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# Immune markers and oxidative stress among welders prolongedly exposed to heavy metals

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## ABSTRACT

Chronic occupational exposure to heavy metals is among the most significant health risks faced by workers in environmentally hazardous industries, particularly in welding. These elements are associated with cumulative toxic effects on the immune and renal systems, as well as redox balance, often leading to long-term chronic health disorders. A cross-sectional study was conducted in Ramadi, Iraq, from January 1 to February 1, 2024, including 40 welders exposed to metal fumes and a control group of 20 healthy, unexposed individuals. Participants were aged 25–60 years, with welding work ranging from 5 to 30 years, stratified into four groups with 5-year exposure intervals. Blood and urine samples were collected from all subjects to assess immunoglobulins (IgE) and cytokines (IL-5), neutrophil gelatinase-associated lipocalin (NGAL), oxidative stress biomarkers (GSH-PX), and blood metal levels (Pb, Cd, Cr).

Significant increases in mean ± SD IgE levels were observed, rising from 86.2 ± 22.7 IU/mL (5 years exposure) to 139.8 ± 39.0 IU/mL (25–30 years), compared with 66.9 ± 21.7 IU/mL in controls ($p < 0.001$). The mean ± SD IL-5 increased numerically from 12.8 ± 3.1 to 17.2 ± 5.1 pg/mL, compared with 9.2 ± 2.7 pg/mL in controls, but did not reach statistical significance ($p = 0.088$). Urinary NGAL mean ± SD rose significantly from 120.8 ± 21.3 to 191.6 ± 39.2 ng/mL, compared with 104.5 ± 23.3 ng/mL in controls ($p = 0.001$), suggesting progressive tubular kidney damage. GSH-PX mean ± SD activity decreased markedly from 45.2 ± 9.2 to 26.7 ± 9.1 units/mL, against 47.6 ± 11.6 units/mL in controls ($p = 0.002$), indicating diminished antioxidant defense. Lead and cadmium blood levels showed linear increases, with mean ± SD Pb rising from 25.7 ± 9.1 to 66.1 ± 13.7 µg/dL and mean ± SD Cd rising from 3.2 ± 1.1 to 8.0 ± 2.4 µg/L, while chromium remained elevated but not statistically significant. The study reveals a clear cumulative impact of chronic heavy metal exposure in welding environments on workers' immune, renal, and oxidative systems, with severity increasing alongside years of service, notably beyond 10 years. These findings support the urgent need for enhanced preventive measures and ongoing medical surveillance among long-term exposed workers to curb progressive health risks.

**Keywords:** *Heavy metals, Immune biomarkers, Industrial pollutants, Oxidative stress*

## 1 INTRODUCTION

Heavy metals such as cadmium (Cd), lead (Pb), and chromium (Cr) are recognized as trace elements in the human body; however, prolonged exposure through environmental and industrial pollutants has toxic effects on human health, especially with chronic occupational exposure in settings such as welding. These metals, characterized by high density and toxicity even at low concentrations, can accumulate in biological tissues, resulting in a range of health disorders affecting the nervous, immune, and renal systems [1]. Chronic occupational exposure refers to repeated and prolonged contact with

these metals via inhalation or dermal absorption over months and years, leading to bioaccumulation and long-term adverse health effects [2].

In assessing the health effects of this exposure, immune markers such as immunoglobulin E (IgE) are informative. IgE is an immune protein that mediates enhanced immune responses in allergy and chronic inflammation. In addition, interleukin-5 (IL-5), a cytokine that stimulates eosinophils involved in inflammatory processes, plays a key role in immune responses, particularly in regulating and activating eosinophils (a type of white blood cell). IL-5 is also implicated in the development of eosinophilia (an increased number of eosinophils in the blood), allergy, and asthma. Additionally, the urinary biomarker neutrophil gelatinase-associated lipocalin (NGAL) is used to detect and assess kidney injury, particularly acute kidney injury (AKI). NGAL is stored in neutrophils and produced by various tissues, including the kidney, and its levels increase rapidly in blood and urine during kidney damage [3–6], providing early indicators of immune system dysregulation caused by heavy metal exposure.

Furthermore, oxidative stress, a state induced by excessive reactive oxygen species (ROS) that damages DNA, proteins, and lipids, is a key mechanism underlying heavy metal toxicity [7]. Glutathione peroxidase activity (GSH-PX) serves as a measure of the body's antioxidant defense, neutralizing ROS and protecting cellular integrity [8]. The mechanisms by which heavy metals negatively affect human health include inhalation or dermal absorption, distribution into the bloodstream, tissue deposition, ionic imbalance within cells, disruption of cellular signaling, and activation of chronic inflammation via cytokines such as TNF-$\alpha$ and IL-6, culminating in tissue damage and organ dysfunction. These metals also impair natural antioxidant systems, leading to ROS accumulation and genetic and enzymatic changes that increase mutation and carcinogenesis risks. These multifactorial effects on the nervous system, liver, kidneys, and immunity have been extensively documented [7–9].

Global statistics indicate that occupational heavy metal exposure triggers over 1.5 million annual disease cases, with elevated mortality due to kidney disease and cancers [2]. Recent findings corroborate that chronic exposure induces immune dysregulation and oxidative imbalances linked to multiple chronic diseases [5, 9] Cadmium, lead, and hexavalent chromium (Cr(VI)) are classified carcinogens that promote oxidative stress and cellular damage [7].

Research by Jaishankar et al. (2014) demonstrates that these metals disrupt biological functions through oxidative mechanisms and immune modulation, leading to chronic illnesses, including cancer and neurological disorders [9]. A 2023 review reported elevated IgE, IL-5, and NGAL levels in occupationally exposed individuals, reflecting chronic inflammation [5]. Mousavi et al. (2023) highlighted decreased antioxidant enzyme activity (e.g., GSH-PX), impairing cellular defenses [8]. Kumar and Singh (2021) confirmed genotoxic and oxidative damage induced by Cr(VI), lead, and cadmium [7]. The WHO (2023) emphasizes environmental monitoring and preventive interventions in high-risk industries such as welding [2]. Wang et al. (2022) observed that occupational exposure impairs immune balance and upregulates inflammatory gene expression, increasing chronic disease risk [10]. Li et al. (2023) further confirmed that exposure reduces the efficacy of antioxidant enzymes, thereby fostering oxidative stress and cellular damage [11].

Collectively, chronic heavy metal exposure triggers complex immune disturbances, as evidenced by elevated IgE levels, with implications for allergic and inflammatory responses [5]. IL-5 plays a central role in eosinophil activation, while NGAL effectively indicates kidney injury and inflammation [3, 4]. These markers are valuable for elucidating the negative immunological effects of occupational exposure to heavy metals. By reviewing scientific literature, the importance of this study becomes clear: these findings underscore the need to assess immune and oxidative stress biomarkers among welders to detect health risks early and develop targeted preventive and therapeutic strategies. Measuring IgE, IL-5, NGAL, and antioxidant enzyme activity (GSH-PX) provides an integrated view of the biological impact of occupational exposure, supporting the implementation of health programs that ensure worker safety.

## 2 MATERIALS AND METHODS

### 2.1 Sample collection

Samples were collected between January 1 and February 1, 2024. The study included 40 welders and 20 healthy individuals from Ramadi, serving as the control group. Participants' ages ranged from 25 to 60 years, and their welding exposure durations ranged from 5 to 30 years. The selection aimed to represent age groups and exposure durations appropriately, enabling an accurate assessment of chronic heavy metal effects in the work environment.

### 2.2 Study samples

Blood samples were collected from all participants, with a volume of 8 mL per individual, using two types of sterile tubes, depending on the analysis required. A total of 5 mL was collected, with 2 mL transferred into EDTA tubes. The remaining 3 mL were placed into dry tubes without anticoagulant to obtain serum for immunological marker analyses, including IgE and IL-5, as well as for assessing oxidative stress via glutathione peroxidase (GSH-PX) enzyme activity. For neutrophil gelatinase-associated lipocalin (NGAL) analysis, urine samples were collected in sterile containers.

Immediately after collection, samples were centrifuged at 3,000 rpm for 10 min to separate plasma or serum, depending on the sample type. Plasma and serum were transferred into sterile Eppendorf tubes and frozen at -20 °C or below until analysis to maintain biomarker stability and prevent degradation, in accordance with internationally accepted protocols [3, 8, 9].

Additionally, part of the blood sample was allocated for measuring heavy metal concentrations (lead, cadmium, chromium) using an atomic absorption spectrophotometer (AAS) with high precision and sensitivity at low concentrations. Samples were prepared according to validated protocols, including appropriate chemical treatments for metal separation to ensure accuracy. Combining metal concentration measurements with immunological and antioxidant activity analyses provides a comprehensive assessment of occupational exposure effects, enhancing the study's reliability and thoroughness. These procedures ensure result accuracy and sample integrity, aligning with international standards for biological sample collection and storage.

### 2.3 Human immunoglobulin E (IgE)

The concentration of human immunoglobulin E (IgE) was determined using a SunLong Biotech ELISA kit (Catalog No. SL0913Hu) by sandwich ELISA. All reagents and samples were prepared according to the manufacturer's instructions. 100 µL of each sample or standard was pipetted into microtiter wells precoated with monoclonal anti-IgE antibodies. The plates were incubated for 90 min at room temperature to allow antigen–antibody binding. Following five washes with wash buffer to remove unbound substances, 100 µL of HRP-conjugated detection antibody was added and incubated for 30 min. The wells were washed again, and 90 µL of TMB substrate was added, incubated in the dark for 15–20 min to develop color. The reaction was stopped with 50 µL of diluted sulfuric acid, and the optical density was measured at 450 nm. Each sample was assayed in duplicate, and IgE concentration was calculated from a standard curve.

### 2.4 Human interleukin-5 (IL-5)

Human interleukin-5 was measured using a SunLong Biotech ELISA kit (Catalog No.: SL0998Hu) by sandwich ELISA. Similar to IgE, 100 µL of sample or standard was incubated in precoated wells for 90 min at room temperature, washed five times, incubated with 100 µL of HRP-conjugated detection antibody for 30 min, and then washed again. 90 µL of TMB substrate was added, incubated for 15–20 min, then 50 µL of stop solution was added, and the mixture was read at 450 nm. Measurements were conducted in duplicate and calculated using the standard curve.

### 2.5 Urinary NGAL (Lipocalin-2)

Urine NGAL was assessed with a SunLong Biotech ELISA kit (Catalog No. EL0252Hu) via sandwich immunoassay. Samples and standards (100 µL) were added to microtiter wells coated with anti-lipocalin-2 antibodies and incubated for a build-up period at room temperature. After washing, 100 µL of HRP-conjugated antibody was added, incubated, and washed. TMB substrate was incubated in the dark for 15–20 min, then stopped with acid, and absorbance was measured at 450 nm. All assays were performed multiple times to ensure accuracy, and NGAL concentration was derived from calibration curves.

### 2.6 Glutathione peroxidase (GSH-PX) Activity

The activity of the human GSH-PX enzyme was measured using a SunLong Biotech ELISA kit (Catalog No.: SL0778Hu). 10 µL of sample was incubated in antibody-coated wells at 37 °C for 90 min, washed three times, followed by 100 µL of biotinylated detection antibody (60 min, 37 °C), washed, then 100 µL of HRP conjugate (30 min, 37 °C). Following five washes, 90 µL of TMB substrate was incubated for 15 min in the dark, stopped with 50 µL of stop solution, and absorbance was measured at 450 nm. Values were calculated using a standard curve (0.3–20 ng/mL range).

### 2.7 Heavy metal quantification in blood

Heavy metal levels (Pb, Cd, Cr) were quantified using an atomic absorption spectrophotometer. A total of 5 mL of venous blood was collected per participant (2 mL EDTA, 3 mL plain). For metal analysis, samples were digested with concentrated $HNO_3$ (65%) and heated at 95–100 °C for 2–3 h to ensure complete breakdown of organic matter. After cooling, samples were diluted with deionized water to 25–50 mL. Calibration with standard solutions (0.1, 0.5, 1, 5, 10 µg/dL) was performed. Metal-specific wavelengths were used, 283.3 nm for Pb, 228.8 nm for Cd, and 357.9 nm for Cr, under optimized flames or a graphite furnace. Flow rates of acetylene/oxygen were adjusted to maintain flame temperatures of 2300–2500 K. Each sample reading took approximately 3–5 min, based on absorbance by free atoms, and concentrations were determined from calibration curves. This method is extremely sensitive and employs robust QA/QC procedures in accordance with international guidelines [2, 9, 12].

### 2.8 Statistical analysis

Descriptive statistics (mean ± SD) were calculated for all biomarkers and heavy metal concentrations across each occupational exposure subgroup and the control group. A one-way analysis of variance (ANOVA) was performed to detect overall differences among the groups (welders stratified by exposure duration and control). Tukey's HSD post hoc test was applied for pairwise comparisons to clarify which specific time-exposure categories showed significant differences. A p-value < 0.05 was considered statistically significant.

## 3 RESULTS

The statistical data from this study provide an extensive analysis of the impact of chronic occupational exposure to heavy metals on welders' biological health. This was evaluated using a set of biomarkers, including immune indicators (IgE, IL-5), oxidative-stress-related biochemical markers (GSH-PX), renal indicators (NGAL), and blood concentrations of heavy metals (Pb, Cd, Cr). Comparisons were made between welders and a control group of healthy, unexposed individuals, and differences in exposure duration were examined across multiple time categories.

All statistical analyses used descriptive methods (mean ± SD) and inferential techniques, including one-way ANOVA and Tukey post hoc tests, to detect significant differences between groups and the cumulative impact of exposure duration. The results are illustrated in the figures below, highlighting significant changes in biomarkers across study groups along with p-values for each comparison.

### 3.1 Immunoglobulin E (IgE)

The data indicated that mean ± SD serum IgE levels steadily increased with longer durations of welding exposure. The lowest IgE mean ± SD level was observed in the group with 5 years of service (86.2 ± 22.7 IU/mL). This means ± SD rose to 122.5 ± 41.4 IU/mL in the 5–<10 years group, then 131.7 ± 39.5 IU/mL in the 10–<15 years group. It further increased to 135.9 ± 38.7 IU/mL in the 15–<20 years cohort, 138.8 ± 39.2 IU/mL in the 20–<25 years group, and peaked at 139.8 ± 39.0 IU/mL in welders with 25+ years of experience. In contrast, the mean ± SD of the control group was 66.9 ± 21.7 IU/mL. ANOVA revealed highly significant differences ($p < 0.001$), and Tukey HSD tests demonstrated statistically significant differences between most duration-based subgroups and the control group (Figure 1).

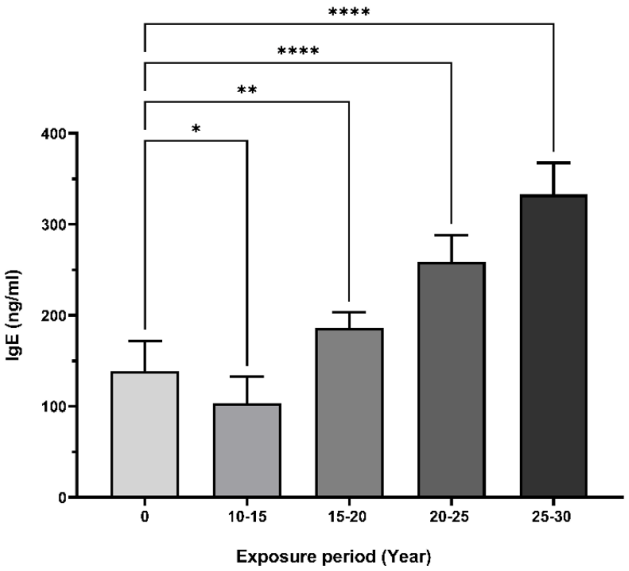


**Fig. 1** Mean±SD IgE concentration among welders exposed to heavy metals, stratified by exposure duration, compared with the healthy control group

### 3.2 Interleukin-5 (IL-5)

The results show a notable rise in mean ± SD IL-5 concentrations with increasing years of service: 12.8 ± 3.1 pg/mL (5 years), 15.4 ± 4.4 pg/mL (5–<10 years), 16.2 ± 4.6 pg/mL (10–<15 years), 16.7 ± 5.1 pg/mL

(15–<20 years), 17.1 ± 5.2 pg/mL (20–<25 years), and 17.2 ± 5.1 pg/mL (25–30 years). The control group was 9.2 ± 2.7 pg/mL. Although the upward trend aligns with exposure duration, ANOVA did not show statistically significant differences across groups (p = 0.088); however, the difference from controls remains evident (Figure 2).

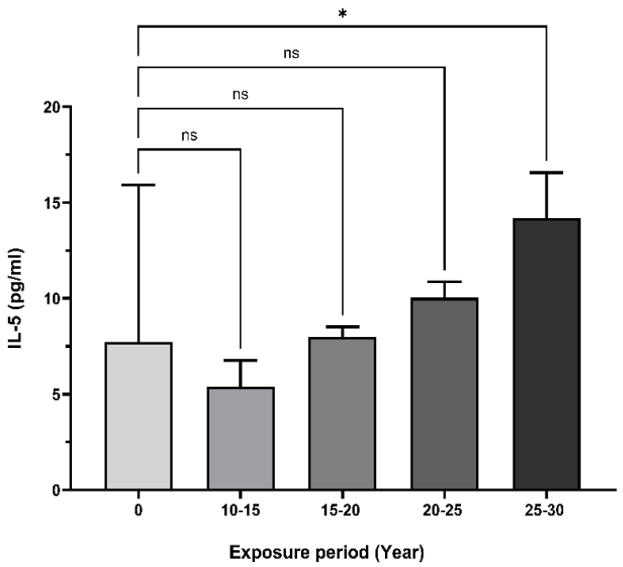


**Fig. 2** Mean±SD IL-5 concentration among welders exposed to heavy metals, according to exposure duration, compared with the healthy control group

### 3.3 Renal biomarker: Urinary NGAL

A consistent, statistically significant increase in urinary NGAL was observed with increasing years of service among welders. Mean ± SD NGAL was 120.8 ± 21.3 ng/mL (5 years), 171.2 ± 31.7 ng/mL (5–<10 years), 179.1 ± 34.4 ng/mL (10–<15 years), 186.7 ± 37.8 ng/mL (15–<20 years), 190.4 ± 37.9 ng/mL (20–<25 years), peaking at 191.6 ± 39.2 ng/mL (25–30 years). In contrast, the control mean ± SD was 104.5 ± 23.3 ng/mL. This trend was highly significant (p = 0.001), confirming substantial intergroup differences by exposure duration (Figure 3).

### 3.4 Oxidative-stress enzyme activity: GSH-PX

Analysis of the antioxidant enzyme glutathione peroxidase (GSH-PX) revealed a clear, progressive decline with increasing years of service. Mean ± SD levels were 45.2 ± 9.2 units/mL (5 years), 33.2 ± 7.8 units/mL (5–<10 years), 29.3 ± 8.3 units/mL (10–<15 years), 27.8 ± 9.2 units/mL (15–<20 years), 26.9 ± 9.2 units/mL (20–<25 years), reaching a minimum of 26.7 ± 9.1 units/mL (25–30 years). The control mean ± SD was 47.6 ± 11.6 units/mL. Differences were statistically significant (p = 0.002) (Figure 4).

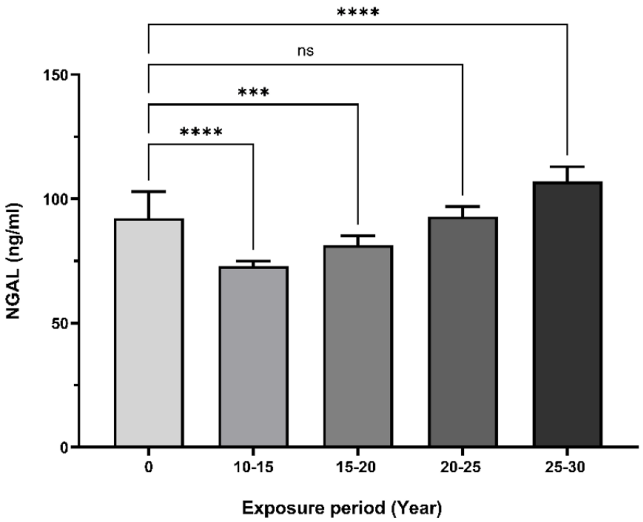


**Fig. 3** Mean±SD NGAL concentration among welders exposed to heavy metals, stratified by exposure duration, compared with the healthy control group

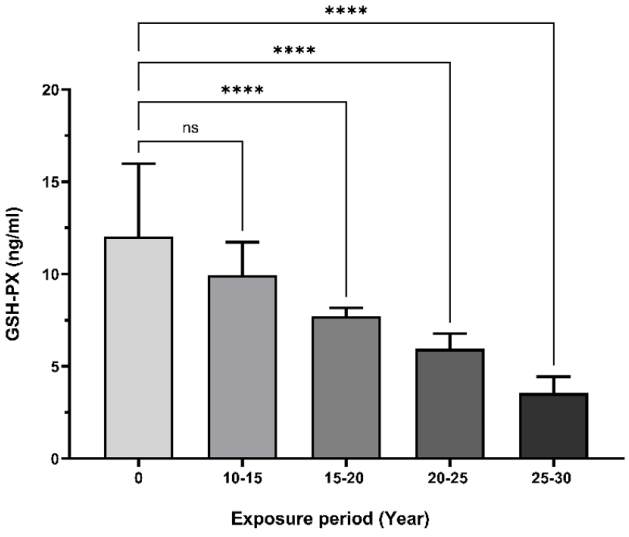


**Fig. 4** Mean±SD GSH PX enzyme activity among welders compared with the healthy control group, according to the duration of exposure to heavy metals

### 3.5 Heavy metal blood concentrations

#### 3.5.1 Lead (pb)

Blood lead levels rose significantly with years of exposure. The control mean ± SD was 19.3 ± 7.9 µg/dL. Among welders, the mean ± SD gradually increased: 25.7

± 9.1 µg/dL (5 years), 53.0 ± 12.2 µg/dL (5–<10 years), 59.6 ± 12.9 µg/dL (10–<15 years), 63.8 ± 13.5 µg/dL (15–<20 years), 65.5 ± 13.7 µg/dL (20–<25 years), and 66.1 ± 13.7 µg/dL (25–30 years). ANOVA showed strong significance ($p < 0.001$), and Tukey HSD confirmed marked differences between most sequential exposure categories (Figure 5).

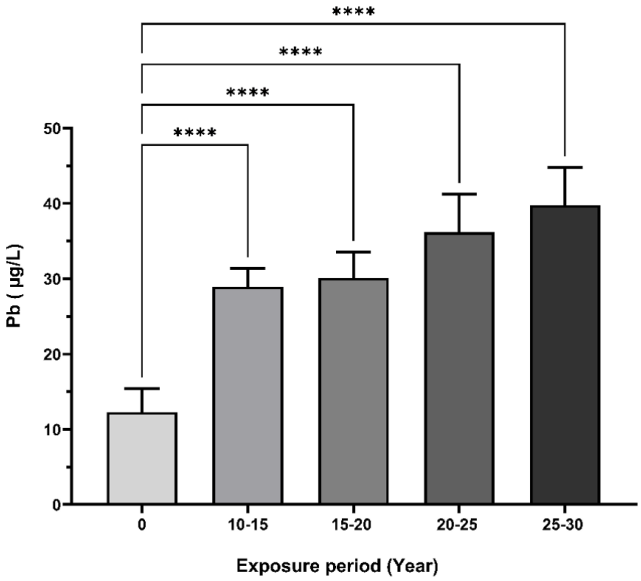


**Fig. 5** Mean±SD Pb concentration among welders exposed compared with the healthy control group, according to the duration of exposure to heavy metals

### 3.5.2 Cadmium (cd)

Cadmium levels increased significantly with service duration. The control mean ± SD was 2.1 ± 0.9 µg/L. Welders in the 5-year group showed 3.2 ± 1.1 µg/L, rising to 6.8 ± 1.8 µg/L (5–<10 years), 7.2 ± 1.9 µg/L (10–<15 years), 7.8 ± 2.2 µg/L (15–<20 years), 7.9 ± 2.3 µg/L (20–<25 years), and reaching 8.0 ± 2.4 µg/L (25–30 years). The variation was statistically significant ($p = 0.004$), with Tukey HSD indicating that most higher exposure categories significantly exceeded lower and control groups (Figure 6).

### 3.5.3 Chromium (cr)

Mean ± SD blood chromium levels were as follows: control group, 1.2 ± 0.6 µg/L; welders, 2.9 ± 1.1 µg/L (5 years), 3.3 ± 1.5 µg/L (5–<10 years), 3.5 ± 1.5 µg/L (10–<15 years), 3.7 ± 1.6 µg/L (15–<20 years), 3.8 ± 1.6 µg/L (20–<25 years), and 3.9 ± 1.6 µg/L (25–30 years) (Figure 7).

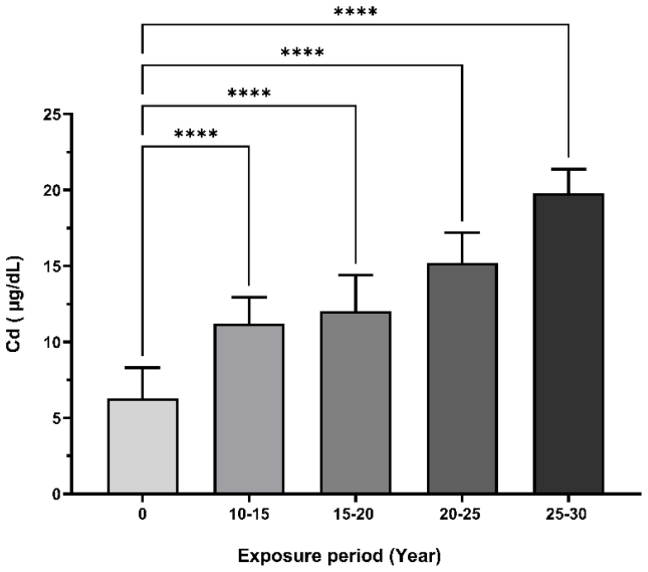


**Fig. 6** Mean±SD Cd concentration among welders compared with the healthy control group, according to duration of exposure to heavy metals

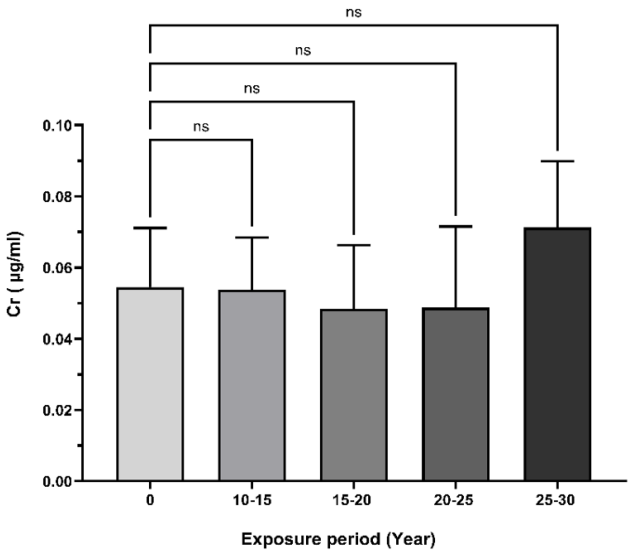


**Fig. 7** Mean±SD Cr concentration among welders compared with the healthy control group, according to duration of exposure to heavy metals

## 4 DISCUSSION

Results from this study underscore the complex biological effects of chronic exposure to heavy metals among welders, including abnormal immune responses, early signs of renal injury, and disruption of cellular antioxidant balance. These findings align with the established

understanding that heavy metals function as multisystem toxicants, exerting cumulative effects even at subclinical exposure levels.

### 4.1 Immune biomarkers: IgE and IL-5

The current study observed marked increases in immunoglobulin E (IgE) and interleukin-5 (IL-5), reflecting an activated immune response pathway mediated by type 2 T-helper (Th2) cells, a pattern commonly observed in allergic conditions and chronic exposure to environmental stimuli. The study by Kim et al. (2019), conducted among battery factory workers, reported similar findings, with elevated IgE and IL-5 levels and a decrease in interferon-gamma (IFN-Y), indicating a shift in the immune response from the Th1 to the Th2 phenotype under the influence of lead exposure. The researchers explained this phenomenon by activation of the IL-4/IL-5 axis by lead, which stimulates Th2 helper T cells, leading to excessive IgE production and eosinophil proliferation. Furthermore, the findings of Ahmed et al. (2021) support this hypothesis by establishing an association between increased cadmium levels and elevated IL-5 in patients with respiratory allergies, demonstrating that cadmium enhances inflammatory and allergic responses through IL-5 production and eosinophil activation [13]. Therefore, heavy metals affect T and B lymphocytes by modulating immune gene expression and increasing reactive oxygen species (ROS) production, which interfere with intracellular signaling pathways within immune cells [14].

In a multicenter study conducted in Asia, the mean IgE level among exposed individuals was 220 ng/mL, compared with 120 ng/mL in non-exposed individuals, with a statistically significant difference ($p < 0.001$) [15]. A study [16] demonstrated that higher blood levels of cadmium and lead are associated with increased immune-inflammatory markers, reflecting a shift in immune response pathways frequently involving cytokines such as IL-5. Wells, Schwartz, and Krishnan (2014) reviewed the immunotoxic effects of heavy metal exposure, focusing on lead- and cadmium-mediated inflammation relevant to asthma. They concluded that both lead and cadmium induce immune dysregulation by promoting inflammatory pathways, particularly by shifting immune responses toward a Th2 phenotype characterized by increased cytokine production, including IL-5. This shift promotes eosinophilic inflammation, a central feature in the pathology of allergic asthma. Chronic exposure to these metals results in heightened immune activation and exacerbates asthma symptoms, underscoring the need to reduce environmental and occupational exposure to mitigate asthma risk [17]. A broader epidemiological investigation by Zhang et al. (2023) showed that heavy metal exposure compromises immune responsiveness and vaccine efficacy in children by reducing antibody and cytokine production [5].

### 4.2 Renal indicator: Urinary NGAL

The elevated urinary NGAL observed in welders aligns with [18], which identified NGAL as an early biomarker for cadmium-induced renal tubular damage even before traditional markers shift. Fujishiro et al. (2020) demonstrated a positive correlation between urinary cadmium and NGAL, indicating subclinical tubular injury. Cadmium tends to accumulate in the renal cortex, inducing tubular damage via ROS, altered transport, and proteinuria [19]. A 2024 review [20] suggested that NGAL, along with KIM-1 and NAG, serves as an early renal injury indicator and independently predicts chronic renal impairment and mortality. Kim et al. (2022) reported that NGAL elevation precedes increases in serum creatinine or overt renal failure [21]. Liu et al. (2022) reported that welders exposed to heavy metals exhibited early signs of kidney injury, as indicated by elevated urinary levels of neutrophil gelatinase-associated lipocalin (NGAL), a sensitive biomarker of renal tubular damage [22]. Cadmium- and lead-induced tubular cell damage triggers NGAL secretion, which accumulates in blood and urine, allowing early detection before changes in creatinine or glomerular filtration rate.

### 4.3 Oxidative stress: GSH-PX activity

A significant decline in GSH-PX activity was observed, indicating heavy metal-induced suppression of antioxidant defenses. Flora et al. (2012) concluded that Pb and Cd reduce GSH-PX activity by depleting selenium and downregulating antioxidant enzyme gene expression [23]. Jomova and Valko (2011) attributed the mechanism to heavy metal-induced ROS generation overwhelming cellular antioxidant systems, leading to protein and DNA damage [24]. A Turkish study (Demir et al., 2023) demonstrated that co-exposure to Pb, Cd, and Cr significantly lowered GSH-PX activity and elevated oxidative stress markers, raising chronic disease risk [25]. A study (Laoye et al., 2025) confirmed that these metals increase ROS production and inhibit antioxidant enzymes, contributing to protein/DNA oxidation, cancer, cardiovascular disease, and renal disease [26]. Another study indicated that heavy metals bind sulfhydryl groups in

antioxidant enzymes, inhibiting them and exacerbating oxidative stress [27]. Heavy metal-induced ROS impairs GSH-PX's ability to reduce oxygen, destabilizing antioxidant defenses, triggering membrane damage, and oxidizing proteins.

### 4.4 Heavy metal accumulation (pb, cd, cr)

This study documented clear accumulation of lead and cadmium in welders, consistent with [1], which reported gradual deposition in the kidney, nervous system, and immunity. Järup (2003) noted that chronic cadmium exposure leads to renal damage and osteoporosis even in asymptomatic people. The effects of cadmium are slow and covert, yet cumulative and hazardous [28]. Satarug et al. (2020) highlighted that even low-level cadmium exposure, such as that observed in control subjects, may lead to chronic biological effects if exposure persists and renal excretion is inadequate [29]. Co-exposure to multiple heavy metals results in cumulative and synergistic health impacts, amplifying immune, renal, and oxidative disturbances beyond those caused by single-metal exposure [15]. These findings underscore the value of including advanced biomarkers such as NGAL and GSH-PX in occupational health monitoring, alongside immune markers that detect subclinical chronic risks.

## 5 CONCLUSION

According to the findings, welders are a high-risk occupational group due to chronic toxic exposure to heavy metals. Their silent physiological changes, elevated hypersensitivity markers (IgE, IL-5), early renal damage (NGAL), and disturbed cellular redox balance (GSH-PX) carry significant biological and clinical relevance, calling for proactive surveillance and preventive strategies in occupational health programs.

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**Data availability**

N/A

## DECLARATIONS

**Conflict of interest**

The authors declare no conflict of interest.

**Consent to publish**

N/A

**Ethical approval**

The University of Anbar's ethical committee gave its approval for this investigation (approval ID: ref 101, 6/8/2023).

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