



Health risk characterization for exposure to benzene in service stations and petroleum refineries environments using human adverse response data



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ARTICLE INFO

Article history:

Received 6 April 2015

Received in revised form 25 May 2015

Accepted 2 June 2015

Available online 6 June 2015

Key words:

Benzene exposure

Human epidemiological data

Hazard quotient

Monte-Carlo simulation

Overall risk probability

ABSTRACT

Health risk characterization of exposure to benzene in service stations and petroleum refineries has been carried out in previous studies using guideline values set by various agencies. In this work, health risk was characterized with the exposure data as cumulative probability distribution (CPD) plots but using human epidemiological data. This was achieved by using lowest observable adverse effects levels (LOAEL) data plotted as cumulative probability lowest effects distribution (CPLED). The health risk due to benzene was characterized by using probabilistic methods of hazard quotient (HQ_{50/50} and HQ_{95/5}), Monte-Carlo simulation (MCS) and overall risk probability (ORP). CPD relationships of adverse health effects relationships and exposure data were in terms of average daily dose (ADD) and lifetime average daily dose (LADD) for benzene. For service station environments HQ_{50/50} and HQ_{95/5} were in a range of 0.000071–0.055 and 0.0049–21, respectively. On the other hand, the risk estimated for petroleum refinery environments suggests higher risk with HQ_{50/50} and HQ_{95/5} values ranging from 0.0012 to 77 and 0.17 to 560, respectively. The results of Monte-Carlo risk probability (MRP) and ORP indicated that workers in petroleum refineries (MRP of 2.9–56% and ORP of 4.6–52% of the affected population) were at a higher risk of adverse health effects from exposure to benzene as compared to exposure to benzene in service station environments (MRP of 0.051–3.4% and ORP of 0.35–2.7% affected population). The adverse effect risk probabilities estimated by using the Monte-Carlo simulation technique and the ORP method were found to be generally consistent.

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1. Introduction

Petroleum refinery workers and service station attendants are exposed to benzene due to the emission of volatile aromatic hydrocarbons (VAHs) [45,50]. The evaluation of the health risk resulting from benzene, toluene and xylene (BTX) in service stations and benzene in petroleum refinery environments has been carried out previously by using guideline values developed by various agencies [17]. The results indicated that service station attendants were at a higher risk of adverse health effects with HQ values of 1.4–7.8 than customers using the service stations for 10–30 min per/week, workers in the offices with minimal exposure and people external to the service stations with HQ values of 0.0081–0.27 [16]. The evaluation of health risk for benzene in petroleum refinery

environments suggested a higher risk of adverse health effects for petroleum refinery workers with HQ values of 0.2–210 than people external to the petroleum refineries where HQ was estimated at 0.024 to 0.85 [17].

Probabilistic risk assessment techniques use cumulative probability distribution (CPD) plots and are considered to give a more detailed understanding of the hazard and the associated risks. It also provides additional information indicating the possibility of specific adverse effects. Several probabilistic methods can be used in quantifying the health risks. The hazard quotient method (HQ_{50/50} or HQ_{95/5}) is considered the simplest method. It uses data points from the probabilistic plots to estimate the risk as a ratio of exposure at 50% and 95% cumulative probability (CP) to the adverse effects at 50% and 5% CP levels, respectively. However, information from HQ_{50/50} or HQ_{95/5} does not give the complete risk characteristics of the whole population as HQ is estimated at single point (50% and 95% CP level) in the CPD. HQ_{95/5} represents a conserva-

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Table 1
Benzene inhalation data from service stations and petroleum refinery environments

Scenario	Number of data points	Mean	Standard Deviation	Range (Min–Max)	Reference
Service station environments					
S1 (Attendants)	74	510	570	1.9–2900	[14,43,31,7]
S2 (Mechanics)	10	230	160	50–540	[53]
S3 (Within)	168	21	33	1.1–220	[13,54][35,63,25,11]
S4 (Office)	9	4.7	5.9	1.6–21	[14]
S5 (Customers)	7	1800	1600	150–4900	[18]
S6 (External)	546	15	24	0.7–190	[13,40,63]
Petroleum refinery environments					
R1 (Base Estimates)	95	3600	5800	3.3–28,000	[22,23,34]
R2A (Workers)	66	110	170	0.22–830	[4]
R2B (Workers)	8	17000	24000	3500–78,000	[38]
R3A (Inside)	52	120	290	3.8–2000	[45]
R3B (Inside)	28	14000	12000	4000–65,000	[38]
R4 (Outside)	68	64	110	0.8–580	[45,25,36]

^aNumber of data points represents the affected population in the cumulative probability distribution (CPD) plots.

tive measure of risk since it uses the most exposed population (95% CP level) and the most sensitive sample (5% CP level).

The full range of cumulative probability distribution (CPD) can be used in estimating the risk more comprehensively [42], with other probabilistic methods. Hamidin et al. used the Monte-Carlo simulation (MSC) with exposure and dose adverse effects curves to characterize the health risks due to exposure to chlorinated disinfection by-products in treated drinking water [27]. This method allowed the identification of a number of possible adverse health effects. It was also used by Connell et al. to evaluate the risk of contaminant interference with breeding success of birds [12]. The overall risk probability (ORP) for risk characterization was introduced by Cao et al. [6] and this method has been applied in previous studies of [16,41,60,17,42], in the risk characterisation for exposure to environmental chemicals. The various studies using the MCS and ORP methods have an advantage in that they indicate a number of possible adverse health effects resulting from the exposure.

The aim of this study was to characterize the health risk for exposure to benzene in service stations and petroleum refinery environments by using human adverse effects data, with the probabilistic methods of hazard quotient (HQ_{50/50} and HQ_{95/5}), Monte-Carlo simulation and overall risk probability.

2. Methodology

2.1. Data collection

2.1.1. Exposure data

The exposure data used for the risk characterization were obtained from previous work [16,17]. The data consisted of exposure data for benzene concentrations in air of service stations and petroleum refinery environments sourced from various investigations as reported and presented in Table 1.

The inhalation data sets were grouped into different scenarios and plotted as cumulative probability distributions (CPD) plots by using the following equation.

$$CP(\%) = i/(n + 1) \times 100\% \quad (1)$$

where CP is cumulative probability (%), *i* is the *i*th point in an ascending sequence of the data points, and *n* is the total number of data points.

The scenarios for exposure to benzene through inhalation in service station environments were categorized as follows:

Scenario S1 (attendants)–exposure of service station attendants to benzene concentrations in air

This scenario was for service station attendants dispensing petrol to drivers wearing personal air sampling pumps during their work shift [14,43,31,7].

Scenario S2 (mechanics)–exposure to concentrations of benzene in air for mechanics repairing petrol pumps

This scenario was for exposure to ambient benzene concentrations as 8 h TWA for mechanics repairing and maintaining petrol dispensing pumps [53].

Scenario S3 (internal)–exposure of people to concentrations of benzene in air internal to the service stations

This scenario was for exposure to ambient benzene concentrations at various points within the forecourt of the service station [13,54,63,35,25,11]

Scenario S4 (office)–exposure of workers to concentrations of benzene in air in the offices of service stations

This scenario was for exposure to workers in the offices of service stations by collecting air samples inside the offices of the service stations by using personal air sampling pumps [14].

Scenario S5 (customers)–exposure to concentrations of benzene in air for customers during car refuelling

This scenario was for exposure of customers to ambient benzene concentrations during car refuelling [18].

Scenario S6 (external)–exposure to concentrations of benzene in air for people external to the service stations

This scenario was for benzene concentrations from the service stations in the immediate surroundings giving exposure to people living near service stations [13,40], Terres et al., 2010, [10,30,11].

The scenarios for exposure to benzene through inhalation in petroleum refinery environments were;

Scenario R1 (base estimates)–exposure to benzene as base estimate concentrations for petroleum refinery workers

This scenario was for benzene concentrations collected as base estimate concentrations for retrospective benzene exposures in petroleum industries from studies using similar methods in deriving the base estimates from benzene measurements [22,23,34].

Scenario R2 (workers)–exposure to benzene for petroleum refinery workers

This scenario was for petroleum refinery workers in different occupations within the petroleum refineries exposed to benzene and divided into 2A [4] and 2B [38]. The concentrations of benzene in air were collected by the workers wearing personal air sampling pumps.

Scenario R3 (internal)–benzene concentrations in air internal to the petroleum refineries

The data sets were derived from air samples of benzene taken within various work locations inside the petroleum refineries and grouped as 3A [45] and 3B [38].

Table 2
Reported toxicity data for human exposure to benzene.

Short term exposure in terms of add						
LOAEL ($\mu\text{g}/\text{m}^3$)	ED(day)	EL (day/day)	LOAEL-ADD ($\mu\text{g}/\text{kg}/\text{day}$)	Observed Health Effects	Reference	
6.38×10^7	1	0.01	1.82×10^5	Death	[20]	
1.91×10^5	1	0.1	5.47×10^3	Drowsiness, dizziness, headaches, strong odour, fatigue	[37]	
9.57×10^5	0.5	0.04	1.09×10^4	Drowsiness, headaches, dizziness	[20]	
1.91×10^5	1	0.1	5.47×10^3	Mucous membrane irritation	[37]	
1.91×10^5	21	0.33	1.81×10^4	Mucous membrane irritation, dyspnea	[37]	
Long term exposure—non carcinogenic effects in terms of add						
LOAEL ($\mu\text{g}/\text{m}^3$)	ED(day)	EL (day/day)	LOAEL-ADD ($\mu\text{g}/\text{kg}/\text{day}$)	Observed Health Effects	Reference	
4.79×10^5	5475	0.33 ^a	4.51×10^4	Hematotoxicity– Pancytopenia	[3]	
9.57×10^3	1095	0.33 ^a	9.02×10^2	Hematotoxicity–Anemia, lymphocytosis, leukopenia,	[15]	
6.69×10^5	365	0.33 ^a	6.32×10^4	Hematotoxicity– Pancytopenia, hypercellular bone marrow	[2]	
1.28×10^5	365	0.33 ^a	1.21×10^4	Hematotoxicity–Decrease in white blood cells counts	[8]	
9.25×10^4	6935	0.33 ^a	8.72×10^3	Hematotoxicity–Aplastic, anemia	[59]	
6.38×10^1	7665	0.33 ^a	6.02	Hematotoxicity–Aplastic, anemia	[61]	
7.98×10^4	10585	0.33 ^a	7.52×10^3	Hematotoxicity–Increased mean corpuscular volume	[19]	
1.82×10^3	2190	0.33 ^a	1.71×10^2	Hematotoxicity–Reduced WBC and platelet counts	[33]	
7.21×10^3	3541	0.33 ^a	6.79×10^2	Hematotoxicity–Reduced neutrophils and RBC counts	[44]	
2.42×10^4	2300	0.33 ^a	2.29×10^3	Hematotoxicity–Reduced absolute lymphocyte count	[47]	
6.69×10^5	365	0.33 ^a	6.32×10^4	Immunological– hypoplastic to hyperplastic bone marrow	[2]	
1.28×10^5	365	0.33 ^a	1.21×10^4	Immunological– Decreased lymphocytes	[8]	
3.53×10^4	1852	0.33 ^a	3.33×10^3	Immunological–Anemia, macrocytosis, thrombocytopenia	[24]	
2.39×10^5	9125	0.33 ^a	2.26×10^4	Immunological– Leukopenia	[32]	
6.67×10^5	365	0.33 ^a	6.29×10^4	Immunological– Pancytopenia	[2]	
1.28×10^5	365	0.33 ^a	1.21×10^4	Immunological–Decreased lymphocytes	[8]	
3.51×10^4	1852	0.33 ^a	3.31×10^3	Immunological–Anemia, macrocytosis, thrombocytopenia	[26]	
2.20×10^3	365	0.33 ^a	2.08×10^2	Immunological– Leukopenia	[56]	
6.06×10^3	6205	0.33 ^a	5.71×10^2	Reproductive–increased in chromosome aberration	[5]	
3.19×10^3	2190	0.33 ^a	3.01×10^2	Reproductive–increased in chromosome aberration	[57]	
6.87×10^1	2210	0.33 ^a	6.48	Reproductive–increased in chromosome aberration	[62]	
Long term exposure – carcinogenic effects in terms of add & ladd						
LOAEL ($\mu\text{g}/\text{m}^3$)	ED(day)	EL (day/day)	LOAEL-ADD ($\mu\text{g}/\text{kg}/\text{day}$)	LOAEL-LADD ($\mu\text{g}/\text{kg}/\text{day}$)	Observed health effect	Reference
4.79×10^5	5475	0.33 ^a	4.51×10^4	9.67×10^3	Cancer–Leukemia	[3]
3.19×10^5	2590	0.33 ^a	3.01×10^4	3.05×10^3	Cancer–Leukemia	[9]
9.25×10^4	6935	0.33 ^a	8.72×10^3	2.37×10^3	Cancer– Human lymphocytic leukemia	[59]
3.19×10^3	14,600	0.33 ^a	3.01×10^2	1.72×10^2	Cancer–Leukemia	[1]
9.57×10^2	548	0.33 ^a	9.02	1.94	Cancer– Leukemia	[39]
5.1×10^4	5110	0.33 ^a	4.81×10^3	9.62×10^2	Cancer– Leukemia	[46]
2.74×10^3	2555	0.33 ^a	2.59×10^2	2.58×10^1	Cancer– Acute myeloid leukemia	[66]
5.1×10^4	5110	0.33 ^a	4.81×10^3	9.62×10^2	Cancer– Leukemia	[28]
6.87×10^3	365	0.33 ^a	6.48×10^2	9.25	Cancer– Chronic erythroid leukemia	[58]
6.38×10^5	10950	0.33 ^a	6.02×10^4	2.58×10^4	Cancer– Leukemia	[55]
3.19×10^4	3650	0.33 ^a	3.01×10^3	4.29×10^2	Cancer– Leukemia	[28]

LOAEL – Lowest observed adverse effects levels.

ED – Exposure duration (day).

EL – Exposure length (day/day).

LT – Lifetime (70 years \times 365 days = 25,550 day).

IR – Inhalation rate ($20 \text{ m}^3/\text{day}$).

BW – Body weight (70 kg).

^a 0.33 day/day (8 h/d) is assumed to be equivalent occupational exposure.

Scenario R4 (external)–benzene concentrations in air external the petroleum refineries

The data sets obtained for this scenario were for emissions of benzene from petroleum refineries to the immediate surroundings giving exposure of people living near the petroleum refineries [45,25,36].

Both the exposure data and the adverse effects data are based on inhalation exposure. Hence, it is most suitable to use the inhalation dose for risk assessment in this study. The exposure data from concentration in air ($\mu\text{g}/\text{m}^3$) were converted into inhalation dose in terms of ADD and LADD ($\mu\text{g}/\text{kg}/\text{day}$) by following the method recommended by the USEPA [52] as shown in Eqs. (2) and (3), respectively. The inhalation rate (IR) used was $20 \text{ m}^3/\text{day}$ and the absorption rate of the inhaled benzene was assumed to be 100%.

$$\text{ADD}(\mu\text{g}/\text{kg}/\text{day}) = \frac{[\text{C} \times \text{IR} \times \text{EL}]}{[\text{BW}]} \quad (2)$$

$$\text{LADD}(\mu\text{g}/\text{kg}/\text{day}) = \frac{[\text{ADD} \times \text{ED}]}{[\text{LT}]} \quad (3)$$

where ADD and LADD is inhaled dose ($\mu\text{g}/\text{kg}/\text{day}$); C, concentration in air ($\mu\text{g}/\text{m}^3$); IR, inhalation rate (m^3/day); BW, body weight (kg); ED, exposure duration (days); EL, exposure length (day/day); LT, lifetime exposure (day). The values for the quantities used in the equations are given in Table 2. For example, the exposure concentration of $479,000 \mu\text{g}/\text{m}^3$ [3] were converted into inhalation doses as follows:

In terms of ADD,

$$\text{ADD}(\mu\text{g}/\text{kg}/\text{day}) = \frac{\left[\frac{479000 \mu\text{g}}{\text{m}^3} \times \frac{20 \text{ m}^3}{\text{day}} \times \frac{0.33 \text{ day}}{\text{day}} \right]}{70 \text{ kg}} = 45,200 \mu\text{g}/\text{kg}/\text{day} \quad (4)$$

In terms of LADD,

$$\text{LADD}(\mu\text{g}/\text{kg}/\text{day}) = \frac{[45,163 \mu\text{g}/\text{kg}/\text{day} \times 5475 \text{ day}]}{[25,550 \text{ day}]} = 9680 \mu\text{g}/\text{kg}/\text{day} \quad (5)$$

The life time exposure was evaluated as lifetime average exposure (LAE) in terms of LADD which was estimated from the average daily dose.

Table 3
Health risk outcomes derived for ORP, MRP and HQ_{95/5}.

Scenario	STE (ADD) {1–28 days (<1 h/day)},				LTE (ADD) {>6months 8 h/day}				LAE (LADD) (70 years)			
	ORP (%)	MRP (%)	HQ _{50/50}	HQ _{95/5}	ORP (%)	MRP (%)	HQ _{50/50}	HQ _{95/5}	ORP (%)	MRP (%)	HQ _{50/50}	HQ _{95/5}
Service station environments												
S1 (attendants)	1.7	0.78	0.0012	0.55	17	15	0.0038	8.8	2.7	3.4	0.055	21
S2 (mechanics)	0.57	0.34	0.00093	0.27	12	7.9	0.0029	4.4	2.1	2.4	0.041	7.5
S3 (within)	0.35	0.15	0.000024	0.23	6.7	3.2	0.000074	3.8	1.3	0.96	0.0011	0.72
S4 (office)	0.28	0.043	0.000071	0.0049	2.2	0.93	0.00022	0.078	0.57	0.30	0.0031	0.23
S5 (customers)	0.35	0.086	0.000029	0.037	4.4	1.8	0.000094	0.59	0.57	0.15	0.0013	0.31
S6 (external)	0.35	0.051	0.000036	0.0061	2.8	1.3	0.00011	0.096	0.57	0.18	0.0016	0.68
Petroleum refinery environments												
R1 (base estimates)	15	13	0.10	9.2	22	30	0.31	150	38	37	4.5	120
R2A (workers)	6.5	3.4	0.0077	0.92	9.5	7.5	0.024	14	17	29	0.34	5.0
R2B (workers)	29	23	1.8	24	47	49	5.5	375	52	56	77	560
R3A (inside)	6.5	3.3	0.0023	0.31	9.5	6.9	0.0071	5.0	17	28	0.10	4.7
R3B (inside)	27	20	1.1	19	44	47	3.5	310	47	52	51	380
R4 (outside)	4.6	2.9	0.0012	0.17	6.4	3.8	0.0039	2.7	11	18	0.055	2.3

LOAEL–ADD – Lowest observed adverse effects average daily dose.

LOAEL–LADD – Lowest observed adverse effects lifetime average daily dose.

2.1.2. Human epidemiological data on lowest observable adverse effects levels (LOAEL)

Adverse health effect data from epidemiological studies on human populations were obtained from the scientific literature and used for risk characterization as summarized in Table 2. From each data set, the lowest observable adverse effects level (LOAEL) corresponding to adverse health effects was evaluated and the LOAEL data were plotted as cumulative probability distributions. The LOAEL data were categorized into short-term {1–28 days (<1 h/day)}, long-term {>6months (8 h/day)} and lifetime average exposure (70 years). It was converted to LOAEL–ADD for short-term exposure (STE), LOAEL–ADD for long-term exposure (LTE) with Eq. (2) and LOAEL–LADD for lifetime average exposure (LAE) to benzene with Eq. (3). These data sets were then used in developing cumulative probability of lowest effects distribution (CPLED) by using Equation (1).

The LOAEL data for benzene is generally derived from biological observations on human populations from epidemiological studies. Different LOAEL values were reported from the various investigations as a result of the different experimental designs, different adverse effects and different population size. However, the LOAEL data presented in Table 2 can be collated to give support to general relationships between the dose of benzene and the observed adverse effects. The LOAEL data on benzene can be organized into a sequence of exposures and adverse effects of increasing magnitude in developing a probabilistic plot. However, the probabilistic plots could be expected to show deviations from the normal distribution since the data are of diverse sources [64].

2.2. Methods for risk characterization based on probabilistic techniques

Three probabilistic methods were used in the risk characterization for exposure to benzene. The first method used for evaluation of the risk was the hazard quotient with HQ_{50/50} and HQ_{95/5}. The HQ_{50/50} was calculated as the ratio of the median exposure dose at 50% CP to the median dose (50% CP) of adverse effects; thus, this represents the evaluation of health risk for the median population exposed. The HQ_{95/5} was calculated by using a highly exposed group in the population at 95% CP (or 5% the most exposed) and the sensitive group of the population at 5% CP of adverse effect curve. It is therefore, a conservative evaluation of the health risk.

The second method used was the Monte-Carlo simulation technique. The hazard quotient (HQ_{MC}) was obtained as a probability density. A process of repeated simulations was used to calculate

the HQ at random values for 10,000 times giving the probability distribution density (PDD) for the whole population [65]. From the PDD, the Monte-Carlo risk probability (MRP) was estimated as the percentage probabilities of HQ > 1 in the distribution.

The third method used was the of overall risk probability method [6]. The ORP curve is the plot of exposure exceedance values in cumulative percentage against affected population also in cumulative percentage. The overall risk probability of adverse effects is estimated as the area under the ORP curve. The ORP was estimated as the area under the curve of the relationships between exposure exceedance values and affected population [6].

3. Risk characterization for exposure to benzene in service station and petroleum refinery environments

3.1. CPD relationship of LOAEL for human health effects

The LOAEL data include data for a range of different types of human health effects such as haematotoxicity, immunotoxicity, genotoxicity, and cancer (Table 1). This data can be collected and organized into a sequence of adverse effects from the lowest non-lethal effects to lethal effects. The LOAEL data from human epidemiological studies were used in developing CPLED plots for short term, long term and lifetime exposure [16]. The evaluation of the LOAEL data as CPLED relationships are shown in Figs. 1 and 2 (LOAEL–ADD and LOAEL–LADD). They have correlation coefficients ranging from 0.90 to 0.94 and are considered suitable for use in risk characterization of benzene.

3.2. Hazard quotients calculated at HQ_{50/50} and HQ_{95/5}

3.2.1. Hazard quotients (HQ_{50/50} and HQ_{95/5}) for service station environments

Benzene inhalation dose for attendants (S1), mechanics (S2), inside the service station (S3), office of service stations (S4), and customers (S5) as well as outside the service stations (S6) were plotted as exposure CPD plots in Fig. 1. In addition the CPLED relationships (LOAEL evaluation of adverse effects) for short term (STE), long term (LTE) and lifetime average exposure (LAE) to benzene from human epidemiological studies are shown calculated as the ADD and LADD (LOAEL–ADD and LOAEL–LADD).

The calculated HQ_{50/50} and HQ_{95/5} values were presented in Table 3 for STE, LTE and LAE. HQ_{50/50} was < 1 for all the service station Scenarios (S1–S6) suggesting minimal or no risk of adverse effects for the median population for STE, LTE and LAE. Also, the

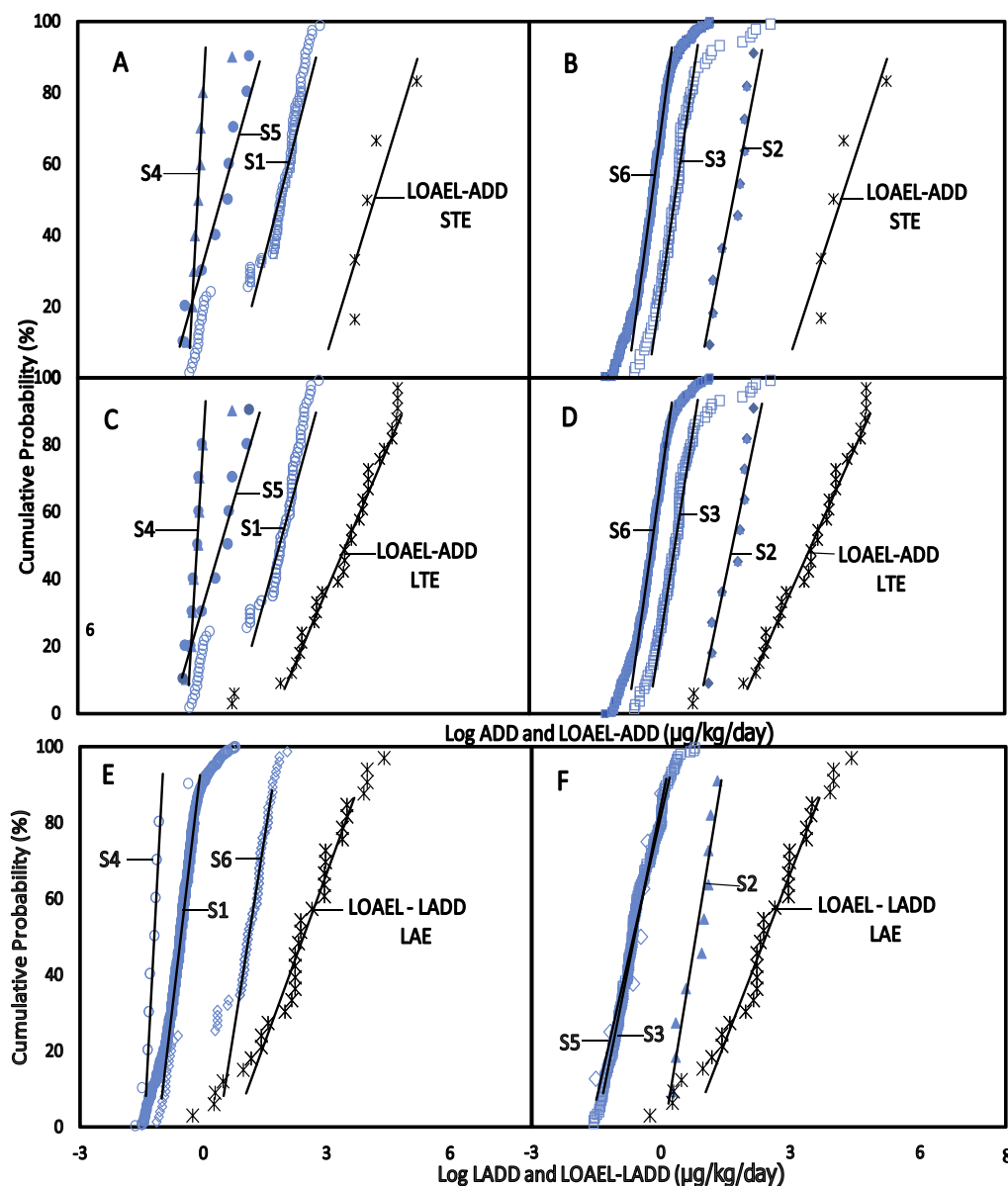


Fig. 1. Math Image deleted from here. CPD for exposure scenarios for benzene in service station environments (S1–S6 see Section 2.1) with corresponding CPLED adverse health effects for short term exposure [LOEL – ADD (A and B)]; long term exposure [LOEL – ADD (C and D)]; lifetime average exposure [LOEL – LADD (E and F)].

estimated $HQ_{95/5}$ for S1–S6 was <1 for STE (<1 h/day). This suggests minimal or no risk for STE to benzene in the service station environments. $HQ_{95/5}$ was <1 for Scenarios S4, S5 and S6 for LTE and Scenarios S3, S4, S5 and S6 for LAE suggesting no risk of adverse effects for the highly exposed group. However, $HQ_{95/5}$ for LTE was >1 with values of 8.8, 4.4 and 3.8 for Scenarios S1, S2 and S3, respectively. For LAE with benzene $HQ_{95/5}$ of 21 and 7.5 were estimated for Scenarios S1 and S2 respectively. This implies potential risk of adverse effects summarized in Table 3 for LTE and lifetime average exposure to benzene for the scenarios with $HQ > 1$ for the highly exposed group (5% of the population).

HQ was previously estimated for lifetime exposure to benzene in terms of LADD at 50% and 95% CP for service stations and using United State Environmental Protection Agency (USEPA) Reference Dose (RfD) [16]. In comparing the HQ values of [16] to this study, $HQ_{50/50}$ and $HQ_{50/RfD}$ (in terms of LADD) with service station scenarios were in range of 0.0011–0.055 and 0.0081–1.4, respectively. $HQ_{95/5}$ and $HQ_{95/RfD}$ for service station scenarios were

0.023–21 and 0.084–7.8, respectively. This suggests a higher risk of adverse effects with human data than with USEPA RfD for the highly exposed (95% CP). On the other hand at 50% CP the estimated HQ values suggests higher risk of adverse effects with USEPA RfD than with human data.

3.2.2. Hazard quotients ($HQ_{50/50}$ and $HQ_{95/5}$) for petroleum refinery environments

The exposure dose estimated for Scenarios R1 (base estimates), R2A and R2B (workers), 3A and R3B (internal) and R4 (external) were plotted as CPD relationships together with CPLED of adverse effects (LOEL evaluation) for STE, LTE and LAE to benzene and shown in Fig. 2. From the calculated $HQ_{50/50}$ and $HQ_{95/5}$ (Table 3), $HQ_{50/50}$ values of 1.8, 5.5 and 77 were estimated for Scenario R2B and 1.1, 3.5 and 51 for R3B in terms of STE, LTE and LAE, respectively, suggesting risk of adverse effects for the median population. On the other hand, $HQ_{50/50} < 1$ were estimated for STE and LTE for Scenario R1. But then $HQ_{50/50}$ of 4.5 was estimated for LAE suggesting risk

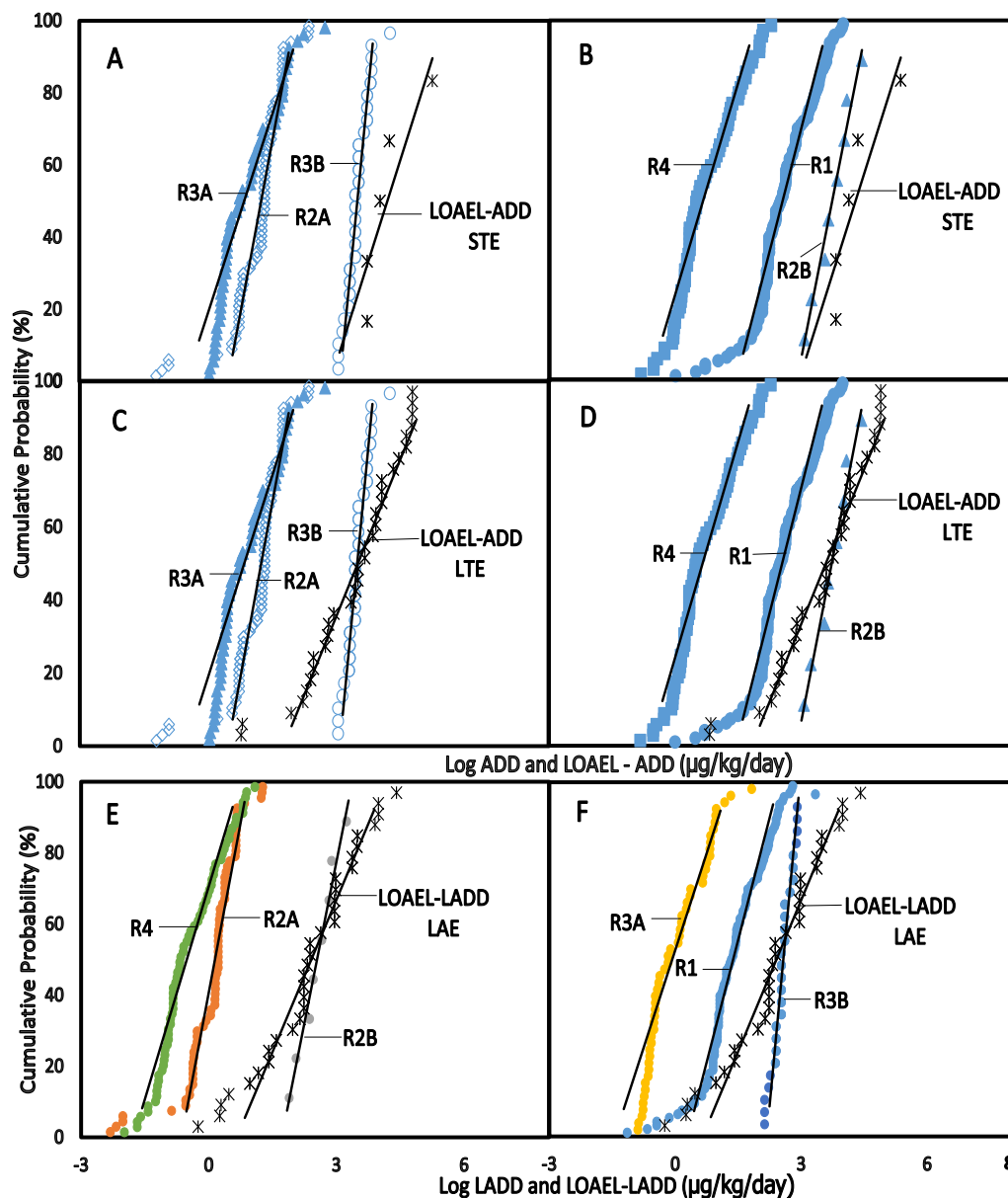


Fig. 2. CPD for exposure scenarios for benzene in petroleum refinery environments (R1, R2A,B, R3A, B, R4 see Section 2.1) with corresponding CPLED adverse health effects for short term exposure [LOEL – ADD (A and B)]; long term exposure [LOEL – ADD (C and D)]; lifetime average exposure [LOEL – LADD (E and F)].

of adverse effects for lifetime to benzene in Scenario R1. However, minimal or no risk was estimated for Scenarios R2A, R3A and R4 with $HQ_{50/50} < 1$ for STE, LTE and LAE for the median population. These results suggest minimal or no risk for STE (<1 h/day) exposure to benzene for the affected population in Scenario R2A, R3A and R4. For LTE (8 h/day) and LAE (70 years) to benzene, $HQ_{95/5} > 1$ (2.3–560) for all the Scenarios (R1, R2A, R2B, R3A, R3B and R4) suggesting risk of adverse effects (Table 3) for the affected highly exposed population in all the scenarios exposed to benzene for long term and lifetime.

The HQ values estimated at 50% and 95% for lifetime exposure (LADD) in this study was compared to a previous study by [17], that evaluated the risk of adverse effects in terms of LADD using USEPA RfD. $HQ_{50/50}$ and $HQ_{50/RfD}$ for petroleum refinery scenarios were in range of 0.055–77 and 0.024–43, respectively. $HQ_{95/5}$ and $HQ_{95/RfD}$ were 2.3–560 and 0.85–210, respectively. This suggests a higher risk of adverse effects by using human data than with USEPA RfD at 50% and 95% CP, respectively.

3.3. Distribution of hazard quotients from Monte-Carlo simulation

3.3.1. Hazard quotient distribution from Monte-Carlo simulation (HQ_{MC}) for service station environments

CPD plots for Scenarios S1–S6 of exposure to benzene in service station environments and the CPLED plots (LOEL evaluation of adverse effects) plots (Fig. 1) were used in the Monte-Carlo simulation. The HQ_{MC} (Fig. 3A) was <1 for most of the population for STE (<1 h/day), LTE (8 h/day) and LAE (70 years). The percentage of the population with $HQ_{MC} > 1$ is presented in Table 3 as the Monte-Carlo risk probability (MRP). LTE for Scenario S1–S6 has the highest MRP (2.2–17%) as compared to STE (0.043–0.78%) and LAE (0.23–2.1%).

The highest MRP value was for attendants (S1) at 17%. This suggests that 17% of the exposed group in S1 have a risk of adverse effects from long term exposure to benzene as compared to the lowest Monte-Carlo risk probabilities value of 0.043% of the exposed group in S4 (Table 3).

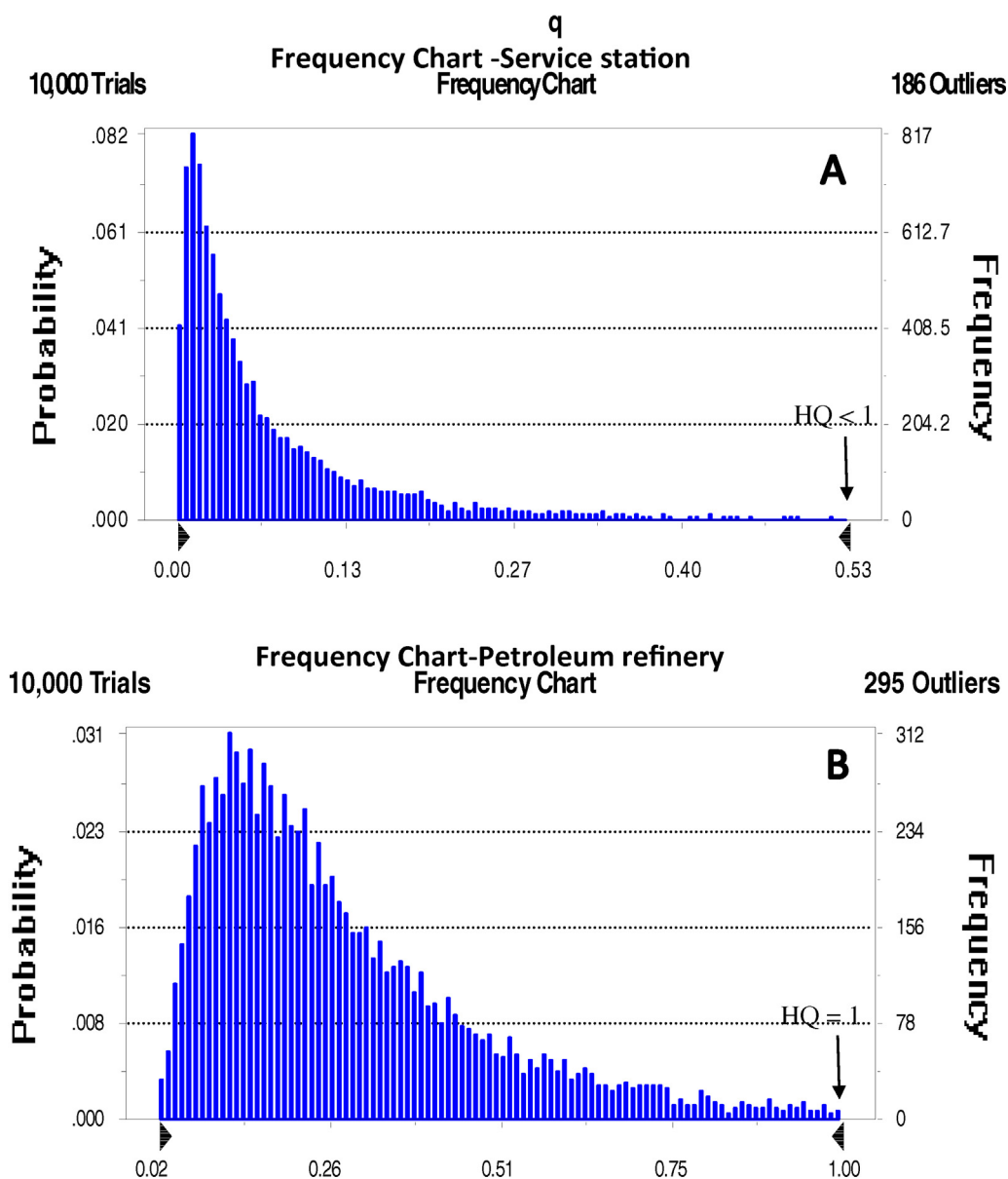


Fig. 3. Examples of simulated probability distribution density for A, service station attendants (Scenario S1) exposure to benzene evaluated for STE and B, petroleum refinery workers exposure to benzene (Scenario R2A) evaluated for STE using human data.

3.3.2. Hazard quotient distribution from Monte-Carlo simulation (HQ_{MC}) for petroleum refinery environments

CPD plots made for exposure for Scenario R1, R2A,B, R3A,B, and 4R together with CPLED plots of adverse effects (Fig. 2) were used in developing HQ_{MC} probabilistic distribution density (PDD) (Fig. 3B). The MRP values are presented in Table 3. The maximum MRP was in terms of LADD for Scenarios R2B and R3B were 56% and 52%, respectively, implying that the exposed population were at risk of adverse effects for lifetime average exposure to benzene. $MRP > 1$ was estimated for lifetime exposure in Scenarios R1–R4 with values ranging from 18 to 56% (Table 3).

These results imply that the magnitude of risk of adverse effects is from moderate for STE (2.9–23%) and LTE (3.8–49%) to high level of risk of adverse effects for lifetime average exposure to benzene (18–56%) for the affected population in petroleum refinery environment.

3.4. Overall risk probability

3.4.1. Overall risk probability for service station environments

The CPD plots of exposure for Scenarios S1–S6 and the CPLED plots of adverse effects were used in developing ORP curves as shown in Fig. 4. The ORP of adverse health effects with human data for exposure to benzene in S1–S6 were estimated (Fig. 4). In general, the ORP values for STE and LAE (<2.7%) were lower than the ORP values for LTE (<17%) for S1–S6 as shown in Table 3, suggesting less than 17% of the affected population were at risk of adverse health effects for LTE. This implies that about 17% of attendants (S1), 15% mechanics (S2) and 12% of exposed population inside the service stations (S3) were at risk of adverse effects. While less than 2.7% of exposed population in all the scenarios for STE and LAE were at risk of adverse health effects which is mainly leukaemia.

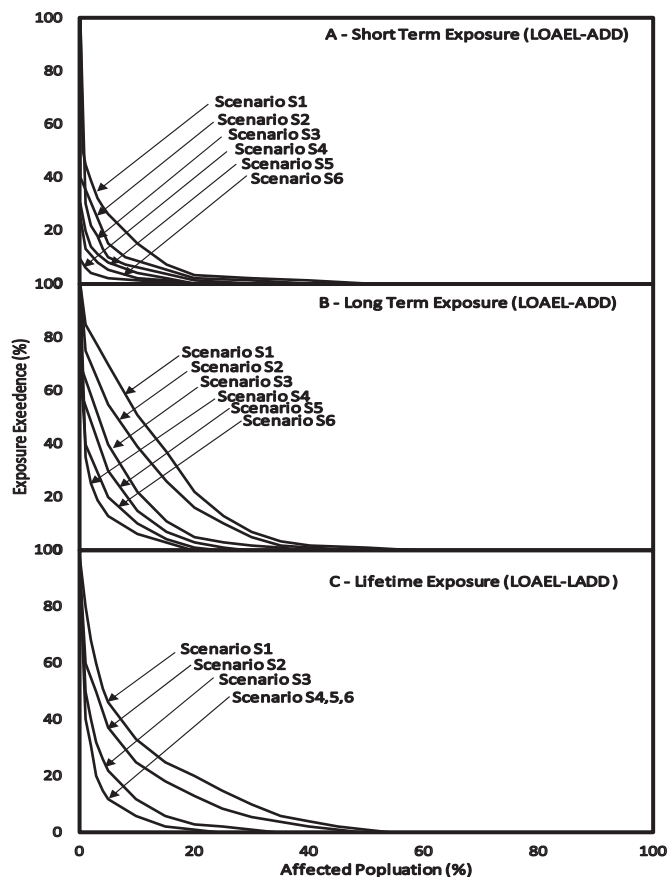


Fig. 4. Overall risk probability curve derived for A, short term; B, long term and C, lifetime exposure to benzene in service station environments (see Fig. 1).

3.4.2. Overall risk probability for petroleum refinery environments

The CPD plots for Scenarios R1–R4 and the CPLED plots of adverse effects (LOAEL evaluation) were used in developing ORP curves shown in Fig. 5. The ORP of adverse health effects with human data for R1–R4 are presented in Table 3 for STE, LTE and LAE. Higher values of ORP for adverse effects were recorded for Scenarios R2B (workers) and R3B (Internal) as the percentage of affected population. As shown in Table 3, close to half of the affected population in Scenarios R2B and R3B would be expected to be at risk of adverse health effects for LTE and LAE. Generally, higher percentage values of ORP were observed for lifetime exposure (11–52%) than LTE (6.4–47%) and STE (4.6–29%). This implies that higher level of risk of cancer and non-cancer effects will be expected for LAE to benzene for the affected population in Scenarios R2B and R3B than risk for STE and LTE in all the Scenarios (R1–R4).

4. Overall health risk characterization

The risk characterization values with the service station environment from the three methods indicated higher risk of adverse effects for attendants (S1) ($HQ_{50/50}$ 0.00071–0.055, $HQ_{95/5}$ 0.55–21, MRP 0.78–15 and ORP 1.7–15) followed by mechanics (S2) and exposed population inside the service stations (S3) for service station environments (Table 3). Although, low risk of adverse effects were observed for workers in the offices of service stations (S4), customers (S5) as well as people external to the service station (S6) for STE and LTE exposure to benzene. However, lifetime exposure indicated potential risk of adverse health effects in all the scenarios (Table 3).

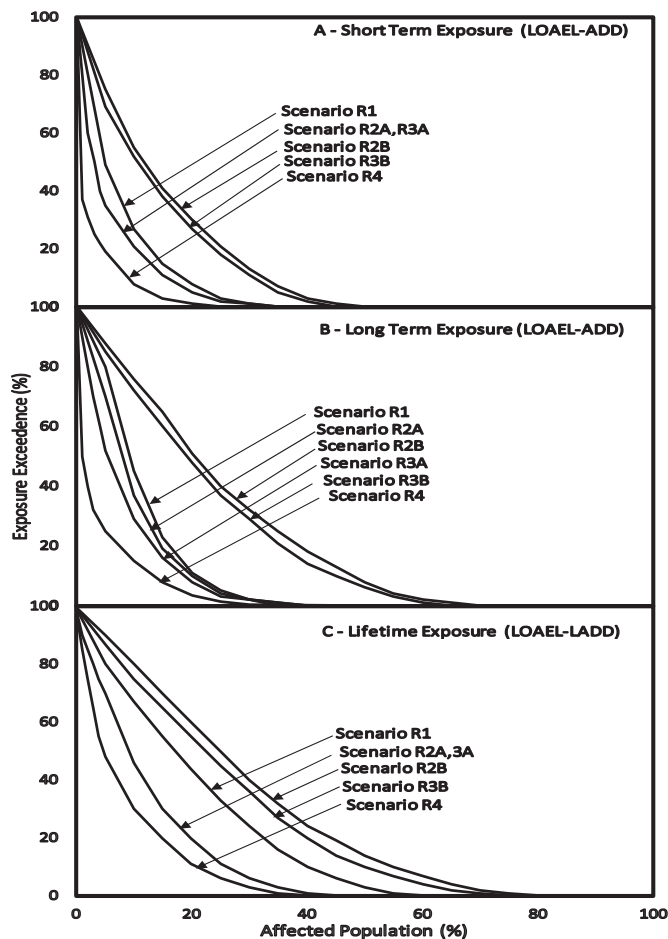


Fig. 5. Overall risk probability curve derived for A, short term; B, long term and C, lifetime exposure to benzene in petroleum refinery environments (see Fig. 1).

For petroleum refinery environments, higher health risks were estimated for petroleum refinery workers (R2B) ($HQ_{50/50}$ 1.8–77, $HQ_{95/5}$ 24–560, MRP 23–56 and ORP 29–52) and exposure to benzene concentrations inside the petroleum refinery R3B ($HQ_{50/50}$ 1.1–51, $HQ_{95/5}$ is 19–380, MRP 20–52 and ORP 27–47) in Bulgaria. This was followed by base estimate exposure (R1) from refineries in Australia, Canada and United Kingdom. The risk values for R2A (workers in Italian refinery) and R3A (internal, Indian refinery) were effectively the same but higher than R4 (external, Indian and Italian refineries).

5. Evaluation of $HQ_{50/50}$, $HQ_{95/5}$, MRP and ORP methods

A summary of the $HQ_{50/50}$, $HQ_{95/5}$, MRP and ORP values are presented in Table 3 estimated for exposure to benzene in service station and petroleum refinery environments. The $HQ_{95/5}$ method estimates the risk of adverse effects for not only the highly exposed population group (95% CP) but also the most sensitive population group to adverse health effects (5%CP, LOAEL). Furthermore, the risk of adverse effects is difficult to interpret with the $HQ_{95/5}$ for the total exposed population. However, $HQ_{50/50}$ can be compared to the MRP and ORP since it represents the median group in the population.

The MRP and ORP methods take into account the exposure for all the population in estimating the risks of adverse effects at all sensitivity levels. Thus, estimating risk of adverse effects with the MRP and ORP methods give the quantitative risk value for all the exposed groups in the population at all sensitivities. With the ORP method,

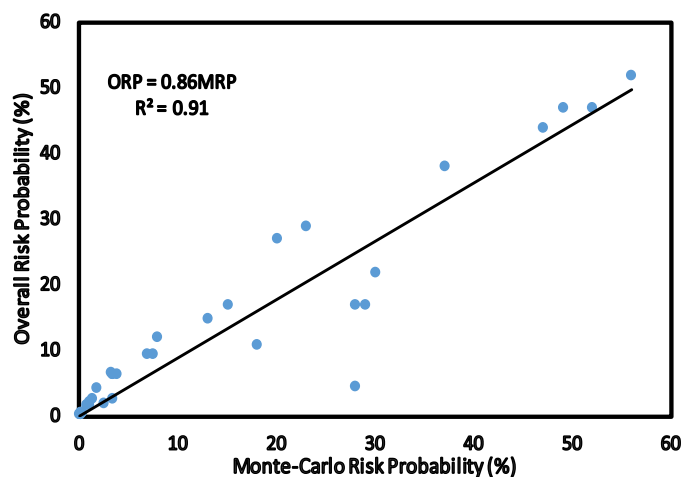


Fig. 6. Relationship between overall risk probability of affected population (%) and Monte-Carlo risk probability (MRP) of HQ greater than unity (%) for exposure to benzene in service stations and petroleum refinery environments.

the area under the curves (Figs. 4 and 5) gave ORP values (Table 3) that can be interpreted as the percentage of affected population at risk of adverse effects. MRP provides the risk probability (percentage probability in Table 3). Both methods can be used to estimate non-cancer and cancer effects for exposure to benzene. However, the advantage of using the $HQ_{50/50}$ and $HQ_{95/5}$ is that these measures can be used to evaluate the risk for specific groups such as the highly exposed group (5% CP) and median exposed group (50%CP) of the exposed population.

6. Comparison of MRP and ORP

Fig. 6 is an evaluation of the relationship between the MRP and the ORP values. If the risk characterization were a perfect relationship, there would be a slope of unity and R^2 value of unity. From the linear relationship (Fig. 6), of the ORP and MRP values using values from Table 3 for exposure to benzene in service station and petroleum refinery environments. A good correlation with R^2 of 0.91 was obtained for the relationship as well as a slope of 0.86. The results, suggest that the risk probabilities of non-cancer and cancer effects estimated by using MRP and ORP for the whole population are relatively consistent.

The relationship between ORP and HQ is presented in Fig. 7. The ORP is a method of estimating the risk for the whole population as compared to the HQ that is single point representing risk at a particular CP level. However, the ORP values can be compared to the $HQ_{50/50}$ values since both methods involve the main exposed group in the population. This suggests, that the simple $HQ_{50/50}$ may provide a reasonable evaluation of the risk to the whole population. However, this does not take into account deviations from a normal distribution in the data for both exposure and the CP for adverse effects. The most accurate method for evaluation of health risk for the whole of a population is the ORP since this accounts for deviations for a normal distribution.

7. Conclusions

Human health risks for exposure to benzene in service station and petroleum refinery environments were characterized by using human adverse response data and three probabilistic methods of hazard quotient ($HQ_{50/50}$ and $HQ_{95/5}$), Monte-Carlo simulation and ORP (overall risk probability). The results indicated that workers in petroleum refineries ($HQ_{50/50}$ 1.1–77, $HQ_{95/5}$ 19–560, MRP 20–56% and ORP 27–52%) have a higher risk of adverse health

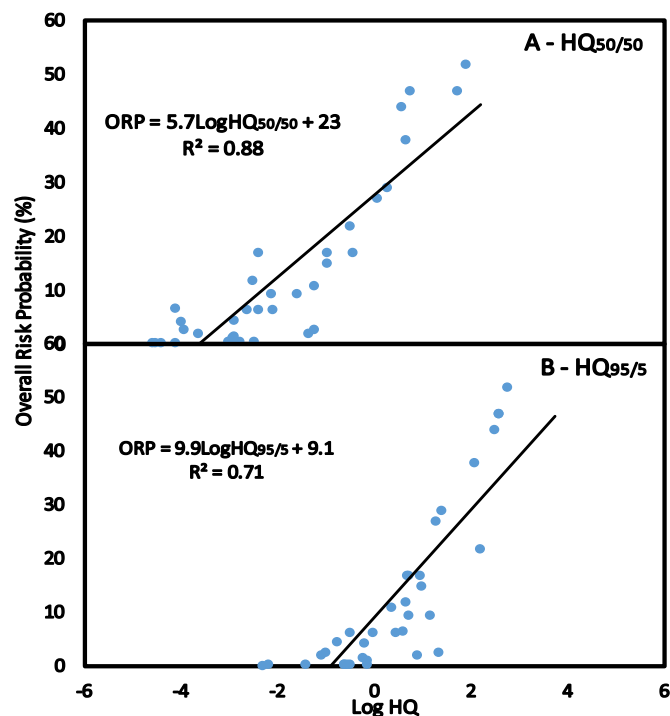


Fig. 7. Relationship between overall risk percentage probability (ORP) of affected population (%) and hazard quotient A, 50%CP ($HQ_{50/50}$) and B, 95%CP ($HQ_{95/5}$) for exposure to benzene in service stations and petroleum refinery environments.

effects from exposure to benzene as compared to exposure to benzene in service station environments. The risks of adverse effects from benzene exposure for customers during car refueling, workers in the offices of service stations and people external to service stations and petroleum refineries were found to be low ($HQ_{50/50}$ and $HQ_{95/5} < 1$). In the service station environments, ORP was low for STE and LAE (0.28–2.1%) as compared to risk probabilities for LTE to benzene (2.2–17%) and the values were similar to MRP (0.043–3.4% for STE, 0.93–15% for LTE and 0.15–3.4% for LAE). On the other hand, in petroleum refinery environments, higher ORP of affected population likely to have health effects for LAE (11–52%) as compared to STE (4.6–29%) and LTE (6.4–47%) for exposure to benzene. The MRP values were also similar at 2.9–13% for STE, 3.8–30% for LTE, 18–37% for LAE. The results of ORP and MRP obtained for various scenarios were generally in good agreement for service stations and petroleum refinery environment for risk characterization.

In comparing the HQ values for lifetime exposure from this study with previous studies conducted on service stations and petroleum refinery environments, the USEPA RfD was used as a reference point to measure the potential effects of benzene at different exposure dose. $HQ_{50/50}$ and $HQ_{95/5}$ method is evaluated at the 5% and 50% CP threshold dose that relates to adverse health effects observed in the sensitive group and main group respectively of the human population as observed in epidemiological studies.

Acknowledgments

The authors are grateful for the financial support from Delta State Government, Nigeria and Griffith University Brisbane, Queensland, Australia (scholarship for doctoral study).

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