Human Health Risk Assessment

Abstract

Risk assessment has been performed in three typical exposure scenarios in Kolkata Metropolitan city due to exposure towards a few mono-aromatic hydrocarbons. Cancer risk estimated from the exposure of ambient level with general people taken as target population or receptors was found to be higher (ranging between 3.0E+05 and 8.9E+06) than acceptable, although the non-cancer health risk was found to be within acceptable limit. Carcinogenic health risk due to exposure of benzene and ethylbenzene level in typical urban residential indoor, generally inhabited by middle and lower middle income group, was also found sufficiently higher (2.9E-05) than the acceptable risk of 1.0E-06 for 15 years of exposure.

The occupational exposure and corresponding risk values for the sub-population of petrol pump workers (engaged in filling work) and passenger car drivers were found to be very high. The integrated lifetime cancer risks (ILCR) due to two carcinogens, namely benzene and ethylbenzene, were calculated to be 1.46E-04 and 4.28E-05 respectively. Overall Hazard Index due to chronic exposure to BTEX was calculated to be 1.7 and 1.1 for petrol pump workers and car drivers respectively indicating chance of chronic health effect for the exposed population.

The carcinogenic risk estimated using external exposure value using conventional method was found to be much less for the occupational groups than the same estimated from internal dose. The internal dose or body burden reflects the overall exposure through all the exposure routes, nature of exposure and individual physiological variability, which made it a superior indicator of individual susceptibility to benzene toxicity.
6.1 Introduction

Intuitive risk assessment and risk management have been fundamental for human survival and evolution. Those who appreciated risk were more likely to survive and reproduce whereas those who could not were more likely to perish from environmental hazards (Thomas and Hrudey, 1997).

A specific quantitative concept of 'risk' estimation based on probability only was developed in late seventeenth century. For prediction of risk, there are various factors to be considered: (a) a hazard (the source of danger); (b) an uncertainty of occurrence and outcomes (expressed by probability distributions); (c) possible adverse health outcomes; (d) a target; (e) a time frame; and (f) the importance of the risk for people affected by it (Thomas and Hrudey, 1997).

Risk assessment is the process of estimating the potential impact of a chemical, physical, microbiological or psychosocial hazard on a specified human population or ecological system under a specific set of conditions and for a certain timeframe. It provides a systematic approach for characterizing the nature and magnitude of the risks associated with environmental health hazards. The ultimate aim of risk assessment is to provide the best possible scientific, social and practical information about the risks, so that these can be discussed more broadly and the best decisions could be made.

The procedure of risk assessment may not always provide a compelling or definitive outcome. There are limitations of risk assessment procedure such as:

- Often the default values and assumptions made during the process are not sufficiently realistic, which may lead to an overestimation or underestimation;
- Proper consideration of the effect of multiple agents and the individual susceptibility may not be possible;
• The use of default values and assumption may become too rigid so that situation-specific data are not applied;
• The uncertainties of risk assessment are often inadequately described;
• There may be an emphasis on cancer risk while the possibility of other adverse effects e.g. reproductive and developmental outcomes may be neglected;
• In some situations there may be insufficient scientific knowledge to be able to perform credible risk assessments.

Air pollution and its public health impacts are drawing increasing concern from the environmental health research community, environmental regulatory agencies, industries, as well as the public. The use of risk assessment as a tool in the decision-making process has become increasingly important over recent years. Compared with the large volume and varieties of studies carried out in the developed countries, risk assessment studies in developing nations are relatively scarce. Despite the revised emission standards and technical improvement in pollution control measures, expanding industrialization and increasing traffic volumes in the developing countries is drastically increasing emissions of many air pollutants which amplify the necessity of such study in representative scenario.

The human health risk assessment process includes consideration of risks to individuals and populations, including sensitive sub-populations, (e.g., children, pregnant women, the elderly and individuals with respiratory impairments). This includes four discrete steps: hazard identification, dose-response assessment, exposure assessment, and risk characterization (USEPA 1997).

1. The hazard identification: This step defines exposure conditions and catalogs environmental stressors (i.e., pollutants), receptors i.e. the human sub-population, and pathways by which receptors contact stressors i.e. the routes of exposure. Hazard
identification is to recognize the conditions that may cause adverse effects, which may be acute (e.g., nausea or headaches), chronic (e.g., emphysema or central nervous system depression) or life-threatening (e.g., central nervous system disability or cancer). The nature of hazards caused by a single, repeated or lifelong exposure to pollutant(s) may be carcinogenic, non-carcinogenic or both. Carcinogenic hazards describe the development of any type of cancer in any part of the body. All health effects other than cancer are classified under this hazard category.

2. The dose-response assessment: This step establishes a relationship between the extent of contact with a stressor via a specific route (e.g. oral, inhalation, dermal) and the nature and severity of adverse effects that result. Dose-response information of a pollutant can be obtained from epidemiology studies as well as animal studies.

Dose-response for non-carcinogens: For non-carcinogenic effect, toxicants have a "threshold dose" below which the body's defensive mechanisms protect against adverse health effects. Exposure to the contaminant at sub-threshold doses is less likely to cause harm. The more an exposure exceeds the threshold, the more likely and severe the health effect is likely to be. According to USEPA, a "reference dose" (RfD) is a daily dose, in milligrams (mg) of contaminant per kilogram (kg) of body weight per day, which is expected to be without adverse effect to human populations, including sensitive individuals (USEPA 1997). The uncertainty surrounding the RfD is expected to be an order of magnitude or more. Discrete RfDs may be calculated for chronic (lifetime), sub-chronic (long term but less than lifetime), and acute (short term; hours or days) exposure periods.

Dose-response for carcinogens: Carcinogenic health effects do not have a simple dose-response relationship. Instead, they are considered to pose an incremental risk of
causing cancer for each exposure increment above zero. Thus carcinogenic contaminants have no threshold limit for effects.

In assessing carcinogenic potential of a chemical, the USEPA classifies the chemical according to the credence of evidence from epidemiologic and animal studies, as follows:

- **Group A** - Human Carcinogen (sufficient evidence of carcinogenicity in humans)
- **Group B** - Probable Human Carcinogen (B1-limited evidence of carcinogenicity in humans; B2-sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
- **Group C** - Possible Human Carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data)
- **Group D** - Not Classifiable as to Human Carcinogenicity (inadequate or no evidence)
- **Group E** - Evidence of Non-carcinogenicity for Humans (no evidence of carcinogenicity in adequate studies)

The USEPA follow a linear multi-stage model for carcinogenesis, which extrapolates relatively high doses used in animal studies to low environmental doses via a straight line. The slope of this line is the Carcinogenic Potency Slope (CPS), or Slope Factor (SF) which is the upper bound excess cancer risk per unit dose, \((\text{mg kg}^{-1} \text{day}^{-1})^{-1}\).

3. **The exposure assessment**: The third step of a human health risk assessment that is the exposure assessment determines i) which receptors are likely to be exposed to the contaminants of concern, ii) how the exposures may occur and iii) the magnitude and duration of the exposures. Human exposure can occur by inhalation (direct) and by indirect pathways such as dermal contact and ingestion of hazardous air pollutants.
deposited to soil and water. The duration of exposure is affected by the fraction of time that the sources of pollution are in operation as well as the fraction of time that individuals are present at a particular location. The extent to which an individual will be impacted by direct inhalation exposure can be expressed as a function of the duration of exposure, volume of air inhaled, and contaminant concentrations.

4. **The risk characterization**: The final step of risk assessment integrates information from the three preliminary steps to estimate the likelihood of adverse effects. The risk characterization assumes that the carcinogenic effects as well as the non-carcinogenic hazard from different contaminants are additive. The calculated carcinogenic risk is the upper bound lifetime excess risk of contracting cancer from the exposure. The non-carcinogenic risk, calculated as Hazard Quotient (HQ) of individual pollutant, indicates the existence of some potential for adverse effects when the HQ value exceeds unity. The higher the HQ, the greater the possibility of effects.

This study is on the last two steps of risk assessment process i.e., exposure assessment and risk characterization for human exposure towards a few mono-aromatic hydrocarbons as obtained in the various scenarios in Kolkata metropolitan city, which have been discussed in details in earlier Chapters. Information on hazard identification and dose-response assessment has been taken from previously reported studies and as documented in the online database of the Risk Assessment Information System (USEPA, RAIS). The toxicity values (RfD and SF) used in the risk calculation in this study associated with the target pollutants are also taken from the above database.

Assuming inhalation to be the primary exposure pathway of air pollutants to humans, only the inhalation pathway of exposure to human receptors has been considered here.
6.2 Methodology

Exposure calculation

The daily exposure ($E$ in mg kg$^{-1}$day$^{-1}$) of an individual due to intake process (considering inhalation only) was calculated (US EPA 1997) from the equation (6.1)

$$E = C \cdot IR_a \cdot ED / BW_a$$ ........... (6.1)

Where, $C$ is concentration (mg m$^{-3}$) of the target pollutant; $IR_a$ is the adult Inhalation Rate (0.83 m$^3$ hr$^{-1}$, USEPA 1997); $ED$ is Exposure Duration (in hour day$^{-1}$ depending upon the nature of exposure); $BW_a$ is adult Body Weight (70 kg).

Effective yearly exposure ($E_Y$ in mg kg$^{-1}$ day$^{-1}$, equation 6.2) and effective lifetime exposure ($E_L$ in mg kg$^{-1}$ day$^{-1}$, equation 6.3) have been calculated separately.

$$E_Y = E \cdot (D/7) \cdot (WK/52)$$ ........... (6.2)

$$E_L = E \cdot (D/7) \cdot (WK/52) \cdot (YE/YL)$$ ........... (6.3)

Where, $D$ is Days per Week Exposure, $WK$ is Weeks of Exposure per year (50 weeks),

Where, $YE$ is Years of chronic exposure $YL$ is Years in Lifetime (75 Years)

The chronic non-cancer hazard quotient of individual pollutant (HQ) was estimated using $E_Y$ and the lifetime cancer risk (ILCR) was estimated from $E_L$.

Calculation of chronic Non-Cancer Risk

Non-cancer risk, expressed as Hazard Quotient (HQ), is defined as the ratio between the yearly average daily dose received, $E_Y$ and the response dose, RfD (a level below which adverse health effects are not likely to occur). This algorithm were used to calculate chronic non-cancer risk (i.e., risk associated with long-term exposures), using chronic RfDs. Summation of HQs for individual contaminants gives Hazard Index (HI).

Calculation of Cancer Risk

Cancer risks was calculated from the equation (6.4)
$$Risk = E_L(mg \ kg^{-1}d^{-1}) \cdot SF(mg^{-1}kg \ d) \quad \cdots \cdots \ (6.4)$$

Where, SF is the carcinogenic potency slope or slope factor.

### 6.4 Result and Discussion

Risk assessment due to exposure towards a few mono-aromatic hydrocarbons has been performed in three exposure scenarios in Kolkata Metropolitan city namely (a) exposure from ambient level, with general people taken as target population or receptors; (b) exposure from some typical urban residential indoor air where the target sub-population is the community representing middle and lower middle income group inhabiting such residences; (c) occupational exposure of the sub-population of petrol pump workers engaged in filling work and drivers of passenger car.

#### 6.4.1 Ambient exposure and risk

The concentrations of the BTEX were found to be quite high in Kolkata metropolitan city and their levels could be a real threat to the health of the city inhabitants (Chapter 3, Section 3.1.3). Table 6.1 gives the average daily exposure, average life time exposure, individual Hazard Quotient (HQ) and ILCR (for 15 years residence time, (i.e. YE=15) for an individual. The exposure duration, ED and exposure day per week, D is taken as 10 hr d$^{-1}$ and 7 days week$^{-1}$ respectively.

The overall exposure is maximum for both xylene mixture and toluene because of their higher ambient concentration followed by benzene and ethylbenzene. According to WHO the lifetime risk of chronic leukemia for benzene exposure of 1 $\mu g$ m$^{-3}$ is 4.4 - 7.6x10$^{-6}$ (WHO, 2000) while ethylbenzene is classified as a group B2 carcinogen (USEPA, RAIS).
Table 6.1: Estimate of individual pollutant exposure, associated non-cancer hazard and cancer risk

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Site</th>
<th>Daily exposure (mg kg(^{-1})day(^{-1}))</th>
<th>Effective life time exposure (mg kg(^{-1})day(^{-1}))</th>
<th>Individual HQ</th>
<th>ILCR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Benzene</strong></td>
<td>Site N</td>
<td>1.5E-03</td>
<td>3.0E-04</td>
<td>1.8E-01</td>
<td>8.3E-06</td>
</tr>
<tr>
<td></td>
<td>Site C</td>
<td>3.3E-03</td>
<td>6.6E-04</td>
<td>3.9E-01</td>
<td>1.8E-05</td>
</tr>
<tr>
<td></td>
<td>Site S</td>
<td>5.2E-03</td>
<td>1.0E-03</td>
<td>6.0E-01</td>
<td>2.8E-05</td>
</tr>
<tr>
<td><strong>Toluene</strong></td>
<td>Site N</td>
<td>3.1E-03</td>
<td>6.2E-04</td>
<td>2.2E-03</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Site C</td>
<td>5.1E-03</td>
<td>1.0E-03</td>
<td>3.6E-03</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Site S</td>
<td>6.0E-03</td>
<td>1.2E-03</td>
<td>4.2E-03</td>
<td></td>
</tr>
<tr>
<td><strong>Ethylbenzene</strong></td>
<td>Site N</td>
<td>8.2E-04</td>
<td>1.6E-04</td>
<td>2.9E-03</td>
<td>6.3E-07</td>
</tr>
<tr>
<td></td>
<td>Site C</td>
<td>1.5E-03</td>
<td>3.0E-04</td>
<td>5.2E-03</td>
<td>1.2E-06</td>
</tr>
<tr>
<td></td>
<td>Site S</td>
<td>1.7E-03</td>
<td>3.5E-04</td>
<td>6.1E-03</td>
<td>1.3E-06</td>
</tr>
<tr>
<td><strong>Xylene mixture</strong></td>
<td>Site N</td>
<td>3.6E-03</td>
<td>7.1E-04</td>
<td>1.2E-01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Site C</td>
<td>5.0E-03</td>
<td>1.0E-03</td>
<td>1.8E-01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Site S</td>
<td>5.7E-03</td>
<td>1.1E-03</td>
<td>2.0E-01</td>
<td></td>
</tr>
</tbody>
</table>

The cancer risk calculated in the current study suggests the exposure level to be far from being safe for city population residing for 15 years near the monitoring sites. For all three sites, the estimated cancer risk is most for benzene due its high carcinogenicity. Estimated cancer risk in all the sites and for all the individual components (except for ethylbenzene in Site N) exceeded the threshold value of 1E-06 indicating significant cancer risk. In general, residents of Site S receive higher dose of exposure from the pollutants in comparison to the other two sites and as a result the probability of cancer risk is higher in Site S. Assuming that the carcinogenic effect from different pollutants is additive, the cumulative cancer risk from benzene and ethylbenzene is maximum (3.0E-05) in Site S, followed by Site C (1.9E-05) and Site N (8.9E-06).
Due to the low reference dose for adverse non-cancer health effect, benzene gives the highest non-cancer HQ, in spite of its lower exposure value. Benzene is closely followed by xylenes in causing non-cancer health hazard. The individual HQs or the HI for BTEX did not exceed unity at any site indicating no serious threat of chronic health effect in pollutant specific target organs for the city population.

**6.4.2 Indoor air and health risk**

The estimate of mean individual pollutant exposure, associated non-cancer hazard and cancer risk from exposure in urban residential indoors is given in Table 6.2 for all the households studied (Chapter 4, Section 4.3). ED and D are taken as 10hr d\(^{-1}\) and 7days week\(^{-1}\) respectively for 15 years of residing period (i.e., YE=15) in such dwelling conditions.

As a guideline, excess lifetime cancer risk below 10\(^{-6}\) may be acceptable (EPA, 1989). Indoor level of benzene alone exceeded the safe level in all cases and ethylbenzene exceeded the level in 76% residences studied.

**Table 6.2: Estimate of pollutant exposure, associated non-cancer hazard and cancer risk in residential indoors**

<table>
<thead>
<tr>
<th>Compound</th>
<th>Average daily exposure</th>
<th>Effective life time exposure</th>
<th>Hazard Quotient</th>
<th>Cancer Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzene</td>
<td>4.98E-03</td>
<td>9.97E-04</td>
<td>5.81E-01</td>
<td>2.72E-05</td>
</tr>
<tr>
<td>Toluene</td>
<td>8.21E-03</td>
<td>1.64E-03</td>
<td>5.74E-03</td>
<td></td>
</tr>
<tr>
<td>Et-Benzene</td>
<td>2.70E-03</td>
<td>5.41E-04</td>
<td>9.46E-03</td>
<td>2.08E-06</td>
</tr>
<tr>
<td>Xylene mixture</td>
<td>8.75E-03</td>
<td>1.75E-03</td>
<td>3.06E-01</td>
<td></td>
</tr>
</tbody>
</table>

162
The average integrated lifetime cancer risk considering the exposure to benzene and ethylbenzene for residing in such indoor conditions for 15 years is estimated to be $2.9 \times 10^{-5}$. The mean HQs for the individual component or the HI value are less than 1 indicating no significant threat of non-cancer health effect. However, the HQ and HI values corresponding to benzene level exceeded the acceptable limit of 1 in 13% and 33% cases respectively of the residences studied.

### 6.4.3 Occupational exposure and health risk

The occupational exposures of the two sub-population namely, petrol pump workers and the car drivers, to the target pollutants were estimated considering an occupational exposure of 40 years ($YE=40$), with ED and D taken as 8 hr d$^{-1}$ and 5 days week$^{-1}$ respectively. The exposures along with the cancer and non-cancer risk are tabulated in Table 6.3. The exposure was found to be highest for toluene, but due to its greater toxicity, benzene showed maximum non-cancer hazard as well as cancer risk for both the occupational groups.

Although the exposure level for BTEX observed in the current study was well below the Threshold Limit Value-Time weighted Average (TLV-TWA) value proposed by American Conference of Industrial Hygienists (ACGIH) (1600 $\mu$g m$^{-3}$ for benzene), and Recommended Exposure Limit proposed by NIOSH (325 $\mu$g m$^{-3}$), the individual cancer risks for benzene and ethylbenzene were found to be higher than the acceptable limit. The integrated lifetime cancer risks from benzene and ethylbenzene were found to be quite high with cancer risk for 146 and 42 workers in a million for petrol pump workers and the drivers respectively.
Table 6.3: Estimate of individual pollutant exposure, associated non-cancer hazard and cancer risk for occupationally exposed groups

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Workers</th>
<th>Benzene</th>
<th>Toluene</th>
<th>Ethylbenzene</th>
<th>Xylene mixture</th>
</tr>
</thead>
<tbody>
<tr>
<td>$E$ (mg kg$^{-1}$ day$^{-1}$)</td>
<td>Petrol Pump Workers</td>
<td>1.30E-02</td>
<td>6.11E-02</td>
<td>1.12E-02</td>
<td>2.64E-02</td>
</tr>
<tr>
<td>Drivers</td>
<td>9.28E-03</td>
<td>1.81E-02</td>
<td>5.72E-03</td>
<td>1.49E-02</td>
<td></td>
</tr>
<tr>
<td>$E_Y$ (mg kg$^{-1}$ day$^{-1}$)</td>
<td>Petrol Pump Workers</td>
<td>8.96E-03</td>
<td>4.19E-02</td>
<td>7.69E-03</td>
<td>1.81E-02</td>
</tr>
<tr>
<td>Drivers</td>
<td>6.38E-03</td>
<td>1.24E-02</td>
<td>3.93E-03</td>
<td>1.02E-02</td>
<td></td>
</tr>
<tr>
<td>$E_L$ (mg kg$^{-1}$ day$^{-1}$)</td>
<td>Petrol Pump Workers</td>
<td>4.78E-03</td>
<td>2.24E-02</td>
<td>4.10E-03</td>
<td>9.65E-03</td>
</tr>
<tr>
<td>Drivers</td>
<td>1.28E-03</td>
<td>2.49E-03</td>
<td>7.86E-04</td>
<td>2.05E-03</td>
<td></td>
</tr>
<tr>
<td>HQ</td>
<td>Petrol Pump Workers</td>
<td>1.05E+00</td>
<td>2.93E-02</td>
<td>2.69E-02</td>
<td>6.33E-01</td>
</tr>
<tr>
<td>Drivers</td>
<td>7.44E-01</td>
<td>8.71E-03</td>
<td>1.37E-02</td>
<td>3.58E-01</td>
<td></td>
</tr>
<tr>
<td>ILCR</td>
<td>Petrol Pump Workers</td>
<td>1.30E-04</td>
<td>1.58E-05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drivers</td>
<td>3.48E-05</td>
<td>7.98E-06</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The individual HQ value did not exceed unity except for petrol pump workers' benzene exposure but the HI value due to all of BTEX exceeded 1, (1.7 and 1.1 for petrol pump workers and the drivers respectively) for both the occupational groups. This indicates that though the level of the pollutants may not pose serious non-cancer health threat individually, their cumulative exposure, which is the actual scenario here, may cause some non-cancer health damage to the workers.

It is therefore wise to calculate the cancer risk for benzene and the non-cancer hazard quotient for BTX corresponding to the limiting occupational exposure values set by various authorities around the world. The corresponding risk values are alarmingly high considering 40 years of occupational exposure (Table 6.4).
Table 6.4: Exposure, hazard and risk values corresponding to the limiting concentration recommended by various authorities

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Limit</th>
<th>Concentration</th>
<th>$E$</th>
<th>$E_Y$</th>
<th>$E_L$</th>
<th>HQ</th>
<th>ILCR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\mu g , m^{-3}$</td>
<td>$mg , kg^{-1} , day^{-1}$</td>
<td>$mg , kg^{-1} , day^{-1}$</td>
<td>$mg , kg^{-1} , day^{-1}$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benzene</td>
<td>TLV-TWA</td>
<td>1600</td>
<td>1.5E-01</td>
<td>1.0E-01</td>
<td>5.6E-02</td>
<td>1.2E+01</td>
<td>1.5E-03</td>
</tr>
<tr>
<td></td>
<td>REL</td>
<td>325</td>
<td>3.1E-02</td>
<td>2.1E-02</td>
<td>1.1E-02</td>
<td>2.5E+00</td>
<td>3.1E-04</td>
</tr>
<tr>
<td></td>
<td>PEL, LV, VLA</td>
<td>3250</td>
<td>3.1E-01</td>
<td>2.1E-01</td>
<td>1.1E-01</td>
<td>2.5E+01</td>
<td>3.1E-03</td>
</tr>
<tr>
<td>Toluene</td>
<td>MAK, TLV-TWA, VLA</td>
<td>1.91E+05</td>
<td>1.8E+01</td>
<td>1.2E+01</td>
<td>6.6E+00</td>
<td>8.7E+00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>REL</td>
<td>3.82E+05</td>
<td>3.6E+01</td>
<td>2.5E+01</td>
<td>1.3E+01</td>
<td>1.7E+01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PEL</td>
<td>7.64E+05</td>
<td>7.2E+01</td>
<td>5.0E+01</td>
<td>2.7E+01</td>
<td>3.5E+01</td>
<td></td>
</tr>
<tr>
<td>Xylene</td>
<td>MAK, TLV-TWA, VLA, REL</td>
<td>4.42E+05</td>
<td>4.2E+01</td>
<td>2.9E+01</td>
<td>1.5E+01</td>
<td>1.0E+03</td>
<td></td>
</tr>
<tr>
<td>mixture</td>
<td>LV, VLA</td>
<td>2.21E+05</td>
<td>2.1E+01</td>
<td>1.4E+01</td>
<td>7.7E+00</td>
<td>5.0E+02</td>
<td></td>
</tr>
</tbody>
</table>

MAK (Maximum Allowable Concentration): (DFG, German Research Foundation) (2002)
REL (Recommended Exposure Limit): NIOSH (1994).
LV (Limit Values): European Directives 2000/39/EC and 97/42/EC.

Permissible exposure limit (PEL) value suggested by Occupational Safety and Health Association (OSHA) gives a probability of cancer risk for 3 workers in thousand workers and a non-cancer HQ of 25 resulting from benzene-exposure only. This demand the occupational exposure limit values be modified considering the inherent health risk of the workers.

It is to be noted that the risk values computed in the present study considered only the eight hours of occupational exposure of the petrol pump workers while other environmental exposure for remaining part of the day may aggravate both the actual cancer and non-cancer risk.
6.4.4 Comparison of health risks of benzene exposure assessed from external and internal dose:

In this section a comparative analysis of the carcinogenic and non carcinogenic health risk has been carried out using conventional environmental exposure against the same estimated using the internal dose obtained as body burden as described earlier (Chapter 5, Section 5.3.4) for two occupational exposure groups and the control group due to inhalation of benzene. The ED and YE for control group was taken same as that for occupationally exposed groups to keep the result equitable. For risk calculation from internal dose or body burden, the TBA has been assumed to be equivalent to daily exposure E and EY, Els, Risk, HQ were calculated subsequently.

The conventional occupational benzene exposure (E) via inhalation for eight hours was estimated to be 13.0 and 9.3 μg kg⁻¹ body weight for petrol pump workers and car drivers respectively (Table 6.3) and the corresponding body burden or the inhalation intake (TBₗ in mg kg⁻¹ body weight) was assumed to be 50% of E considering 50% of respiratory absorption of benzene (USEPA, 2002). The body burden of benzene estimated from urinary t₁,t-MA measurement using PBPK model was found to be nearly 169% and 140% more for petrol pump workers and car drivers respectively, even after metabolism. There may be four fold reasons behind it.

1. The occupationally exposed workers are also exposed environmentally for the rest of the day. If we consider the 16 hr environmental exposure additive to the occupational exposure, then the body burden remains 78% and 39% more than the total inhalation intake respectively.

2. The occupational as well as environmental exposure being chronic in nature, there may be residual body burden from previous exposure.
3. We have considered only inhalation route of exposure, whereas other routes like oral or dermal route of exposure may contribute towards body burden. Especially for petrol pump workers dermal route is likely to contribute significantly.

4. As already mentioned, body burden may not be proportional to exposure due to non-linear increase of metabolic capacity at comparatively higher exposure level. The body burden of control group is only about 90% of the inhalation intake if we consider a 24 hr environmental exposure.

Figure 6.1 gives the comparative carcinogenic risk and non-carcinogenic hazard estimate using both conventional method and the body burden or internal dose. Carcinogenic risk estimated from body burden is significantly high in both petrol pump workers and car drivers with probability of cancer of 176 and 111 workers in a million respectively. The HQ value of 1.4 indicates that the prevailing benzene level may also produce non-cancer health effect in petrol pump workers but may not be in drivers with HQ value of 0.9 which is in the borderline. The carcinogenic risk estimated from internal dose is 35% and 20% more for petrol pump workers and car drivers respectively than the same estimated using external exposure values.

It is evident from risk assessment study that internal dose or body burden is a superior indicator of individual susceptibility to benzene toxicity as it reflects the overall exposure considering all the exposure routes, nature of exposure and individual physiological variability.
6.5 Conclusion

It was found that the prevailing benzene and ethylbenzene level is estimated to pose significant cancer risk due to the inhalation exposure to the general city population. The carcinogenic risk was also more than the acceptable probability of one in a million in typical urban residences generally inhabited by population of middle or lower-middle income group. It was estimated that high level of exposure of refueling station attendants and car drivers make them a population of ‘High Risk’ (both cancer and non cancer) although the exposure levels were well below the prescribed limit set by the regulating authorities. The total body burden well represents the actual internal dose through all possible exposure routes, nature of exposure and individual physiological variability due to chronic occupational and environmental exposure at varying level. Toxicity of benzene can be more accurately described by the total body burden rather than the external dose derived from exposure.
Reference:

ACGIH, 2003 Threshold limit values for chemicals substances and physical agents and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienist.


USEPA, RAIS. Online Database of The Risk Assessment Information System (RAIS); url: http://rais.ornl.gov