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RESEARCH ARTICLE

Investigating the Role of Some Biomarkers in Assessing the Proposed Air Pollution Effects in Selected Areas in Erbil Governorate

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ABSTRACT

The aim of this study was to evaluate the levels of urinary Hydroxyproline, urinary Malondialdehyde, urinary Creatinine, and urinary Albumin as potential biomarkers for air pollution in two distinct sites: Site 1, an industrial area and Site 2, a non-industrial area, also measuring the urine albumin-to-creatinine ratio (UACR) to further investigate the effects of air pollution. The study employed a cross-sectional design with 90 participants. Site 1 had 56 participants (42 adults, 14 children, 11.2% of the total population), while Site 2 had 34 participants (23 adults, 11 children, 11.3% of the total population). The findings of the study indicated that the concentrations of Hydroxyproline and Malondialdehyde were significantly higher at Site 1 for both adults and children compared to Site 2. Biomarkers indicate more sensitive to industrial air pollution exposure. On the other hand, the concentrations of Creatinine and Albumin were found to be higher at Site 1; however, the difference was not statistically significant when compared to Site 2 for both adults and children. UACR values were measured at Site 1 and Site 2. For adults, the UACR values were 10.093 and 8.870 mg/g, respectively, while for children, they were 11.061 and 9.882 mg/g, respectively. All values were within the normal range, suggesting that air pollution did not significantly impact kidney function in this study. Elevated hydroxyproline levels indicate collagen alterations, which could be caused by air pollution-induced tissue injury. Increased malondialdehyde and urine albumin levels indicate oxidative stress and renal impairment caused by exposure to air pollution.

Keywords: Albumin, Biomarker, Creatinine, Hydroxyproline, Malondialdehyde

Introduction

Air pollution is one of the most important health challenges confronting almost every country on the planet. The world's growing urbanization has resulted in a high rate of emissions produced by industry and motor vehicles. Currently, 50% of individuals who live in cities and metropolitan areas are continually exposed to air pollution.[1] Biomarkers are biological markers that show how xenobiotics interact with specific cellular, biochemical, or molecular targets. These biomarkers can be measured to reveal any early, reversible, or irreversible modifications that occur before major structural or functional alterations of molecules or cells.[2] Studies on biological monitoring are crucial for determining the risk variables for occupational pollution exposure.[3] People who breathe polluted air suffer from both acute and chronic illnesses.[4] Three categories of biomarkers—exposure, effect, and susceptibility—are used to assess occupational human health.[5] Biomonitoring is the measurement of metabolites, chemicals, or reaction products in human blood, sputum, urine, saliva, and exhaled breath condensate to determine internal exposure to dangerous compounds.[6] Biomarkers of exposure are regarded as an essential tool since they measure the amounts at which chemical compounds are absorbed by measuring hazardous chemicals or

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metabolites in biological specimens.[7] Blood and urine samples have been used to set limit values for human biomonitoring measures such as biological exposure indices because they are regarded as trustworthy indicators of recent body burden.[8]

Hydroxyproline stands out as a non-proteinogenic collagen amino acid that contributes to the integrity of the triple helix. It came about as a result of prolyl-hydroxylase hydroxylating proline post-translationally.[9] Oxidative stress arises when there is an imbalance between the creation of reactive oxygen species or reactive nitrogen species and the body's antioxidant defense mechanisms. Initially, an adaptive response occurs in the form of induction of antioxidant production. However, when antioxidant depletion occurs, cellular injury and dysfunction can follow. Therefore, oxidative stress can lead to detrimental effects on cells and tissues.[10] Oxygen free radicals are very reactive chemicals that can cause damage to biological components. The proteins, nuclear DNA, and lipids of living organisms are all prone to damage from these radical species. ROS damage to biological cells is referred to as oxidative stress.[11] Malondialdehyde (MDA) is an organic molecule that occurs naturally and can be utilized as a predictor of oxidative stress.[12] MDA is regarded as a crucial biomarker for assessing levels of total lipid peroxidation and as a sign of free radical damage brought on by oxidative stress. MDA, a byproduct of lipid peroxidation, is widely used to measure the body's level of oxidative stress. Since free radicals and other ROS can harm cells and tissues, their presence indicates that they are being produced. As a result, monitoring MDA levels is a helpful method for determining the degree of oxidative stress present in the body.[13] Creatinine is an amino acid molecule that is generated from creatine. When creatine is metabolized in skeletal muscle and releases Creatinine into circulation, it is readily filtered by the nephron tubule and eliminated through the urine.[14]

Albumin is the most prevalent protein in the blood, and very little intact Albumin is eliminated by the kidneys under normal renal function. The glomerular filtration of Albumin is enhanced after renal injury, and the tubules' capacity to reabsorb and degrade Albumin is reduced. As a result, there are more intact Albumin molecules in the urine.[15] As a result, air pollution may have an effect on renal function, which may be measured using the estimated urine Albumin/Creatinine ratio (UACR).[16] Previous studies have used these biomarkers as indications of air pollution. A study conducted on air pollution measured as a particular matter with an aerodynamic diameter of 2.5 m or less (PM2.5) was associated with higher urine MDA values.[17] According to this study, exposure to PM2.5 during the early stages of pregnancy is associated with greater levels of systemic oxidative stress, as demonstrated by elevated levels of malondialdehyde (MDA).[18] The study investigated outdoor air pollution's impact on health in two Iranian cities. In individuals exposed to pollution, there were increased levels of malondialdehyde (MDA).[19] A meta-analysis found links between higher particulate matter (PM2.5 and PM10) exposure and chronic kidney disease. Carbon monoxide (CO) and nitrogen dioxide (NO2) have also been linked to an increased risk of chronic kidney disease. Exposure to particulate matter (PM10 and PM2.5) was linked to a decrease in estimated glomerular filtration rate (eGFR).[20] A cross-sectional study examined urine hydroxyproline (UHP) levels in urban and rural joggers exposed to nitrogen dioxide (mean UHP: 25.02 mg/24 h/m^2), indicating a possible relationship between greater UHP and nitrogen dioxide exposure.[21] The objective of this study is to assess the levels of Hydroxyproline, Malondialdehyde, Creatinine and Albumin and (urine Albumin/Creatinine ratio) in urine as potential biomarkers for measuring air pollution and to compare the concentrations of these biomarkers between two locations: an industrial site (S1) and a non-industrial site (S2).

Materials and methods

Study area

Erbil City has had substantial air pollution problems in recent decades as a result of increased industrialization and urbanisation.[22] Tymar village, located at 36°06'32.98"N 43°58'44"E, with 500 inhabitants, is roughly 13 km south of Erbil city, which represents an industrial region (S1). There are several industries and industrial activities in Tymar village. Haji Wsu village, on the other hand, positioned at coordinates 36°09'10.58"N 44°19'04.19"E, with 300 inhabitants, is located about 42 km east of Erbil city and represents non-industrial regions (S2), which is free of any pollution sources such as industries and industrial facilities, as illustrated in Fig. 1.

Collection of samples and measurements

The first-morning spot urine sample was collected from each participant and placed in a sealed polyethene plastic bottle. To measure hydroxyproline, the participants followed a diet that excluded collagen for 24 hours before and during the sample collection. The collected urine samples were then frozen at -20 °C until they were ready for analysis. The research study received approval from the Human

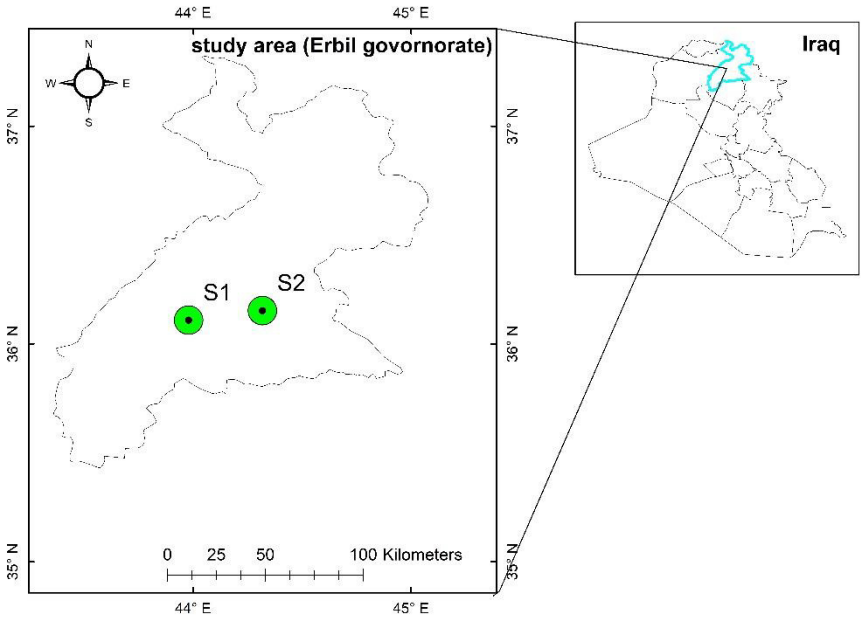

Fig. 1. Map of the study area.

Research Ethics Committee (HREC) at Salahaddin University, specifically the College of Science, Biology Department, ensuring its compliance with ethical standards.

Measurement of urinary hydroxyproline (Hyp)

Hydroxyproline is a modified amino acid containing a hydroxyl group on the pyrrolidine ring's fourth carbon. It is a non-proteinogenic amino acid produced by prolyl hydroxylase on pre-existing proteins, most often collagen components. Hydroxyproline was measured using Hydroxyproline Assay Kit Abbexa (abx298833, UK) and a microplate reader system. Hydroxyproline reacts with tosylchloramide and para-dimethylamino-benzaldehyde (DMAB) to produce an absorbance at 560 nm. The optical density is proportional to the concentration of hydroxyproline. The data were presented in µmol/L.

Measurement of urinary malonaldehyde (MDA)

The sample preparation protocol for determining MDA in urine was the following: 400 µl of urine was mixed with 250 µl of TBA (0.6%) and 750 µl of o-H_3PO_4 (1%). In a temperature-controlled heating block, the reaction mixture was heated to 90 ˚C for 30 minutes. Putting samples on ice stopped the reaction. MDA concentration was determined by the spectrometer at = 532 nm.[23]

Measurement of urinary creatinine

The measurement of urinary Creatinine was conducted utilizing the Creatinine Jaffé Gen.2 method on COBAS INTEGRA® 400 plus analyzer. The reagents employed for this analysis were acquired from Roche Diagnostics based in Germany, with the reference number REFF# 04810716190. Creatinine concentrations were expressed in mg/dL.

Measurement of urinary albumin

The measurement of urinary Creatinine was conducted utilizing the Tina Quant Albumin Gen.2 method on COBAS INTEGRA® 400 plus analyzer. The reagents employed for this analysis were acquired from Roche Diagnostics based in Germany, with the reference number REFF# 04469658190. Albumin concentrations were expressed in mg/L.

The measurement of urine Albumin-to- Creatinine Ratio (UACR)

UACR is a marker for Albuminuria, which is defined as abnormally high amounts of Albumin protein in the urine.[24] Clinically, UACR can be regarded as a quick and accurate indication of CKD, considering UACR is noninvasive and may be identified with a spot urine sample rather than a 24-hour urine sample, it has the advantage of having low sample needs.[25] Albumin-to- Creatinine ratio (ACR) was computed by

dividing the urine Albumin concentration by the urinary Creatinine concentration.[26] The reference range for (ACR) is categorized as follows: ACR levels below 30 mg/g are considered within the normal range, ACR levels ranging from 30 to 300 mg/g indicate the presence of microalbuminuria, and ACR levels exceeding 300 mg/g are indicative of clinical Albuminuria.[27]

Statistical analysis

To show the data, the mean and standard error of the mean (SEM) were used. Unpaired t-tests. $P \leq 0.05$ was used to evaluate statistical significance. The statistical analysis was carried out using the GraphPad Prism software 8.

Results and discussion

In this study, the levels of Hydroxyproline, Malondialdehyde, Creatinine, and Albumin were measured at two different sites: S1 (an industrial area) and S2 (a non-industrial area). The mean concentration of Hydroxyproline was found to be higher at S1, with a standard error of 66.02 ± 0.623 for adults, 65.57 ± 1.417 for children. In contrast, the mean concentration at S2 was lower, with a standard error of 64.00 ± 0.462 for adults, 61.60 ± 0.832 for children. The statistical analysis revealed a significant difference in hydroxyproline concentrations between S1 and S2 as shown in Fig. 2 and Table 1.

In the study, it was observed that the high concentration of Malonaldehyde was at S1, with a standard error of 1.534 ± 0.069 in adults and 1.675 ± 0.146 in children. Conversely, the lower concentration was recorded at S2, with a standard error of 1.325 ± 0.072 in adults and 1.162 ± 0.112 in children. The statistical analysis showed a significant difference between malonaldehyde concentrations between S1 and S2 as represented in Fig. 3 and Table 2.

The mean levels of Creatinine and Albumin were higher at S1 compared to S2. For adults, the mean with the standard error was 123.0 ± 5.790 for Creatinine and 8.365 ± 0.322 for Albumin at S1, while at S2 it was 104.4 ± 3.543 for Creatinine and 7.705 ± 0.361 for Albumin. For children, the mean with standard error at S1 was 68.33 ± 2.340 for Creatinine and 7.318 ± 0.702 for Albumin, and at S2 it was 65.84 ± 1.817 for Creatinine and 6.455 ± 0.486 for Albumin. However, statistical analysis revealed no significant differences in Creatinine and Albumin concentrations between S1 and S2. The (ACR) values were 10.093 for adults and 11.061 for children at S1, while at S2 they were 8.8709 for adults and 9.882 for children as shown in Figs. 4 and 5 and Table 3.

Discussion

The mean concentration of Hydroxyproline was found to be higher at S1, with a standard error of 66.02 ± 0.623 for adults, 65.57 ± 1.417 for children. In contrast, the mean concentration at S2 was lower, with a standard error of 64.00 ± 0.462 for adults, 61.60 ± 0.832 for children. The statistical analysis revealed a significant difference in hydroxyproline

Table 1. Presents the Mean ± S.E. values Hydroxyproline for adults and children at Site 1 and 2.

	Site 1	Site 2		Site 1	Site 2	
Variable	Adult (n = 42) Mean ± SE	Adult (n = 23) Mean ± SE	*P*-value	Child (n = 14) Mean ± SE	Child (n = 11) Mean ± SE	*P*-value
Hyp (µmol/L)	66.02 ± 0.623	64.00 ± 0.462	0.0113	65.57 ± 1.417	61.60 ± 0.832	0.0253

Hyp: Hydroxyproline. S1: Industrial area. S2: Non-industrial area. Statistical significance level $p < 0.05$

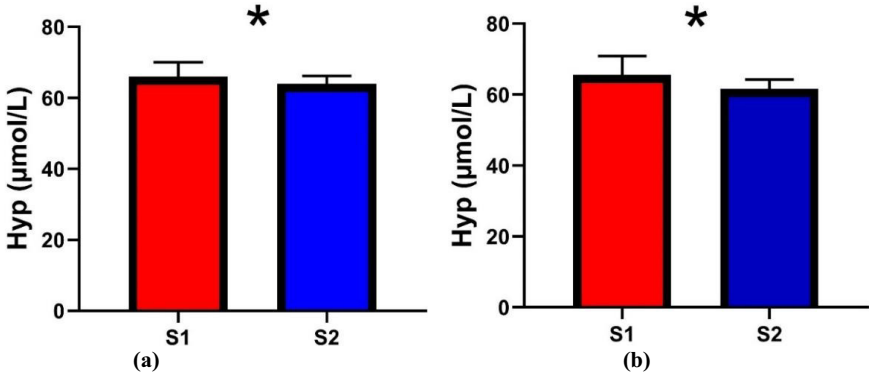

Fig. 2. The levels of Hydroxyproline in two groups: (a) Adults and (b) Children.

Table 2. Presents the Mean ± S.E. values malonaldehyde for adults and children at Site 1 and 2.

Variable	Site 1 Adult (n = 42) Mean ± SE	Site 2 Adult (n = 23) Mean ± SE	*P*-value	Site 1 Child (n = 14) Mean ± SE	Site 2 Child (n = 11) Mean ± SE	*P*-value
MDA (µmol/L)	1.534 ± 0.069	1.325 ± 0.072	0.0427	1.675 ± 0.146	1.162 ± 0.112	0.0117

MDA: malonaldehyde. S1: Industrial area. S2: Non-industrial area. Statistical significance level $p < 0.05$

Table 3. Presents the mean ± S.E. values creatinine and albumin for adults and children at Site 1 and 2.

Variable	Site 1 Adult (n = 42) Mean ± SE	Site 2 Adult (n = 23) Mean ± SE	*P*-value	Site 1 Child (n = 14) Mean ± SE	Site 2 Child (n = 11) Mean ± SE	*P*-value
Creatinine (mg/dL)	123.0 ± 5.790	104.4 ± 3.543	0.136	68.33 ± 2.340	65.84 ± 1.817	0.5748
Albumin (mg/L)	8.365 ± 0.322	7.705 ± 0.361	0.1787	7.318 ± 0.702	6.455 ± 0.486	0.3235
ACR (mg/g)	10.093	8.870		11.061	9.882	

S1: Industrial area. S2: Non-industrial area. Statistical significance level $p < 0.05$

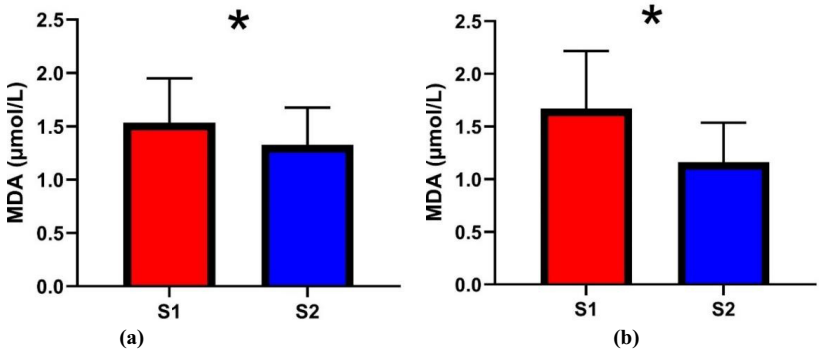

Fig. 3. The levels of Malonaldehyde in two groups: (a) Adults and (b) Children.

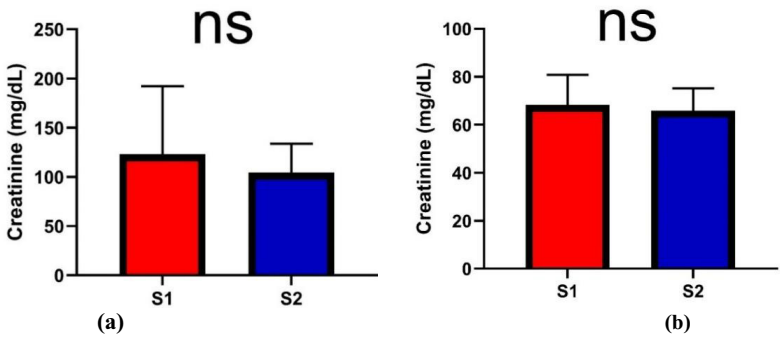

Fig. 4. The levels of Creatinine in two groups: (a) Adults and (b) Children.

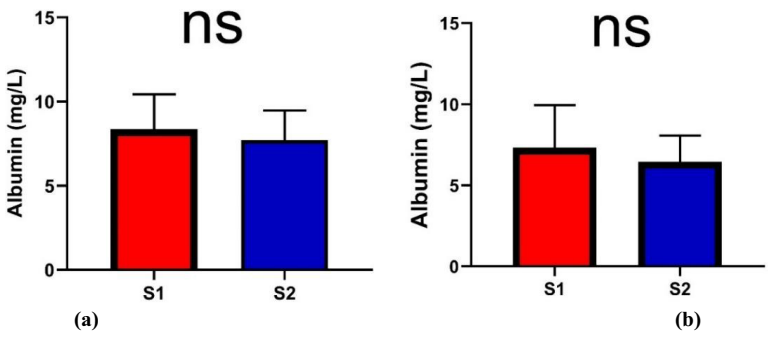

Fig. 5. The levels of Albumin in two groups: (a) Adults and (b) Children.

concentrations between S1 and S2. The mean concentration of hydroxyproline was found to be higher and significantly different at S1 (an industrial area) for adults and children compared to S2 (a non-industrial area). The hypothesis is that urinary hydroxyproline, which is secreted in the urine as a result of lung collagen catabolism in response to NO_2 damage, may be employed as a biochemical measure of the health impacts of NO_2 exposure.[21] Perdelli *et al.*,[21] did a study to assess urine hydroxyproline as a biomarker of impact after nitrogen dioxide exposure. A cross-sectional study was undertaken on two groups of participants who were exposed to nitrogen dioxide in different ways. The results revealed that participants practicing in nitrogen dioxide-polluted locations had greater levels of hydroxyproline than those training in non-polluted areas.

Collagen hydroxyproline breakdown products may serve as biological indicators of the early effects of nitrogen dioxide exposure.[28] Azari *et al.*,[28] conducted a study on the potential biomarkers of exposure and consequences for glass artisans and braziers exposed to nitrogen oxides revealed. When compared to controls, all of these exposed workers' hydroxyproline levels were higher. According to some epidemiological research, increased hydroxyproline excretion in the urine is linked to NO_2 exposure.[29–31]

In the study, it was observed that the high concentration of Malonaldehyde was at S1, with a standard error of 1.534 ± 0.069 in adults and 1.675 ± 0.146 in children. Conversely, the lower concentration was recorded at S2, with a standard error of 1.325 ± 0.072 in adults and 1.162 ± 0.112 in children. The statistical analysis showed a significant difference between malonaldehyde concentrations between S1 and S2. Malonaldehyde was found to have a high concentration and a significant difference at S1 in adults and children when compared to S2. MDA in the urine is one of the byproducts of the breakdown of several primary and secondary lipid peroxidation products. Typically, they are used to describe the prevalence of lipid or oxidative DNA damage in populations.[32] Air pollution can directly cause the oxidizing effects of ROS in the body or trigger additional ROS-producing events in the lungs. In macrophages, neutrophils, endothelial cells and epithelial cells, and, these prooxidants that are present in the ambient air trigger oxidases and myeloperoxidases (MPOs). The internal environment may become more oxidative due to ROS generated from these internal sources that may damage mitochondrial DNA and disrupt the electron transport chain.[33] One of these crucial pathways is oxidative stress responses to ROS and consequent inflammation caused by air pollution exposure.[34] Outdoor air pollution varies in time and area, and the number of hazardous substances as well as their physical-chemical properties may impact xenobiotic absorption by breathing.[35] The biotransformation pathways may be responsible for the harmful effects seen following exposure to airborne xenobiotics. These may encourage the production of reactive metabolites and ROS and RNS, which might then cause cellular damage, ongoing inflammation, cancer, autoimmune disorders, and other disease processes in the body.[36] The respiratory system is most likely to be readily accessible by fine airborne particulates and gases, which can produce reactive oxygen species, and consequently cause macromolecule damage. Exposure to these particles and gases can also activate inflammatory mediators that can exacerbate lung inflammation, induce an increase in blood coagulability, and cause endothelial dysfunction.[37] Occupational exposure to air pollution causes oxidative damage due to the lack of available antioxidant defenses.[38]

Air pollution and the inflammatory process might be the reason for vasoconstriction and endothelial dysfunction, resulting in autonomic nervous system instability.[39] Particulate matters (PM)-mediated oxidative stress can result from direct generation of ROS from the surface of soluble compounds; altered mitochondrial function or decreased nicotinamide adenine dinucleotide phosphate (NADPH)-oxidase; and activation of inflammatory cells capable of producing ROS and RNS, as well as oxidative DNA damage.[40] Furthermore, physiologically catalyzed oxidation events that take place in the cell membrane and mitochondria of target cells, such as airway epithelial cells and macrophages, produce ROS in response to particle absorption.[41] Ambient particulate matter can cause oxidative stress and pro-inflammatory processes in the vascular and central nervous systems.[42] Furthermore, ambient particulate matter can be directly transferred into the central nervous system via the olfactory epithelium and damage the brain, or it can penetrate deeply into the pulmonary system, irritate and erode the alveolar membrane, and therefore compromise lung function.[43] $PM_{2.5}$ can cause an increase in oxidative stress by causing an increase in the generation of ROS.[44]

MDA is produced in vivo by the peroxidation of polyunsaturated fatty acids, which occurs as a result of the ROS produced as a result of oxidative stress.[45] Inhalation of particulate matter triggered oxidative stress by translocating from the lungs into the vascular tree, resulting in the production of pro-inflammatory mediators and the activation of phosphatidylinositol 3-kinase pathways.[46] Li *et al.*,[47]

investigated the relationship between oxidative stress indicators and brief exposure to ambient particle air pollution. Short-term exposure to ambient PM2.5 was linked to a considerably higher level of MDA, according to a meta-analysis, suggesting that increased oxidative stress may be a result of ambient particle air pollution. Lin *et al.*,[48] performed a study on the relationship between modifications to pollution in the air and biomarkers of oxidative stress in children before and during the Beijing Olympics, and the findings show that exposure to black carbon causes systemic oxidative stress in children. According to research by Gokirmak *et al.*,[49] occupational exposure to high quantities of Sulphur dioxide increases oxidative stress and lipid peroxidation. This study focused on the function of oxidative stress in bronchoconstriction caused by SO_2 exposure.

The mean levels of Creatinine and Albumin were higher at S1 compared to S2. For adults, the mean with the standard error was 123.0 ± 5.790 for Creatinine and 8.365 ± 0.322 for Albumin at S1, while at S2 it was 104.4 ± 3.543 for Creatinine and 7.705 ± 0.361 for Albumin. For children, the mean with standard error at S1 was 68.33 ± 2.340 for Creatinine and 7.318 ± 0.702 for Albumin, and at S2 it was 65.84 ± 1.817 for Creatinine and 6.455 ± 0.486 for Albumin. However, statistical analysis revealed no significant differences in Creatinine and Albumin concentrations between S1 and S2. The mean levels of Creatinine and Albumin were higher at S1 compared to S2 and statistical analysis showed no significant differences in Creatinine and Albumin concentrations between S1 and S2. Conducted research in Mianyang City, Southwest China, to evaluate the link between the combined toxicity of air pollutants and renal function in adult women, the results revealed that air pollutants were positively connected to Creatinine. Blum *et al.*,[50] did a study to know if there was a link between long-term PM2.5 exposure and renal disease, such as eGFR, Albuminuria, and acute CKD. The findings of the study indicated a positive correlation between higher yearly average PM2.5 concentrations and increased levels of Albuminuria. Okoye *et al.*,[51] conducted a study to determine the relationship between long-term air pollution and renal outcomes in areas near oil and natural gas (ONG). The findings revealed that residents of exposed communities are more likely to develop chronic kidney disease and had higher blood Creatinine levels than residents of less exposed places. Blum *et al.*,[50] researched to explain the connection between chronic exposure to PM2.5 and kidney disease, including eGFR, Albuminuria levels, and acute CKD. and their findings revealed that in a community-based population, exposure to greater annual average PM2.5 concentrations was linked to a higher degree of Albuminuria and a higher risk for developing incident CKD. Chin *et al.*,[52] researched the effects of air pollution on Albuminuria in type 2 diabetic patients, and the results revealed that high CO and PM2.5 levels enhanced Albuminuria in type 2 diabetic patients.

The (ACR) values were 10.093 for adults and 11.061 for children at S1, while at S2 they were 8.8709 for adults and 9.882 for children. The (ACR) values for adults and children are under the normal range (ACR<30 mg/g) at both sites. O'Neill *et al.*,[53] conducted a study on urine Albumin and Creatinine levels in members of the Multi-Ethnic research of Atherosclerosis throughout three visits from 2000 to 2004. PM2.5 and PM10 exposure were studied, and the findings indicated that chronic and recent particle exposures were not associated with current UACR or microalbuminuria. Li *et al.*,[54] did a study in Beijing on the relationship of metals and metalloids with urinary Albumin/Creatinine ratio, and the results showed that urinary Cu content was strongly positively related to UACR.

Conclusion

Based on the study conducted in the two study sites in Erbil Governorate, some urinary biomarkers were evaluated as indicators of proposed air pollution specifically at Site 1 (an industrial area) and Site 2 (a non-industrial area). The concentrations of Hydroxyproline and Malondialdehyde, which are biomarkers associated with oxidative stress and inflammation, were found to be significantly higher in both adults and children at Site 1 compared to Site 2. This suggests that these biomarkers are more sensitive to proposed air pollution exposure in industrial areas. Nevertheless, for both adults and children, the creatinine and albumin concentrations, biomarkers linked to kidney function, were higher at Site 1 but not substantially different from Site 2. This suggests that although not statistically significant, air pollution may have some impact on these biomarkers. We also measured the urine albumin-to-creatinine ratio (UACR) in order to look into the effects of air pollution. For every participant, the UACR values at both locations were within the normal range. According to the study, oxidative stress and inflammation biomarkers (hydroxyproline and malondialdehyde) are significantly impacted by exposure to proposed air pollution, especially in industrial areas. These results highlight the significance of air pollution monitoring and the need for additional studies to fully comprehend the long-term impacts of air pollution on human health.

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Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images, that are not ours, have been included with the necessary permission for republication, which is attached to the manuscript.
- No animal studies are present in the manuscript.
- Authors sign on ethical consideration's approval.
- Ethical Clearance: The project was approved by the local ethical committee at Salahaddin University.

Authors' contribution statement

J. K. M. Conceptualization, Methodology, Software, Visualization, Investigation. Y. A. S. Supervision, Data Validation, Reviewing and Editing.

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دراسة دور بعض المؤشرات الحيوية في تقييم آثار تلوث الهواء المقترحة في مناطق مختارة من محافظة أربيل

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الخلاصة

يهدف الدراسة الحالية الى تقييم المستويات هيدروكسي برولين البولي ، ومالونديالديهايد البولي ، والكرياتينين البولي والألبومين البولي كمؤشرات حيوية محتملة لتلوث الهواء في موقعين متميزين: الموقع 1 المنطقة الصناعية والموقع 2 المنطقة الغير الصناعية ، أيضًا تم قياس نسبة الألبومين إلى الكرياتينين (UACR) في البول وذلك لمزيد من التحري في تأثيرات تلوث الهواء. تم استخدام تصميم مقطعي في هذه الدراسة بمجموع تسعين مشاركًا. في الموقع 1 كان هناك 56 مشاركًا يشمل 42 بالغًا و 14 طفلاً ، والتي بلغت حوالي 11.2% من إجمالي السكان. وفي الوقت نفسه في الموقع 2 كان هناك 34 مشاركًا يشمل 23 بالغًا و 11 طفلاً، يمثلون حوالي 11.3% من السكان. أشارت نتائج هذه الدراسة إلى أن تراكيز الهيدروكسي برولين و المالونديالدهيد كانت أعلى بشكل ملحوظ في الموقع 1 لكل من البالغين والأطفال مقارنة بالموقع 2. وهذا يشير إلى أن هذه المؤشرات الحيوية أكثر حساسية للتلوث الهواء في المناطق الصناعية. ومن جهة أخرى وجد أن تراكيز الكرياتينين والألبومين أعلى في الموقع 1 مقارنتا بالموقع 2 لكل من البالغين والأطفال على الرغم من عدم وجود فروقات الاحصائية. تم قياس قيم UACR في الموقع 1 والموقع 2 بالنسبة للبالغين ، كانت قيم UACR 10.093 و 8.870 mg/gعلى التوالي ، بينما للأطفال كانت 11.061 و 9.882 mg/g على التوالي. كانت جميع القيم ضمن المدى الطبيعي ، مما يشير إلى أن تلوث الهواء لم يؤثر بشكل كبير على وظائف الكلى في السكان المدروسة. يمكن أن تشير مستويات الهيدروكسي برولين المرتفعة إلى تغيرات في أيض الكولاجين، والتي يمكن ربطها بتلف الأنسجة الناجم عن تلوث الهواء. تشير زيادة مستويات المالونديالدهيد إلى زيادة الإجهاد التأكسدي وتدهور المكونات الخلوية، مما يعني تأثير تلوث الهواء على تراكيب الدهنية. يعد ارتفاع مستويات الألبومين في البول علامة حساسة لإصابة الكلى أو خلل وظيفي ناتج عن التعرض لتلوث الهواء، دلالة على الآثار السلبية على وظائف الكلى.

الكلمات المفتاحية: الزلال، الدلائل الحيوية، الكرياتينين، هيدروكسي برولين، مالونديالدهيد.