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The Impact of Occupational Exposure to Aromatic Hydrocarbons on the Functional State of the Liver and Hematopoietic System

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Abstract

Workers in coke plants are potentially exposed to emissions from coke ovens, which contain hundreds of chemicals and are primarily composed of aromatic hydrocarbons and volatile organic compounds. Within the framework of this study, the primary objective was to investigate the impact of aromatic hydrocarbon vapors on the functional state of the liver and hematopoietic system, as well as to determine the association between occupational exposure to aromatic hydrocarbons and toxic damage to the liver and hematopoietic system. The liver function profile included serum levels of liver enzymes aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transpeptidase (GGT), and total bilirubin. To assess the functional state of the hematopoietic system, basic blood parameters (erythrocytes, leukocytes, hemoglobin, and platelets) were determined. This study was conducted as a retrospective study including 100 workers from the coke industry, who were divided into two groups: the experimental group consisting of 50 coke plant workers employed at workplaces exposed to aromatic hydrocarbon vapors, and the control group consisting of 50 administrative workers and others not exposed to aromatic hydrocarbon vapors. The obtained results indicate that there is a statistically significant difference in AST, ALT, GGT, and total bilirubin levels between the experimental and control groups. AST values were 39% higher in the experimental group compared to the control group, ALT values were higher by 67.7%, GGT values by 91.5%, and total bilirubin values by 14.5%. Statistically significant differences in blood parameters (erythrocytes, hemoglobin, leukocytes, and platelets) between the experimental and control groups were not found. These findings suggest that routine liver func-

tion monitoring may serve as an effective early screening measure for workers chronically exposed to aromatic hydrocarbon vapors.

Keywords

Organic Solvents, Aromatic Hydrocarbons, Blood Parameters, Liver Enzymes

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) are highly problematic environmental pollutants due to their chemical stability and inertness, which result from the conjugated π electrons of aromatic rings. These compounds can exist in gaseous or solid phases; PAHs with a smaller number of rings and lower molecular mass are typically found in the gaseous phase, whereas those with a larger number of rings and higher molecular mass exhibit more pronounced carcinogenic effects [1]. Concerns regarding the exposure of workers in chemical industries engaged in the production of benzene, tar, coke, and other derivatives have been growing in recent decades. Numerous studies have demonstrated that coke plant workers are frequently exposed to elevated concentrations of carcinogenic monocyclic and polycyclic aromatic hydrocarbons, which pose serious occupational and public health risks [2]-[4].

Acute benzene poisoning typically occurs accidentally, most often during maintenance or leakage incidents, leading to neurological and cardiovascular symptoms, while chronic poisoning results from prolonged inhalation of lower concentrations [5]. These conditions have a long latent period, with symptoms potentially appearing years after exposure has ceased [5].

Coke production emits large volumes of coke oven gas, leading to environmental pollution and exposure not only for workers but also for nearby populations, similar to other industrial processes. During the coking cycle, significant amounts of chemicals are released during coal charging, coke pushing, and through leakage from numerous cracks in oven doors and charging ports. The primary chemicals involved include polycyclic aromatic hydrocarbons (PAHs), such as benzo[a]pyrene, and monocyclic aromatic hydrocarbons (MAHs), such as benzene [3]. The working environment in coke plants can negatively affect workers exposed to coke oven emissions containing PAHs, which are formed and released into the environment through the pyrolysis of coal. Some studies have shown that exposure to PAHs from coke ovens continues to pose a health risk for both workers and the general population. Workers in coke plants remain at risk and should be monitored for adverse effects of long-term exposure. Occupational exposure to aromatic hydrocarbons generally indicates that such workplaces involve specific working conditions, which include periodic medical examinations of workers. These routinely comprise monitoring liver enzyme levels, namely the activity of aspar-

tate aminotransferase (AST), alanine aminotransferase (ALT), and gamma-glutamyl transferase (GGT) in serum as well as complete blood count (CBC) analysis. Although these indicators are primarily considered to reflect recent exposure, elevated liver enzyme values may also suggest previous exposure to hepatotoxic substances in the work environment that has since been discontinued or significantly reduced [6].

The primary routes of potential human exposure to coke oven emissions are inhalation and dermal contact. Inhalation is the main absorption route for volatile organic substances, as these are liposoluble vapors that readily cross the alveolar-capillary membrane [4]. Absorption via skin and the digestive tract is also possible. Although benzene is readily absorbed through the skin, a significant portion of dermally applied benzene evaporates from the skin surface. Data suggest that benzene is distributed throughout the body following absorption into the bloodstream. Once absorbed, solvents are transported by the blood to tissues and organs, where they undergo biotransformation (primarily in the liver and kidneys) or accumulate [5]. Distribution is greatest in lipid-rich tissues (adipose tissue, nervous system, and liver) and blood-rich organs (heart and muscles). Individuals with higher adiposity accumulate more solvents and eliminate them more slowly. At low exposure levels, aromatic hydrocarbons are rapidly metabolized and excreted mainly as conjugated metabolites in urine. At higher exposure levels, metabolic pathways appear to become saturated, and a large proportion of absorbed hydrocarbons are excreted unchanged in exhaled air.

The metabolism of most volatile organic substances occurs in the liver through the cytochrome P450 enzyme system, producing metabolites that are generally less biologically active [6]. However, some metabolites and intermediates may be more toxic than the parent compound, as in the case of benzene metabolism into toxic benzoquinones and trans, trans-muconaldehyde. Benzene and related aromatic hydrocarbons undergo hepatic biotransformation primarily through the cytochrome P450 system, generating metabolites that contribute to systemic toxicity. However, the current study does not address mechanistic aspects, focusing instead on functional changes in hepatic and hematological parameters among exposed workers [7].

Accumulation in the body can lead to poisoning even years after exposure has ceased. Elimination occurs primarily via the lungs in unchanged form, and to a lesser extent through the kidneys, skin, and glands. The rate and proportion of pulmonary excretion depend on the dose and mode of exposure. Qualitatively, the metabolism and elimination of benzene appear similar in humans and laboratory animals, although directly comparable studies are lacking [8]. Some compounds from this group cause damage during their elimination, *i.e.*, while passing through the body.

Workers employed in chemical industries producing coke, benzene, tar, and other products are exposed to emissions from coke ovens, released into the environment during coal pyrolysis. These emissions consist of various volatile organic

compounds, particularly aromatic hydrocarbons, which are primarily metabolized in the liver and excreted via bile. An epidemiological study demonstrated that workers with long-term exposure to aromatic hydrocarbon vapors had an increased risk of lung and prostate cancer [9]. Respiratory effects have been documented in humans after acute exposure to benzene vapors [10]. Mucosal irritation was observed in 80% and dyspnea in 67% of workers occupationally exposed to more than 60 ppm for up to 3 weeks. Nasal irritation and sore throat were reported in workers exposed to 33 - 59 ppm of benzene for over a year [11]. In addition to lung damage, coke oven emissions also have adverse effects on the cardiovascular system [12]. Workers exposed to high concentrations of coke oven emissions are more likely to develop hypertension and abnormal electrocardiographic findings compared to those exposed to lower concentrations.

Metabolism in the liver can generate toxic metabolites, raising concerns that the liver may be particularly sensitive to injury associated with aromatic hydrocarbon vapors. The liver plays a key role in the metabolism of proteins, lipids, and carbohydrates, as well as detoxification processes through urea synthesis, amino acid metabolism, and deamination. Chronic occupational exposure to low concentrations of aromatic hydrocarbons is believed to cause kidney and liver damage in exposed individuals, which is often difficult to identify [13].

Exposure to aromatic hydrocarbons also affects hematological parameters and the central nervous system, causing neurological dysfunction. Benzene exposure impacts the central nervous system (depression, ataxia, and confusion) and the hematopoietic system (anemia, leukopenia, thrombocytopenia, or pancytopenia after chronic exposure).

Epidemiological studies of individuals occupationally exposed to various levels of benzene over medium and long-term periods also point to hematological effects. The main limitations of these studies include uncertainty in historical exposure assessment, concurrent exposure to other chemicals, and the lack of adequate control groups. Nevertheless, sufficient evidence indicates that the hematopoietic system is a critical target of benzene toxicity. Inhalation of benzene at levels exceeding occupational exposure limits (8 hours, 1 ppm) over several years can lead to reductions in circulating blood cell counts, which may be severe enough to constitute clinical pancytopenia. Continuous exposure to benzene can also result in aplastic anemia or leukemia [14].

The objective of this study is to determine the association between occupational exposure to aromatic hydrocarbons and toxic damage to the liver and hematopoietic system by analyzing serum liver enzyme levels (AST, ALT, GGT, and total bilirubin) in workers occupationally exposed to aromatic hydrocarbons.

Although numerous international studies have established the hepatotoxic and hematotoxic effects of aromatic hydrocarbons, data from industrial settings in Southeastern Europe remain scarce. Occupational hygiene practices, workplace conditions, and medical surveillance programs vary considerably between regions. Therefore, this study aimed to assess the functional impact of aromatic hy-

drocarbon exposure in a local industrial cohort to contribute region-specific data and highlight the need for systematic biological monitoring in the regional coke industry.

2. Materials and Methods

2.1. Ethic Consideration

The study was conducted only upon obtaining a positive opinion from the Ethics Committee, ensuring that the research plan does not compromise the subjectivity and integrity of individuals involved, in accordance with Good Clinical Practice guidelines (ICH-GCP).

2.2. Study Participants and Inclusion Criteria

This research was conducted as a retrospective study including 100 individuals employed in the chemical industry engaged in the production of coke, tar, and benzene in the Federation of Bosnia and Herzegovina. The experimental group consisted of 50 workers with at least two years of professional experience at workplaces exposed to aromatic hydrocarbon vapors (including positions such as raw benzole distillers, pipe and washer operators, coke oven door operators, coke and coal handlers, maintenance fitters, and gas condensation analysts). Workers in the experimental group used personal protective equipment as prescribed in the systematic examination guidelines provided by Plava Medical Group. The control group included 50 administrative employees who were not directly exposed to aromatic hydrocarbon vapors during working hours. Occupational exposure classification was determined based on official safety records, job descriptions, and periodic workplace air monitoring data from the company's occupational health department. Both groups underwent biochemical and hematological testing as part of routine medical examinations between 05 January 2023 and 01 June 2023, with blood sampling performed at "PZU Plava Medical Group" in Tuzla. Individuals with less than two years of work experience, as well as those with the following conditions, were excluded from the study: diabetes, hypertension, alcohol consumption, smoking, pre-existing liver or hematopoietic system disorders, and other diseases that could negatively affect liver or hematopoietic system function. Adherence to exclusion criteria, including alcohol consumption and smoking, was verified through self-reported questionnaires and review of medical records obtained during routine medical examinations. This approach minimized the potential influence of confounding factors unrelated to occupational exposure.

2.3. Sample Collection and Biochemical-Hematological Assessment

Blood samples were collected between 08:00 and 10:00 a.m. after an overnight fast of at least 8 hours to minimize diurnal and postprandial variability in biochemical and hematological parameters. Serum levels of AST, ALT, GGT, total bilirubin, and complete blood count (CBC) parameters were measured during routine med-

ical examinations, and the obtained values were retrieved from the medical records of PZU "Plava Medical Group". Blood samples were collected by venipuncture: 6 mL of blood was drawn into a tube containing a clot activator. After centrifugation for 10 minutes at 3500 rpm, serum was obtained for liver enzyme analysis. Serum AST, ALT, GGT, and total bilirubin were determined using a Biobase BK 600 biochemical analyzer via spectrophotometry, with internal and external quality controls performed prior to analysis. Complete blood count was determined from 3 mL of whole blood collected in an EDTA-anticoagulant tube. After inverting the tube 8 - 10 times, the analysis was performed using an automated hematology analyzer Medonic M51, which provides a 5-part differential count using impedance or electrical resistance measurement. Reference values for the analyzed parameters were as follows: AST (0 - 41 μ/L), ALT (0 - 40 μ/L), GGT (0 - 51 μ/L), total bilirubin (0 - 17 mmol/L), RBC (3.50 - 5.10 × 10^{12}/L), WBC (3.50 - 9.50 × 10^{9}/L), HGB (11.5 - 16.5 g/dL), PLT (125 - 350 × 10^{9}/L).

2.4. Statistical Analysis

The data were analyzed using non-parametric statistical methods. Basic statistical parameters, frequencies, and percentages were calculated, with results presented in tables and graphs. To evaluate the study objectives, the Mann-Whitney U test and independent-sample T-test were applied. Statistical analyses were performed using IBM SPSS Statistics software, Version 26.0 (IBM Corp., Armonk, NY, USA).

3. Results

The following section presents the results of the study, including the biochemical and hematological parameters measured in both the experimental group exposed to aromatic hydrocarbon vapors and the control group not exposed. The obtained results were subjected to statistical analysis in order to determine potential differences between the experimental and control groups. These findings provide a basis for evaluating the impact of occupational exposure to aromatic hydrocarbon vapors on workers' health. Table 1 shows the demographic characteristics and laboratory parameters of participants exposed to aromatic hydrocarbon vapors and the control group.

Table 1. Demographic characteristics and laboratory parameters of participants exposed to aromatic hydrocarbon vapors and the control group.

Parameter	Participants exposed to aromatic hydrocarbon vapors	Control Group
Number (N)	50	50
Age (Mean ± SD)	46.22 ± 9.24	49.9 ± 11.4
Duration of exposure (years)	6.3 ± 4.7	-
AST (μ/L)	28.04 ± 16.47	18.7 ± 6.10
ALT (μ/L)	39.7 ± 26.5	19.6 ± 9.88
GGT (μ/L)	52.7 ± 43.1	19.6 ± 11.8

Continued

Total bilirubin (μmol/l)	15.5 ± 3.91	13.4 ± 4.11
WBC (*L)	7.73 ± 2.30	7.07 ± 1.95
RBC (*L)	5.04 ± 0.33	4.93 ± 0.36
HGB (g/dL)	15.4 ± 0.93	15.1 ± 0.91
PLT (*L)	240.9 ± 48.7	240.5 ± 62.4

Table 2 shows the differences in AST, ALT, GGT, and total bilirubin between exposed and control groups.

Table 2. Differences in AST, ALT, GGT, and total bilirubin between exposed and control groups.

Parameter	Group of participants	N	Mean ± SD	Mann-Whitney test P < 0.05
AST (μ/L)	Participants exposed to aromatic hydrocarbon vapors	50	28.04 ± 16.47	P < 0.001
AST (μ/L)	Control group of participants	50	18.7 ± 6.10	
ALT (μ/L)	Participants exposed to aromatic hydrocarbon vapors	50	39.7 ± 26.5	P < 0.001
ALT (μ/L)	Control group of participants	50	19.6 ± 9.88	
GGT (μ/L)	Participants exposed to aromatic hydrocarbon vapors	50	52.7 ± 43.1	P < 0.001
GGT (μ/L)	Control group of participants	50	19.6 ± 11.8	
Total bilirubin (μmol/L)	Participants exposed to aromatic hydrocarbon vapors	50	15.5 ± 3.91	P < 0.001
Total bilirubin (μmol/L)	Control group of participants	50	13.4 ± 4.11	

Using the Mann-Whitney U test (Table 2), the values of AST, ALT, GGT, and total bilirubin were analyzed in participants exposed to aromatic hydrocarbon vapors (working in coke, tar, and benzene production) and in the control group of participants not exposed to aromatic hydrocarbon vapors (working in administrative positions, as porters, or drivers). A statistically significant difference was observed in ALT values ($P < 0.001$). The results of the statistical test showed that ALT values (39.7 ± 26.5) in participants exposed to aromatic hydrocarbon vapors were significantly higher than in participants not exposed to vapors at the workplace (19.6 ± 9.88). A statistically significant difference was also found in GGT values ($P < 0.001$). The results showed that GGT values (52.7 ± 43.1) were significantly higher in participants exposed to aromatic hydrocarbon vapors compared to participants who were not exposed (19.6 ± 11.8). According to the Mann-Whitney U test, differences were also observed in AST values ($P < 0.001$) and total bilirubin ($P = 0.004$).

Table 3 and Table 4 show the differences in AST and ALT concentrations between participants exposed to aromatic hydrocarbon vapors and unexposed con-

trols.

Table 3. Differences in AST concentrations between participants exposed to aromatic hydrocarbon vapors and unexposed controls.

Parameter	Group of participants	N	Mean ± SD	Independent samples T-test, P < 0.05
AST (μ/L)	Participants exposed to aromatic hydrocarbon vapors	50	28.04 ± 16.47	P < 0.001
AST (μ/L)	Control group of participants	50	18.7 ± 6.10	

Using the independent samples T-test, significant differences were found in AST values (P < 0.001) between the group of participants exposed to aromatic hydrocarbon vapors and the control group of participants who were not exposed.

Table 4. Differences in ALT concentrations between participants exposed to aromatic hydrocarbon vapors and unexposed controls.

Parameter	Group of participants	N	Mean ± SD	Independent samples T-test, P < 0.05
ALT (μ/L)	Participants exposed to aromatic hydrocarbon vapors	50	39.7 ± 26.5	P < 0.001
ALT (μ/L)	Control group of participants	50	19.6 ± 9.88	

Using the independent samples T-test, ALT values were analyzed in the group of participants exposed to aromatic hydrocarbon vapors (working in coke, tar, and benzene production) and the control group of participants not exposed to aromatic hydrocarbon vapors (working in administration, as gatekeepers, or drivers). A statistically significant difference in ALT values was confirmed (P < 0.001).

Table 5 shows the differences in in GGT concentrations between participants exposed to aromatic hydrocarbon vapors and unexposed controls. The difference in gamma-glutamyl transferase values was statistically significant with P < 0.001. The test results were obtained using the independent samples T-test.

Table 5. Differences in GGT concentrations between participants exposed to aromatic hydrocarbon vapors and unexposed controls.

Parameter	Group of participants	N	Mean ± SD	Independent samples T-test, P < 0.05
GGT (μ/L)	Participants exposed to aromatic hydrocarbon vapors	50	52.7 ± 43.1	P < 0.001
GGT (μ/L)	Control group of participants	50	19.6 ± 11.8	

Table 6 shows the differences in total bilirubin concentrations between the experimental group and the control group not exposed to aromatic hydrocarbon vapors. By comparing total bilirubin values between the group of participants exposed to aromatic hydrocarbon vapors and the control group not exposed, a sta-

tistically significant difference was found (P = 0.009).

Table 6. Differences in total bilirubin concentrations between the experimental group and the control group not exposed to aromatic hydrocarbon vapors.

Parameter	Group of participants	N	Mean ± SD	Independent samples T-test, P < 0.05
Total bilirubin (μmol/L)	Participants exposed to aromatic hydrocarbon vapors	50	15.5 ± 3.91	P < 0.009
Total bilirubin (μmol/L)	Control group of participants	50	13.4 ± 4.11	

Table 7 shows the differences in blood parameters (leukocytes, erythrocytes, hemoglobin, and platelets) between exposed and control groups. According to the Mann-Whitney U test, no statistically significant differences were found in leukocyte, erythrocyte, hemoglobin, and platelet parameters.

Table 7. Differences in blood parameters (leukocytes, erythrocytes, hemoglobin, and platelets) between exposed and control groups.

Parameter	Group of participants	N	Mean ± SD	Mann-Whitney test P < 0.05
WBC (10^9/L)	Participants exposed to aromatic hydrocarbon vapors	50	7.73 ± 2.30	P = 0.18
WBC (10^9/L)	Control group of participants	50	7.07 ± 1.95	
RBC (10^{12}/L)	Participants exposed to aromatic hydrocarbon vapors	50	5.04 ± 0.33	P = 0.09
RBC (10^{12}/L)	Control group of participants	50	4.93 ± 0.36	
HGB (g/dL)	Participants exposed to aromatic hydrocarbon vapors	50	15.4 ± 0.93	P = 0.11
HGB (g/dL)	Control group of participants	50	15.1± 0.91	
PLT (10^9/L)	Participants exposed to aromatic hydrocarbon vapors	50	240.9 ± 48.7	P = 0.88
PLT (10^9/L)	Control group of participants	50	240.5 ± 62.4	

4. Discussion

Exposure of humans to benzene and other polycyclic aromatic hydrocarbons (PAHs) has been associated with multiple toxicities affecting hematological, hepatic, immunological, and chromosomal functions, as well as with an increased risk of carcinogenesis. However, the precise mechanisms underlying the toxic effects induced by benzene are not yet fully understood. Therefore, a thorough understanding of the health consequences of benzene exposure is crucial for developing risk assessment approaches in affected communities [15]. Elevated levels of liver serum enzymes may result from the overproduction or release of enzymes from hepatocytes in response to hepatocellular injury or cell death, although the exact mechanisms underlying these processes in benzene-exposed individuals re-

main to be clarified.

In 1998, Michailova *et al.* reported a significant increase in serum AST and ALT activity among workers in the petroleum industry [16]. Similar findings were reported by Akintonwa and Oladele in 2003 among gas station attendants exposed to benzene [17]. Likewise, in 2004, Saadat and Ansari-Lari, in a study involving 56 gas station workers in Shiraz, reported significantly elevated plasma creatinine levels, increased serum ALT and AST activity, and reduced albumin and protein levels in the exposed group compared to the control group [18].

The aim of the present study was to assess the effects of benzene and other volatile organic compounds on hematological parameters and liver function. Since previous studies have confirmed that benzene and other PAHs contribute to liver and hematopoietic system impairment, we evaluated liver function by measuring serum levels of AST, ALT, GGT, and total bilirubin, and assessed hematopoietic function by examining red blood cells, white blood cells, platelets, and hemoglobin in individuals occupationally exposed to benzene and compared them with unexposed subjects (control group). ALT and GGT are useful biomarkers before liver damage becomes irreversible [19]. Solvent-induced liver disease can be prevented from progressing to irreversible liver injury by eliminating exposure, provided it is detected in its early stages [20]. Therefore, ALT and GGT in workers exposed to volatile organic compounds are valuable biomarkers for the early detection and monitoring of liver injury. Aromatic hydrocarbons such as benzene are metabolized primarily in the liver through cytochrome P450 enzymes, producing reactive intermediates such as benzene oxide and benzoquinones. These metabolites can induce oxidative stress, lipid peroxidation, and mitochondrial dysfunction, leading to hepatocellular injury and enzyme leakage into the bloodstream.

The study was designed as a retrospective analysis, and data were collected from the medical records of coke industry workers undergoing routine systematic medical examinations at Plava Medical Group. Liver enzyme and hematological tests were performed. All study participants were male, aged between 31 and 64 years. The mean age of exposed participants was 46.22 ± 9.24 years, while the mean age in the control group was 49.9 ± 11.4 years. The mean duration of employment among exposed participants was 6.3 ± 4.7 years (**Table 1**).

Our findings revealed significant differences in liver enzyme levels and total bilirubin between the experimental group exposed to vapors and the control group. The mean serum AST level in the exposed group was 28.04 ± 16.47 compared to 18.7 ± 6.10 in the control group, indicating a significant difference (**Table 2**). Similarly, the mean serum ALT level was 39.7 ± 26.5 in the exposed group, compared to 19.6 ± 9.88 in the control group, showing a statistically significant difference (**Table 2**). The mean GGT level in the experimental group was 52.7 ± 43.1, while in the control group it was 19.6 ± 11.8 (**Table 2**). Regarding total bilirubin, its mean value in the exposed group was 15.5 ± 3.91 compared to 13.4 ± 4.11 in the control group, which, according to the Mann-Whitney test, also

demonstrated a significant difference (P = 0.004; Table 2).

AST levels were 39% higher in the experimental group compared to controls, ALT levels were 67.7% higher, GGT levels were 91.5% higher, and total bilirubin levels were 14.5% higher in exposed participants compared to controls. Among 50 exposed participants, 4 (8%) had elevated AST above the reference value, 18 (36%) had elevated ALT compared to 2 (4%) in the control group (a statistically significant difference). In addition, 17 (34%) exposed participants had elevated GGT compared to 3 (6%) in the control group. Regarding total bilirubin, 6 (12%) of exposed participants had elevated values compared to 4 (8%) in the control group. Overall, liver enzymes and total bilirubin were significantly higher in benzene-exposed individuals compared to non-exposed controls (Table 2).

Similar findings were reported in a 2015 study of 200 gasoline station workers in Shiraz, which also identified significantly elevated liver enzymes among exposed individuals compared to controls [21]. It should be noted that shift work and circadian variation in hepatic metabolism may have influenced the results of that study, making direct comparison difficult.

Previous studies have reported varying results regarding benzene hematotoxicity depending on exposure time and concentration. In the occupational context, benzene hematotoxicity is well documented, and evidence of hematological consequences is largely convincing. Numerous studies of workers exposed to aromatic hydrocarbons have demonstrated that chronic exposure to benzene concentrations of 10 ppm or higher leads to adverse hematological effects, which worsen with increased levels of exposure. Animal studies support these findings, showing significant reductions in all three blood cell lineages (RBC, WBC, platelets), along with bone marrow hypocellularity, granulocytic hyperplasia, erythroid progenitor damage, and reduced colony-forming units at benzene concentrations ranging from 10 - 300 ppm.

Many authors have reported an increased risk of hematological malignancies in exposed workers [22]-[24]. In addition, altered blood and urine parameters, as well as DNA and chromosomal damage, have been identified [25] [26]. Hematological effects of benzene exposure generally result in reduced levels of WBC, hemoglobin, and platelets, as observed in multiple studies [27] [28]. A study among residents of Nanjing, one of the most industrialized regions of China with a strong petrochemical presence, included 240 residents from a contaminated area and 181 from control communities. It compared blood levels of benzene, toluene, xylene, and o-xylene, along with hematological parameters. Exposure durations ranged from 5 to 71 years. The contaminated group had significantly higher levels of aromatic hydrocarbons in the blood, unaffected by smoking status. Moreover, neutrophil, platelet, RBC, hematocrit, and hemoglobin counts were significantly lower in the exposed group, while monocyte and basophil counts were significantly higher compared to controls [29].

By contrast, some studies at lower benzene concentrations report different findings. Collins *et al.* (1991) found no significant correlation between benzene expo-

sure and abnormal hematological parameters in 200 exposed workers (0.01 - 1.4 ppm) compared to 268 unexposed workers in the same facility [30]. Similarly, in a Texas refinery with a mean benzene concentration of 0.53 ppm, hemoglobin, hematocrit, RBC, WBC, and platelet counts among workers with up to 21 years of exposure were within normal ranges. Rajia and Hall (2014), in a study of 60 workers (40 exposed to benzene vs. 20 unexposed), concluded that lower concentrations of benzene were not associated with reductions in RBC count [31].

The results of our study showed no significant differences in hematological parameters (RBC, WBC, platelets, hemoglobin) between exposed and control groups. Similar findings were reported in an Italian study evaluating hematological parameters (WBC, neutrophils, lymphocytes, monocytes, eosinophils, basophils, RBC, hemoglobin, platelets) among 153 Bulgarian petrochemical workers exposed to benzene (0.01 - 23.9 ppm) with at least one year of employment. The control group consisted of 50 unexposed employees from the same facility. Basophil counts were found to increase with exposure, while no differences were observed in other hematological outcomes [32].

Reduced blood cell counts were documented in two independent studies among Chinese workers, where exposure was measured as 4-week average benzene levels prior to blood collection. Qu *et al.* (2002) [32] reported decreased RBC, WBC, and neutrophils in exposed subjects compared to controls. Lan *et al.* (2004) [26] found significantly reduced levels of all leukocyte subtypes and platelets in workers exposed to 0.1 - 75 ppm compared to controls. One possible explanation is that benzene hematotoxicity in Chinese workers reflects higher cumulative exposures in the months before phlebotomy, resulting in specific toxicity to hematopoietic progenitor cells and a dose-dependent decline in peripheral blood counts. Differences in exposure duration, workplace concentrations, gender distribution (our study included only men vs. ~70% women in the Chinese study), alcohol use, nutrition, and other unmeasured confounders may explain the different outcomes.

This study has several limitations that should be acknowledged. First, its retrospective design limits the ability to establish causal relationships between exposure and outcomes. Second, the sample size of 100 participants may reduce the statistical power to detect subtle hematological differences. Third, quantitative data on the exact concentrations and duration of aromatic hydrocarbon exposure for each worker were not available, preventing dose-response analysis. Future research using prospective study designs and detailed exposure assessment is recommended to confirm these findings.

The composition and processing of raw coke oven gas is highly complex, containing not only ammonia, benzene, sulfur, and crude tar, but also a wide range of other hazardous pollutants. Therefore, individuals occupationally exposed to benzene and aromatic hydrocarbons should be carefully monitored over time for long-term toxic effects on bone marrow, liver, kidneys, and other target organs, as well as for the potential development of secondary malignancies. To achieve this, healthcare professionals must conduct regular examinations and laboratory

blood analyses in exposed populations, along with periodic assessments of pulmonary, cardiac, neurological, and other organ functions to monitor long-term effects of benzene and aromatic hydrocarbon exposure.

5. Conclusion

Evaporation of aromatic hydrocarbons has harmful effects on health, particularly in highly exposed groups of the population, including workers in certain industrial sectors. The mechanisms through which aromatic hydrocarbon vapors affect human health are not fully understood. Exposure of workers to volatile organic substances, primarily aromatic hydrocarbon vapors and especially benzene, leads to changes in the functional state of the liver and causes alterations in laboratory parameters of liver function (primarily transaminases and total bilirubin). Elevated levels of transaminases cannot be attributed to metabolic syndrome, since diabetes mellitus was an exclusion criterion, and individuals who consume alcohol and cigarettes were also not included in this study. Although no changes were observed in hematopoietic parameters, the findings may suggest that hepatotoxic effects manifest earlier or at lower cumulative doses than hematotoxic effects. This indicates that liver function may serve as a more sensitive indicator of early toxic impact from aromatic hydrocarbon exposure. Based on the obtained results, it can be concluded that aromatic hydrocarbon vapors are involved in altering liver function, while hematological parameters are still preserved and no significant differences were found compared to the control group. From an occupational health perspective, routine monitoring of liver function should be implemented as a primary screening measure for early detection of solvent-induced hepatotoxicity among coke plant workers.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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