Risk Analysis, Vol. 27, No. 5, 2007 DOI: 10.1111/j.1539-6924.2007.00956.x

Assessment of Inhalation Exposures and Potential Health Risks to the General Population that Resulted from the Collapse of the World Trade Center Towers

Matthew Lorber,[1]* Herman Gibb,[2] Lester Grant,[1] Joseph Pinto,[1] Joachim Pleil,[3] and David Cleverly[1]

In the days following the collapse of the World Trade Center (WTC) towers on September 11, 2001 (9/11), the U.S. Environmental Protection Agency (EPA) initiated numerous air monitoring activities to better understand the ongoing impact of emissions from that disaster. Using these data, EPA conducted an inhalation exposure and human health risk assessment to the general population. This assessment does not address exposures and potential impacts that could have occurred to rescue workers, firefighters, and other site workers, nor does it address exposures that could have occurred in the indoor environment. Contaminants evaluated include particulate matter (PM), metals, polychlorinated biphenyls, dioxins, asbestos, volatile organic compounds, particle-bound polycyclic aromatic hydrocarbons, silica, and synthetic vitreous fibers (SVFs). This evaluation yielded three principal findings. (1) Persons exposed to extremely high levels of ambient PM and its components, SVFs, and other contaminants during the collapse of the WTC towers, and for several hours afterward, were likely to be at risk for acute and potentially chronic respiratory effects. (2) Available data suggest that contaminant concentrations within and near ground zero (GZ) remained significantly elevated above background levels for a few days after 9/11. Because only limited data on these critical few days were available, exposures and potential health impacts could not be evaluated with certainty for this time period. (3) Except for inhalation exposures that may have occurred on 9/11 and a few days afterward, the ambient air concentration data suggest that persons in the general population were unlikely to suffer short-term or long-term adverse health effects caused by inhalation exposures. While this analysis by EPA evaluated the potential for health impacts based on measured air concentrations, epidemiological studies conducted by organizations other than EPA have attempted to identify actual impacts. Such studies have identified respiratory effects in worker and general populations, and developmental effects in newborns whose mothers were near GZ on 9/11 or shortly thereafter. While researchers are not able to identify specific times and even exactly which contaminants are the cause of these effects, they have nonetheless concluded that exposure to WTC contaminants (and/or maternal stress, in the case of developmental effects) resulted in these effects, and have identified the time period including 9/11 itself and the days and few weeks afterward as a period of most concern based on high concentrations of key pollutants in the air and dust.

KEY WORDS: Inhalation exposure; risk assessment; World Trade Center

[1] National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency, 1200 Pennsylvania Ave., Washington, DC 20460, USA.
[2] Sciences International, Alexandria, VA, 22314, USA.
[3] National Exposure Research Laboratory, Office of Research and Development, Research Triangle Park, NC, 27709, USA.
* Address correspondence to Mr. Matthew Lorber, U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC, USA; lorber.matthew@epa.gov.

1. INTRODUCTION

Environmental Protection Agency (EPA) Region 2, which includes the New York City metropolitan area in New York and New Jersey, is the EPA's lead office on activities associated with the collapse of the World Trade Center (WTC) towers. In November 2001, 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 collapse. The resulting evaluation was an inhalation exposure and human health risk assessment. That assessment was released as a public review draft in December 2002 (U.S. EPA, 2002a) and was reviewed by a sitting panel of independent experts in New York City in July 2003. During 2004 and 2005, the assessment was revised to reflect reviewer comments and new information that had come out of continuing studies on environmental and human health impacts from the collapse. Several aspects of this assessment have been published in the open literature (Lorber, 2003; Lorber *et al.*, 2004; Pleil *et al.*, 2004; Vette *et al.*, 2004a, 2004b; Pinto *et al.*, 2005); this article describes the final results of the assessment.

Numerous other efforts are ongoing or have been completed to address aspects of exposure and potential risk associated with the collapse of the WTC towers that are not addressed by this study. For example, this study does not assess ground zero (GZ) firefighter or rescue worker exposure. Data on worker exposures were collected by the Occupational Safety and Health Administration (OSHA; http://www.osha.gov/nyc-disaster/wtc.html) and the National Institute of Occupational Safety and Health (NIOSH; http://www.cdc.gov/niosh/wtcsampres.html). This evaluation also does not assess indoor exposures as would occur in apartments or offices. The Agency for Toxic Substances and Disease Registry (ATSDR) completed a study of indoor air quality in 2002 (ATSDR, 2002). Efforts by EPA Region 2 to clean apartments and also to evaluate the quality of indoor air were completed during 2002/2003 (U.S. EPA, 2003b) and efforts continue today (see http://www.epa.gov/wtc). Finally, epidemiologic studies of the exposed populations conducted by organizations other than EPA have provided a scientifically robust evaluation of two types of health effects that were attributed to WTC exposures: respiratory and developmental. These studies have addressed general population as well as worker exposures, and as epidemiological studies, they have identified health effects that resulted from outdoor as well as indoor exposures, from exposures to more than one contaminant, exposures to airborne contaminants as well as contaminants in dust, and so on. This article summarizes these studies as a way of providing a complete picture of the health impacts from collapse of the WTC towers, and a perspective to the contaminant-by-contaminant evaluations of outdoor ambient monitoring presented in this study.

Following a review of the methodology, an overview of general findings and contaminant-specific results is provided. A separate section describes epidemiological studies that have attempted to identify health impacts from exposed populations, including the general population and WTC worker populations. This article concludes with comments on uncertainty and a reiteration of the three primary conclusions of the assessment.

2. METHODOLOGY

The evaluation focuses on particulate matter (PM); metals (lead, chromium, and nickel compounds); polychlorinated biphenyls (PCBs); dioxin-like compounds (CDD/Fs); asbestos; volatile organic compounds (VOCs); particle-bound polycyclic aromatic hydrocarbons (PAHs); silica; and synthetic vitreous fibers (SVFs). These substances were included because monitoring indicated that they were present near GZ at levels significantly above background levels, and because they posed a potential concern for health impacts. The evaluation focuses on outdoor inhalation exposures incurred by the "general population," defined as individuals living and working in neighborhoods surrounding GZ. This evaluation does not address exposures and potential impacts that could have occurred to rescue workers, firefighters, and other site workers, and also to exposures that could have occurred in the indoor environment. In most instances, the evaluation involved comparing the measured air levels at locations near GZ with established health benchmarks for inhalation exposure, with typical urban background levels, and sometimes with occupational and regulatory standards. Established benchmarks for inhalation exposure include EPA's Air Quality Index (AQI), National Ambient Air Quality Standard (NAAQS), Asbestos Hazard Emergency Response Action (AHERA) Standard, and Reference Concentration (RfC); OSHA's Permissible Exposure Levels (PELs); NIOSH's Recommended Exposure Limits (RELs); and ATSDR's Minimal Risk Levels (MRLs). Of these benchmarks, those established to protect against acute and subchronic exposures to the general population are preferred because the focus of this assessment is on

this population and for a limited time period when air concentrations were elevated above typical background. Benchmarks that are intended to protect against exposures lasting more than 1 year or throughout a lifetime, such as RfCs, or those used primarily for occupational exposures, such as PELs and RELs, were only used if other more appropriate benchmark values were not available. In addition to this simple comparison, cancer inhalation risks were assessed for PCBs, asbestos, PAHs, and dioxin-like compounds. The dioxin assessment was additionally unique in that it included an exercise that examined the potential increase in body burdens of dioxin-like compounds as a result of the WTC-related exposure.

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 a large majority of samples (e.g., 99%) are much less than a benchmark (e.g., lower by an order of magnitude), then in most cases concluding that a health impact is unlikely is appropriate. On the other hand, if a significant number of samples exceed the benchmark (e.g., greater than 10%), then it may be appropriate to consider the possibility that a health impact may have occurred, or could occur, depending on the circumstances. Even a single exceedance of a benchmark may be of concern, particularly if the benchmark is for an acute effect, like a 24-hour AQI for PM. The evaluations below should be understood in that context; they do not predict disease or actual adverse health outcomes, they can only suggest where concerns may or may not lie.

In order to characterize exposure and risks, it became necessary to characterize the duration of exposure in conjunction with the location of the 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 September 14, this "restricted zone" included Lower Manhattan south of 14th Street. Pedestrian and vehicular traffic was limited to emergency management and rescue personnel and other credentialed people within restricted zones. The extent of the restricted zone diminished quickly, and after September 14, it extended only 3–5 blocks from GZ. By the end of October, essentially only GZ was restricted. Residency in homes located in the restricted zones was prohibited. Although some people in certain areas might have come and gone quickly (for example, to collect pets), no one was living or spending an extended amount of time in these areas unless they were part of the rescue, recovery, and cleanup operations. The evaluations below discuss whether exceedences of health benchmarks occurred within an area that was restricted when the sample was taken. The implication, of course, is that the general population would not be substantially exposed to elevated concentrations that occurred within restricted zones.

In addition to understanding restricted zone implications, also important is understanding the intent of monitoring. For example, samples were taken in order to characterize VOC emissions within smoldering piles on GZ and on other work areas within and bordering GZ to determine if the area was safe for entry by rescue workers and firefighters. These "grab samples," as they were termed, comprise the bulk of all measurements for VOCs. They were taken by mobile monitors in short time periods, usually about 4 minutes, for the purpose of obtaining a quick analysis to determine the safety of work areas. When these samples showed extremely high concentrations of VOCs, entry into those locations was prohibited. Because the purpose of the monitoring was to evaluate the safety of the work areas, high concentrations found by grab sample monitoring are inappropriate for evaluating general population exposure. Still, high levels from these grab samples are noted in summaries below with the disclaimer that they do not represent exposure levels.

3. FINDINGS

3.1. Early Monitoring

Ambient monitoring within and near GZ did not begin immediately. 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 collapse of the WTC towers. A small number of dust samples were collected for analysis on 9/11, and some air samples were taken in Brooklyn and New Jersey also on 9/11. However, the first air samples of some of the critical contaminants, such as asbestos, were not taken within and near GZ until September 14, while other contaminants, such as PAHs, dioxins, and others, were not sampled until September 16. Fig. 1 shows a timeline of when the contaminants in this assessment were first collected within and near GZ. There were some existing PM monitoring stations operational in New York City, mostly at public schools far from the WTC

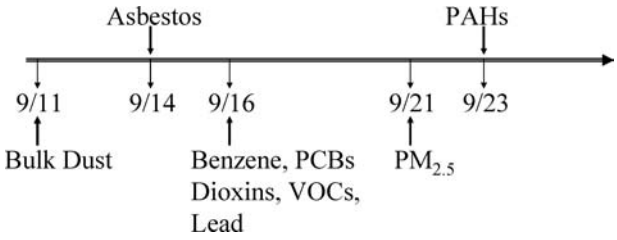

Fig. 1. Timeline showing when sampling began for key contaminants in the vicinity of ground zero (GZ).

site in Lower Manhattan, and PM results from 9/11 onward in these samplers were examined for trends.

An examination of the concentrations measured during September show that the highest concentrations were the ones taken closest in time to 9/11 and closest in proximity to GZ. For example, the first measurements of dioxin in the monitors nearest GZ were the highest measurements taken in the entire program, and they were the highest ambient measurements of dioxin ever recorded anywhere in the world. Five measurements of dioxin toxic equivalent (TEQ) concentrations, in particular, were more than 100 pg TEQ/m^3; all others were less than 100 pg TEQ/m^3. These five were among the first measurements in the three nearest downwind monitors: the first three measurements in the WTC Building 5 monitor on September 23 (160 pg TEQ/m^3), October 2 (170 pg TEQ/m^3), and October 4 (170 pg TEQ/m^3); the second measurement at the Church and Dey Streets monitor on September 23 (130 pg TEQ/m^3; the first measurement on September 16 was elevated as well at 60 pg TEQ/m^3); and the first measurement at the Liberty Street and Broadway monitor on September 23 (100 pg TEQ/m^3). In contrast to these measurements, typical urban concentrations of dioxin are about 0.1 pg TEQ/m^3, and elevated measurements downwind of incinerators have been measured in the range of 1–5 pg TEQ/m^3 (Smith *et al.*, 1989; Walker *et al.*, 2002). Similar observations showing that the highest measurements of all the contaminants evaluated here were the first measurements taken are included in the contaminant-specific discussions below.

3.2. Particulate Matter, PM

People caught in the initial dust/smoke cloud that encompassed Lower Manhattan after collapse of the WTC buildings on 9/11 were exposed for several hours (4–8 hours) to very high levels of airborne PM. The dust cloud was optically dense, as can be seen from 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 mg/m^3 (thousands of μg/m^3). The following formula relates visibility to the concentration of $PM_{2.5}$ (Stevens *et al.*, 1984):

$$0.5(\text{km} - \text{mg/m}^3) = \text{V(km)}^* \text{C(mg/m}^3), \quad (1)$$

where V is the visibility range (km) and C is the concentration of $PM_{2.5}$ (mg/m^3). During the collapse of the WTC towers, visibilities were reduced to less than 100 m (about 1 city block) on many streets. Assuming that visibility on streets in lower Manhattan affected by the dust cloud was controlled by fine particles, then application of the above formula indicates that $PM_{2.5}$ concentrations could have been about 5 mg/m^3 (5,000 μg/m^3). However, the collapse of the WTC towers mainly produced coarse particles (Lioy *et al.*, 2002) that, as mentioned above, are less effective than fine particles in controlling visibility. Thus, a value of 5,000 μg/m^3 represents a lower limit on the abundance of total PM, and it seems likely that total PM concentrations could exceed 5,000 μg/m^3 at times during the first several hours after the WTC collapse.

Measurements of PM from permanent monitors established within and at the perimeter of GZ were not available until September 21. However, Wolff *et al*. (2005) modeled the exposures to PM of a cohort of 187 pregnant women who worked or lived near GZ from September 11 to October 8. A complex air dispersion model was used to simulate the dispersion of PM emitted from the collapse of the towers, and satellite photography was used to determine the area affected by these PM emissions. The results of these analyses were combined with self-reported time-activity patterns of the 187 women to give an Exposure Index, or EI. This exercise suggests that this cohort experienced a more than 100-fold decrease in EI between September 11, after the collapse of the WTC Towers, and September 14. This drop was due to the fact that these women left the area and were not exposed to the remaining high levels in the air. The EI dropped further because of rain on September 16 and then increased to within 10 to 100-fold of initial post-collapse September 11 values through October. The increase in EI after the rain on September 16 was not due to an increase in concentration of particulates but rather to an increase in reported activities near GZ as the women returned to their jobs and their homes.

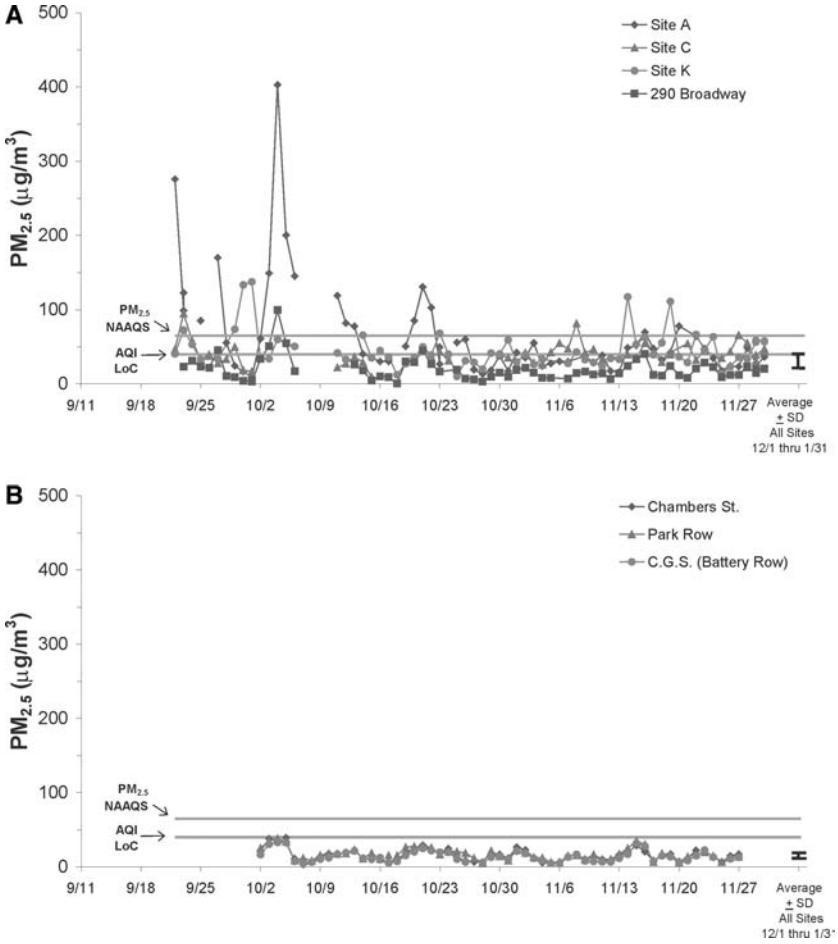

Fig. 2. *Panel A (top)*: Daily $PM_{2.5}$ concentrations monitored at sites designated "A" (directly north of GZ), "C" (south), and "K"(southwest) on GZ perimeter and at 290 Broadway 6 blocks northeast of GZ. *Panel B (bottom):* $PM_{2.5}$ concentrations observed at several extended monitoring network sites in Lower Manhattan within 3 to 10 blocks of GZ.

Monitoring results near GZ were available from nine monitors after September 21, and results from three established monitors at more distant locations were also available for the entire period of the collapse and its aftermath. During the first several days after September 21, PM levels in the air at the GZ perimeter were very high: daily average $PM_{2.5}$ concentrations were typically above 40 $\mu g/m^3$ (the EPA AQI level of concern) except when it rained, with occasional excursions in the range from 100–400 $\mu g/m^3$. Fig. 2 shows a subset of the 24-hour measurements of $PM_{2.5}$ at sites on the perimeter of GZ (Panel A of Fig. 2) and then at locations 3–10 blocks from GZ (Panel B). The measurements at the GZ perimeter exceeded EPA's daily $PM_{2.5}$ NAAQS of 65 $\mu g/m^3$, and $PM_{2.5}$ concentrations at some other nearby Lower Manhattan sites exceeded EPA's 40 $\mu g/m^3$ 24-hour AQI. Even into November, the average $PM_{2.5}$ concentration at sites on the perimeter of GZ were above 40 $\mu g/m^3$, with occasional excursions above 100 $\mu g/m^3$ (see Fig. 1).

Beyond 10 blocks, elevations in PM were also noted, but these elevations were not out of line with historic levels in Lower Manhattan. Fig. 3 shows monitoring results from Public School 64, which is about 5 km (3 miles) in the predominant northeast, downwind direction from GZ. Whereas elevations above 40 $\mu g/m^3$ occurred on September 13 and October 4 at this location, with both elevations attributed to the WTC collapse (based on plume movement evaluations), it is easily seen from the lower portion of Fig. 3 that the frequency and magnitude of such excursions were not out of line with historic trends of $PM_{2.5}$ concentrations at that site.

PM_{10} (PM $\leq$ 10 μm in diameter) measurements were initiated at several sites in Lower Manhattan on October 2. At no time did PM_{10} concentrations at these sites exceed the daily NAAQS for PM_{10} of 150 $\mu g/m^3$.

The issue of alkalinity also arises for WTC dust, including the inhalable size $PM_{2.5}$ particles. McGee *et al.* (2003) found that pH levels of water-extracted

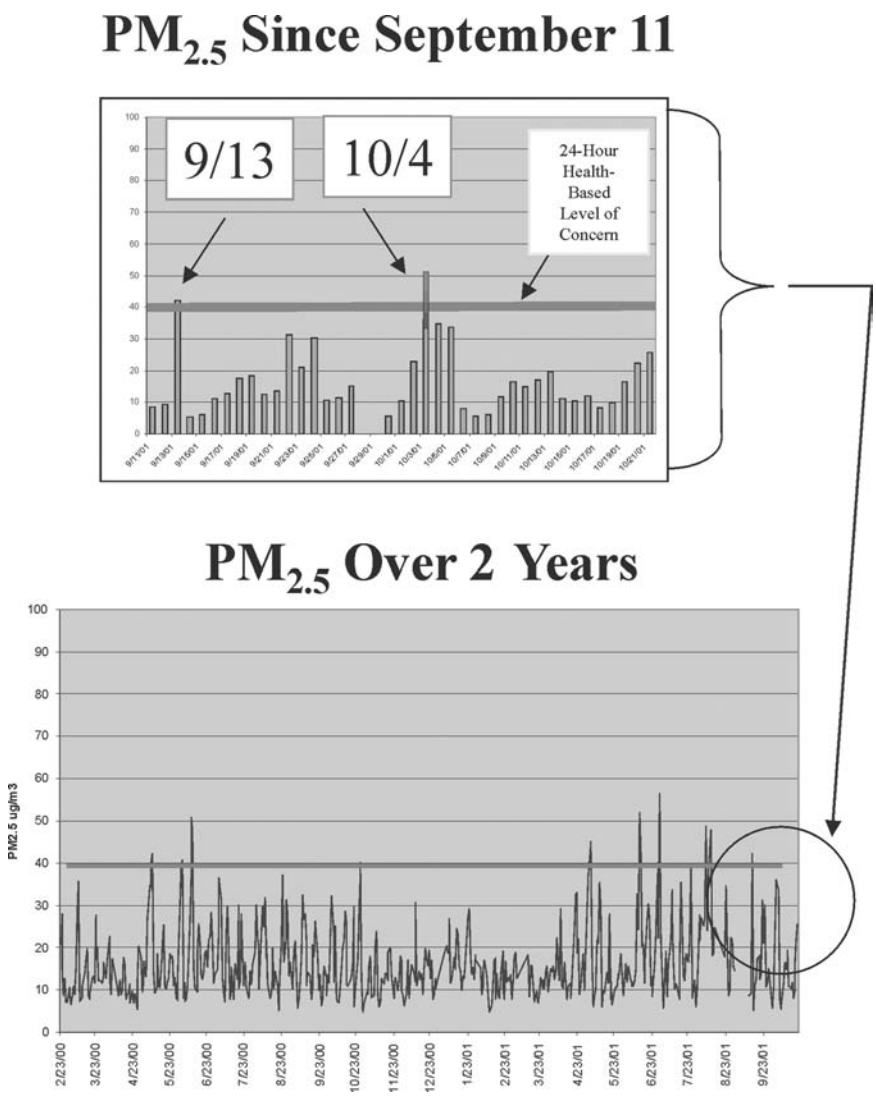

Fig. 3. Daily $PM_{2.5}$ concentrations recorded at NYSDEC PS 64 monitoring site after September 11, 2001 (9/11/01 to 10/27/01) compared to historic record of 24-hour $PM_{2.5}$ values at the same site during prior 2 years (2/23/00 to 9/01/01).

$PM_{2.5}$ before lyophilization ranged from 8.88–10.00. They state that the alkaline pH results from the building materials comprising much of the dust, most likely the alkaline earth (calcium, magnesium) compounds, as well as calcium carbonate, which is a major component of cement and other building materials. Chen and Thurston (2002) state that the pH of most of the suspensions of the WTC settled dust were greater than 10. They found that the dust's alkalinity decreased with decreasing particle size, with particles less than 2.5 μm at about neutral pH. Additional health concern arises not only because of the alkaline nature of some constituent particles but also because of other unusual features, such as that the particles included slender microscopic glass fibers with toxic materials attached to them or that the very fine particles were composed of unusual combinations of silica coalesced with lead or other toxic materials. Chen and Thurston (2002) suggest that the properties of the WTC dust were responsible for the chronic cough noted for WTC workers in the months after 9/11.

3.3. Lead (Pb), Chromium (Cr), and Nickel (Ni)

Persons caught in the initial WTC-related dust cloud likely experienced brief exposures to high levels of Pb, based on analyses of deposited dust samples. In late September 2001, air Pb concentrations at the WTC perimeter sites reached levels above the EPA NAAQs for Pb of 1.5 $\mu g/m^3$ on some days. However, the air Pb levels averaged over 90 days (late September through late November) did not exceed

1.5 $\mu g/m^3$. After mid-October, air Pb at all sites in Lower Manhattan outside GZ dropped to levels more comparable with background concentrations typical of New York City and other northeastern U.S. urban areas. On the basis of ambient air and dust data, there is little indication of substantial health risks associated with Pb exposures to the general population in Lower Manhattan areas around the WTC site. The same is true of the other two metals evaluated, Cr and Ni. Samples evaluated for total Cr at GZ and at sites surrounding GZ never exceeded the NIOSH REL of 0.5 mg/m^3 or the OSHA PEL of 1 mg/m^3 for Cr metal and insoluble salts, or the ATSDR Intermediate MRL of 1 $\mu g/m^3$ for Cr VI particulates. Ni samples evaluated at GZ and at sites surrounding GZ never exceeded the NIOSH REL of 0.015 mg/m^3 or the OSHA PEL of 1 mg/m^3 for Ni metal. The ATSDR Intermediate MRL for Ni of 0.2 $\mu g/m^3$ was exceeded only once, on November 10, 2001, by a measurement of 0.49 $\mu g/m^3$. Overall, monitored levels were rarely above background. It should be noted, however, that Cr and Ni were selected for evaluation in this assessment because both can be irritating and sensitizing, and it is possible that early exposures to these contaminants sorbed to particulates may have contributed to observed respiratory health effects.

3.4. Polychlorinated Biphenyls, PCBs

Of the several hundred PCB air measurements available, only one sample was elevated above 100 ng total PCB/m^3 (153 ng/m^3) and only three samples were above 50 ng total PCB/m^3. Typical urban background PCB concentrations are in the range of 1–8 ng total PCB/m^3. After a month, nearly all readings were in the range of typical urban PCB concentrations or were not detected. There were no exceedances of the NIOSH REL of 1,000 ng/m^3 or the OSHA PEL of 500,000 ng/m^3. There are no ATSDR acute or intermediate inhalation MRLs for PCBs.

A cancer risk screening exercise was conducted for PCBs. EPA currently classifies PCBs as B2 carcinogens, a probable human carcinogen (IRIS, 2006). From the dose-response data derived from animal studies, the EPA has calculated an upper bound cancer unit risk (UR) factor of $1 * 10^{-4}$ $[\mu g/m^3]^{-1}$ associated with continuous lifetime inhalation exposure to total PCBs (IRIS, 2006). For exposure to dioxin-like PCB congeners alone, the slope factor developed for dioxin-like compounds should be applied (Van den Berg *et al.*, 1998). This assessment does not consider exposure and risk from dioxin-like PCBs because these congeners were not measured separately.

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

$$\mathrm{LAC} = \mathrm{AC} * [\mathrm{ED}/\mathrm{LT}] \tag{2a}$$

$$\mathrm{Risk} = \mathrm{LAC} * \mathrm{UR}, \tag{2b}$$

where LAC is the air concentration averaged over a lifetime ($\mu g/m^3$), AC is the average air concentration during the period of exposure ($\mu g/m^3$), ED is the exposure duration (days) during this period of exposure, LT is lifetime (days), typically 70 years or 25,500 days, and UR is the unit risk factor, $1 * 10^{-4}$ $[\mu g/m^3]^{-1}$ (IRIS, 2006).

Proper application of Equation (2) requires a representative air concentration and a time during which exposure to that concentration occurred. The areas with elevated PCB air concentrations were generally located within the "restricted zone." Still, even if an individual were exposed to the highest concentration found at 153 ng PCB/m^3 for a period of 1 month (all the data suggest that elevations did not exist beyond 1 month), the lifetime cancer risk would be estimated at about $2 * 10^{-8}$ (calculated as: $[0.153\ \mu g/m^3] * [30\ \mathrm{d}/(25{,}500\ \mathrm{d})] * [1 * 10^{-4}\ (\mu g/m^3)^{-1}]$). EPA regulatory programs, such as the Superfund Program, typically consider individual incremental cancer risk estimates of less than 10^{-6}, when they are made in the context of a scenario-based risk assessment, to be below regulatory concern. When the risks range from 10^{-6} to 10^{-4}, they are assumed to be of potential significance and worthy of further analysis. On this basis, an incremental cancer risk estimate in the range of 10^{-8} is judged to be insignificant.

The ATSDR Toxicological Profile for PCBs (ATSDR, 2000) is a comprehensive review and summary of existing health effects information relevant to human exposures. This review established that the no-observed-adverse-effect levels (NOAELs) for chronic exposures in experimental animals ranged from $9 * 10^3$ ng/m^3 for hepatic effects to $1.5 * 10^6$ ng/m^3 for renal effects. These NOAELs are one to six orders of magnitude higher than the highest PCB air levels measured in Lower Manhattan.

3.5. Dioxins and Furans, CDD/Fs

A total of 29 congeners are considered to be "dioxin-like": 7 polychlorinated dibenzo-*p*-dioxins (abbreviated dioxins), 10 polychlorinated dibenzofurans (furans), and 12 coplanar PCBs (Van den Berg *et al.*, 1998). 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 are described in terms of picograms per cubic meter, pg/m^3. Concentrations of dioxin-like congeners are also expressed on a toxic equivalent, or TEQ basic. A congener's TEQ concentration is calculated by multiplying its concentration (C_i) by 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-Tetrachlorodibenzo-p-Dioxin (TCDD) (this 17th congener is naturally assigned a TEF of 1.0). An overall TEQ air concentration is, therefore, $\Sigma(TEF_i * C_i)$. When a congener was not detected in a WTC sample, a value of one-half the detection limit was used for that congener in calculating the TEQ concentration. The TEQ concentrations reported in the WTC monitoring program were developed using the "International" set of TEF values developed in 1989 (I-TEF) (U.S. EPA, 1989). In 1998, the World Health Organization (WHO) proposed a new set of TEF values (WHO-TEF) (Van den Berg *et al.*, 1998). Calculating TEQs using the WHO-TEFs tend to result in concentrations slightly higher than when using I-TEFs; a spot check on WTC data suggested that WHO-TEQ concentrations would be about 10% higher than I-TEQ concentrations. An even newer set of TEF values was published in 2006 (Van den Berg *et al.*, 2006), and a similar spot check suggests a similar increase of about 10% as compared to I-TEQ. To avoid confusion with the EPA website, I-TEQ values are used in this assessment, and all results described below are described simply as TEQ.

Table I shows the TEQ concentrations from three air monitors: a monitor at the GZ site (identified as WTC-Building 5 in Table I), a monitor at a location immediately off-site of GZ in the predominant easterly wind direction (the Church and Dey monitor), and a monitor located about 5 blocks away, also in the predominantly easterly wind direction (the Park Row monitor). TEQ levels in air near the WTC were up to three orders of magnitude higher (1,000 times higher) than is typical for urban areas in the United States. Typical levels for urban areas are 0.1–0.2 pg TEQ/m^3 (U.S. EPA, 2003a), while levels found in GZ and near GZ, starting September 16 (the date of the first sample taken) and continuing through late November, 2001, ranged from 10–170 pg TEQ/m^3. Before this, the highest TEQ concentration reported in the United States was about 1.0 pg/m^3, downwind of an incinerator in Niagara Falls, NY (Smith *et al.*, 1989). Air concentrations near an incinerator in Japan adjacent to a U.S. Naval Air Base were regularly measured on the base for CDD/Fs. Measurements at the nearest downwind monitor, at about 200 meters, averaged 3.5 pg TEQ/m^3 for weekly samples over a 15-month period (Walker *et al.*, 2002). Concentrations measured several blocks from GZ were still elevated above typical urban background but considerably lower than sites in or near GZ, ranging from 1–10 pg TEQ/m^3 from September through November. The levels dropped during November, and the data suggest that by December 2001, levels decreased to typical urban background levels.

Understanding the context of these high air concentrations is important. Over 95% of an average individual's total exposure to dioxin is attributed to dietary intake in normal background settings, and approximately 1% is attributed to inhalation exposures (U.S. EPA, 2003a). Further, the risk from exposure to dioxin is long term and cumulative. Therefore, while the air concentrations through November appear extremely elevated, they may not result in a long-term cumulative effect since elevations only occurred for a matter of weeks after 9/11.

In order to evaluate this possibility, a cancer risk exercise was conducted. The average daily inhalation dose to TEQs is given by:

$$\mathrm{ADD} = [\mathrm{IN} * \mathrm{C} * \mathrm{ABS}]/[\mathrm{BW}], \quad (3)$$

where ADD is average daily dose (pg TEQ/kg-day), IN is the inhalation rate (m^3/day), C is the average concentration during the period of exposure (pg TEQ/m^3), ABS is the fraction of contaminant inhaled which is absorbed (unitless), and BW is the body weight (70 kg for an adult). An average daily dose estimate rather than simply an average concentration is required because, unlike PCBs, an inhalation UR factor is not available for dioxin-like compounds. The draft Dioxin Reassessment (U.S. EPA, 2003a) assumes an absorption fraction of 0.8 for TEQ exposures, both from inhalation and food consumption, and this assumption is used here. The average adult inhalation rate is given as 13.3 m^3/day (US EPA, 1997). Cancer risk is then given by:

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

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

where LADD is the lifetime daily dose (pg TEQ/kg-day), ADD is the average daily dose during the period of exposure (pg TEQ/kg-day), ED is the exposure duration (days), LT is lifetime (25,500 days, equal to

Table I. Measured Dioxin TEQ Air Concentrations at the WTC Building 5 Monitor, the Church & Dey Monitor, and the Park Row Monitor (All Units = pg TEQ/m^3; NR = Not Reported; All TEQ Calculated at ND = $^1/_2$ DL Except Values in Parenthesis, Which Are Calculated at ND = 0)

Date	WTC - Bldg 5	Date	Church & Dey	Date	Park Row
9/16/01	NS	9/16/01	60.0	9/16–10/11	No data taken
9/23	161.0 (161.0)	9/23	139.0 (139.0)	10/12/01	8.35
9/27	NS	9/27	50.0	10/14	0.34
10/2	175.0 (170.0)	10/2	59.3 (57.2)	10/15	4.78
10/4	176.0 (140.0)	10/4	51.9 (50.6)	10/16	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	NS	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	NS	11/12	1.3 (0.6)	11/19	2.50
11/15	5.4 (1.6)	11/15	3.4 (1.6)	11/22	1.30
11/21	4.1 (3.1)	11/21	10.0 (8.3)	11/26	0.80
		11/27	5.6 (5.5)	11/29	0.16
No samples taken from 11/21/01 to 1/15/02		12/1/2001–5/17/2002		12/3/2001–3/14/2002:	
1/15–5/28/02; $n = 46$		$n = 46$		$n = 29$	
Reported range: 0.4–5.5		Reported range: 0.2–4.1		All samples reported $< = 0.16$	
Average: 1.4 at ND = $^1/_2$ DL and 0.0 at ND = 0.		Average: 1.1 at ND = $^1/_2$ DL and 0.0 at ND = 0.			

70 years), Risk is the upper bound incremental excess lifetime cancer risk that results from the exposure described by LADD, and SF is the upper bound cancer slope factor, expressed in inverse units to LADD, or [pg TEQ/kg-day]$^{-1}$. The SF of.000156 [pg/kg-day]$^{-1}$ was developed by EPA in 1984 for 2,3,7,8-TCDD exposures (U.S. EPA, 1984). It is applied to TEQ exposures in this cancer risk screening exercise.

The concentration to which the individual was exposed was derived from the data on Church and Dey shown in Table I. The on-site concentrations from the monitor at WTC-Bldg 5 were clearly the highest and might be representative of site worker exposure, so they are not appropriate for characterizing general population exposure. The Park Row data demonstrate the gradient of dioxin concentrations with distance, and concentrations there were lower than the Church and Dey border-monitoring site. With the intent of being conservative, therefore, general population exposures to dioxins were assumed to be characterized by the Church and Dey monitor. As seen in Table I, the concentrations in December 2001, through the end of the monitoring in May 2002 were nondetects for the Church and Dey site. As discussed in Lorber (2003), the low volume of air taken into the Church and Dey monitor was insufficient to characterize background air concentrations of dioxins, though it was sufficient to quantify the elevated levels seen through about the end of November. The Park Row monitor, in contrast, took in a sufficient volume of air to characterize background levels, and as seen in Table I, concentrations dropped well below 1 pg/m^3 toward the end of November into December and beyond. Exposure was assumed to begin on September 16, the first day for which a measurement from Church and Dey was available, and approximately the time when the restricted zone was opened for individuals to return to work and their homes, and to continue through the end of November, for a total of 77 days. The time-weighted average concentration (derived by linearly extrapolating between measurement dates) was 27.4 pg TEQ/m^3 in the Church and Dey monitor. Using other parameters described above, the lifetime average cancer risk is calculated to be $2 * 10^{-6}$.

U.S. EPA (2003a) derived an average cancer risk for the general population based on a lifetime of exposure to dioxin-like compounds, dominated by food-related exposures, as noted before. The cancer risk EPA derived is about 50 times higher than this WTC-related risk at about $1 * 10^{-4}$. As discussed in Section 3.4, EPA typically considers individual incremental cancer risk estimates made in the context of a scenario-based risk assessment at less than 10^{-6} to

be below regulatory concern, and risks in the range of 10^{-6}–10^{-4} to be of potential significance and worthy of further analysis. Exposure to dioxin-like compounds represents a unique circumstance because background exposures are already within the range of concern. Therefore, while the incremental $2 * 10^{-6}$ cancer risk due to 9/11 is within this 10^{-6}–10^{-4} range, it is judged to be of minimal concern, given much higher average exposures to dioxins.

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. Adults are assumed to be 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.

With only typical background exposures, dioxins build up and decline over prolonged periods of time because the overall biological half-life of dioxin-like compounds in the human body ranges from 7 to 14 years (U.S. EPA, 2003a). The use of the body burden as the measure of dose has implications for short-term unique exposures, such as those near the WTC site, where elevated exposures 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) (U.S. EPA, 2003 a).

In the draft Dioxin Reassessment, it is 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 (U.S. EPA, 2003a). 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:

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

where IBB is the increment of body burden on a lipid basis (pg/g, or ppt, lipid basis), ADD is the average daily dose over the period of exposure (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) over the period of exposure will provide an estimate of body burden at the end of the exposure, when the incremental body burden will be at its largest. 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 $*$ 1,000 g/kg), and a k of 0.000267 day^{-1} ($= 0.098\ \mathrm{yr}^{-1}$, corresponding to a 7.1 year half-life) (U.S. EPA, 2003a) will be used. Results for this exercise include both an incremental body burden estimate (the IBB of Equation (5)), calculated at the end of the exposure period, and the 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. Given the conservative scenario of being exposed 24 hours/day to high concentrations seen at the border of GZ for 2.5 months, an increment of 3.1 pg/g lipid, or about 17% over the background level of 18 pg/g lipid, is calculated.

There is evidence in the literature that suggests exposures to dioxin-like compounds near WTC could have resulted in a rise in body burdens. Edelman *et al.* (2003) published results of a biomonitoring study where blood and urine of 321 firefighters who were present and working at GZ were measured for numerous contaminants and then compared with those of 47 firefighters who did not work at GZ and served as controls. The firefighters themselves were split into groups corresponding to time of arrival at the site and whether or not they were "Special Operations Command" firefighters. Samples were taken from October 1 to 5, 2001, about 3/4 weeks after 9/11, and all of the "exposed" firefighters worked on most days between 9/11 and this sampling time. Most of the findings showed insignificant or no differences between the firefighters and the controls or within the different firefighter groups. However, six contaminants showed a statistically significant different geometric mean concentration in the firefighters as a whole when compared with the control firefighters, and one of those contaminants was the hepta dioxin congener, 1,2,3,4,6,7,8-heptachlorodibenzodioxin (HpCDD). The mean concentration of this congener for all firefighters was 27.8 versus 19.2 pg/g lipid for the controls. There were also differences, although not statistically significant, based on arrival times and whether or not the firefighter was in the Special Operations Command.

Specifically, those arriving on Day 1 at the time of collapse had a geometric mean level of 30.1 pg/g lipid compared with 26.4 for those arriving on Days 1–2, but not at the time of collapse. Special Operations Command firefighters had a geometric mean level of 30.6 pg/g lipid compared with 25.9 pg/g lipid for all other firefighters. No statistically significant differences were noted between different groups for the 16 other dioxin-like congeners measured. Still, measurements were taken between October 1 and 5, and elevations of dioxins in the air continued through the end of November, as modeled here. Although not a direct verification of the finding in the body burden exercise described above, this firefighter biomonitoring study supports the modeling showing a rise in body burden in the 10–20% range.

The *margin of exposure* (MOE) is defined as the ratio of body burden where effects are found to 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 within an order of magnitude of where noncancer effects are seen (U.S. EPA, 2003a). Also, U.S. EPA (2003a) discusses variability of background dioxin exposures and finds that exposures in the general population extend up to three times the mean exposure, based on intake estimates. With these two facts—that the general population variability is a factor of three above background body burdens, and that noncancer risks are seen with a factor of 10 of background body burdens—an IBB of 17% is judged to be of minimal concern.

3.6. Polycyclic Aromatic Hydrocarbons, PAHs

Only a few specific PAH samples were collected as part of the regular WTC monitoring program. However, a method was developed to exploit existing $PM_{2.5}$ samples to assay specific particle-bound PAHs that represent the species associated with carcinogenicity, mutagenicity, and reproductive effects (Pleil *et al*., 2004). These include the following nine PAHs: benz(*a*)anthracene, chrysene, benzo(*b*)fluoranthene, benzo(*k*)fluoranthene, benzo (*a*)pyrene, benzo(*e*)pyrene, indeno(1,2,3-*cd*)pyrene, dibenz(*a,h*)anthracene, and benzo(*g,h,i*)perylene. PM samples were collected daily at three perimeter sites located at the fenceline, 1 block away, and 3 blocks from GZ and one about 7 blocks away at the EPA building at 290 Broadway from September 23, 2001 to March 27, 2002. A subset of 243 samples were randomly selected and analyzed by gas chromatography/mass spectroscopy (GC/MS) for the presence of the nine PAHs (Pleil *et al.*, 2004). A few of the highest levels (expressed as a sum of nine target PAHs) exceeded 100 ng/m^3 in the first few days of sampling; the estimated mean value on 9/11, based on back-extrapolation of time series data, is 35 ng/m^3, and the estimated NYC background is 1.4 ng/m^3. Initial PAH levels decreased, with an estimated 15-day half-life, reaching a WTC mean background level of 5.3 ng/m^3 after 50 days.

Carcinogenic risk from ambient PAH exposure is generally estimated by the relative potency factor model (RPFM). Calculations are based on seven PAHs designated as probable human carcinogens by EPA (IRIS, 2006) and their relative potency with respect to benzo*(a)*pyrene, BaP, considered the most toxic of the seven. The RPFM assumes linear additive effects and does not account for potential chemical interactions. This methodology is essentially the same as the dioxin TEQ methodology described above; a total concentration toxically equivalent to BaP is calculated as the sum of each congener's concentration times its potency equivalency factor, or PEF. The State of California has used the RPFM to set regulations (CEPA, 1999) and it has been adopted in various forms by other states as well (e. g., Minnesota, Massachusetts, New Jersey). Similar to PCBs, cancer risk from PAH exposure is calculated as a unit risk factor (UR) times a concentration deemed to be representative of lifetime average air concentration due to a WTC incremental exposure. The UR for BaP inhalation has been estimated from different hamster studies at $0.37 * 10^{-6}/(\text{ng/m}^3)$, $1.1 * 10^{-6}/(\text{ng/m}^3)$, and $1.7 * 10^{-6}/(\text{ng/m}^3)$. Initially, EPA recommended $1.7 * 10^{-6}/(\text{ng/m}^3)$, but owing to some concerns over study quality, no value is listed in EPA's integrated risk information system (IRIS, 2006). For this exercise, $1.1 * 10^{-6}/(\text{ng/m}^3)$ is used, following the practice of the State of California (CEPA, 1999). Further, California published PEFs for 23 additional PAHs, including seven that are used to develop the concentrations used for this exercise (CEPA, 1999).

To apply the RPFM, the "equivalent BaP" lifetime average air concentration (CA) needs to be calculated. The data on PAHs sorbed to PM during the period of elevation due to WTC can be compartmentalized into three groups: early, middle, and late after 9/11. The early group (from Day 12 to Day 50), "group1," is representative of major WTC fires, activity from diesel engine activity of GZ power generation, demolition equipment (cranes, bulldozers), and debris removal (trucks). The middle group (from Day 51 to Day 100), "group2," is representative of

sporadic fires, some scaled-back rescue activity, and truck traffic. Finally, the late group (from Day 101 to Day 200), "group3," is the "plateau" period, when the fires were officially out, most WTC demolition was done, WTC area power had been restored, and only truck traffic and background remained as PAH sources. With these definitions, the lifetime average BaP-toxically equivalent concentration, CA_{WTC}, due to WTC is calculated as:

$$\begin{aligned} CA_{WTC} = &[CA_{group1} * 50/25,550] \\ &+ [CA_{group2} * 50/25,550] \\ &+ [CA_{group3} * 100/25,550], \end{aligned} \quad (6)$$

where 25,550 is the number of days in a 70-year lifespan. Based on data from the sampler directly at GZ, the three concentrations are: group1—2.32 ng/m^3; group2—1.06 ng/m^3; and group3—0.47 ng/m^3, and the CA_{WTC} is 0.0084 ng/m^3. Using the UR of $1.1 * 10^{-6}$/(ng/m^3) described above, the estimated cancer risk is $9 * 10^{-9}$. Similar to the PCB assessment above having an incremental risk in this range, this is judged to be of minimal concern.

Available literature does not give a good definition for chronic noncancer risk endpoints for PAHs, and the IRIS database contains reference doses (RfDs) for only five PAHs (acenaphthene, anthracene, fluoranthene, fluorene, and pyrene). However, ambient PAHs are strongly associated with diesel particulate matter (DPM), which comprises about 13.6% (on average) of $PM_{2.5}$ in Manhattan (Kinney *et al.*, 2000; NTP, 2002; IRIS, 2006). EPA has estimated that 20–40% of DPM is composed of organic compounds, of which 1% are PAHs and nitrated PAHs (U.S. EPA, 2002b). DPM is implicated in human pulmonary inflammation and histopathology; animal studies indicate immunological effects and reproductive toxicity (IRIS, 2006). The inhalation RfC for DPM is 5 μg/m^3; it is defined as the lifetime exposure level below which the human population, including sensitive subgroups, will not have an appreciable risk of deleterious effects. The average $PM_{2.5}$ concentration measured for GZ samples for 200 days after 9/11 was 37.7 μg/m^3; the average $PM_{2.5}$ concentration for Manhattan for 2001 and 2002 was 16 μg/m^3 (NYSDEC, 2003). Thus, DPM represents about 5.1 μg/m^3 and 2.17 μg/m^3 on average for the 200-day WTC aftermath and the New York baseline, respectively. Even in the unlikely scenario in which all of the chronic noncancer effects of DPM could be ascribed to the PAH fraction, the RfC for a lifetime exposure is still not exceeded by the contribution from the WTC disaster.

Finally, developmental studies have demonstrated impacts to newborns, and these impacts could be the result of exposures to PAHs. Discussion of these developmental studies and their possible link to PAH exposures is discussed below in Section 3.10 on observed health effects.

3.7. Asbestos

The large majority of air measurements of asbestos was below established benchmarks and within the range of typical urban background levels. However, as with other contaminants, the few measurements above benchmarks occurred near 9/11 in time and in close proximity to the WTC. Only 22 of more than 9,400 transmission electron microscopy (TEM; used to identify structures greater than 0.5 μm in length) exceeded the Asbestos Hazard Emergency Response Act (AHERA) Standard of 70 structures per square millimeter (S/mm^2). Of the 22 exceedances, 12 occurred in September and were at sites bordering GZ. These sites were still in the restricted zone during September. The same general trend can be seen with the phase contrast light microscopy (PCM) (used to identify structures greater than 5 μm in length) data. Of more than 19,000 measurements, only four were above the OSHA PEL of 0.1 fibers per cubic centimeter (f/cc), and these were taken near GZ during September and October from temporary monitoring sites. The seven highest measurements from the fixed monitors ranged from 0.04 to 0.08 f/cc and were also taken near in time to 9/11 and near GZ.

In addition to reporting TEM results in terms of S/mm^2, the EPA TEM data were expressed in terms of total fiber concentrations as well as concentrations of structures greater than 5 μm. This allowed for a cancer risk assessment using the IRIS inhalation UR factor of 0.23 (f/cc)$^{-1}$ (IRIS, 2006). Multiplying this factor by an estimate of a concentration of fibers greater than 5 μm to which an individual is exposed over a lifetime will estimate the risk of incurring cancer as a result of this lifetime inhalation exposure. An estimate of a lifetime urban air concentration was made on the basis of measurements taken in monitoring sites near GZ. This estimation included a month of higher concentrations surrounded by years of background concentrations. Providing an average concentration for TEM measurements specific to structures greater than 5 μm is a nontrivial exercise. This is because the limit of detection for fibers of this length was fairly high in the EPA sampling program at about 0.004 f/cc. Therefore,

assuming 1/2 detection limit for nondetects in calculation of an average would imply that when at least one large fiber was not counted, then the overall concentration in the air would be higher than most urban background settings at 0.002 f/cc.

According to ATSDR (2001), average urban background concentrations of fibers of this length are about 0.00007 PCM f/cc. For this reason, nondetects for fibers at this length were assumed to be 0.0, and weighted average concentrations were derived for each month. For example, the concentration during the highest month, the month of September 2001, was calculated to be 0.0036 f/cc. This was calculated based on the data, which showed an average positive concentration of 0.0095 f/cc and positives detected 38% of the time ($0.38 * 0.0095 = 0.0036$). The average of monthly averages from October 2001 to June 2002 equaled 0.00017 f/cc, and the percent of detections of fibers of this length were much lower, always under 10%. It was assumed that 0.00017 f/cc represents the average monthly concentration for the New York urban background, so a cancer risk estimate is developed only for exposures that occurred in September as represented by the concentration of 0.0036 f/cc. Assuming that concentration occurred for a month, the LAC of asbestos is estimated as $[0.0036 * 1 \text{ month}/(70 \text{ years} * 12 \text{ months} - 1)] = 4.3 * 10^{-6}$ f/cc. The cancer risk associated with this lifetime average concentration is $1 * 10^{-6}$. The cancer risk associated with the average background concentration of 0.00017 f/cc is $3.9 * 10^{-5}$. Therefore, similar to dioxins, while the incremental cancer risk due to 9/11 is within a range of possible regulatory concern at $1 * 10^{-6}$, background risk is about 40 times higher, so this incremental risk is judged to be of minimal concern.

Overall, it is reasonable to conclude from these data 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 percentage of measurements of asbestos above established benchmarks were reported.

3.8. Volatile Organic Compounds, VOCs

A total of 11 VOCs were evaluated at sites surrounding GZ. No exceedances of screening benchmarks outside of GZ were seen for 1,4-dioxane, ethanol, styrene, tetrahydrofuran, and xylenes. Exceedances of screening benchmarks were seen for acetone, benzene, 1,3-butadiene, chloromethane, ethylbenzene, and toluene in sites outside of GZ. Except for benzene, exceedances for these chemicals occurred in restricted zones. Also, the exceedances were all grab samples. As noted in the introduction, grab samples comprise the bulk of all measurements for VOCs. They are taken by mobile monitors in short time periods, usually about 4 minutes, for the purpose of obtaining a quick analysis to determine whether areas are safe for entry and working. A limited number of 24-hour samples were taken on the perimeter of GZ. A comparison of these 24-hour samples with grab samples demonstrates how cumulative exposure point concentrations can be much lower than transient high concentrations that might be captured with a 4-minute grab sample. The 24-hour samples of 1,3-butadiene, ethylbenzene, and toluene all were about three orders of magnitude (1,000 times) lower than the grab samples. On the basis of available monitoring data, it is concluded that the exceedances of the screening benchmarks outside of GZ in restricted zones for acetone, 1,3-butadiene, chloromethane, ethylbenzene, and toluene do not represent a health risk to the general population.

The data for benzene were not as definitive. The exceedances for benzene were more frequent, some were farther from GZ than the other VOCs, and the 24-hour samples were lower, but within a factor of 10 of the grab sample exceedances. The fact that the 24-hour samples were measured at levels that were closer in magnitude to the grab sample exceedances than the other VOCs, within a factor of 10, suggests 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 historic average for New York City of about 0.0005 ppm (ATSDR, 1997). Specifically, six of 14 24-hour samples were above the detection limit of 0.0007 ppm, with three at 0.0007 or 0.0008, and three at 0.002, 0.0025, and 0.005 ppm. The data suggest that the exposures to benzene at levels that approach the Intermediate MRL were not likely to have lasted longer than 45 days. Whether or not specific health effects occurred owing to exposure to benzene is unknown, but the exceedances and elevations above typical background were near GZ and mostly within restricted zones. Thus, the data suggest that exposures for the general population were likely to be of limited concern.

3.9. Silica and Man-Made Vitreous Fibers, MMVF

Silica samples evaluated at GZ and at sites surrounding GZ never exceeded the NIOSH REL of 0.05 mg/m^3, which is a 10-hour time-weighted average

concentration. Silica was sampled between September 27, 2001, and June 20, 2002, at 14 sites, including the GZ site, WTC Building 5. Approximately 1% of nearly 1,800 samples taken for silica were positive, with the highest positive at 0.03 mg/m^3 and no positive samples found among 159 samples taken at the GZ site. On the basis of samples evaluated, exposures to silica were not likely to have caused any adverse health effects. However, it is important to note that the first measurement of silica was not made until September 27. It is likely that the pulverized concrete building materials resulted in exposures to respirable crystalline silica on September 11 and the first few days thereafter.

Only 32 air samples were analyzed for SVFs (glass fibers, fiberglass, mineral wool) in ambient air. Six samples were taken on October 4; for 3 days at the end of February and beginning of March 2002, 26 samples were taken within the breathing zone in the cabs of heavy equipment used by workers on the pile. The samples in October were nondetects at detection limits of about 0.02 f/cc, and the samples in February and March 2002 had 11 nondetects, but there were 15 detects, ranging from 0.01–0.04 f/cc. With essentially no data for the period of concern, inhalation exposures for the general population could not be evaluated.

However, SVFs were measured in both outdoor dust and indoor dust, and exposures to SVFs on 9/11 have been identified as one of the possible causes, along with high pH dust and agglomerated particles, of the persistent cough and other respiratory symptoms that occurred among GZ workers and nearby residents shortly after 9/11. Lioy *et al.* (2002) took three dust samples in weather-protected areas on September 16 and 17 and found that glass fiber constituted 40% of settled dust in each of the three samples. They found the width of the glass fibers to be about 1 μm (up to 10 μm), with the length ranging from 5–100 μm. Lioy concluded that the high pH of the dust, along with glass fibers and agglomerated fine particles, were likely to be the cause of initial lung irritation reported for workers and residents in the initial days and weeks following the disaster. The only other study that measured SVFs was conducted by the ATSDR in November 2001. This study focused on indoor dust in residential apartments in Lower Manhattan, but also took outdoor dust samples. Indoor settled dust contained SVFs in 40 of 83 sites, ranging from 2–35% of the dust content. SVFs were detected in 11 of 14 (79%) outdoor locations at levels ranging from 1% to 72% of the sample. No SVFs were detected in dust in comparison areas above 59th Street (ATSDR, 2002). Landrigan *et al.* (2004) cite the Lioy conclusion that exposure to SVFs, along with high pH dust and agglomerated particles, was responsible for at least the early pulmonary symptoms observed in GZ workers as well as some local residents.

3.10. Observed Health Effects

While the analysis described above conducted by EPA evaluated the potential for health impacts based on measured air concentrations, epidemiological studies conducted by organizations other than EPA have attempted to identify actual impacts. These studies have focused on developmental and respiratory effects. This section provides a summary of the key findings of these studies as described by the authors of the studies; further detail on the study designs, limitations, and uncertainties can be found in the provided citations. In one of the reproductive/developmental studies, the principal identified health effect was a finding of intra-uterine growth restriction (IUGR) resulting in a two-fold increase in small-for-gestational-age (SGA) (<10th percentile for gestational age) infants in a study of 187 women who were pregnant and either inside or near the WTC on September 11, 2001 (Berkowitz *et al.*, 2003). The study's authors hypothesized that this effect could be due to exposures to PAHs and particulates. A later evaluation on this cohort of 187 women looked at PAH-DNA adducts in maternal blood mononuclear cells starting in February 2002, with measurements through October 2002 (Wolff *et al.*, 2005). Although 88 of the 160 total measurements (55%) were nondetects, a higher percentage of women reportedly had measurable adducts during February and March (46 of 72, or 64%), with a median value of 46.7 adducts/million nucleotides (apmn), compared with the April through October findings (26 of 88 or 30%), with a median of 20 apmn.

The second study (Lederman *et al.*, 2004) reported small, but significant reductions in birth length (−0.74 cms) and birth weight (−122 gms) among infants of a cohort of 300 women who lived or worked within a 2-mile radius of the WTC during the fall of 2001, as compared with those of a control, who neither lived or worked within this 2-mile radius during this time period (results adjusted for gestational period and socioeconomic and biomedical risk factors). Although previous work suggests a general IUGR link to PAH exposure, in the case of the WTC event, the researchers who conducted this study hypothesized that such a link may be confounded with effects from

exposure to other constituents of airborne PM. Similarly, the authors suggest a potential contribution to the reproductive effects from maternal psychological stress caused by the WTC disaster. To the extent that pollution was responsible, it is reasonable to assume that both outdoor and indoor inhalation exposures contributed to observed effects. The authors acknowledge that the effects, although statistically significant, are modest.

Two general population studies have focused on respiratory health effects. One study, conducted at New York University (Reibman *et al*., 2005), reported an increase in many respiratory-related symptoms in Lower Manhattan residents (e.g., cough) in an exposed area, as compared with a control group. A total of 2,812 residents completed a symptom-based questionnaire and did on-site spirometry tests 12 ± 4 months after the collapse of the WTC towers. Reibman *et al*. (2005) reported that new-onset respiratory symptoms were described by 55% of the exposed group compared with 20% in the control group, and persistent new-onset symptoms were reported by 26% in the exposed group versus 8% in the control group. Also, there was an increased response to a methacholine challenge (an indicator of reactive airway disease) in a small subset of subjects who had persistent new-onset symptoms and who agreed to participate in a small pilot study apart from the main questionnaire/spirometry study. However, spirometry (forced vital capacity) was not significantly different between the two groups. Reibman *et al*. (2005) observed that the symptoms are consistent with those identified in the rescue worker and responder population but that they are unable to document the exposure level of the residents to the dust and fumes. They suggest that the residents may have had high-level exposures to the initial dust cloud or to settled dust and persistent fires.

The other study (Szema *et al*., 2004) investigated asthma exacerbation in previously diagnosed asthmatics pre- and post-September 11 from a retrospective chart review of children in Chinatown. Chinatown is located about 0.5–1 mile northeast of GZ. Overall, the researchers looked at patients living within and beyond a 5-mile radius of GZ. The chart review assessed asthma severity for a 1-year period directly before and a 1-year period directly after 9/11 and compared the results with those of a control population. They found increases in clinic visits for both groups of patients—those living within and beyond 5 miles of GZ; the increase they found for those living within 5 miles was deemed to be significant, $p = 0.013$, whereas the increase for those living beyond 5 miles was not significant. They found that mean percent predicted peak expiratory flow rates decreased solely for those patients living within 5 miles of GZ during the 3 months after 9/11.

It is noteworthy that Szema *et al*. (2004) looked at asthma severity in patients living in two groups defined by their general proximity to GZ: those within and those beyond a 5-mile radius of GZ. The Lederman *et al*. (2004) study of birth weights looked at women living within or beyond a 2-mile radius of GZ. However, neither study investigated the possible presence of a dose-response trend, as measured by proximity to the WTC, within the rather large exposure zones. Consequently, generalizing the findings should be done with caution.

The principal health effects experienced by GZ workers were pulmonary effects: a persistent cough, bronchial hyperactivity, and an increased risk of asthma. The pulmonary effects observed in the GZ workers have been attributed to inhalation of highly alkaline WTC dust, and also inhalation of SVFs, such as glass fibers. Fine particles or metals, such as Cr and Ni in the initial dust cloud, could have irritated or sensitized individuals to further response. Prezant *et al*. (2002) reported on NYC firefighters exposed during the first 6 months after September 11, 2001. WTC cough occurred in 128 of 1,636 (8%) firefighters with a high level of exposure, 187 of 6,958 (3%) with a moderate level of exposure, and 17 of 1,320 (1%) with a low level of exposure, for a total of 332 firefighters with WTC cough. Chest radiographs were unchanged from precollapse findings in 319 of the 332 with WTC cough. In addition, 95% had symptoms of dyspnea, 87% had gastroesophageal reflux disease, and 54% had nasal congestion. A small subset of these firefighters without WTC cough, a total of 295 moderately and highly exposed firefighters, were tested for bronchial hyperreactivity with a methacholine challenge during the period between October 1 and October 14. Among this cohort of 295 without WTC cough, bronchial hyperreactivity was present in 77 (26%) firefighters with a high level of exposure and 26 (9%) with a moderate level of exposure.

Izbicki *et al.* (2007) report on the abnormally high frequency of sarcoidosis or "sarcoid-like" granulomatous pulmonary disease (SLGPD) experienced in firefighters who participated in the rescue/recovery effort at GZ. Briefly, they identified 15,048 FDNY (Fire Department of New York) workers who were present at any time during the WTC disaster rescue, recovery, and cleanup operation between September 11, 2001,

and July 1, 2002. Of these, 14,092 (94% of total) were evaluated as part of the FDNY WTC medical monitoring and treatment programs. Between 2001 and 2006, they identified 26 workers from this population who had pathologic evidence of sterile granulomatous pulmonary disease consistent with the diagnosis of sarcoidosis or SLGPD. Thirteen patients were identified in the first year (9/11/01–9/10/02), 1 in the second year, 4 in the third year, 4 in the fourth year, and 4 in the fifth year. All 26 patients had normal pulmonary evaluations prior to September 11, 2001. The annual incidence of sacoidosis or SLGPD increased significantly in the 5 years after the WTC exposure: it corresponded to a rate of 86 per 100,000 in Year 1 and 22 of 100,000 in Years 3–5, compared to 15 of 100,000 in the previous 15 years prior to September 11, 2001.

Another occupational study (Skloot *et al*., 2004) reported that respiratory symptoms occurred in a majority of ironworkers tested at the WTC disaster site and were not attributable to smoking. Exposure on September 11 was associated with a greater prevalence of cough. Objective evidence of lung disease was less common. Spirometry underestimated the prevalence of lung function abnormalities in comparison with forced oscillation (Skloot *et al*., 2004). The authors reported that continuing evaluation of symptoms, chest radiographs, and airway dysfunction should determine whether long-term clinical sequelae will exist.

Herbert *et al*. (2006) summarized the results from a 5-year assessment of a medical screening program from a portion of the GZ worker population. Approximately 11,000 first responders (rescue workers, paramedics, volunteers, and others) not eligible to participate in other programs (such as the program for the Fire Department of New York workers) were administered a medical screening, including questionnaires, spirometry, blood chemistry, urinalysis, and other tests. Of the 9,442 responders providing consent to use their results for publication, 69% reported new or worsened respiratory symptoms while performing WTC work, and 59% reported that the symptoms persisted up until the time of examination. Of the total tested, 28% had abnormal spirometry. Respiratory symptoms and spirometry abnormalities were significantly associated with early arrival at the site, including any time on 9/11 or within the first week after 9/11. This is consistent with the general finding of this assessment that the highest concentrations, and thus highest possible exposures, were those that occurred close in time to 9/11 and near GZ.

Wheeler *et al*. (2007) report on the prevelance of asthma diagnosed after 9/11 among WTC workers and volunteers, as reported in the WTC Health Registry. This registry is a collaborative effort of the New York City Department of Health and Mental Hygiene and the federal ATSDR, and is comprised of over 32,000 individuals who participated in any recovery, rescue, cleanup, or volunteer tasks from 9/11 through June 2002. Compared to a 0.3% 3-year risk of asthma in the general population, they found a 3.6% increase from this registry. The highest increase, 7%, was from workers who arrived on September 11 and worked for 90 days. In addition to early arrival and work duration, they also found a significant association with being caught in the initial dust cloud on 9/11.

Finally, health effects that have not yet surfaced could still occur. Landrigan *et al*. (2004) recommended follow-up studies to determine whether there were any long-term health impacts associated with exposure to asbestos (mesothelioma) and dioxin (cancer, diabetes, and other chronic disease).

What cannot be ascertained with all the data collected to date is exactly when and where significant exposures occurred, although Landrigan *et al*. (2004) observed that the most extreme exposures occurred very close in time to 9/11 and near GZ in proximity. As stated in their article,

> In the first few hours, extremely heavy exposures to high levels of dust and smoke as well as to gaseous products of combustion predominated. This pattern continued for the next 2 days, when there occurred rapid decline of smoke and dust levels and continuing decline in levels of combustion products as jet fuel and flammable building contents were consumed. A large fraction of the outdoor dust was eliminated over the first weekend after the disaster by rain that fell on Friday, 14 September, and by the U.S. EPA's clean up of the Wall Street area.

Of course, this critical time when the highest exposures were likely to have occurred is a time when there is very limited information.

4. DISCUSSION AND CONCLUSIONS

The analysis of air quality near GZ and the implications for potential health impacts should be considered limited for several reasons. For one, there is uncertainty as to the quality of air during the first few hours and days after 9/11. Difficulties associated with site access and security, power supply sources, equipment availability, and analytical capacity hindered efforts to begin regular monitoring for several days. The

limited data available during these very early times after 9/11 suggest, as might be expected, that concentrations in the air were highest during this time. Other limitations of this analysis include a lack of consideration of exposures to dust through pathways other than inhalation, indoor exposures, and the potential for cumulative exposures. Ingestion exposures to dust would be of concern mostly for GZ workers, but another population of concern for this pathway would be infants and small children in indoor environments into which WTC dust may have deposited and may not have been thoroughly removed through cleaning. The ATSDR sampled air and dust within residences in November 2001 (ATSDR, 2002). In Lower Manhattan, asbestos was found in indoor dust in 15 of 83 (18%) samples from residential units and common areas at levels ranging from less than 1% to 1.5%. SVFs were detected in 40 of 83 (43%) indoor dust samples, at levels ranging from 2% to 35% of the dust content. No asbestos or SVF was detected in dust in the comparison areas above 59th Street. Air and dust within two apartments located near the WTC were sampled on September 18, 2001, in a different study (Chatfield and Kominsky, 2001). Very low concentrations of dioxin, PCBs, and metals were found. However, asbestos readings were elevated in both air and dust in both apartments. EPA Region 2 conducted a study on a highly impacted residential building near GZ in 2002 (U.S. EPA, 2003b). The primary purpose of this study was to evaluate different cleaning methods, as the Region then embarked on a program to clean apartments near GZ. Elevated levels of several contaminants were found in this confirmation cleaning study.

Besides dust exposures and indoor exposures, there remains the uncertainty of inhalation (or other pathway) exposures to multiple contaminants. The cumulative risk from so many different exposures at the elevated concentrations that occurred close to 9/11 and GZ may well have produced effects that cannot be fully discerned by examination of exposure to individual substances. Because the air monitoring data and procedures for cumulative impacts were not sufficiently robust, this assessment does not address cumulative impacts.

This discussion has been offered simply to provide perspective on the three principal conclusions of this assessment by EPA, which are:

1. Persons exposed to extremely high levels of ambient PM and its components, SVF, and other contaminants during the collapse of the WTC Towers and for several hours afterward were likely to be at risk for acute and potentially chronic respiratory effects.
2. Following the extremely high levels of contaminants associated with the collapse of the WTC Towers on 9/11, available data suggest that the concentrations within and near GZ remained significantly elevated above background levels for a few days. EPA began taking limited site-related measurements the afternoon of September 11. By September 14, fixed air monitoring sites had been established for asbestos and other contaminants and by September 16 the first samples of dioxins, PAHs, and PCBs were taken from these fixed sites.
3. Except for inhalation exposures that may have occurred on 9/11 and a few days afterward, the ambient air concentration data suggest that persons in the general population were unlikely to suffer short-term or long-term adverse health effects caused by inhalation exposures. While these air concentrations were substantially elevated above typical background for the early days, they only occasionally exceeded health benchmarks after the first few weeks, and they had returned to typical background levels by November and December 2001.

In the years since 9/11, epidemiologic studies conducted by organizations other than EPA have identified respiratory effects in worker and general populations, and developmental effects in newborns whose mothers were near GZ on 9/11 or shortly thereafter. Respiratory impacts, such as exacerbated asthma and "WTC cough," have been observed in residents and other individuals living and working on the perimeter of GZ, and these impacts have persisted in some individuals to the current time. Researchers studying the respiratory impacts have hypothesized that they resulted from inhalation exposures that occurred near GZ and very close in time to 9/11, when concentrations of critical respiratory contaminants (PM, SVFs, asbestos, and others) were thought to be substantially elevated over typical background levels in air. In addition to respiratory effects, adverse developmental effects were observed in two studies. In both studies, the cohorts were pregnant women selected based on attributes such as being near GZ on 9/11 and/or living or working in the area during the several weeks afterward. Both outdoor and indoor exposures may have contributed to the observed effects. In one study, the reproductive effect of intra-uterine growth

restriction resulted in small-for-gestational-age babies. In the second study, small but significant reductions in gestation time and birth weight were observed. Although attribution is not certain, the researchers concluded that the observed reproductive effects suggest an impact of pollutants (PAHs and particulates) and/or stress related to the WTC disaster.

ACKNOWLEDGMENTS

The authors are indebted to the following individuals in EPA's Region 2 office for their contributions and very careful review of the EPA report (2002a) upon which this article is based: Marcus Kantz, Mark Maddaloni, Dennis Santella, Pat Evangelista, and Beverly Kolenberg, and the authors thank also these individuals from the National Center for Environmental Assessment: Jeff Frithsen, David Bussard, Ila Cote, Peter Preuss, and Linda Tuxen.

DISCLAIMER

The views expressed in this article are those of the authors and do not necessarily reflect the views or policies of the U.S. EPA.

REFERENCES

ATSDR. (1997). *Toxicological Profile for Benzene*. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, GA.

ATSDR. (2000). *Toxicological Profile for Polychlorinated Biphenyls (PCBs)*. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, GA.

ATSDR. (2001). *Toxicological Profile for Asbestos*. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services. Atlanta, GA.

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. (http://www.atsdr.cdc.gov/asbestos/asbestos˙WTC.html).

Berkowitz, G. S., Wolff, M. S., Janevic, T. M., Holzman, I. R., Yehuda, R., Landrigan, P. J. (2003). The World Trade Center disaster and intrauterine growth restriction. *Journal of the American Medical Association*, *290*(5), 595–596.

CEPA. (1999). *Technical Support Document for Describing Available Cancer Potency Factors*. California Environmental Protection Agency, Sacremento, CA. Available at http://www. oehha.ca.gov/pdf /HSCA2.pdf; accessed March, 2004.

Chatfield, E. J., Kominsky, J. R. (2001). *Summary Report: Characterization of Particulate Found in Apartments After Destruction of the World Trade Center*. Requested by "GZ" 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 at http://www.eqm.com.

Chen, L. C., Thurston, G. (2002). World Trade Center cough. *The Lancet 360*, Suppl, s37–s38.

Edelman, P., Osterloh, J., Pirkle, J., Caudill, S. P., Grainger, J., Jones, R., Blount, B., Calafat, A., Turner, W., Feldman, D., Baron, S., Bernard, B., Lushniak, D. B., Kelly, K., Prezant, D. (2003). Biomonitoring of chemical exposure among New York City firefighters responding to the World Trade Center fire and collapse. *Environmental Health Perspectives*, *111*(16), 1906–1911.

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

Herbert, R., Moline, J., Skloot, G., Metzker, K., Baron, S., Luft, B., Markowitz, S., Udasin, I., Harrington, D., Stein, D., Todd, A., Enright, P., Stellman, J. M., Landrigan, P. J., Levin, S. (2006). The World Trade Center disaster and the health of workers: Five-year assessment of unique medical screening program. *Environmental Health Perspectives*, *114*(12), 1853–1858.

IRIS. (2006). *Integrated Risk Information System*. Office of Research and Development, National Center for Environmental Assessment, U.S. Environmental Protection Agency, Washington, DC. Accessible http://www.epa.gov/iris/index.html.

Izbicki, G., Chavko, R., Banauch, G. I., Weiden, M. D., Berger, K. I., Aldrich, T. K., Hall, C., Kelly, K. J., Prezant, D. J. (2007). World Trade Center "sarcoid-like" granulomatous pulmonary disease in New York City Fire Department Rescue Workers. *Chest*, *131*, 1414–1423.

Kinney, P. L., Aggarwal, M., Northbridge, M. E., Janssen, N. A. H., Shepard, P. (2000). Airborne concentrations of PM2.5 and diesel exhaust particles on Harlem sidewalks: A community-based pilot study. *Environmental Health Perspectives*, *108*(3), 213–218.

Landrigan, P. J., Lioy, P. J., Thurston, G., Berkowitz, G., Chen, L. C., Chillrud, S. N., Gavett, S. H., Georgopoulos, P. G., Geyh, A. S., Levin, S., Perera, F., Rappaport, S. M., Small, C., the NIEHS World Trade Center Working Group. (2004). Health and environmental consequences of the World Trade Center disaster. *Environmental Health Perspectives*, *112*(6), 731–739.

Lederman, S. A., Rauh, V., Weiss, L., Stein, J. L., Hoepner, L. A., Becker, M., Perera, F. P. (2004). The effects of the World Trade Center event on birth outcomes among term deliveries at three lower Manhattan hospitals. *Environmental Health Perspectives*, *112*(17), 1772–1778.

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. *Environmental Health Perspectives*, *110*(7), 703–714.

Lorber, M. (2003). Assessment of dioxin inhalation exposures and potential health impacts following the collapse of the World Trade Center Towers. *Organohalogen Compounds*, *63*, 324–327.

Lorber, M., Gibb, H., Grant, L., Lioy, P. (2004). Assessment of inhalation exposures and potential health risks that resulted from the collapse of the World Trade Center Towers. *Environmental Manager*, February 2004, 27–29.

McGee J. K., Chen, L. C., Cohen, M. D., Chee, G. R., Prophete, C. M., Haykal-Coates, N., Wasson, S. J., Conner, T. L., Costa, D. L., Gavett, S. H. (2003). Chemical analysis of World Trade Center fine particulate matter for use in toxicological assessment. *Environmental Health Perspectives*, *111*(7), 972–980.

NTP. (2002). *10th Report on Carcinogens*. National Toxicology Program, Public Health Service, U.S. Department of Health and Human Services, Research Triangle Park, NC. Available at http://ntp-server.niehs.nih.gov.

NYSCDE. (2003) *New York State Department of Environmental Conservation Data Summary*. Available at http://www.dec.ny.gov/chemical/8888.html.

Pinto, J. P., Grant, L. D., Vette, A. F., Huber, A. H. (2005). Evaluation of potential human exposures to airborne particulate matter (PM) following the collapse of the World Trade Center Tragedy. In *Urban Aerosols and Their Impact: Lessons Learned from the World Trade Center Tragedy* (Eds. J. Gaffney and N.A. Marley) American Chemical Society Symposium Series 919, pp. 190–238.

Pleil, J. D., Vette, A. F., Johnson, B. A., Rappaport, S. M. (2004). Air levels of polycyclic aromatic hydrocarbons following the World Trade Center disaster. *Proceedings of the National Academy of Sciences*, *101*(32), 11685–11688.

Prezant, D. J., Weiden, M., Banauch, G. I., McGuinness, G., Rom, W. N., Aldrich, T. K., Kelly, K. J. (2002). Cough and bronchial responsiveness in firefighters at the World Trade Center site. *New England Journal of Medicine*, *347*(11), 806–815.

Reibman, J., Lin, S., Hwang, S. A., Gulati, M., Bowers, J. A., Rogers, L., Berger, K. I., Hoerning, A., Gomez, M., Fitzgerald, E. F. (2005). The World Trade Center residents' respiratory health study: New-onset respiratory symptoms and pulmonary function. *Environmental Health Perspectives*, *113*(4), 406–411.

Skloot, G., Goldman, M., Fischler, D., Goldman, C., Schechter, C., Levin, S., Teirstein, A. (2004). Respiratory symptoms and physiologic assessment of ironworkers at the World Trade Center site. *Chest*, *125*, 1248–1255.

Smith, R. M., O'Keefe, P. W., Hilker, D. R., Aldous, K. M., Mo, S. H., Stelle, R. M. (1989). Ambient air and incinerator testing for chlorinated dibenzofurans and dioxins by low resolution mass spectrometry. *Chemosphere*, *18*, 585–592.

Stevens, R. K., Dzubay, T. G., Lewis, C. W. Shaw, R. W. (1984). Source apportionment methods applied to the determination of the origin of ambient aerosols that affect visibility in forested areas. *Atmospheric Environment*, *18*(2), 261–272.

Szema, A. M., Khedkar, M., Maloney, P. F., Takach, P. A., Nickels, M. S., Patel, H., Modugno, F., Tso, A. Y., Lin, D. H. (2004). Clinical deterioration in pediatric asthmatic patients after September 11, 2001. *Journal of Allergy and Clinical Immunology*, *113*(3), 420–426.

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. (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. (1997). *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. (2002a). *Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster*. External Review Draft. National Center for Environmental Assessment. Office of Research and Development. EPA/600/P-2/002A. October 2002. (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=54667).

US EPA. (2002b). *Health Assessment Document for Diesel Engine Exhaust*. Prepared by the National Center for Environmental Assessment, Washington, DC, for the Office of Transportation and Air Quality. EPA/600/8-90/057F. Available from National Technical Information Service, Springfield, VA, PB2002-107661, and at http://www.epa.gov/ncea.

US EPA. (2003a). *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. NAS Review Draft. December, 2003. EPA/600/P-00/001C(a-f). Available at http://www.epa.gov/ncea/dioxin.htm.

US EPA. (2003b). *Residential Confirmation Cleaning Study*. Interim Final. Prepared by S EPA Region 2, New York Response and Recovery Operations. May, 2003.

Van Den Berg, M., Birnbaum, L., Bosveld, A. T. C., Brunstrom, B., Cook, P., Feeley, M., Giesy, J. P., Hanberg, A., Hasegawa, R., Kennedy, S. W., Kubiak, T., Larsen, J. C., van Leeuwen F. X. R., Liem, A. K. D., Nolt, C., Peterson, R. E., Poellinger, L., Safe, S., Schrenk, D., Tillitt, D., Tysklind, M., Younes, M., Warn, F., Zacharewski, T. (1998). Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives*, *106*, 775–792.

Van Den Berg, M., Birnbaum, L., Denison, M., de Vito, M., Farland, W., Feeley, M., Fiedler, H., Hakansson, H., Hanberg, A., Haws, L., Rose, M., Safe, S., Schrenk, D., Ohyama, C., Tritscher, A., Tuomisto, J., Tysklind, M., Walker, N., Peterson, R. E. (2006). The 2005 World Health Organization re-evaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. *Toxicological Sciences*, *93*(2), 223–241.

Vette, A., Seila, R., Swartz, E., Pleil, J., Webb, L., Landis, M., Huber, A., Vallero, D. (2004a). Air pollution measurements in the vicinity of the World Trade Center. *Environmental Manager*, Feb. 2004.

Vette, A., Gavett, S., Perry, S., Heist, D., Huber, A., Lorber, M., Lioy, P., Georgopoulos, P., Rao, S. T., Petersen, W., Hicks, B., Irwin, J., Foley, G. (2004b). Environmental research in response to 9/11 and homeland security. *Environmental Manager*, February, 14–22.

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.

Wheeler, K., McKelvey, W., Thorpe, L., Perrin, M., Cone, J., Kass, D., Farfel, M., Thomas, P., Brackbill, R. (2007). Asthma diagnosed after September 11, 2001, among rescue and recovery workers: Findings from the World Trade Center Health Registry. *Environmental Health Perspectives*, Advance online 27 August 2007.

Wolff, M. S., Teitelbaum, S. L., Lioy, P. J., Santella, R. M., Wang, R. Y., Jones, R. L., Caldwell, K. L., Sjödin, A., Turner, W. E., Li, W., Georgopoulos, P. Berkowitz, G. S. (2005). Exposures among pregnant women near the World Trade Center site on 9/11. *Environmental Health Perspectives*, *113*(6), 739–748.