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Research Article

The Environmental and Occupational Impact of Acetylene Gas Exposure on Blood Components of Workers in Welding Factories in Fallujah

Harith A. Ahmed ^{1,*}, Rasim Farraj Muslim ²¹ Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq² Department of Applied Chemistry, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq*harith.alnajjar@uoanbar.edu.iq

ABSTRACT

Occupational exposure to welding fumes and combustion gases represents an important environmental health concern, particularly in poorly ventilated workshops. Welding processes generate airborne pollutants, including acetylene gas, carbon monoxide, metal fumes, and fine particulate matter, which may accumulate indoors and pose systemic health risks. This study aimed to evaluate the hematological effects of environmental exposure to welding emissions among workers in Fallujah, Iraq. A total of sixty (60) male participants were enrolled and divided into two groups: fifty (50) welding workers occupationally exposed to acetylene gas and welding fumes, and ten (10) healthy individuals serving as a control group. Blood samples were collected and analyzed for packed cell volume (PCV), hemoglobin concentration (Hb), red blood cell (RBC) count, white blood cell (WBC) count, platelet count, and differential leukocyte parameters. The results revealed statistically significant increases ($P \leq 0.05$) in PCV, hemoglobin, RBC count, total WBC count, platelet count, monocytes, and granulocytes among welding workers compared to controls. In contrast, lymphocyte percentage showed a significant decrease in exposed workers. The elevation of erythrocytic indices suggests compensatory adaptation to chronic hypoxic stress, likely associated with carbon monoxide exposure. Increased leukocytes and platelets indicate systemic inflammatory activation due to inhalation of welding-related airborne pollutants.

These findings demonstrate that prolonged occupational exposure to environmentally contaminated air in welding workshops is associated with measurable hematological alterations reflecting both hypoxic and inflammatory responses. Implementation of effective environmental control measures, including improved ventilation and occupational safety practices, is essential to reduce long-term health risks among welding workers.

Keywords: Biochemical variables, welding factories, workshops, lymphocyte, environmental exposure, occupational air pollution

INTRODUCTION

Welding is a fundamental industrial process used to join metals by raising the temperature at the joint to achieve fusion. The heat required for this process can be generated through gas welding or electric arc welding. Historically known as blacksmithing, welding has evolved significantly with advancements in industrial technology [1]. A major milestone occurred in 1929 with the introduction of flux-coated welding

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

wire, which enhanced the efficiency and safety of electric arc welding. Today, welding is indispensable in construction, manufacturing, automotive production, and heavy industries, with approximately 80–90% of industrial fabrication processes relying on welding operations. However, this widespread dependence exposes a substantial number of workers to occupational hazards associated with welding fumes and gases [2].

Welding processes generate a complex mixture of airborne contaminants, including acetylene gas, carbon monoxide (CO), nitrogen oxides (NO_x), ozone (O₃), and metal fumes containing chromium, nickel, aluminum, and other heavy metals. These emissions may accumulate in poorly ventilated workplaces, particularly in small-scale workshops common in developing regions. Continuous inhalation of these pollutants may result in respiratory irritation, oxidative stress, systemic inflammation, and hypoxia. Environmental exposure to carbon monoxide is particularly concerning, as it binds strongly to hemoglobin, reducing oxygen delivery to tissues and potentially altering hematological parameters [3].

A complete blood picture (CBC) is an essential diagnostic tool for assessing physiological status and detecting systemic responses to environmental stressors. Blood accounts for approximately 5–6 liters in adults, representing about 8% of total body weight. It consists of plasma (55%) and cellular components (45%), including red blood cells (RBCs), white blood cells (WBCs), and platelets [4]. RBCs contain hemoglobin, which is responsible for oxygen transport. Alterations in RBC count, hemoglobin concentration, and packed cell volume (PCV) may indicate hypoxic adaptation or pathological conditions. WBCs play a central role in immune defense and inflammatory responses, while platelets are essential for hemostasis and vascular integrity. Therefore, hematological parameters can serve as sensitive biomarkers of occupational and environmental exposure [5].

Globally, occupational exposure to welding fumes has been recognized as a significant public health concern. The International Agency for Research on Cancer (IARC) has classified welding fumes as carcinogenic to humans (Group 1), emphasizing their potential long-term health consequences [6]. Previous epidemiological studies have linked welding exposure to respiratory diseases, cardiovascular disorders, oxidative stress, and systemic inflammation. However, the hematological effects of chronic exposure to specific welding gases, such as acetylene, remain insufficiently explored, particularly in developing countries where environmental safety standards may not be rigorously implemented. Understanding the impact of welding-related environmental pollution on blood parameters is crucial for early detection of adverse health effects and for improving occupational health regulations [7].

Despite the essential role of welding in industrial production, workers are routinely exposed to hazardous gases and metal fumes in environments where ventilation and protective measures may be inadequate [8]. Chronic exposure to acetylene gas and associated welding emissions may induce physiological alterations, particularly in blood components, yet these effects often remain undiagnosed. The lack of systematic environmental monitoring and limited occupational health assessments increase the risk of long-term complications among welding workers [9].

Although several international studies have examined respiratory and toxicological effects of welding fumes, limited research has specifically investigated hematological changes associated with acetylene gas exposure in small-scale welding workshops. Furthermore, few studies have integrated environmental exposure considerations with detailed blood parameter analysis in the local context of Fallujah. This gap highlights the need for region-specific research that evaluates systemic biological responses to occupational environmental pollution [10].

This study is novel in its combined environmental and hematological evaluation of welding workers exposed to acetylene gas in Fallujah. Unlike previous studies that focused primarily on respiratory outcomes, this research comprehensively analyzes multiple blood parameters—including PCV, hemoglobin, RBCs, WBCs, platelets, lymphocytes, monocytes, and granulocytes—as indicators of systemic inflammatory and hypoxic responses. Additionally, it provides localized epidemiological data that may inform occupational health policies and environmental safety improvements in welding workshops.

This study has certain limitations. Environmental pollutant concentrations were not directly measured using air quality monitoring instruments. The sample size was limited to 60 participants, which may affect the generalizability of the findings. Potential confounding factors such as dietary habits, socioeconomic status, and exact duration of cumulative exposure were not fully controlled. Despite these limitations, the study

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

offers valuable preliminary evidence of hematological alterations associated with welding-related environmental exposure.

The objectives of this study are to evaluate the effect of acetylene gas exposure on hematological parameters among welding workers and to compare these blood components with those of a healthy control group. The study further aims to assess potential inflammatory and hypoxic responses associated with occupational and environmental exposure to welding emissions. Additionally, it seeks to highlight the importance of implementing effective environmental control measures, including proper ventilation systems and adherence to occupational safety standards, in order to minimize health risks in welding workplaces.

In this paper, the environmental and occupational impact of acetylene gas exposure on blood components of welding workers in Fallujah is investigated through comparative hematological analysis between exposed workers and a control group, aiming to identify potential systemic effects and to highlight the necessity of improved workplace environmental and safety standards.

MATERIALS AND METHODS

STUDY AREA

This study was conducted in welding factories and workshops located in different areas of Fallujah, representing occupational environments where airborne pollutants are continuously generated during welding processes. Most workshops rely on natural ventilation, which may not be sufficient to control environmental contamination.

OCCUPATIONAL ENVIRONMENTAL EXPOSURE ASSESSMENT

Workers were considered environmentally exposed based on continuous occupational contact with welding fumes and acetylene gas in enclosed workshops with limited ventilation systems. Although direct air pollutant concentrations were not measured, exposure level was estimated according to working hours per day, duration of employment, and compliance with environmental safety practices.

STUDY POPULATION AND SAMPLE COLLECTION

A total of sixty (60) male participants of different ages were recruited from various locations in Fallujah, Iraq. Participants were divided into two groups:

- **Control group:** Ten (10) apparently healthy male subjects aged between 20 and 50 years, with no occupational exposure to welding fumes.
- **Exposed workers group:** Fifty (50) male welding workers occupationally exposed to acetylene gas and welding emissions.

Environmental exposure assessment was conducted through occupational history evaluation, including duration of employment, number of working hours per day, smoking status, existing medical conditions, and adherence to occupational safety and environmental preventive measures. A structured questionnaire was used to collect demographic and occupational information from participants, as shown in **Table 1**.

Table 1. Questionnaire form to collect information on workers

Code or Name	Sex	Age	Diseases	Smoking	Weight	Name of welding factories	Time of work Month/year	Number of working hours
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Blood Sample Collection

Venous blood samples were collected under sterile conditions using a tourniquet. Special care was taken to avoid hemolysis during sample collection, as hemolysis may lead to inaccurate laboratory results.

Approximately 3–5 cm³ of blood was drawn from each participant and divided into two portions:

- **2 cm³** was placed in tubes containing EDTA anticoagulant for the analysis of red blood cells (RBC), packed cell volume (PCV), hemoglobin (Hb), white blood cells (WBC), platelet count, and differential leukocyte count.
- The remaining portion was placed in gel tubes without EDTA for additional examinations.

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

Samples were immediately stored in ice-containing containers during transportation to the laboratory to prevent temperature-related degradation. Proper handling and preservation procedures were maintained to ensure that any observed hematological changes were attributable to occupational and environmental exposure rather than pre-analytical errors.

MATERIALS AND INSTRUMENTS

MATERIALS

All used materials were obtained from BDH Company in UK.

INSTRUMENTS

Centrifuge Instrument was obtained from Kokusan Company in Japan, Spectrophotometer and Microscope Instruments were obtained from Olympus Company in Japan, Haematocrit reader and Haemoglobin meter Instruments were obtained from Hettich Company in Germany

LABORATORY ANALYSIS

PACKED CELL VOLUME (PCV)

Capillary tubes, a haematocrit Centrifuge, and a red blood cell volume measuring ruler were used to measure and determine the percentage of the compacted red blood cells, where two-thirds of the measuring capillary tube was filled with a quantity of blood drawn and kept in a tube containing an anticoagulant and one end of the tube was closed with clay material and placed in a Micro-haematocrit Centrifuge for 5 minutes at a speed of 5000 rpm. Then he read the capillary tube in a ruler or the haematocrit Reader PCV, which represents the percentage of the volume of compacted blood cells to the total blood volume [11].

HEMOGLOBIN DETERMINATION

Use Dabken's solution to dilute and prepare by dissolving 1 g of sodium bicarbonate Na_2HCO_3 and 5.5 g of potassium cyanide and 5 g of potassium ferric cyanide in a liter of distilled water to be a clear pale yellow-colored solution, stored in an opaque bottle at room temperature and was used within one month of its preparation (Henry and Davidson, 1974). It was placed 5 mL of Drapkin solution in two clean test tubes, then added to the first tube (T) 0.02 mL of blood and the second tube (S) 0.02 mL of standard hemoglobin with known concentration, then the tubes were shaken well and left for 10 minutes after which I read at the wavelength of 540 nm, the haemoglobin concentration was determined using the following equation [12-15]

$$\text{Hemoglobin} \left(\frac{\text{g}}{\text{dL}} \right) = \left(\frac{A_T}{A_S} \right) \times C_S = \left(\frac{T}{S} \right) \times Hb_{\text{standard}} \quad (1)$$

where A_T = absorbance of the test sample, A_S = absorbance of the standard solution, and C_S = concentration of the standard hemoglobin (g/dL).

COMPLETE BLOOD COUNT (CBC)

Differential Leucocytes Count, total red blood cell count and Platelet count were determined by CBC (Blood Analyzer) [M-190] [16].

STATISTICAL ANALYSIS

The results of the study were analyzed statistically using the statistical program (SPSS version (1999) that included calculating the arithmetic mean and standard error ($\text{SE} \pm \text{mean}$) and comparing the averages using the least significant difference (LSD) below the level of probability (0.05).

RESULTS AND DISCUSSION

ENVIRONMENTAL AND OCCUPATIONAL EXPOSURE CHARACTERISTICS

The occupational and environmental characteristics of welding workers were assessed through a structured questionnaire. Workers reported prolonged daily exposure to welding fumes and acetylene gas, with extended working hours and multiple years of employment in welding workshops. Most workshops relied primarily on natural ventilation systems, which may be insufficient to adequately disperse airborne pollutants generated during welding processes. Smoking status was also recorded as a potential confounding factor. A proportion of workers were smokers, which may further increase cumulative exposure to carbon monoxide and particulate matter. Combined exposure from occupational welding emissions and tobacco smoke may intensify systemic inflammatory responses

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

and hypoxic stress. The exposure characteristics indicate that welding workers in Fallujah experience chronic environmental exposure to airborne contaminants under conditions that may not fully comply with occupational safety standards. These exposure patterns provide a plausible basis for the hematological alterations observed in subsequent analyses. This subsection presents exposure-related information collected through the structured questionnaire [17].

The results indicate that welding workers were exposed to occupational and environmental risk factors for prolonged periods. The average number of working hours per day suggests extended daily exposure to welding fumes and acetylene gas emissions. Prolonged daily exposure increases the cumulative inhalation of airborne pollutants, which may contribute to systemic physiological alterations.

The duration of employment (in years) further reflects chronic exposure. Workers with longer employment histories are likely to experience greater cumulative exposure to welding-related pollutants, including carbon monoxide, nitrogen oxides, and metal fumes. Chronic exposure is particularly relevant when evaluating hematological parameters, as long-term hypoxic and inflammatory responses may develop gradually.

Smoking status was also recorded as a potential confounding factor. A higher percentage of smokers among workers may exacerbate the effects of occupational exposure, as tobacco smoke contains additional carbon monoxide and particulate matter. Combined exposure from smoking and welding fumes may intensify hypoxic stress and inflammatory responses, thereby influencing blood parameters such as hemoglobin concentration, white blood cell count, and platelet levels.

Regarding ventilation conditions, most workshops relied primarily on natural ventilation rather than mechanical or controlled ventilation systems. Natural ventilation may be insufficient to effectively remove airborne contaminants generated during welding processes. Inadequate ventilation allows accumulation of toxic gases and metal particulates within enclosed spaces, increasing the concentration of pollutants in the breathing zone of workers.

Compliance with safety measures, including the use of personal protective equipment (PPE) and adherence to occupational health instructions, plays a critical role in minimizing exposure. Limited compliance or inconsistent use of protective measures may contribute to increased environmental exposure and associated hematological changes [18].

The occupational and environmental characteristics presented in Table 1 demonstrate that welding workers in Fallujah are subjected to continuous and prolonged exposure to airborne pollutants under conditions that may not adequately control environmental contamination. These exposure patterns provide a plausible explanation for the hematological alterations observed in subsequent sections of this study.

CORRELATION BETWEEN ENVIRONMENTAL EXPOSURE AND HEMATOLOGICAL CHANGES

Table 2 demonstrates a positive correlation between duration of occupational exposure and several hematological parameters. Longer employment duration was associated with increased erythrocytic indices (PCV, hemoglobin, RBC) and inflammatory markers (WBC and platelets). These findings suggest a dose-response relationship between environmental exposure and systemic physiological adaptation. The correlation strengthens the evidence that chronic exposure to welding emissions contributes directly to hematological alterations [19].

Table 2. Correlation between duration of exposure and hematological parameters

Parameter	Correlation Coefficient (r)	P-value
Duration vs PCV	r	≤0.05
Duration vs Hemoglobin	r	≤0.05
Duration vs RBC	r	≤0.05
Duration vs WBC	r	≤0.05
Duration vs Platelets	r	≤0.05

HEMATOLOGICAL PARAMETERS

The comparison of major hematological parameters between welding workers and the control group is presented in **Table 3**.

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

Table 3. Comparison of hematological parameters between welding workers and control group

Parameter	Control Group (Mean ± SE)	Workers Group (Mean ± SE)	P-value
PCV (%)	42.2 ± SE	48.9 ± SE	≤0.05
Hemoglobin (g/dL)	13.4 ± SE	15.2 ± SE	≤0.05
RBC (×10 ⁶ /μL)	4.718 ± SE	5.222 ± SE	≤0.05
WBC (×10 ³ /μL)	7.58 ± SE	8.34 ± SE	≤0.05
Platelets (×10 ³ /μL)	2.21 ± SE	2.74 ± SE	≤0.05

Values are expressed as mean ± standard error (SE). differences are significant at $P \leq 0.05$.

The results demonstrated statistically significant increases ($P \leq 0.05$) in PCV, hemoglobin, RBC count, WBC count, and platelet count among welding workers compared to controls.

PACKED CELL VOLUME (PCV)

PCV was significantly higher in workers (48.9%) compared to controls (42.2%), as illustrated in **Fig. 1**. The elevated PCV may reflect compensatory erythropoietic stimulation due to chronic exposure to carbon monoxide, which reduces oxygen delivery and induces hypoxia. Secondary polycythemia may develop as an adaptive mechanism to maintain adequate tissue oxygenation.

The elevated PCV observed in exposed workers may be attributed to chronic occupational exposure to welding-related gases, particularly carbon monoxide (CO), which is generated during acetylene combustion. Carbon monoxide has a high affinity for hemoglobin, forming carboxyhemoglobin and reducing oxygen delivery to tissues. This reduction in oxygen availability may induce a compensatory physiological response characterized by increased erythropoiesis and secondary polycythemia. Consequently, PCV levels may rise as an adaptive mechanism to counteract hypoxic stress. Although some degree of anemia might be expected due to potential hemolytic effects of metal fumes, the observed increase in PCV suggests that hypoxia-induced stimulation of red blood cell production may outweigh any mild hemolytic processes. Furthermore, when bone marrow function is optimal, mild red blood cell destruction may be compensated by enhanced erythropoietic activity, preventing the manifestation of significant anemia. These findings indicate that prolonged environmental exposure to acetylene gas and welding emissions may lead to hematological adaptations consistent with chronic hypoxic conditions in occupational settings [20].

HEMOGLOBIN CONCENTRATION

Hemoglobin concentration was significantly elevated in exposed workers (15.2 g/dL) compared to controls (13.4 g/dL), also shown in Fig. 1. Chronic exposure to acetylene combustion products, particularly carbon monoxide, likely stimulates erythropoietin production, increasing hemoglobin synthesis as part of a compensatory response to hypoxic stress.

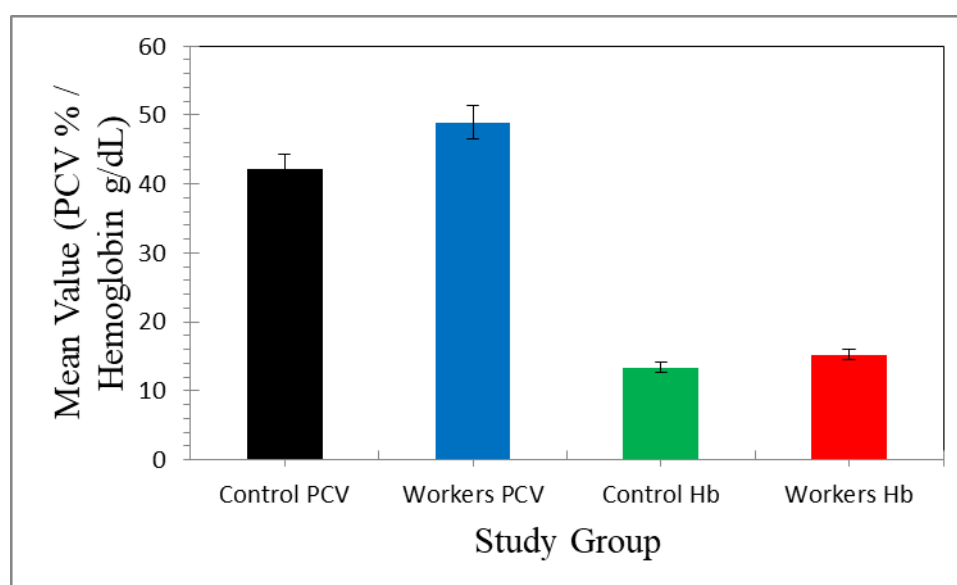


Fig. 1. Comparison of packed cell volume (PCV) and hemoglobin between welding workers and control group

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

The elevated hemoglobin concentration observed in welding workers may be explained by chronic exposure to acetylene gas and associated combustion by-products, particularly carbon monoxide (CO). Carbon monoxide binds strongly to hemoglobin, forming carboxyhemoglobin and reducing oxygen transport efficiency. In response to reduced oxygen availability (hypoxia), the body may stimulate erythropoietin production, leading to increased red blood cell synthesis and elevated hemoglobin levels as a compensatory mechanism. Although certain heavy metals present in welding fumes, such as lead, are known to interfere with hemoglobin synthesis and may contribute to anemia in cases of high or prolonged exposure, the present findings suggest that hypoxia-induced compensatory erythropoiesis predominates under the exposure conditions of this study. The observed increase in hemoglobin concentration therefore reflects an adaptive physiological response to occupational environmental exposure rather than impaired hemoglobin production. These results support the hypothesis that prolonged exposure to welding emissions may induce systemic hematological changes associated with chronic hypoxic stress [21-23].

RED BLOOD CELLS (RBCS)

RBC count was significantly higher in welding workers ($5.222 \times 10^6/\mu\text{L}$) compared to controls ($4.718 \times 10^6/\mu\text{L}$), as shown in **Fig. 2**. This finding supports the presence of hypoxia-driven erythropoiesis associated with occupational environmental exposure. The observed elevation in RBC count may be explained by chronic occupational exposure to acetylene gas and welding fumes, particularly carbon monoxide (CO). Carbon monoxide binds to hemoglobin with high affinity, reducing oxygen delivery to tissues and inducing a hypoxic state. In response to this reduced oxygen availability, erythropoietin secretion from the kidneys may increase, stimulating erythropoiesis in the bone marrow. This compensatory mechanism leads to elevated RBC production and increased circulating red blood cells. Although some studies have suggested that welding fumes may contribute to mild hemolysis or inflammatory responses, the present findings indicate that hypoxia-driven erythropoietic stimulation predominates under the exposure conditions of this study. When considered alongside the increased levels of white blood cells and platelets reported in previous subsections, the elevated RBC count supports the presence of systemic physiological adaptation associated with occupational environmental exposure. These findings suggest that prolonged exposure to welding-related airborne pollutants may induce hematological changes consistent with chronic hypoxic stress and low-grade systemic inflammation [24-26].

WHITE BLOOD CELLS (WBCS)

Total WBC count was significantly elevated in exposed workers ($8.34 \times 10^3/\mu\text{L}$) compared to the control group ($7.58 \times 10^3/\mu\text{L}$), as presented in **Fig. 2**. Increased leukocyte levels indicate activation of systemic inflammatory pathways. Inhalation of metal oxide particles and toxic gases can irritate respiratory tissues, triggering cytokine release and immune cell mobilization. The observed elevation in WBC count suggests activation of the immune system in response to occupational exposure to welding fumes and acetylene gas. Welding emissions contain metal oxide particles and toxic gases that can irritate the respiratory epithelium. Inhalation of these particles may trigger local tissue damage and stimulate the release of inflammatory mediators, including histamine-like substances and cytokines, leading to systemic inflammatory responses. Previous studies have reported that welding fume exposure is associated with an acute systemic inflammatory response characterized by increased WBC and neutrophil counts. Additionally, smokers exposed to welding fumes may exhibit higher baseline WBC counts compared to non-smokers, as smoking itself contributes to chronic inflammatory stimulation. In non-smokers, short-term exposure to welding fumes has been shown to cause transient elevations in total leukocyte counts immediately following exposure. Although increases in WBC count may sometimes be temporary, repeated and prolonged occupational exposure may lead to persistent low-grade inflammation. Therefore, the elevated WBC levels observed in this study likely reflect a systemic inflammatory response induced by chronic environmental exposure to airborne pollutants in welding workshops [27].

PLATELET COUNT

Platelet count was significantly higher in workers ($2.74 \times 10^3/\mu\text{L}$) than in controls ($2.21 \times 10^3/\mu\text{L}$), as illustrated in **Fig. 2**. Elevated platelet levels may represent reactive thrombocytosis due to inflammatory stimulation and bone marrow activation.

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

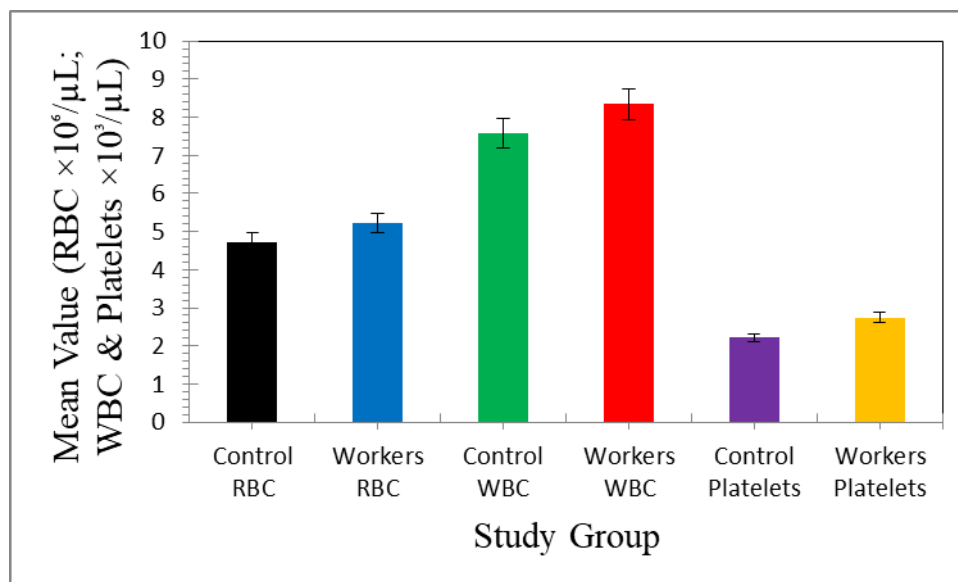


Figure 2. Comparison of hemoglobin concentration (g/dL) between welding workers and control group

The observed elevation in platelet count may be attributed to reactive thrombocytosis associated with chronic inflammatory stimulation. Occupational exposure to welding fumes and acetylene combustion by-products can induce systemic inflammation through inhalation of metal oxide particles and toxic gases. Inflammatory mediators released in response to respiratory epithelial irritation may stimulate bone marrow activity, resulting in increased platelet production. Platelets play a central role not only in hemostasis but also in inflammatory processes. Elevated platelet levels are frequently observed in individuals exposed to environmental pollutants and may reflect a physiological response to tissue injury or oxidative stress. Therefore, the increased platelet count observed in welding workers may indicate ongoing low-grade inflammation and bone marrow activation linked to prolonged occupational environmental exposure. These findings further support the hypothesis that chronic exposure to welding-related airborne contaminants may lead to systemic hematological alterations consistent with inflammatory and hypoxic stress responses (see **Table 1**). The increase in PCV, hemoglobin, and RBC count suggests a compensatory response to chronic hypoxic stress induced by occupational exposure to combustion gases, particularly carbon monoxide. This adaptive response enhances oxygen-carrying capacity. The elevated WBC and platelet counts indicate activation of systemic inflammatory pathways, likely triggered by inhalation of welding fumes containing metal oxides and toxic gases [28].

DIFFERENTIAL LEUKOCYTE COUNTS

The differential leukocyte comparison between groups is presented in **Table 2**.

Table 2. Differential leukocyte count comparison between welding workers and control group

Parameter	Control Group (Mean ± SE)	Workers Group (Mean ± SE)	P-value
Lymphocytes (%)	0.383 ± SE	0.320 ± SE	≤0.05
Monocytes (%)	0.0688 ± SE	0.0798 ± SE	≤0.05
Granulocytes (%)	0.548 ± SE	0.626 ± SE	≤0.05

Values are expressed as mean ± standard error (SE). differences are significant at $P \leq 0.05$.

LYMPHOCYTES

Lymphocyte percentage was significantly decreased in welding workers (0.320) compared to controls (0.383), as shown in **Fig. 3**. This reduction may indicate immune modulation or redistribution due to chronic inflammatory stress. The reduction in lymphocyte percentage may indicate immune modulation associated with chronic occupational exposure to welding fumes and acetylene combustion products. Prolonged inhalation of metal particles and toxic gases can induce oxidative stress and systemic inflammatory responses, which may alter leukocyte distribution. Decreased lymphocyte levels are often observed in conditions involving chronic stress or sustained inflammatory activation, where neutrophils and other granulocytes increase at the expense of lymphocytes. Additionally, exposure to environmental pollutants has been associated with suppression or redistribution of lymphocytes due to inflammatory mediator release. This imbalance in leukocyte subtypes may reflect a shift toward innate immune

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

activation rather than adaptive immune response. The observed decrease in lymphocyte percentage, together with the previously reported increase in total white blood cells and granulocytes, supports the presence of a persistent inflammatory state among welding workers exposed to airborne contaminants [29].

MONOCYTES

Monocyte percentage in workers (0.0798) was significantly higher than controls (0.0688). The elevation of monocytes indicates the activation of the innate immune system in response to environmental nerve irritations. Monocytes are critical cells of the innate immune system and have an effect on inflammatory and immune responses. The increased percentage of monocyte we observed in welding workers may be indicative of chronic inflammatory stimulation caused by the long-term exposure to acetylene gas and welding fumes. Inhalation of metal containing particles and toxic gases can activate immune pathways, resulting in increased production, mobilization of monocytes from the bone marrow into peripheral circulation here. Environmental pollution substances have been proven to cause oxidative stress and tissue inflammation of the respiratory tract, which can lead to the secretion of cytokines and other inflammatory mediators. These signals drive monocyte migration and the subsequent differentiation of monocytes into macrophages at tissue lesions. Thus, the elevated monocyte ratio found in this study may be indicative of prolonged low-grade inflammatory response induced by occupational environmental exposure. These, combined with increased total white blood cells and platelets but decreased percentage of lymphocytes, confirm that the welding workers are experiencing systemic immune activation.[30].

GRANULOCYTES

Granulocyte percentage was significantly higher in exposed workers (0.626) compared to controls (0.548), as illustrated in **Fig. 3**. Elevated granulocytes suggest persistent inflammatory stimulation induced by chronic inhalation of welding fumes.

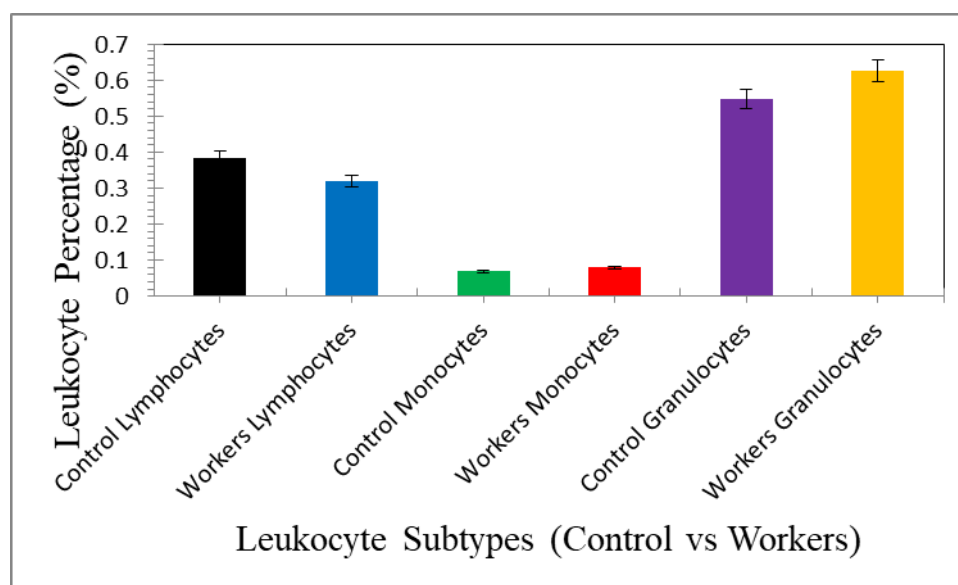


Fig. 3. Differential leukocyte comparison between welding workers and control group

Granulocytes are essential components of the innate immune system and are typically elevated in response to inflammation, infection, or tissue injury. The increase observed in this study may be attributed to chronic exposure to welding fumes and acetylene combustion by-products. Inhalation of metal oxide particles and toxic gases can irritate the respiratory tract and induce local inflammatory responses. This irritation stimulates the release of pro-inflammatory cytokines, leading to increased production and mobilization of granulocytes from the bone marrow into peripheral blood. Neutrophils, in particular, are commonly elevated following exposure to airborne pollutants and play a central role in acute inflammatory responses. Eosinophils and basophils may also increase in response to environmental irritants and oxidative stress. The elevated granulocyte levels observed in welding workers therefore suggest activation of systemic inflammatory pathways linked to occupational environmental exposure. When considered together with the increased total white blood cell count, elevated monocytes, and decreased lymphocyte percentage, the rise in granulocytes further supports the presence of persistent low-grade inflammation among welding workers exposed to airborne contaminants (see **Table 2**). The reduction in lymphocytes may indicate immune modulation or redistribution associated with chronic inflammatory stress. The elevation in monocytes and granulocytes reflects activation of innate immune responses due to prolonged exposure to airborne pollutants in

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

welding workshops. These findings further support the presence of persistent low-grade systemic inflammation among welding workers [21].

INTEGRATED ENVIRONMENTAL IMPACT SUMMARY

Fig. 4 summarizes the relative percentage change in hematological parameters among welding workers compared to controls. The largest increase was observed in PCV and hemoglobin, followed by inflammatory markers, while lymphocytes showed a relative decrease. These findings illustrate the combined hypoxic and inflammatory responses associated with occupational environmental exposure. Percentage change (%) can be calculated via the following formula [14]

$$\text{Percentage change (\%)} = \frac{(\text{Workers} - \text{Control})}{\text{Control}} \times 100 \quad (2)$$

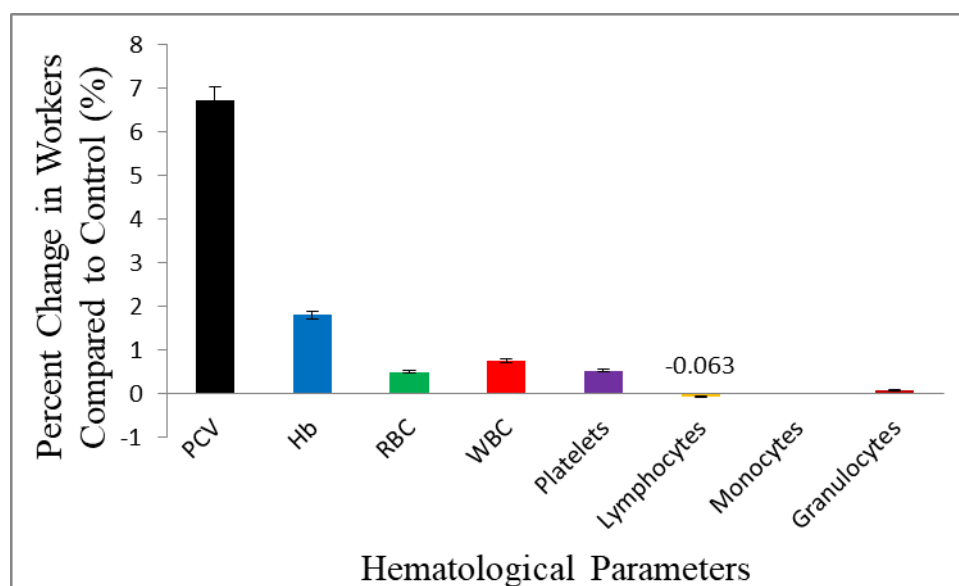


Fig. 4. Summary of significant hematological changes due to environmental exposure

The findings in this work indicated that hematological parameters of welders exposed to acetylene and welding fumes were altered heavily. Compared with the controls, welders from exposed group had significantly higher levels of PCV, hemoglobin (Hb) concentration, RBC count and WBC count, whereas lower level of lymphocyte count was observed in this group. The simultaneous rise of PCV, hemoglobin and RBC is indicative of a compensatory physiological reaction to chronic hypoxia due to exposure in the workplace to combustion byproducts (in particular carbon monoxide). Carbon monoxide decreases the binding of oxygen by promoting the formation carboxyhemoglobin and erythropoietin, which stimulates erythropiesis. This adaptation could account for the raised red cell mass in welders. At the same time, the rise in white blood cells, monocytes, granulocyte and platelets demonstrates an activation of a systemic inflammatory process. Inhalation of welding fumes with toxic metal oxides and gases participates in respiratory epithelial irritation, oxidative stress and pro-inflammatory cytokines production. These effects activate bone marrow and induce mobilization of immune cells into the peripheral blood. The reduced number of lymphocytes also indicates immune modulation, which might correlate with the slow dominance of innate immune response in chronic environmental stress. To summarize, our results support the hypoxic adaptation in welders and low-grade inflammation. The hematological variations described in this work are physiological modifications due to prolonged occupational exposure to air pollutants under poorly ventilated workshop conditions. These findings demonstrate the importance of environmental and industrial exposure to welding fume emissions on systemic hematopoietic condition. These changes demonstrate a need for the provision of effective environmental protection measures, such as improvement in ventilation systems; regular monitoring of air quality data; and adherence to stringent occupational safety guidelines with an aim to minimise welders' long term risk of ill-health. [9].

CONCLUSION

The present study demonstrates that occupational exposure to acetylene gas and welding fumes in welding workshops in Fallujah is associated with significant hematological alterations. Welding workers exhibited statistically significant increases in packed cell volume (PCV), hemoglobin concentration, red blood cell (RBC) count, white

*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

blood cell (WBC) count, platelet count, monocyte percentage, and granulocyte percentage, alongside a significant decrease in lymphocyte percentage compared to the control group.

The elevation of erythrocytic indices (PCV, hemoglobin, and RBC count) suggests a compensatory physiological response to chronic hypoxic conditions, likely induced by carbon monoxide exposure during acetylene combustion. Carbon monoxide reduces oxygen transport efficiency, stimulating erythropoietin secretion and enhanced erythropoiesis. Concurrently, the increase in total leukocytes, monocytes, granulocytes, and platelets indicates activation of systemic inflammatory pathways, possibly resulting from inhalation of metal oxide particles and toxic gases. The reduction in lymphocytes may reflect immune modulation associated with prolonged environmental stress.

Collectively, these findings indicate that welding-related environmental exposure contributes to both hypoxic adaptation and persistent low-grade inflammation. Although the observed changes may initially represent adaptive mechanisms, prolonged exposure may increase the risk of long-term health complications.

The study highlights the urgent need for improved occupational and environmental control measures in welding workplaces, including enhanced ventilation systems, regular air quality monitoring, and strict adherence to safety protocols. Implementation of these measures is essential to minimize exposure and protect the hematological and overall health of welding workers.

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*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq

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*Corresponding author

Harith A. Ahmed,

Department of Environmental, College of Applied Sciences-Hit, University Of Anbar, Anbar, Iraq

e-mail: harith.alnajjar@uoanbar.edu.iq