Research Article

Section: Health Sciences

Check for updates

Published by Royallite Global in the *Research Journal in Medical and Health Sciences*.

Volume 3, Issue 1, 2022

Article Information

Submitted: 22nd May 2022

Accepted: 30th July 2022

Published: 4th August 2022

Additional information is available at the end of the article

To read the paper online, please scan this QR code

How to Cite:

Sani, A. H., Keke, C. O., Harvey, F., Konamah, A., & Muhammad, T. H. (2022). Renal function parameters and hematological indices among artisans occupationally exposed to lead in Suleja metropolis, Nigeria. *Research Journal in Medical and Health Sciences*, 3(1). Retrieved from https://royalliteglobal.com/rjmhs/article/view/860

Renal function parameters and hematological indices among artisans occupationally exposed to *Lead* in Suleja metropolis, Nigeria

Haruna Sani Aliyu[1], Collins Obinna Keke[2], Florence Harvey[3], Ama Konamah[4] & Tasallah Huawa Muhammad[5]

[1] Department of Public Health Sciences, National Open University of Nigeria, Nigeria

[2] Department of Biochemistry, School of Biological Sciences, Federal University of Technology, Nigeria

[3] Department of Science, Methodist College of Education, Ghana

[4] Department of Science, Kibi College of Education, Ghana

[5] Department of Microbiology, Faculty of Life Sciences, Ahmadu Bello University, Nigeria

Correspondence: elharun014@gmail.com

https://orcid.org/0000-0002-4518-9439

Abstract

Lead exposure at work has been linked to significant health impacts, including a detrimental impact on organ functions, including nephrotoxicity and hematologic impairment. The above cross-sectional study was carried out to examine renal function status and hematological indicators of artisans working in the Suleja metropolis who had occupational lead exposure. There was a maximum of 137 persons studied: 67 artisans (car mechanics, generator repairers, petrol attendants, battery chargers, and spray painters) compared with 70 age-matched healthy control subjects between 17 and 50 years. Everyone had a 10ml venous blood sample drawn in order to determine biological markers (potassium, sodium, urea, uric acid, chloride and creatinine levels) and the complete blood count using standard laboratory methods. Results revealed that occupationally exposed artisans have significantly elevated blood lead of (82.25%, 37.6±0.8 µg/dL), White blood cell count (WBC) 8.5**±1.4 x10**^{9}/L and decreased heamoglobin (Hb) (72.51%, 9.8±1.1 g/dL, P<0.05), mean corpuscular volume (MCV) (76.2 ± 5.4 fL P<0.05), mean corpuscular hemoglobin (MCH) (25.3±1.9 pg, P<0.05) as compared with unexposed subjects (lead: 12.7±1.8 µg/dL, Hb: 14.3 ± 1.5 µg/dL. WBC: 6.5**±1.8 x10**^{9}/L, MCV: 83.4 ± 2.7 fL, MCH: 28.7±1.4 pg; p<0.05 in all cases). Significant elevations were also observed in the mean concentrations of serum urea (8.1±1.6 mmol/L), creatinine (1.3±0.3 mg/dL), sodium (163±32 mmol//L), potassium (6.1±1.3 mEq/L), as well as a considerable decrease in the mean serum chloride levels (86.7±18 mmol/L) of occupationally exposed artisans compared to unexposed subjects (110.5±21 mmol/L), indicating impaired renal and hematologic functions in occupationally exposed individuals.

Keywords: lead exposure, occupationally exposed, artisans, renal impairment, hematologic impairment

Introduction

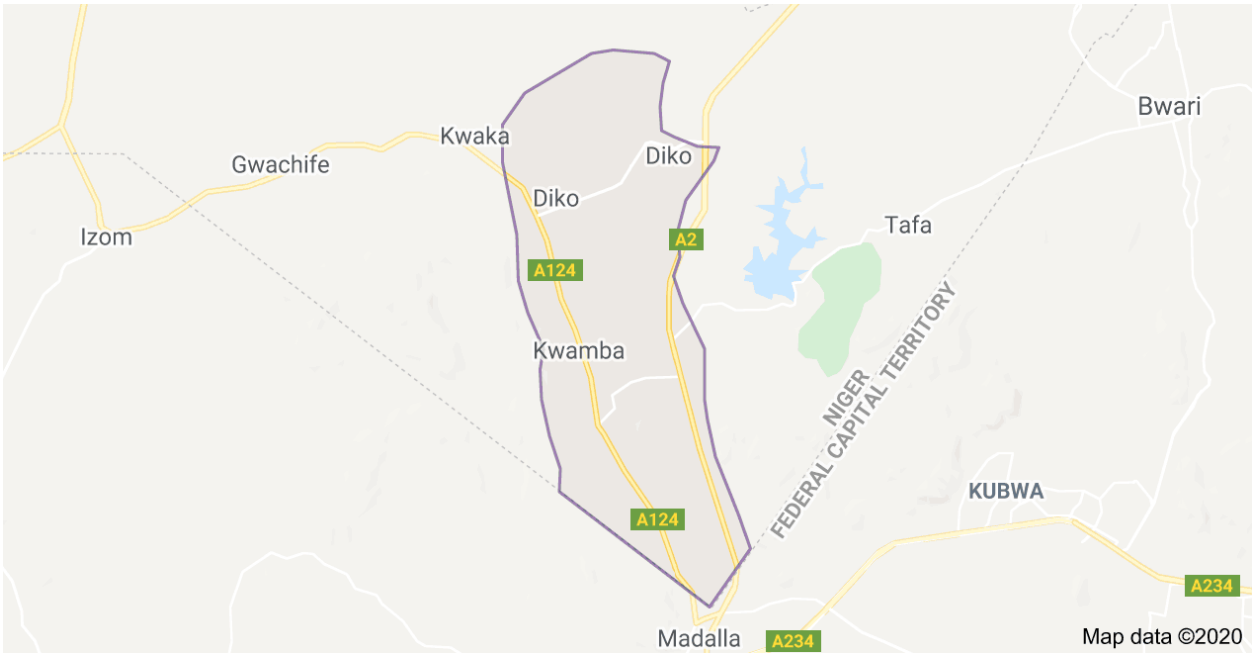
One of the most pervasive hazardous metals and toxicants in the world is lead (Ghaemi *et al*., 2017; Liao *et al*., 2016). Both artificial and natural sources can yield lead. The battery business, paint industry, jobs with radiators, ceramics ,gas stations, cable manufacture, plumbing, contaminated food stuffs , exposure to polluted soil, and in some regions of the world, abuse of opium are possible sources of lead-related dangers (Nakhaee and Mehrpour, 2018). Touch, ingestion, or inhalation are the three ways that lead gets into the body and induce poisoning (Fathabadi *et al*., 2018; Mehrpour, 2012). When ingested into a biological system, more than 95% is bound to erythrocytes and is eventually deposited in bone (Dumková *et al*., 2017). In the near term, the liver and kidneys are the main lead reservoirs (Dongre, 2010). It is devoid of any known biological role in both animals and humans (Loghman - Adham, 1997). Adults with blood lead levels (BLLs) equivalent to or higher than 5µg/dL, as defined by the Centers for Disease Control and Prevention and the National Institute for Occupational Safety and Health (NIOSH), are considered to have increased blood lead levels. Also, CDC and NIOSH believe lead poisoning to be the greatest environmental health think that lead poisoning is the biggest environmental health risk for kids in the US (CDC, 2014). The US Department of Health and Human Services further advises lowering adult blood lead levels to fewer than 10 µg/dL. Nevertheless, research shows that lead-associated effects and difficulties may develop even at low levels, and there is no established safe threshold for lead exposure (CDC, 2015; Kshirsagar *et al*., 2015). The following are non-specific signs and symptoms of lead toxicity in adults: irritability, loss of short-term memory, inattention, depression, loss of coordination, paresthesias, nonspecific abdominal discomfort, weight loss, nausea, headache, loss of deep tendon reflexes and weakness (Karrari *et al*., 2012). It has been discovered over time that lead toxicity plays a substantial role in the development of chronic renal failure (Ernesto and Ludivina, 2012). Moreover, heme biosynthesis disruption has an impact on the hematological system, causing a reduction in the production of globin and heme, as well as erythrocyte development and function hence producing varying degrees of hematological deficiencies or anemia. Environmental and occupational exposure to nephrotoxic chemicals can result in glomerular dysfunction and renal tubular (Marek, 2005). The structure and function of the kidney make it particularly susceptible to these impacts. At rest, a kidney gets one-fifth of the heart's blood flow of which 10% is filtered in the glomerulus. This transports substantial quantities of solutes and particles to the glomeruli and tubules (Marek, 2005).Lead and the majority of assimilated heavy metals are removed from the body primarily via the kidneys and chronic exposure to high lead concentration levels can cause long term nephrotoxic implications namely, interstitial tubular damage, nephritis, and in advanced phases glomerular injury, resulting in persistent renal failure. (Sabath and Robles-Osorio, 2012; Soderland *et al*., 2010). Numerous empirical research have frequently demonstrated a relationship between natural and anthropogenic exposure to lead and an elevated risk of kidney disease, and its accumulation over time which ultimately causes kidney damage, has been widely documented in different regions of the world (Ekong *et al*., 2006; Evans and Elinder, 2011). Despite the significant risk of lead toxicity in Nigeria, information on the prevalence of lead poisoning in the workplace and its effects on renal function and hematologic abnormalities are limited. Consequently, the purpose of this study was to examine occupational exposure to lead and the connection between lead exposure measurements, hematological indices, and kidney function parameters among occupationally exposed artisans in the Nigerian metropolis of Suleja.

Materials and methods

Study area

Suleja is a city in the Nigerian state of Niger that has a population and a land area of 114.6 km2 with a considerable number of artisans and a population of population of 260,240 (NPC, 2016). Situated just north of Abuja, with a geographical coordinate at longitude 7°10›45.80»E and latitude 9°10'50.12"N. The chief occupation of the inhabitants is farming, pottery, cotton weaving and dyeing.

Map of Suleja



(Source: Google/Web/Flicker)

Study Design and Sample

Cross-sectional methodology was used in this investigation. In total, 137 participants were included in the study, including 67 people with confirmed lead exposure at work and 70 control subjects who were not at risk for lead exposure (control).Auto technicians, generator mechanics, welders, radiator repairers, gas station workers, battery chargers, and splatter painters volunteered for this research. Such occupations are high on the list of those that put workers at risk of lead poisoning. (Alli, 2015; Alasia *et al*., 2010). Part-time employees and those with fewer than six months of employment were omitted from this study. The 70 unexposed volunteers (mainly non-smokers and without a history of hypertension or diabetes) were randomly selected residents and students from satellite towns and campuses within the study area. All ages were represented, from 17 to 50. Participants were interviewed face-to-face and asked to complete a self-designed, semi-structured questionnaire, with 20 questions for those who had been exposed and 15 for those who had not. The extra 5 questions for those who were exposed were designed to ascertain their occupational exposure to lead. Participants were guaranteed that their identities and responses will remain anonymous. Interviews were conducted with participants before they took the questionnaire to measure the test's reliability, precision and usefulness.

Blood Collection and Storage

Each person gave 10 mL of venous blood for testing. Blood samples (5 mL) were drawn via venipuncture and placed in potassium ethylene diamine tetra-acetic acid (K-EDTA) tubes, which are used for metal

detection and hematological tests; 5mL were dispensed into sealed plain tubes; suitable for serum electrolytes (Aliyu and Amanabo, 2021) analysis; and lithium heparin containers were utilized for collection of samples for serum urea, creatinine, and uric acid investigations. After being placed on ice at the site of blood collection, the labeled tubes holding the blood samples were transferred to a refrigerator set at 4 °C for storage (Cornelis et al., 2005). Blood was drawn from a vein after the skin at the puncture site was thoroughly swabbed using methylated spirit and dry cotton wool to exclude any possibility of contamination from the outside.

Ethical Clearance

All participants in this study provided informed consent after being educated on the benefits of the research, and the Ethical Committee of General Hospital Suleja, Niger State, granted their approval (GHS/EC/HN: 971-324-121).

Analysis of blood samples for lead

Atomic absorption spectrometry (AAS) was used to assess blood lead levels using the Hassel method (1968). 5 mL of thoroughly mixed EDTA blood were combined with 1 mL of 5 % triton x-100 to induce lysis of the erythrocytes and release the lead. Faster haemolysis could be achieved by putting the tube's contents in a vortex mixer and shaking it up. To chelate the lead, 1 mL of a 2% solution of ammonium pyrrolidine dithiocarbamate was added. Next, 5 mL of methyl-isobutyl ketone was used to extract the solution, and the resulting organic supernatant was placed in screw-capped specimen containers before being analyzed in a Perkin-Elmer 703 Atomic Absorption Spectrophotometer set to a wavelength of 283.3 nm.

Electrolytes, Urea and Creatinine Determination

Determination of serum electrolytes

Using the procedures of Mazzachi *et al*. (1994), serum sodium, potassium, and chloride concentrations were measured using an M508/UV-VIS spectrophotometer set at 630 nm and 578 nm, respectively.

Estimation of serum urea level

The Berthlot Method, as reported by Ochei and Kolhatkar, was used to make an estimate of the serum urea concentration (2007).

Estimation of serum creatinine level

As described by Laron, the Jaffe-Slot Alkaline Picric Acid procedure was used to measure serum creatinine concentration (1972).

Determination of uric acid levels

The method of Fujihara *et al* (1987) for measuring uric acid was used. At 546 nm with a RA-50 spectrophotometer (Ames/Technicon, France), this technique is quite similar to the one proposed by Praetorius and Poulsen (1958).

Determination of hematological parameters

The DIATRON Abacus 380 Hematology Analyzer was utilized in order to investigate the hematologic parameters of the complete blood count (CBC) (Diatron-A380, Hungary). The method developed by Tasevski and colleagues was used to calculate the mean corpuscular volume (MCV), the amount

of hemoglobin (Hb), the red cell distribution width (RDW), and the number of reticulocytes with basophilic stippling (2002).

Statistical Analysis

Using Statistical Package for the Social Sciences (Version 19), we compared the means of the control and experimental groups, with values provided as mean± SD, using the Students t-test and Pearson's bivariate correlation coefficient (SPSS Software, Chicago, IL, USA). For this study, the threshold for statistical significance was set at $P \leq 0.05$.

Results

Table 1: Demographic characteristics of research subjects.

Characteristics	Exposed individuals	Unexposed individuals (control)
Sex		
Male	56	38
Female	11	32
Total	67	70
Age		
17−24	22	24
25−35	31	25
36−50	14	21
Occupation		
Automobile mechanics	25	-
Petrol attendants	14	-
Radiator repairers	11	-
Battery chargers	08	-
Spray painters	09	-

The average age of the 167 individuals was 28.7 ± 11.8 years (range: 17-50 years). 69 % of the participants were men with a mean age of 32.7± 10.2 (Table 1). There were 11 and 32 females in the exposed and unexposed groups, respectively. Age differences between the two groups were not statistically significant ($Z = 0.69$, $P = 0.49$). (Table1).

Table 2: Mean blood lead (BPb) levels in artisans occupationally exposed and unexposed subjects

Group	Blood Pb (µg/dL)
Exposed subjects	37.6±0.8*
Unexposed subjects	12.7±1.8*

All values are means ± SDs, * = statistically significant at $p < 0.05$

The mean blood lead level in exposed artisans was 37.6 ± 0.8 µg/dL, significantly higher than 12.7±1.8 in unexposed subjects (Table 2).

Table 3: Mean serum urea, electrolytes, creatinine, and uric acid levels in artisans occupationally exposed to lead and in unexposed subjects.

Group	Urea (mmol/L)	Sodium (mEq/L)	Potassium (mmol/L)	Chloride (mmol/L)	Creatinine (mg/dL)	Uric acid (mg/dL)
Exposed subjects	8.1±1.6*	163±32*	6.1±1.3*	86.4±18	1.3±0.3	4.6±1.3*
Unexposed subjects	4.6±0.6*	138±17*	4.5±0.9*	110.5±21	1.1±0.6	3.9±0.9*

*Statistically significant P-value.

Table 4: Mean blood hematological parameters in artisans occupationally exposed and unexposed subjects.

Group	MCV (fL)	Hb (g/dL)	WBC ($x10^9$cells/L)	MCH (pg)
Exposed subjects	**76.2 ± 5.4***	**9.8±1.1***	**7.5±1.4***	**25.3±1.9***
Unexposed subjects	**83.4±2.7***	**14.3±1.5***	**6.5±1.8***	**28.7±1.4***

*Statistically significant P-value.

As shown in Tables 3 and 4, the biochemical and hematologic values were compared between the exposed and unexposed groups. White blood cell levels were considerably higher in the exposed group of artisans. (8.5**±1.4 x10**^{9}/L); with a corresponding lower hemoglobin (Hb) level, mean corpuscular volume (MCV) (76.2±5.4 vs 83.4±2.7, P = 0.005) and mean corpuscular hemoglobin (MCH) (25.3±1.9 vs 28.7±1.4, P = 0.005) respectively as seen in Table 4.

Discussion

In this study, artisans in the Suleja metropolitan region of Niger State, Nigeria, who were exposed to lead at work, were compared to a control group of healthy persons to determine the relationship between blood lead levels, renal function and hematologic markers. According to the findings of this study, serum electrolytes, urea, and creatinine were significantly correlated with BLL and WBC. It shown that exposure to occupational lead has a sizable impact on electrolytes, urea, and creatinine as they relate to renal function. BLLs were substantially greater in occupationally exposed artisans than in control participants. This is comparable to earlier research on lead exposure at work (Goldman *et al*., 1998). The artisans in this study had a mean BLL of 59.6 µg/dL, which is commensurate with the threshold indicative of severe lead poisoning (Orisakwe, 2009). This result is also in line with findings from a different study conducted in Nigeria, which found that occupationally exposed patients had a mean BLL of 56.3 (0.95) µg/dL as opposed to controls and studies conducted in other nations, which had a BLL of 30.47 (1.4) µg/dL. A research conducted on Korean lead workers found that their mean BLL was 32.00 (15.00) µg/dL, while the mean BLL of the control group was 5.8(1.8) 22µg/dL (Weaver *et al*., 2003). Similarly, Jung *et al.* found that the mean BLL for highly exposed workers was 74.6 (7.8) µg/dL, for moderately exposed workers it was 46.5 (5.9) µg/dL, and for mildly exposed workers it was 24.3 (2.7) µg/dL, with a mean BLL of 7.9 (1.4) µg/dL for the control group.

The slightly elevated mean BLLs seen in the control group here and in many other studies such as those by (Ogunfowokan *et al*., 2002; Galadima and Garba , 2012 ; Orisakwe, 2009) are suggestive of the widespread nature of lead pollution in the environment in Nigeria .

The WHO permissible range for lead in adults is less than 10 µg/dL. Regardless of one's

line of work, this raises major concerns about environmental lead exposure for virtually everyone. Artisans who were exposed to lead at work had considerably higher levels of urea and creatinine than the control group. Higher than normal levels of urea have been seen in other researches (Amah *et al*., 2014; Dongre *et al*., 2010). Therefore these results are consistent with them. In line with findings from related investigations by Okpogba *et al* (2018). Dioka *et al*. (2004) and Orisakwe *et al*. (2009) found lower urea concentrations than the present concentrations observed in this paper (2007). Zinat *et al.* (2012) found that lead-exposed Bangladeshi car workers had significantly lower creatinine levels, however this finding is in contrast with their findings. According to the findings, elevated levels of urea and creatinine may indicate impaired renal function in lead-exposed artisans due to their employment. Creatinine and urea are both indicators of renal function and are always maintained within the recommended ranges in healthy people (Ahmed, 2011; Allen, 2012). Their buildup in the blood suggests kidney disease. (Appel *et al*., 2008; Perazella and Khan, 2006).Furthermore, there was no noticeable difference in uric acid levels between the exposed group and the control group (72%; $p>0.05$), with all values being within the acceptable range. Uric acid is produced by the activity of the enzyme xanthine oxidase on xanthine and hypoxanthine during the breakdown of purines in humans. This result contradicts the consistently reported higher amounts of uric acid in lead intoxication (Loghman –Adham, 1997). Electrolyte panels are often employed to check for an electrolyte or acid-base imbalances as well as to diagnose or track the responses to treatment on a confirmed imbalance that is affecting the function of the kidneys.

The electrolytes test measures bicarbonate, chloride, potassium and sodium for the diagnosis and treatment of renal, hormonal, acid-base, and water balance problems, among others. Potassium is a powerful electrolyte indicator of renal failure. During renal failure, increased plasma potassium is caused by both reduced filtration and reduced potassium production in the distal tubule. The most serious and potentially fatal consequence of renal failure is hyperkalemia (James and Mitchel, 2006). Lead occupationally exposed artisans' sodium ion (Na^+) (163±32) and potassium (K^+) (6.1±1.3) levels were considerably higher ($p<0.05$) than control participants (138±17) and (4.5±0.9). However, chloride (Cl^-) levels in lead-exposed artisans (84.11±7.26) were substantially lower ($p<0.05$) than in controls (26.73±0.20). This finding is consistent with the findings of Onuegbu *et al*. (2011) on kidney parameters of those exposed to lead at work. It, nonetheless contradicts the findings of Babalola and Babajide (2009), who found that while there was a large rise in BLLs in a group of industrial employees in the Ewekoro, Abeokuta granite sector, there was no distinction in Na^+ and K^+. The increased Na^+ concentration found in the serum of factory workers in this research may have resulted from water loss caused by improper osmolarity regulation, which occurs every once in a while due to renal or hepatic disease or persistent sweating without intake of water, and high K^+ due to the kidneys' inability to remove ingested potassium, most likely caused by dehydration (Leaf and Santos, 1961). Maintaining adequate K^+ concentrations in extracellular fluids is critical, especially for proper heart function. High amounts produce broad intra-cardiac block, whilst low quantities decrease heart muscle contractility. Furthermore, the current study found a substantial, positive relationship between BLLs and WBC, as well as an inverse relationship between Hb, MCV, and MCH counts in occupationally exposed artisans. This study supports the findings of Mazumdar and Goswami (2014). Kim and Lee in 2008 found a substantial and inverse relationship between RBCs and BLL and haemoglobin at even lead levels under 10µg/dL. They discovered that increasing lead levels in red blood cells increases the probability of borderline anaemia by 36.2 % and clinical anaemia by 134.4 % in the Korean population, respectively (Moayedi *et al*., 2008; Stoleski, 2008). Lead's effects on RBCs could very well be due to either a direct damaging effect on the erythrocyte or an impairment of hemoglobin synthesis. Lead has been shown

to impede haem production (Mazumdar and Goswami, 2014). It also affects red blood cell membranes by disrupting the transport and energy mechanisms (Kshirsagar, 2015) consequently lowering their life-span. Lead may also result in insufficient maturation of red blood cells, poor iron absorption, incorrect erythropoietin production, and anemia in the long run. It's possible that greater exposure to lead dust or fumes is to blame for the noticeably elevated total WBC count in these occupationally exposed artisans. Therefore, estimating hematological characteristics is helpful for identifying artisans who work in environments where lead is present. In people exposed to lead at work, the cardiovascular system is known to be impacted. Therefore, lead-caused nephrotoxicity could be a likely cause of these artisans' secondary hypertension.

Conclusion

This research has successfully demonstrated that there were noticeable upsurges in the mean BLLs, WBC, serum urea, creatinine, Na, and K levels with an equivalent significant decline in the Hb, MCV, MCH, and mean serum chloride levels in the artisans occupationally exposed to lead in Suleja metropolis. These findings indicate a compromised renal function and hematologic impairment in exposed artisans.

References

Alasia, D. D., Emem-Chioma, P. C., & Wokoma, S. F. (2010). Occupational and environmental lead exposure in Port Harcourt, Nigeria: analysis of its association with renal function indices. *Nigerian Journal of Medicine*, *19*(4), 407-414.

Alessio, L. (1988). Relationship between "chelatable lead" and the indicators of exposure and effect in current and past occupational exposure. *Science of The Total Environment*, *71*(3), 293-299.

Appel, L. J., Wright, J. T., Greene, T., Kusek, J. W., Lewis, J. B., Wang, X., ..& Agodoa, L. Y. (2008). Long-term effects of renin-angiotensin system–blocking therapy and a low blood pressure goal on progression of hypertensive chronic kidney disease in african americans. *Archives of Internal Medicine*, *168*(8), 832-839.

Babalola, O. O., & Babajide, S. O. (2009). Selected heavy metals and electrolyte levels in blood of workers and residents of industrial communities. *African Journal of Biochemistry Research*, *3*(3), pp-037.

Babalola, O. O., Ojo, L. O., & Aderemi, M. O. (2005). Lead levels in some biological samples of auto-mechanics in Abeokuta, Nigeria.

Cave, M., Appana, S., Patel, M., Falkner, K. C., McClain, C. J., & Brock, G. (2010). Polychlorinated biphenyls, lead, and mercury are associated with liver disease in American adults: NHANES 2003–2004. *Environmental health perspectives*, *118*(12), 1735-1742.

CDC. Preventing lead poisoning in young children. Atlanta, GA: CDC [Accessed June 5, 2014] http://www.cdc.gov/nceh/lead/publications/prevleadpoisoning.pdf

Centers for Diseases Control and Prevention 2015. http:// www.cdc.gov/niosh/topics/ABLES/description.html. Accessed July, 2018. 9. Keramati MR, Ghasemi MN, Mood MB. Correlation between iron deficiency and lead intoxication in the workers of a car b

Centers for Diseases Control and Prevention 2015. http://www.cdc.gov/niosh/topics /ABLES/description. html.[Accessed July,2018].9.Keramati.

Cornelis, R., Heinzow, B., Herber, R. F. M., Christensen, J. M., Poulsen, O. M., Sabbioni, E., ... & Vesterberg, O. (1995). Sample collection guidelines for trace elements in blood and urine (technical report). *Pure and applied chemistry*, *67*(8-9), 1575-1608.

Dioka, C. E., Orisakwe, O. E., Adeniyi, F. A. A., & Meludu, S. C. (2004). Liver and renal function tests in artisans occupationally exposed to lead in mechanic village in Nnewi, Nigeria. *International Journal of Environmental Research and Public Health*, *1*(1), 21-25.

Dongre Nilima, N., & Rathi, S. A. P. A. J. (2010). Occupational Lead Exposure In Automobile Workers In North Karnataka (India): Effect On Liver And Kidney Functions.

Dumková, J., Smutná, T., Vrlíková, L., Le Coustumer, P., Večeřa, Z., Dočekal, B., ... & Buchtová, M. (2017). Sub-chronic inhalation of lead oxide nanoparticles revealed their broad distribution and tissue-specific subcellular localization in target organs. *Particle and fibre toxicology*, *14*(1), 1-19.

Ekong, E. B., Jaar, B. G., & Weaver, V. M. (2006). Lead-related nephrotoxicity: a review of the epidemiologic evidence. *Kidney international*, *70*(12), 2074-2084.

Evans, M., & Elinder, C. G. (2011). Chronic renal failure from lead: myth or evidence-based fact?. *Kidney international*, *79*(3), 272-279.

Fathabadi, B., Dehghanifiroozabadi, M., Aaseth, J., Sharifzadeh, G., Nakhaee, S., Rajabpour-Sanati, A., ... & Mehrpour, O. (2018). Comparison of blood lead levels in patients with Alzheimer's disease and healthy people. *American Journal of Alzheimer's Disease & Other Dementias®*, *33*(8), 541-547.

Galadima, A., & Garba, Z. N. (2012). Heavy metals pollution in Nigeria: causes and consequences. *Elixir pollution*, *45*(1), 7917-7922.

Ghaemi, K., Ghoreishi, A., Rabiee, N., Alinejad, S., Farzaneh, E., Zadeh, A. A., ... & Mehrpour, O. (2017). Blood lead levels in asymptomatic opium addict patients; a case control study. *Emergency*, *5*(1).

Jakubowski, M. A. R. E. K. (2005). Influence of occupational exposure to organic solvents on kidney function. *International Journal of Occupational Medicine and Environmental Health*, *18*(1), 5-14.

Jung, K. Y., Lee, S. J., Kim, J. Y., Hong, Y. S., Kim, S. R., KIM, D. I., & Song, J. B. (1998). Renal dysfunction indicators in lead exposed workers. *Journal of occupational health*, *40*(2), 103-109.

Karrari, P., Mehrpour, O., & Abdollahi, M. (2012). A systematic review on status of lead pollution and toxicity in Iran; Guidance for preventive measures. *DARU Journal of Pharmaceutical Sciences*, *20*(1), 1-17.

Kasiske, B. L. (2000). Laboratory assessment of renal disease: clearance, urinalysis, and renal biopsy. *The kidney.*

Kim, Y., & Lee, B. K. (2013). Increased erythrocyte lead levels correlate with decreased hemoglobin levels in the Korean general population: analysis of 2008–2010 Korean National Health and Nutrition Examination Survey data. *International archives of occupational and environmental health*, *86*(7), 741-748.

Kshirsagar, M., Patil, J., Patil, A., Ghanwat, G., Sontakke, A., & Ayachit, R. K. (2015). Biochemical effects of lead exposure and toxicity on battery manufacturing workers of Western Maharashtra (India): with respect to liver and kidney function tests. *Al Ameen J Med Sci*, *8*(2), 107-114.

Leaf, A., & Santos, R. F. (1961). Physiologic mechanisms in potassium deficiency. *New England Journal of Medicine*, *264*(7), 335-341.

Liao, L. M., Friesen, M. C., Xiang, Y. B., Cai, H., Koh, D. H., Ji, B. T., ... & Purdue, M. P. (2016). Occupational lead exposure and associations with selected cancers: the shanghai Men's and Women's health study cohorts. *Environmental health perspectives*, *124*(1), 97-103.

Loghman-Adham, M. (1997). Renal effects of environmental and occupational lead exposure. *Environmental health perspectives*, *105*(9), 928-939.

Mazumdar, I., & Goswami, K. (2014). Chronic exposure to lead: a cause of oxidative stress and altered liver function in plastic industry workers in Kolkata, India. *Indian Journal of Clinical Biochemistry*, *29*(1), 89-92.

Mehrpour, O., Karrari, P., & Abdollahi, M. (2012). Chronic lead poisoning in Iran; a silent disease. *DARU Journal of pharmaceutical Sciences*, *20*(1), 1-2.

Moayyedi, S. N., Mehbod, A. S. A., Fanei, A., & Mohajerani, H. R. (2008). The relationship between blood lead levels and clinical syndromes. *Research in Medicine*, *32*(1), 75-79

Nakhaee, S., & Mehrpour, O. (2018). Opium addiction as new source of lead poisoning: an emerging epidemic in Iran. *EXCLI journal*, *17*, 513.

Ochei, J., & Kolhatkar, J. (2007). Estimation of plasma urea by Berthelot reaction. *Medical Laboratory Science, Theory and Practice, 6th reprint, Tata McGraw Hill Publishing Company Limited, New Delhi*, 114-115.

Okpogba, A. N., Ogbodo, E. C., Dike, C. C., Nwoko, S. O., Ugwu, E. C., Amah, A. K., ... & Onyeneke, E. C. (2019). Evaluation of heavy metal levels in blood of cable manufacturing factory workers in Nnewi. *Int J Clin Biochem Res*, *6*(3), 430-436.

Okpogba, A. N., Ogbodo, E. C., Okpogba, J. C., Analike, R. A., Amah, A. K., Odeghe, B. O., ... & Onyeneke, C. E. (2018). Assessment of kidney function status in chickens (Gallus gallus domestica) in rural (Elele) and urban (Nnewi) Areas. *J Med Sci Clin Res*, *6*(12), 1048-52.

Orisakwe, O. E. (2009). Environmental pollution and blood lead levels in Nigeria: who is unexposed?. *International journal of occupational and environmental health*, *15*(3), 315-317.

Sabath, E., & Robles-Osorio, M. L. (2012). Renal health and the environment: heavy metal nephrotoxicity. *Nefrología (English Edition)*, *32*(3), 279-286.

Sani, A. H., & Amanabo, M. (2021). Lead: A concise review of its toxicity, mechanism and health effect. *GSC Biological and Pharmaceutical Sciences*, *15*(1), 055-062.

Shaik, A. P., & Jamil, K. (2009). Polymorphisms in MGP gene and their association with lead toxicity. *Toxicology Mechanisms and Methods*, *19*(3), 209-213.

Soderland, P., Lovekar, S., Weiner, D. E., Brooks, D. R., & Kaufman, J. S. (2010). Chronic kidney disease associated with environmental toxins and exposures. *Advances in chronic kidney disease*, *17*(3), 254-264.

Stoleski, S., Karadžinska-Bislimovska, J., Stikova, E., Risteska-Kuc, S., Mijakoski, D., & Minov, J. (2008). Adverse effects in workers exposed to inorganic lead. *Arhiv za Higijenu rada i Toksikologiju*, *59*(1), 19-29.

US Department of Health and Human Services. (1999). Agency for Toxic Substances and Disease Registry-ATSDR.

Weaver, V. M., Lee, B. K., Ahn, K. D., Lee, G. S., Todd, A. C., Stewart, W. F., ... & Schwartz, B. S. (2003). Associations of lead biomarkers with renal function in Korean lead workers. *Occupational and environmental medicine*, *60*(8), 551-562.

Zaki, A., El Shazly, M., Abdel Fattah, M., El Said, K., & Curtale, F. (1998). Lead toxicity among working children and adolescents in Alexandria, Egypt. *EMHJ-Eastern Mediterranean Health Journal, 4 (3), 520-529, 1998*.

Zinat, A., Hossain, M., Bhowmik, S., Khanom, M., Islam, L. N., & Nabi, A. H. M. N. (2012). Modulation of renal function indices of the occupationally lead exposed Bangladeshi automobile workers. *International Journal of Environmental Sciences*, *3*(2), 910.