Received: 25 July 2022 | Revised: 30 August 2022 | Accepted: 05 September 2022
DOI: 10.22034/ecc.2023.357811.1524

Journal of Medicinal and Pharmaceutical Chemistry Research
SAMI

FULL PAPER

# Study of some hematological and biochemical parameters among petrol stations workers in Baghdad city

**Abdulameer Hassan Abdulameer*** |**Saba Zuhair Hussein**

*Department of Chemistry, College of Science, University of Baghdad, Baghdad, Iraq*

***Corresponding Author:**
Abdulameer Hassan Abdulameer
Email: abdulameerhassan68@gmail.com
Tel.: +9647711659028

Filling workers at petrol stations are particularly susceptible to the occupational exposure to toxins that cause hematotoxicity and blood disorders like leukemia, aplastic anemia, and dysplastic bone marrow. Thus, this study aimed to evaluate hematological and biochemical alterations among petrol stations worker in Baghdad city. A 102 male filling station workers from different regions of Baghdad governorate were divided into three groups according to exposed duration (exposed groups) and 50 subjects (control group), were included. Each samples were analyzed and compared for CBC, protein profile, and renal function between the groups. The results indicated no significant differences in BMI, MCH, MCHC, and WBC for the exposed compared with control groups. A highly significant increase was found in HB, RBC, HCT, and BASO with a highly significant decrease in PLT, RDW, and MPV in exposed group than control. Moreover, the long duration of exposure to petrol showed a significant decrement in MCV, NEU, and MONO with a significant increment in LYM and EOS. Also, there was a significant decrease in total protein and globulins levels with a significant increase in albumin, urea, and creatinine in exposed than control groups. In conclusion, the current results showed that prolonged exposure to petrol products for petrol filling workers may increase the chance of developing blood and kidney related disorders.

**KEYWORDS**

Petrol station; CBC; urea; creatinine; total protein; albumin.

## Introduction

Gasoline is a complex mixture of low-molecular-mass compounds, primarily paraffinic, naphthenic, olefinic, and aromatic with carbon numbers typically between 3 and 11. The type of crude oil used to make it and the kind of processing of various refinery streams from which it is blended both affect the precise chemical makeup of gasoline. Benzene, toluene, and xylenes make up a significant group of the aromatic components of gasoline [1]. It is a petroleum-derived volatile and flammable liquid mixture primarily used for internal machine combustion. It is used in vehicles, synthetic solvents, pesticides, and cleaners among other things. It is made up of various aromatic and inorganic-organic compounds. Any of its products is particularly carcinogenic to humans [2]. Every day, in the outdoors, at work and at home, everyone is exposed to a small amount of gasoline however people who live in cities or the industrial areas are generally exposed to higher levels of gasoline in the air than those living in the rural areas [3]. The International Agency for Research on Cancer (IARC) has suggested that human exposure to gasoline vapors may be carcinogenic, based on the well-known carcinogenicity of some components like benzene [4].

Benzene, toluene, ethylene, and xylene which are also known as volatile organic compounds (VOCs), are responsible for many toxicological effects associated with gasoline exposure [5].

Fuel stations are a well-known source of air pollution, containing complex mixtures of various petroleum products and vapors. Petrol vapors are made up of a complicated mix of hydrocarbons that are aliphatic and cyclic compounds (about 95%), with only 2% being aromatic. The benzene concentration about 1-5 % which is a highly flammable, colorless, and volatile liquid found in the air as a result of coal combustion, gasoline service stations, motor vehicle exhaust, and evaporation [6]. Inhaling small amounts of petrol vapors can cause irritation to the nose and throat, as well as headaches, dizziness, nausea, vomiting, confusion, and breathing problems. Rashes, redness, and swelling are also some side effects of coming into contact with gasoline on the skin [7].

Hematological abnormalities have been linked to the occupational exposure to dangerous chemicals. Employees who work at gas stations, such as attendants, are at risk of being exposed to gasoline and developing cancers [8]. These substances levels may accumulate in the air that have negative health, economic, or aesthetic consequences. Gasoline vapors, cause damage to constructive tissue, genetic mutations, decreased bone marrow cell generation, severe anemia, and immunodeficiency [9]. Several chemicals have been linked to changes in the hematological profile. The negative health effects of gasoline exposure may be primarily related to hematopoietic system impairment, including bone marrow depression, pancytopenia, aplastic anemia, and an increased risk of developing cancer (acute myeloblastic leukemia). In addition, microcytosis, as a morphological effect occurs on red blood cells (RBCs) [10].

Exposure to petroleum hydrocarbons has also been linked to renal dysfunction due to oxidative stress, which causes lipid peroxidation and lowers the antioxidant defense mechanism [11]. The kidneys are responsible for maintaining blood volume, regulating mineral content in the bloodstream, and removing metabolic waste from the body. The kidneys filter unwanted waste materials from the blood and maintain body water and chemical levels [12]. Total serum protein, also known as total plasma protein or complete protein, is a biochemical test used to determine the amount of protein in blood plasma or serum. The total serum protein is made up of a number of different proteins like albumin and globulins with any changes in their amounts can reveal diagnostic information [13]. The total protein levels or an imbalance of albumin to globulins ratio can associated with many diseases and these medical conditions include chronic inflammation, viral hepatitis, or HIV, and bone marrow diseases such as multiple myeloma [14]. Thus, the present study aimed to assess the effect of fuel vapor exposure on some hematological and biochemical parameters among petrol station workers in Baghdad city.

## Materials and methods

In this study, 102 male Iraqi filling station workers from different regions of the Governorate of Baghdad were included. The employees were chosen from the filling stations in Baghdad's Al-Kilani, Al-Mustansiriya, Al-Khalisa, Al-Muthanna, Palestine, Al-Idrisi, and Bab Al-Moadham and the samples took place from October 2021 to the end of November 2021. According to the length of fuel exposure, the participants were split into three groups. A group of fifty (50) healthy volunteers in the same age range were chosen as the control group. Table 1 lists the groups' specifications.

**TABLE 1** The details of individuals groups

| Characteristics | Exposed groups | | | Non-exposed group |
|---|---|---|---|---|
| | Group 1 G1 (n=25) | Group 2 G2 (n=50) | Group 3 G3 (n=27) | Control C (n=50) |
| Age (Years) | 25-50 | 30-55 | 37-60 | 24-60 |
| Duration of exposure (Years) | 1-10 | 11-19 | ≥ 20 | - |
| No. of working hours/day | 10 | 10 | 10 | - |

The following conditions were disqualified: alcoholism, drug addiction, diabetes mellitus, use of antioxidant, vitamin supplements, obesity, rheumatoid arthritis, and liver conditions that might affect the study. The University of Baghdad, College of Science and the management of the country's gas stations gave their approvals for this study to be carried out. All volunteers provided their permission before having their samples used for research and this information was disclosed to them.

The BMI (Kg/m$^2$) was calculated based on divided the weight (Kg) by height (m$^2$) for all subjects [15]. The World Health Organization (WHO) defined underweight as having a BMI under 18 and obese as having a BMI over 30. The venous blood around 5 mL was collected from the volunteers included in this study. Each blood sample was split into two parts. The first part (4 mL) of blood was used on the same day to measure the CBC, while the second part (1mL) was allowed to clot in room temperature and centrifuged at (1500 x g) for 10 min to collect serum. Then, the serum was stored at (-20 °C) until used foranalysis. Complete Blood Counts were obtained from the whole blood specimens on the same day of collection by using an automated Ruby Hematology Analyzer. The total proteins and albumin content was determined by using a commercially available assay kit (spinreact, spain), and the serum globulins concentration was calculated via the following equation:

Globulins conc. (g/dl) = TP conc. (g/dl) – Albumin conc. (g/dl)

The serum urea and creatinine content was determined by using a commercially available assay kit (Linear chemicals, spain). A spectrophotometer was used to measure the serum levels of urea and creatinine [16]. An enzymatic colorimetric method was utilized to assess the level of blood urea (BU). The color intensity is proportional to the urea concentration in the sample. While the method for estimating the serum creatinine is based on the picrate kinetic reaction.

The data throughout this work was expressed in the form of mean value ± the standard deviation (mean±SD). The data were compared by using SPSS version 26 and Excel 2013 by One-Way ANOVA; where the difference is considered as highly significant when ($p<0.001$), significant when ($p<0.05$), and non-significant when ($p>0.05$).

## Results and discussion

The city of Baghdad suffers from the large and continuous environmental problems in the workplace, especially workers at petrol stations. The workers at petrol stations were chosen for this study because they are at a high risk of exposure to benzene and it was crucial because the long-term exposure to benzene has been linked to known effects like cancer, bone marrow depression, and neurological, and hematological toxic effects [8]. The petrol stations workers are frequently exposed to the various dangerous toxins and noxious vapors; benzene fumes are among the most dangerous toxins. They are linked to an increased risk of hematological malignancies, particularly acute nonlymphocytic (myeloid) leukemia, and can lead to abnormal alterations in the functioning of many vital organs [17].

The exposed groups included in this study had the mean ages of 42.20±8.99, 44.46±7.15, and 50.22±5.91 years old for G1, G2, and G3, respectively, compared with 42.44±9.43 for the control (C) group. Table 2 presented the BMI levels and hematological parameters in the exposed and control groups. The mean value of BMI, MCH, MCHC, and WBC revealed no significant differences for the exposed groups compared with that of control group. The results showed the presence of a highly significant increase of HB in exposed groups of G2 and G3 compared with control except G1 that showed no significant difference, while no significant differences is found upon the comparison of this parameter between exposed groups (G1, G2, and G3). A highly significant increase was found in RBC, HCT, and BASO in all exposed groups compared with the control group and no significant differences between exposed groups (G1, G2, and G3).

**TABLE 2** The comparison of BMI and hematological parameters in all studied groups.

| Dependent Variables | Groups | | | |
|---|---|---|---|---|
| | C | G1 | G2 | G3 |
| BMI (Kg/m$^2$) | 26.92±2.48 | 26.48±2.16 | 26.64±2.15 | 27.02±1.94 |
| HB(g/dl) | 14.08±1.18 | 14.77±1.61 | 15.14±1.58 $^{a**}$ | 15.22±1.71 $^{a**}$ |
| RBC (10e$^3$ /uL) | 4.85±0.42 | 5.12±0.55 $^{a**}$ | 5.34±0.54 $^{a**}$ | 5.34±0.64 $^{a**}$ |
| HCT (%) | 41.67±2.92 | 44.80±4.93 $^{a**}$ | 44.78±4.68 $^{a**}$ | 44.11±3.84 $^{a**}$ |
| MCV(fL) | 86.81±5.87 | 84.17±5.50 | 84.97±7.24 | 83.97±5.57 $^{a*}$ |
| MCH (pg) | 28.97±1.93 | 28.70±2.29 | 28.58±2.78 | 28.66±3.28 |
| MCHC(g/dl) | 33.86±2.15 | 34.05±0.98 | 33.96±1.49 | 33.63±1.41 |
| RDW (%) | 12.84±1.18 | 11.15±0.64 $^{a**}$ | 11.63±1.42 $^{a**}$ | 11.80±1.36 $^{a**}$ |
| PLT (10e$^3$ /uL) | 254.18±65.95 | 234.40±43.52 | 213.56±48.96 $^{a**}$ | 212.81±35.92 $^{a**}$ |
| MPV (fL) | 8.82±1.08 | 6.48±1.31 $^{a**}$ | 6.31±1.22 $^{a**}$ | 6.76±1.48 $^{a**}$ |
| WBC (10e$^3$ /uL) | 7.53±1.82 | 7.75±1.88 | 7.92±1.79 | 8.01±1.64 |
| NEU% | 56.56±6.94 | 56.44±6.37 | 54.68±10.06 | 53.33±6.34 $^{a*}$ |
| LYM% | 32.84±7.02 | 33.98±6.51 | 34.02±8.03 $^{a*}$ | 35.60±6.86$^{ab*}$ |
| MONO% | 6.87±1.64 | 6.28±1.88 | 6.54±2.40 | 5.64±1.38 $^{a*}$ |
| EOS% | 2.81±1.59 | 2.75±1.22 | 3.68±2.33 $^{a*}$ | 3.68±2.42 $^{a*}$ |
| BASO% | 0.89±0.20 | 1.15±0.28 $^{a**}$ | 1.08±0.36 $^{a**}$ | 1.07±0.28 $^{a**}$ |

(a)-significant difference between C and (G1,G2, and G3)
(b)-significant difference between G1 and (G2 and G3)
(c)- significant difference between G2 and G3
*p<0.05 ; **p<0.01

There are no statistically significant differences in BMI between workers in petrol stations (exposed groups) and healthy people, indicated that is all not obese (below 30) which is consistent with the previous studies [18,19].

The measured hematological parameters included hemoglobin (Hb), red blood cells (RBC), pack cell volume (PCV), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) as well as white blood cell (WBC) and its differentials [20]. The initial parameters impacted by systemic toxicity are hematological parameters. Therefore, determining the negative impact of foreign substances on blood components *in vivo* can be done by evaluating hematological parameters [21]. According to the present study, the mean values of HB and RBC for exposed groups showed a significant increment compared with control. These findings was in agreement with the previous studies [17,22]. In contrast, these results were not in agreement with other study [23]. On the other hand, there were no discernible differences between the mean serum RBC counts in relation to the work length in petrol stations, which was in accordance with other study [2]. Our results also revealed a high significant increase in HCT value in all exposed groups than control which was agreed with a previous study [24]. However, another study was not agreed with our finding [23]. The possible reason for variations with our study because the exposure years in the previous study was not considered. Generally, the fact that gasoline is known to contain the high amounts of carbon monoxide, which can enter the blood via the respiratory system, may be the cause of an increase in RBC parameters in gasoline filling employees. The carboxyhemoglobin formation, which interferes with the ability of blood to carry oxygen and causes tissue hypoxia, was facilitated by the strong affinity of the molecule for HB up to 200 times greater than that for oxygen. The erythropoiesis was stimutes by tissue hypoxia which then caused to the production of more RBC, then leads to rises the levels of HB and HCT [22].

Moreover, our results indicated that the counts of MCH, MCHC, and WBC had no significant differences in exposed groups than those of control which were within the normal ranges and this findings was in the line with the prior studies [17,23].

Our results also reported that the mean value of MCV in long duration exposed workers (G3) showed a significant decrement compared with the control which was a consistent result had been previously reported [22]. The evidence revealed that the altered membranes might be the cause of a decrease in cellular size. Although its mechanisms are unclear, benzoene may affect the cell's pliability and permeability. In addition, the cell membrane is damaged due to the free radicals formation as a result of the petrol chemicals metabolism [12].

The current study indicate a significant decrease in PLT count in exposed groups than control which was supported by previous finding [17]. On the other hand, this result was contrary to the prior study who noted a significant increase in PLT count in petrol filling workers compared with healthy controls [22]. Another study which was proved that there are no statistically significant differences in PLT between gas station workers and healthy people that do not agree with our study [25]. However, the variations in sample size, socio-demographic variables, and exposure duration might be the possible reason for the discrepancies between the studies.

Furthermore, according to our results, the mean RDW and MPV value of exposed groups demonstrated a significant decrement compared with control group. A prior study confirmed that there are statistically significant decreased in RDW and MPV, which agree with our study [42]. Whereas, other study [22] reported a significant increase in RDW in petrol station workers which was disagreed with our findings.

Furthermore, our study showed a significant increase in mean values of LYM, EOS, and BASO with significant decrease in NEU and MONO in exposed groups (especially long work duration) than control. A previous study found a significant increases in LYM in petroleum exposed workers than non-exposed [27]. Also in another study, there was a statistically significant increase in LYM count with no significant difference in NEU count of petrol filling workers than healthy control [22]. Meanwhile, this study confirmed that there are no statistically significant differences in LYM and EOS between petrol station workers and healthy people [28]. The study reported no statistically significant differences in NEU with a significant decrease in LYM and MONO between petrol station workers and healthy people [26]. A significant decrease was noted in LYM, NEU, and MONO in exposed workers than non-exposed group by previous study [29]. However, the variation in sample size and environmental conditions may be the possible reasons for this discrepancy between the study's findings. Thus, this finding suggests that aromatic hydrocarbons inhalation may contribute to a significant effect on different types of WBC.

The results for the protein profile were listed in Table 3. Based on the results, it is obvious that there was a significant decrease in serum total protein with a significant increase in albumin in exposed groups (G1, G2, and G3) compared with control group. Furthermore, there was a highly significant decrease was found in globulins level with a highly significant increase in Albumin/Globulin ratio in exposed groups (G1, G2, and G3) compared with control group, while no significant difference was found in all these parameters between exposed groups.

**TABLE 3** The comparison of protein profile in all studied groups

| Dependent Variables | Groups | | | |
|---|---|---|---|---|
| | C | G1 | G2 | G3 |
| Total serum protein (g/dl) | 7.28±0.50 | 6.84±0.81 a* | 6.90±1.02 a* | 6.88±1.30a* |
| Albumin (g/dl) | 4.25±0.28 | 4.61±0.79 a* | 4.57±0.81a* | 4.64±0.92 a* |
| Globulin (g/dl) | 3.03±0.44 | 2.22±0.89 a** | 2.33±0.89 a** | 2.34±0.99 a** |
| Albumin/Globulin ratio | 1.43±0.25 | 2.52±1.35 a** | 2.41±1.48 a** | 2.80±3.25 a** |

(a)-significant difference between C and (G1,G2, and G3)
(b)-significant difference between G1 and (G2 and G3)
(c)- significant difference between G2 and G3
*p<0.05 ; **p<0.01

Plasma proteins are involved in several processes, including carrying food metabolites, and hormones throughout the body, maintaining adequate water distribution between the blood and tissues, coagulation, and protecting against infection. The measurement of total protein concentration provides general information about the general nutritional status and reverses disease states of the patients in several organ systems [30]. Hence, the change in serum total protein concentration is commonly used in clinical practice as a non-specific indicator to identify disease, or monitor disease severity. Albumin is an extracellular antioxidant with albumin making up to 49% of the total antioxidant status in plasma while globulins make up 38% of blood proteins, transport ions, hormones, and lipids that help in immune function and fibrinogen comprises 7% of blood proteins [31]. An imbalance in albumin to globulins ratio may indicate persistent inflammation, liver problems, or, in rare cases, immunodeficiency [32].

In the current study, the mean level of total serum protein in exposed groups was significantly decreased when compared with healthy control. The results of the present study agreed with the previous studies [2,33].

One of the primary endogenous carriers for the molecules' biodistribution through blood plasma is human serum albumin [34]. In addition, the results of this study found that the mean value of serum albumin level for the exposed groups was increased significantly than the control group. Furthermore, in [35], the higher albumin levels were detected significantly in petrol station workers compared with control group. While [2] found no significant differences between fuel station workers than control regarding the serum albumin level. Moreover, the results of the present study revealed that there was a significant decrease in globulins level in exposed groups than in the control group, which is in agreement with the previous study [36], while it was also demonstrated in the same study that there is a significant decrease in Albumin/Globulins ratio, which does not agree with our study.

Table 4 indicated the findings for the renal function parameters (urea and creatinine).

**TABLE 4** The comparison of renal function parameters in all studied groups.

| Dependent Variables | Groups | | | |
|---|---|---|---|---|
| | C | G1 | G2 | G3 |
| Urea (mg/dl) | 28.82±6.80 | 39.46±8.55 [a**] | 40.01±6.69 [a**] | 42.33±7.37 [ab**] |
| Creatinine (mg/dl) | 0.83±0.09 | 0.98±0.21 [a**] | 0.98±0.16 [a**] | 1.24±0.25[abc**] |

(a)-significant difference between C and (G1,G2, and G3)
(b)-significant difference between G1 and (G2 and G3)
(c)- significant difference between G2 and G3
*p<0.05 ; **p<0.01

Actually, the workers at petrol stations are frequently exposed to various harmful toxins, among the most dangerous toxins are the vapors of gasoline, kerosene, and diesel linked to an increased risk of renal cancer and can cause abnormal changes in the way of function formany vital organs. Blood urea (BU) produced by the liver and dispersed throughout intracellular and extracellular fluid, is a significant nitrogenous end product of protein and amino acid catabolism [37]. Muscles produce creatinine (inner anhydride of creatine) through irreversible, non-enzymatic dehydration, and phosphate cleavage from creatine phosphate, which is used as an energy source for muscle contraction. The creatinine retention in the blood is a key marker of renal dysfunction [38]. The present study showed a high significant elevated in both serum urea and creatinine levels in exposed groups compared with control. Similarly, the levels of serum urea and creatinine were reported to be significantly increased in gasoline filling workers more than control group in previous studies [12,39]. The reason for the increase in urea levels may be caused by the reduction in glomerular filtration rate brought on by exposure to petroleum products, and it may also be caused by structural damage to the nephrons structural integrity. In addition, the elevation in creatinine level may be caused by the creatinine retention as a result of petroleum products ingestion, inhalation, or skin absorption by workers and the renal function impairment brought on by exposure to nephrotoxic substances like petroleum products [39].

## Conclusion

Based on the results, due to significant alterations in hematological parameters and the increased in urea and creatinine when comparing the petrol exposed groups with control group, the long-term exposure to petroleum products may be the main cause of the blood disorders or renal dysfunction in filling station employees, and further it has a number of toxicological effects on bodily tissues and biochemical alterations that make it a major health risk for all people.

Thus, it is advised that the workplace be improved and developed with a frequent medical attention to slow down the advancement of blood disorders or renal insufficiency among the petrol stations workers.

## Acknowledgments

The authors would like to express their thanks to all the workers at the filling stations. Also, they would like to provide their sincere gratitude to the healthy volunteered to give blood samples.

**Conflict of Interest**

The authors declare that there is no conflict of interests regarding the publication of this manuscript.

***Orcid:***

*Abdulameer Hassan Abdulameer:*
*https://www.orcid.org/0000-0002-1006-8127*
*Saba Zuhair Hussein:*
*https://www.orcid.org/0000-0002-6750-8437*

**References**

[1] W.H. Al-Dahhan, S.M.A. Mahmood, *ANJS.*, **2019**, *22*, 35-42. [Crossref], [Google Scholar], [Publisher]

[2] M. Hasb Elnabi, S. Mohamed, H. Abo-El Wafa, H. Khalaf, and W. Allam, *Ain-Shams J. Forensic Med. Clin. Toxicol.*, **2021**, *37*, 57-66. [Crossref], [Google Scholar], [Publisher]

[3] I. Manisalidis, E. Stavropoulou, A. Stavropoulos, E. Bezirtzoglou, *Front Public Health.*, **2020**, *8*, 1-13. [Crossref], [Google Scholar], [Publisher]

[4] L. Benbrahim-Tallaa, R.A. Baan, Y. Grosse, B. Lauby-Secretan, F.El Ghissassi,V. Bouvard, N. Guha, D. Loomis, K. Straif, *The Lancet Oncology*, **2012**, *13*, 663-664. [Crossref], [Google Scholar], [Publisher]

[5] C.J. Davidson, J.H. Hannigan, S.E. Bowen, *Environ. Toxicol. Pharmacol.*, **2021**, *81*, 103518. [Crossref], [Google Scholar], [Publisher]

[6] S.A.N. AL-Jadaan, A.S.J. Alkinany, *Int J Pharm Sci Res.*, **2017**, *2*, 31-36. [Google Scholar], [Publisher]

[7] A.H.A. Elsukar, N.A. Ahmed, M.A. Khaleel, *J. med. pharm. sci.*, **2018**, *2*, 12-21. [Crossref], [Google Scholar], [Publisher]

[8] G. Teklu, M. Negash, T. Asefaw, F. Tesfay, G. Gebremariam, G. Teklehaimanot, M. Wolde, A. Tsegaye, *J. Blood Med.*, **2021**, *12*, 839-847. [Crossref], [Google Scholar], [Publisher]

[9] M. Chehrehei, S.A.H. Mirzahosseini, N. Mansouri, M.H. Behzadi, Y. Rashidi, *Research Square*, **2021**, 1-14. [Crossref], [Google Scholar], [Publisher]

[10] R. Sun, K. Xu, S. Ji, Y. Pu, L. Yu, L. Yin, J. Zhang, Y. Pu, *Ecotoxicol. Environ. Saf.*, **2021**, *207*, 111490. [Crossref], [Google Scholar], [Publisher]

[11] F.O. Owagboriaye, G.A. Dedeke, A.A. Aladesida, J.A. Bamidele, W.E. Olooto, *J. King Saud Univ. Sci.*, **2018**, *30*, 393-399. [Crossref], [Google Scholar], [Publisher]

[12] T. Asefaw , M. Wolde, A. Edao, A. Tsegaye, G. Teklu, F. Tesfay, and G. Gebremariam, *PLoS One*, **2020**, *15*, 1-9. [Crossref], [Google Scholar], [Publisher]

[13] C. Tothova, O. Nagy, G. Kovac, *Veterinarni Medicina*, **2016**, *61*, 475-496. [Crossref], [Google Scholar], [Publisher]

[14] L. Franco-Serrano, M. Huerta, S. Hernández, J. Cedano, J. Perez-Pons, J. Piñol, A. Mozo-Villarias, I. Amela, E. Querol, *The Protein Journal*, *37*, 444-453. [Crossref], [Google Scholar], [Publisher]

[15] S.A. Wahyuti, Siswantoyo, Suhadi, R. Meikahani, Paryadi, A.S. Putro, R.P. Perdana, M.W. Dewangga, *J. Med. Chem. Sci.*, **2022**, *5*, 1102-1108. [Crossref], [Google Scholar], [Publisher]

[16] B.H.O. Saleh, F.M. Khaleel, *Chemical Methodologies*, **2022**, *6*, 438-445. [Crossref], [Google Scholar], [Publisher]

[17] S. Mohammed, *ARO-The Scientific Journal of Koya University*, **2014**, *2*, 6–11. [Crossref], [Google Scholar], [Publisher]

[18] C. Costa, E. Ozcagli, S. Gangemi, F. Schembri, F. Giambò, V. Androutsopoulos, A. Tsatsakis, C. Fenga, *Food and Chemical Toxicology*, **2016**, *90*, 30-35. [Crossref], [Google Scholar], [Publisher]

[19] A. Davoudi-Kiakalayeh, R. Mohammadi, A. A. Pourfathollah, Z. Siery, S. Davoudi-Kiakalayeh, *Int. J. Prev. Med.*, **2017**, *8*, 10–13. [Crossref], [Google Scholar], [Publisher]

[20] A. John, and R. A. Oluwafemi, *Progress in Chemical and Biochemical Research*, **2019**, *2*,

170-177. [Crossref], [Google Scholar], [Publisher]

[21] J.H. Mahmoud, O.A. Ghareeb, Y.H. Mahmood, *J. Med. Chem. Sci.*, **2022**, *5*, 76-81. [Crossref], [Google Scholar], [Publisher]

[22] S. Getu, E. Shiferaw, M. Melku, *Environ. Health Prev. Med.*, **2020**, *25*, 1–9. [Crossref], [Google Scholar], [Publisher]

[23] H.S. Abou-ElWafa, A.A. Albadry, A.H. El-Gilany, F.B. Bazeed, *BioMed Research International*, **2015**, *2015*, 1–6. [Crossref], [Google Scholar], [Publisher]

[24] H.A. Mistry, A. Mathur, D. Parmar, R. Dixit, *NJPPP*, **2016**, *6*, 167–169. [Crossref], [Google Scholar], [Publisher]

[25] B.N. Obodo, K.O. Iyevhobu, I.U. Idara, T.J. Okobi, S.A. Abayomi, E.R. Usoro, A.H. Bisiriyu, L.E. Omolumen, I.A. Omisakin, *International Journal of Research Publications*, **2020**, *58*, 1. [Crossref], [Google Scholar], [Publisher]

[26] Z. Zhang, X. Liu, X. Zhang, Y. Zhang, N. Deng, G. Lai, A. Yang, Y. Huang, S. Dang, Y. Zhu, X. Xing, Y. Xiao, Q. Deng, *Res Sq.*, **2022**. [Crossref], [Google Scholar], [Publisher]

[27] T.I. Ndubuisi Ezejiofor, *Journal of Clinical Toxicology*, **2016**, *6*, 1–13. [Crossref], [Google Scholar], [Publisher]

[28] A.M. Moro, N. Brucker, M.F. Charão, E. Sauer, F. Freitas, J. Durgante, G. Bubols, S. Campanharo, R. Linden, A.P. Souza, C. Bonorino, Rafael Moresco, D. Pilger, A. Gioda, S. Farsky, A. Duschl, S.C. Garcia., *Environ. Res.*, **2015**, *137*, 349–356. [Crossref], [Google Scholar], [Publisher]

[29] A.S. Jabbar, E.T. Ali, *J Environ Public Health*, **2020**, *2020*, 1–7. [Crossref], [Google Scholar], [Publisher]

[30] R.M. Dawood, H.R. Hasan, *Baghdad Sci. J.*, **2013**, *10*, 934–944. [Crossref], [Google Scholar], [Publisher]

[31] E. Malmstrom, O. Kilsgård, S. Hauri, E. Smeds, H. Herwald, L. Malmstrom, J. Malmstrom, *Nat. Commun.*, **2016**, *7*, 1–10. [Crossref], [Google Scholar], [Publisher]

[32] B.R. Verma, B. Williams, A. Honushefsky, S. Kaur, S. Doddamani, *J. Card. Fail.*, **2016**, *22*, S30. [Crossref], [Google Scholar], [Publisher]

[33] A.O. Odewabi, O.A. Ogundahunsi, M. Oyalowo, *Br. J. Pharmacol.*, **2014**, *5*, 83–87. [Crossref], [Google Scholar], [Publisher]

[34] M. Shalbafan, G.R. Behbehani, H. Ghasemzadeh, *Chem. Methodol.*, **2019**, *3*, 348-353. [Crossref], [Google Scholar], [Publisher]

[35] A.A.H. Mohamed, M.I. Hassan, Al. Ibrahim, O. Suliman, G.M. Elimairi, *Int. J. Curr. Res.*, **2016**, *8*, 30683–30685. [Crossref], [Google Scholar], [Publisher]

[36] A. Firoozichahak, S. Rahimnejad, A. Rahmani, A. Parvizimehr, A. Aghaei, and R. Rahimpoor, *Toxicology Reports*, **2022**, *9*, 269–275. [Crossref], [Google Scholar], [Publisher]

[37] I.D. Weiner, W.E. Mitch, J.M. Sands, *Clinical Journal of the American Society of Nephrology*, **2015**, *10*, 1444-1458. [Crossref], [Google Scholar], [Publisher]

[38] J.C. Okpala, I. Sani, R. Abdullahi, H.N. Ifedilichukwu, J.C. Igwe, *Afr. J. Biochem. Res.*, **2014**, *8*, 52–64. [Crossref], [Google Scholar], [Publisher]

[39] A.H. Awadalla, N.A. Ahmed, A.Y.E. Yagoob, *EJPMR*, **2017**, *4*, 395–399. [Crossref], [Google Scholar], [Publisher]

**How to cite this article:** Abdulameer Hassan Abdulameer*, Saba Zuhair Hussein. Study of some hematological and biochemical parameters among petrol stations workers in Baghdad city Journal of Medicinal and Pharmaceutical Chemistry Research, 2023, 5(1), 103-111.