A preliminary cancer health risk assessment of a population of inner-city teenagers in New York City

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ABSTRACT

One of the objectives of the Toxics Exposure Assessment Columbia and Harvard (TEACH) Project was to assess the potential health risks associated with exposures to a suite of urban air pollutants in a population of high-school teenagers in New York City. Forty-six high-school students were recruited and most participated in the study in the winter and summer of 1999. Personal, indoor home, and outdoor home 48-h samples were collected in each season. Dual-sorbent thermal desorption tubes were used for the collection VOCs and C_{18} DNPH-coated cartridges were used for carbonyl samples. Filter collection was used for $PM_{2.5}$, and ICP/MS analysis of filters yielded metals composition. Individual cancer risks for VOCs and metals were determined using published cancer potencies. Cancer risks were summed to provide an estimate of overall cancer risk for each subject. Most VOCs had median cancer risks that exceeded 10^{-6}, the EPA benchmark for cancer risk. Only three of the metals had median cancer risks greater than 10^{-6} chromium, nickel and arsenic. Of the top five compounds with the highest cancer risk, only benzene is classified by EPA as a known human carcinogen. These risks are within an order of magnitude range of risks found in the literature for urban areas; however, most studies included polycyclic organic matter in their estimates, which contributes greatly to the overall cancer risk.

INDEX TERMS

VOCs; Carbonyls; Metals; Risk assessment; Personal exposures

INTRODUCTION

Little is known about the adverse health impacts of urban air pollutants, in particular the mix of over 100 hazardous air pollutants (HAPs), as defined by the US EPA in the 1990 Clean Air Act Amendments. One of the potential health impacts associated with exposures to HAPs is cancer. In 1994, the US EPA began the Cumulative Exposure Project, a large modelling effort to assess exposures and risks of toxic contaminants found in air, food, and water across the US (Rosenbaum *et al.*, 1999). Model validation against monitored concentrations showed a tendency for underestimation of concentrations. Furthermore, the US EPA TEAM studies (Wallace *et al.*, 1985, 1988; Wallace, 1987) and other studies that followed (Brown *et al.*, 1992, 1994) have found consistently higher concentrations of various HAPs inside of homes compared to ambient concentrations and personal concentrations often exceeded indoor concentrations. Even given the likely underestimation of population exposures, modelled ambient concentrations of HAPs still exceeded cancer health benchmarks, in at least some of the census tracts (Woodruff *et al.*, 1998). In New York City, modelled median concentrations of benzene, 1,3-butadiene, carbon tetrachloride, chloroform and *p*-dichlorobenzene exceeded cancer health benchmarks by at least a factor of 2, but as much as a factor of 100 for 1,3-butadiene. Few studies have looked at comparative cancer risks associated with HAPs exposures. The Toxics Exposure Assessment Columbia–Harvard (TEACH) Project was designed to collect data on personal, indoor and outdoor concentrations to various HAPs

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including a suite of VOCs and particulate-bound elements, with the goal of determining levels of exposure and potential cancer risks. The study population was inner city high-school students, a never before sampled population. Two-day samples were collected in New York City in two seasons, winter and summer of 1999. This analysis presents a preliminary assessment of the cancer health risks associated with exposures to VOCs and particle-bound elements, based on personal, indoor and outdoor air concentrations.

METHODS

Recruitment of participants was conducted in a single school in east Harlem in New York City (NYC). Each participant provided a personal sample, a home indoor sample and a home outdoor sample. Sampling was conducted in two seasons, winter (February–April 1999) and summer (June–August 1999). The seasons were chosen to maximize the potential differences in pollutant concentrations, with higher concentrations typical in the winter and lower concentrations in the summer for most VOCs and metals. A total of 46 individuals and homes were sampled with 38 in the winter and 41 in the summer. Of these, a total of 33 homes were monitored in both winter and summer. The majority of the homes were located in the upper Manhattan and the Bronx (>80%) and the rest in Brooklyn and Queens.

The personal sampler was run by a BGI pump with the flow split three ways to collect one $PM_{2.5}$ filter at 4 l/min (LPM), one VOC thermal desorption tube (TDT) at 1.8 standard cubic cm per minute (SCCM) and one 2,4-dinitrophenylhydrazine(DNPH)-coated C_{18} carbonyl sampler at ca. 100 SCCM. This personal sampler was housed in a customized daypack that the students carried over their shoulder. The indoor measurements were carried out typically in the living room and the outdoor sampler was setup to monitor through a window in the home. In each city and season, three to five different homes were sampled each week for one 48-h period (typically Tuesday through Thursday), for about 6–8 weeks. Two sampling boxes containing three seven LPM pumps (Medo, Inc.) were used to collect samples inside and outside of each subject's home.

Concentrations of target VOCs were determined using an active sampling method. Samples were collected on multi-sorbent 'Air Toxics' tubes (Perkin-Elmer). The sampling and analytical methods are described in US EPA's Compendium Method TO-17 (Woolfenden and McClenny, 1997). Analysis of VOC tubes was carried out using a Perkin-Elmer Automatic Thermal Desorber (ATD), Model 400 connected to a Hewlett Packard (HP) 5890II GC/5971 MSD. Carbonyls were sampled by the method of Fung and Grosjean (1981), with air pumped through a cartridge packed with C_{18} coated with acidified 2,4-dinitrophenylhydrazine (DNPH). The coated samplers were obtained from ATMAA (Calabasas, CA). The DNPH-derivatives (hydrazones) were eluted with acetonitrile and then analysed using an HPLC (Hewlett Packard 1100) with a UV detector (360 nm).

Field blanks were used to determine background contamination and for calculation of limits of detection (LODs). LODs were mostly lower than 1 $\mu g/m^3$ except for methylene chloride, benzene and 1,4-dichlorobenzene in both seasons and formaldehyde and acetaldehyde in the winter only. The mean relative difference (MRD) was calculated as a measure of the method precision by taking the absolute difference of a pair of duplicates divided by the mean of the pair. For most compounds the mean relative difference was below 25%, 1,3-butadiene had the highest MRD (41%). VOC and carbonyl breakthrough were also tested. Only benzene showed breakthrough in a few samples and this was probably a result of background contamination. Mean analytical recoveries ranged from 73 to 149%. Samples that were lost due to equipment or analytical problems were excluded from data analysis. All concentrations were blank corrected and any negative or zero values were set to half the LOD.

Personal concentrations were used to assess the individual cancer risks for a total of 13 VOCs and seven particle-bound metals using published cancer potencies. The cancer risk estimate was derived from the inhalation unit risk (in $\mu g/m^3)^{-1}$. These inhalation unit risk factors were taken from Caldwell *et al.* (1998) and represent the probability that an individual will develop cancer as a result of exposure to 1 $\mu g/m^3$ of the compound over a lifetime (70 years). They are typically non-threshold linear, high dose to low dose extrapolations from animal or occupational studies. The unit risks are calculated by using either maximum likelihood estimates from a dose–response relationship or represent the 95% upper bound estimate. If we multiply the unit risk by the personal concentration of each individual we obtain the individual lifetime risk associated with exposure to any given carcinogen. An additive model was assumed to determine the cumulative risks associated with VOCs and metals. Time-weighted indoor and outdoor concentrations were calculated using time–activity data and cumulative cancer risk estimates were calculated based on these concentrations.

RESULTS

Tables 1 lists the carcinogenic compounds, mean personal exposures averaged across the two seasons, and cancer unit risks for the VOCs and metals included in the analysis. Average personal concentrations generally exceeded both indoor and outdoor concentrations, often by an order of magnitude or more for VOCs and less so for particle-bound metals. Figures 1 and 2 show the distribution of cancer risks for VOCs and metals, respectively. Most of the VOCs had median cancer risks that exceeded 10^{-6}, which is the EPA benchmark for cancer risk (Woodruff *et al.*, 1998). Only three of the metals had median cancer risks greater than 10^{-6}, chromium, nickel and arsenic. The highest risk contributions come from formaldehyde, 1,3-butadiene, 1,4-dichlorobenzene, chloroform and acetaldehyde. Interestingly, benzene (the only known human carcinogen) ranked sixth highest due to low personal concentrations. VOC cumulative risks were similar for personal concentrations and time-weighted indoor concentrations, with risks spanning two orders of magnitude (10^{-4}–10^{-2}) across individuals. Cumulative risks associated with time-weighted outdoor concentrations were much lower and spanned almost three orders of magnitude (10^{-7}–10^{-5}). For metals, personal cumulative risks were almost an order of magnitude higher compared with time-weighted indoor concentrations and were substantially higher than outdoor cumulative risks.

DISCUSSION

The carcinogenic risk associated with personal exposures was estimated both for individual compounds and cumulatively for all the sampled VOCs and metals that are known or suspected carcinogens. Morello-Frosch *et al.* (2000) assessed air toxics cancer risks for California based on modelled ambient concentrations and found that 16 of 89 pollutants analysed accounted for 97% of estimated excess lifetime cancers. Of these four (polycyclic organic matter (POM), 1,3-butadiene, formaldehyde, and benzene) accounted for 70% of the cancer cases. POM alone accounted for 34% of the cancer risk. The median cancer risk for all air toxics was found to be 2.7×10^{-4}, an order of magnitude lower than our results. Similarly, a risk assessment by Pratt *et al.* (2000) yielded a maximum cancer risk of 4.7×10^{-5} based on monitored ambient concentrations in Minnesota. An estimate across all of the US yielded a median cancer risk estimate of 1.8×10^{-4} (Woodruff *et al.*, 2000). All of these studies used modelled or monitored ambient concentrations. The concentrations from personal exposures can be at least 10 times higher than ambient concentrations and thus may explain the 10-fold difference in cancer risk. One study of cancer risks using the TEAM data obtained mean

Table 1 Summary statistics of personal, indoor home and outdoor home concentrations of VOCs and metals for New York City teenagers, averaged across two seasons

		Personal			Indoor			Outdoor		
Compound	**Cancer Unit Risk[1]**	**N**	**Median**	**Range**	**N**	**Median**	**Range**	**N**	**Median**	**Range**
VOCs (μg/m^3)										
1,3 Butadiene	2.8E-04	44	0.52	0.03-5.9	41	0.49	0.03-10	42	0.03	0.03-22
1,4-Dichlorobenzene	6.7E-06	43	13	2.3-353	40	9.8	0.41-1637	40	2.6	0.61-38
Acetaldehyde	2.2E-06	45	13	5.2-86	45	13	5.3-92	44	3.2	1.5-7.5
Benzene	7.7E-06	44	3.6	0.54-12	41	2.9	0.02-23	41	1.8	0.33-5.6
Carbon Tetrachloride	1.5E-05	44	0.64	0.05-1.1	41	0.65	0.05-1.0	42	0.64	0.32-0.97
Chloroform	2.3E-05	44	2.6	0.06-9.5	41	2.7	0.06-9.2	42	0.14	0.06-2.2
Ethylbenzene	5.0E-07	43	2.5	0.96-10	40	1.9	0.49-20	40	1.2	0.34-6.0
Formaldehyde	1.3E-05	45	18	4.7-55	45	16	6.6-46	44	3.1	0.53-7.3
Methylene Chloride	4.8E-07	44	2.1	0.35-169	41	1.9	0.41-199	42	0.92	0.20-14
MTBE	1.7E-07	44	14	5.4-253	41	13	4.9-209	42	11	2.2-82
Styrene	5.0E-07	43	1.1	0.48-6.9	40	0.89	0.24-2.8	40	0.33	0.13-1.2
Tetrachloroethylene	5.9E-07	43	4.2	0.09-62	40	3.6	1.1-88	40	1.6	0.44-98
Trichloroethylene	1.7E-06	43	0.37	0.08-37	40	0.36	0.08-22	40	0.24	0.08-0.92
Metals (ng/m^3)										
Antimony (Sb)	5.0E-07	40	0.40	0.18-2.6	45	0.87	0.27-71	45	1.1	0.20-3.8
Arsenic (As)	4.3E-03	45	5.3	2.1-1667	38	0.34	0.13-0.90	35	0.28	0.17-078
Beryllium (Be)	2.4E-03	33	0.002	6.7E-04-0.004	30	0.001	2.8E-05-0.003	23	0.002	1.5E-06-0.01
Cadmium (Cd)	1.8E-03	45	0.18	0.07-1.1	45	0.15	0.04-0.77	45	0.12	0.02-0.40
Chromium (Cr)	1.2E-02	39	1.0	0.14-7.8	38	0.50	0.06-1.3	33	0.35	0.05-2.2
Lead (Pb)	7.7E-05	45	16	1.6-353	45	4.8	1.4-198	45	5.3	0.96-20
Nickel (Ni)	2.4E-04	45	0.90	0.31-48	45	15	1.3-348	45	19	0.14-94

[1]Inhalation Unit Risk in μg/m^3 taken from Caldwell *et al.* (1998).

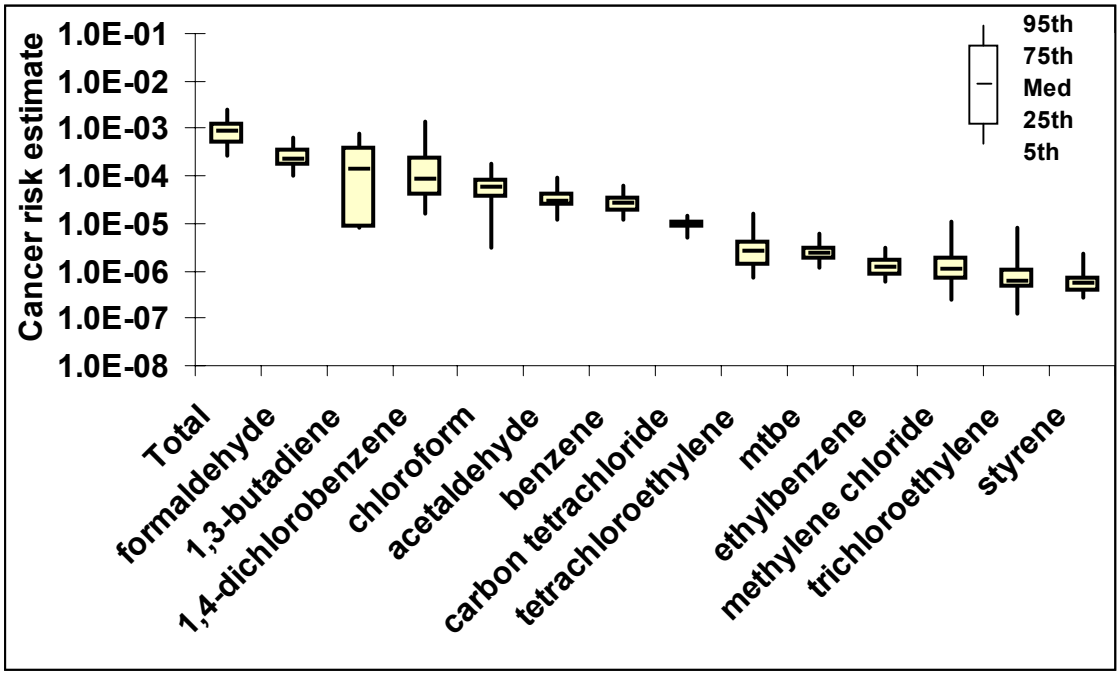

Figure 1 Distribution of cancer risks associated with personal exposures to VOCs.

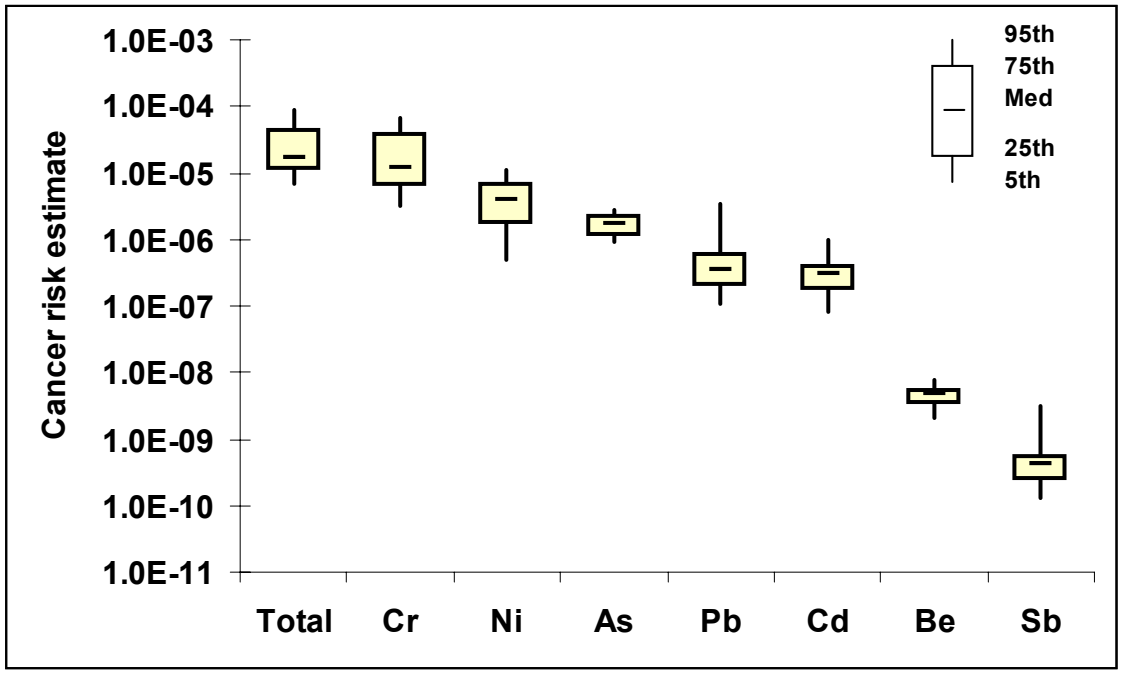

Figure 2 Distribution of cancer risks associated with personal exposures to metals.

cancer risks of 1.9×10^{-2} for New Jersey (Tancrede *et al.*, 1987). In that study all monitored pollutants were considered to have a cancer risk, which may account for the high cancer risk. Our estimates fall in the middle of the studies using indoor concentrations (Tancrede *et al.*, 1987) and those using the ambient concentrations (Woodruff *et al.*, 2000; Morello-Frosch *et al.*, 2000; Pratt *et al.*, 2000) and are considerably higher than the benchmark risk level of 10^{-6} established by the EPA. It should be noted that of the top five compounds with the highest cancer risk, only benzene is classified by EPA as a known human carcinogens. Both MTBE and ethyl benzene are not classifiable as carcinogens because of insufficient human and animal data.

CONCLUSIONS AND IMPLICATIONS

These risk estimates should be used as a guide to help prioritize research and can be indicative of potential hazards faced by these urban dwelling teenagers. There are several inherent limitations to this risk analysis, however, that may under- or overestimate the cancer risks. There are many known uncertainties associated with the inhalation unit risks. The toxicity data derived from animal studies has uncertainty associated with extrapolations from high doses used in animals to the low human exposures. Also, extrapolating from animals to humans provides additional uncertainty. Data collected from occupational studies has uncertainty associated with the high doses and also from the occupational cohorts that may not be representative of the overall human population (i.e. health male workers). This risk analysis is not a comprehensive analysis of all potential carcinogenic compounds. Of particular importance to cancer risks are the myriad of polycyclic aromatic hydrocarbons (PAHs) of which benzo(a)pyrene is a well-characterized and potent carcinogen. As noted above, studies have found that PAHs could contribute as a large percentage to the total cancer risk for ambient air toxics. Since we do not consider PAHs and many other carcinogens in our analysis, the cancer risks may be underestimated. An additional limitation is the lack of

knowledge regarding the speciation of the metals. For chromium, for example, only the hexavalent chromium is considered to be carcinogenic, and we only have data on total chromium. This may overestimate the cancer risks associated with chromium exposure. Despite the limited number of compounds monitored in this study and the inherent limitations of the risk assessment, there appears to be evidence of health concerns for these urban-dwelling teenagers. Only cancer endpoints were considered in this analysis, future work will consider other toxicity endpoints.

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