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Prediction of health risk due to polycyclic aromatic hydrocarbons present in urban air in Rio de Janeiro, Brazil

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ABSTRACT. Risk assessment can provide a comprehensive estimate of potential effects of contaminants under specific, well-defined, and well-described circumstances, providing quantitative relationships between exposure and effects to identify and to define areas of concern. We investigated the mutagenic activity of particulate matter in air samples collected from three sites in Rio de Janeiro city. Samples were collected using a high-volume sampler at Avenida Brasil, at Campus of Universidade do Estado do Rio de Janeiro, and at Rebouças Tunnel. Six polycyclic aromatic hydrocarbons were quantified by gas chromatography/mass spectrometry. *Salmonella typhimurium* TA98 and the derivative strains TA98/1.8-DNP$_6$, YG1021, and YG1024,

commonly used in mutagenicity assays, were treated (10-50 μg/plate), with and without exogenous metabolization. The highest values for the polycyclic aromatic hydrocarbons were detected at Rebouças Tunnel. For chrysene, as an example, the concentration was nearly 200 times higher than that established by the US Environmental Protection Agency. Frequent traffic jams can place bus drivers who go through the Rebouças Tunnel at risk of exposure to up to 0.69 ng/m^3 benzo(a) pyrene. Independent of exogenous metabolization, mutagenicity was detected in strains YG1021 and YG1024 at all the sites, suggesting nitro and amino derivatives of polycyclic aromatic hydrocarbons. Rebouças Tunnel air samples gave the highest values for rev/μg and rev/m^3. This could be due to the fact that the long, enclosed passageway through a mountain restricts ventilation. The cancer risk estimate in this study was 10^{-3} for the benzo(a)pyrene, at the two sites, indicating a high risk.

Key words: Respirable particulate matter; PAHs; Risk assessment; *Salmonella*/microsome assay; Mutagenicity

INTRODUCTION

The risk assessment process involves the characterization of toxicities and estimation of possible adverse outcomes from specific chemical exposures (Environment Canada, 1997). The US Environmental Protection Agency (USEPA Draft Cancer Risk Assessment Guidelines, 1996) defines risk characterization as the step in the risk assessment process that integrates hazard identification, dose-response assessment, and exposure assessment, using a combination of qualitative and quantitative information.

The World Health Organization (WHO, 2005) considers air pollution to be an environmental exposure situation that can affect human health, where it is implicated in acute respiratory infections, cancer, and chronic respiratory and cardiovascular diseases. Studies around the world have consistently demonstrated that particulate matter (PM) with an aerodynamic diameter <10 μm (PM10) and, more recently, <2.5 μm (PM2.5), poses a significant threat to human health (Vinitketkumnuen et al., 2002) as it can penetrate deep into alveolar sacs in the lungs. It has been suggested that fine particles from automotive emissions are responsible for a 3% rise in mortality rate for every 10 μg/m^3 increase, while fine coal combustion emissions account for only 1%, and fine crystal aerosols have no discernible effect (Laden et al., 2000).

Urban airborne PM is a complex variable mixture containing many different chemical species (USEPA, 1996; Cassoni et al., 2004). Ambient air genotoxins can originate from fuel combustion (motor vehicle exhausts, central heating, and power generation), waste incineration, and industrial processes, and are also formed by atmospheric reactions (Claxton and Woodall Jr., 2007; Umbuzeiro et al., 2008). Studies on organic extracts of urban PM have proven their genotoxicity (Vinitketkumnuen et al., 2002; Cassoni et al., 2004), revealing the risk it poses to exposed populations. Generally, the mutagenicity of airborne combustion particles is primarily attributed to polycyclic aromatic hydrocarbons (PAHs), but recent reviews have demonstrated that these compounds are not the most predominant class of mutagens in airborne particulate matter, although they significantly contribute to mutagenicity. A wide

range of aromatic compounds, such as nitroarenes, are found in ambient air and are present in emissions from direct sources or may be products of atmospheric reactions in the presence of NO_2 and NO_3 radicals (Coronas et al., 2009).

The objective of the present study was to make a risk assessment of the PM samples collected at three sites (Avenida Brasil, Rebouças Tunnel, and Campus of Universidade do Estado do Rio de Janeiro) in Rio de Janeiro between April and July 2010 for mutagenic activity using a *Salmonella*/microsome assay, as described by Kado et al. (1983).

MATERIAL AND METHODS

Sampling sites

The samples were collected at three sites in Rio de Janeiro: Avenida Brasil (site 1), Campus of Universidade do Estado do Rio de Janeiro (site 2), and Rebouças Tunnel (site 3) between April and July 2010. Site 1 has heavy traffic (~250,000 vehicles/day) and is the city's biggest highway, spanning 58 km in length and crossing 27 neighborhoods. Site 2, with little traffic, is located in a residential area in the city's north zone. Site 3 has heavy traffic (~190,000 vehicles/ day). It connects the north and south zones of the city and is 2.8 km long.

Sampling of airborne particulate matter and extraction of organic compounds

Airborne PM2.5 samples were collected on fiberglass filters (E558 X 10IN, 254 mm x 203 mm) using a high-volume collector (Energética Indústria e Comércio Ltda., AVG MP 2.5, 1.13 m^3/min) for 24 h at Avenida Brasil and Universidade Federal do Rio de Janeiro, and 6 h in Rebouças Tunnel. Samples were collected each week from April to July 2010.

Half of each filter was extracted with dichloromethane (CASRN. 75-09-2, TediaBrazil, Brazil, purity 99.9%) at 40°C by sonicating for three rounds of 10 min each (Vargas et al., 1998). The extracts were concentrated to 15 mL in a rotary evaporator and filtered on a Teflon membrane (0.5 µm). The concentration of extractable organic matter (EOM, in µg/m^3) was calculated. Prior to bioassays, the organic extract was dried at 4°C and resuspended in dimethyl sulfoxide (DMSO, CASRN. 67-68-5, Synth, Brazil, purity 99.9%) (Vargas et al., 1998).

Analysis of PAHs

PAHs were quantified by gas chromatography/mass spectrometry (GC/MS). They were identified and quantified using a Varian system consisting of a GC (450-GC) with a split/splitless injector 1177S/SL (kept at 300°C) coupled to a mass spectrometer detector (MS 220). The ion trap (250°C), manifold (280°C), and transfer line (280°C) were maintained at constant temperatures. PAHs were identified by mass similarity and by the retention time of the components in a commercial standard kit (Supelco, PAH610-S).

Quantification was based on five calibration points, which were constructed from each standard for all the target analytes, ranging from 10 to 250 pg/µL. Injections (2.0 µL) were splitless, with the split opened after 0.5 min, and helium was used as the carrier gas. A VF-5MS column (30 m x 0.25 mm x 0.25 µm) was employed. The column and septum purge flows were set at 1.6 and 3 mL/min, respectively. The oven temperature program was as follows:

70°C for 4 min and 70-300°C at 10°C/min. These conditions were designed for the analysis of six PAHs: phenanthrene, fluoranthene, pyrene, benzo(a)anthracene, chrysene, and benzo(a) pyrene. The limits of quantification were determined from the minimum point in the calibration curves. Limits of detection were determined from PAH concentrations, which resulted in a signal-to-noise ratio of 3:1. The results were expressed in ng/m^3.

Salmonella/microsome assay

The organic extracts were assayed for mutagenicity using the microsuspension version (Kado et al., 1986) of the *Salmonella*/microsome assay (Maron and Ames, 1983). *Salmonella typhimurium* TA98 (frameshift strain) and the derivative strains YG1021 (nitroreductase-overproducing), TA98/1.8-DNP$_6$ (O-acetyltransferase deficient) and YG1024 (O-acetyltransferase-overproducing) were used with and without metabolic activation (S9 mix fraction). Five concentrations of each sample (10, 20, 30, 40, and 50 µg/plate) were tested in triplicate. The samples were pre-incubated for 90 min. All assays were carried out under yellow light along with a negative control (DMSO solvent, 5 µL/plate) and positive controls (4-nitroquinoline oxide, 0.5 µg/plate, CASRN. 56-57-5 and 2-aminofluorene, 1 µg/plate, CASRN. 153-78-6, Sigma Chemical Company, St. Louis, MO, USA). Plates were incubated in the dark at 37°C for 72 h, after which time revertants were counted. The assay response for mutagenicity was considered positive when the number of revertant colonies in the test was at least twice the number of spontaneous revertants; the responses were expressed in rev/µg and rev/m^3. In the cytotoxicity test, the solution containing the sample and the bacterial culture (100-200 cells) were plated on nutrient agar plates and incubated at 37°C for 24 h, and the surviving colonies were counted (Vargas et al., 1998).

Statistical analysis

The mutagenic response was considered positive when the number of revertant colonies in the test was at least twice the number of spontaneous revertants and no cytotoxicity was detected (survival rates > 60%). The significant responses were identified by statistical analysis (ANOVA, $P \leq 0.05$) (Vargas et al., 1998).

Risk assessment

The risk assessment was calculated for each detected PAH by potency equivalency factors (PEFs) as described in Collins et al. (1998).

RESULTS AND DISCUSSION

Airborne PM

The PM2.5 concentration and EOM of the samples in µg/m^3 are shown in Table 1.

WHO (2005) has established guidelines for long-term and short-term PM2.5 concentrations: 10 µg/m^3 (annual mean) and 25 µg/m^3 (24-h mean). Most of our results were above the levels established by WHO (2005; Table 1). A previous study (Godoy et al., 2009) carried

out near site 2 also detected low concentrations of PM2.5 (18.10 µg/m^3), probably due to the fact that this area has low traffic flows and is far from any industry. A previous study done by Instituto Estadual do Ambiente (2009) to monitor PM10 in the metropolitan area of Rio de Janeiro showed a high annual average of this pollutant (64 µg/m^3). WHO (2005) has established a guideline for long-term PM10 concentrations of 20 µg/m^3 (annual mean). PM10 can only penetrate the upper respiratory tract, whereas PM2.5 can penetrate the lungs and cause various diseases (Claxton and Woodall Jr., 2007). It has been demonstrated that for each 10 µg/m^3 increase in PM concentration, the risk of mortality from cardiopulmonary diseases increases 6%, while the risk of mortality from lung cancer rises 8% (Ianistcki et al., 2009). Our results showed high concentrations of PM2.5 at sites 1 and 3. Most of the PM released into the atmosphere is from diesel-powered vehicles (Claxton and Woodall Jr., 2007). In Brazil, the fleet of gasoline-fueled vehicles rose by 8% between 1996 and 2000, while for the same period there was a 60% increase in the number of diesel vehicles (SISAET, Sistema de Informações do Anuário Estatístico dos Transportes, 2011). The composition of diesel and gasoline has been changed (Braun et al., 2003) in a bid to reduce their pollutant emissions. Nevertheless, the PM concentration was found to be high at sites 1 and 3 - Avenida Brasil and Rebouças Tunnel - where the traffic of diesel vehicles is heavy. The concentrations of PM2.5 detected at sites 1 and 3 are in agreement with other studies performed in urban areas: Santiago, Chile (33.00 µg/m^3) (Seguel et al., 2009), Palermo, Italy (34.20 µg/m^3) (Dongarrá et al., 2010), and Hong Kong (68.60 µg/m^3) (Cheng et al., 2010).

Table 1. Collection sites, air volume, PM2.5 concentration, and extractable organic matter (EOM) of the samples analyzed.

Site	Month	Air volume (m^3) ± SD	PM2.5 (µg/m^3) ± SD	EOM (µg/m^3)
Avenida Brasil (1)	April	1545 ± 1	40 ± 12	7.76
	May	1521 ± 22	60 ± 22	34.51
	June	1523 ± 9	34 ± 21	32.99
	July	1518 ± 1	35 ± 8	4.93
UERJ (2)	April	1618 ± 87	14 ± 7	6.02
	May	1518 ± 65	21 ± 9	8.16
	June	1514 ± 63	35 ± 28	8.23
	July	1545 ± 1	36 ± 15	4.85
Rebouças Tunnel (3)	July	413 ± 1	83 ± 24	18.18

PM = particulate matter; UERJ = Universidade do Estado do Rio de Janeiro.

Analysis of PAHs

At site 1 in April, 0.07 ng/m^3 chrysene was detected. In May, the following PAHs were detected: phenanthrene (0.07 ng/m^3), fluoranthene (0.13 ng/m^3), pyrene (0.22 ng/m^3), benzo(a) anthracene (0.15 ng/m^3), chrysene (0.26 ng/m^3), and benzo(a)pyrene (0.74 ng/m^3). In June, none of the PAHs evaluated were detected. In July, the following were detected: pyrene (0.07 ng/m^3), benzo(a)anthracene (0.08 ng/m^3), chrysene (0.17 ng/m^3), and benzo(a)pyrene (0.19 ng/m^3). None of the PAHs under study was detected at site 2 during the study period.

At site 3, we detected phenanthrene (0.23 ng/m^3), fluoranthene (0.42 ng/m^3), pyrene (0.53 ng/m^3), benzo(a)anthracene (0.80 ng/m^3), chrysene (1.78 ng/m^3), and benzo(a)pyrene (1.65 ng/m^3).

All PAHs evaluated in the present study are considered a priority in environmental monitoring, but only benzo(a)pyrene is believed to be a human carcinogen (group 1). Chrysene and benzo(a)anthracene are considered to be possibly carcinogenic to humans (group 2B), and the other PAHs evaluated in these study are classified as non-carcinogenic to humans (group 3) (IARC, 2011). By the estimated unit of risk, the chronic dose of benzo(a)pyrene for cancer is 1.10 ng/m^3, followed by 0.11 ng/m^3 for benzo(a)anthracene, and 0.01 ng/m^3 for chrysene (USEPA, 1996). Benzo(a)pyrene is the most carcinogenic, with the doses of benzo(a)anthracene and chrysene being calculated from benzo(a)pyrene. Benzo(a)pyrene has been identified in environmental studies as having the highest carcinogenic potential, with the capacity to form adducts and produce base substitutions and frameshifts (Fahl et al., 1981) in the DNA chain. Our study detected concentrations above the recommended levels estimated by USEPA, for chrysene (at site 1 - April, May, and July, and at site 3 - July), benzo(a)anthracene (at site 1 - May and at site 3 - July), and benzo(a)pyrene (at site 3 - July). The urban atmosphere in Rio de Janeiro is influenced by several factors, such as the uneven topography, the irregular occupation of space, the presence of open sea, and Guanabara Bay, which result in a complex regime for winds and irregular distribution and dispersion of pollution (Azevedo et al., 1999). A previous study performed from December 1998 to March 1999, close to site 1, reported similar values for benzo(a)pyrene (0.57-0.75 ng/m^3) (Fernandes et al., 2002). These results indicate that this pollutant is present during different periods of the year at this site. In another study in 1999, at site 3, 0.58 ng/m^3 benzo(a)pyrene was determined, while we detected 1.65 ng/m^3. This difference may be related to changes in the flow of vehicles, from about 7200 vehicles/day back then (Azevedo et al., 1999) to the present day level of 190,000 vehicles/day (CET-Rio - Companhia de Engenharia de Tráfego do Rio de Janeiro, 2011).

The existence of phenanthrene, fluoranthene, pyrene, benzo(a)anthracene, chrysene, and benzo(a)pyrene at sites 1 and 3 can be attributed to the heavy traffic and the absence of dispersion factors such as rainfall (especially at site 3). Moreover, the results found for sites 1 and 3 are in agreement with other studies performed in urban areas: Hong Kong (Zheng and Fang, 2000), Santiago (Kavouras et al., 1999) and São Paulo (Bourottea et al., 2005).

Salmonella/microsome assay

Tables 2 and 3 show the mutagenicity data for the organic extracts from airborne PM in rev/μg and rev/m^3, respectively.

At site 1, positive responses were observed for TA98 both in the absence (April and May) and in the presence (July) of metabolic activation. A positive response was observed for TA98/1.8-DNP_6 in July in the absence and in the presence of S9 mix. For YG1021, a positive response was observed in April, also in the absence and presence of S9 mix, while a positive response was observed in May, June, and July only in the presence of S9 mix. For YG1024, a positive response was observed in April in the presence of S9 mix, in May in the absence of S9 mix, and in July in the absence and presence of S9 mix.

At site 2, positive responses were observed for TA98 in the presence of metabolic activation in May, and in the absence and presence of metabolic activation in July. For TA98/1.8-DNP_6, a positive response was observed in the absence of metabolic activation in June and July. For YG1021 a positive response was observed in the presence of S9 mix in June and July. For YG1024, a positive response was observed in April in the absence of S9 mix and in July in the absence and presence of S9 mix.

Table 2. Mutagenicity of airborne particulate matter organic extracts in rev/µg.

Site	Month	TA98		TA98/1.8-DNP$_6$		YG1021		YG1024	
		-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9
1	April	2.39 ± 0.05	nd	nd	nd	1.30 ± 0.35	1.18 ± 0.14	nd	3.35 ± 1.48
	May	4.48 ± 1.02	nd	nd	nd	nd	1.86	1.40 ± 0.20	nd
	June	nd	nd	nd	nd	nd	1.77 ± 0.25	nd	nd
	July	nd	4.53 ± 0.14	6.50 ± 0.57	1.53 ± 0.29	nd	3.53 0.08	5.60 ± 1.69	4.73 ± 0.39
2	April	nd	nd	nd	nd	nd	nd	0.90 ± 0.14	nd
	May	nd	2.51± 1.40	nd	nd	nd	nd	nd	nd
	June	nd	nd	1.61 ± 0.05	nd	nd	2.55 ± 0.40	nd	nd
	July	2.85	3.28 ± 0.35	2.51 ± 0.84	nd	nd	2.05 ± 0.25	3.40 ± 0.53	1.59 ± 0.51
3	July	3.43 ± 0.67	1.80 ± 0.13	1.93 ± 0.63	nd	3.80 ± 0.57	1.95 ± 0.07	7.50 ± 4.38	5.47 ± 0.29

nd = not detected. Negative control = DMSO for the mutagenicity assay without S9 mix: TA98 (35 ± 5); TA98/1.8-DNP$_6$ (14 ± 2); YG1021 (36 ± 12); YG1024 (21 ± 3). For the mutagenicity assay with S9 mix: TA98 (59 ± 10); TA98/1.8-DNP$_6$ (22 ± 2); YG1021 (24 ± 8); YG1024 (44 ± 2). Positive controls for the mutagenicity assay without S9 mix: 0.5 µg/plate 4-nitroquinoline oxide for TA98 (948 ± 56); TA98/1.8-DNP$_6$ (1214 ± 119); YG1021 (953 ± 62); YG1024 (1154 ± 97). For the mutagenicity assay with S9 mix: 1 µg/plate 2-aminofluorene for TA98 (184 ± 15); TA98/1.8-DNP$_6$ (246 ± 47); YG1021 (1216 ± 104); YG1024 (235 ± 70).

At site 3, positive responses were observed for TA98 in the absence and presence of metabolic activation. For TA98/1.8-DNP$_6$, a positive response was observed only in the absence of S9 mix. For YG1021 and YG1024, positive responses were observed in the absence and presence of metabolic activation (Table 3).

The mutagenicity observed in the presence of metabolic activation may be associated with the presence of promutagens, such as PAHs. Benzo(a)pyrene was present in the samples from site 1 (May and July) and site 3 (July). The positive response for TA98 in the presence of S9 mix might have been related to this PAH at sites 1 and 3 in July. It is known that benzo(a)pyrene can induce frameshift mutations in DNA at a concentration of 0.5 µg/plate for TA98 in the presence of metabolic activation (Aouadene et al., 2008). In our results, we detected benzo(a)pyrene at site 1 (0.05 µg/plate) in July, and at site 3 (0.61 µg/plate) also in July. Although we detected the presence of benzo(a)pyrene in May at site 1, no mutagenic response related to this PAH was observed.

The positive response observed at the three sites for the O-acetyltransferase strains (TA98/1.8-DNP$_6$ and YG1024) suggests the presence of amino compounds. The positive response observed at the three sites for the nitroreductase-overproducing strain (YG1021) could be related to the presence of nitro derivatives of PAHs.

At sites 1 and 2 (outside), the highest values for rev/µg and rev/m^3 for different strains (Tables 3 and 4, respectively) were observed in July (winter). In Rio de Janeiro, there is normally little rainfall in winter. The lack of rain in this season favors the accumulation of these pollutants in the atmosphere. A previous study evaluating the mutagenicity of PM10 near site 2 in the winter of 1984 showed a positive response for TA98 in the presence of S9 mix (2.60 rev/µg and 5.98 rev/m^3) (Miguel et al., 1990). At this site in the same season, we found higher values for TA98 in the presence of S9 mix (3.28 rev/µg and 15.98 rev/m^3) (Tables 2 and 3, respectively). These data indicate that the PM2.5 particles contained more PAHs/µg than did the PM10 particles (Claxton and Woodall Jr., 2007).

Site 3 (inside) exhibited the highest rev/µg and rev/m^3 values of the three sites. This finding might have been related to the fact that the long enclosed tunnel running through the mountain has limited ventilation and high traffic volume.

Table 3. Mutagenicity of airborne particulate matter organic extracts in rev/m³.

Site	Month	TA98		TA98/1.8-DNP$_6$		YG1021		YG1024	
		-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9
1	April	18.50 ± 0.38	nd	nd	nd	10.08 ± 2.74	9.15 ± 1.09	nd	26.00 ± 11.52
	May	154.60 ± 35.14	nd	nd	nd	nd	64.18	48.50 ± 6.36	nd
	June	nd	nd	nd	nd	nd	58.49 ± 8.25	nd	nd
	July	nd	22.32 ± 0.70	32.04 ± 2.78	7.52 ± 1.42	nd	17.41 ± 0.37	27.60 ± 8.36	23.29 ± 1.92
2	April	nd	nd	nd	nd	nd	nd	5.41 ± 0.85	nd
	May	nd	20.48 ± 11.42	nd	nd	nd	nd	nd	nd
	June	nd	nd	13.24 ± 0.45	nd	nd	19.33 ± 2.32	nd	nd
	July	13.82	15.90 ± 1.71	12.20 ± 4.07	nd	nd	9.94 ± 1.20	16.51 ± 2.61	7.73 ± 2.50
3	July	62.41 ± 12.10	32.72 ± 2.40	35.14 ± 11.43	nd	69.08 ± 10.28	35.45 ± 1.28	136.34 ± 79.70	99.50 ± 5.22

nd = not detected. Negative control = DMSO for the mutagenicity assay without S9 mix: TA98 (35 ± 5); TA98/1.8-DNP$_6$ (14 ± 2); YG1021 (36 ± 12); YG1024 (21 ± 3). For the mutagenicity assay with S9 mix: TA98 (59 ± 10); TA98/1.8-DNP$_6$ (22 ± 2); YG1021 (24 ± 8); YG1024 (44 ± 2). Positive controls for the mutagenicity assay without S9 mix: 0.5 µg/plate 4-nitroquinoline oxide for TA98 (948 ± 56); TA98/1.8-DNP$_6$ (1214 ± 119); YG1021 (953 ± 62); YG1024 (1154 ± 97). For the mutagenicity assay with S9 mix: 1 µg/plate 2-aminofluorene for TA98 (184 ± 15); TA98/1.8-DNP$_6$ (246 ± 47); YG1021 (1216 ± 104); YG1024 (235 ± 70).

Table 4. Estimating risk assessment for each polycyclic aromatic hydrocarbon (PAH) in relation to the benzo(a)pyrene in Avenida Brasil and Rebouças Tunnel.

Sites	PAHs	PEF	Individual cancer risk
Avenida Brasil (1)			
April	Chrysene	0.01	$7.7\ 10^{-7}$
May	Phenanthrene	0.001	$7.7\ 10^{-8}$
	Fluoranthene	0.001	$1.4\ 10^{-7}$
	Pyrene	0.001	$2.4\ 10^{-7}$
	Benzo(a)anthracene	0.1	$1.7\ 10^{-5}$
	Chrysene	0.01	$2.9\ 10^{-6}$
	Benzo(a)pyrene	1	$0.8\ 10^{-3}$
July	Pyrene	0.001	$7.7\ 10^{-8}$
	Benzo(a)anthracene	0.1	$8.8\ 10^{-6}$
	Chrysene	0.01	$1.9\ 10^{-6}$
	Benzo(a)pyrene	1	$0.2\ 10^{-3}$
Rebouças Tunnel (3)	Phenanthrene	0.001	$2.5\ 10^{-7}$
	Fluoranthene	0.001	$4.6\ 10^{-7}$
	Pyrene	0.001	$5.8\ 10^{-7}$
	Benzo(a)anthracene	0.1	$8.8\ 10^{-5}$
	Chrysene	0.01	$1.9\ 10^{-5}$
	Benzo(a)pyrene	1	$1.8\ 10^{-3}$

The detailed derivation of each potency equivalency factors (PEF) can be found in OEHHA (1994).

Risk assessment

Table 4 shows the risk assessment. In the estimate of individual risk of cancer, values on the order of 10^{-8} to 10^{-3} were detected at site 1 in April, May and July. At site 3, observed values were on the order of 10^{-7} to 10^{-3} for the PAHs listed in Table 4.

The evaluation of health effects and quantitative risk could be carried out for every PAH, where estimating the risk of cancer using PEFs is usually measured relative to benzo(a) pyrene, since it is the only PAH for which a complete quantitative risk assessment has been done (Collins et al., 1991; OEHHA, 1994). The use of PEFs for estimating risk from exposure to PAHs is an improvement for those PAHs for which there are reliable collection and measurement techniques. However, there are a large number of PAHs for which PEFs have not yet

been determined and/or for which measurement techniques are unavailable. PEFs are primarily based on chronic internal dosing experiments and skin-painting studies (Collins et al., 1998). Explanations of the derivation of each PEF, the type of data used for the derivation, and the relevant references were presented in a technical report that underwent public and scientific peer review (OEHHA, 1994); the derivations are detailed in that report (Collins et al., 1998).

In this study, the highest risks were detected for benzo(a)anthracene and for benzo(a)pyrene in May at site 1, and for benzo(a)anthracene, chrysene and benzo(a)pyrene at site 3. According to Collins et al. (1998), a risk of about 10^{-5} could lead to notification of the public. These data reflect the risk to which people using these routes are exposed daily. In general, bus drivers who work at sites 1 and 3 spend, respectively, 4 h and 10 min daily on these roads. Considering these data and the PAH concentrations detected, we can extrapolate how much these workers are at risk of PAH exposure. Traffic jams are frequent at site 3, lasting up to 2 h (http://www.jb.com.br/rio/noticias) and subjecting bus drivers to a risk of exposure of up to 0.69 ng/m^3 benzo(a)pyrene. This concentration is above the USEPA limit, and several studies have demonstrated an increase in DNA adducts at this level (Topinka et al., 1997; Lewtas et al., 1997; Kyrtopoulos et al., 2001).

In conclusion, nitro and amino derivatives of PAHs contributed to the mutagenicity detected for PM2.5. Furthermore, the population that uses routes 1 and 3, especially the bus drivers, are more exposed to cancer risk. This study reinforces the importance of using cleaner fuels and having better indoor ventilation. These measures could result in a reduction in diseases related to air pollution caused by PM2.5, and a consequent improvement in quality of life.

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