Risk assessment of BTEX concentration from combustion of coal in a controlled laboratory environment

Daniel Masekameni13,*, Raeesa Moolla2, Mary Gulumian45 and Derk Brouwer1

1 Occupational Health Division, School of Public Health: University of the Witwatersrand, Parktown, 2193. 
Derk.brouwer@wits.ac.za
2 School of Geography, Archaeology and Environmental Studies, University of the Witwatersrand, Private Bag X3, WITS, 2050. Raeesa.molla@wits.ac.za
3 Department of Geography, Environmental Management and Energy Studies, University of Johannesburg, Johannesburg, South Africa.
4 National Institute for Occupational Health, National Health Laboratory Services, Braamfontein, Johannesburg, South Africa. mary.gulumian@nioh.nhls.ac.za
5 Haematology and Molecular Medicine, School of Pathology, University of the Witwatersrand University of the Witwatersrand, Parktown, Johannesburg, 2193
* Corresponding author: danielmasekami@gmail.com, +27 11-717 2355

Abstract: A D-grade type coal was burned under simulated domestic practices in a controlled laboratory set-up, in order to characterize emissions of volatile organic compounds (VOCs); viz. benzene, toluene, ethylbenzene and xylenes (BTEX). Near-field concentrations were collected in a shack-like structure constructed using corrugated iron, simulating a traditional house found in informal settlements in South Africa. Measurements were carried out using the Synspec Spectras GC955 real-time monitor over a three-hour burn cycle. The 3-hour average concentrations (in µg/m³) of benzene, toluene, ethylbenzene, p-xylene and o-xylene were 919 ± 44, 2051 ± 91, 3838 ±19, 4245 41 and 3576 ± 49, respectively. The cancer risk for adult males and females in a typical SA household exposure scenario, was found to be 1.1 -1.2 and 110-120 folds higher than the US EPA designated risk severity indicator (1E+), respectively. All four TEX compounds recorded the Hazard Quotient (HQ) of less than 1, indicating a low risk of developing related non-carcinogenic health effects. The HQ for TEX ranged from 0.001 – 0.05, with toluene concentrations being the lowest and ethylbenzene the highest. This study has demonstrated that domestic coal burning may be a significant source of BTEX emission exposure.

Keywords: Coal, BTEX, Hazardous air pollutants, domestic fuel burning

1. Introduction

The introduction of several chemicals in the atmosphere has been widely associated with increased health risks (1,2). Anthropogenic sources of higher exposure to air pollutants is suggested to be attributed to industrial activities (3,4). Several studies have been conducted globally, investigating the emissions of larger industrial activities such as power generation on the external environment (5,6). The mechanisms as to how pollutants are emitted and distributed are well understood, especially on larger stationary sources in developed countries and parts of developing Asia.

There is a growing concern globally, regarding pollutant inventories in order to understand major sources of emissions and their impacts (7). There is an emerging body of knowledge which suggests that indoor household burning presents a major threat to public health (8,9) arising from lack of access to clean energy sources, which has been identified as a major contributor to local indoor...
Emission of volatile organic compounds (VOCs) under these conditions may present an important class of pollutants as it has been associated with several health and environmental impacts (14–16). It is reported that VOCs, even at low concentrations, can produce several health effects including nausea, eye and throat irritation, induction of asthma attacks, fatigue, dizziness and mental confusion (17–21). VOCs in general are quite numerous however; emphasis is given to mono-aromatic volatile organic compounds termed BTEX (benzene, toluene, ethyl benzene and xylenes). This group of VOCs are often considered carcinogenic (22,23). Particularly, benzene and ethylbenzene exposure is linked with increased risk of leukemia and hematopoietic cancers (24–26). Toluene and xylene are non-carcinogenic but may produce reproductive adverse effects; especially when exposures are chronic at low to high concentrations (27).

Efforts to create an exposure inventory for BTEX is mainly done in occupational environments, while less information is available at non-occupational settings (28–34). The sources of BTEX in residential areas are diverse including domestic care products; life style related chemicals such cigarette smoke; and combustion energy-related sources (35). It has also been suggested that the risk of exposure is higher in indoor environments relative to outdoor environments (36–41).

Exposure to airborne pollutants is influenced by many factors such as emission rate at the source, air exchange rate, pollutant concentration and time spent indoors and meteorological conditions (32,35,38,42). Children and the elderly are the most vulnerable groups as they spend most of their time indoors and also due to weaker immune system (42). Moya, Bearer and Etzel (2004), have emphasized that infants and children are at greater risk than adults due to their high metabolic and resting rate compared to adults (32,35). It was further found that children spend most of their time indoor next to their mothers and are thus exposed to elevated concentrations of combustion pollutants, during cooking and heating conditions (20).

In regulating exposure to toxic compounds on human health, many countries use risk assessments as a tool to determine the relative risk and develop action plans based on the emissions or concentration. However, risk assessment considers various factors in estimating a possibility of a biological response. Factors such as hazard source identification, exposed group, exposure pathway, concentration of the contaminant, target organ and potential biological response dose which might trigger a response are investigated (32,42). Hematotoxicity and immunotoxicity have been widely used as indicators for non-carcinogenic effects of benzene exposure (44–46). Chronic exposure to benzene have been reported in several studies and reviews indicating the risk of anemia, bone marrow hyperplasia, aplastic anemia, leukopenia, lymphocytopenia, thrombocytopenia and pancytopenia has been shown (24,46,47).

Exposure to high concentrations of BTEX have been widely associated with several adverse health effect in countries such as USA, India and China (46–50). Despite several human health effects reported elsewhere regarding exposure to BTEX, in South Africa very few studies have been conducted to quantify indoor and environmental exposure to BTEX, especially from domestic activities where coal burning has been consistently linked to severe health effects (51,52). The present study aims to quantify concentrations of BTEX from domestic coal burning process and evaluate potential health risk in respect of cancer and non-cancer effects. The study uses experimental data on BTEX emission were used as proxies for near field concentration to estimate exposure mimicking indoor use of coal in a brazier applicable in the South African informal settlements. The study is
hoped to assist in contributing knowledge on domestic solid fuel burning technologies and might aid in supporting future epidemiological and other studies in South Africa and in other low to medium income countries with domestic coal burning activities.

2. Materials and Methods

2.1. BTEX sampling condition

BTEX were sampled under the laboratory conditions simulating community based activities. The combustion laboratory was constructed using corrugated iron and combustion materials included coal, wood kindling and paper. The selection of the stove, known as brazier (imbaula), to fuel combination (Top-Lit Updraft (TLUD) and High ventilated stove), was based on the government project roll-out program of TLUD ignition method as an interim air pollution reduction strategy initiative and the selection of high ventilated stoves was based on local studies which proven that the use of high ventilated stove lit with TLUD leads to the reduction of emissions (13,53). Tests were performed over a period of three hours and further details on the burn sequences are provided in (13,54). Stove was lit up using the (TLUD) method in a high ventilated brazier, using D-grade bituminous coal. Further, details on the stove and fuel combination can be obtained in published literature as contained in the references (13,16,55,56).

The study was carried out at the University of Johannesburg's Sustainable Energy and Research Centre in South Africa. The stove was placed at the center of the combustion lab. The combustion laboratory is built simulating a typical informal house colloquially known as a shack, constructed using corrugated irons, with a small window (300 mm x 400 mm) and a standard door (840 mm x 1.8 m) as shown in Figure 1.

Figure 1: Schematic diagram of a traditional corrugated iron house in a typical South African informal settlement. (Not drawn to scale)

2.2. Domestic combustion scenario in South African low income settlement

Prior to stove ignition, all openings leading to the outside of the shack were closed/ sealed mimicking field based practices. Nevertheless, it must be noted that air leaks could occur since the...
sealing of openings were not comprehensive enough to contain all emitted pollutants, which might be similar to a typical shack. The stove was placed at the center of the combustion lab and measurements were taken at 1 m above the floor and 1.2 m away from the stove as shown in Figure 1. Domestic coal fire is generally associated with high heat generation, simultaneously increasing the indoor temperature significantly. Due to the sensitivity of the monitoring equipment care was given to separate the experimental and data capturing rooms. The detection device was placed in the analysis room next to the combustion laboratory. The sampling probe of 1.9 m in length was used to draw in sample to the detection device/ gas analyzer. The isolation or removal of the detection device from the hot environment was to avoid similar challenges experienced during field monitoring in Moolla et al. (2014), where higher temperature led to instrumentation malfunctioning and loss of data.

Samples were taken and averaged for each distinct time aggregate (15 minutes, 45 minutes and 2 hours, to coincide with burn cycles). The first sample was taken from the time the fuel is lit until the establishment of the flame i.e. the first 15 minutes of the combustion where the condition is smoldering (i.e. burning slowly with visible smoke but without flames) with insufficient air supply and low fuel bed temperature, the next stage is when a flame is well established and the combustion process is at the mixing stage and takes about 45 minutes, the last stage where there is no visible flame and only coke/ fixed carbon burns and char formation often takes place (~120 minutes). The laboratory experiments were done three times per each combustion time interval, where the average concentrations over three experiments was used in the study.

2.3. BTEX sampling instruments

In the present study, five VOCs were monitored using the Synspec Spectras gas chromatography (GC955, series 600). This instrument is widely used to monitor BTEX and has been approved as per service specification EN 14662-3. The samples were drawn in through the inlet feeder operated at a flowrate of 5 ml/min connected at the back of the instrument. A 37 mm filter was connected between the monitoring instrument and the inlet probe to isolate or exclude foreign particles. Drawn in hydrocarbons are firstly pre-concentrated in the Tenax GR, where they were pre-heated and desorbed, and thereafter separated according to columns. The instrument is coupled with a photo ionizer detector (PID) which assists in increasing sensitivity for benzene and other aromatic hydrocarbons. The running cycle can be from 15 minutes upwards, which can be adjusted and operates at a temperature of <70 °C. Helium was used as a carrier gas set at a pressure of 350 kPa, connected using Teflon tubing at a distance of 1.8 m (from the gas cylinder to the GC).

2.4. Risk assessment

BTEX emissions were monitored at near field (inside the room at 1.2 m away from the emitting source) and were then used as room concentrations. In a typical coal burning house in South Africa, coal stoves are often used indoors during the winter season between 18h00 pm to 21h00 pm. This is due to the inherent heat production of the technology while in summer it is unlikely that the stove will be used indoors for either cooking or space heating. The winter period in South Africa is from June to August (equivalent to 92 days). During winter, all outlets leading to the external environment are closed with the intention to contain all produced or radiated heat from the device. Due to the hazardous nature of coal and associated carbon monoxide poisoning, households often extinguish the fire and remove the
stove from the indoor spaces before going to bed. We therefore, used this scenario to estimate the exposure duration.

In general, we have conducted risk assessment to estimate the potential exposure to BTEX from domestic coal burning and similar to other studies, used the data to assess the risk to human health (48,57,58). Risk assessment is a comprehensive process which includes hazard source identification, evaluation, characterisation and control, aiming at prevention of possible health outcomes. Thus, this research has aimed at assessing both carcinogenic and non-carcinogenic potential risk by estimating exposure to BTEX.

a) Hazard identification

During data collection, BTEX concentrations from the coal burning device was monitored. The focus of the monitoring was limited to BTEX emissions due to their inherent carcinogenic and non-carcinogenic health impacts on exposed groups. As mentioned earlier, benzene is classified as a Group A human carcinogenic according to US Environmental Protection Agency (EPA) (59,60). Using the approach similar to the one described below, a lifetime exposure duration of 30 years was assumed for residential dwellers as a default value based on the USEPA (61). Common occupants in South African informal settlements are often unemployed individuals or those who falls in the low income brackets. In the study conducted by (62,63), it was found that majority of household live on average income of R3 500. Predominately, adult males and females resides in informal settlements while just a small number of school going children (64). Informal settlements are often built closer to employment sites or in industrial zones (63,65). The average age of dwellers was estimated to be 35 years, while majority of shack owners are males at 61% relative to females (64). A small proportion of children below 18 years were recorded during census 2010 at 1% of the total dwellers in the informal sector (65).

b) Exposure assessment

For the exposure assessment we have considered the estimated dose expressed as chronic daily intake (mg/kg/day). Due to inadequate available methodologies to determine the internal dose we used near field breathing zone concentration for the exposure assessment. We assumed that the breathing zone concentration is equal to the near field concentration or emission zone (61). The driving factors in dose estimation were exposure pathway (air) including route of entry (inhalation), frequency to which one is expected to be exposed, duration of exposure and population age group (Adults male and females). Since, this was a laboratory based study simulating the experience of residents, where population index is not present, the study adopted some of the parameters for the exposure scenario from the US EPA’s risk assessment guidelines and South African Statistics as in Table 1 (61,64).

Table 1: Summary of exposure scenario factors and values used in this study

| Parameter | Description | Value | Unit |
|-----------|-------------|-------|------|
| C         | Room concentration | -     | mg/m³ |
| IR        | Inhalation rate   | 20    | m³/day |
| BW        | Body weight       | 70 males/ 60 kg females | Kg |
Dose response relationship was used to estimate potential biological response for each pollutant. Similar to (66,67), average concentration for the entire burn cycle was used to calculate the chronic intake concentration. Chronic daily intake (CDI) (equation 1) for both carcinogenic and non-carcinogenic pollutants was calculated using values as shown in Table 1. The average CDI \text{year} provides estimated daily intake corresponding to an annual dose.

\[
CDI (\text{averaged daily intake}) = \frac{C \times CF \times IR \times ED}{BW \times AT}
\]  

Equation 1

The chronic daily intake (CDI) determination was used as a basis for risk assessment calculation similar to current risk assessment studies (66,68–70) where:

- C is the breathing zone concentration of a BTEX in (µg/m\(^3\)) derived from three identical experiments taken over 3-hour burn cycle;
- CF is the concentration conversion (mg/µg= 0.001 or 1 µg) factor;
- IR is the inhalation rate (default adults 20 m\(^3\)/day);
- ED is the exposure duration as in equation 2 (11.5 days);
- BW is the average body weight (70 kg, 60 kg for male and female adult respectively);
- AT is the number of days per year.

However, default values as contained in Table 1, assume a daily intake of pollutant over a 24-hour period, is often constant and can be extrapolated over a year. In our study, there was a variation on exposure duration due to the nature on how households use the technology.

In equation 2, we determined a procedure used to estimate exposure duration in a typical winter period in South Africa. The limitation of this method is that the exposure duration seeks to be confined to coal combustion period (3 hours), without taking into account exposure resulting from accumulated concentrations which might take time to vent out from indoor to outdoor. Since, this was a laboratory study the authors intentionally left out other variables in an ordinary house in informal settlements. Such variables may include ventilation rate or the building envelope which influences the air ratio taking into account the exchange from inside to outside. The exposure duration obtained in equation 2 indicates a daily average exposure, given that exposure involves a 3-hour duration over 92-day period in a year from this source (to allow for a full season).

\[
ED = \frac{\text{Actual exposure duration}}{24 \text{ hours}} \times 92 \text{ days}
\]  

Equation 2
Where:

- ED is the exposure duration (days/year);
- Actual exposure duration is the 3-hour combustion period;
- 24 hours is the total hours in a day; and
- 92 days is the number of exposure days in a year.

In equation 1, an average annual chronic daily intake was determined. However, for risk assessment, a cumulative lifetime exposure concentration intake needs to be completed. In equation 3 the average 30 years chronic dose (CDI_{30 years}) is calculated using the 30 year residential exposure duration as obtained from USEPA default value

\[
CDI(30 \text{ years dose}) = \sum CDI \times 365 \times YE \\
\text{60/67}
\]

Equation 3

Where:

- CDI is the cumulative average 30-year dose (mg/kg/day);
- CDI is the chronic daily intake (mg/kg);
- YE is estimated lifetime residential exposure duration which is equivalent to 30 years;
- 365 is the total number of days in a year;
- 60 is the male life expectancy and 67 is the female life expectancy in South Africa.

Therefore, for risk assessment calculation we need the adjusted lifetime chronic daily intake (CDI_{adj}), taking in to account the life expectancy for a female and male South African adult resident. In equation 4, we calculated the average CDI_{adj}, assuming a lifetime daily dose intake.

\[
CDI_{adj} = \frac{CDI(30 \text{ years average dose})}{\text{life expectancy in days}}
\]

Equation 4

We assume that the average chronic daily adjusted dose over a lifetime amongst female and male adults will better simplify risk assessment calculation as in equation 4.

c) Toxicity assessment and risk characterisation

Risk characterisation is the last step in risk assessment which provides information on the hazard status of a contaminant or pollutant (71). For both carcinogenic and non-carcinogenic effects, the use of inhalation Reference Concentration (RfC) assists in determining the health risk associated in exposed population. For carcinogenic pollutants (such as benzene) the use of the slope factor can be used to estimate the relative risk. Furthermore, the use of the inhalation reference concentration was based on toxicological/ occupational epidemiology studies focusing on several health outcomes such as cellular necrosis. In summary, inhalation Reference Concentration (RfC) is an estimated daily human inhalation exposure which is suggested not to cause a health effect in a lifetime. (46,47,72).

A lifetime inhalation dose of BTEX was determined based on the absolute lymphocyte count (ALC) at the benchmark concentration adjusted (BMCL) of 8.2 mg/m³. The inhalation benzene lifetime exposure was therefore calculated using the benchmark dose modelling and was found to be 0.03 mg/m³. The 0.03 mg/m³ was therefore, described as the RfC for benzene (72). The non-carcinogenic effects of TEX inhalation reference concentration for each pollutant was used to calculate the hazard quotient as in Table 2 (73–75).
Since benzene is the only confirmed human carcinogenic (category A) pollutant amongst the BTEX pollutants, the slope factor was used to calculate the cancer risk. We have adopted the methodology for calculating the cancer risk using the slope factor from previous similar studies (44–46, 72, 76). It must be noted that there is no threshold for carcinogenic compounds. Therefore, the use of reference levels is used as a guide to probably support a decision. In our study we used both designated cancer severity indicator of 1 case /10^4 (1E-4) and 1/10^6 (1E-6) (59, 77).

Table 2: Benzene slope factor and TEX inhalation reference values

| Chemical      | Inhalation Reference concentration (RfC) (mg/m^3) | Inhalation Slope Factor (SF) (mg/kg/day)-1 |
|---------------|-----------------------------------------------|---------------------------------------|
| Benzene       | 0.03                                          | 0.0273                                |
| Toluene       | 5                                             | N/A                                   |
| Ethylbenzene  | 1                                             | N/A                                   |
| O-xylene      | 0.1                                           | N/A                                   |
| P-xylene      | 0.1                                           | N/A                                   |

For carcinogenic pollutants, it must be noted that there is no safe threshold, therefore risk characterisation followed was similar to the method described by the US EPA’s Risk Assessment Guidance for Superfund (61); We thus calculated the risk of cancer by using equation 5 (72).

\[ CR = CDI_{adj} \times SF \]  

Equation 5

Where:

- SF is the slope factor for carcinogenic pollutant (0.0273);
- CR is the carcinogenic risk; and
- CDI_{adj} is the cumulative lifetime adjusted dose (equation 4) over an estimated exposure in a lifetime of 60 or 67 years for male and female adult, respectively.

Therefore, Cancer risk > 1E-6 and 1E-4 means carcinogenic effects of concern while the cancer risk < 1E-6 and <1E-4 means a designated cancer severity indicator.

For non-carcinogenic pollutants a hazard quotient (HQ) was used to estimate the potential health risk of dwellers. Where a HQ value is greater than one, it is regarded as a hazardous exposure; while HQ value of less than one means there is a low probability of developing associated health effects. In equation 6, the procedure for calculating HQ is shown.

\[ HQ = CDI_{adj} \times (mg/kg)/(day)/(RfC(mg/m^3) \times 20m^3/(70 kg)) \]  

Equation 6

Where:

- HQ is the hazard quotient;
- CDI_{adj} is the cumulative intake dose;
- RfC is the reference;
- 20 m^3 is the default value for average adult daily air volume; and
70 kg is the average body weight for a male adult while 60 kg will be used for female adult.

2.5. Quality control

All monitoring instruments were maintained and operated, according to the manufacturer’s instructions, and returned to the suppliers for factory calibration at prescribed intervals. Before each test, the gas probes were cleaned by means of compressed air, to remove any residue from prior tests, which might negatively affect the next results. All monitoring instruments were zero checked, according to the manufacturer’s instructions, before monitoring/sample collection.

The GC955 instrument was tested in accordance the EMC directive 89/336/EMC, test specification EN 50081–1:1991 and EN 50082–2: 1994. The monitoring instruments were calibrated before use (calibration was done in the range of 0 to 10 ppb). Quality control checks were conducted during and after the monitoring campaign and a correction factor of 2 ppb and 4 ppb for benzene and toluene, respectively were used, to counter systematic under-sampling of the instrument.

Background concentrations were accounted for as BTEX from outside the testing facility could possibly infiltrate the testing laboratory and contribute to the final concentration readings. The instrument was run for 30 minutes before the three-hour testing duration and the background concentrations were calculated using equation 7.

\[ C_{\text{combustion}} = C_{\text{activity}} - C_{\text{without}} \]

Equation 7

Where:

- the \( C_{\text{combustion}} \) is the final concentration;
- \( C_{\text{activity}} \) is the actual sample collected during the BTEX generating activity was taking place + background concentration;
- \( C_{\text{without}} \) is the concentration of BTEX obtained in the absence of the activity under investigation.

In experimental studies the use of equipment which are accurately calibrated is an important quality control feature and it assist in reducing the uncertainty of data set. Trial runs before the actual tests might help in the identification of instruments malfunctioning and detection signal faults.

3 Results and Discussion

3.1. BTEX concentration under laboratory conditions

The results from the coal combustion brazier; under a laboratory controlled environment are presented herein. In Figure 2, time aggregates concentration for each BTEX compound is presented as an average concentration for the specified time (15, 45 and 120 minutes). Using a 3-hour average concentration Benzene is the lowest emitted VOC, while ethyl benzene was found to be the most emitted pollutant throughout the combustion cycle. From the results, it is shown that the concentration of BTEX are consistent throughout the entire burn cycle of the three-hour period.

Benzene and ethyl benzene concentration steadily increases as the combustion process progresses. The minimum concentration as can be depicted from Figure 2, is associated with the first 15 minutes of the combustion. Contrary, to benzene and ethyl benzene the concentration of toluene and xylene were highest in the 45 minutes and 120 minutes respectively. The observed BTEX profile reported in our study is similar to the one presented in the study by (29). However, the observed differences may require additional statistical analysis to provide more details on concentration...
variation at different time intervals. Unfortunately, the differences on BTEX concentration at different time aggregates was not within the scope of the current project. The implication of this finding indicates for the first time in South African domestic sector the determination of domestic coal combustion as might be an important source of BTEX in indoor air spaces.

![Figure 2: Time series BTEX concentration for a 3-hour combustion cycle](image)

In table 3, BTEX near field room concentrations are presented for replicates three experiments as averages over a 3-hour burn cycle. Benzene concentration ranged from 857 – 942 µg/m³ with the mean of 919 µg/m³ over a three-hour burn cycle. The benzene concentration observed in our study varied from those conducted in India where the concentrations have ranged from 44 – 167 µg/m³ (50). However, in the latter study, the emissions of benzene were associated with kerosene burning which is different from our present study. Lower values of indoor benzene concentrations were also reported in several other studies where the concentration ranged from 0.7 – 7.2 µg/m³ (78–80). In Hong Kong Special Administrative Region of China similar low benzene indoor level were reported which was mainly associated with vehicular emissions at 0.5 – 4.4 µg/m³ (30,81). However, studies conducted in petrol refineries reported concentrations for benzene varied between 12 – 17 000 µg/m³, with the highest exposure concentrations being mainly from refinery workers working in indoor environment (82–84).

Toluene, ethylbenzene and xylenes (TEX) results are comparable with several studies conducted elsewhere; however, most of this studies were conducted in occupational settings (34,58,69). The ethylbenzene concentration measured in our study was 2 folds higher than the concentration reported by (80). Toluene, ethyl benzene and xylenes also present several health effects even at lower concentrations (85–87).

Table 3: Time weighted average BTEX room concentrations
In table 4 we investigated a percentage contribution of individual BTEX compound. From the total BTEX indoor air concentration benzene was found to have contributed less at 6% while ethyl benzene was the highest at 29%. Fairly, comparable percentage contribution between P-xylene and O-xylene were observed at 26 and 25, respectively. However, despite benzene being the least quantified VOC it is worrying given its hazardous nature to human health. Toluene was found to be the lowest contributed VOC amongst the TEX at 14%.

Table 4: Percentage contribution of each BTEX pollutant averaged over a 3-hour burn cycle

| Duration | Benzenes | Toluene | P-Xylene | Ethyl benzene | O-Xylene |
|----------|----------|---------|----------|---------------|----------|
| 15 minutes | 857 ± 32.40 | 1922 ± 127.5 | 3864 ± 48.33 | 4189 ± 87.11 | 3589 ± 48.74 |
| 45 minutes | 958 ± 5.73 | 2137 ± 27.04 | 3831 ± 15.12 | 4257 ± 31.26 | 3510 ± 13.66 |
| 2 hours | 942 ± 13.36 | 2095 ± 36.59 | 3819 ± 9.60 | 4288 ± 91.51 | 3628 ± 9.42 |
| 3 hours Average conc. | 919 ± 44 | 2051 ± 93 | 3838 ± 19.04 | 4245 ± 41.13 | 3576 ± 49 |
determined $1.2 \times 10^{-4}$ and $1.1 \times 10^{-4}$ respectively. The cancer risk for woman was found to be higher than that of males. This finding suggest that women will be more vulnerable than men even though the exposure concentration is the same. As shown in table 5, the cancer risk for women suggest that 120 people will be at risk of cancer per $1 \times 10^6$ of the exposed population. Furthermore, in table 6, results show that 110 men per million exposed will be at risk of carcinogenic health effects. In both exposure scenarios (male and female) the cancer risk was found to be higher than the acceptable risk levels of $1 \times 10^{-6}$ and $1 \times 10^{-4}$. 
### Table 5: Carcinogenic and non-carcinogenic risk for adult female

| Pollutant      | Average concentration | CDI\(_{\text{year}}\) | CDI\(_{30\ \text{year}}\) | CDI\(_{\text{adj.}}\) | CR | HQ | CR/ 1E\(_6\) | CR/ 1E\(_4\) |
|----------------|-----------------------|------------------------|-------------------------|-----------------------|----|----|-------------|-------------|
| Benzene        | 919 µg/m\(^3\)       | 0.0097                 | 1.06E+02                | 4.32E-03              | 1.2E-04 | N/A | 120         | 1           |
| Toluene        | 2051 µg/m\(^3\)      | 0.0215                 | 2.36E+02                | 9.64E-03              | N/A | 0.001 | n/a         | n/a         |
| P-Xylene       | 3838 µg/m\(^3\)      | 0.0403                 | 4.41E+02                | 1.73E-02              | N/A | 0.050 | n/a         | n/a         |
| Ethyl benzene  | 4245 µg/m\(^3\)      | 0.0446                 | 4.88E+02                | 2.00E-02              | N/A | 0.006 | n/a         | n/a         |
| O-Xylene       | 3576 µg/m\(^3\)      | 0.0376                 | 4.11E+02                | 1.68E-02              | N/A | 0.049 | n/a         | n/a         |

### Table 6: Carcinogenic and non-carcinogenic risk for adult male

| Pollutant      | Average concentration | CDI\(_{\text{year}}\) | CDI\(_{30\ \text{year}}\) | CDI\(_{\text{adj.}}\) | CR | HQ | CR/ 1E\(_6\) | CR/ 1E\(_4\) |
|----------------|-----------------------|------------------------|-------------------------|-----------------------|----|----|-------------|-------------|
| Benzene        | 919 µg/m\(^3\)       | 0.0083                 | 9.06E+01                | 3.70E-03              | 1.1E-04 | N/A | 110         | 1           |
| Toluene        | 2051 µg/m\(^3\)      | 0.0185                 | 2.02E+02                | 8.27E-03              | N/A | <0.001 | n/a         | n/a         |
| P-Xylene       | 3838 µg/m\(^3\)      | 0.0345                 | 3.78E+02                | 1.55E-02              | N/A | 0.045 | n/a         | n/a         |
| Ethyl benzene  | 4245 µg/m\(^3\)      | 0.0382                 | 4.18E+02                | 1.71E-02              | N/A | 0.005 | n/a         | n/a         |
| O-Xylene       | 3576 µg/m\(^3\)      | 0.0322                 | 3.52E+02                | 1.44E-02              | N/A | 0.042 | n/a         | n/a         |
We presented the cancer risk for adult females and males where reference was given to the female/male body weight (default value from US EPA, 2010) and life expectancy as in table 1. The cancer risk in adult female and male were found to be 120 and 110 folds higher than the designated cancer severity indicator of $10^{-6}$, respectively. These findings confirm those reported by WHO (WHO.int: Media Centre fact sheets Internet Geneva, Switzerland: World Health Organization; updated 8 May 2018; cited 9 November 2018. Available from: http://www.who.int/mediacentre/factsheets/fs292/en/) and the World Bank (Household Cookstoves, Environment, Health, and Climate Change THE WORLD BANK A NEW LOOK AT AN OLD PROBLEM (2011)) where 17% of premature lung cancer deaths in adults were found to be attributable to exposure to carcinogens from household air pollution caused by cooking with kerosene or solid fuels such as wood, charcoal or coal and the risk for women was higher, due to their role in food preparation.

For non-carcinogenic health effects, we used the hazard quotient to determine the risk. A hazard quotient of greater than 1, was used as a reference value; whereby, a value greater than one indicated higher probability of contracting a related health effect. For both adult male and female, the hazard quotient was found to be below 1 for the TEX’s. Toluene indicated the lowest hazard quotient, whilst ethylbenzene was found to potentially have the highest hazard quotient, at a value of 0.05. The results presented in our study indicate that there is a lower probability of non-carcinogenic health effects as a result of exposure to domestic coal combustion technology as described in this study.

Despite, the non-carcinogenic effects rated hazard quotient of less than one, this might change significantly especially in households were coal burning devices are used indoors for longer durations. This includes overstretched winter periods and prolonged exposure durations based on activity. For instance, in some households especially during winter this type of technology can be used to warm for the entire day time (06:00 am to 18:00 pm) and some part of the night period (18:00 pm and 21:00 pm). This implies that exposure to TEX from this combustion activity may significantly vary from one household to the other depending on the use case scenarios.

4 Study limitations

In the absence of field exposure data, results presented herein had several limitations. Individual information used for risk assessment are average person default values. As known that there is no an average person in the world, this might significantly affect the accuracy of risk determination. Individual varies based on the biological make-up which might affect parameters such as breathing rate and moreover the exposure scenario. Furthermore, using average values overlook the issue of individual susceptibility which might affect the risk score. In addition, we have used a room concentration to assess the risk, assuming that a three-hour exposure at near field breathing zone is the average exposure duration. The influence of pollutants leakages also was not addressed, where there might be loss due to leakages. The BTEX concentration reported in this study was obtained from a laboratory environment which might vary from field concentrations.
Despite these limitations this study has shown that exposure to domestic coal combustion pollutants may lead to risk of carcinogenic effect while the non-carcinogenic effects were found to be unlikely. However, it must be noted that the results presented herein was based on laboratory experiment study where several variables which might influence stove to fuel performance where controlled. Such performance determinants includes stove operational method, fire ignition method, coal particle size, moisture content and coal grade (13,53,88–90).

5 Conclusion

The study attempted to quantify BTEX concentration from domestic coal combustion in a brazier, simulating its use in the South African informal settlements. Based on the results presented in this study, it can be concluded that domestic coal burning might be significant source of BTEX in indoor spaces. The results showed a constant concentration of BTEX throughout the combustion cycle of 3-hours.

The study further, attempted to utilise breathing zone near field BTEX concentration as averaged over a 3-hour burning cycle in adult female and male to estimate carcinogenic and non-carcinogenic health effects, simulating practices in informal settlements. The cancer risks were found to be 110 to 120 folds higher than the designated cancer severity indicator of 1E⁻⁴.

The health risk assessment of TEX, through calculating the hazard quotient, was below the reference value of 1; indicating potentially low exposure to these pollutants and possibly a reduced risk of associated health effects. Lessons drawn from this laboratory experimental study indicate the need for further studies in this field in order to have a better understanding on exposure scenarios and better inform risk characterisation from this source. The study presented the first risk assessment arising from domestic coal burning activities in a laboratory environment while mimicking field practices relevant to South African situation.

Notably, risk assessment is a comprehensive and iterative process to assess the relative risk for several exposure scenarios. It must be understood that risk assessment has several uncertainties, the accuracy of the results depends on the correct risk identification and use of accurate exposure information. Despite all uncertainties, in our studies we attempted to ensure that exposure scenarios are accurately defined which might be used in future for future studies.

Author Contributions: Daniel Masekameni conceptualized and prepared the manuscript. He also carried out the experiment and writing up of the paper. Raeesa Moolla developed the methodology for data analysis. She further analyzed the data and assisted in the editing of the manuscript. Mary Gulumian edited the manuscript and validated methodology for risk assessment. Derk Brouwer supervised the data analysis process, interpretation, presentation of arguments and assisted in the editing of the manuscript.

Acknowledgments: This work was done in collaboration with several people or groups. Sincere appreciations to Mr. Shalala Mgwambani and Mr. Kevin Kasangana for their assistance during laboratory experiments. Mr. Marlin Patchappa for assisting with a regulator and the helium gas. Appreciatiions to Dr. Moolla for funding the project. Dr. Tafadzwa Makonese and Prof Isaac Rampedi for the supervision and guidance they have shown in this work.

Conflicts of Interest: There is no conflict in any part of this work.
References

1. Atash F. The deterioration of urban environments in developing countries: Mitigating the air pollution crisis in Tehran, Iran. Cities. 2007;24(6):399–409.

2. Atabi F, Moattar F, Mansouri N, Alesheikh AA, Mirzahosseini SAH. Assessment of variations in benzene concentration produced from vehicles and gas stations in Tehran using GIS. Int J Environ Sci Technol. 2013;10(2):283–94.

3. Atkinson R, Arey J. Atmospheric Degradation of Volatile Organic Compounds - Atmospheric Degradation of Volatile Organic Compounds. Chem Rev. 2003;103(3):4605–38.

4. Xu M, Yu D, Yao H, Liu X, Qiao Y. Coal combustion-generated aerosols: Formation and properties. Proc Combust Inst [Internet]. 2011;33(1):1681–97. Available from: http://dx.doi.org/10.1016/j.proci.2010.09.014

5. Borhani F, Noorpoor A. Cancer Risk Assessment Benzene , Toluene , Ethylbenzene and Xylene ( BTEX ) in the Production of Insulation Bituminous. 2017;1:311–20.

6. Garg A. Pro-equity Effects of Ancillary Benefits of Climate Change Policies: A Case Study of Human Health Impacts of Outdoor Air Pollution in New Delhi. World Dev [Internet]. 2011;39(6):1002–25. Available from: http://dx.doi.org/10.1016/j.worlddev.2010.01.003

7. Kumar A, Singh BP, Punia M, Singh D, Kumar K, Jain VK. Assessment of indoor air concentrations of VOCs and their associated health risks in the library of Jawaharlal Nehru University, New Delhi. Environ Sci Pollut Res. 2014;21(3):2240–8.

8. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: A systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2224–60.

9. Gordon S, Bruce N, Grigg J, Hibberd P, Kurmi O, Lam K, et al. Respiratory risks from household air pollution in low and middle income countries. Lancet Respir Med [Internet]. 2014;2(10):823–60. Available from: http://pubmedcentralcanada.ca/pmcc/articles/PMC5068561/pdf/emss-70175.pdf

10. Balakrishnan K, Cohen A, Smith KR. Addressing the Burden of Disease Attributable to Air Pollution in India: The Need to Integrate across Household. Environ Health Perspect. 2014;122(1):A6–7.

11. Bonjour S, Adair-Rohani H, Wolf J, Bruce NG, Mehta S, Prüss-Ustün A, et al. Solid fuel use for household cooking: Country and regional estimates for 1980-2010. Environ Health Perspect. 2013;121(7):784–90.

12. Edwards RD, Jurvelin J, Koistinen K, Saarela K, Jantunen M. VOC source identification from personal and residential indoor, outdoor and workplace microenvironment samples in EXPOLIS-Helsinki, Finland. Atmos Environ. 2001;35(28):4829–41.

13. Masekameni D, Makone T, Annegarn HJ. Optimisation of ventilation and ignition method for reducing emissions from coal-burning imbaulas. Proc 22nd Conf Domest Use Energy, DUE 2014. 2014;

14. Garte S, Taioli E, Popov T, Bolognesi C, Farmer P, Merlo F. Genetic susceptibility to benzene toxicity in humans. J Toxicol Environ Heal - Part A Curr Issues. 2008;71(22):1482–9.

15. Abbate C, Giorgianni C, Munao F, Brecciaroli R. Neurotoxicity induced by exposure to toluene. An electrophysiologic study. Int Arch Occup Env Heal [Internet]. 1993;64(6):389–92.
Ernstgård L, Gullstrand E, Löf A, Johanson G. Are women more sensitive than men to 2-propanol and m-xylene vapours? Occup Environ Med. 2002;59(11):759–67.

Midzenski MA, McDiarmid MA, Rothman N, Kolodner K. Acute high dose exposure to benzene in shipyard workers. Am J Ind Med. 1992;22(4):553–65.

Cometto-múiz JE, Cain WS. Relative sensitivity of the ocular trigeminal, nasal trigeminal and olfactory systems to airborne chemicals. Chem Senses. 1995;20(2):191–8.

Ahaghotu E, Babu RJ, Chatterjee A, Singh M. Effect of methyl substitution of benzene on the percutaneous absorption and skin irritation in hairless rats. Toxicol Lett. 2005;159(3):261–71.

Bruce N, Perez-Padilla R, Albalak R. The health effects of indoor air pollution exposure in developing countries. Geneva World Heal Organ Rep WHO/SDE/OEH/0205. 2002;1–40.

Wah C, Yu F, Kim T. Indoor and B uilt Building Pathology , Investigation of Sick Buildings VOC Emissions. 2010;(82):30–9.

IARC. Agents Classified by the IARC Monographs , Volumes 1 – 104. IARC Monogr. 2012;7(000050):1–25.

Marć M, Zabiegała B, Namieśnik J. Application of passive sampling technique in monitoring research on quality of atmospheric air in the area of Tczew, Poland. Int J Environ Anal Chem [Internet]. 2014;94(2):151–67. Available from: http://www.tandfonline.com/doi/abs/10.1080/03067319.2013.791979

Bond GG, Mclaren EA, Baldwin CL, Cook RR. An update of mortality among chemical workers exposed to benzene. Br J Ind Med. 1986;43:685–91.

Schnatter AR, Glass DC, Tang G, Irons RD, Rushton L. Myelodysplastic Syndrome and Benzene Exposure Among Petroleum Workers: An International Pooled Analysis. 2012;104(22).

Lan TTN, Binh NTT. Daily roadside BTEX concentrations in East Asia measured by the Lanwatsu, Radiello and Ultra I SKS passive samplers. Sci Total Environ [Internet]. 2012;441:248–57. Available from: http://dx.doi.org/10.1016/j.scitotenv.2012.08.086

McKenzie LM, Witter RZ, Newman LS, Adgate JL. Human health risk assessment of air emissions from development of unconventional natural gas resources. Sci Total Environ [Internet]. 2012;424:79–87. Available from: http://dx.doi.org/10.1016/j.scitotenv.2012.02.018

Al Zabadi H, Ferrari L, Laurent AM, Tiberghien A, Paris C, Zmirou-Navier D. Biomonitoring of complex occupational exposures to carcinogens: The case of sewage workers in Paris. BMC Cancer. 2008;8(2007):1–10.

Chang E-E, Wei-Chi W, Li-Xuan Z, Hung-Lung C. Health risk assessment of exposure to selected volatile organic compounds emitted from an integrated iron and steel plant. Inhal Toxicol [Internet]. 2010;22(sup2):117–25. Available from: http://www.tandfonline.com/doi/full/10.3109/08958378.2010.507636

Lee CW, Dai YT, Chien CH, Hsu DJ. Characteristics and health impacts of volatile organic compounds in photocopy centers. Environ Res. 2006;100(2):139–49.

Azari MR, Konjin ZN, Zayeri F, Salehpour S. Occupational Exposure of Petroleum Depot Workers to BTEX Compounds. 2012;3(1).
32. Rumchev K, Brown H, Spickett J. Volatile Organic Compounds: Do they present a risk to our health? Volatile Organic Compounds: Do they present a risk to our health? 2016;(October).

33. Vitali M, Ensabella F, Stella D, Guidotti M. Exposure to Organic Solvents among Handicraft Car Painters: A Pilot Study in Italy. 2006;3:30–7.

34. Moolla R, Curtis CJ, Knight J. Assessment of occupational exposure to BTEX compounds at a bus diesel-refueling bay: A case study in Johannesburg, South Africa. Sci Total Environ [Internet]. 2015;537:51–7. Available from: http://dx.doi.org/10.1016/j.scitotenv.2015.07.122

35. Annesi-Maesano I, Baiz N, Banerjee S, Rudnai P, Rive S. Indoor air quality and sources in schools and related health effects. J Toxicol Environ Heal - Part B Crit Rev. 2013;16(8):491–550.

36. Schneider P, Gebefugi I, Richter K, Wolke G. Indoor and outdoor BTX levels in German cities. 2001;

37. Haghighat F, Lee CS, Ghaly WS. Measurement of diffusion coefficients of VOCs for building materials: Review and development of a calculation procedure. Indoor Air. 2002;12(2):81–91.

38. Katsoyiannis A, Leva P, Kotzias D. VOC and carbonyl emissions from carpets: A comparative study using four types of environmental chambers. J Hazard Mater. 2008;152(2):669–76.

39. Katsoyiannis A, Leva P, Barrero-Moreno J, Kotzias D. Building materials. VOC emissions, diffusion behaviour and implications from their use. Environ Pollut [Internet]. 2012;169(9296):230–4. Available from: http://dx.doi.org/10.1016/j.envpol.2012.04.030

40. Nazaroff WW, Weschler CJ. Cleaning products and air fresheners: exposure to primary and secondary air pollutants. 2004;38:2841–65.

41. Wang S, Ang HM, Tade MO. Volatile organic compounds in indoor environment and photocatalytic oxidation: State of the art. 2007;33(x):694–705.

42. Bruinen De Bruin Y, Koistinen K, Kephalopoulos S, Geiss O, Tirendi S, Kotzias D. Characterisation of urban inhalation exposures to benzene, formaldehyde and acetaldehyde in the European Union: Comparison of measured and modelled exposure data. Environ Sci Pollut Res. 2008;15(5):417–30.

43. Moya J, Bearer CF, Etzel RA. VARIOUS LIFE STAGES. 2004;113(4).

44. Aksoy M, Dincol K, Erdem S, Akgun T, Dincol G. Details of blood changes in 32 patients with pancytopenia associated with long-term exposure to benzene. Br J Ind Med. 1972;29(1):56–64.

45. Gelman FYBA, Maszle JJDR, Alexeef LZG. OR I G I NA L I NV ES T I G A T I O N Population toxicokinetics of tetrachloroethylene. 1996;104(December):347–55.

46. Crump KS. Risk of benzene-induced leukemia predicted from the pliofilm cohort. Environ Health Perspect. 1996;104(SUPPL. 6):1437–41.

47. Paxton MB. Leukemia risk associated with benzene exposure in the pliofilm cohort. Environ Health Perspect. 1996;104(SUPPL. 6):1431–6.

48. Dutta C, Som D, Chatterjee A, Mukherjee AK, Jana TK, Sen S. Mixing ratios of carbonyls and BTEX in ambient air of Kolkata, India and their associated health risk. Environ Monit Assess. 2009;148(1–4):97–107.

49. Chen X, Zhang G, Zhang Q, Chen H. Mass concentrations of BTEX inside air environment of buses in Changsha, China. Build Environ [Internet]. 2011;46(2):421–7. Available from: http://dx.doi.org/10.1016/j.buildenv.2010.08.005

50. Pandit GG, Srivastava PK, Mahan Rao a M. Monitering of Indoor Volatile Organic Compounds and Polycyclic Aromatic Hydrocarbons Arising From Kerosene Cooking Fuel. Sci Total Environ. 2001;279:159–65.
51. GroundWork. The Destruction of the Highveld: Digging Coal. 2016.

52. Forouzanfar MH, Alexander L, Bachman VF, Biryukov S, Brauer M, Casey D, et al. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990-2013: A systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2015;386(10010):2287–323.

53. Makonese T, Masekameni DM, Annegarn HJ, Forbes PBC. Influence of fire-ignition methods and stove ventilation rates on gaseous and particle emissions from residential coal braziers. J Energy South Africa [Internet]. 2017;26(4):16. Available from: http://journals.assaf.org.za/jesa/article/view/2089

54. Makonese T. Systematic investigation of smoke emissions from packed-bed residential coal combustion devices. 2015;(April).

55. Le Roux LJ, Zunckel M, Mccormick S. Reduction in air pollution using the ‘bas a njengo magogo’ method and the applicability to low-smoke fuels. J Energy South Africa. 2009;20(3):3–10.

56. Surridge AD, Asamoah JK, Chauke GR, Grobelaar CJ. STRATEGY TO COMBAT THE NEGATIVE IMPACTS OF DOMESTIC COAL COMBUSTION Basa Njengo Magogo Methodology Classical Fire-lighting Methodology. 2004;14(1):13–6.

57. Karachaliou T, Protonotarios V, Kaliampakos D, Menegaki M. Using Risk Assessment and Management Approaches to Develop Cost-Effective and Sustainable Mine Waste Management Strategies. Recycling [Internet]. 2016;1(3):328–42. Available from: http://www.mdpi.com/2313-4321/1/3/328

58. Edokpolo B, Yu QJ, Connell D. Health risk assessment for exposure to benzene in petroleum refinery environments. Int J Environ Res Public Health. 2015;12(1):595–610.

59. Durmusoglu E, Taspinar F, Karademir A. Health risk assessment of BTEX emissions in the landfill environment. J Hazard Mater. 2010;176(1–3):870–7.

60. Robinson SN, Shah R, Wong BA, Wong VA, Farris GM. Immunotoxicological effects of benzene inhalation in male Sprague-Dawley rats. Toxicology. 1997;119(3):227–37.

61. USEPA. Risk assessment guidance for superfund (RAGS). Volume I. Human health evaluation manual (HHEM). Part E. Supplemental guidance for dermal risk assessment USEPA, 2004. Risk assessment guidance for superfund (RAGS). Volume I. Human health evaluation manual (H. US Epa. 2004;1(540/R/99/005):1–156.

62. Makonese T, Masekameni DM, Annegarn HJ. Energy use scenarios in an informal urban settlement in Johannesburg, South Africa. Proc 24th Conf Domest Use Energy, DUE 2016. 2016;

63. Chikoto T. Informal Settlements in South Africa. BSc Treatise. 2009;1–55.

64. StatsSA. P0302: Mid-year population estimates 2017. Stats Sa [Internet]. 2017;(July):10. Available from: http://www.statssa.gov.za/publications/P0302/P03022017.pdf

65. Housing Development Agency HDA. South Africa: Informal settlements Status. 2013;(August):60.

66. Masih A, Lall AS, Taneka A, Singhvi R. Inhalation exposure and related health risks of BTEX in ambient air at different microenvironments of a terai zone in north India. Atmos Environ [Internet]. 2016;147:55–66. Available from: http://dx.doi.org/10.1016/j.atmosenv.2016.09.067

67. Masih A, Lall AS, Taneka A, Singhvi R. Exposure profiles, seasonal variation and health risk assessment of BTEX in indoor air of homes at different microenvironments of a terai province
of northern India. Chemosphere [Internet]. 2017;176:8–17. Available from: http://dx.doi.org/10.1016/j.chemosphere.2017.02.105

Hosny G, Elghayish M, Noweir K. Health risk assessment for benzene-exposure in oil refineries. 2017;5(1):23–30.

Edokpolo B, Yu QJ, Connell D. Health risk assessment of ambient air concentrations of benzene, toluene and Xylene (BTX) in service station environments. Int J Environ Res Public Health. 2014;11(6):6354–74.

Badjagbo K, Loranger S, Moore S, Tardif R, Sauvé S. BTEX exposures among automobile mechanics and painters and their associated health risks. Hum Ecol Risk Assess. 2010;16(2):301–16.

Hazrati S, Rostami R, Farjaminezhad M, Fazlzadeh M. Preliminary assessment of BTEX concentrations in indoor air of residential buildings and atmospheric ambient air in Ardabil, Iran. Atmos Environ [Internet]. 2016;132:91–7. Available from: http://dx.doi.org/10.1016/j.atmosenv.2016.02.042

72. Environmental Protection Agency. Benzene; CASRN 71-43-2. 2003;1–43.
73. Environmental Protection Agency. Toluene; CASRN 108-88-3. 2005;3:1–33.
74. Epa U. Ethylbenzene; CASRN 100-41-4. 1987;1–20.
75. U.S. Environmental Protection Agency. Xylenes; CASRN 1330-20-7. 2003;1–32.
76. Paustenbach DJ, Bass RD, Price P. Benzene toxicity and risk assessment, 1972-1992: Implications for future regulation. Environ Health Perspect. 1993;101(SUPPL. 6):177–200.
77. Tunsaringkarn, Tanasorn; Tassanee Prueksasit, Mingkwan Kitwattanavong, Watatsit Siriwong, Saowanee Sematong; Kalaya Zapuang AR. Cancer risk analysis of benzene, formaldehyde and acetaldehyde on gasoline station workers. Jounal Environ Eng Ecol Sci. 2012;11(1):1–6.
78. Azuma K, Uchiyama I, Ikeda K. The risk screening for indoor air pollution chemicals in Japan. Risk Anal. 2007;27(6):1623–38.
79. Guo H, Lee SC, Li WM, Cao JJ. Source characterization of BTEX in indoor microenvironments in Hong Kong. Atmos Environ. 2003;37(1):73–82.
80. Jia C, Batterman S, Godwin C. VOCs in industrial, urban and suburban neighborhoods, Part 1: Indoor and outdoor concentrations, variation, and risk drivers. Atmos Environ. 2008;42(9):2083–100.
81. Lee SC, Guo H, Li WM, Chan LY. Inter-comparison of air pollutant concentrations in different indoor environments in Hong Kong. Atmos Environ. 2002;36(12):1929–40.
82. Rao PS, Ansari MF, Gavane AG, Pandit VI, Nema P, Devotta S. Seasonal variation of toxic benzene emissions in petroleum refinery. Environ Monit Assess. 2007;128(1-3):323–8.
83. Gariazzo C, Pelliccioni A, Filippo PDI, Sallusti F, Cecinato A. Compounds Around an Oil Refinery. Saf Heal. 2005;17–38.
84. Lin TY, Sree U, Tseng SH, Chiu KH, Wu CH, Lo JG. Volatile organic compound concentrations in ambient air of Kaohsiung petroleum refinery in Taiwan. Atmos Environ. 2004;38(25):4111–22.
85. Moolla R, Valsamakis SK, Curtis CJ, Piketh SJ. Occupational health risk assessment of benzene and toluene at a landfill site in Johannesburg, South Africa. WIT Trans Built Environ. 2013;134:701–12.
86. Keretetse GS, Laubscher PJ, Du Plessis JL, Pretorius PJ, Van Der Westhuizen FH, Van
Deventer E, et al. DNA damage and repair detected by the comet assay in lymphocytes of African petrol attendants: A pilot study. Ann Occup Hyg. 2008;52(7):653–62.

Mc Donald R, Biswas P. A methodology to establish the morphology of ambient aerosols. J Air Waste Manag Assoc. 2004;54(9):1069–78.

Makonese T, Masekameni D, Annegarn H, Forbes P, Pemberton-pigott C. Domestic Lump-Coal Combustion: Characterization of Performance and Emissions from Selected Braziers. 2012;(SEPTEMBER).

Masondo L, Masekameni D, Makonese T, Annegarn HJ, Mohapi K. Influence of coal-particle size on emissions using the top-lit updraft ignition method. 2016;26(1):15–20.

Makonese T, Masekameni D, Annegarn H, Forbes P. Influence of fuel-bed temperatures on CO and condensed matter emissions from packed-bed residential coal combustion. Domest Use Energy (DUE), 2015 Int Conf. 2015;(October):63–9.