Health and carcinogenic risk evaluation for cohorts exposed to PAHs in petrochemical workplaces in Rawalpindi city (Pakistan)

Atif Kamal\textsuperscript{a}, Alessandra Cincinelli\textsuperscript{b}, Tania Martellini\textsuperscript{b}, Ilaria Palchetti\textsuperscript{b}, Francesca Bettazzi\textsuperscript{b} and Riffat Naseem Malik\textsuperscript{a*}

\textsuperscript{a}Environmental Biology and Ecotoxicology Laboratory, Department of Environmental Sciences, Faculty of Biological Sciences, Quaid-I-Azam University, Islamabad, Pakistan; \textsuperscript{b}Department of Chemistry, University of Florence, Florence, Italy

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This study presents the analyses of urinary biomarkers (1-OHPyr, α- and β-naphthols) of polycyclic aromatic hydrocarbons (PAHs) exposure and biomarkers of effect (i.e. blood parameters) in petroleum-refinery workers (RFs) and auto-repair workers (MCs). Exposed subjects had higher concentrations of white blood cell (WBC) count than control subjects (CN) subjects (5.31 \times 10^3 \mu L^{-1} in exposed vs. 5.15 \times 10^3 \mu L^{-1} in CN subjects), while the biomarker of oxidative DNA damage (8-OHdG) was significantly higher in MCs. The exposure among these two cohorts could be influenced by the ambience of the workplaces; in fact, MCs’ shops are relatively damp and enclosed workplaces in comparison with the indoor environment of refineries. PAHs in the dust samples from mechanical workshops probably originated from mixed sources (traffic exhaust and petroleum spills), while the incremental lifetime cancer risk (ILCR) for MCs showed moderate-to-low cancer risk from exposure to dust-bound PAHs. The study shows that increasing PAH exposure can be traced in MC workstations and needs to be investigated for the safety of public health.

\textbf{Keywords:} auto-mechanic shops; oxidative stress; Pakistan; petrol refinery; traffic pollution

Introduction

Polycyclic aromatic hydrocarbons (PAHs), which are well-known mutagens, teratogens (Kennish 1997), and endocrine disrupting chemicals (Augulyte et al. 2009), are primarily formed during the incomplete combustion of organic materials, and are also released during industrial activities. PAHs have also been detected at low levels in cigarette smoke motor vehicle emissions, and in some foods such as char-broiled meat, vegetables, fruit, and cereals. Occupational exposure to PAHs may also occur in workers breathing in exhaust fumes such as auto-repair mechanics, street vendors, motor vehicle drivers as well as those involved in mining, metal working, and oil refining. If exposed to PAHs, the harmful effects that may occur largely depend on the way people are exposed. Various studies on workers that breathed in or had a long-term contact with PAHs have suggested that PAHs may cause not only breathing problems, chest pain, irritation, and coughing lung but also kidney, pancreatic, prostate, or skin cancer.

According to the International Agency for Research on Cancer (IARC), some PAHs are carcinogenic to humans. In particular, occupational exposure to PAHs may occur in

*Corresponding author. Email: r_n_malik2000@yahoo.co.uk
workers continuously inhaling the fumes of used gasoline engine oil (UGEO) and volatile fraction (mist), such as auto-repair workers (Kamal et al. 2011). Grimmer et al. (1982) showed that carcinogenic contents are present in both used and virgin engine oils, and in fact, they evidenced that UGEO induces local tumor after prolonged application in mice, while PAHs containing more than three rings were responsible for more than 70% of carcinogenicity of UGEO. Long-term petroleum or crude oil exposure is also related to other human health effects in addition to tumors (Malins & Ostrander 1994), such as blood disorders (Yamato et al. 1996), reproductive problems (Eisler 1987), nephrotoxicity (Vyskocil & Cizkova 1996; Ezejiofor et al. 2014), reduced growth (Oliver et al. 1993), and morphological abnormalities (Kennish 1997).

In developing countries such as Pakistan, there is lack of awareness among workers regarding workplace chemicals and other related hazards. In fact, there is a large number of workers in Pakistan who are self-employed and work as auto-mechanics, and do not use personal-protective equipment to minimize the risk of skin cancer and respiratory tract diseases. Some of these workers deal with auto-spare parts; they work in unhygienic conditions, and they come into contact with UGEO on daily basis. This study aimed to conduct a comparative analysis of PAHs exposure among auto-repair station and petrol-refinery workers and to evaluate impact of occupational exposure to petrochemicals on selected biochemical parameters. Moreover, the carcinogenic risk from ingestion, inhalation, and ingestion of dust-bound PAHs was also evaluated.

Materials and methods

Study area and hot spots description

Rawalpindi is a rapidly growing city in the Pothohar region of northern Punjab (Pakistan), located only 14 km south from the capital city of Islamabad and 275 km to the North West of Lahore city. The growth rate of the urban areas of Rawalpindi city is 3.39% (Shabbir & Ahmad 2010), and due to the low literacy rate in the country and increasing unemployment, the trend of self-employment is often followed by young generation. There are several mismanaged and self-established workplaces (i.e. auto-mechanic and spare part shops) on main roads, amid narrow streets, and residential areas. These hot spots not only release large quantity of untreated petrochemical residues into the environment, but they also pose health risk to the surrounding population. These hot spots are mainly located in the Murree road and its surrounding areas in Rawalpindi; thus, exposed human subjects and workplace dust samples were respectively selected and collected in the area (see Plate S1-Supplementy material S1).

Selection of subjects and their self-certification of health status

Sampling was done at random between car mechanics (MCs, \( n = 25 \)), including diesel engine mechanics, motor-truck mechanics, engine-repair mechanics, differential and brakes repairers, general automobile service-station mechanics and refinery workers (RFs, \( n = 30 \)) located in the district Rawalpindi. Participants for an age-matched non-occupationally exposed group (CNs, \( n = 34 \)) were selected from non-chemical-related occupations of the same city.

All participants filled a short questionnaire to provide confidential information on their occupation. The information included the work/exposure hour/day (h/d), work
experience, and types of chemicals used in the workplace. Moreover, the health and socio-demographic status, such as age, height, body mass index (BMI), education, history of known medical problems, and lifestyle factors, including frequency and amount of smoking were also documented. The inclusion criteria for workers were that the subject should be in good health, non-smoker, do not take any kind of medication, work or be exposed 6 h/d and six d/week, and have a working experience of ≥5 years. Same criteria (except that they were non-occupationally exposed) were used for the recruitment of the CN group. All study subjects were recruited with informed written consent under an approved protocol from the ethical review committee of the Quaid-I-Azam University Islamabad (Pakistan). The subjects, who were former smoker or recently recruited staff/worker, those on some kind of medication, and having the extreme body mass index (BMI < 17 and > 30 kg/m²), were excluded from the analyses.

The subjects were also asked for the prevalence of any respiratory symptoms. A respiratory disturbance was characterized by having one or more of the following symptoms: coughing, sneezing, fatigue, upper respiratory congestion, and rhinitis, while headache symptoms included dizziness, fatigue, and headache. Subjects experiencing these symptoms only once in a month were ranked as no or low, with more than once in two weeks were ranked as medium, while with more than one symptom in a week were ranked as high. Similarly, headache and other work-related health symptoms (if any) reported by subjects were also documented.

**Reagents**

All solvents were pesticide grade and purchased by Supelco (Bellefonte, PA, USA) and tested for contaminants before use. Standard PAHs mixture (EPA) was commercially purchased from Supelco (Bellefonte, PA, USA). Benzo(e)pyrene and coronene were purchased from Alltech (Deerfield, USA). Silica (100–200 mesh) and sodium sulfate (Na₂SO₄) were purchased from Merck (Darmstadt, Germany). Sodium sulfate was heated for 12 h at 450 °C to remove any organic matter and kept at 120 °C until use.

**Blood sampling**

Post-shift blood samples of workers and non-occupationally exposed subjects were collected on the same day during sampling. From each subject, 3–4 ml blood was collected in disposable vacutainers tubes (without anticoagulant for serum and with EDTA for whole blood). The blood samples were always withdrawn by a trained technician. The blood specimen was immediately shipped to the analytical laboratory and were kept refrigerated until analysis, except for the analysis of hematologic parameters and superoxide dismutase (SOD) activity, which was performed the same day. The gel-vacutainers were centrifuged for 20 min to separate serum from blood cells within one hour after blood collection. The separated serum samples were transferred to Eppendorf-tubes with an identification code and kept refrigerated. All biological samples were sent to an accredited bio-medical chemistry laboratory for selected biochemical analysis after proper labeling/tagging each sample with accurate information.

**Urine sample collection and analysis**

Post-shift urine samples (100–200 mL) were collected during blood sampling from each participant in polyethylene screw-cap bottles. The urine samples were shipped in dry ice
to the laboratory and were stored at \(-25^\circ C\). The monohydroxy-PAHs analyzed included the 1-OHPyr (1-pyrenol), \(\alpha\)-naphthol (\(\alpha\)-naphthol or 1-hydroxynaphthalene), and \(\beta\)-naphthol (\(\beta\)-naphthol or 2-hydroxynaphthalene). Sample preparation and analysis of urinary PAHs metabolites were performed according to the methodologies reported previously (Xu et al. 2004; Elovaara et al. 2006), and the details of analytical and instrumental conditions have been described previously (Kamal et al. 2014; also see Supplementary material S1-1.1). The results of urinary OH-PAH biomarkers were adjusted with urinary creatinine (measured using conventional Jaffe’s colorimetric method) and were expressed in \(\mu\text{mol/mol-creatinine (mol-Cr}^{-1})\).

**Analysis of hematological parameters and SOD**

Fifty milliliter of each whole blood sample was analyzed using an automated hematology analyzer Sysmec (KX-21) to measure blood parameters, including hemoglobin (Hb g dL\(^{-1}\)), platelet counts (PLTs \(\times 10^3\) \(\mu\)L\(^{-1}\)), red blood cells (RBCs \(\times 10^6\) \(\mu\)L\(^{-1}\)), white blood cells (WBCs \(\times 10^3\) \(\mu\)L\(^{-1}\)), and mean corpuscle volume (MCV, fL). The serum c-reactive proteins CRP levels were measured on a Roche/Hitachi MODULAR automated analyzer (Roche Diagnostics). The SOD in erythrocytes was determined using the method described previously (Marklund & Marklund 1974; Winterbourn et al. 1975) consisting in using pyrogallol as a substrate and reading the absorbance at 420 nm (see Supplementary material S1-1.2).

**Urinary 8-OHdG assay**

Before the analyses of 8-OHdG, the thawed urine samples were centrifuged at 3000 g for 5 min to collect the supernatant, which was further diluted with the diluents provided with the kit and vortexed to mix well. The solution was re-centrifuged, to collect supernatant which was further used for the quantification of 8-OHdG, according to the instruction provided by manufacturer’s of competitive enzyme-linked immunosorbent assay kit (ELISA; Cell Bio-labs Inc. San Diego CA). A logarithmic standard curve was used to interpolate the results; the results were further adjusted with urinary creatinine values and presented as 8-OHdG ng mg-Cr\(^{-1}\).

**Dust sample collection, preparation, extraction, cleanup, and analysis**

A total of 19 soil/dust samples were collected from automobile repair stations/small shops located on the main roads and slum areas of Rawalpindi city, using dustpans and plastic brushes (new pair for each site) by gentle sweeping motion to collect fine particulates. Collection of pure dust was often not possible, and instead, the samples comprised dust particles with small factions of soil in most of the cases. All the shops falling within an area of 5 m were considered a single sampling site, and soil/dust samples from about five different work areas were combined into one composite sample. Soil/dust samples were collected in refinery (\(n = 17\)) belonged to seven different location of same refinery; each sample was a composite of three soil/dust samples from the same site collected in three consecutive days. In order to protect all samples from sunlight exposure, aluminum foil was used to wrap each soil/dust sample separately. After that, the samples were sealed in zip-locked polyethylene bags and stored until analysis.

The details of sample preparation and instrumental analysis have been described in detail previously (Martellini et al. 2012; Kamal et al. 2014). All the prepared samples were analyzed using a Hewlett-Packard 6890 gas chromatography-mass spectrometer.
(GC-MS), equipped with a 5973 MSD and a HP-5MS capillary column (J&W Scientific, Folsom, CA, USA, 30 m, 0.25 mm I.D., 0.25 mm film thickness). Compound identification was based on the MSD database (NIST, 98) and GC retaining time of PAHs standards. The MSD was operated in selected ion monitoring mode (SIM).

Quality control and quality assurance
Average PAHs recoveries and relative standard deviations were measured to evaluate the method performance by multiple analyses of clean sand samples spiked with PAHs standard mixture containing 18 PAHs (acenaphthene (Ace), acenaphthylene (Acy), anthracene (Ant), benzo(a)anthracene (BaA), benzo(a)pyrene (BaP), benzo(b)fluoranthene (BbF), benzo(e)pyrene (BeP), benzo(gi)perylene (BghiP), benzo(k)-fluoranthene (BkF), chrysene (Chry), coronene (Cor), dibenzo(a,h)anthracene (DBA), fluoranthene (Fla), fluorene (Fl), indeno(1,2,3-cd)pyrene (IP), naphthalene (Naph), phenanthrene (Phe), and pyrene (Pyr)). BbF and BkF were reported together as BbkF. In order to determine any potential laboratory contamination, the procedural blanks were evaluated and periodically performed. The recovery percentage of 18 PAHs ranged between 84.1 and 122.4 %, while that of surrogate PAHs was 93 ± 13 % for benzo(a)pyrene-d12, 97 ± 12 % for fluoranthene-d10, and 94 ± 11 % for p-therpenyl-d4. We also used SRM1649a (urban dust) as a control. The diluted standards used were between the range of 0.16 and 10 ng μL−1, and a six point calibration curve was drawn for quantification of PAHs in the soil/dust samples. Detection limits of PAHs were in the range of 20–60 pg g−1 (dry weight basis).

ILCR assessment and model and parameters
The incremental lifetime cancer risk (ILCR), which represents the estimated increase in the lifetime cancer risk due to exposure to a particular carcinogen over 70 years of life time (Meiners & Yandle 1995), was calculated using the following equations for ingestion, dermal contact, and inhalation pathways,

\[
\text{ILCR (Ingestion)} = \frac{\sum \text{TEQ} \times \{ \text{CSF (ingestion)} \times (\text{BW/70})^{1/3} \} \times \text{ED} \times \text{EF} \times \text{IngR}} {\text{BW} \times \text{AT} \times \text{CF}}
\]

\[
\text{ILCR (Inhalation)} = \frac{\sum \text{TEQ} \times \{ \text{CSF (inhalation)} \times (\text{BW/70})^{1/3} \} \times \text{ED} \times \text{EF} \times \text{InhR}} {\text{BW} \times \text{AT} \times \text{PEF}}
\]

\[
\text{ILCR (Dermal contact)} = \frac{\sum \text{TEQ} \times \{ \text{CSF (dermal contact)} \times (\text{BW/70})^{1/3} \} \times \text{ABS} \times \text{AF} \times \text{SA} \times \text{ED} \times \text{EF}} {\text{BW} \times \text{AT} \times \text{CF}}
\]

where \(\Sigma \text{TEQ}\) = sum of toxic equivalent concentrations, that is, the sum of BaP equivalent concentrations for each PAH calculated by multiplying the individual PAH concentration by its corresponding TEF value (Nisbet & LaGoy 1992; Fang et al. 2006; Cincinelli et al. 2007); IngR = ingestion rate; CSF = carcinogenic slope factor, which was based on the cancer-causing ability of benzo(a)pyrene; EF = outdoor exposure frequency; ED = outdoor exposure duration; BW = body weight (kg); AT = average life time; CF = conversion factor; PEF = particle emission factor for BaP; AT (h) = average life time; SA = workers exposed skin area; AF (soil) = dermal adherence factors; ABS = fraction of contaminant absorbed dermally from soil (unitless and specific to contaminant).
Statistical analyses

Data distribution was tested using Kolmogorov–Smirnov (K–S) test. The comparative analysis of urinary PAHs biomarkers (being non-normally distributed) was performed using the Mann–Whitney’s U-test, whereas, the independent sample t-test was used for comparative analysis of demographic parameters including age, BMI weight, height, and daily work hour, the data are presented in median, mean ± standard deviation, and minimum and maximum values, while the categorical/nominal data are tabulated as frequencies (percentage). The comparison of nominal/ordinal parameters was performed using Chi-square analysis. All other statistical analyses were performed using SPSS 20 and Ms-Excel 2010. Urine dilution was corrected by adjusting urinary biomarkers with urinary creatinine, and values were expressed in μmol/mol-creatinine (μmol mol-Cr⁻¹).

Results and discussion

The occupationally exposed and non-exposed groups recruited in this study were age matched, with almost similar work experience and BMI. Table 1 summarizes the socio-demographic information of the subjects, with the comparative analyses revealing long working hours and low life status of MC subjects as compared to CN (p < 0.05).

However, the RFs workers had the highest educational background as compared to CNs and MCs (who are self-employed), showing that in the refinery sector, generally technical staff with sound educational background is recruited. On the contrary, MCs do not have a technical education in their work sector, because they have acquired their expertise working as apprentices since their childhood.

Environmental pollution is considered one of main cause of muscular problems and headache symptoms (Pick et al. 2002), and there are some additives in the gasoline which are known to cause neuropathies and psychosis (Epstein & Selber 2002). Taking into account these aspects, the self-reported health symptoms, including headache and respiratory disturbances, were examined in connection with exposure to petroleum mist in the work environment. Naphthalene, which is present at high concentrations in the petroleum-contaminated workplaces (Kamal et al. 2011, 2012; Klasing & Brodberg 2013), causes numerous respiratory effects, especially damages in ciliated and Clara cells of the bronchiolar epithelium have been reported in the mouse model (OEHH 2001). Headache symptoms were more experienced by RFs and MCs than CN subjects, while a more positive response for respiratory effects was reported by RFs.

A comparative overview of biomarkers of exposure and effect

In this study, we analyzed biomarkers of PAHs exposure and effect to evaluate exposure outcomes and observed different results in biochemical parameters of the subjects compared with results obtained in a previous investigation on exposure of workers to pyrogenic sources of PAHs (Kamal et al. 2014). In particular, most of the comparison between CN and exposure groups was non-significant. It was generally observed that the DNA damage (as indicated by urinary 8-OHdG concentration) was quite high in both groups exposed to petroleum products/fumes in occupational environments. Moreover, MCV increased more in MCs than RFs (94.6 fL than 92.5 fL, respectively). Similarly, packed cell volume (PCV) was also higher in MCs than RFs (47.8 % in MCs and 45.4 % in RFs). Overall hematocrit of exposed subjects was higher than CNs (46.5 % vs. 45 % respectively, p < 0.05). Both these symptoms indicated significant
| Variables               | CN (34) | RFs (30) | MCs (25) | All exposed (55) | \( P \text{-value}^a \) | \( P \text{-value}^b \) | \( P \text{-value}^c \) | \( P \text{-value}^d \) |
|------------------------|---------|----------|----------|------------------|----------------|----------------|----------------|----------------|
| Work experience (in years) | 12.5   | 10.74    | 10.8     | 10.7             | 0.17\( f \) | 0.21\( f \) | 0.98          | 0.11\( f \) |
|                        | 14.2 ± 5.9 | 12 ± 5.8 | 12 ± 5.9 | 12 ± 5.8         | 0.17\( f \) | 0.21\( f \) | 0.98          | 0.11\( f \) |
| Daily working hours | 8.5    | 8        | 8.8      | 8.86             | 0.28\( f \) | <0.001\( f \) | <0.001        | <0.01\( f \) |
|                        | 7.8 ± 1 | 8 ± 1    | 9.8 ± 1  | 8.8 ± 1.5        | 0.28\( f \) | <0.001\( f \) | <0.001        | <0.01\( f \) |
| Age (years)             | 36.7   | 38.7     | 36.7     | 37.8             | 0.87\( f \) | 0.32\( f \) | 0.27          | 0.68\( f \) |
|                        | 37 ± 6.7 | 39.8 ± 9.7 | 38 ± 6.7 | 39 ± 8.5         | 0.87\( f \) | 0.32\( f \) | 0.27          | 0.68\( f \) |
| BMI (kg/m²)             | 21.4   | 20.98    | 21.4     | 21.2             | 0.55\( e \) | 0.18\( e \) | 0.69          | 0.33\( e \) |
|                        | 20.8 ± 1.8 | 21.2 ± 3.2 | 21.5 ± 2 | 21 ± 2.7         | 0.55\( e \) | 0.18\( e \) | 0.69          | 0.33\( e \) |
| Height (Feet)           | 5.64   | 5.61     | 5.6      | 5.6              | 0.54\( f \) | 0.27\( f \) | 0.29          | 0.47\( f \) |
|                        | 5.65 ± 0.32 | 5.61 ± 0.16 | 5.61 ± 0.16 | 5.58 ± 0.5       | 0.54\( f \) | 0.27\( f \) | 0.29          | 0.47\( f \) |
| Weight (kg)             | 60     | 56.1     | 57.6     | 60.7             | 0.13\( f \) | 0.10\( f \) | 0.16          | 0.74\( f \) |
|                        | 61 ± 8 | 64 ± 11  | 57.8 ± 4.8 | 61.3 ± 9         | 0.13\( f \) | 0.10\( f \) | 0.16          | 0.74\( f \) |
| Life status             | Low    | 6(17.64) | 0(0)     | 11(44)           | 0.013\( h \) |<0.01\( h \) |<0.001        |<0.001\( h \) |
|                        | Medium | 21(61.76) | 10(33.3) | 14(56)           | 24(43.63) | 24(43.63) | 24(43.63)     | 24(43.63)     |
|                        | High   | 7(20.58) | 20(66.66) | 0(0)             | 20(63.36) | 20(63.36) | 20(63.36)     | 20(63.36)     |

(Continued)
| Variables                      | CN (34) | RFs (30) | MCs (25) | All exposed (55) | P-value<sup>a</sup> | P-value<sup>b</sup> | P-value<sup>c</sup> | P-value<sup>d</sup> |
|-------------------------------|---------|----------|----------|------------------|----------------------|----------------------|----------------------|----------------------|
| Headache<sup>g</sup>          |         |          |          |                  | <0.01<sup>h</sup>    | 0.004<sup>h</sup>    | <0.001               | <0.01<sup>h</sup>    |
| No                            | 16(47.05) | 0(0)     | 2(8)     | 2(3.63)          |                      |                      |                      |                      |
| Sometimes                     | 10(29.41)| 6(20)    | 16(64)   | 22(40)           |                      |                      |                      |                      |
| Often                         | 8(23.52) | 24(80)   | 7(28)    | 31(56.36)        |                      |                      |                      |                      |
| Respiratory problems<sup>g</sup> |         |          |          |                  | <0.01<sup>h</sup>    | 0.19<sup>h</sup>     | 0.29                 | 0.29                 |
| No                            | 5(14.7)  | 6(20)    | 7(28)    | 13(23.63)        |                      |                      |                      |                      |
| Sometimes                     | 15(44.12)| 22(73.33)| 13(52)   | 35(63.63)        |                      |                      |                      |                      |
| Often                         | 14(41.17)| 2(6.66)  | 5(20)    | 7(12.72)         |                      |                      |                      |                      |
| Education                     |         |          |          |                  | <0.01<sup>h</sup>    | <0.01<sup>h</sup>    | <0.001               | <0.001<sup>h</sup>   |
| None                          | 0(0)    | 0(0)     | 0(0)     | 0(0)             |                      |                      |                      |                      |
| Under-Primary                 | 4(11.76)| 0(0)     | 8(32)    | 8(14.54)         |                      |                      |                      |                      |
| Primary                       | 13(38.23)| 0(0)     | 17(68)   | 17(30.9)         |                      |                      |                      |                      |
| ≥Secondary                    | 17(50)  | 30(100)  | 0(0)     | 30(54.54)        |                      |                      |                      |                      |

Note: Edu = education level; BMI = body mass index; P = probability value; CN = control group; RFs = refinery workers; MCs = car mechanics.

<sup>a</sup>CN vs. RFs.

<sup>b</sup>CN vs. MCs.

<sup>c</sup>RFs vs. MCs.

<sup>d</sup>CN vs. all exposed (MCs and RFs).

<sup>e</sup>Median/mean ± std. dev/min-max.

<sup>f</sup>Comparative analysis of parametric variables using independent sample t-test.

<sup>g</sup>Comparative analyses of percentage using Chi-square test ($\chi^2$).
changes and implication of blood disorders. We did not observe any significant change in PLT and CRP concentrations of either exposure groups compared with CNs; however, the exposed group had higher concentration of WBC count than CN subjects ($5.3 \times 10^3 \mu\text{L}^{-1}$ and $5.15 \times 10^3 \mu\text{L}^{-1}$ in all exposed and CN subjects, respectively), and the MCs had significantly high concentration of WBC than CN ($5.42 \times 10^3 \mu\text{L}^{-1}$ vs. $5.15 \times 10^3 \mu\text{L}^{-1}$; $p < 0.05$).

The exposure of RFs to PAHs is rarely reported in the past literature. Even if it is expected that new oil should contain lower content of PAHs, huge exposure to volatile organic compounds (evidenced from high petroleum odor in workplaces) is expected in refineries. The petrogenic sources are known to emit lower molecular weight (MW) PAHs like naphthalene (Klasing & Brodberg 2013), as well as refined petroleum products (Granella & Clonfero 1991). Therefore, α- and β-naphthols, which are representative of volatile and gas phase (inhalable) PAHs at low levels (Kim et al. 1999), were also analyzed. Moreover, the combustion of fuel, vehicular emission, and oil refining are all sources of PAHs emissions (Baek 1991). The PAHs which are semi-volatile and have low MW (< 206), such as pyrene, can emerge from incomplete combustion of organic materials and also from petrogenic sources, including evaporation of petroleum products and leakages of such oil (Zhang & Tao 2009; Ma et al. 2010). The results provide an indication that MCs were more exposed to PAHs than RFs. This was also evidenced from the higher concentration of urinary 1-OHPyr (median 1.02 μmol mol-Cr$^{-1}$) in the exposed group than in CN subjects (0.62 μmol mol-Cr$^{-1}$); 1-OHPyr was also high in RFs, but the difference vs. MCs was non-significant. The difference of exposure among these two cohorts seems to be influenced by the ambience of the workplace; in fact, it was noticed that MC-workshops are relatively damp and enclosed places as compared to the environment of refineries, where the workers have to perform the routine tasks in an indoor environment for most of the time. MCs come in contact with body parts of automobile engines and handling tools, and they smear most of the body parts with petrochemical fractions. Ventilation in the workplace is also a key factor that influences the indoor concentration significantly (Kamal et al. 2011). 1-OHPyr is a very useful biomarker of exposure to air born mixture of PAHs and has been used in numerous biomonitoring studies in the past. The survey of such workplaces has shown that the MCs work in a highly hazardous situation, that is, they do not use self-protective equipment and their hands are routinely smeared with UGEO, which facilitates cutaneous absorption of PAHs. A previous study from same country has reported that MCs are exposed to volatile PAHs in their workplace and the main exposure among MCs occurs through UGEO on daily basis (Kamal et al. 2011; Kamal & Malik, 2012). In both exposed groups, we did not observe much effect of occupational exposure on blood parameters and any sound association of any blood parameter and PAHs biomarkers. Despite the PCV (47.8 %), RBC ($5.42 \times 10^6 \mu\text{L}^{-1}$) and WBC counts ($5.4 \times 10^3 \mu\text{L}^{-1}$) were exclusively higher in MCs than CN subjects (Table 2). In general, the exposed subjects had higher level of 8-OHdG; in particular, it was significantly higher in MCs (48 ng mg-Cr$^{-1}$) than CNs (25.8 ng mg-Cr$^{-1}$) ($p < 0.001$). All other parameters were not so much different among groups, and despite relatively increased blood parameters in MCs, the values were within the normal ranges. Since the risk of exposure was also high among MCs, they work without self-protective equipment (Kamal et al. 2012), and therefore, they are highly exposed to genotoxins present UGEO, including petroleum residues and carcinogenic PAHs which are accumulated in the lubricating oil over time.
Table 2. Comparative analyses of biochemical parameters, among exposed (MCs and RFs) and control (CN) groups.

| Variables | CN group (34) | MCs (25) | RFs (30) | All exposed (55) | $P$-value$^a$ | $P$-value$^b$ | $P$-value$^c$ | $P$-value$^d$ |
|-----------|---------------|----------|----------|------------------|--------------|--------------|--------------|--------------|
| $Hb^e$ (g dL$^{-1}$) | 15.1 | 15.1 ± 0.3 | 15.2 | 15.1 | 0.18 | 0.49 | 0.07 | 0.64 |
| | 14.1–15.1 | 14.18 ± 0.47 | 14–15.2 | 13.8–15.98 | | | | |
| $MCV^e$ (fL) | 92 | 91.6 ± 4.5 | 91 | 93 | 0.49 | 0.08 | 0.15 | 0.4 |
| | 85–100 | 92.5 ± 6.21 | 86–109 | 93.5 ± 5.5 | | | | |
| $PCV^e$ (%) | 44.7 | 45 ± 2.1 | 45.1 | 46.6 | <0.001 | 0.59 | 0.001 | 0.01 |
| | 41.5–50.7 | 45.4 ± 2.4 | 46.5 ± 2.8 | | | | | |
| $PLTe^e$ ($\times 10^3$ µL$^{-1}$) | 211.5 | 218.9 ± 23.6 | 216 | 0.7 | 0.69 | 0.54 | 0.99 |
| | 183–276 | 229.8 ± 46 | 223.4 ± 39 | | | | | |
| $RBC^e$ ($\times 10^6$ µL$^{-1}$) | 5.1 | 5.09 ± 0.28 | 5.01 | 5.2 | 0.003 | 0.61 | 0.02 | 0.21 |
| | 4.1–5.6 | 5.1 ± 0.55 | 95.2 ± 0.56 | | | | | |
| $WBC^e$ ($\times 10^3$ µL$^{-1}$) | 5.1 | 5.15 ± 0.61 | 5.3 | 5.4 | 0.04 | 0.13 | 0.53 | 0.31 |
| | 4.1–6.6 | 5.26 ± 0.89 | 5.3 ± 0.79 | | | | | |
| $CRPe^e$ (mg L$^{-1}$) | 0.54 | 0.65 ± 0.39 | 0.64 | 0.64 | 0.08 | 0.12 | 0.73 | 0.056 |
| | 0.1–1.4 | 0.82 ± 0.38 | 0.8 ± 0.33 | | | | | |
| $1$-OHPyr$^f$ (µmol mol-Cr$^{-1}$) | 0.62 | 0.69 ± 0.22 | 0.78 | 0.81 | 0.048 | 0.57 | 0.18 | 0.11 |
| | 1.02 | 0.96 ± 0.42 | 0.87 ± 0.39 | | | | | |

(Continued)
Table 2.  (Continued).

| Variables | CN group (34) | MCs (25) | RFs (30) | All exposed (55) | P-value<sup>a</sup> | P-value<sup>b</sup> | P-value<sup>c</sup> | P-value<sup>d</sup> |
|-----------|---------------|----------|----------|------------------|-----------------|----------------|----------------|----------------|
| α-naphtholf (µmol mol-Cr<sup>-1</sup>) | 0.64 ± 0.18 | 1.69 ± 0.19 | 1.55 ± 1.2 | 1.6 ± 0.68 | <0.001 | <0.001 | 0.37 | <0.001 |
| β-naphtholf (µmol mol-Cr<sup>-1</sup>) | 0.61 ± 0.16 | 5.48 ± 2.16 | 4 ± 1.57 | 4.97 ± 3.2 | <0.001 | <0.001 | 0.29 | <0.001 |
| 8-OHdG<sup>e</sup> (ng mg-Cr<sup>-1</sup>) | 25.8 ± 7 | 48 ± 15 | 37 ± 13 | 45.8 ± 14 | <0.001 | <0.01 | 0.06 | <0.01 |
| SOD<sup>e</sup> (U/g Hb) | 1005.8 | 1036 | 1134.7 | 1121.2 | 0.15 | 0.057 | 0.67 | 0.06 |

Notes: 1-OHPyr = 1-hydroxypyrine; 8-OHdG = 8-hydroxydeoxyguanosine; CRP = c-reactive proteins; Hb = hemoglobin; MCV = mean cell volume; PCV = packed cell volume (hematocrit); PLT = platelet count; RBC = red blood cells; SOD = superoxide dismutase; WBC = white blood cells.

<sup>a</sup>CN vs. MCs.
<sup>b</sup>CN vs. RFs.
<sup>c</sup>RF vs. MC.
<sup>d</sup>CN vs. all exposed (combined RFs and MCs) Each set of values are represents the median/mean ± std. dev/min–max.
<sup>e</sup>Independent sample t-test.
<sup>f</sup>Mann–Whitney’s U-test.
| PAH congeners (ring #) abbreviations | Exposure sites | MC-dust samples (19) | RF-dust samples (17) | \( P^a \) |
|-------------------------------------|---------------|----------------------|---------------------|--------|
|                                     | TEFs*         | Mean ± Std. dev. Med | Min–Max             | Mean ± Std. dev. Med | Min–Max | P^a |
| Naphthalene (3) (Naph)              | 0.001         | 134 ± 22             | 29–221              | 115 ± 59             | 27–230   | 0.5 |
|                                     |               | 125                  |                     | 111                 |         |     |
| Acenaphthene (3) (Ace)              | 0.001         | 35 ± 7               | 11–83.8             | 47 ± 6               | 13–89    | 0.44|
|                                     |               | 37                   |                     | 45                  |         |     |
| Acenaphthylene (3) (Acy)            | 0.001         | 113 ± 27             | 33.6–217            | 96 ± 23              | 54–190   | 0.58|
|                                     |               | 115                  |                     | 87                  |         |     |
| Anthracene (3) (Ant)                | 0.01          | 42.2 ± 8             | 13.8–136            | 24 ± 4               | 12–48    | 0.24|
|                                     |               | 43.1                 |                     | 22                  |         |     |
| Fluorene (3) (Fl)                   | 0.001         | 36.7 ± 9             | 22.5–53             | 33 ± 9               | 15.5–69  | 0.6 |
|                                     |               | 38.6                 |                     | 32                  |         |     |
| Phenanthrene (3) (Phe)              | 0.001         | 75 ± 8               | 14.1–157            | 119 ± 34             | 48–144   | 0.06|
|                                     |               | 70                   |                     | 114.9               |         |     |
| Benzo(a)anthracene (4) (BaA)        | 0.1           | 24 ± 8               | 13.9–37             | 18 ± 5               | 9.9–25   | 0.17|
|                                     |               | 19                   |                     | 18                  |         |     |
| Chrysene (4) (Chry)                 | 0.01          | 137 ± 19             | 85–190              | 108 ± 23             | 72–137   | 0.10|
|                                     |               | 129                  |                     | 99.5                |         |     |
| Fluoranthene (4) (Fla)              | 0.001         | 54 ± 5               | 33–106              | 43 ± 6               | 27.8–67  | 0.33|
|                                     |               | 42.5                 |                     | 38                  |         |     |
| Pyrene (4) (Pyr)                    | 0.001         | 84 ± 7               | 61–119              | 88 ± 8               | 63–117   | 0.7 |
|                                     |               | 86                   |                     | 88                  |         |     |
| Benzo(a)pyrene (5) (BaP)            | 1             | 47 ± 11              | 17–85               | 19 ± 3               | 9–31     | <0.01|
|                                     |               | 45                   |                     | 18                  |         |     |
| Benzo(b + k)fluoranthene (5) (BbkF) | 0.1           | 35 ± 7               | 10–98               | 20.9 ± 6             | 10–46    | 0.24|
|                                     |               | 29                   |                     | 18                  |         |     |
| Dibenzo(a,h)anthracene (5) (DBA)    | 1             | 28 ± 7               | 3.5–75              | 10 ± 2               | 2.3–18.6 | 0.09|
|                                     |               | 27                   |                     | 12                  |         |     |

(Continued)
| PAH congeners (ring #) abbreviations | TEFs* | Mean ± Std. dev. (MC-dust samples (19)) | RF-dust samples (17) | P* |
|-------------------------------------|-------|---------------------------------------|---------------------|-----|
|                                     |       | Min–Max                               | Min–Max             |     |
|                                      |       | Med                                   | Std. dev.           |     |
| Benzo(c)pyrene (BeP) (5)            | 0.01  | 39 ± 6                                | 9.7–101             | 19 ± 9 | 7–29 | 0.15 |
|                                     |       | 25                                    |                     | 21   |      |
| Indeno(1,2,3,c,d)pyrene (IP) (6)    | 0.1   | 17 ± 4                                | 10.8–28             | 14 ± 6 | 5.7–22 | 0.36 |
|                                     |       | 13                                    |                     | 15   |      |
| Benzo (g,h,i)perylene (BghiP) (6)   | 0.01  | 85 ± 8                                | 64–121              | 75 ± 7 | 41–117 | 0.44 |
|                                     |       | 85                                    |                     | 75   |      |
| Coronene (Cor) (7)                  | 0.001 | 28.6 ± 11                             | 10.6–57             | 34 ± 9 | 9.4–53 | 0.53 |
|                                     |       | 26                                    |                     | 40   |      |
| ∑PAHs                               | –     | 1014 ± 55                             | 690–1496            | 883 ± 60 | 692–1004 | 0.22 |
|                                     |       | 912                                   |                     | 916  |      |
| ∑COMB                               | –     | 522 ± 56                              | 387–801             | 405 ± 81 | 263–510 | 0.09 |
|                                     |       | 473                                   |                     | 408  |      |
| ∑7-Carcinogens                      | –     | 292 ± 82                              | 206–421             | 235 ± 57 | 149–338 | 0.14 |
|                                     |       | 255                                   |                     | 234  |      |
| ∑HMPAHs                             | –     | 550 ± 75                              | 390–876             | 368 ± 62 | 269–460 | 0.13 |
|                                     |       | 502                                   |                     | 350  |      |
| ∑LMPAHs                             | –     | 436 ± 44                              | 250–597             | 433 ± 24 | 403–459 | 0.95 |
|                                     |       | 465                                   |                     | 433  |      |
| ∑456-rings PAHs/∑PAHs               | –     | 0.54 ± 0.07                           | 0.45–0.63           | 0.46 ± 0.1 | 0.39–0.52 | 0.03 |
|                                     |       | 0.52                                  |                     | 0.47  |      |
| ∑LMPAHs/∑HMPAHs                     | –     | 0.83 ± 0.26                           | 0.5–1.19            | 1.2 ± 0.16 | 1–1.5 | <0.01 |
|                                     |       | 0.84                                  |                     | 1.2   |      |
| Ant/(Phe + Ant)                     | –     | 0.38 ± 0.2                            | 0.1–0.7             | 0.17 ± 0.09 | 0.08–0.28 | 0.02 |
|                                     |       | 0.31                                  |                     | 0.13  |      |
| BaA/(BaA + Chry)                    | –     | 0.15 ± 0.04                           | 0.1–0.2             | 0.14 ± 0.09 | 0.11–0.2 | 0.81 |
|                                     |       | 0.14                                  |                     | 0.13  |      |

(Continued)
| Exposure sites | MC-dust samples (19) | RF-dust samples (17) |
|---------------|----------------------|----------------------|
|               | TEFs*    | Mean ± Std. dev. | Min–Max | Mean ± Std. dev. | Min–Max | P* |
|               | Med      | Min–Max          | Med      | Min–Max          |         |
| BaP/BghiP     | –        | 0.55 ± 0.2       | 0.2–0.88 | 0.26 ± 0.1       | 0.20–0.34 | <0.01 |
|               |          | 0.52             |          | 0.25             |         |
| BeP/(BeP + BaP) | –      | 0.41 ± 0.2       | 0.14–0.67 | 0.49 ± 0.09       | 0.32–0.59 | 0.24 |
|               |          | 0.42             |          | 0.53             |         |
| Fl/(Pyr + Fl) | –        | 0.31 ± 0.1       | 0.22–0.38 | 0.26–0.09       | 0.17–0.37 | 0.21 |
|               |          | 0.30             |          | 0.21             |         |
| Fla/(Pyr + Fla) | –    | 0.38 ± 0.1       | 0.28–0.47 | 0.32 ± 0.08       | 0.20–0.42 | 0.18 |
|               |          | 0.38             |          | 0.31             |         |
| IP/(IP + BghiP) | –     | 0.16 ± 0.07     | 0.13–0.19 | 0.16 ± 0.03       | 0.12–0.19 | 0.78 |
|               |          | 0.16             |          | 0.17             |         |
| Phe/(Phe + Ant) | –    | 0.6 ± 0.2        | 0.28–0.88 | 0.8 ± 0.09       | 0.72–0.92 | 0.02 |
|               |          | 0.69             |          | 0.87             |         |

Notes: ΣPAHs = sum of non-alkylated total PAHs. ΣCOMB = PAHs originating from the combustion process, that is, BaA, BaP, BbkF, BeP, BghiP, Chry, Fla, IP, Pyr (Prahl & Carpenter 1983). Σ7-Carcinogenic PAHs = BaA, BaP, BbkF, BghiP, IP, Pyr. LMPAHs = low molecular weight PAHs; HMPAHs = high Molecular weight PAHs.

*TEF for individual PAHs relative to BaP as reported by Nisbet and LaGoy (1992), except TEF values for Cor and BeP which were adopted from Malcom and Dobson (1994).

*P = probability value, significant at α < 0.05.
Profile and source apportionment of dust-bound PAHs

The profile of dust-bound PAHs of all samples from both exposure sites is reported in Table 3. PAH congeners were present in relatively low concentrations in RF workplaces in comparison to MCs. The order of individual PAH congeners from highest to lowest concentration in MC workplaces is the following: Chry (137 ng g$^{-1}$d.w.) > Naph (134 ng g$^{-1}$d.w.) > Acy (113 ng g$^{-1}$d.w.) > (BghiP (85 ng g$^{-1}$d.w.) > Pyr (84 ng g$^{-1}$d.w.) > Phe (75 ng g$^{-1}$d.w.) > Fla (54 ng g$^{-1}$d.w.) > Ant (42.2 ng g$^{-1}$d.w.) > Ace (35 ng g$^{-1}$d.w.) > BbkF (35 ng g$^{-1}$d.w.) > Cor (28.6 ng g$^{-1}$d.w.) > DBA (28 ng g$^{-1}$d.w.) > BaA (24 ng g$^{-1}$d.w.). Likewise, individual PAHs were found in the following order in RF soil/dust samples: Phe (119 ng g$^{-1}$d.w.) > Naph (115 ng g$^{-1}$d.w.) > Chry (108 ng g$^{-1}$d.w.) > Acy (96 ng g$^{-1}$d.w.) > Pyr (88 ng g$^{-1}$d.w.) > BghiP (75 ng g$^{-1}$d.w.) > Ace (47 ng g$^{-1}$d.w.) > Fla (43 ng g$^{-1}$d.w.) > Cor (34 ng g$^{-1}$d.w.) > Fl (33 ng g$^{-1}$d.w.) > Ant (24 ng g$^{-1}$d.w.) > BbkF (20.9 ng g$^{-1}$d.w.) > BeP (19 ng g$^{-1}$d.w.) = BaP (19 ng g$^{-1}$d.w.) > BaA (18 ng g$^{-1}$d.w.) > IP (14 ng g$^{-1}$d.w.) > DBA (10 ng g$^{-1}$d.w.). Among all congeners, BaP, which is the most important carcinogenic PAH, was significantly higher in MC (47 ng g$^{-1}$d.w.) than RF (19 ng g$^{-1}$d.w.) soil/dust samples ranging between 17 and 85 a ng g$^{-1}$d.w. and from 9 to 31.2 ng g$^{-1}$d.w. in MC and RF soil/dust samples, respectively ($p < 0.01$). The concentrations of $\sum$PAHs, $\sum$COMB, and $\sum$7-carcinogenic PAHs were also higher in MC soil/dust samples than in RFs (ranging between 690 and 1496 ng g$^{-1}$d.w.; between 387 and 801 ng g$^{-1}$d.w.; and between 206 and 421 ng g$^{-1}$d.w., respectively) (Table 3). It was also observed that almost half of the total PAHs derived by combustion origin, and the $\sum$7-carcinogenic PAHs were higher in MC soil/dust samples than RFs (Figure 1, see also Figure S1 and S3). However, the proportion of combustion origin PAHs was lower in RF than MC soil/dust samples. The soil/dust sample represents a mixed pattern in which 3, 4, and 6 rings PAHs dominated the profile. In order to identify the sources of PAHs in both sites, diagnostic ratios were used for apportionment and identification of source tracers (Yunker et al. 2002; Kamal et al. 2014).

The diagnostic ratios (Table 3) and the score plot (Figure 2 and Figure S3) showed dominance of petrogenic sources in RF samples, whereas predominance of petroleum
combustion and mixed sources in MC soil/dust samples. The ratios suggest dominance of petrogenic sources and traces of petroleum combustion, however, in MCs soil/dust; the petroleum combustion and other petrogenic sources were major contributors. The Σ456-PAHs/ΣPAHs ratios < 0.40 is an indicator of petrogenic source while it represents...
petroleum and other biomass combustion burning when it is > 0.5, and in the case of
MC soil/dust samples, the ratio shows a mixed PAH source between petrogenic emis-
sion and petroleum burning origins, whereas a predominant petroleum source in RFs
(Biache et al. 2014 and reference therein).

In this study, the following PAH concentration diagnostic ratios, characteristic of
the anthropogenic emissions, were calculated as follows: IP/(IP + BghiP), BaP/(BeP + BaP),
Fl/(Pyr + Fl), BghiP/IP, BaP/BghiP, and Ant/(Ant + Phe).

The ratio BaP/(BeP + BaP) provides information on the photodegradation of PAHs
in ambient air, because BaP degrades faster than BeP in the atmosphere. Thus, a ratio
value < 0.5 indicates that PAHs have undergone aging and photodegradation, as shown
by the ratio of the MC soil/dust samples. The ratio of IP/IP + BghiP) presumes that
PAHs ratios < 0.2 indicate petroleum sources; ratios in the range ≥ 0.2 and ≤ 0.5 as
petroleum combustion sources; and > 0.5 as grass, wood, and coal combustion sources
(Biache et al. 2014 and reference therein). In the case of both MC and RF soil/dust
samples, the ratio has been found ranging between 0.13 and 0.19, which indicated
petroleum as a major contributing source. The Ant/(Ant + Phe) ratio presumes that ratios
> 0.1 indicate combustion origin, while ratios < 0.1 indicate PAHs source to be of petro-
leum origin (Yunker et al. 2002; Mannino & Orecchio 2008). This ratio ranged between
0.08 and 0.28 in RFs samples, which indicated mixed petroleum and petroleum combus-
tion signature, and was significantly lower than that found in MC soil/dust samples, and
the ratio indicated combustion of petroleum as dominant source. The Fl/(Pyr + Fl) ratio
presumes that ratios < 0.4 also indicate petroleum sources, while ratios in the range
≥ 0.4 and ≤ 0.5 indicate petroleum combustion. Ratios < 0.4 for both MC and RFs soil/
dust samples indicated petrogenic source. According to BaP/BghiP ratio, a value < 0.6
indicates non-traffic emission, while > 0.6 may be related with traffic emission. This
ratio ranging between 0.2 and 0.88 in MC soil/dust samples showed some signatures of
traffic exhaust emissions (Moyo et al. 2013 and reference there in). LMPAHs/HMPAHs
ratios also indicated that RFs PAHs were mostly derived from petrogenic sources (i.e.
> 1) while that of MC soil/dust samples (< 1) showed prevalence of some traces of
pyrogenic sources (Zhang et al. 2008).

Evaluation of ILCR

The ILCR approach was used to estimate PAH exposure risk for adults (18–70 years) and
children (5–17 years, considered as child-labors in MC working areas) via ingestion, inha-
lation, and dermal contact pathways (Table 4). The results showed that dermal contact and
ingestion pathways contributed more than inhalation route to PAHs exposure in MCs.
ILCR-values of dermal route of exposure ranged from 4.69 × 10^{-6} to 9.39 × 10^{-4} and
from 3.44 × 10^{-6} to 6.88 × 10^{-4} for adults and children, respectively, whereas the ILCR-
values for ingestion route of exposure varied from 2.53 × 10^{-6} to 5.06 × 10^{-4} and from
1.85 × 10^{-6} to 3.71 × 10^{-4} for adults and children, respectively. It is worth noting that chil-

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dren working as apprentices in auto-repair work areas are at high risk of soil/dust-bound
PAHs exposure via dermal contact and ingestion routes because their high frequency of
hand-to-mouth activities as well as their immune and nervous system vulnerability
(Maertens et al. 2008). The total cancer risk due to PAHs exposure from all the routes ran-
ged between 7.22 × 10^{-6} and 1.45 × 10^{-3} and from 5.29 × 10^{-6} to 1.06 × 10^{-3} for adults
and children, respectively. According to United States Environmental Protection Agency
(US EPA) criteria, which reported a ILCR value of 10^{-4} and/or greater as moderate-high
Table 4. Individual and total ILCR from three different routes of exposure.

| Exposure sites | Dermal | Ingestion | Inhalation | Total risk |
|----------------|--------|-----------|------------|------------|
|                | Adult  | Children  | Adults     | Children   | Adults     | Children   |
| MC             | 4.37E-04 | 3.20E-04 | 2.36E-04   | 1.73E-04   | 1.83E-08   | 5.03E-09   | 6.73E-04 | 4.93E-04 |
|                | 2.82E-04 | 2.07E-04 | 1.52E-04   | 1.12E-04   | 1.18E-08   | 3.24E-09   | 4.34E-04 | 3.19E-04 |
|                | 3.82E-04 | 2.80E-04 | 2.06E-04   | 1.51E-04   | 1.60E-08   | 4.39E-09   | 5.88E-04 | 4.31E-04 |
|                | 4.69E-06 | 3.44E-06 | 2.53E-06   | 1.85E-06   | 1.96E-10   | 5.39E-11   | 7.22E-06 | 5.29E-06 |
|                | 9.39E-04 | 6.88E-04 | 5.06E-04   | 3.71E-04   | 3.93E-08   | 1.08E-08   | 1.45E-03 | 1.06E-03 |
| RF             | 4.60E-06 | 3.37E-06 | 2.48E-06   | 1.82E-06   | 1.92E-10   | 5.28E-11   | 7.08E-06 | 5.19E-06 |
|                | 5.21E-07 | 3.81E-07 | 2.81E-07   | 2.06E-07   | 2.18E-11   | 5.98E-12   | 8.02E-07 | 5.87E-07 |
|                | 4.74E-06 | 3.47E-06 | 2.56E-06   | 1.87E-06   | 1.98E-10   | 5.45E-11   | 7.30E-06 | 5.34E-06 |
|                | 3.64E-06 | 2.67E-06 | 1.96E-06   | 1.44E-06   | 1.52E-10   | 4.19E-11   | 5.60E-06 | 4.11E-06 |
|                | 5.17E-06 | 3.79E-06 | 2.79E-06   | 2.04E-06   | 2.16E-10   | 5.94E-11   | 7.96E-06 | 5.83E-06 |
cancer risk, all ILCR-values reported for RF soil/dust samples (lower than $10^{-6}$) suggested no significant risk for any individual or combined exposure pathway risk for RF workers (as proposed by the US EPA (2005)).

**Conclusions**

In this study, we assessed the potential exposure of workers to PAHs in chemical work places via dermal, ingestion, and inhalation pathways. All participants, in particular the self-employed in auto-mechanical workshops, were found to be working without any personal protective equipment and regular use of coveralls and gloves when handling petrochemicals during daily working hours. In comparison with RF workplaces, exposure to aromatic solvents, driven by UGEO and residues of petroleum products, was much more prevalent in the ambience of MCs. In fact, RFs were found to be under low carcinogenic risk. The hazardous occupational environments have been demonstrated to be potential risks to workers’ health, causing mild impact on in blood parameters. Exposure to PAHs and other toxic chemicals in the work environments can be reduced considerably by the use of personal protections, work practice controls, and specific regulations.

**List of abbreviations**

- 1-OHPyr: 1-hydroxypyrene
- BMI: body mass index
- CN: control group
- ILCR: incremental lifetime cancer risk
- MC: auto-repair mechanic
- MCV: mean cell volume
- MW: molecular weight
- PAHs: polycyclic aromatic hydrocarbons
- PCV: packed cell volume
- PLTs: platelets
- RBCs: red blood cells
- RF: refinery workers
- US EPA: United States Environmental Protection Agency
- UGEO: used gasoline engine oil
- WBCs: white blood cells

**Supplementary material**

The supplementary material for this paper is available online at [http://dx.doi.10.1080/09603123.2015.1007843](http://dx.doi.10.1080/09603123.2015.1007843)

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