**LITERATURE REVIEW**

# Literature Review:

# BENZENE

**Oleh:**

**dr. Dewi Indah Lestari, MKK, Sp.OK**

**FAKULTAS KEDOKTERAN**
**UNIVERSITAS TARUMANAGARA**
**JAKARTA**

**2026**

# LITERATURE REVIEW

# BENZENE

## Background

In formal industry where benzene has been identified as one of occupational hazard, strategies are made to recognize, monitor, and prevent excessive workers exposure to benzene, and its health effect. Benzene monitoring is a critical component of health and safety programs in oil and gas companies, as workers in these industries are exposed to certain levels of this toxic substance. The oil and gas sector, particularly during exploration, extraction, and refining processes, involves several stages where benzene is a byproduct of crude oil, natural gas, and petroleum refining operations[1]

Occupational exposure often occurs in industries such as petrochemical production, oil refining, rubber manufacturing, and printing, where workers may come into contact with benzene via inhalation or dermal absorption2. Additionally, benzene is a component of gasoline, leading to exposure risks for workers involved in fuel handling, transportation, and storage. Maintenance operations in equipment used to process benzene-containing materials also present exposure risks[3].

However, benzene is not an isolated health hazard to oil and gas workers, it is a common air pollutant in urban environments. Individuals working in traffic-intensive areas, such as motorcycle drivers, are at risk of exposure to ambient benzene levels. Studies have shown that traffic-related air pollution, including benzene, poses a significant health risk to individuals who are frequently exposed to vehicle emissions. Motorcycle drivers, especially those in urban areas with heavy traffic, often inhale high concentrations of benzene, as this compound is a byproduct of the combustion of gasoline and other fuels[4].

Ambient benzene concentrations are typically higher in traffic hotspots, where vehicle emissions contribute to pollution levels, particularly in areas with high vehicle density. Motorcycle drivers are especially vulnerable due to their close proximity to exhaust gases, with studies showing higher benzene levels in the air surrounding vehicles, including motorcycles, compared to areas with lower traffic volume[2]. Furthermore, motorcycles are less regulated in terms of emissions control compared to other vehicles, leading to increased benzene exposure for drivers[1].

Benzene exposure can also cause cardiovascular damage, apart from its already acknowledged health risks to hematopoietic system and a carcinogenic agent. It has been associated with an elevated risk of cardiovascular events in occupational groups and individuals with mild to high cardiovascular disease (CVD) risk5. Studies have shown that benzene contributes to oxidative stress and inflammation, which may impair vascular function, lead to endothelial injury, and promote atherosclerosis [6 7]. The compound has been associated with endothelial dysfunction, a precursor to the development of cardiovascular diseases, particularly in workers exposed to high levels of benzene over long periods[1].

Studies have shown that benzene metabolites generate reactive oxygen species (ROS), which damage cellular components, leading to endothelial dysfunction and impairing vascular function[4]. This endothelial damage is a key factor in the development of atherosclerosis, hypertension, and other cardiovascular diseases[2]. Additionally, benzene exposure can lead to increased blood clotting and platelet aggregation, further contributing to the risk of cardiovascular events such as stroke or myocardial infarction1. Given these

risks, biomonitoring plays a crucial role in assessing benzene exposure and its effects on the cardiovascular system. Biomonitoring typically involves measuring biomarkers of benzene exposure, such as trans, trans-muconic acid (t,t-MA) in urine, which provides an indication of cumulative benzene exposure. Elevated levels of t,t-MA have been associated with increased risk of cardiovascular dysfunction in both occupational and environmental exposure scenarios[2]. In a study, benzene exposure is associated with a decrease of circulating angiogenic cells that has been predictive of cardiovascular event in human[5]. However, currently we still have limited knowledge regarding utilization of CACs as biomarker of effect for benzene cardiovascular toxicity. There is a need for further study about possibility of using CACs in biomonitoring for protection of workers exposed to benzene.

## Production and Route of Exposure of Benzene

Benzene, as a byproduct of working processes, has been identified in oil and gas and manufacture industry. It is also an environmental chemical hazard for workers in transportation sector. The upstream oil and gas industry is a major source of benzene production in occupational settings, especially in sectors such as conventional oil and gas extraction, heavy oil processing, drilling, and pipeline operations, where benzene exposure can occur.

The study found that highest personal long-term exposure to benzene was reported for a dehydration operator at a gas plant, with a measurement of 57.6 mg/m³. There were five other samples over the occupational exposure limit (OEL) for benzene, ranging from 4.13 to 28.2 mg/m³, with a calculated frequency of overexposure of less than 1%. In the conventional gas section, two personal short-term samples exceeded the short-term exposure limit (STEL) for benzene, with values of 28.8 mg/m³ and 35.2 mg/m³. These samples were collected from workers replacing a mechanical seal on a condensate booster pump. The conventional oil/gas section had the highest frequency of samples over the OEL for both benzene and total hydrocarbons (THC) for area long-term samples, with 13% and 50% respectively. However, the sample size was small, with only 13 data points. No samples exceeded the exposure limit for either benzene or THC in the heavy oil processing, drilling, and pipeline sections. The data points for drilling and pipeline sections were very limited. The study highlighted that the conventional gas section had the highest area long-term exposure to benzene, with a sample measuring 39.778 mg/m³ taken in a glycol utilities area. Another sample over the OEL was reported at 6.219 mg/m³ in the area of a condensate loading rack. The study concluded that while compliance with the benzene OEL of 1 ppm (3.2 mg/m³) for an 8-hour TWA was generally not a problem, greater effort would be required to meet the lower ACGIH TLV-TWA of 0.5 ppm (1.6 mg/m³) and STEL of 2.5 ppm (8 mg/m³) as the standard of that time. Current standard as per 2024 for benzene in ACGIH as below:

| | ADOPTED VALUES | | | | |
|---|---|---|---|---|---|
| Substance [CAS No.] (Documentation date) | TWA | STEL | Notations | MW | TLV Basis |
| * Benzene [71-43-2] (2023) | 0.02 ppm | — | Skin; A1; BEI | 78.11 | Myelodysplastic syndrome; acute myeloid leukemia; leukemia; hemato-logic eff; chromosomal dam |

Table 1. TWA levels in ACGIH 2024

TLV–CS

| Substance [CAS No.] (Documentation date) | ADOPTED VALUES TWA | STEL | Notations | MW | TLV Basis |
|---|---|---|---|---|---|
| Arsine [7784-42-1] (2007) | 0.005 ppm | — | — | 77.95 | PNS & vascular system impair; kidney & liver impair |
| Asbestos [1332-21-4], all forms (1998) | 0.1 f/cc (F) | — | A1 | — | Pneumoconiosis; lung cancer; mesothelioma |
| Asphalt (Bitumen) fumes [8052-42-4], as benzene-soluble aerosol (2000) | 0.5 mg/m³ (I) | — | A4; $BEI_P$ | — | URT & eye irr |
| Atrazine [1912-24-9] (and related symmetrical triazines) (2014) | 2 mg/m³ (I) | — | A3 | 215.69 | Hematologic, repro, & developmental eff |
| Azinphos-methyl [86-50-0] (2014) | 0.2 mg/m³ (IFV) | — | Skin; DSEN; A4; $BEI_C$ | 317.34 | Cholinesterase inhib |
| Barium [7440-39-3] and soluble compounds, as Ba (1996) | 0.5 mg/m³ | — | A4 | 137.30 | Eye, skin, & GI irr; muscular stimulation |
| Barium sulfate [7727-43-7] (2014) | 5 mg/m³ (I, E) | — | — | 233.43 | Pneumoconiosis |
| Bendiocarb [22781-23-3] (2018) | 0.1 mg/m³ (IFV) | — | Skin; A4; $BEI_C$ | 223.20 | Cholinesterase inhib |
| Benomyl [17804-35-2] (2014) | 1 mg/m³ (I) | — | DSEN; A3 | 290.32 | URT irr; male repro, testicular, & embryo/fetal dam |
| Benz[a]anthracene [56-55-3] (1993) | — (L) | — | A2; $BEI_P$ | 228.30 | Skin cancer |
| ‡ Benzene [71-43-2] (1997) | (0.5 ppm) | (2.5 ppm) | Skin; A1; BEI | 78.11 | Leukemia ( ) |

Table 2. TWA levels in ACGIH 2023

In Indonesia, the standard for benzene in the workplace regulated by ministry of health as follows:

| No. | Parameter | Nomor CAS | Notasi | NAB *TWA* | | NAB *STEL* | | NAB *Ceiling (C)* | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | ppm | mg/m³ | ppm | mg/m³ | ppm | mg/m³ |
| 31. | Barium, soluble compounds (sebagai Ba) | 7440-39-3 | A4 Eye, Skin, GI Irr, Muscular stim | | 0,5 | | | | |
| 32. | Barium Sulfate | 7727-43-7 | - | | | | | | |
| | Total dust | | | | 10 | | | | |
| | Respirable fraction | | | | 5 | | | | |
| 33. | Benzene | 71-43-2 | Skin; A1; BEI Leukemia | 0,5 | | 2,5 | | | |

Table 3 Ministry of Health Regulation No.70 2016

Benzene is commonly present in the ambient air at fuel service stations due to fuel evaporation during gasoline refueling operations[8]. Research has shown that workers exposed to benzene face increased health risks, including a heightened risk of cancer from occupational exposure and adverse health effects associated with prolonged benzene exposure. Symptoms of benzene poisoning include headaches, fatigue, throat and nasal irritation, nausea, dizziness, and depression. The extent of benzene exposure at gasoline stations can be evaluated by measuring its concentration in the workplace air and through biomarker monitoring. A health risk assessment based on benzene concentration levels has indicated an increased cancer risk among gasoline station workers.

Exposure biomarkers, specifically trans, trans-muconic acid (tt-MA) and S-phenylmercapturic acid (S-PMA), are used to measure benzene level in workers after work shifts, which according to The American Conference of Governmental Industrial Hygienists (ACGIH), the value of biological exposure index (BEI) for tt-MA should be no more than 500 µg/g creatinine (Cr) in post-shift samples. In Thailand, a study on gasoline station workers found tt-MA levels ranging from 3.0 to 1127 µg/g Cr, while research on Indonesian gasoline workers reported an average level of 480.74 (219.7) µg/g Cr. Another study examining tt-MA levels and DNA oxidative stress among fueling workers and university staff in Egypt found that fueling workers had significantly higher tt-MA levels, along with observable

hematological effects. Additionally, a study investigating the impact of benzene exposure at gasoline stations identified several abnormalities, including a decrease in nearly all blood cell counts, which was considered a phenotypic effect of benzene exposure.

In manufacture sector, benzene exposure comes with the use of solvent. A study by Kahar et al (2024) assessed airborne benzene levels in a manufacturing environment, focusing on a painting production unit. The findings indicated that benzene concentrations were 0.0098 BDS in the production area and 0.0001 BDS in the office, both of which were well below the Occupational Exposure Limit (OEL) of 0.5 BDS/ppm, ensuring a safe working environment for employees. A total of 16 participants were involved in the research, with urine and blood samples collected to measure trans,trans-muconic acid (tt-MA) levels and liver function indicators, including SGOT and SGPT. The results showed that tt-MA levels were significantly higher among production unit workers, averaging 331.5 µg/creatin, compared to 106.6 µg/creatin in administrative staff. Statistical analysis revealed a highly significant difference in tt-MA levels between the exposed and unexposed groups, with a p-value of less than 0.001, confirming that production unit workers had increased benzene exposure. Liver function tests also showed a significant difference in SGOT levels ($p < 0.033$) between the two groups, suggesting that benzene exposure may impact liver function.

Although airborne benzene levels were within safety limits, the study highlighted notable differences in tt-MA and SGOT levels between production and administrative workers. These findings underscore the importance of continuous monitoring and protective measures to safeguard workers from potential benzene exposure risks.

Workers in the transportation industry are also exposed to benzene and other volatile organic compounds in traffic environments. A study conducted in Egypt by Kasemy et al on 2019 to taxi drivers' population. The study examined environmental benzene exposure and its health effects on taxi drivers in Egypt, comparing them to a control group of unexposed individuals. A total of 280 taxi drivers and 120 matched controls participated in the study. Measurements of ambient air pollutants revealed significantly high concentrations of hazardous substances. The average levels recorded were 0.81 ppm benzene, 26.69 ppm toluene, 29.36 ppm ethylbenzene, and 25.11 ppm xylene. These values exceeded international permissible limits, highlighting a serious environmental health concern ($P < 0.001$). Health symptoms were notably more prevalent among taxi drivers compared to the control group. Among the drivers, 27.1% reported pallor, 24.3% experienced dizziness, and 45.7% suffered from fatigue, whereas these symptoms occurred at much lower rates among the control participants. This disparity suggests that benzene exposure is contributing to adverse health effects in taxi drivers. Additionally, chronic diseases were more frequently reported among the taxi drivers.

**Chemical Identity of Benzene**

**Table 4-1. Chemical Identity of Benzene**

| Characteristic | Information | Reference |
|---|---|---|
| Chemical name | Benzene | NLM 2023 |
| Synonym(s) | [6] Annulene, benzeen (Dutch), benzen (Polish), benzol, benzole; benzolo (Italian), coal naphtha, cyclohexatriene, fenzen (Czech), phene, phenyl hydride, pyrobenzol, pyrobenzole | NLM 2023 |
| Registered trade name(s) | Polystream | IARC 1982 |
| Chemical formula | $C_6H_6$ | NLM 2023 |
| SMILES | C1=CC=CC=C1 | NLM 2023 |
| Chemical structure |  | NLM 2023 |
| CAS Registry Number | 71-43-2 | NLM 2023 |

CAS = Chemical Abstracts Service; SMILES = simplified molecular-input line-entry system

Table 5.Chemical Identity of Benzene from ATSDR

**Physical and Chemical Properties of Benzene**

**Table 4-2. Physical and Chemical Properties of Benzene**

| Property | Information | Reference |
|---|---|---|
| Molecular weight | 78.11 g/mol | Budavari et al. 2001 |
| Color | Clear, colorless liquid | Budavari et al. 2001 |
| Physical state | Colorless to light yellow liquid | NLM 2023 |
| Melting point | 5.558°C | NLM 2023 |
| Boiling point | 80.08°C | NLM 2023 |
| Density at 20°C, g/cm³ | 0.8756 | NLM 2023 |
| Odor | Aromatic (petroleum-like) | NFPA 1994; NLM 2023 |
| Odor threshold: | | |
| Water | 2.0 mg/L | NLM 2023 |
| Air* | Detection range: 34–119 ppm (geometric mean: 61 ppm)<br>Recognition: 97 ppm | AIHA 1989 |
| Taste threshold | 0.5–4.5 mg/L | NLM 2023 |
| Solubility: | | |
| Water at 25°C | w/w: 1,790 mg/L | NLM 2023 |
| Organic solvent(s) | Alcohol, chloroform, ether, carbon disulfide, acetone, oils, carbon, tetrachloride, glacial acetic acid | Budavari et al. 2001 |
| Partition coefficients: | | NLM 2023; Karickhoff 1981; Kenaga 1980 |
| Log $K_{ow}$ | 2.13 | |
| Log $K_{oc}$ | 1.8–1.9 | |
| Vapor pressure at 25°C | 94.8 mm Hg | NLM 2023 |
| Henry's law constant at 25°C | $5.5 \times 10^{-3}$ atm-m³/mol | Mackay and Leinonen 1975 |
| Autoignition temperature | 498°C | NFPA 1994 |
| Flashpoint | -11°C (closed cup) | Budavari et al. 2001 |
| NFPA hazard classification: | | OSHA 2021 |
| Health | 2 | |
| Flammability | 3 | |
| Reactivity | 0 | |
| Flammability limits in air | 1.2% (lower limit); 7.8% (upper limit) | OSHA 2021 |
| Conversion factors | 1 ppm=3.26 mg/m³ at 20°C and 1 atm pressure; 1 mg/m³=0.31 ppm | NLM 2023 |
| Explosive limits | 1.4% (lower limit); 8% (upper limit) | NLM 2023 |

Table 6. Chemical Properties of Benzene from ATSDR

Benzene is formed from human activities as well as natural processes. It evaporates rapidly to air and slightly soluble in water. Commercially refined benzene-535 is free of hydrogen sulphide and sulphur dioxide but contains a maximum of 1 ppm thiophene and a maximum of 0.15% nonaromatic9. Benzene is also commercially available as nitration-grade (99% pure), thiophene-free, 99 mole%, 99.94 mole%, and nano grade quality.

**Benzene Toxicokinetic**

Human exposure to benzene primarily occurs through inhalation, though ingestion and skin contact are also possible. Benzene is efficiently absorbed through both inhalation and oral intake. While it can also penetrate the skin, a significant portion evaporates before being absorbed. Once absorbed, benzene is quickly distributed throughout the body, with a tendency to accumulate in fatty tissues. The liver and other tissues, such as lymphocyte progenitor cells in the bone marrow, metabolize benzene. This process generates reactive metabolites believed to contribute to benzene's toxic effects. At low exposure levels, benzene is rapidly broken down and primarily excreted as conjugated urinary metabolites. However, at higher exposure levels, metabolic pathways become saturated, leading to the excretion of a substantial portion of unchanged benzene in exhaled air. The metabolic processing of benzene is qualitatively similar across humans and various laboratory animals, though quantitative differences exist in the proportions of its metabolites.

**Absorption**

Inhalation is likely the primary route of human exposure to benzene. Several studies have assessed benzene absorption by measuring the difference between inhaled and exhaled concentrations10 11. Evidence suggests that benzene is rapidly absorbed upon inhalation. A study involving 23 participants exposed to 47–110 ppm benzene for 2–3 hours found that absorption was highest in the initial minutes of exposure but declined over time10. Within the first 5 minutes, absorption ranged from 70–80%, decreasing to approximately 50% (ranging from 20–60%) after an hour. In another study, six male and female volunteers exposed to 52–62 ppm benzene for 4 hours had a respiratory extraction rate of around 47%.

Similarly, research on three healthy nonsmoking individuals exposed to benzene at 1.6 or 9.4 ppm for 4 hours estimated absorption by calculating the difference between inhaled and exhaled concentrations11. The study found a respiratory extraction rate of 48% at the higher dose and 52% at the lower dose, aligning with the findings of. In occupational settings, benzene exposure can occur through both inhalation and skin absorption . Studies examining workplace exposure suggest that benzene is absorbed via both routes in many industrial environments. A study conducted in Finland in 1992 assessed benzene exposure among car mechanics12. Measurements were taken at five garages, and blood samples from eight nonsmoking mechanics were collected 3–9 hours after exposure and then adjusted to estimate levels at 16 hours post-exposure.

Benzene concentrations during carburettor-related tasks ranged from 0.5 to 1.1 $cm^3/m^3$ (0.2–0.3 ppm), while changing a fuel filter in an electronic fuel-injection system resulted in exposure levels of 0.9–3.4 $cm^3/m^3$ (0.3–1.1 ppm). Estimated benzene concentrations in blood at 16 hours post-exposure were significantly higher than anticipated based on air measurements alone (8-hour TWA). A comparison of expected benzene levels in the absence of dermal absorption with actual blood concentrations suggested that skin exposure contributed to overall absorption, with dermal exposure levels ranging from 1.1% to 88.2%. Two of the eight workers exhibited minimal skin absorption (0–1.1%), while the remaining six had substantial dermal exposure (79.4%). Benzene can also be absorbed from fire-related emissions. A comparison of blood benzene levels between U.S. engineers and firefighters working at burning oil wells in Kuwait and non-exposed U.S. citizens was conducted[12]. The median benzene concentrations in whole blood were 0.035 µg/L (range: not detected–0.055 µg/L) for engineers, 0.18 µg/L (range: 0.063–1.1 µg/L) for firefighters, and 0.066 µg/L (range: not detected–0.54 µg/L) for the general U.S. reference group. The median benzene concentration in firefighters was generally higher than that in engineers[12].

**Distribution**

Most of the information regarding benzene distribution in humans comes from case studies. Findings indicate that after being absorbed into the bloodstream, benzene disperses throughout the body. Due to its lipophilic nature, it is expected to accumulate in fatty tissues. Following inhalation exposure, benzene has been detected in biological fluids and tissues [11] [12] . Benzene levels in bodily fluids and tissues have been documented in both accidental and intentional fatal exposures. In one case, a worker who died from exposure to extremely high benzene concentrations had levels of 0.38 mg% in blood, 1.38 mg% in the brain, and 0.26 mg% in the liver[12]. However, the specific unit of measurement for mg% (e.g., mg/100 mL or mg/100 g) was not provided.

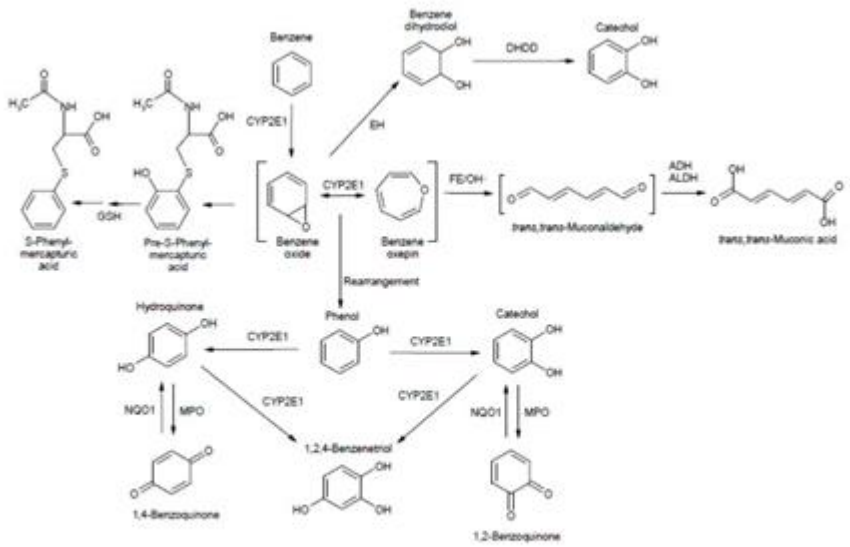
Another autopsy, performed on a young individual who died after inhaling reagent-grade benzene, detected benzene concentrations of 2.0 mg% in blood, 3.9 mg% in brain, 1.6 mg% in liver, 1.9 mg% in kidney, 1 mg% in stomach, 1.1 mg% in bile, 2.23 mg% in abdominal fat, and 0.06 mg% in urine[12]. Similar to Tauber's study, the exact units for mg% were not specified. Findings from animal studies suggest that absorbed benzene is distributed across multiple compartments in the body.

**Metabolism**

Benzene metabolism has been extensively researched, and it is widely recognized that both cancerous and noncancerous effects stem from one or more of its reactive metabolites. Evidence suggests that metabolites formed in the liver are transported to the bone marrow, where benzene toxicity manifests. Additionally, benzene metabolism occurs within the bone marrow and other tissues. Most data on benzene metabolism in humans come from studies involving inhalation exposure. The compound is excreted both unchanged through the lungs and as metabolites in the urine, with small amounts also eliminated as the parent compound.

The rate and extent of excretion via the lungs depend on the exposure dose and route. In terms of metabolic processes and elimination, benzene appears to be processed in a qualitatively similar manner in both humans and laboratory animals.

**Figure 3-1. Metabolic Pathways for Benzene**




ADH = alcohol dehydrogenase; ALDH = aldehyde dehydrogenase; CYP2E1 = cytochrome P450 2E1; DHDD = dihydrodiol dehydrogenase; EH = epoxide hydrolase; GSH = glutathione; MPO = myeloperoxidase; NQO1 = NAD(P)H:quinone oxidoreductase

Sources: Bowman et al. 2023; Nebert et al. 2002; Ross 2000; Sabourin et al. 1998

Figure : Metabolic Pathways for Benzene

The metabolic pathway of benzene, is derived from extensive mechanistic studies on benzene metabolism (Henderson et al., 1989; Ross, 1996, 2000). The initial step involves the oxidation of benzene by the enzyme CYP2E1, leading to the formation of benzene oxide, which exists in equilibrium with benzene oxepin[13]. Benzene oxide undergoes several metabolic pathways. The primary metabolic route involves a nonenzymatic rearrangement that produces phenol, the major initial metabolite of benzene[14]. Phenol is further oxidized by CYP2E1 into catechol or hydroquinone, both of which are converted by myeloperoxidase (MPO) into reactive metabolites: 1,2-benzoquinone and 1,4-benzoquinone, respectively. These quinones can be reduced back to catechol and hydroquinone through the action of NAD(P)H:quinone oxidoreductase 1 (NQO1). Additionally, CYP2E1 can convert catechol and hydroquinone into 1,2,4-benzenetriol, another reactive metabolite.

A study involving 131 participants found that median trans,trans-muconic acid levels were approximately 2.5 times higher in smokers than in nonsmokers. Moreover, individuals who smoked more than 20 cigarettes per day had higher median trans,trans-muconaldehyde concentrations compared to those who smoked 10 or fewer cigarettes per day or those in the 11–20 cigarette-per-day range. Another study examining 136 smokers found a correlation between urinary trans,trans-muconaldehyde levels and the number of cigarettes smoked daily. Research conducted in Iran's Golestan Province, where cancer incidence is high, showed that cigarette and hookah smokers had significantly elevated levels of trans,trans-muconic acid. The median creatinine-adjusted levels were 65.1 µg/g for cigarette smokers and 82.4 µg/g for hookah smokers, compared to a median level of 30.2 µg/g among nonsmokers.

## REFERENCES


1. Benzene Toxic and Hazardous Substances. Occupational Safety and Health Administraton (OSHA) [Internet]. 2021. Available from: https://www.osha.gov/laws-regs/regulations/standardnumber/1910/1910.1028
2. Chaiklieng S, Tongsantia U, Suggaravetsiri P, Autrup H. Assessment of Exposure to Benzene Among Gasoline Station Workers in Thailand: Risk Assessment Matrix Methods. Int J Environ Res Public Health. 2025;22(3):397.
3. Services USD of H and H. Agency for Toxic Substances and Disease Registry-ATSDR. 1999;
4. American Conference of Governmental Industrial Hygienists (ACGIH). 2024 TLVs and BEIs Special Note to User. 2024;1–302.
5. Abplanalp W, DeJarnett N, Riggs DW, Conklin DJ, McCracken JP, Srivastava S, et al. Benzene exposure is associated with cardiovascular disease risk. PLoS One. 2017;12(9):e0183602.
6. Malovichko M V, Abplanalp WT, McFall SA, Taylor BS, Wickramasinghe NS, Sithu ID, et al. Subclinical markers of cardiovascular toxicity of benzene inhalation in mice. Toxicol Appl Pharmacol [Internet]. 2021;431:115742. Available from: https://doi.org/10.1016/j.taap.2021.115742
7. Zelko IN, Dassanayaka S, Malovichko M V, Howard CM, Garrett LF, Uchida S, et al. Chronic Benzene Exposure Aggravates Pressure Overload-Induced Cardiac Dysfunction. Toxicol Sci. 2021 Dec;185(1):64–76.

8. Chaiklieng S, Tongsantia U, Suggaravetsiri P, Autrup H. Altered haematological parameters in gasoline station workers due to benzene exposure. Safety. 2024;10(2):38.
9. Nomiyama K, Nomiyama H. Respiratory elimination of organic solvents in man: Benzene, toluene, n-hexane, trichloroethylene, acetone, ethyl acetate and ethyl alcohol. Int Arch Arbeitsmed. 1974;32(1–2):85–91.
10. Srbova J, Teisinger J, Skramovsky S. Absorption and elimination of inhaled benzene in man. 1950;
11. Pekari K, Vainiotalo S, Heikkilä P, Palotie A, Luotamo M, Riihimäki V. Biological monitoring of occupational exposure to low levels of benzene. Scand J Work
12. Etzel RA, Ashley DL. Volatile organic compounds in the blood of persons in Kuwait during the oil fires. Int Arch Occup Environ Health. 1994;66(2):125–9.
13. Vogel E, Günther H. Benzene Oxide-Oxepin Valence Tautomerism. Angew Chemie Int Ed English. 1967;6(5):385–401.
14. Parke D V, Williams RT. Studies in detoxication. 54. The metabolism of benzene.(a) The formation of phenylglucuronide and phenylsulphuric acid from [14C] benzene.(b) The metabolism of [14C] phenol. Biochem J. 1953;55(2):337.