Air levels of carcinogenic polycyclic aromatic hydrocarbons after the World Trade Center disaster

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The catastrophic collapse of the World Trade Center (WTC) on September 11, 2001, created an immense dust cloud followed by fires that emitted soot into the air of New York City (NYC) well into December. The subsequent cleanup used diesel equipment that further polluted the air until the following June. The particulate air pollutants contained mutagenic and carcinogenic polycyclic aromatic hydrocarbons (PAHs). By using an assay developed for archived samples of fine particles, we measured nine PAHs in 243 samples collected at or near Ground Zero from September 23, 2001, to March 27, 2002. Based on temporal trends of individual PAH levels, we differentiated between fire and diesel sources and predicted PAH levels between 3 and 200 d after the disaster. Predicted PAH air concentrations on September 14, 2001, ranged from 1.3 to 15 ng/m^3; these values are among the highest reported from outdoor sources. We infer that these high initial air concentrations resulted from fires that rapidly diminished over 100 d. Diesel sources predominated for the next 100 d, during which time PAH levels declined slowly to background values. Because elevated PAH levels were transient, any elevation in cancer risk from PAH exposure should be very small among nonoccupationally exposed residents of NYC. However, the high initial levels of PAHs may be associated with reproductive effects observed in the offspring of women who were (or became) pregnant shortly after September 11, 2001. Because no PAH-specific air sampling was conducted, this work provides the only systematic measurements, to our knowledge, of ambient PAHs after the WTC disaster.

The catastrophic collapse of the World Trade Center (WTC) on September 11, 2001, released an estimated 1 million tons of dust and smoke into the air of New York City (NYC) (1). More than 90% of the airborne mass consisted of large particles (>10 μm in diameter), generated from pulverization of concrete, metals, silica, and organic materials in the structures (1, 2). Although these large alkaline particles were irritating to the upper airways (3), they settled quickly from the air and did not penetrate deeply into the lungs of city residents. Thus, the vast majority of the dust generated by the collapse of the WTC posed a relatively small health risk to the general public. However, fine particles [particulate matter ($PM)_{2.5}$, diameters <2.5 μm] were continually emitted from Ground Zero by fires, and thus became widely dispersed throughout greater NYC [based on the deposited dust (1), an estimated 11,000 tons of $PM_{2.5}$ were released]. Given their deep penetration into the lungs, particles in the $PM_{2.5}$ fraction are of potentially great concern to the public health.

In addition to constituents from the pulverized WTC structures, the $PM_{2.5}$ fraction contained soot particles emanating from fires that persisted from September 11 through December 20, 2001. Fires started with the ignition of ≈91,000 liters of jet fuel (from the two commercial aircraft that initiated the collapse of the WTC) and spread to an estimated 100,000 tons of organic debris, 490,000 liters of transformer oil, 380,000 liters of heating and diesel oil, and fuel from several thousand automobiles (stored in subterranean structures of the WTC) (4–6). Soot from the fires contained numerous carcinogens, notably polycyclic aromatic hydrocarbons (PAHs) that are ubiquitous products of incomplete combustion. Even after the fires were extinguished, PAHs were generated by diesel trucks, cranes, generators, and construction equipment that were used to clear 1,500 million kg of rubble from Ground Zero through May 2002 as described in contemporary major media reports (e.g., CNN, USA Today, etc.) and other official reports (4–6).

Since Pott (7) first linked squamous cell carcinomas with exposure to soot among British chimney sweeps in 1775, PAHs have frequently been associated with human cancers of the skin, lungs, and bladder (8). In fact, our modern understanding of the relationship between cancer and the environment is largely conditioned by investigations involving exposures to PAHs (9–11). Several individual PAHs, including benzo(*a*)pyrene, chrysene, indeno(*1,2,3-c,d*)pyrene, and benzo(*b*)fluoranthene have produced carcinogenic, mutagenic, and genotoxic effects in animal experiments (12–15). Air pollution, presumably enriched with PAHs, has been shown to induce heritable (paternal germ-line) mutations in mice (16). More recently, PAHs have been associated with elevated levels of DNA adducts and P53 mutations in humans (17–19). Airborne PAHs have also been implicated in human reproductive effects, notably, DNA adducts and *hypoxanthine-guanine phosphoribosyltransferase* mutations in newborns as well as preterm birth and intrauterine growth restriction (20–22).

Despite the potential importance of PAH exposures to human health and the generation of large quantities of soot, remarkably little is known about air levels of PAHs in NYC after September 11, 2001. Based on analysis of 13 portions of the settled dust, Offenberg *et al.* (23) estimated that the collapse generated between 100 and 1,000 tons of PAHs. Yet, because the settled dust contained relatively little mass from the $PM_{2.5}$ fraction, it is still an open question as to whether the public was exposed to high levels of airborne PAHs in the wake of September 11, 2001. In fact, only five relevant air measurements of PAHs have been reported in $PM_{2.5}$ samples (24) and these measurements were sufficiently large to motivate "... the most serious kind of concern ... " (25).

Materials and Methods

Air Sampling. In response to the WTC disaster, the U.S. Environmental Protection Agency rapidly began collecting air samples at or near Ground Zero to monitor a variety of pollutants. Four of the sites deployed monitors for fine particulate matter ($PM_{2.5}$) by using Teflon membrane filters through which air was drawn at 5–15 l/min. Three of the sites (designated A, C, and K) were at the fence line of Ground Zero, whereas the fourth site

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Abbreviations: WTC, World Trade Center; NYC, New York City, PAH, polycyclic aromatic hydrocarbon; PM, particulate matter.

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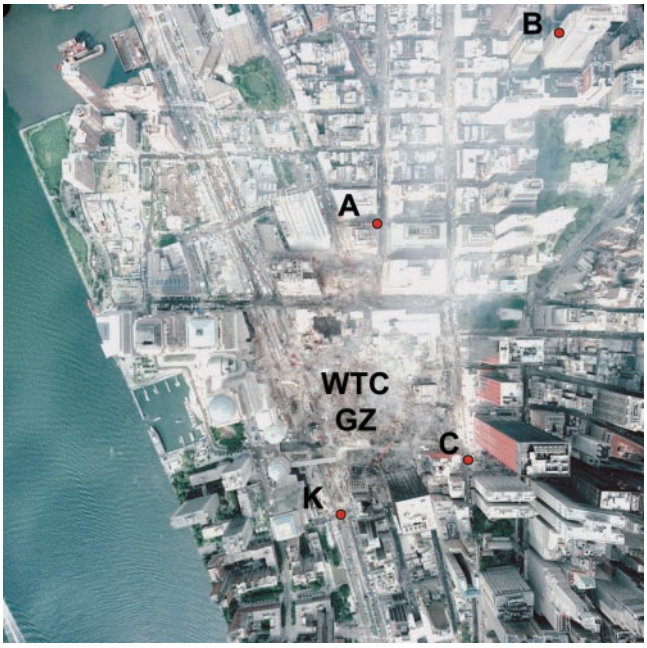

Fig. 1. Aerial photograph of lower Manhattan taken on September 23, 2001, from an altitude of 3,300 feet. Ground Zero (GZ) of the WTC and four sampling sites are labeled. Site A: Park Place and West Broadway. Site B: 290 East Broadway between Reade Street and Duane Street. Site C: Trinity Place and Cedar Street. Site K: West Street and Albany Street. Photo courtesy of the National Oceanographic and Atmospheric Administration (NOAA).

(B) was on the 16th floor of an office building at 290 Broadway, ≈0.5 km from Ground Zero (see Fig. 1). Samples were collected daily at each site (≈24-h duration) between September 23, 2001, and March 27, 2002. After performing nondestructive assays for particle mass (gravimetric) and metal content (x-ray fluorescence), the U.S. Environmental Protection Agency archived the Teflon filters.

Analysis for PAHs. Because PAH-specific samplers were not deployed, we assayed some of the most hazardous four- to six-ring PAHs in 243 of the archived $PM_{2.5}$ filters described above, by using a method we developed for this purpose (26). Briefly, the Teflon filters were extracted with 10 ml of dichloromethane-containing deuterated PAHs as internal standards. After concentration of the extracts to 50 μl under N_2, 2-μl aliquots were analyzed by GC MS in selective ion-monitoring mode. Results are reported for nine carcinogenic and/or mutagenic PAHs, namely, benz(*a*)anthracene, chrysene, benzo(*b*)fluoranthene, benzo(*k*)fluoranthene, benzo(*e*)pyrene, benzo(*a*)pyrene, indeno(*1,2,3-c,d*)pyrene, dibenz(*a,h*)anthracene, and benzo(*g,h,i*)perylene.

Results and Discussion

The overall distribution of airborne PAHs in the 243 air samples is illustrated in Fig. 2, which shows the sum of the nine analyte concentrations versus time after September 11, 2001. Despite considerable variability within and between sites, PAH levels were consistently greater at Ground Zero than at 290 Broadway and were much greater during the first 2 months after September 11, 2001, than thereafter. There is also evidence of declining trends in PAHs levels, particularly in the period before December 20, 2001 (day 100, vertical dashed line).

Relative amounts of individual PAHs differ among combustion sources and thereby serve as potential source signatures (27–29). We observed that the relative contributions of several

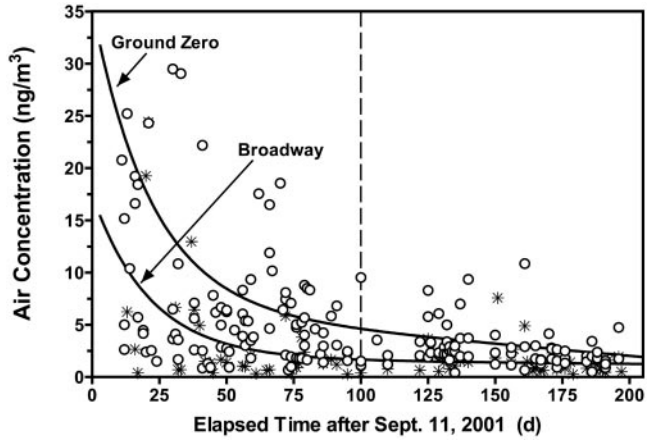

Fig. 2. Scatter plot of PAH levels (sum of nine analytes, ng/m^3) versus time after September 11, 2001. Open symbols, mean values for sites A, K, and C at Ground Zero; filled symbols, site B at 290 Broadway; vertical dashed line, December 20, 2001. Curves are calculated for the summed data based on the regression model presented in Eqs. **1** and **2**.

PAHs changed significantly during the period of observation. This finding is illustrated in Fig. 3 for the increasing percentage of benzo(*g,h,i*)perylene and the decreasing percentage of benzo(*b*)fluoranthene with respect to all measured PAHs. Such behavior suggests that the predominant source(s) of PAHs changed during the investigation, almost certainly from fires to diesel exhaust. This conjecture is supported by Governor Pataki's declaration that all fires were extinguished by December 20, 2001, but WTC rubble continued to be removed by diesel equipment through May 2002 (from contemporary media reports, e.g., U.S. World News, CNN, or USA Today). This conjecture also consistent with the chronology of events offered by Landrigan *et al.* (30) who identified the primary sources of combustion products between September 14 and December 20, 2001, as "smoldering fires (with occasional flare-ups)" and diesel exhaust.

The trends in PAH levels, illustrated in Figs. 2 and 3, can be explained by three underlying processes. First, after the initial collapse, explosions, and fierce jet-fuel fires of the initial two days, PAHs were continually generated by smoldering fires as the

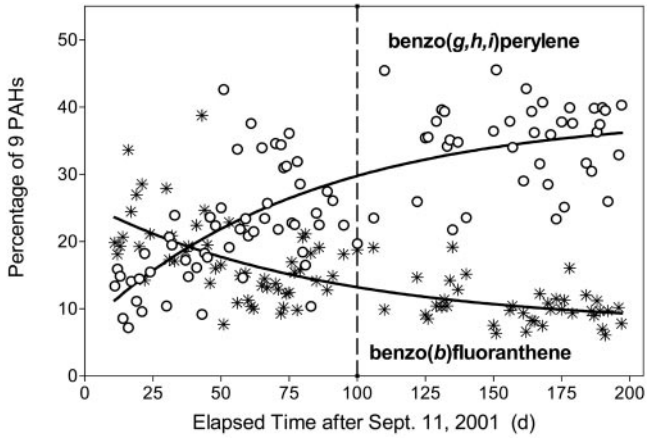

Fig. 3. Percentages of benzo(*g,h,i*)perylene (open symbols) and benzo(*b*)-fluoranthene (filled symbols) to the total of nine PAHs (averaged over all sites) versus time after September 11, 2001. Vertical dashed line represents December 20, 2001, the date that all fires were declared extinguished. Curves represent least-squares fits of a single-compartment model to the data.

Table 1. Predicted air concentrations and 95% confidence intervals (in parentheses) of nine PAHs (ng/m^3) measured at Ground Zero and 290 Broadway on t_j = 3, 100 and 200 d after September 11, 2001

Compound	Ground Zero (n = 170)			290 Broadway (n = 73)			Los Angeles (1998–2002)	Teplice* (Winter 1993–1994)
	t_j = 3	t_j = 100	t_j = 200	t_j = 3	t_j = 100	t_j = 200		
Benz(*a*)anthracene	1.3 (0.18, 2.8)	0.17 (0.14, 0.20)	0.16 (0.13, 0.19)	0.40 (0.00, 0.92)	0.03 (0.02, 0.04)	0.02 (0.01, 0.03)	—	5.8 (5.1, 6.5)
Chrysene	1.5 (0.24, 2.4)	0.16 (0.14, 0.19)	0.15 (0.12, 0.18)	0.37 (0.00, 0.79)	0.04 (0.03, 0.05)	0.03 (0.01, 0.04)	—	7.5† (6.6, 8.4)
Benzo(*b*)fluoranthene	15 (0.82, 29)	0.53 (0.43, 0.63)	0.25 (0.14, 0.35)	5.6 (0.00, 13)	0.36 (0.25, 0.46)	0.07 (0.02, 0.12)	0.17 (0.14, 0.19)	6.2 (5.4, 6.9)
Benzo(*k*)fluoranthene	4.8 (0.72, 9.0)	0.31 (0.26, 0.36)	0.23 (0.17, 0.29)	1.7 (0.00, 3.6)	0.12 (0.09, 0.16)	0.05 (0.02, 0.07)	0.07 (0.06, 0.08)	4.0‡ (3.5, 4.5)
Benzo(*e*)pyrene	7.0 (1.6, 12)	0.42 (0.35, 0.49)	0.30 (0.22, 0.38)	4.1 (0.00, 8.2)	0.21 (0.15, 0.27)	0.09 (0.05, 0.13)	—	4.7 (4.2, 5.3)
Benzo(*a*)pyrene	2.2 (0.07, 4.4)	0.44 (0.36, 0.52)	0.34 (0.25, 0.43)	1.0 (0.00, 2.4)	0.17 (0.12, 0.23)	0.07 (0.03, 0.11)	0.11 (0.04, 0.18)	6.0 (5.3, 6.8)
Indeno(1,2,3-*c*,*d*)pyrene	4.0 (0.06, 7.9)	0.57 (0.47, 0.68)	0.31 (0.19, 0.43)	1.5 (0.00, 3.2)	0.39 (0.28, 0.50)	0.13 (0.06, 0.21)	0.23 (0.19, 0.27)	6.0 (5.3, 6.7)
Dibenz(*a*,*h*)anthracene	1.3 (0.00, 2.9)	0.10 (0.08, 0.12)	0.03 (0.01, 0.06)	0.69 (0.00, 1.7)	0.08 (0.06, 0.11)	0.01 (0.00, 0.03)	0.03 (0.02, 0.03)	2.1 (1.9, 2.4)
Benzo(*g*,*h*,*i*)perylene	4.1 (0.00, 8.8)	1.1 (0.93, 1.3)	0.85 (0.60, 1.1)	1.3 (0.00, 3.1)	0.68 (0.49, 0.87)	0.40 (0.23, 0.58)	0.50 (0.42, 0.57)	5.3 (4.7, 6.0)

Air concentrations in Los Angeles, CA (California Air Resources Board data), and Teplice, Czech Republic (21, 22), are shown for comparison.
*Confidence intervals estimated by imputing variances from Dejmek *et al.* (22).
†Chrysene/triphenylene.
‡Benzo(*k*+*j*)fluoranthene.

available fuel was consumed; this finding suggests a first-order process leading to an exponential decline in PAH emissions over time. Second, PAHs were generated from diesel-fueled activity related to removal of debris from Ground Zero; this result would lead to a quasi-linear decline in PAH emissions as diesel sources diminished from mid-September 2001 to May 28, 2002. Third, PAHs were continuously generated by background sources, notably vehicular engine exhausts in NYC; this result would lead to a near-constant rate of PAH emission throughout the period of observation. With these processes in mind, we constructed the following statistical model to characterize PAH levels in our samples:

$$C_{j,s} = \beta_{0,s}e^{-\alpha_s t_j} + \beta_{1,s}\left(1 - \frac{t_j}{T}\right) + \beta_{2,s} + \varepsilon_{j,s} \text{ for } 0 < t_j \leq t_{0,s}, \quad [1]$$

and

$$C_{j,s} = \beta_{1,s}\left(1 - \frac{t_j}{T}\right) + \beta_{2,s} + \varepsilon_{j,s} \text{ for } t_{0,s} < t_j \leq T, \quad [2]$$

where $C_{j,s}$ represents the level of a given PAH on the jth day (time t_j after September 11, 2001) at site s. The first three terms in Eq. **1** refer, respectively, to the PAH contributions from fires, WTC-derived diesel exhausts (where T = 289 d represents May 28, 2002), and background city contributions. The term $\varepsilon_{j,s}$ represents all random errors arising from sources, changes in wind speed and direction, and assays, whereas $t_{0,i,s}$ is the time at which the fire contribution became negligible for a given site. (The derivation of this model and detailed results are given in *Supporting Text*, which is published as supporting information on the PNAS web site). Although our model includes $t_j < 12$ d (our first observation), we recognize that the nature of the fire-related source changed from combustion of jet fuel during the first two days to smoldering fires thereafter. Thus, in what follows, we restrict our predictions of PAH levels to begin at day 3 when smoldering fires were the primary combustion sources (30).

The above model was applied to each of the nine PAHs by means of nonlinear segmented regression. After preliminary analyses indicated that there were no statistical differences in PAH levels at the three Ground Zero sites (A, C, and K), data from these sites were combined and models were rerun. Observed and predicted levels of the individual PAHs in our samples were similar in appearance to the plots representing the sum of these nine analytes shown in Fig. 2. Because no significant differences were observed in estimates of the time constant α_s (for the fire-derived PAHs) at the different sites, all final models assumed a common time constant $\alpha_{1-4} = 0.0483$ d^{-1} obtained as the estimated mean value for the nine PAHs (SE = 0.0092 d^{-1}); this result corresponds to a half-life of 14.4 d. Given this decay rate, fires contributed insignificant quantities of PAH to the air of NYC after 100 d had elapsed from September 11, 2001. Likewise, no significant differences were detected in the linear (diesel-related) slopes $\beta_{1,s}$ across sites for a given PAH and common values were used for each analyte in the final model.

The estimated model parameters and SE were used to predict air levels and confidence intervals for each PAH at t_j = 3 (September 14, 2001), t_j = 100 (December 28, 2001, fires officially extinguished), and t_j = 200, (March 29, 2002, final day of the study). Table 1 summarizes these predictions for the nine PAHs, and presents comparison values from downtown Los Angeles (mean values for 1998–2002) (furnished by the California Air Resources Board, Sacramento, CA) and from Teplice, in the coal-burning region of the Czech Republic (winter 1993–1994; ref. 31). Our results indicate that, in the immediate aftermath of the WTC disaster (t_j = 3), PAH concentrations at Ground Zero were 10- to 214-fold greater (median = 65-fold) than the background values observed when t = 200 d at 290 Broadway. Indeed, predicted PAH concentrations at t_j = 3 in lower Manhattan were in the range of those observed in Teplice in winter, when some of the highest outdoor PAH concentrations in the world have been reported. However, air levels rapidly declined with the dissipation of fires during the first 100 days and slowly declined thereafter as diesel equipment was phased out, approaching Los Angeles mean values after 200 days. Nonetheless, even then (t_j = 200 d) the contributions of diesel sources close to Ground Zero produced PAH levels that were 2- to 8-fold greater (median = 4-fold) than those at 290 Broadway.

Conclusion

Our retrospective analysis of PAHs in archived $PM_{2.5}$ filters provides an important insight into the environmental implications of the WTC disaster. Without this opportune investigation, inferences about air levels of PAHs would have been restricted to 13 settled dust samples (23) and 5 $PM_{2.5}$ samples (24), which were insufficient for modeling PAH sources and trends.

Given the rapid decline of PAH levels in the aftermath of September 11, 2001, we consider it unlikely that the long-term risks of cancer arising from PAHs would have been significantly elevated above those from background PAH exposures in NYC over 70 years among city residents. Indeed, employing standard methods, we estimate only a 10^{-8} increase in lifetime cancer risk

at Ground Zero from WTC-related PAHs in our samples (details are given in *Supporting Text*, specifically in *Risk Estimates* published as supporting information on the PNAS web site). We temper this observation with knowledge that workers engaged in the cleanup efforts could have been exposed to much higher levels of PAHs than those in our samples and, thus, could bear higher cancer risks. Also, our conclusion regarding cancer risks from PAHs cannot be extended to other carcinogenic substances that were also released to the air after the WTC disaster.

However, because PAH levels were very high during several weeks after the WTC disaster, the potential for adverse reproductive effects cannot be ruled out among the offspring of women who were (or became) pregnant during that period. In fact, a recent report that levels of aromatic DNA adducts were greater in the blood of newborns than of their PAH-exposed mothers in Krakow, Poland (20) suggests that the fetus may be at particular risk to PAH damage. Certainly PAH exposures in early gestation (especially the first month of pregnancy) have been implicated as a source for reproductive effects in the Czech Republic (21, 22), and preliminary results point to a 2-fold elevation of intrauterine growth restriction among the offspring of women residing close to the WTC during those fateful weeks after September 11, 2001 (32). By carefully mapping PAH levels in space and time after the WTC disaster, it may be possible to determine the nature and extent of links between PAH exposure and developmental effects in the local population.

Finally, we caution that the transient nature of exposures to high levels of PAHs from the WTC disaster is only indicative of outdoor air, where massive dilution rapidly reduced the air concentrations. However, dust and soot from the WTC disaster also deeply penetrated residential and commercial buildings, leaving thick layers of residue (33–35). Human indoor activities and contaminated central ventilation, heating, and cooling systems can continually resuspend settled dusts into the air; therefore, indoor air may represent a continuing source of exposure to PAHs (and other particle-related pollutants) that should be considered.

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