Occupational Exposure to Cadmium Increases Inflammation, Leading to Cardiovascular Disease and Reducing Selenium Levels

Nagham J. Kadam[1,2]* , Nadhum A.N. Awad[1], Saad S. Hamadie[3]

1. Department of Chemistry, College of Sciences, University of Basra, IRAQ
2.Iraqi Ministry of Education, The Basrah Education Directorate, IRAQ
3. Internal Medicine Branch, College of Medicine, University of Basra, IRAQ

*Corresponding author E-mail: *pgs.nagham.jwad@uobasrah.edu.iq

https://doi.org/10.29072/basjs.20240220

ARTICLE INFO

Keywords

Cadmium; Occupational health; Selenium, high-sensitivity C-reactive protein; cardiovascular health; Inflammation

ABSTRACT

This study offers significant insights into the potential risks associated with oil well workers' exposure to cadmium and heart disease. The study comprised two groups of men. Ninety workers in the first group were exposed to crude oil well sites in Basra, whereas ninety persons in the control group were unexposed. The investigation involved measuring cadmium concentrations, high-sensitivity C-reactive protein, and selenium levels in the blood of both groups. High-sensitivity C-reactive protein (hs-CRP) was additionally quantified. Levels of cadmium and selenium were evaluated. The main finding indicated that blood cadmium concentrations were markedly elevated in oil well workers compared to controls ($P < 0.0001$). The study identified a strong association ($P < 0.001$) between elevated levels of high-sensitivity C-reactive protein (hs-CRP) in workers and cadmium exposure. High-sensitivity C-reactive protein (hs-CRP) serves as a crucial biomarker for inflammation, with elevated levels signifying an augmented risk of cardiovascular disease. The blood tests of the workers indicated a shortage in selenium ($P < 0.0001$), underscoring the antagonistic interaction between cadmium and selenium. The study concludes that cadmium exposure may elevate the risk of cardiovascular disease by enhancing inflammation and compromising the body's antioxidant defenses.

Received 20 Nov 2024; Received in revised form 08 Dec 2024; Accepted 21 Dec 2024, Published 31 Dec 2024

1. Introduction

Basrah suffers greatly from pollution due to the expansion of the manufacture and production of oil, which has broad negative effects on human health and the environment [1,2]. The exposure of the work environment to petroleum products that contain heavy minerals and organic compounds can significantly weaken the functions of the various physiological systems of workers in places near the production of petroleum [3,4]. Elements classified as heavy metals have atomic masses of more than 20 and densities greater than 5 g/cm^3 [5]. They are toxic in small amounts and are harmful because they accumulate over time [6,7]. One divalent cation linked to cardiovascular disease is cadmium. Even at low doses, it can disrupt various biological systems and has a half-life of 10 to 30 years [8,9]. Through oxidative processes and elevated levels of reactive oxygen species in mitochondria, cadmium can damage vascular tissue, cause vascular dysfunction, and promote atherosclerosis. It can also participate in Fenton reactions by indirectly displacing endogenous Fenton metals (such as Fe^{2+}) [10]. Because cadmium is mostly maintained in smooth muscle cells after being carried across endothelial cells, it can also damage endothelial cell integrity and result in cell death. This can lead to lipid buildup and a shift toward atherosclerotic [11]. Cadmium absorption in the gut is increased in iron, calcium, or zinc shortages because of their chemical similarities in valence +2 [12]. Cadmium displaces important biominerals, changing their equilibrium and disrupting biological functions [9]. For instance, zinc and cadmium compete with one another for binding sites in metal-absorbing proteins and antioxidant enzymes, decreasing their capacity to scavenge free radicals [13]. Cadmium can also target the thiol (-SH) groups of cysteines found in proteins. The inactivation of sulfhydryl enzyme groups leads to many functional deficits in the nucleus, endoplasmic reticulum, and mitochondria [14]. Prolonged exposure to Cadmium increases the opportunity to develop different cancers by damaging DNA or changing genetic expression through interactions with basic minerals[15,16]. Cardiovascular diseases (CVDs) are the leading cause of death globally, and they are considered a significant public health concern [17,18]. Exposure to environmental stressors such as air pollution, noise, and climate change raises the risk of noncommunicable diseases (NCDs) in addition to the conventional risk factors for cardiovascular disease, which include diabetes, high blood pressure, smoking, high cholesterol, and hereditary factors [19]. The liver produces C-reactive protein (CRP) in response to inflammation in the body and raises the level of this protein in the blood [20]. High-sensitivity C-reactive protein (hs-CRP) is a more sensitive type of CRP, used to detect lower levels of this protein. High levels of hs-CRP are associated with an increased risk of cardiovascular diseases such as heart attack and stroke [21–23] Along with low-density lipoprotein cholesterol (LDL), it is an actual risk factor for CVD [24]. The operation of the immune system and the control of inflammation depend on the basic minerals necessary to work properly for fungal and immunosuppressive systems. Maintaining a balance between supportive and anti-inflammatory organizations and defense systems is important. The deficit may disrupt the continuous regulation of inflammation or temporarily reduce immune efficiency [25]. Selenium (Se) is a trace element vital to the body in metabolic processes and protection from oxidative stress. Selenium deficiency can lead to cardiovascular diseases such as myocardial infarction, heart failure, coronary heart disease, and atherosclerosis. Proteins require selenium, which is also involved in calcium efflux, parathyroid hormone metabolism, and redox regulation [26–28]. It is essential for the glutathione peroxidase system and may reduce cancer risk by decreasing lipid peroxidation [29,30]. This study investigates the role of inflammation in the development of cardiovascular disease by assessing the predictive ability of inflammatory biomarkers, such as raised levels of high-sensitivity C-reactive protein brought on by occupational cadmium exposure. Another investigation examined the impact of cadmium levels on low selenium levels at oil well locations.

2. Materials and Method

2.1. Study Design

The study involved two groups of participants. The first group comprised ninety workers exposed to working conditions at crude oil well sites in Basrah. Each worker had at least two years of experience working near oil wells and worked eight hours a day, five days a week. The second group was not exposed to these conditions (control group).

2.2 Questionnaire

Participants' demographic information (age and gender), personal lifestyle (drinking and smoking), work history, exposure duration (number of years of work and hours of work per day), and health status (genetic disorders, drugs consumed, other blood disorders such as diabetes, kidney disease, and hypertension) were requested using a questionnaire. At 9:00 a.m., data collection began

2.3 Sample Collection

The study protocol was approved by the Research Ethics Committee of Basra Oil Company, after which all participants in the study provided written informed consent. Five milliliters of blood were drawn from each participant by venous infusion and transferred into two tubes. Blood samples were obtained and stored in a humidified ice box until measured. This was on the same day that the samples were measured. Each laboratory task was completed in a designated analytical laboratory. Cadmium and selenium were measured in two milliliters of venous blood in a tube filled with EDTA (diethylenediamine tetraacetate).

2.4 Determination of Cadmium and Selenium

Cadmium and selenium levels were determined using inductively coupled plasma optical emission spectroscopy (ICP-OES- HORIBA Scientifical, JY2000-2, France). To evaluate the levels of cadmium and selenium at wavelengths of 226.502 and 196.025 nm, respectively. The amounts of cadmium and selenium were multiplied by a dilution factor, and the standard curve was extrapolated.

2.5 Biomarker analysis of high-sensitivity C-reactive protein (hs-CRP)

Wondfo Finecare Plus (models FS-112, FS-113, FS-205) in China was used to test serum hs-CRP levels according to the manufacturer's instructions. A fluorescence immunoassay was used to measure serum hs-CRP levels in the laboratory. High-sensitivity C-reactive protein (hs-CRP) testing is a robust and reliable method for predicting the risk of atherosclerosis and cardiovascular disease (CVD) and identifying infections and inflammations.

2.6 Statistical analysis

Data analysis was carried out using the available statistical package of IBM SPSS-22 (IBM Statistical Packages for Social Sciences, version 22, Chicago, IL, USA). Current study data were analyzed using the Chi-square test or Fisher exact probability (F.E.P.) test to compare percentages (qualitative data). The T-test was used to compare two numeric variables. Pearson Correlation: to examine the degree of relation between variables. The correlation coefficient value (r) is either positive (direct correlation) or negative (inverse correlation). The sensitivity and specificity of the parameters studied were evaluated using a receiver operating characteristic (ROC) curve. The comparison of significance (p-value) in any test was considered as follows:

- P-value greater than 0.05 ($P>0.05$) was non-statistically significant (NS).
- P-value of less than 0.05 ($P<0.05$) was statistically significant (S).
- P-value of less than 0.01 ($P<0.01$) was highly statistically significant (HS).

3. Results and discussion

3.1 Study demographics

Table 1 contains the demographic information of the exposed workers who worked at crude oil well sites in Basrah City and the non-exposed workers (control group). The workers' ages ranged from 23 to 55 years; the mean value was 41.44 ± 1.07. The second group included 90 unexposed individuals (students and faculty members). Their ages ranged from 21 to 54 years; the mean value was 41.93 ± 1.05.

Table 1: Demographics of the study

Demographics	Exposed workers	Nonexposed control
Age	41.44 ± 1.07 (55–23)	41.93 ± 1.05 (54–21)
Smoking	42 (46.6 %)	40 (44.4 %)
Number	90	90

3.2 Chronic Diseases

Table 2 shows the chronic diseases in this study's exposed workers and nonexposed control groups. The workers showed higher rates of diabetes, hypertension, allergies, and heart disease compared to the control group with a significant difference ($P<0.001$).

Table 2: Chronic diseases in both groups studied

Demographic Picture	Status	Exposed Workers n=90	Non-exposed Control n =90	
Chronic Diseases	None	22	85	Chi-square =31.7 *P*-value= 0.001
	DM	26	2	
	Hypertension	17	3	
	Heart disease	13	None	
	Allergy	12	None	

3.3 Comparison between exposed workers in oil sites and an unexposed control sample in cadmium concentration and high-sensitivity C-reactive protein (hs-CRP):

The results in Table 3 showed that the presence of Cadmium (ng/ml) in the blood of workers exposed to high concentrations was greater than that in the unexposed control group, with a mean value of (85.8 ± 0.83 vs. 43.7 ± 3.07), respectively. These differences were statistically highly significant ($P \leq 0.0001$), and Selenium (ng/ml) (1462.27 ± 20.00 vs. 1650.57 ± 19.25) and ($P \leq 0.0001$). The results also documented the presence of hs-CRP in workers' blood at higher concentrations compared to unexposed control (2.14 ± 0.26 vs. 0.49 ± 0.07), respectively. ($P \leq 0.001$), however, the data revealed a significant difference in the mean hs-CRP value between the two groups.

Table 3: Comparative the mean values of Cd(ng/ml), Se(ng/ml), and hs-CRP (mg/ml) between Exposed workers n=90 and Nonexposed control (n=90)

Group	Cd(*ng/ml*)	Se(*ng/ml*)	hs-CRP (*mg/L*)
Exposed workers	0.83 ± 85.8	462.2± 20.00	2.14± 0.26
Nonexposed control	3.07 43.7±	9.25 1650.5±	0.49± 0.07
P-value	100.00 ≤	0.0001 ≤	≤0.001

***P* is significant statistics at level < 0.05.**
Data are reported as mean ± standard deviation

3.4 Correlation Assessment between hs-CRP and (Cd & Se):

The following metrics showed a substantial association with one another, according to our evaluation of the interdependence of the laboratory values: Table 4 illustrates the significant inverse link between hs-CRP and Se (r = - 0.193**, P = 0.01) and the positive correlation between hs-CRP and Cd hazardous ions (r = 0.04, P = 0.56).

Table 4: Correlation Assessment between hs-CRP and (Cd& Se)

		Cd(ng/ml)	Se(ng/ml)
hs-CRP (*mg/L*)	Pearson Correlation	0.33**	-0.193**
	P-value	0.000	0.01

****Correlation is significant at the 0.01 level (2-tailed).**
***Correlation is significant at the 0.05 level (2-tailed).**

3.4. Correlation Assessment between Se and Cd:

The most important relationship to focus on is between Se and Cd. Data analysis revealed a strong inverse relationship between Se and Cd (r = - 0.417**, *P* = 0.00), as shown in Table 5.

Table 5: Correlation Assessment between Se and Cd

		Cd(*ng/ml*)
Se(*ng/ml*)	Pearson Correlation	-0.417**
	P-value	0.000

4. Discussion

The current research included a clinical study to determine the levels of cadmium emitted from crude oil wells in the blood of oil workers. It also aimed to investigate the relationship between high levels of high-sensitivity C-reactive protein (hs-CRP) and low levels of selenium and their potential effects on cardiovascular diseases. The clinical results showed that the blood levels of cadmium in oil workers were higher than those in the control group. Moreover, the results revealed lower levels of selenium and increased levels of high-sensitivity C-reactive protein, indicating the presence of oxidative stress and endogenous oxidative imbalances. Oxidative stress is a cellular response resulting from an imbalance in the redox system, which activates pathways important for cell survival. The imbalance stimulates pathways that determine cell survival. It increases the risk of cancer, neurological conditions, diabetes, infertility, developmental disorders, kidney failure, and cardiovascular diseases [31]. Heavy metal toxicity works through many similar mechanisms, such as ROS production, impaired antioxidant defense, enzyme inactivation, and multiple organ toxicity. Heavy metals are known to inhibit the enzyme glutathione peroxidase. However, some metals have a specific mechanism that enables them to bind to a particular protein. Heavy metals mostly affect thyroid and steroid hormones in humans, which can disrupt the endocrine system. Cadmium is known to cause thyroid toxicity. Cadmium exposure induces inflammation of the central nervous system, leading to neurotoxicity due to its effect on proinflammatory cytokines. Cadmium also has a toxic impact on the liver and blood, damaging the kidneys and respiratory system [14]. Heavy metals can interfere with cellular processes such as growth, proliferation, differentiation, damage healing, and apoptosis. High-sensitivity C-reactive protein (CRP) is associated with the formation of atherosclerotic plaques. Together with low-density lipoprotein (LDL) cholesterol, it is a major risk factor for cardiovascular disease [23,24,32].

Lifestyle factors, including diet, smoking, lack of physical activity, and sleep deprivation, significantly influence the development of atherosclerosis [33]. Environmental factors such as high temperatures also affect the concentration of pollutants in oil work sites in southern Iraq [1]. To prevent occupational hazards in the oil industry, it is essential to implement safety protocols and training programs for workers. Strategies like hazard assessment, emergency plans, personal protective equipment provision, ongoing training, routine equipment maintenance, accident investigations, adequate ventilation, and routine medical examinations should be implemented. These measures aim to reduce exposure to harmful substances and improve worker health and safety. Comprehensive accident investigations are also necessary to identify causes and take corrective measures. This shared responsibility between workers and company management ensures a safer working environment.

5. Conclusions

The study suggests that cadmium exposure may increase cardiovascular disease risk by promoting inflammation and disrupting the body's antioxidant defenses. This highlights the importance of environmental factors in cardiovascular health.

The main findings were:

- Occupational exposure to cadmium (Cd) and cardiovascular disease risk.
- Elevated levels of high-sensitivity C-reactive protein: The study found a direct relationship between increased levels of high-sensitivity C-reactive protein (a marker of inflammation) and cadmium exposure. This suggests that cadmium exposure may contribute to the development of cardiovascular disease by promoting inflammation.
- Inflammation and cardiovascular disease: Chronic inflammation is a well-established risk factor for cardiovascular disease, including atherosclerosis, heart attack, and stroke.
- Antagonism between cadmium and selenium.

Antioxidant defense: Selenium (Se) is crucial in the body's antioxidant defense system. The study highlights the antagonistic interaction between cadmium and selenium, suggesting that cadmium may interfere with the body's ability to protect itself from oxidative stress.

- Oxidative stress and cardiovascular disease: Studies have linked the development of cardiovascular disease to oxidative stress, an imbalance between free radicals and antioxidants.

The study calls for more research to understand the complex mechanisms by which exposure to petroleum emissions (and other environmental pollutants) can lead to inflammation, oxidative stress, and cardiovascular disease.

Acknowledgments

The authors thank Basra Oil Company for agreeing to take blood samples from workers at oil sites. I would also like to thank the Nahran Bin Omar oil field workers north of Basra for their cooperation in donating blood samples and providing information. The authors would also like to thank the Department of Chemistry, College of Science, and the University of Basra in Iraq for their support. The corresponding author would also like to thank the Iraqi Ministry of Education and Basra Education Directorate for their support.

References

[1] N.A. Salman, M.K. Al-Mishrey, H.T. Al-Saad, A. Rushdi, Air Pollution in the Southern Part of Iraq and Its Health Risks, in: Aerosol Opt. Depth Precip. Meas. Part. Conc. Heal. Risks Environ. Impacts, Springer, 2024,107–122 DOI:10.1007/978-3-031-55836-8_6

[2] A.S. Alshaheen, I.M. Al-Naiema, D.M. Tuama, W.H. Al-Mosuwi, Characterization, risk assessment, and source estimation of PM10-bound polycyclic aromatic hydrocarbons during wintertime in the ambient air of Basrah City, Iraq, Chemosphere 326 (2023) 138444, https://doi.org/10.1016/j.chemosphere.2023.138444

[3] A. Sajid Jabbar, E.T. Ali, Impact of Petroleum Exposure on Some Hematological Indices, Interleukin-6, and Inflammatory Markers of Workers at Petroleum Stations in Basra City, J. Environ. Public Health 2020 (2020) 7693891, DOI: 10.1155/2020/7693891

[4] M. Priyadarshanee, U. Mahto, S. Das, Mechanism of toxicity and adverse health effects of environmental pollutants, in: Microb. Biodegrad. Bioremediation, Elsevier, 2022: pp. 33–53, https://doi.org/10.1016/B978-0-323-85455-9.00024-2

[5] E.L. Ungureanu, G. Mustatea, Toxicity of heavy metals, in: Environ. Impact Remediat. Heavy Met., IntechOpen, 2022, DOI: 10.5772/intechopen.102441

[6] R.N. Khalef, A.I. Hassan, H.M. Saleh, Heavy metal's environmental impact, in: Environ. Impact Remediat. Heavy Met., IntechOpen, 2022, DOI:10.5772/intechopen.97895

[7] Kiran, R. Bharti, R. Sharma, Effect of heavy metals: An overview, in: Mater. Today Proc., Elsevier, 2022, 880–885, https://doi.org/10.1016/j.matpr.2021.06.278

[8] D. Diaz, F. Ujueta, G. Mansur, G.A. Lamas, A. Navas-Acien, I.A. Arenas, Low-level cadmium exposure and atherosclerosis, Curr. Environ. Heal. Reports, 8 (2021) 42–53. https://doi.org/10.1007/s40572-021-00304-w

[9] G. Genchi, M.S. Sinicropi, G. Lauria, A. Carocci, A. Catalano, The effects of cadmium toxicity, Int. J. Environ. Res. Public Health 17 (2020) 3782, doi: 10.3390/ijerph17113782

[10] M. Peana, A. Pelucelli, C.T. Chasapis, S.P. Perlepes, V. Bekiari, S. Medici, M.A. Zoroddu, Biological effects of human exposure to environmental cadmium, Biomolecules 13 (2022) 36, https://doi.org/10.3390/biom13010036

[11] H.-C. Lin, W.-M. Hao, P.-H. Chu, Cadmium and cardiovascular disease: An overview of pathophysiology, epidemiology, therapy, and predictive value, Rev. Port. Cardiol. (English Ed. 40 (2021) 611–617, https://doi.org/10.1016/j.repce.2021.07.031

[12] M.M. Brzóska, J. Moniuszko-Jakoniuk, Interactions between cadmium and zinc in the organism, Food Chem. Toxicol. 39 (2001) 967–980. https://doi.org/10.1016/S0278-6915(01)00048-5

[13] A. Cirovic, A. Cirovic, S. Yimthiang, D.A. Vesey, S. Satarug, Modulation of Adverse Health Effects of Environmental Cadmium Exposure by Zinc and Its Transporters, Biomolecules 14, (2024) 650, https://doi.org/10.3390/biom14060650

[14] M. Balali-Mood, K. Naseri, Z. Tahergorabi, M.R. Khazdair, M. Sadeghi, Toxic mechanisms of five heavy metals: mercury, lead, chromium, cadmium, and arsenic, Front. Pharmacol., 12 (2021) 643972, DOI: 10.3389/fphar.2021.643972

[15] A. Cirovic, S. Satarug, Toxicity tolerance in the carcinogenesis of environmental cadmium, Int. J. Mol. Sci. 25 (2024) 1851, https://doi.org/10.3390/ijms25031851

[16] F. Qu, W. Zheng, Cadmium Exposure: Mechanisms and Pathways of Toxicity and Implications for Human Health, Toxics, 12 (2024) 388, https://doi.org/10.3390/toxics12060388

[17] O. Gaidai, Y. Cao, S. Loginov, Global cardiovascular diseases death rate prediction, Curr. Probl. Cardiol., 48 (2023) 101622, https://doi.org/10.1016/j.cpcardiol.2023.101622

[18] G.A. Roth, G.A. Mensah, C.O. Johnson, G. Addolorato, E. Ammirati, L.M. Baddour, N.C. Barengo, A.Z. Beaton, E.J. Benjamin, C.P. Benziger, Global burden of cardiovascular diseases and risk factors, 1990–2019: update from the GBD 2019 study, J. Am. Coll. Cardiol., 76 (2020) 2982–3021, doi: 10.1016/j.jacc.2020.11.010

[19] T. Münzel, O. Hahad, M. Sørensen, J. Lelieveld, G.D. Duerr, M. Nieuwenhuijsen, A. Daiber, Environmental risk factors and cardiovascular diseases: a comprehensive expert review, Cardiovasc. Res., 118 (2022) 2880–2902, DOI: 10.1093/cvr/cvab316

[20] I.M. Rajab, P.C. Hart, L.A. Potempa, How C-reactive protein structural isoforms with distinctive bioactivities affect disease progression, Front. Immunol., 11 (2020) 2126. DOI: 10.3389/fimmu.2020.02126

[21] S.A. Rizo-Téllez, M. Sekheri, J.G. Filep, C-reactive protein: a target for therapy to reduce inflammation, Front. Immunol., 14 (2023) 1237729, https://doi.org/10.3389/fimmu.2023.1237729

[22] T. Banait, A. Wanjari, V. Danade, S. Banait, J. Jain, Role of high-sensitivity C-reactive protein (Hs-CRP) in non-communicable diseases: a review, Cureus, 14 (2022). doi: 10.7759/cureus.30225

[23] A. Denegri, G. Boriani, High sensitivity C-reactive protein (hsCRP) and its implications in cardiovascular outcomes, Curr. Pharm. Des., 27 (2021) 263–275. DOI: 10.2174/1381612826666200717090334

[24] S.E.C. Bafei, X. Zhao, C. Chen, J. Sun, Q. Zhuang, X. Lu, Y. Chen, X. Gu, F. Liu, J. Mu, Interactive effect of increased high sensitive C-reactive protein and dyslipidemia on cardiovascular diseases: a 12-year prospective cohort study, Lipids Health Dis., 22 (2023) 95, DOI: 10.1186/s12944-023-01836-w

[25] C. Weyh, K. Krüger, P. Peeling, L. Castell, The role of minerals in the optimal functioning of the immune system, Nutrients, 14 (2022) 644, doi: 10.3390/nu14030644

[26] B.K. Shimada, N. Alfulaij, L.A. Seale, The impact of selenium deficiency on cardiovascular function, Int. J. Mol. Sci. 22 (2021)10713, doi: 10.3390/ijms221910713

[27] C.M. Munguti, M. Al Rifai, W. Shaheen, A rare cause of cardiomyopathy: A case of selenium deficiency causing severe cardiomyopathy that improved on supplementation, Cureus, 9 (2017)e1627, DOI: 10.7759/cureus.1627

[28] M. Kieliszek, I. Bano, H. Zare, A comprehensive review on selenium and its effects on human health and distribution in middle eastern countries, Biol. Trace Elem. Res. 200 (2022) 971–987, doi: 10.1007/s12011-021-02716-z.

[29] N. Thirumoorthy, R. Senthilkumaran, L. Panayappan, B. Thandapani, K. Ranganathan, Glutathione as Oxidative Stress Marker in Cancer, Handb. Oxidative Stress Cancer Mech. Asp., 94(2022) 353–372, DOI: 10.1007/s00204-020-02838-8

[30] J.M. Stolwijk, R. Garje, J.C. Sieren, G.R. Buettner, Y. Zakharia, Understanding the redox biology of selenium in the search of targeted cancer therapies, Antioxidants, 9 (2020) 420, doi: 10.3390/antiox9050420

[31] J.G. Paithankar, S. Saini, S. Dwivedi, A. Sharma, D.K. Chowdhuri, Heavy metal associated health hazards: An interplay of oxidative stress and signal transduction, Chemosphere, 262 (2021) 128350, https://doi.org/10.1016/j.chemosphere.2020.128350

[32] W. Koenig, High-sensitivity C-reactive protein and atherosclerotic disease: from improved risk prediction to risk-guided therapy, Int. J. Cardiol., 168 (2013) 5126–5134. DOI: 10.1016/j.ijcard.2013.07.113

[33] K. Lechner, C. von Schacky, A.L. McKenzie, N. Worm, U. Nixdorff, B. Lechner, N. Kränkel, M. Halle, R.M. Krauss, J. Scherr, Lifestyle factors and high-risk atherosclerosis: Pathways and mechanisms beyond traditional risk factors, Eur. J. Prev. Cardiol., 27 (2020) 394–406, doi: 10.1177/2047487319869400

[34] S.A.R. Al-Asadi, T.J.A. Almula, Y.S. Abdulrazzaq, A.M. Al-Abadi, Modeling the impact of land use changes on the trend of monthly temperature in Basrah province, Southern Iraq, Model. Earth Syst. Environ., 10 (2024) 3727–3744,https://doi.org/10.1007/s40808-024-01975-8

يؤدي التعرض المهني للكادميوم إلى زيادة الالتهاب، ويخفض مستويات السيلينيوم مما يسبب أمراض القلب والأوعية الدموية

نغم جواد كاظم[1]، ناظم عبد النبي عواد[2]، سعد شاهين حمادي[3]

(1,2) جامعة البصرة، كلية العلوم، قسم الكيمياء
(1) وزارة التربية العراقية، مديرية تربية البصرة
(3) جامعة البصرة ، كلية الطب، الطب الباطني

المستخلص

تناولت الدراسة التأثيرات الصحية للتعرض للمعادن الثقيلة للكادميوم في مواقع ابار النفط الخام، وشارك فيها مجموعتان من الذكور: 90 عاملاً في آبار النفط في البصرة و90 طالباً جامعياً وأعضاء هيئة تدريس .وهدف هذا البحث إلى فهم المخاطر الصحية المحتملة .وتبحث الدراسة في العلاقة بين مستويات البروتين التفاعلي عالي الحساسية في الدم، وهو مؤشر حيوي مرتبط بالالتهاب ومخاطر القلب والأوعية الدموية، والتعرض للكادميوم .كما بحثت في الدور الذي يلعبه الكادميوم في تقليل السيلينيوم .وأظهرت النتائج أن دم العمال يحتوي على مستويات أعلى من الكادميوم ($P \leq 0.0001$) والبروتين التفاعلي عالي الحساسية ($P \leq 0.001$) من تلك الموجودة في المجموعة الضابطة .كما كشفت عينات دم الموظف عن انخفاض كبير في السيلينيوم($P \leq 0.0001$) . كشفت النتائج أن زيادة مستويات البروتين التفاعلي سي عالي الحساسية بسبب التعرض للكادميوم تزيد من خطر الإصابة بأمراض القلب والأوعية الدموية وتسلط الضوء على التفاعل المضاد بين الكادميوم والسيلينيوم، حيث أن السيلينيوم ضروري كمضاد للأكسدة.