (but none in the WTCX-100 group) had minimal free bronchiolar pigment; again suggesting that this finding is not treatment-dependent. However, it may be more difficult to see the WTC PM which is lighter in color than the ROFA or MSH PM. Focal bronchiolar macrophage pigment was found in 2 of 8 mice in the MSH-100 group at an average severity of 0.3. These findings indicate that both ROFA-100 and MSH-100, but not the pooled WTCX-100 or any lower dose, caused focal subacute bronchiolar inflammation.

6. Summary. Results from investigation of the dose-response relationships of pooled WTCX PM showed that the two lower doses of WTCX (10 µg and 31.6 µg) did not have any significant effects on inflammatory parameters, lung histopathologic findings, or respiratory responses. The 100 µg dose of WTCX caused a slight but significant increase in BAL neutrophils (7% of total cells) as determined by BAL parameters, and no inflammation as determined by histopathologic examination, while the toxic PM control, ROFA, caused minimal inflammation by histopathologic examination, significant increases in BAL neutrophils and other cell types, and significant increases in biochemical indicators of lung injury. Despite the lack of effect of WTCX on lung injury and the relatively low level of neutrophilic inflammation, mice in the WTCX-100 group were significantly more responsive to Mch aerosol challenge than all other groups. A lack of correlation between lung inflammation and airway hyperresponsiveness is not uncommon (e.g. Alvarez et al., 2000; Smith and McFadden Jr., 1995). The significant degree of airway hyperresponsiveness induced by WTC PM_{2.5} implies that components of the dust can promote mechanisms of airway obstruction.

C. Experiment B: Effects of Nose-Only Inhalation Exposure

1. Exposure results. The gravimetric concentration for the WTC3 exposure chamber was 10.64 ± 3.10 mg/m³. The mass median aerodynamic diameter (MMAD) was 1.05 µm, and the geometric standard deviation (σg) was 2.67. Chamber temperature and relative humidity was 74 °F and 11% in the control chamber and 75 °F and 11% in the WTC3 chamber. The low humidity was required to prevent the PM from sticking to the string in the aerosol generation system; the humidity within the exposure tubes

was significantly higher due to body heat from the mice in a confined environment. At the end of the exposure, two control mice (#131 and #146) and one WTC3-exposed mouse (#203) were found dead in the exposure tubes, apparently from attempting to turn around in the exposure tubes and suffocating. The incidence of this problem was not unusual considering the large number of mice exposed simultaneously (AD Ledbetter, personal communication). The two spare mice (designated #146a and #203a) were used to replace the dead ones, and were killed on day 6 (December 3, 2001). An additional control mouse (designated #131a) was exposed to air for 5 hours on November 30, 2001 to replace the second dead control mouse, and was killed on day 3 (December 3, 2001). Therefore all groups of mice had the full number of 8 per group per time point.

Table 11. Experiment B: Body Weights of Mice in Nose-Only Inhalation Exposure Study ^a

Day:	Body Weight (g)					
	-1	0 (Pre-)	0.25 (Post-)	1	3	6
Group						
Air	23.91 0.16	23.81 0.18	21.83 0.18	23.00 0.18	24.04 0.23	24.78 0.35
WTC 3	23.93 0.15	23.47 0.18	21.82 0.16	23.05 0.18	23.89 0.15	24.66 0.34

^a Values shown are means (in bold) and SEM immediately below means (n=48 days -1 through 1, n=32 day 3, n = 16 day 6). Body weight was measured in the morning except on day 0.25 (immediately after nose-only exposure). There was no significant difference between the two groups.

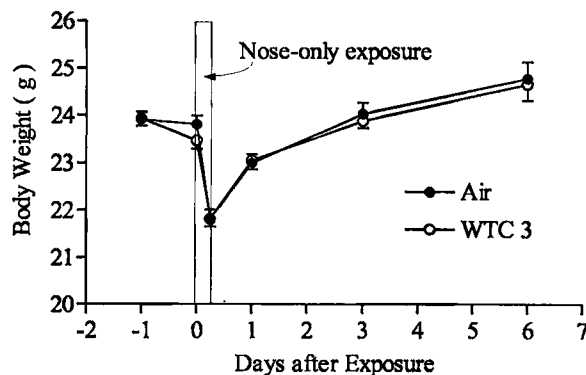


Figure 10. Experiment B: Body weights in nose-only inhalation exposure experiment. Values shown are means and SEM (numbers of mice shown in Table 11). Nose-only inhalation exposure caused a significant drop in body weight but there was no significant difference between groups.

Table 12. Experiment B: Immediate Airway Responses ^a

Group	Breathing Frequency (min ⁻¹)			PenH (unitless)		
	Pre-	Post-	% increase	Pre-	Post-	% increase
Air	549.2	357.7	-35.0	0.88	1.09	29.5
	9.1	24.4	4.1	0.05	0.09	13.8
WTC 3	560.3	389.3	-30.1	0.94	1.47	60.1
	10.3	19.0	3.9	0.06	0.16	18.2

^a Values shown are means (in bold) and SEM immediately below means (n=12). Respiratory parameters were measured immediately before (Pre-) and after (Post-) nose-only inhalation exposure on day 0. No significant differences in percent change in frequency or PenH between groups were found.

2. Body weights. Animal weights were monitored on days -1 (before exposure), 0 (both before and after exposure), 1, 3, and 6 (Table 11). Body weight was measured between 7:00 and 8:00 each morning, except immediately after exposure. There were no significant differences between the two groups at any time point. The nose-only exposure caused a significant 2 g drop in body weight in both groups of mice (Figure 10).

3. Immediate airway responses to nose-only exposure. Ventilatory parameters were measured in 12 mice from each group before and after the nose-only exposure. Ventilatory rate decreased after exposure in both groups but there was no significant difference between them (Table 12). It should be noted that many physiological responses are readily reversible, and the time required to unload the mice from the exposure tubes and

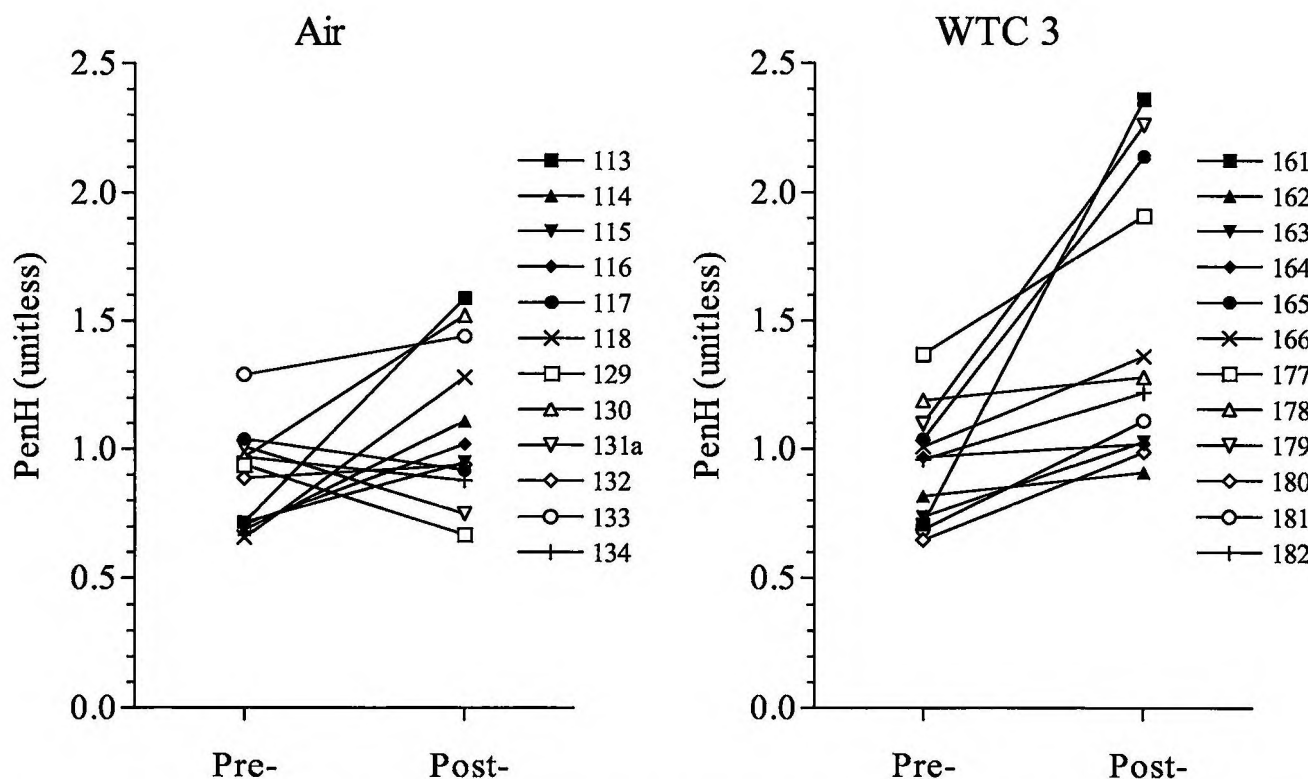


Figure 11. Experiment B. PenH values measured immediately before and after nose-only exposure to WTC 3 PM or Air only. Legends refer to individual mouse numbers. Immediate response (calculated as [(Post-value - Pre-value) / Pre-value x 100%]) was not significantly different between the two groups but data indicate the possibility of individual sensitivity to dust exposure.

Table 13. Experiment B: Diffusing Capacity of the Lung for Carbon Monoxide ^a

Treatment Group	Day after Treatment	Subjects 1 - 4	Subjects 5 - 8	Average n = 2
Air	1	-3.761	-4.185	-3.973
WTC 3	1	-3.715	-3.981	-3.848
Air	3	-4.102	-3.826	-3.964
WTC 3	3	-3.818	-4.078	-3.948
Air	6	-3.528	-4.162	-3.845
WTC 3	6	-3.487	-3.809	-3.648

^a Diffusing capacity of the lung for carbon monoxide was determined 1, 3, or 6 days after exposure on four mice from each treatment group placed together in a single bell jar. Values shown are slopes of chamber [CO] vs. time (ppm/min), after subtraction of value from empty chamber.

begin the measurement of breathing parameters (~20 minutes) may have caused us to miss some changes. PenH was increased by an average of 30% after exposure to air and by an average of 60% after exposure to WTC3 ($P = 0.20$). Although this difference was not significant, examination of the changes in individual mice showed that PenH increased in all 12 mice exposed to WTC3, but only 8 of 12 mice exposed to Air (Figure 11). Furthermore some of the increases in WTC3-exposed mice were quite large. These data indicate the possibility that individual

mice in this outbred strain may be susceptible to bronchoconstrictive effects of WTC PM.

4. DLCO measurements. DLCO was determined 1, 3, and 6 days after exposure on 4 mice from each group placed together in the test chamber. Since there were 8 mice per group per time point, only two tests of DLCO were conducted within each group, and no statistical comparison was possible between Air and WTC3 mice. Examination of the data showed little apparent difference in DLCO at different times in the two groups (Table 13).

5. Responsiveness to methacholine aerosol. Analysis of baseline PenH values (immediately before Mch aerosol) between the two groups showed a significant difference depending on day, but not due to treatment (day 6 baselines were lower in both groups; $P = 0.0007$; Table 14). Responsiveness to increasing concentrations of Mch aerosol was assessed and quantified as described in the Methods section (Table 14). Unlike Experiment A, the results could be modeled with linear equations (Figure 12). Significant interactions of treatment, day, and Mch concentration were detected ($P = 0.01$), implying that the results depended upon a combination of factors. Slopes of the Day and Treatment combinations were significantly different ($P = 0.0001$). Analysis of the data showed that one equation could be used to describe the data for Air Day 6 and for WTC3 Day 1 (Figure 12). The slope of this line was significantly different from and less than that of

Table 14. Experiment B: Baseline PenH and Responsiveness to Methacholine Aerosol ^a

Treatment Group	Day after Treatment	Baseline PenH	Dose Mch (mg/ml) and PenH AUC (PenH - sec)					
			0	4	8	16	32	64
Air	1	0.80	-1.5	20.0	61.5	262.0	945.9	1940.7
		0.04	10.8	15.3	41.7	71.4	227.1	597.3
WTC 3	1	0.68	21.8	18.8	88.9	227.2	697.8	492.3
		0.05	7.9	20.8	12.7	50.0	175.4	104.0
Air	3	0.76	-13.7	50.4	238.4	384.9	1188.1	2031.4
		0.05	9.8	20.9	68.7	77.4	172.5	178.8
WTC 3	3	0.86	-11.4	15.2	37.8	198.6	679.1	1293.3
		0.07	9.7	8.3	12.6	108.8	317.2	356.6
Air	6	0.61	-3.5	-7.6	35.5	106.5	70.1	786.6
		0.05	3.8	12.4	11.7	43.6	59.5	269.9
WTC 3	6	0.58	2.0	31.1	72.0	276.7	1030.4	1935.6
		0.04	2.9	9.0	20.4	97.1	252.8	385.1

^a Values shown are means (in bold) and SEM immediately below means (n=8 per group). Baseline PenH (enhanced pause; unitless) was measured immediately before methacholine aerosol challenge. No significant differences were found between treatment groups, but there was a significant difference in day, with day 6 values being significantly lower than other days (solid line box; $P = 0.0007$). Methacholine aerosol (Mch) was administered (see Methods for details) at the indicated doses, and the airway response was calculated as the area under the curve (AUC) of the PenH response over time in seconds. See Figure 12 for description of statistical analysis of PenH AUC data.

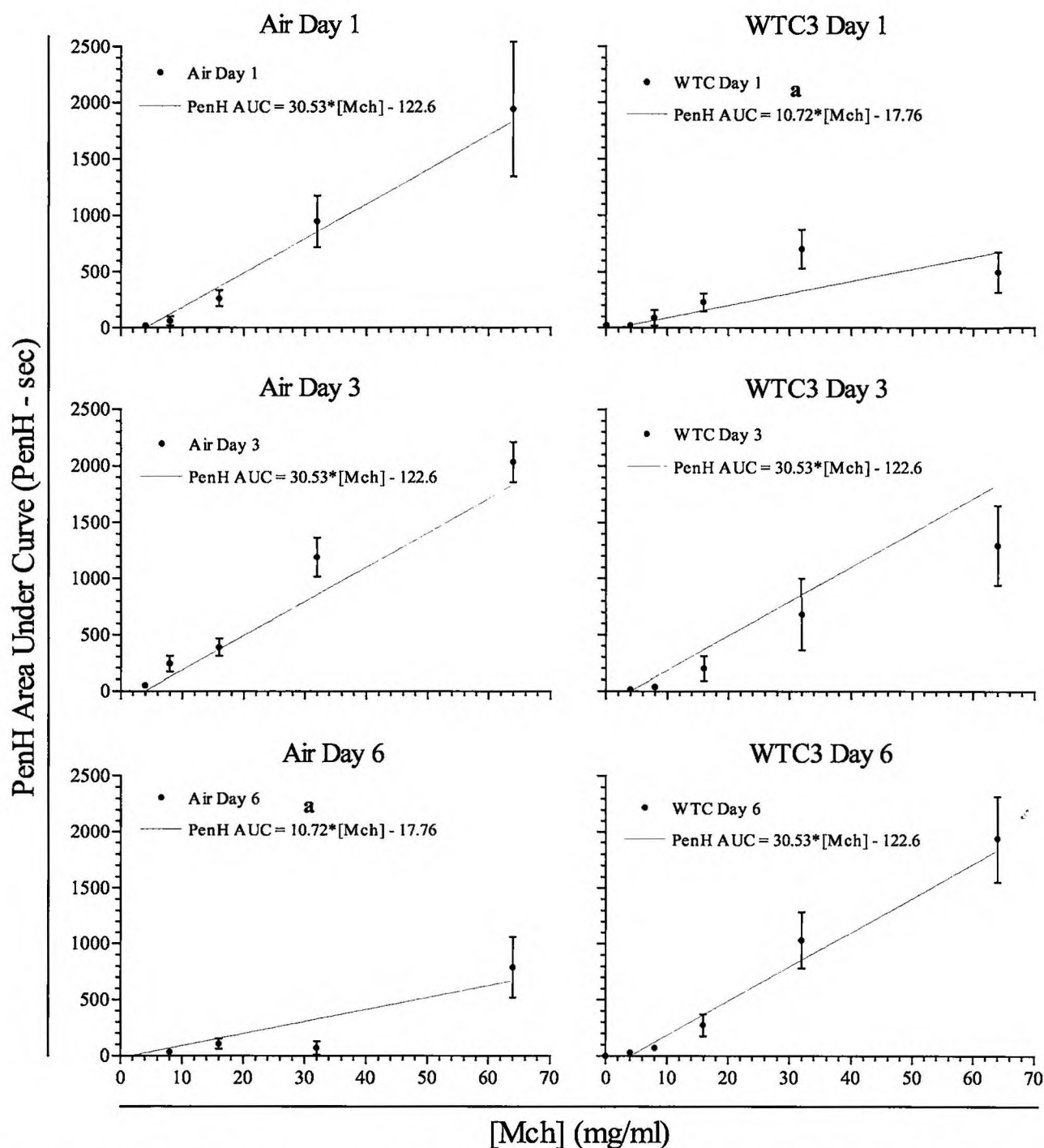


Figure 12. Experiment B: Airway responsiveness to methacholine aerosol challenge in mice exposed nose-only to Air or aerosolized WTC (sample 3) and tested 1, 3, or 6 days later ($n = 8/\text{group}$; data shown are mean $\pm$ SEM). Linear dose-response relationships were found. ^a Slope of Air Day 6 and WTC3 Day 1 were significantly different from and lower than the 4 other groups.

the equation used to fit the other four groups. It should be noted that as in Experiment A, once the lines were determined to come from groups with equal slopes, the

lines were fit simultaneously. This resulted in an equation for the common groups (e.g. Air Day 6 and WTC3 Day 1) that did not fit as well as lines fit to the individual group

Table 15. Experiment B: BAL Cell Numbers after Nose-Only Exposure ^a

Group	Day	BAL Cell Number ($\times 10^4$)			
		Mac	Neut	Eos	Lym
Air	1	14.80	0.012	0.003	0.046
		3.11	0.004	0.002	0.010
WTC 3	1	17.48	0.006	0.000	0.067
		3.13	0.003	0.000	0.013
Air	3	16.56	0.008	0.000	0.125
		1.13	0.004	0.000	0.041
WTC 3	3	26.72	0.034	0.016	0.197
		3.11	0.017	0.009	0.036
Air	6	22.24	0.000	0.000	0.140
		1.13	0.000	0.000	0.026
WTC 3	6	29.86	0.019	0.005	0.281
		2.58	0.008	0.004	0.056

^a Values shown are means (in bold) and SEM immediately below means ($n=8$ per group). Solid-line box: Significant difference ($P=0.01$) between Air and WTC3, Day 6 different from Day 1. Dashed-line boxes: Significant difference ($P=0.02$) between Air and WTC3, Day 3 and Day 6 both different from Day 1.

data. These results could be interpreted as saying that mice exposed to WTC3 became more responsive to Mch in the days following exposure, while Air-exposed mice

became less responsive. However, close examination of the data from experiment B showed it was more variable than that from experiment A. Therefore, although the Air Day 6 and WTC3 Day 1 groups were less responsive to Mch aerosol challenge than the other four groups, the biological significance of this finding is unclear.

6. BAL parameters. Numbers of BAL cells were quantified 1, 3, and 6 days after nose-only exposure to Air or WTC3 (Table 15). Analysis of the data showed that mice exposed to WTC3 had significantly greater numbers of macrophages ($P = 0.01$) and lymphocytes ($P = 0.02$) compared with Air-exposed mice. Macrophage numbers were significantly greater on Day 6 vs. Day 1, and lymphocyte numbers were significantly greater on both Day 3 and Day 6 vs. Day 1. Over all time points, WTC3 mice had 38% more macrophages, and 75% more lymphocytes (Figure 13).

However, macrophages comprised 99% of all recovered cells in both groups at all time points. Lymphocytes constituted about 1% or less of total BAL cells, while both

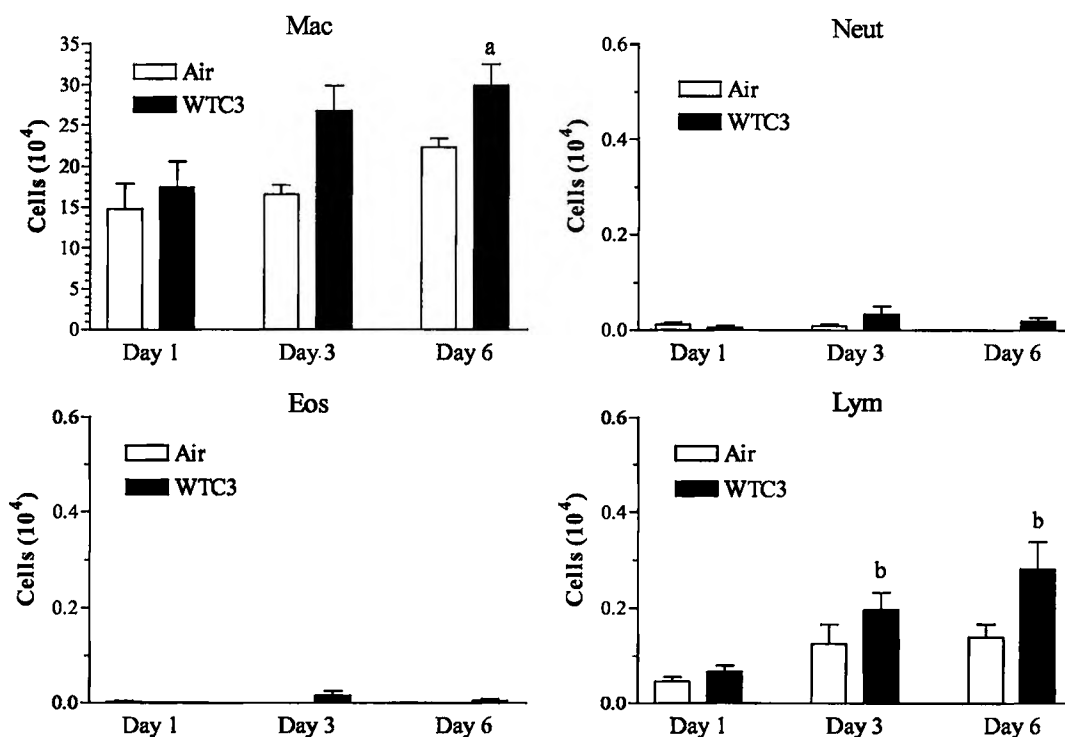


Figure 13. Experiment B: Bronchoalveolar lavage cell numbers recovered from mice 1, 3, or 6 days after 5 hr nose-only inhalation exposure to WTC sample 3 or Air only. Values shown are means and SEM ($n=8$ per group). Cell types shown are macrophages and monocytes (Mac), eosinophils (Eos), neutrophils (Neut), and lymphocytes (Lym). ^a Significant difference ($P = 0.01$) between Air and WTC3, Day 6 different from Day 1. ^b Significant difference ($P = 0.02$) between Air and WTC3, Day 3 and Day 6 different from Day 1.

Table 16. Experiment B: BAL Supernatant Biochemical Values after Nose-Only Exposure ^a

Group	Day	Protein μg/ml	Albumin μg/ml	LDH U/L	NAG U/L
Air	1	165.2 6.1	21.0 1.1	29.0 3.4	1.5 0.1
WTC 3	1	147.1 6.7	16.9 1.2	23.9 3.3	1.6 0.1
Air	3	136.6 10.4	16.2 1.2	33.0 6.4	1.8 0.0
WTC 3	3	138.1 7.8	15.8 1.5	28.9 3.4	1.6 0.1
Air	6	172.6 8.5	22.4 1.3	30.2 2.4	1.4 0.2
WTC 3	6	146.5 6.8	17.5 1.1	27.1 3.1	1.4 0.1

^a Values shown are means (in bold) and SEM immediately below means (n=8 per group). Heavy solid-line boxes: Significant overall treatment effect (WTC3 < Air; no significant day effect); $P = 0.05$ (Protein) or $P = 0.007$ (Albumin).

neutrophils and eosinophils were about 0.1% or less of total BAL cells, indicating that WTC3 did not induce a significant acute inflammatory reaction. The increase in

macrophages and lymphocytes is probably a nonspecific reaction to inhalation of large amounts of dust which induces macrophage recruitment for phagocytosis and clearance of the particles (Adamson and Bowden, 1981).

Levels of proteins and enzymes in the BAL supernatant were assessed in the two groups of mice at 3 time points (Table 16). Significant differences between Air and WTC3 groups were found for total protein ($P = 0.05$) and albumin ($P = 0.007$), but there was no significant day effect. Surprisingly, the levels of protein and albumin were higher in the Air group. However, the overall levels of all proteins and enzymes was low in both groups and at all time points (Figure 14), in comparison with Experiment A. The results indicate that at this exposure concentration and duration, WTC3 PM_{2.5} does not induce severe acute lung injury.

7. Nasal histopathology. No exposure-related nasal lesions were found in mice exposed to air alone (controls). Similarly no nasal lesions were found in the mice exposed to WTC3 and killed 3 or 6 days post-exposure. None of the mice in any group had

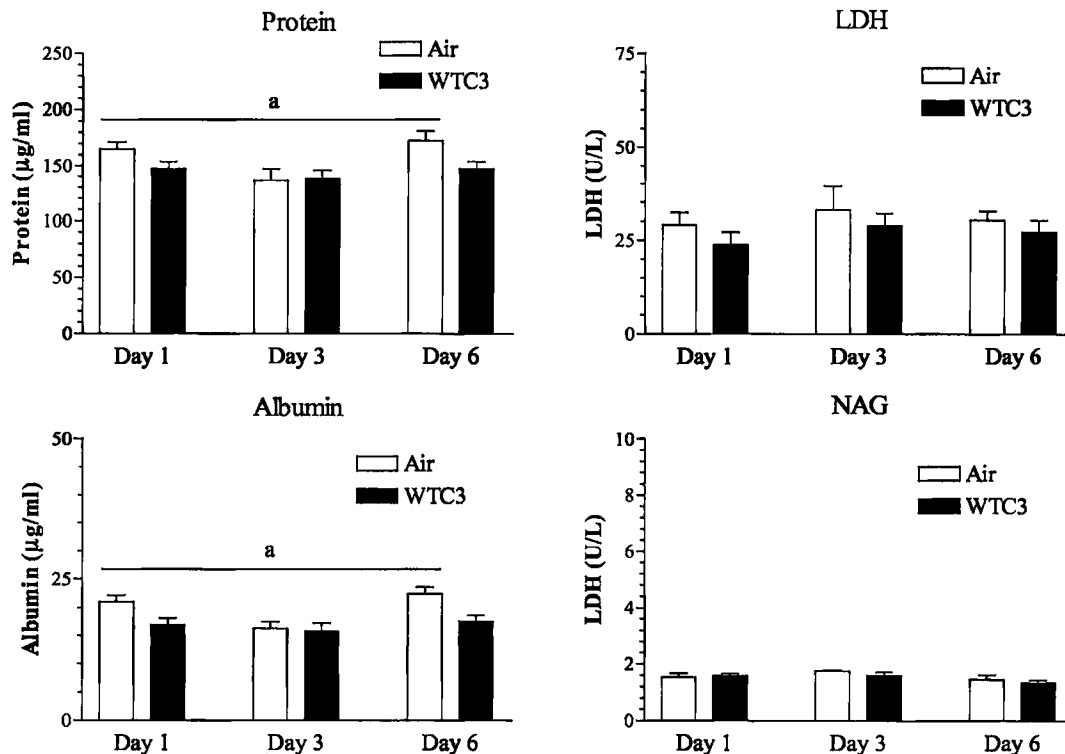


Figure 14. Experiment B: BAL supernatant biochemical values in mice 1, 3, or 6 days after 5 hr nose-only inhalation exposure to WTC3 or Air only. Values shown are means and SEM (n=8 per group). ^a Significant overall treatment effect (WTC3 < Air; no significant day effect); $P = 0.05$ (Protein) or $P = 0.007$ (Albumin).

exposure-related alterations in the mucosal tissues lined by respiratory or olfactory epithelium in the more distal tissue sections examined (T2 and T3).

The only nasal alterations observed by light microscopic examination was minimal to mild acute, focal inflammation (rhinitis) in four of the eight mice exposed to WTC3 PM_{2.5} and killed 24 h post-exposure (animal # 161, 162, 163, 164). This minimal inflammatory response was bilateral and restricted to the most proximal tissue section examined (T1). It was characterized by a slight increase in the number of neutrophils in the mucosal tissues lining the lateral meatus, especially in the ventral lateral meatus, the dorsomedial aspect of the proximal maxilloturbinate, and the ventral aspect of the proximal nasoturbinate in both nasal passages. It must be emphasized, however, that the severity of this focal rhinitis was minimal to mild (i.e., severity score of 1 or 2 out of 4). In addition, there were no associated histologic alterations in the surface epithelium or in the subepithelial tissues in the affected areas. In mouse # 162 there was a small accumulation of mucus and fiber-like material in the lateral meatus of one nasal passage in T1.

In summary, some but not all mice exposed to WTC3 and killed 1 day after exposure had a minimal acute rhinitis that was restricted to the proximal nasal airways. This minimal inflammatory response was probably due to stimulation by the WTC3 exposure. This stimulation, however, did not result in any apparent epithelial cell injury that is often observed with many inhaled agents. No nasal lesions were observed in mice exposed to WTC3 and killed 3 or 6 days post-exposure. This suggests that any acute inflammation that may have been induced by the dust exposure quickly resolved and did not result in any persistent injury to the nasal mucosa that could be detected by light microscopy.

8. Lung histopathology. No remarkable findings were observed in any of the mice exposed to Air or to WTC3 at any time point. Since nasal lesions as described above were restricted to the proximal T1 region and were not found in the more distal T2 and T3 regions, the lack of any findings in the lung suggests that the proximal region of the nose effectively scrubbed out enough of the particulate matter during the exposure to WTC3 to limit deposition further down the respiratory tract. It should be noted that mice are obligate nose-breathers, while humans have significant oral breathing, and therefore significantly more PM can bypass the nasal passages in humans (Schlesinger, 1985). Studies have shown considerably less deposition efficiency in the alveolar region of rodents compared with humans (Asgharian et al., 1995).

9. Summary. Results from investigation of the effects of nose-only exposure to the WTC3 sample indicate that WTC3 PM_{2.5} induced mild transitory neutrophilic inflammation in proximal nasal airways of some mice, but WTC3 PM_{2.5} did not induce neutrophilic inflammation in the lungs of any mice. However, numbers of macrophages were significantly increased after exposure, suggesting that some WTC3 PM_{2.5} penetrated into the lower respiratory tract, which stimulated recruitment of macrophages to phagocytize and clear the particulate matter. Biochemical parameters of lung injury were not increased at all by WTC3. The data suggested that individual mice in this outbred strain may be sensitive to the immediate effects of WTC3 exposure and respond with increased airway obstruction, although this effect was not significant for the group as a whole. Groups of mice exposed to Air or WTC3 PM_{2.5} differed in their responsiveness to Mch aerosol at different times after exposure, but the biological significance of these results was unclear. The dose deposited in the respiratory tract following nose-only inhalation may be estimated as follows: 18.8 ml/min (mouse minute ventilation based on weight; Costa et al., 1992) x 300 min (exposure time) x 0.001 L/ml x 0.001 m³/L x 10.64 mg/m³ (exposure concentration) x 1000 µg/mg x 0.23 (deposition efficiency estimate in total respiratory tract) ≈ 14 µg. Thus, the significant difference in dose deposited into the airways between oropharyngeal aspiration (100 µg) and nose-only inhalation probably accounts for the lack of effect in many of the endpoints examined following nose-only inhalation exposure.

D. Experiment C: Effect of Geographical Location of WTC PM Samples on Responses

1. Sub-experiments and body weights. WTC PM_{2.5} samples from 7 different sites comprised the pooled WTCX sample (Figure 1, Table 1). The effects of the pooled sample may have been dominated by one or more site samples which were toxic in comparison with other site samples. Experiment C was designed to address this possibility and to examine the variability of pulmonary responses associated with WTC PM_{2.5} samples collected from different geographical locations. The 7 sites were located east (WTC11 - 0.1 miles, WTC8 - 0.4 miles), southeast (WTC13 - 0.1 miles, WTCF - 0.25 miles), south (WTCB - 0.25 miles), west-northwest (WTCC - 0.2 miles), and north-northeast (WTCE - 0.25 miles) from the center point of Ground Zero. Sub-experiment C1 examined responses to WTC8, WTC13, WTCF, NIST, and Saline control mice. Sub-experiment C2 examined responses to WTC11, WTCB, WTCC, WTCE, and Saline control mice.

Table 17. Experiment C: Body Weights ^a

Group	Sub-Experiment	Body Weight (g)			
		Day -1	Day 0	Day 1	Day 3
Saline	C1	24.69	24.76	24.72	24.91
		0.39	0.42	0.45	0.42
WTC8	C1	24.76	24.54	24.16	25.35
		0.49	0.52	0.58	0.90
WTC13	C1	24.69	24.26	23.91	24.14
		0.40	0.42	0.38	0.43
WTCF	C1	24.72	24.46	24.17	24.41
		0.43	0.42	0.38	0.49
NIST	C1	24.75	24.32	24.15	24.31
		0.50	0.53	0.50	0.71
Saline	C2	nd ^b	22.55	22.23	22.85
			0.27	0.25	0.55
WTC11	C2	nd	23.72	23.20	23.72
			0.39	0.43	0.68
WTCB	C2	nd	23.66	23.11	23.29
			0.33	0.33	0.61
WTCC	C2	nd	23.67	23.12	23.96
			0.27	0.24	0.41
WTCE	C2	nd	23.69	23.27	23.44
			0.29	0.36	0.41

^a Values shown are means (in bold) and SEM immediately below means (on days -1, 0, and 1, n=16 per group, except Saline sub-experiment C2: n=8; on day 3, n=8 per group, except Saline sub-experiment C2: n=4). ^b nd - Not determined. No treatment-related differences in body weight among groups within each sub-experiment were detected.

Responses were examined in 8 mice per group at 1 and 3 days after oropharyngeal aspiration of 100 µg of each PM sample or saline alone (n = 4 per time point in sub-experiment C2 Saline mice). Responses were examined at both 1 and 3 day time points in order to begin examination of persistence of exposure effects. Statistical analysis of the data was performed within each sub-experiment.

Body weights were determined on days -1 (before oropharyngeal aspiration), 0, 1, and 3 in sub-experiment C1, and on days 0, 1, and 3 in sub-experiment C2 (Table 17). No treatment-related differences in body weight among groups within each sub-experiment were detected, although there were differences on the day the animals were weighed ($P = 0.0001$).

2. Responsiveness to methacholine aerosol. In sub-experiment C1, the WTC8 group had significantly greater baseline PenH values 1 day after exposure compared with the WTC13 group (Table 18). No other significant differences in baseline PenH values in sub-experiments C1

or C2 were found. Responsiveness to methacholine aerosol was quantified as PenH AUC (Table 18). Analysis of the data in both sub-experiments showed that linear regression equations could be fit to the PenH AUC vs. [Mch] data (Figure 15). In both sub-experiments, tests for equal slopes on days 1 and 3 after exposure showed that day was not a significant factor. Therefore, a single equation was fit to the data for each group, and day does not appear in the equations.

In sub-experiment C1, the WTC8, WTCF, and NIST groups could be described with a common slope and intercept. The common slope of these 3 groups was significantly different from and greater than that of the WTC13 or Saline C1 groups ($P < 0.0005$), indicating that WTC8, WTCF, and NIST were hyperresponsive to methacholine aerosol. The slope of the WTC13 group was significantly greater than that of the Saline C1 group, showing that WTC13 mice were hyperresponsive compared with control mice, though less so than WTC8,

Table 18. Experiment C: Baseline PenH and Responsiveness to Methacholine Aerosol ^a

Group	Sub-Experiment	Day	Baseline PenH	Dose Mch (mg/ml) and PenH AUC (PenH - sec)					
				0	4	8	16	32	64
WTC8	C1	1	0.68	35.2	69.6	197.0	429.4	1115.0	1953.5
			0.05	10.1	19.2	92.8	87.0	126.6	203.5
WTC13	C1	1	0.45	35.5	50.5	87.9	264.7	674.8	1151.3
			0.02	5.1	4.4	9.1	44.2	135.4	206.6
WTCF	C1	1	0.55	25.9	62.3	164.4	369.1	1145.3	1966.5
			0.05	2.1	14.4	29.0	47.3	155.8	293.0
NIST	C1	1	0.59	31.6	52.7	97.3	279.3	811.1	1882.2
			0.03	3.1	2.9	11.5	54.0	149.2	324.6
Saline	C1	1	0.57	24.2	40.4	61.4	171.7	534.5	1077.0
			0.02	1.5	4.0	6.6	19.9	74.2	163.2
WTC8	C1	3	0.66	14.5	41.9	62.2	182.5	690.3	1907.0
			0.04	3.0	10.9	25.1	49.7	206.4	370.0
WTC13	C1	3	0.78	27.9	51.2	207.0	271.9	1000.8	1554.5
			0.08	9.0	9.7	62.6	68.3	204.3	190.8
WTCF	C1	3	0.77	18.8	29.3	87.5	280.8	994.4	2160.8
			0.07	6.4	22.3	34.4	130.7	368.1	563.2
NIST	C1	3	0.76	21.1	47.1	86.1	212.3	962.5	1886.2
			0.05	8.0	14.8	19.4	72.2	271.0	437.2
Saline	C1	3	0.71	-2.8	30.6	99.2	280.4	481.0	1287.9
			0.03	4.6	17.2	16.8	67.0	40.8	79.5
WTC11	C2	1	0.69	15.9	66.9	238.8	811.0	1496.5	1835.9
			0.06	6.5	23.3	90.7	223.6	437.7	306.0
WTCB	C2	1	0.67	7.3	46.4	205.4	695.5	1561.4	2333.1
			0.05	9.4	10.0	53.8	193.0	404.0	611.8
WTCC	C2	1	0.62	5.9	27.5	118.7	737.6	1826.9	2800.2
			0.06	5.9	9.9	30.6	249.0	466.3	672.8
WTCE	C2	1	0.62	8.3	28.7	155.6	390.0	1112.5	1722.8
			0.02	3.1	12.6	86.8	177.1	360.2	452.6
Saline	C2	1	0.61	3.2	52.5	111.7	167.7	308.7	825.5
			0.08	4.0	7.5	19.3	29.5	26.0	101.6
WTC11	C2	3	0.59	3.8	24.7	45.0	256.8	1003.0	2304.1
			0.05	6.3	12.5	19.2	112.0	325.5	559.8
WTCB	C2	3	0.59	22.2	31.9	98.0	238.6	738.5	2609.2
			0.05	4.3	14.6	34.7	59.2	216.6	585.5
WTCC	C2	3	0.56	10.0	22.8	118.9	351.7	1282.4	2669.0
			0.04	7.1	6.6	37.4	144.1	365.5	520.9
WTCE	C2	3	0.62	5.7	42.3	138.6	412.4	1257.9	2088.7
			0.05	6.3	11.7	33.2	117.6	297.5	472.9
Saline	C2	3	0.62	3.5	27.5	84.0	114.7	341.1	1058.7
			0.05	3.5	27.0	65.1	17.2	65.2	45.7

^a Values shown are means (in bold) and SEM immediately below means (n=8 per group, except Saline sub-experiment C2: n=4). A significant difference in baseline PenH (enhanced pause; unitless) was found between the WTC8 group and the WTC13 group on day 1 (heavy solid line box). No other significant differences in baseline values on day 1 or day 3 were detected. Methacholine aerosol (Mch) was administered at the indicated doses, and the airway response was calculated as the area under the curve (AUC) of the PenH response over time in seconds. See Figure 15 for description of statistical analysis of PenH AUC data.

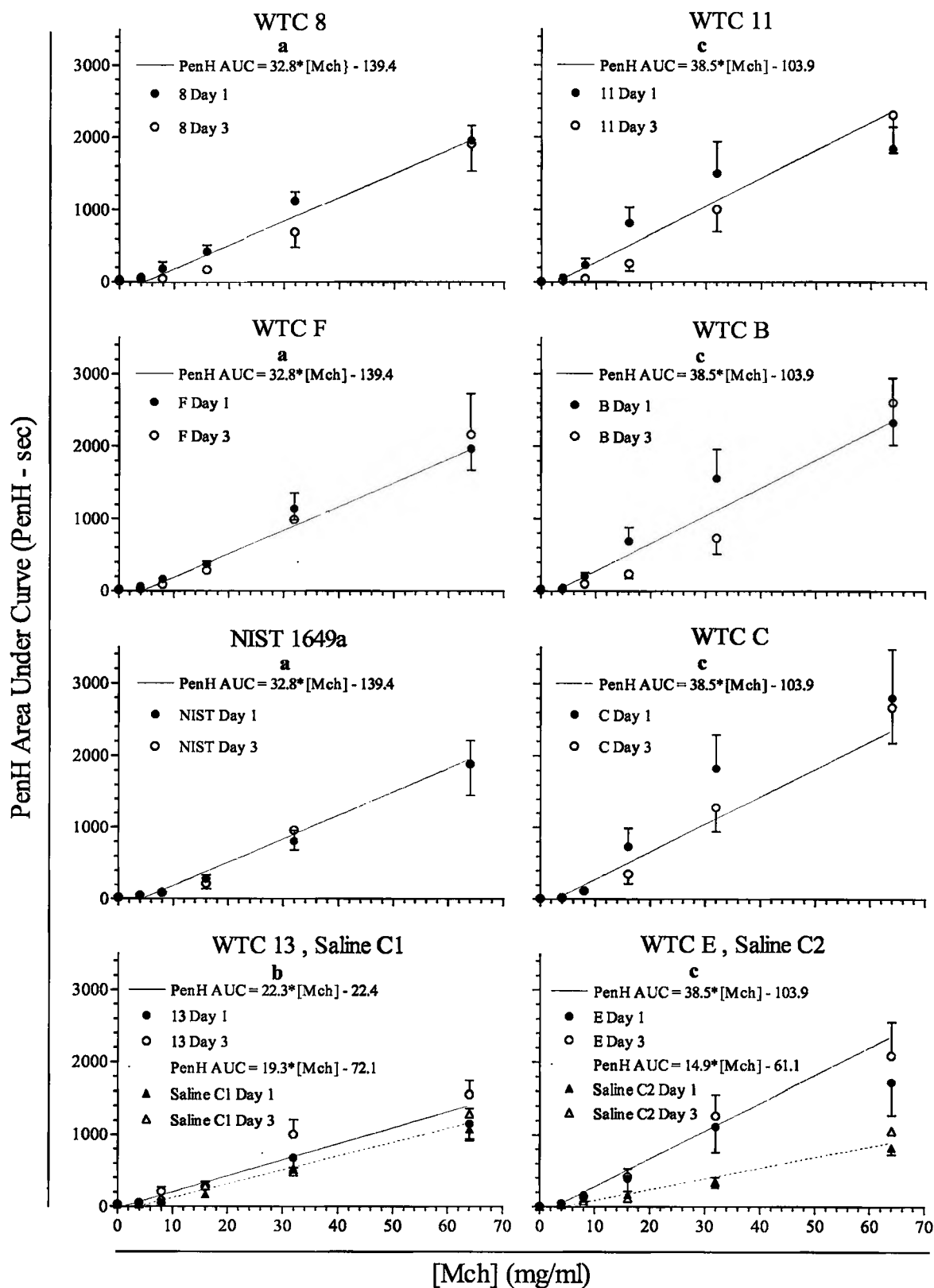


Figure 15. Experiment C: Airway responsiveness. See next page for figure legend.

Figure 15. (previous page.) Experiment C: Airway responsiveness to methacholine aerosol challenge in mice exposed to saline vehicle, NIST 1649a, or WTC PM samples from individual collection sites and tested 1 or 3 days later ($n = 8$ per group except Saline sub-experiment C2: $n = 4$). A single regression equation was fit to the data for both days in each group. ^a In sub-experiment C1 (left panels), a common equation could be fit to the WTC8, WTCF, and NIST data, and the slope of the line was significantly different from and greater than the slopes of the WTC13 and Saline C1 equations. ^b In sub-experiment C1 (left panels), the slope of the equation for the WTC13 group was significantly different from and greater than the slope for the Saline C1 group. ^c In sub-experiment C2 (right panels), a common equation could be fit to the WTC11, WTCB, WTCC, and WTCE data, and the slope of the line was significantly different from and greater than the slope of the Saline C2 equation.

WTCF, and NIST mice.

In sub-experiment C2, the WTC11, WTCB, WTCC, and WTCE groups could all be described with a common slope and intercept, which was similar to that found for WTC8, WTCF, and NIST groups in sub-experiment C1. The common slope of the 4 WTC groups was significantly different from and greater than that of the Saline C2 group ($P = 0.001$), indicating that WTC11, WTCB, WTCC, and WTCE were hyperresponsive to methacholine aerosol.

In general, these results are consistent with those from Experiment A, where the 100 μg dose of the pooled WTCX sample induced significant hyperresponsiveness to methacholine aerosol compared with control PM samples and saline. All but one of the WTC PM samples, as well as the NIST control PM, appeared to cause similar degrees of hyperresponsiveness. However, the WTC13 sample, located just 0.1 miles southeast of Ground Zero, caused a lower degree of hyperresponsiveness compared with WTC8, WTCF, and NIST.

3. BAL cells. After assessment of responsiveness to Mch aerosol, mice were killed and numbers of BAL cells were quantified (Table 19; Figure 16). In sub-experiment C1, significant increases in numbers of neutrophils on Day 1 were found in all PM-exposed groups compared with Saline C1 mice. An average of 14.7×10^4 neutrophils was recovered from NIST mice (45% of total BAL cells). Significantly lower numbers of neutrophils were found in WTC13 (6.1×10^4) and WTCF (6.9×10^4) mice, while numbers of neutrophils were lower still in WTC8 mice (3.2×10^4). The neutrophilic response abated by Day 3, and there were no significant differences among the 5 groups. Numbers of lymphocytes were significantly increased in WTC8, WTC13, WTCF, and NIST mice in

comparison with Saline C1 mice on both Day 1 and Day 3 after oropharyngeal aspiration ($P = 0.0001$). Lymphocyte numbers significantly increased in all groups from Day 1 to Day 3 ($P = 0.0001$). Since there were significant interactions between day and treatment with respect to eosinophil numbers ($P = 0.01$), no significant differences among groups could be discerned. Although a significant difference in macrophage numbers was detected in the WTC13 group compared with saline, it was very marginal and not considered biologically significant.

In sub-experiment C2, significant increases in neutrophils and eosinophils were found in WTC11 and WTCE mice compared with Saline C2 mice. The average number of neutrophils in these 2 WTC groups was comparable to those found in the WTC13 and WTCF groups in sub-experiment C1. In addition, numbers of neutrophils and eosinophils were significantly greater in WTCE mice compared with WTCB mice. Neutrophils numbers declined from Day 1 to Day 3 ($P = 0.0001$). It should be noted that of the four mice in the Saline C2 Day 1 group, two had unusually high neutrophil numbers (individual numbers: 0.15, 0.26, 3.15, and 5.79×10^4 neutrophils), which limited the ability to determine significant increases in neutrophils in mice exposed to WTC PM samples in sub-experiment C2. The reasons for this significant inflammatory response in two control mice are not apparent, but this finding does not detract from the overall conclusion that PM collected from specific locations near the WTC site caused significant inflammation of neutrophils and eosinophils.

These results differ substantially from those found in Experiment A, where 100 μg of pooled WTCX induced only a mild neutrophilic response in the lung one day after oropharyngeal aspiration (average 1.43×10^4). Some WTC individual site samples (WTCF, WTC13, WTC11, WTCE) caused about 4 times the amount of neutrophil recruitment as WTCX, while the others (WTC8, WTCB, WTCC) caused about twice as much recruitment. It is not clear how the individual site samples could all cause more lung inflammation than the pooled WTCX sample which was composed of the individual site samples. This finding may be a result of significant differences in responsiveness of different lots of mice sent on different weeks, which we have found with some studies of other toxic inhalants. Additionally, although these mice were lavaged after Mch challenge, our experience has shown that the challenge itself does not induce cellular inflammation that might account for the observations made here. To adequately address this question, pooled WTCX and individual site samples would need to be tested together in the same experiment.

Table 19. Experiment C: BAL Cell Numbers ^a

Group	Sub-Experiment	Day	BAL Cell Number (x 10 ⁴)			
			Mac	Neut	Eos	Lym
Saline	C1	1	16.41	0.34	0.02	0.14
			1.92	0.20	0.01	0.02
WTC8	C1	1	21.43	3.23	0.27	0.38
			2.38	0.52	0.08	0.05
WTC13	C1	1	24.19	6.10	1.33	0.70
			2.24	0.88	0.43	0.14
WTCF	C1	1	18.47	6.85	0.84	0.58
			1.45	0.85	0.22	0.10
NIST	C1	1	17.36	14.67	0.43	0.48
			3.41	1.17	0.17	0.13
Saline	C1	3	24.03	0.48	0.19	0.36
			6.56	0.45	0.07	0.09
WTC8	C1	3	29.79	0.18	1.48	0.75
			2.70	0.06	0.90	0.15
WTC13	C1	3	25.96	0.46	0.39	0.88
			2.32	0.15	0.10	0.22
WTCF	C1	3	24.60	0.25	1.46	1.16
			2.23	0.07	0.49	0.26
NIST	C1	3	29.09	1.74	0.67	1.97
			4.06	0.26	0.36	0.41
Saline	C2	1	16.25	2.34	0.22	0.16
			1.21	1.34	0.19	0.06
WTC11	C2	1	23.38	5.79	0.75	0.98
			3.84	1.42	0.39	0.26
WTCB	C2	1	25.91	2.98	0.17	0.49
			3.90	0.95	0.05	0.05
WTCC	C2	1	27.19	2.53	0.28	0.29
			4.02	0.42	0.10	0.08
WTCE	C2	1	24.19	5.12	0.37	0.38
			2.94	0.80	0.12	0.10
Saline	C2	3	20.87	0.03	0.02	0.27
			0.69	0.02	0.01	0.11
WTC11	C2	3	25.50	1.13	0.49	0.75
			3.80	0.99	0.16	0.31
WTCB	C2	3	29.60	0.16	0.30	0.46
			2.87	0.05	0.12	0.11
WTCC	C2	3	24.76	0.34	0.39	0.79
			2.24	0.08	0.14	0.19
WTCE	C2	3	30.11	0.21	1.59	1.45
			2.62	0.05	0.43	0.50

^a Values shown are means (in bold) and SEM immediately below means (n=8 per group, except Saline sub-experiment C2: n=4). Significant differences shown are within sub-experiments only. Heavy solid-line boxes: Within sub-experiment C1 day 1 neutrophils, NIST > WTC13 and WTCF > WTC8 > Saline. Solid-line boxes, underlined values: NIST, WTC13, WTCF, and WTC8 all significantly different from Saline. Dashed-line boxes: WTC11 significantly different from Saline. Solid-line shaded boxes: WTCE significantly different from Saline and WTCB.

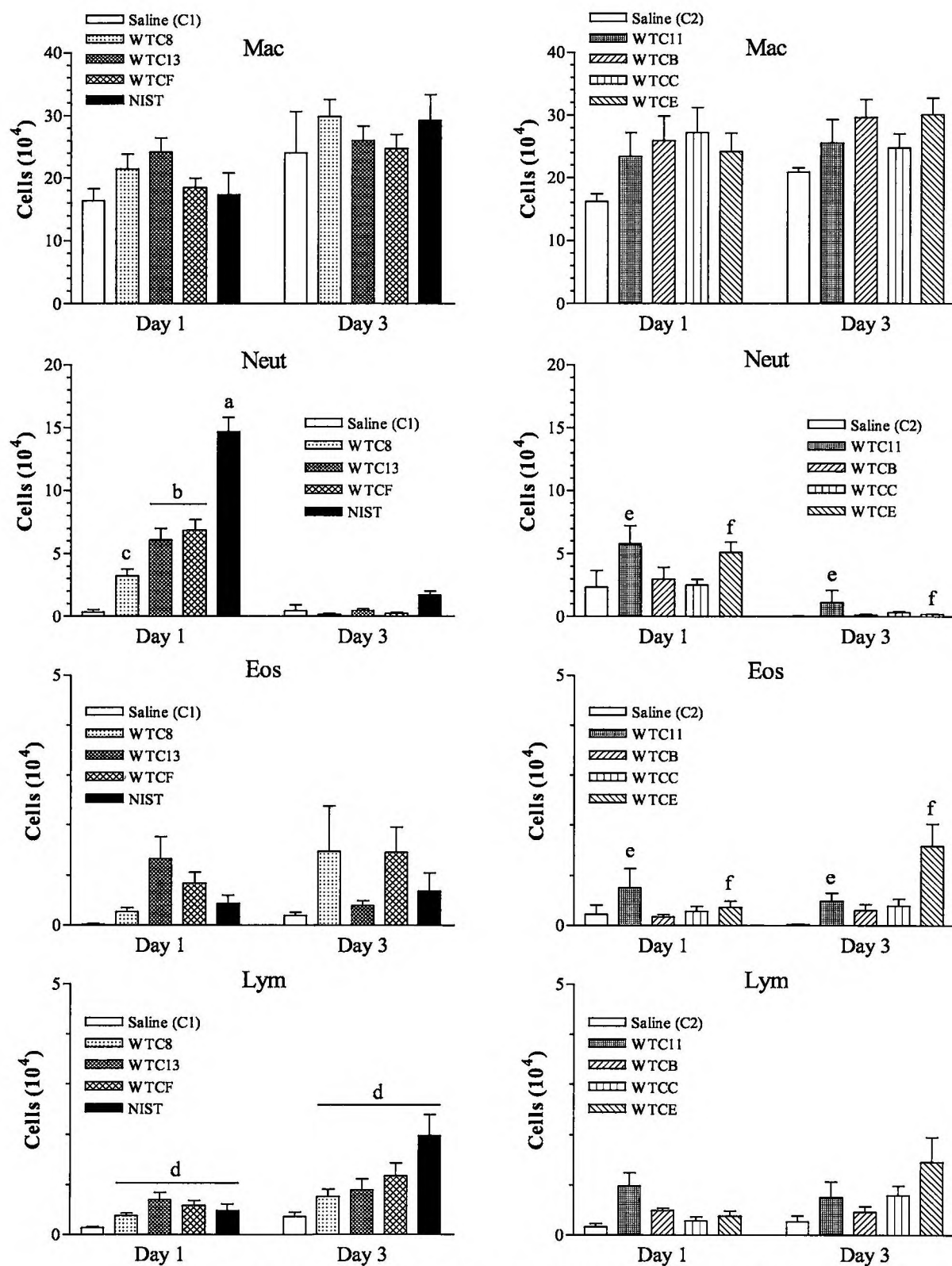


Figure 16. Experiment C: BAL cells. See next page for figure legend.

Figure 16. (previous page.) Experiment C: BAL cell numbers recovered from mice exposed to saline vehicle, NIST 1649a, or WTC PM samples from individual collection sites and tested 1 or 3 days later ($n = 8$ per group except Saline sub-experiment C2: $n = 4$). Cell types shown are macrophages (Mac), neutrophils (Neut), eosinophils (Eos), and lymphocytes (Lym). ^a NIST significantly greater than all other groups. ^b WTC13 and WTCF significantly greater than WTC8 and Saline C1 groups. ^c WTC8 significantly greater than Saline C1 group. ^d Lymphocyte numbers significantly greater in WTC8, WTC13, WTCF, and NIST groups compared with Saline C1 group. ^e Significantly greater numbers of neutrophils and eosinophils in WTC11 group compared with Saline C2 group. ^f Significantly greater numbers of neutrophils and eosinophils in WTCE group vs. WTCB and Saline C2 groups.

4. BAL proteins and enzymes. As for other parameters, BAL protein and enzyme data for sub-experiments C1 and C2 were analyzed separately (Table 20, Figure 17). In sub-experiment C1, significant increases in BAL total protein levels were found in the NIST group compared with the WTC8 group ($P = 0.05$). No significant differences due to Treatment were found with respect to albumin or LDH levels. There were significant interactions between Day and Treatment in NAG values ($P = 0.02$), indicating the results depended on the day animals were killed.

In sub-experiment C2, there were significant effects of Day after treatment for total protein and LDH, but there were no effects of Treatment group. There were significant interactions between Day and Treatment in NAG values ($P = 0.005$), indicating the results depended on the day after treatment.

In both sub-experiment C1 and C2, one of the saline group mice killed on Day 1 had very high values for total protein, albumin, and LDH, which increased the mean values and variability in these groups. Although this result may have limited the ability to detect some statistical differences, overall the biochemical values were not greatly different among the treatment groups, and any additional differences with more consistent control data would likely have been minimal. Therefore the results for the individual site WTC PM samples are comparable to those found with the pooled WTCX sample in Experiment A, where no differences from control saline mice were found.

5. Lung histopathology. Following tests for airway responsiveness to Mch aerosol and lung lavage, lungs were removed and fixed with 4% paraformaldehyde, and

pathological changes were assessed. Although the lungs of all mice in Experiment C were lavaged (they were not lavaged in Experiments A or B), the pattern and the morphology of the PM induced findings were relatively consistent in all treated groups.

Focal subacute bronchiolar inflammation and focal bronchiolar pigmented macrophages (presumably PM) were consistently observed in all groups of mice dosed with each of the different PM samples, and both findings are considered to be PM-induced in all groups (Table 21). Some groups also had findings of focal free bronchiolar pigment, consistent with the pigment in macrophages. No remarkable findings were observed in the lungs of the saline control group (Figure 18A), except for one mouse which had a minimal degree of focal subacute bronchiolar inflammation which was not considered to be treatment-related. Table 21 shows the rankings of the treatment-related histopathologic findings in mice 1 or 3 days after exposure. The degree of focal subacute bronchiolar inflammation was greatest in the NIST (Figure 18C), WTCE, and WTC13 (Figure 18D) groups on Day 1 (average severity scores of 1.9, 2.0, and 2.1, respectively). The scores in the WTCC (Figure 18B), WTCB, WTC8, WTCF, and WTC11 groups were lower (average severity scores of 0.8, 1.1, 1.1, 1.3, and 1.3, respectively). By Day 3, the focal subacute bronchiolar inflammation was greatest in the NIST group (average severity score 2.1; Figure 18E), while the scores were reduced in all of the WTC PM groups relative to their scores on Day 1 (Figure 18F).

The histopathologic scoring system is semi-quantitative, and much larger numbers of mice per group would be necessary to determine statistically significant differences among groups. Nevertheless, these results also show substantial differences from those found in Experiment A with the pooled WTCX sample. Oropharyngeal aspiration of 100 μ g of WTCX did not cause any treatment-related histopathologic findings. In contrast, all individual site samples of WTC PM induced at least minimal focal subacute bronchiolar inflammation, and some samples caused slight/mild and even moderate degrees of inflammation. In addition, pigment associated with PM was visible in macrophages from all WTC PM-exposed mice, but none was visible in mice exposed to pooled WTCX in Experiment A. Re-examination of the slides by a different observer will be necessary to confirm this finding. The findings of pulmonary inflammation in WTC PM groups by histopathologic examination are consistent with the results from the quantification of BAL cell numbers.

Table 20. Experiment C: BAL Supernatant Biochemical Values ^a

Group	Sub-Experiment	Day	Protein µg/ml	Albumin µg/ml	LDH U/L	NAG U/L
Saline	C1	1	240.0 73.9	52.5 15.2	43.1 5.3	1.7 0.2
WTC8	C1	1	191.9 9.5	40.5 3.1	46.1 3.6	2.7 0.3
WTC13	C1	1	201.8 16.3	40.6 4.5	40.6 4.4	2.9 0.2
WTCF	C1	1	200.4 11.9	42.9 3.4	44.6 3.3	1.7 0.2
NIST	C1	1	257.1 16.3	52.6 4.1	56.8 5.0	4.5 0.4
Saline	C1	3	274.6 65.4	55.9 10.0	37.5 11.1	3.1 1.3
WTC8	C1	3	156.1 13.3	34.9 4.6	35.2 1.8	2.3 0.1
WTC13	C1	3	203.8 26.4	47.1 8.1	36.0 2.2	2.7 0.2
WTCF	C1	3	197.5 15.9	45.4 4.5	37.2 2.8	2.9 0.4
NIST	C1	3	220.4 17.3	45.2 4.6	40.2 5.2	4.3 0.6
Saline	C2	1	307.8 95.7	75.5 28.0	44.5 18.9	2.9 0.2
WTC11	C2	1	202.0 30.8	44.8 7.7	48.1 6.2	2.4 0.7
WTCB	C2	1	242.5 56.4	45.2 6.6	55.2 13.0	2.9 0.8
WTCC	C2	1	194.8 13.5	42.2 4.6	34.6 2.6	1.7 0.1
WTCE	C2	1	194.5 11.7	43.5 3.6	39.5 2.7	3.0 0.4
Saline	C2	3	180.8 21.1	46.8 6.1	29.5 4.9	2.3 0.3
WTC11	C2	3	213.0 64.7	49.2 11.5	50.1 15.0	4.4 2.3
WTCB	C2	3	131.8 12.2	32.5 4.6	32.4 3.6	2.2 0.1
WTCC	C2	3	167.2 16.4	44.8 5.8	30.0 1.7	2.9 0.2
WTCE	C2	3	190.2 23.4	51.0 8.1	29.4 2.1	2.5 0.2

^a Values shown are means (in bold) and SEM immediately below means (n=8 per group, except Saline sub-experiment C2: n=4). Significant differences shown are within sub-experiments only. Heavy solid-line boxes: NIST significantly different from WTC8. Significant overall Day effects were found for Protein (sub-experiment C2), and LDH (both sub-experiments).

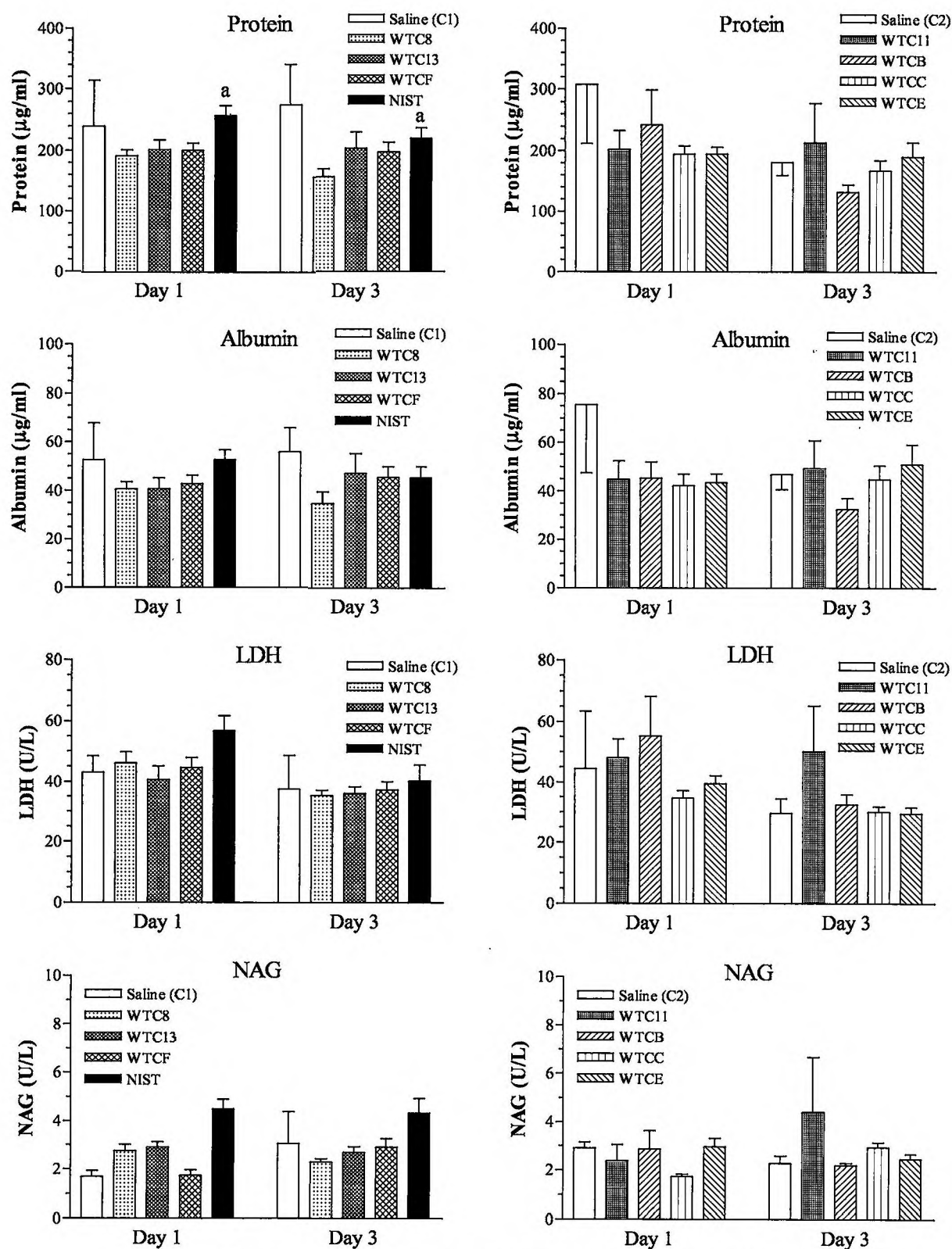


Figure 17. Experiment C: BAL supernatant biochemistry. See next page for figure legend.

Figure 17. (previous page.) Experiment C: Bronchoalveolar lavage supernatant proteins and enzymes recovered from mice exposed to saline vehicle, NIST 1649a, or WTC PM samples from individual collection sites and tested 1 or 3 days later (n = 8 per group except Saline sub-experiment C2: n = 4). ^a Significantly greater protein values in NIST group vs. WTC8 group. Other significant differences were found due to Day of sacrifice or interactions between Day and Treatment, but there were no other effects due to Treatment alone.

6. Summary. Examination of the effects of WTC PM collected from different locations surrounding the WTC site showed that all samples were capable of inducing pulmonary inflammation and hyperresponsiveness to Mch aerosol, although overt lung damage as determined by biochemical parameters of lung injury was minimal. The neutrophilic response was substantially greater for all individual site WTC PM

samples compared with the response induced by the pooled WTCX sample in Experiment A, although differing responsiveness of different shipments of mice could account for this finding, and a direct comparison would be necessary to determine if there is a difference. Numbers of neutrophils declined from Day 1 to Day 3 after oropharyngeal aspiration, as determined by both BAL and histopathologic examination. Other cell types appeared to be more persistent or increase from Day 1 to Day 3 (especially lymphocytes in sub-experiment C1), but these were not large changes. Respiratory responsiveness to Mch aerosol was significantly increased in all WTC groups compared with saline controls, although mice exposed to WTC13 were less responsive than other WTC groups. The degree of Mch hyperresponsiveness in the WTC groups of Experiment C appeared to be comparable to that from the WTCX group in Experiment A.

No particular geographical significance could be deduced from the patterns of responses induced by the individual WTC PM samples. The one group which had

Table 21. Experiment C - Summary of Treatment-Related Histopathologic Findings in Mice 1 or 3 Days after Intratracheal Instillation of Particulate Matter Samples ^a

Treatment Group	Sub-Experiment	Day	Bronchiole, Inflammation, Subacute, Focal		Bronchiole, Pigment, Macrophage, Focal		Bronchiole, Pigment, Free, Focal	
			Incidence	Severity	Incidence	Severity	Incidence	Severity
WTC13	C1	1	8/8	2.1	8/8	2.0	4/8	0.6
WTCE	C2	1	8/8	2.0	8/8	1.9	2/8	0.3
NIST	C1	1	8/8	1.9	7/8	2.0	7/8	0.9
WTC11	C2	1	8/8	1.3	7/8	0.9	0/8	0.0
WTCF	C1	1	8/8	1.3	6/8	0.8	0/8	0.0
WTC8	C1	1	6/8	1.1	6/8	0.8	0/8	0.0
WTCB	C2	1	6/8	1.1	6/8	0.8	0/8	0.0
WTCC	C2	1	6/8	0.8	4/8	0.5	0/8	0.0
Saline	C1	1	1/8	0.1	0/8	0.0	0/8	0.0
NIST	C1	3	8/8	2.1	8/8	2.0	0/8	0.0
WTC11	C2	3	6/8	1.1	2/8	0.3	0/8	0.0
WTCE	C2	3	6/8	0.8	6/8	0.8	0/8	0.0
WTC8	C1	3	4/8	0.8	1/8	0.1	0/8	0.0
WTC13	C1	3	4/8	0.6	3/8	0.4	0/8	0.0
WTCB	C2	3	3/8	0.4	2/8	0.3	0/8	0.0
WTCF	C1	3	3/8	0.4	1/8	0.1	0/8	0.0
WTCC	C2	3	2/8	0.3	1/8	0.1	0/8	0.0
Saline	C1	3	0/7	0.0	0/7	0.0	0/7	0.0

^a Saline-instilled control mice in sub-experiment C2 were not examined. Incidence denotes number of mice in group with finding / total number of mice examined. Average severity score for the group is shown based on the following scoring system: 0 = not present, 1 = minimal, 2 = slight/mild, 3 = moderate, 4 = moderately severe, 5 = severe/high. Groups are arranged in descending order of severity within each post-exposure day, first by severity of focal subacute bronchiolar inflammation, and then by severity of focal bronchiolar pigmented macrophages.

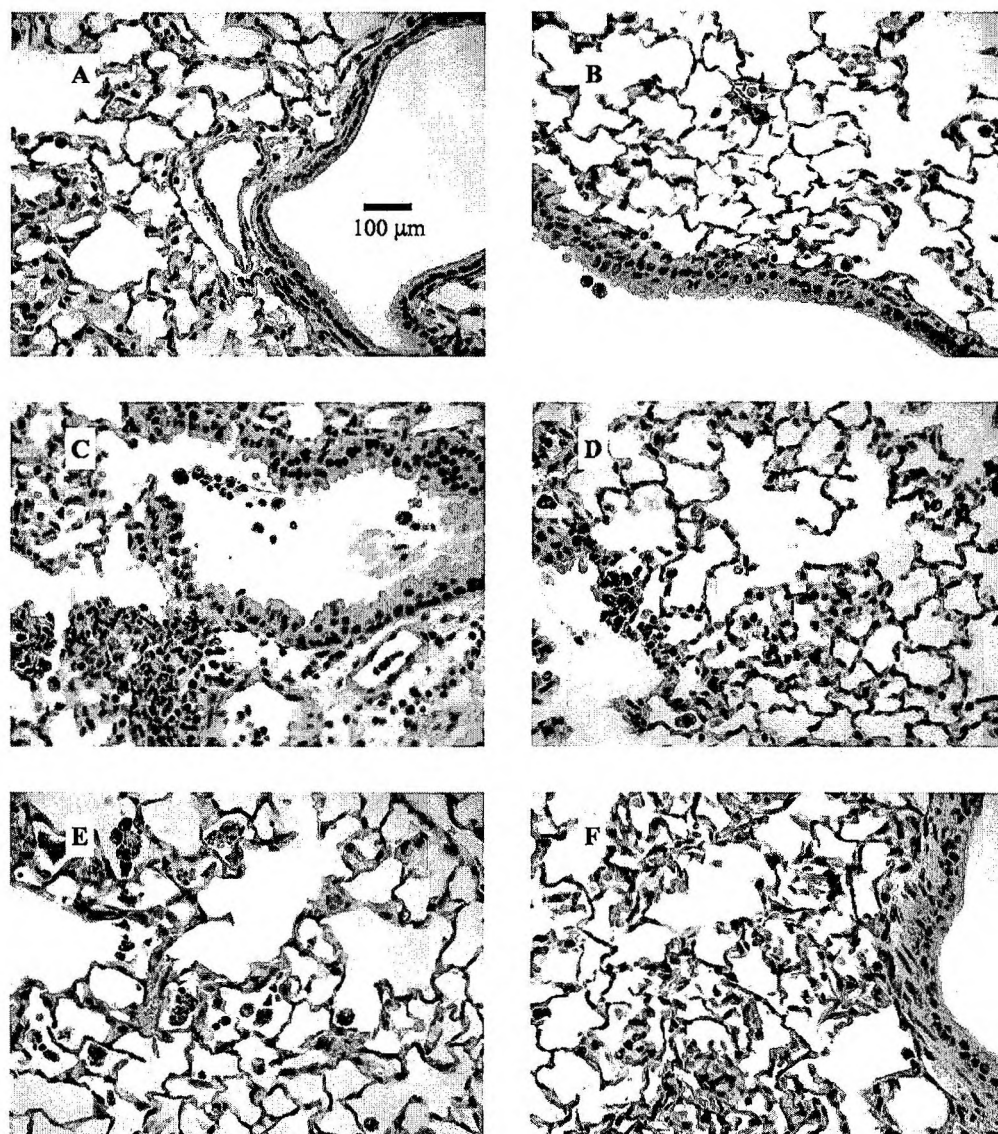


Figure 18. Experiment C. Representative micrographs of lesions occurring in lungs of mice 1 or 3 days after intratracheal instillation of 100 μ g PM sample or saline vehicle (all panels same magnification: bar length = 100 μ m). A. Saline-instilled control mouse (#301), Day 1, with no remarkable findings. B. Mouse #349 instilled with WTCC, Day1, showing minimal degree of focal subacute bronchiolar inflammation (FSBI). C. Mouse #291 instilled with NIST, Day 1, with moderate degree of FSBI. D. Mouse #270 instilled with WTC13, Day 1, with moderate degree of FSBI. E. Mouse #300 instilled with NIST, Day 3, with slight/mild degree of FSBI. F. Mouse #280 instilled with WTC13, Day 3, with minimal degree of FSBI.

lower Mch responsiveness (WTC13) was centrally located only 0.1 mile southeast of the center of Ground Zero. The WTCF sample was blown into a building at 120 Broadway and collected on an undisturbed marble staircase. The responses caused by this “indoor” sample were quite similar to those caused by the other “outdoor” WTC PM samples.

In general, responsiveness to Mch aerosol and pulmonary inflammation were not well correlated. Mice in the WTC13 group had one of the largest neutrophilic and eosinophilic responses, yet had a significantly lower degree of Mch responsiveness. Mice in the WTCC group had perhaps the greatest response to Mch challenge (not significantly different from WTCB, WTCE, or WTC11),

yet their neutrophil and eosinophil responses were low relative to the other WTC groups. As noted previously, a lack of correlation between inflammation and airway

hyperresponsiveness is not uncommon (Alvarez et al., 2000; Smith and McFadden Jr., 1995).

IV. Discussion

Samples of fallen dust were collected at various locations in the immediate vicinity of the WTC site one and two days after the WTC disaster, and were examined by several physical and chemical techniques. Both coarse unfractionated and fine size-fractionated WTC PM samples were composed primarily of calcium-based compounds such as calcium sulfate (gypsum) and calcium carbonate (calcite; the main component of limestone). These and other compounds and elements found in the WTC PM samples are indicative of crustal material-derived building materials such as cement, concrete aggregate, ceiling tiles, and wallboard. Both gypsum and calcite irritate the mucus membranes of the eyes, nose, throat, and upper airways (Stellman, 1998). Calcium carbonate dust causes coughing, sneezing, and nasal irritation (NLM, 2002). These minerals are often contaminated with small amounts of silica, which is the main concern for occupational health hazards (Stellman, 1998). Minor amounts of silica (quartz) were detected in the WTC PM samples.

Our chemical analysis generally agrees with the extensive analysis of WTC PM performed by the USGS (USGS, 2002). Levels of carbon were relatively low, suggesting that combustion-derived particles did not form a significant fraction of these samples recovered in the immediate aftermath of the destruction of the towers. Lastly, there was no evidence of significant asbestos contamination of the samples used in these studies, although the physical analyses conducted were not specifically focused on definitive asbestos quantitation. As of May 23, 2002, the U.S. EPA had analyzed 9,544 air samples in Lower Manhattan since September 11, and found elevated levels of asbestos in only 21 samples (EPA, 2002c).

The effects of exposure to samples of WTC PM_{2.5} on respiratory parameters, pulmonary inflammation, and lung injury were investigated in young adult female CD-1 mice, an outbred strain expected to have significant variability in biological responses, in three separate experiments. A pooled sample of WTC PM_{2.5} composed of roughly equivalent amounts of samples from 7 different locations

around the WTC site caused a mild degree of pulmonary inflammation in mice (7% neutrophils in BAL fluid), and had no effect on parameters of acute lung injury at a dose of 100 µg instilled directly into the lungs. ROFA, a toxic positive control fine PM sample, caused a much higher degree of lung inflammation and lung injury at the same dose. However, mice instilled with 100 µg pooled WTC PM_{2.5} had highly significant increases in airway responsiveness to methacholine (Mch) aerosol challenge, which were significantly greater than that of ROFA. Mice exposed to lower doses of pooled WTC PM_{2.5} (10 µg and 31.6 µg) and mice exposed by nose-only inhalation (estimated to have about 14 µg WTC PM_{2.5} deposited in the respiratory tract) did not have any biologically significant changes in methacholine responsiveness or neutrophilic inflammation. These dose-response relationships and the lack of effect in nose-only exposure suggest that inhalation of relatively high doses of WTC PM_{2.5} are necessary to elicit respiratory effects in people.

Mice exposed to samples of WTC PM_{2.5} from the 7 individual sites around Ground Zero had greater lung inflammation (2 to 4-fold) than mice exposed to the WTC PM_{2.5} sample pooled from these sites. These findings occurred in separate experiments and would need to be confirmed by a direct comparison, but nonetheless all groups of mice exposed to the individual site samples developed hyperresponsiveness to Mch aerosol challenge, similar to mice exposed to the pooled sample. No particular pattern of responses was found corresponding to the geographical location where the samples were taken. Pulmonary inflammation in mice exposed to individual site WTC PM_{2.5} samples diminished from 1 day to 3 days after exposure, although hyperresponsiveness to Mch aerosol did not diminish significantly. Further experiments would be necessary to determine the persistence of pulmonary responses in mice, which may lead to insights into whether any WTC PM-associated effects which may exist in people are persistent.

The results of these studies should be examined in the context of previous studies of the effects of

environmentally relevant PM samples in rodents. Rats were intratracheally instilled with 2.5 mg (~8.3 mg/kg) of various emission source and urban ambient air PM samples (Costa and Dreher, 1997), a dose about twice as high, based on body weight, as the 100 µg WTC PM_{2.5} dose in mice (~4 mg/kg). Oil fly ashes and urban ambient air PM samples (including a ROFA similar to the one used in the present study and NIST1649a) induced strong neutrophilic responses 24 hr after exposure, while biochemical markers of lung injury were lower in the urban air PM samples compared with the oil fly ash samples. ROFA at this dose induced airway hyperresponsiveness in rats which persisted at least 4 days, and was greater than that observed in an urban ambient air PM sample (Pritchard et al., 1996). The fact that WTC PM_{2.5} induced a significantly greater degree of airway hyperresponsiveness in mice than ROFA, which is used as a toxic positive control particle in many studies, suggests a very significant respiratory effect of a relatively high dose exposure to WTC PM_{2.5}.

Some people were exposed acutely to high concentrations of dust in the WTC disaster, and subsequently developed wheezing or symptoms of sensory irritation, such as cough and irritation of the nose and throat. These effects resemble, in some respects, the reactive airways dysfunction syndrome (RADS). RADS can occur after single or multiple high-level occupational exposures to an irritating vapor, fume, or smoke (Gautrin et al., 1999). Effects can occur within minutes or hours after exposure, and include cough, dyspnea, and wheezing. Clinical tests can show airways obstruction, persistent airway hyperresponsiveness, and inflammation. The recovery process appears to be dependent on the initial degree of injury. The effects of a high dose exposure to WTC PM_{2.5} in mice (100 µg) appear to mimic at least some of these responses, especially the significant increase in airway hyperresponsiveness to Mch. It is important to note that WTC PM_{2.5}-induced pulmonary inflammation, although significantly greater than in control mice, was not as robust as one might expect in a realistic animal model of RADS. However, the degree to which inflammation and airway hyperresponsiveness are associated in RADS is not clear (Gautrin et al., 1999). Examination of other time points would be necessary to determine the persistence of WTC PM-induced airway hyperresponsiveness in mice and its similarity to RADS.

Close examination of the data suggested that individual mice within the outbred CD-1 strain vary in sensitivity to the effects of WTC PM_{2.5}. Certain individuals within the human population may also have particular susceptibility to the hazards posed by exposure

to WTC PM_{2.5}. It is known that some asthmatic individuals are hyperresponsive to nonspecific irritants such as cold dry air (Anderson and Daviskas, 2000) or cigarette smoke (Bonham et al., 2001). This subpopulation is likely to be at high risk for development of dust-induced airways obstruction (Donaldson et al., 2000; Peden, 2001; Nel et al., 2001). Very few studies have been published regarding the effects of alkaline aerosols on pulmonary function in asthma. One study reported that inhalation of high concentrations of an alkaline aerosol (pH 9.8 to 10.3) had no significant effect on irritant symptoms or specific airways resistance in mild asthmatic patients (Eschenbacher, 1991). However, this aerosol was composed of a simple mixture of sodium carbonate, sodium bicarbonate, and sodium hydroxide. The chemical composition of the alkaline (pH 8.88 to 10.00) WTC PM_{2.5} is much more complex and interactions of numerous chemical species may be associated with development of airway hyperresponsiveness to methacholine or other bronchoconstrictors.

How does the dose of 100 µg WTC PM_{2.5}, which caused bronchiolar inflammation and airway hyperresponsiveness in mice, relate to exposure of people at the WTC site? Because inflammation was observed mainly in the airways, and airway hyperresponsiveness is mainly due to dysfunction of airway smooth muscle (Fredberg, 2000), the dose metric which is probably most relevant is dose per surface area of the tracheobronchial (TB) region of the respiratory tract. The TB region is defined as the airways (excluding the nasal (head) region) from the trachea down to the terminal bronchioles (Overton et al., 2001). Therefore, to assess the risks of exposure in people, the concentrations of WTC PM_{2.5} in air which could produce doses per TB surface area in humans equivalent to that in mice should be calculated. These WTC PM_{2.5} concentrations may be estimated (Table 22) using the following assumptions: 1) The mouse alveolar pulmonary surface area can be estimated from an allometric equation based on body weight (Jones and Longworth, 1992), and the TB surface area is very small in comparison to the alveolar surface area (Overton et al., 2001); 2) Oropharyngeal aspiration bypasses the mouse nose and spreads the dose of WTC PM_{2.5} evenly over the TB and pulmonary alveolar surface areas of the mouse lung; 3) The human TB dose per surface area, selected to match the mouse dose per surface area, does not clear from the lung in the time frame of exposure to WTC PM_{2.5} (an 8-hour work shift was selected); and 4) The model of the fraction of inhaled PM_{2.5} (model particles with MMAD = 1, σ_g = 2.5, and density = 1 g/cc) deposited in the TB region (Freijer et al., 1999) assumes a reference 30 year-

Table 22 Estimation of WTC PM_{2.5} Concentrations Required to Produce Human Doses Equivalent to Mouse Doses Used in WTC2001 Study

Dose deposited in mouse tracheobronchial and pulmonary regions (µg)	10	31.6	100
Mouse alveolar pulmonary surface area (m ²) ^a	0.103	0.103	0.103
Mouse dose per tracheobronchial (TB) or pulmonary surface area (mg/m ²) ^b	0.097	0.307	0.973
Human TB surface area (m ²) ^c	0.415	0.415	0.415
Total human TB dose equivalent to mouse TB dose (mg/m ² x m ²) ^d	0.040	0.128	0.404
Deposition fraction in human TB region ^e	0.066	0.066	0.066
Total inhaled dose in mg (total human TB dose / TB deposition fraction)	0.612	1.932	6.115
Quantity of air breathed in 8 hr workshift at ventilation of 30 L/min (m ³) ^f	14.4	14.4	14.4
WTC PM _{2.5} concentrations required to produce human doses equivalent to mouse doses used in WTC2001 Study (µg/m ³)	42	134	425

^a From Jones and Longworth (1992) calculated allometric equation: Mammalian alveolar pulmonary surface area in m² = 3.36 x (Wt, kg)^{0.935}, where weight = 0.024 kg (average mouse weight in all studies). Tracheobronchial surface area is minimal in comparison to alveolar surface area and can be ignored in calculation.

^b Assumes dose is spread out evenly over tracheobronchial and pulmonary alveolar regions.

^c Based on 30 year old, 5' 10" male with functional residual capacity (FRC) of 3300 ml (Overton et al., 2001).

^d Calculations assume no clearance of particles after deposition in human respiratory tract.

^e Calculations made with Multiple Path Particle Deposition model version 1.11 (Freijer et al., 1999) which assume human Yeh-Schum 5-lobe model, FRC = 3300 ml (appropriate for 30 year old, 5'10" male), upper respiratory tract volume = 50 ml, density of particles = 1 gm / cc, diameter = 1 µm MMAD, inhalability adjustment on, σ_g = 2.5, breathing frequency = 15 min⁻¹, tidal volume = 2000 ml, minute volume = 30 L/min, inspiratory:expiratory ratio = 1, and oronasal mouth breathing.

^f Estimate of minute ventilation during moderate to heavy sustained work (Åstrand and Rodahl, 1986).

old 5' 10" male breathing oronasally with a minute ventilation of 30 L/min (estimate during moderate to heavy sustained work; Åstrand and Rodahl, 1986). The total human TB dose and the fraction deposited in the TB region are used to back-calculate the total inhaled dose of PM_{2.5}. The total inhaled dose divided by the quantity of air breathed in a typical 8-hour work shift yields the concentration of PM_{2.5} in the WTC work or neighborhood environment required to produce human doses equivalent to the mouse doses used in the WTC2001 study (Table 22). These calculations show that under these conditions, concentrations of 42, 134, and 425 µg/m³ WTC PM_{2.5} would produce human doses per TB surface area equivalent to the mouse doses of 10, 31.6, and 100 µg, respectively. Obviously many factors may cause wide variations in the calculation of dose, and extrapolation of responses from the mouse to the human involves another dimension of uncertainty which was not considered, but it seems reasonable to say that a healthy worker breathing heavily in the dusty environment generated after the collapse of the towers could have inhaled enough PM_{2.5} to

approximate the 100 µg dose in the mouse. Therefore, inhalation of a very high concentration of WTC PM_{2.5} (e.g. ~425 µg/m³) over a short period of time (8 hr) could have contributed to development of pulmonary inflammation, airway hyperresponsiveness, and manifestations of sensory irritation such as cough. Individuals who are especially sensitive to inhalation of dusts, such as asthmatics, may experience these effects at lower doses of inhaled WTC PM_{2.5}. However, most healthy people would not be expected to respond to moderately high WTC PM_{2.5} levels (130 µg/m³ or less for 8 hours) with any adverse respiratory responses. The effects of chronic or repeated exposures to lower levels of WTC PM_{2.5}, or the persistence of any respiratory effects are unknown and were not components of this study. The persistence of any effects of inhaled WTC PM_{2.5}, if similar to RADS, would be expected to depend on the dose initially deposited in the respiratory tract.

It is important to consider several limitations of these studies. First, most of the experiments used oropharyngeal aspiration to deliver PM samples to the respiratory tract

rather than more physiologically relevant inhalation exposure methodology. We believe that utilizing oropharyngeal aspiration, as described in the Experimental Design section, had many advantages and was necessary in these circumstances. However, this report indicates that future studies may be needed to more closely examine bronchoconstriction and sensory irritation during inhalation exposure to WTC PM in mice and in guinea pigs, a species known to be especially sensitive to sensory irritants (Costa and Schelegle, 1999). Secondly, these studies only evaluated short-term toxicological effects (endpoints were examined 1, 3, or 6 days after exposure) after acute exposure and no direct information is provided on the long term effects of acute or chronic exposures to WTC PM_{2.5}. Thirdly, gaseous and vapor-phase toxicants (e.g. dioxin and volatile organic compounds such as benzene) were certainly released, especially during the fires which continued for months after September 11 (EPA, 2002c). The collection and processing techniques described in this report do not allow investigation of these important toxic species, nor are the interactions of particles with gases or organic vapors considered (Mautz et al., 2001). Finally, these studies only examined fine PM_{2.5}, while the toxicity of coarse mode and larger size PM fractions were not investigated. However, it is important to remember that the size-fractionation

techniques employed in this report are not absolute, and significant quantities of PM > 2.5 µm are present in the samples. Furthermore, analysis of the WTC PM_{2.5} and PM₃ samples showed that they were similar in composition (Tables 3 and 5), suggesting that only differences in respiratory tract deposition patterns of fine and coarse WTC PM would affect biological responses. Coarse mode PM may be more relevant for upper airways sensory irritation because larger particles will mainly deposit in the upper airways where sensory innervations are predominant (Costa and Schelegle, 1999). However, chronic effects of fine PM may be greater than coarse PM since it can be inhaled more deeply and deposit in peripheral regions of the lungs, and is more slowly cleared. Coarse PM is much less inhalable in small rodents than in humans, and less is deposited in the respiratory tract (Menache et al., 1995). Consequently, interpretation of results derived from exposure of mice to coarse PM is problematic, and small rodents are probably not the ideal species to study effects of coarse PM. Nevertheless, because upper airways irritant responses seem to be so important in people exposed to WTC-derived dust, future studies should examine the specific toxicity of coarse WTC PM on respiratory responses in appropriate animal models.

V. Quality Assurance Statement

U.S. EPA World Trade Center Research Project:

“Toxicological Effects of Fine Particulate Matter Derived from the Destruction of the World Trade Center”

Page 1 of 2

The study “Toxicological Effects of Fine Particulate Matter Derived from the Destruction of the World Trade Center” was conducted by the Pulmonary Toxicology Branch, Experimental Toxicology Division (ETD), National Health and Environmental Effects Research Laboratory (NHEERL), Office of Research and Development, U.S. Environmental Protection Agency, Research Triangle Park, NC, in compliance with NHEERL QA Guidelines. Results of these inspections were reported directly to the Principal Investigator (PI) of the Study, Dr. Stephen Gavett. Critical phases in the study were audited.

Date of Inspection	Item Inspected
October 29, 2001	Particulate Matter (PM) filters delivered to EPA.
October 31, 2001	Attempted scraping of PM from filters.
November 2, 2001	Approval of study protocol.
November 2, 2001	Extraction of PM from filters.
November 8, 2001	Shipment of WTC dust samples for endotoxin testing.
November 5-14, 2001	Conduct of Experiment A1-A3: weighing of samples and mice, randomization of mice, dosing of mice, BUXCO, DLCO, BAL, cell counts, methacholine responses, lung samples.
November 15, 2001	Delivery of NIST samples and blank filters.
November 19, 2001	Delivery of #3 Cortland Sample. Shipment of NIST samples for endotoxin testing.
November 26, 2001	Delivery of Experiment B inhalation sample (WTC 3).
November 27, 2001	Conduct of Experiment B (Day 0): placing and removal of mice in inhalation chambers, operation of inhalation pump. Shipment of #3 Cortland Sample Back to NYU.
December 3, 2001	Completion of Experiment B (Day 6): Nasal fixation. Receipt of endotoxin results on WTC dust samples.

Quality Assurance Statement

Page 2 of 2

Date of Inspection	Item Inspected
December 4, 2001	XRF/XRD laboratory tour. Shipment of 96 mice heads to Michigan State University.
December 11, 2001	Conduct of Experiment C (Day 0): Dosing of Mice. XRF/XRD technical meeting. Receipt of NIST endotoxin test results.
December 27, 2001	Shipment of 184 lung tissues for histopathological analysis.
January 15-17, 2002	Technical Systems Review of project. Interviews with study personnel and inspection of project data and records.
January 16, 2002	Shipment of six PM samples, and 12 PM samples and 10 filter samples for chemical analysis.
January 16-25, 2002	Data audit of spreadsheets against notebooks.
January 30, 2002	Shipment of 10 liquid samples for chemical analysis.
March 15, 2002	Transfer of custody of 12 PM Samples from the EPA Chemist to the PI.
March 4-19, 2002	Data audit of Draft Final Report.

The Quality Assurance Manager of ETD and the Director of Quality Assurance for NHEERL have determined by the above review process that the conduct of this project was in compliance with EPA quality requirements and the operating procedures and study protocol (Intramural Research Protocol No.: IRP-NHEERL-H/ETD/PTB/SHG/01-01-000). Furthermore, the results accurately reflect the raw data obtained during the course of the study.

_____/ s /_____
Thomas J. Hughes, ETD QA Manager

_____/ 03/22/2002_____
Date

_____/ s /_____
Brenda T. Culpepper, NHEERL Director of QA

_____/ 03/22/2002_____
Date

VI. References

- Adamson IY and Bowden DH (1981). Dose response of the pulmonary macrophagic system to various particulates and its relationship to transepithelial passage of free particles. *Exp. Lung Res.* 2:165-175.
- Alvarez MJ, Olaguibel JM, Garcia BE, Rodriquez A, Tabar AI, and Urbiola E (2000). Airway inflammation in asthma and perennial allergic rhinitis. Relationship with nonspecific bronchial responsiveness and maximal airway narrowing. *Allergy* 55:355-362.
- Anderson SD and Daviskas E (2000). The mechanism of exercise-induced asthma is ... *J. Allergy Clin. Immunol.* 106:453-459.
- Asgharian B, Wood R, and Schlesinger RB (1995). Empirical modeling of particle deposition in the alveolar region of the lungs: a basis for interspecies extrapolation. *Fundam. Appl. Toxicol.* 27:232-238.
- Åstrand P-O, and Rodahl K (1986). *Textbook of Work Physiology* (3rd ed.). New York: McGraw-Hill.
- Biran R., Tang YZ, Brook JR, Vincent R, and Keeler GJ (1996). Aqueous extraction of airborne particulate matter collected on hi-vol teflon filters. *Intern. J. Environ. Anal. Chem.* 63:315-322.
- Birch ME and Cary RA (1996). Elemental carbon-based method for monitoring occupational exposures to particulate diesel exhaust. *Aerosol Sci. Technol.* 25:221-241.
- Bonham AC, Chen CY, Mutoh T, and Joad JP (2001). Lung C-fiber CNS reflex: role in the respiratory consequences of extended environmental tobacco smoke exposure in young guinea pigs. *Environ. Health Perspect.* 109 Suppl 4:573-578.
- Budavari S, ed. (1996). *The Merck Index*. ISBN 0911910123. Whitehouse Station, NJ: Merck & Co., Inc.
- Costa DL, Tepper JS, and Raub JA (1992). Interpretations and limitations of pulmonary function testing in small laboratory animals. In: Parent RA, ed. *Comparative Biology of the Normal Lung*. Boca Raton, FL: CRC Press:367-399.
- Costa DL and Dreher KL (1997). Bioavailable transition metals in particulate matter mediate cardiopulmonary injury in healthy and compromised animal models. *Environ. Health Perspect.* 105 Suppl. 5: 1053-1060.
- Costa DL and Schelegle ES (1999). Irritant air pollutants. In: Swift DL and Foster WM, eds. *Air Pollutants and the Respiratory Tract* (ISBN 0824795210). New York: Marcel Dekker: 119-145.
- Dhingra VK, Uusaro A, Holmes CL, and Walley KR (2001). Attenuation of lung inflammation by adrenergic agonists in murine acute lung injury. *Anesthesiology* 95:947-953.
- Dockery DW, Pope CA 3rd, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, and Speizer FE (1993). An association between air pollution and mortality in six U.S. cities. *N. Engl. J. Med.* 329:1753-1759.
- Donaldson K, Gilmour MI, and MacNee W (2000). Asthma and PM10. *Respir. Res.* 1:12-15.
- Dreher KL, Jaskot RH, Lehmann JR, Richards JH, McGee JL, Ghio AJ, and Costa DL (1997). Soluble transition metals mediate residual oil fly ash induced acute lung injury. *J. Toxicol. Environ. Health.* 50:285-305.
- Driscoll KE, Costa DL, Hatch G, Henderson R, Oberdorster G, Salem H, and Schlesinger RB (2000). Intratracheal instillation as an exposure technique for the evaluation of respiratory tract toxicity: uses and

- limitations. *Toxicol. Sci.* 55:24-35.
- Eschenbacher WL, Gross KB, Muench SP, and Chan TL (1991). Inhalation of an alkaline aerosol by subjects with mild asthma does not result in bronchoconstriction. *Am. Rev. Respir. Dis.* 143:341-345.
- EPA (2002a). Environmental Protection Agency website. EPA Publication SW-846: Test methods for evaluating solid waste, physical/chemical methods. Method 6020a: Inductively Coupled Plasma - Mass Spectrometry. <http://www.epa.gov/epaoswer/hazwaste/test/6020a.pdf> (accessed February 2002).
- EPA (2002b). Environmental Protection Agency website. EPA Publication EPA/600/4-91/010: Methods for the determination of metals in environmental samples. Method 200.7: Determination of metals and trace elements by inductively coupled plasma - atomic emission spectrometry. Online version of EPA Method 200.7 is available by searching: <http://www.epa.gov/cgi-bin/claritgw?op-NewSearch&template=epa> (accessed Feb. 2002).
- EPA (2002c). Environmental Protection Agency website. EPA Response to September 11: Daily Environmental Monitoring Summary. http://www.epa.gov/wtc/data_summary.htm (accessed May 2002).
- Foster WM, Walters DM, Longphre M, Macri K, and Miller LM (2001). Methodology for the measurement of mucociliary function in the mouse by scintigraphy. *J. Appl. Physiol.* 90:1111-1117.
- Fredberg JJ (2000). Frozen objects: small airways, big breaths, and asthma. *J. Allergy Clin. Immunol.* 106:615-624.
- Freijer JI, Cassee FR, Subramaniam R, Asghararian B, Anjilvel S, Miller FJ, van Bree L, and Rombout PJA (1999). Multiple path particle deposition model (MPPDep version 1.11): a model for human and rat airway particle deposition. Bilthoven, The Netherlands: RIVM Publication 650010019. http://www.rivm.nl/index_en.html (accessed March 2002).
- Gavett SH, Madison SL, Dreher KL, Winsett DW, McGee JK, and Costa DL (1997). Metal and sulfate composition of residual oil fly ash determines airway hyperreactivity and lung injury in rats. *Environ. Res.* 72:162-172.
- Gavett SH, Madison SL, Stevens MA, and Costa DL (1999). Residual oil fly ash amplifies allergic cytokines, airway responsiveness, and inflammation in mice. *Am. J. Respir. Crit. Care Med.* 160:1897-1904.
- Gautrin D, Bernstein IL, and Brooks S (1999). Reactive airways dysfunction syndrome, or irritant-induced asthma. In: Bernstein IL, Chan-Yeung M, Malu J-L, and Bernstein DI., eds. *Asthma in the Workplace*. 2nd ed. New York: Marcel Dekker: 565-593.
- Graham JA, Miller FJ, Davies DW, Hiteshew ME, and Walsh III LC (1985). Inhalation studies of Mt. St. Helens volcanic ash in animals. I. Introduction and exposure system. *Environ. Res.* 37:61-71.
- Hamelmann E, Schwarze J, Takeda K, Oshiba A, Larsen GL, Irvin CG, and Gelfand EW (1997). Noninvasive measurement of airway responsiveness in allergic mice using barometric plethysmography. *Am. J. Respir. Crit. Care Med.* 156:766-775.
- Hatch GE, Raub JA, Graham JA (1984). Functional and biochemical indicators of pneumoconiosis in mice: comparison with rats. *J. Toxicol. Environ. Health* 13:487-497.
- Henderson RF, Benson JM, Hahn FF, Hobbs CH, Jones RK, Mauderly JL, McClellan RO, and Pickrell JA (1985). New approaches for the evaluation of pulmonary toxicity: bronchoalveolar lavage fluid analysis. *Fundam. Appl. Toxicol.* 5:451-458.
- Jones JH and Longworth KE (1992). Gas exchange at rest and during exercise in mammals. In: Parent RA, ed. *Comparative Biology of the Normal Lung*. Boca Raton, FL: CRC Press:271-307.
- Kodavanti UP, Hauser R, Christiani DC, Meng ZH, McGee J, Ledbetter A, Richards J, and Costa DL (1998). Pulmonary responses to oil fly ash particles in the rat differ by virtue of their specific soluble metals. *Toxicol. Sci.* 43:204-212.
- Ledbetter AD, Killough PM, and Hudson GF (1998). A

- low-sample-consumption dry-particulate aerosol generator for use in nose-only inhalation exposures. *Inhal. Toxicol.* 10:239-251.
- Levitzky MG (1995). Chapter 6: Diffusion of Gases. In: Houston MJ and Sheinis LA, eds. *Pulmonary Physiology (Fourth Edition)*. New York, NY: McGraw-Hill: 130-141.
- Mamane Y, Willis RD, and Conner TL (2001). Evaluation of computer-controlled scanning electron microscopy applied to an ambient urban aerosol sample. *Aerosol Sci. Technol.* 34:97-107.
- Mautz WJ, Kleinman MT, Bhalla DK, and Phalen RF (2001). Respiratory tract responses to repeated inhalation of an oxidant and acid gas-particle air pollutant mixture. *Toxicol. Sci.* 61:331-341.
- McKetta JJ, ed. (1978). *Encyclopedia of Chemical Processing and Design* (ISBN 0824724577). New York, NY: Marcel Dekker: Cements, Portland - 7:82-93; Concrete - 11:150-160.
- Menache MG, Miller FJ, and Raabe OG (1995). Particle inhalability curves for humans and small laboratory animals. *Ann. Occup. Hyg.* 39:317-328.
- Nel AE, Diaz-Sanchez D, and Li N (2001). The role of particulate pollutants in pulmonary inflammation and asthma: evidence for the involvement of organic chemicals and oxidative stress. *Curr. Opin. Pulm. Med.* 7:20-26.
- New York Times (2001). At least a quarter of ground zero firefighters ill. By Tina Kelley. December 21, 2001.
- NIST (2001). National Institute of Standards and Technology. Certificate of Analysis: Standard Reference Material 1649a. National Institute of Standards and Technology, Gaithersburg, Maryland. Pp. 1 - 22.
- NIST (2002). National Institute of Standards and Technology website. "Table 113.1 Cements (powder form)". <http://srmcatalog.nist.gov/srmcatalog/tables/113-1.htm> (Accessed February 2002).
- NLM (2002). National Library of Medicine. Hazardous substances data bank. <http://toxnet.nlm.nih.gov/> (accessed April 2002).
- Overton JH, Kimbell JS, and Miller FJ (2001). Dosimetry modeling of inhaled formaldehyde: the human respiratory tract. *Toxicol. Sci.* 64:122-134.
- Peden DB (2001). Air pollution in asthma: effect of pollutants on airway inflammation. *Ann. Allergy Asthma Immunol.* 87(6 Suppl 3):12-17.
- Pritchard RJ, Ghio AJ, Lehmann JR, Winsett DW, Tepper JS, Park P, Gilmour MI, Dreher KL, and Costa DL (1996). Oxidant generation and lung injury after particulate air pollutant exposure increase with the concentrations of associated metals. *Inhal. Toxicol.* 8:457-477.
- Raabe OG (1999). Respiratory exposure to air pollutants. In: Swift DL and Foster WM, eds. *Air Pollutants and the Respiratory Tract* (ISBN 0824795210). New York, NY: Marcel Dekker: 39-73.
- Raabe OG, Al-Bayati MA, Teague SV, and Rasolt A (1988). Regional deposition of inhaled monodisperse coarse and fine aerosol particles in small laboratory animals. In: Dodgson J, McCallum RI, Bailey MR, and Fisher DR, eds. *Inhaled Particles VI*. Oxford, UK: Pergamon Press: 53-63.
- Raub JA, Hatch GE, Mercer RR, Grady M, and Hu P-C (1985). Inhalation studies of Mt. St. Helens volcanic ash in animals. II. Lung function, biochemistry, and histology. *Environ. Res.* 37:72-83.
- Schlesinger RB (1985). Comparative deposition of inhaled aerosols in experimental animals and humans: a review. *J. Toxicol. Environ. Health* 15:197-214.
- Sears MR (1997). Descriptive epidemiology of asthma. *Lancet* 350 (supp. II):1-4.
- Smith L and McFadden ER Jr (1995). Bronchial hyperreactivity revisited. *Ann. Allergy Asthma Immunol.* 74:454-469.
- Stellman, JM, ed. (1998). *Encyclopaedia of Occupational Health and Safety*, 4th ed. Geneva, Switzerland: International Labour Office.
- Sunset (2002). Sunset Laboratory website. "Sample Analysis Method for Organic and Elemental Carbon

Aerosols”.

<http://www.sunlab.com/SampleAnalysisMethod.html>
(accessed February 2002).

USGS (2002). U. S. Geological Survey Open File Report OFR-01-0429. Environmental Studies of the World Trade Center area after the September 11, 2001 attack. <http://geology.cr.usgs.gov/pub/open-file-reports/ofr-01-0429/> (accessed March 2002).

Washington Post (2002). In N.Y., taking a breath of fear: illnesses bring new doubts about toxic exposure near

ground zero. By Christine Haughney. January 8, 2002; page A1.

Watkinson WP, Campen MJ, and Costa DL (1998). Cardiac arrhythmia induction after exposure to residual oil fly ash particles in a rodent model of pulmonary hypertension. *Toxicol. Sci.* 41:209-216.

Weast RC, ed. (1985). CRC Handbook of Chemistry and Physics. ISBN 0849304660. Boca Raton, FL: CRC Press, Inc.



Please make all necessary changes on the below label, detach or copy, and return to the address in the upper left-hand corner.

If you do not wish to receive these reports CHECK HERE ☐ ; detach, or copy this cover, and return to the address in the upper left-hand corner.

PRESORTED STANDARD
POSTAGE & FEES PAID
EPA
PERMIT No. G-35

Office of Research and Development
National Health and Environmental
Effects Research Laboratory
Research Triangle Park, NC 27711

Official Business
Penalty for Private Use
\$300

EPA 600/R-02/028
December 2002