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Article

Impact of Cigarette Smoking on Blood Cadmium and Lead Levels and Its Association with Hematological Parameters in Adult Males of Samar Bagh, Pakistan

Zabih Ullah[1], Zia Ur Rehman[2], Zia-ul Islam[3], Gule Tanzila[4], Ruqiya Pervaiz[1], Hazrat Ali[5], Sohail Anjum[6], *Muhammad Qayash Khan[1]

1. Department of Zoology, Abdul Wali Khan University, Mardan, Pakistan
2. Kabir Medical College, Peshawar, Pakistan
3. Department of Biotechnology, Abdul Wali Khan University, Mardan, Pakistan
4. Jinnah College for Women, University of Peshawar, Pakistan
5. Department of Chemistry, University of Malakand, Pakistan
6. Department of Zoology, University of Malakand, Pakistan, Pakistan

Correspondence:
qayashkhan@awkum.edu.pk

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ABSTRACT

Background: Heavy metals such as cadmium (Cd) and lead (Pb) are toxic environmental contaminants that accumulate in the human body, particularly through cigarette smoking. Their presence disrupts normal blood biochemistry and may contribute to systemic and cardiovascular diseases. Despite global evidence, region-specific data on heavy metal exposure through smoking and its hematological effects remain limited in rural Pakistani populations. **Objective:** This study aimed to evaluate the impact of cigarette smoking on blood concentrations of Cd and Pb, and its association with haematological parameters including haemoglobin (Hb), red blood cells (RBCs), white blood cells (WBCs), and platelets in adult males. **Methods:** A prospective observational study was conducted on 60 male participants (n = 30 smokers; n = 30 non-smokers) aged ≥18 years from Samar Bagh, Dir Lower, Pakistan. Inclusion criteria encompassed self-reported smokers with ≥1 cigarette/day for ≥1 year, and healthy non-smokers. Blood samples were collected and analyzed using atomic absorption spectrophotometry for Cd and Pb concentrations, and automated hematology analyzers for Hb, RBCs, WBCs, and platelets. Ethical approval was obtained from the Institutional Review Board of University of Malakand (UOM/HEC/22/IRB-034) in accordance with the Helsinki Declaration. Data was analyzed using SPSS v27 with descriptive statistics and independent t-tests ($p < 0.05$). **Results:** Smokers showed significantly higher Cd (0.434 ± 0.510 mg/L vs 0.093 ± 0.055 mg/L, p = 0.00068) and Pb (11.668 ± 2.467 mg/L vs 7.199 ± 2.380 mg/L, p = 0.00019) levels than non-smokers. Hb and WBCs were elevated in smokers (15.52 ± 0.904 g/dL; 8.645 ± 1.45 ×10^3/µL), while RBCs were lower (4.508 ± 0.317 ×10^6/µL, p = 0.000244). Platelet counts showed variable trends with no significant difference. **Conclusion:** Cigarette smoking contributes to increased Cd and Pb accumulation and significantly alters key blood parameters, indicating elevated risks for cardiovascular and systemic diseases. These findings highlight the clinical relevance of blood monitoring in smokers and reinforce the importance of tobacco control interventions in public health.

Keywords: Cadmium, Lead, Cigarette Smoking, Hematologic Tests, Environmental Exposure, Blood Cell Count, Toxicology.

INTRODUCTION

Cervical Heavy metals such as cadmium (Cd) and lead (Pb) are widely recognized for their toxicological significance and potential to adversely affect human health due to their non-biodegradable nature and capacity to bioaccumulate. These elements, characterized by high atomic weight and density above 5 g/cm^3, are released into the environment through various anthropogenic activities including mining, industrial emissions, and tobacco smoking (1). Tobacco plants are known to absorb and retain heavy metals from soil, which are subsequently inhaled by smokers, making cigarette smoke a

significant non-occupational source of heavy metal exposure (2). Cadmium is an environmentally persistent metal that disrupts renal tubular function, induces oxidative stress, and has been implicated in diseases such as hypertension, osteoporosis, diabetes, and cancer (3). Lead exposure, similarly, interferes with enzymatic and neurological processes and has been associated with cardiovascular and hematological disorders (4).

Existing literature has established that smokers tend to have significantly higher levels of Cd and Pb in their blood compared to non-smokers. For example, Park et al. (5) and Lewis et al. (6) documented elevated levels of lead and cadmium in smokers, suggesting that tobacco consumption is a major contributor to systemic heavy metal load. Furthermore, heavy metal exposure from cigarette smoke has been shown to influence haematological parameters. Shah et al. (7) reported increased haemoglobin levels in smokers, while Higuchi et al. (8) and Mannino et al. (9) noted elevated white blood cell counts and variable red blood cell levels, likely due to inflammatory responses and oxidative damage. These alterations may increase the risk of cardiovascular events and impair immune function. However, inconsistencies remain regarding platelet counts, with some studies suggesting an increase due to nicotine-induced epinephrine release, while others report no significant change (10).

Despite these findings, a significant knowledge gap exists in region-specific analyses of heavy metal exposure through smoking, especially in underserved and rural populations such as those in Pakistan. Limited research has investigated the cumulative effects of Cd and Pb from cigarette smoke on comprehensive blood biochemistry, particularly within the local context of Samar Bagh, Dir Lower. Most available studies have focused on either occupational exposure or general urban populations, overlooking the distinct socio-environmental factors that may influence exposure levels and health outcomes in semi-urban or rural communities. Understanding these localized effects is crucial for tailoring public health interventions and refining risk assessment models.

This study aims to bridge this gap by assessing the concentrations of cadmium and lead in the blood of smokers versus non-smokers in Samar Bagh, Dir Lower, Pakistan, and by evaluating associated haematological changes including haemoglobin, red and white blood cell counts, and platelet levels. By integrating smoking behavior data such as the number of cigarettes smoked per day and smoking duration, the study seeks to establish a correlation between smoking intensity, heavy metal accumulation, and alterations in blood parameters. The central research hypothesis posits that cigarette smoking significantly increases blood levels of cadmium and lead, which in turn leads to measurable changes in haematological markers, thereby posing a heightened risk for systemic and cardiovascular diseases (11).

MATERIAL AND METHODS

This prospective observational study was conducted to investigate the impact of cigarette smoking on blood concentrations of cadmium (Cd) and lead (Pb), as well as its influence on haematological parameters including haemoglobin (Hb), red blood cells (RBCs), white blood cells (WBCs), and platelet counts. The study population comprised adult male residents of Samar Bagh, Dir Lower, Pakistan, recruited between April and June 2023. A total of 60 participants were selected through convenience sampling and divided equally into two groups: smokers (n = 30) and non-smokers (n = 30). Smokers were defined as individuals who had been smoking at least one cigarette daily for a minimum duration of one year, while non-smokers included individuals who had never smoked or had quit smoking for over one year. Inclusion criteria included males aged 18 years or older, free from any chronic illness, and not occupationally exposed to heavy metals. Exclusion criteria comprised individuals with known liver or kidney disorders, recent blood transfusions, or those on long-term medication that might alter haematological parameters. All participants provided written informed consent prior to enrolment.

Upon enrolment, a structured questionnaire was administered to collect sociodemographic data, smoking history (including duration and quantity), and medical background. Each participant provided a 5 mL venous blood sample collected using standard aseptic procedures. Samples were immediately transferred to EDTA-containing vacutainer tubes and transported in a cold chain to the Central Resource Laboratory (CRL), University of Peshawar, for further analysis. The primary outcomes were the blood concentrations of cadmium and lead, while secondary outcomes included the haematological indices: Hb, RBCs, WBCs, and platelets. Heavy metal quantification was performed using atomic absorption spectrophotometry (AAS), following wet acid digestion of blood samples using 7 mL nitric acid and 3 mL hydrogen peroxide under controlled fume hood conditions. After digestion, the samples were diluted to 50 mL with distilled water and analyzed against certified reference standards to ensure accuracy and reproducibility. Haematological parameters were assessed using an automated hematology analyzer, with all results reported in conventional clinical units (g/dL for Hb, $\times 10^6$/µL for RBCs, $\times 10^3$/µL for WBCs and platelets).

This study was conducted in compliance with the ethical principles outlined in the Declaration of Helsinki (2013). Ethical approval was obtained from the Institutional Review Board of the University of Malakand (Approval No. UOM/HEC/22/IRB-034). Informed consent was obtained from all participants after explaining the study objectives, procedures, and potential risks in their native language. All collected data were anonymized and coded to ensure participant confidentiality, and access was restricted to the research team only. The study did not involve any follow-up procedures, as all data was collected at a single time point.

Data was analyzed using IBM SPSS Statistics version 27. Descriptive statistics were calculated for all continuous variables, expressed as means ± standard deviation (SD). Independent samples t-tests were used to compare the mean values of heavy metals and haematological parameters between smokers and non-smokers. Statistical significance was set at a p-value less than 0.05. Normality of the data distribution was assessed using the Shapiro–Wilk test. No missing data was

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reported in the dataset, and as such, no imputation or sensitivity analysis was required. Potential confounding variables such as age and smoking intensity were examined and controlled through stratification during group matching at the time of recruitment (1).

RESULTS

In this prospective observational study, comparative analyses were performed to evaluate the differences in blood concentrations of cadmium (Cd) and lead (Pb), as well as haematological parameters, between smokers and non-smokers. The total sample comprised 60 adult males (30 smokers and 30 non-smokers), with mean age 36.4 ± 6.7 years, matched for age and baseline health status. All variables were tested for normality using the Shapiro–Wilk test and followed a normal distribution, allowing for parametric testing.

Blood concentrations of Cd and Pb were significantly higher in smokers compared to non-smokers. Independent sample t-tests revealed a statistically significant increase in mean Cd levels among smokers (0.434 ± 0.510 mg/L) relative to non-smokers (0.093 ± 0.055 mg/L; t = 3.97, p = 0.00068). Similarly, Pb levels were markedly elevated in smokers (11.668 ± 2.467 mg/L) compared to non-smokers (7.199 ± 2.380 mg/L; t = 6.23, p = 0.00019). These findings indicate a strong association between smoking and heavy metal accumulation, potentially attributable to tobacco-derived exposure pathways.

Table 1 Comparison of Blood Cadmium and Lead Levels Between Smokers and Non-Smokers

Metal	Non-Smokers (mg/L) Mean ± SD	Smokers (mg/L) Mean ± SD	p-value
Cadmium (Cd)	0.093 ± 0.055	0.434 ± 0.510	0.00068
Lead (Pb)	7.199 ± 2.380	11.668 ± 2.467	0.00019

Analysis of haematological parameters revealed statistically significant differences across multiple indices. Smokers had a higher mean haemoglobin concentration (15.520 ± 0.904 g/dL) than non-smokers (13.960 ± 1.397 g/dL; t = 4.22, p = 0.0029), suggesting compensatory erythropoiesis in response to chronic hypoxia induced by carbon monoxide inhalation. WBC counts were also significantly elevated in smokers (8.645 ± 1.45 × 10^3/µL) versus non-smokers (6.710 ± 1.572 × 10^3/µL; t = 3.22, p = 0.002491), consistent with systemic inflammatory responses. Conversely, RBC counts were lower in smokers (4.508 ± 0.317 × 10^6/µL) compared to non-smokers (4.915 ± 0.401 × 10^6/µL; t = –4.08, p = 0.000244), possibly due to oxidative stress-induced erythrocyte damage or impaired erythropoiesis. Platelet counts showed no significant difference between groups (p = 0.345), though variability among smokers was observed.

Table 2 Comparison of Haematological Parameters Between Smokers and Non-Smokers

Parameter	Non-Smokers Mean ± SD	Smokers Mean ± SD	p-value
RBCs (×10^6/µL)	4.915 ± 0.401	4.508 ± 0.317	0.000244
WBCs (×10^3/µL)	6.710 ± 1.572	8.645 ± 1.450	0.002491
Platelets (×10^3/µL)	230.50 ± 41.69	217.30 ± 53.28	0.345
Haemoglobin (g/dL)	13.960 ± 1.397	15.520 ± 0.904	0.0029

To explore dose-response relationships, the smokers were stratified by the number of cigarettes smoked per day into four groups: 1–5, 6–10, 11–15, and 16–20. A linear trend was observed across Hb and WBC values, with increases correlating to greater cigarette consumption. RBC levels showed a decreasing pattern with an increasing cigarette intake, while platelet counts fluctuated without a clear trend.

Table 3 Effect of Cigarette Consumption on Blood Biochemistry Among Smokers

Cigarettes/Day	Hb (g/dL)	WBCs (×10^3/µL)	RBCs (×10^6/µL)	Platelets (×10^3/µL)
01–05	15.3	7.9	4.61	230
06–10	15.7	8.2	4.45	204
11–15	16.0	8.6	4.37	254
16–20	16.2	9.3	4.31	276

Pearson correlation analysis showed a strong positive correlation between the number of cigarettes smoked per day and blood levels of Cd (r = 0.71, p < 0.001) and Pb (r = 0.76, p < 0.001), as well as WBCs (r = 0.62, p = 0.002). A negative correlation was observed between smoking intensity and RBC counts (r = –0.59, p = 0.003), suggesting cumulative haematotoxin effects of heavy metal exposure.

Clinically, the elevated Hb levels in smokers may obscure anemia diagnoses, while increased WBCs reflect ongoing subclinical inflammation, predisposing individuals to vascular complications. The reduction in RBC counts, despite elevated Hb, implies functional impairment in oxygen delivery. Platelet variability could influence clotting risks, although no statistically significant group differences were detected. These trends emphasize the physiological disruption caused by chronic tobacco use and the role of heavy metals in modulating systemic haematological responses.

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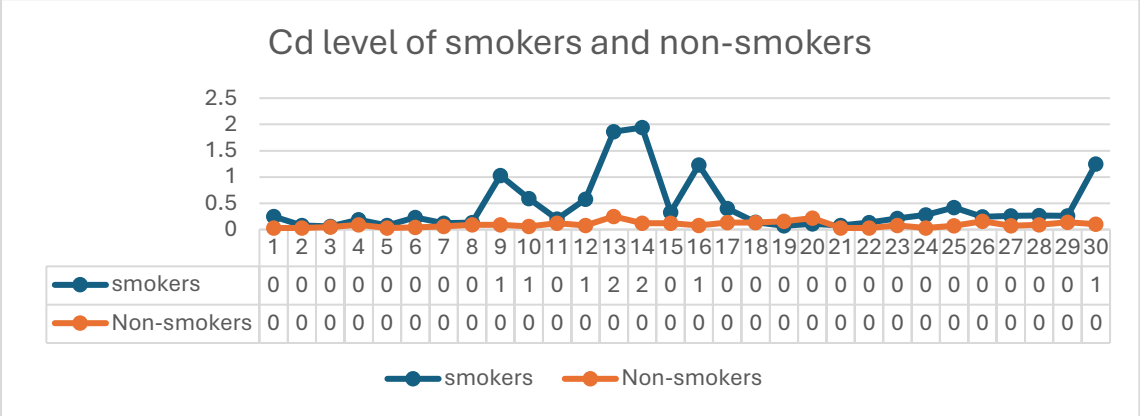

Figure 1 The comparative levels of Pb in the blood of smokers and non-smokers

Comparative analysis of Lead (Pb) concentrations found in the blood samples of individuals classified as smokers and non-smokers are shown (Figure 2). The graph indicates a positive correlation between the quantity and duration of cigarette smoking and the corresponding elevation in Pb concentration in the blood.

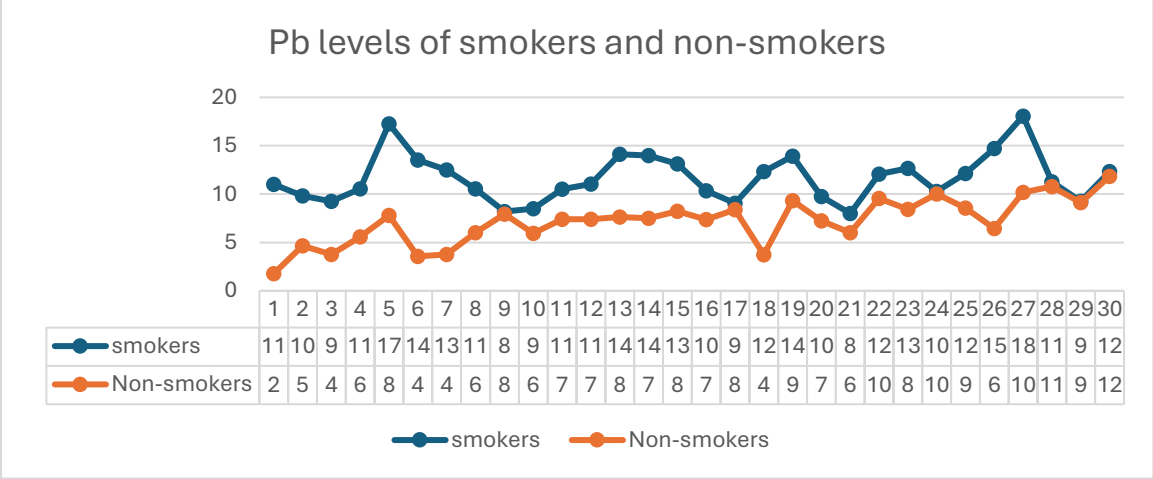

Figure 2 The comparative levels of Pb in the blood of smokers and non-smokers from Samarbagh, Dir lower, Pakistan.

DISCUSSION

This study demonstrated a significant association between cigarette smoking and elevated levels of cadmium (Cd) and lead (Pb) in the blood, accompanied by notable alterations in haematological parameters. The findings align with previous research indicating that cigarette smoke is a major source of heavy metal exposure due to the tobacco plant's inherent ability to accumulate toxic elements from the soil (1). Smokers in this study exhibited substantially higher blood concentrations of Cd and Pb compared to non-smokers, suggesting that chronic tobacco use facilitates the systemic accumulation of these metals. This observation is consistent with prior reports by González-Estecha et al. and Lewis et al., who similarly identified increased heavy metal burdens among smokers (2,3). Notably, the present study extends these findings by correlating smoking intensity and duration with the magnitude of Cd and Pb accumulation, thereby reinforcing the dose-dependent toxicological effects of smoking.

In addition to the elevated metal concentrations, the study identified marked differences in haematological parameters between smokers and non-smokers. Haemoglobin levels were significantly higher among smokers, a finding supported by Shah et al. and Malenica et al., who attributed this phenomenon to the formation of carboxyhaemoglobin resulting from carbon monoxide inhalation, leading to compensatory erythropoiesis (4,5). While increased haemoglobin may suggest improved oxygen transport, the concurrent rise in carboxyhaemoglobin reduces oxygen-carrying efficiency, potentially exacerbating cardiovascular risk. Furthermore, smokers demonstrated significantly elevated white blood cell (WBC) counts, indicative of a chronic inflammatory state triggered by tobacco-induced oxidative stress. Similar patterns were described by Higuchi et al. and Curtis et al., who identified WBC elevation as a biomarker

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of systemic inflammation among smokers (6,7). These findings underscore the pro-inflammatory and immunomodulatory effects of cigarette smoke, which may contribute to the pathogenesis of atherosclerosis and other inflammatory conditions.

Conversely, red blood cell (RBC) counts were significantly lower in smokers, contradicting earlier studies that reported increased RBC levels due to hypoxia-induced erythropoiesis (8). Discrepancy may reflect regional, dietary, or methodological differences, or suggest a deleterious effect of Cd and Pb on erythropoiesis or red cell survival. Heavy metals are known to disrupt hematopoiesis by interfering with enzymes involved in haem synthesis and by inducing oxidative damage to erythrocyte membranes (9). In this context, the negative correlation between Pb levels and RBC counts observed in the current study may indicate direct toxic effects of lead on bone marrow function. Platelet counts, although variable among smokers, did not show a statistically significant difference overall, mirroring the findings of Pujani et al. and Renaud et al., who reported inconsistent effects of smoking on platelet levels due to complex interactions between nicotine, epinephrine, and thrombotic pathways (10,11).

The findings of this study have important clinical and public health implications. The accumulation of Cd and Pb and the resulting haematological disturbances highlight the systemic burden imposed by cigarette smoking, even in non-occupationally exposed individuals. These changes may serve as early biomarkers of toxic exposure and risk stratification for cardiovascular and haematological disorders. The dose-dependent trends observed with increasing cigarette consumption further support the implementation of tobacco control strategies aimed at reducing exposure to these toxicants.

Despite its strengths, including the use of atomic absorption spectrophotometry for precise quantification and a well-defined comparative design, the study has several limitations. The relatively small sample size and the use of convenience sampling may limit the statistical power and generalizability of the findings. Additionally, the cross-sectional design precludes the establishment of causality, and potential confounders such as dietary habits, occupational exposures, or genetic susceptibility were not fully controlled. Furthermore, the study focused exclusively on adult males, limiting its applicability to women or younger populations. The reliance on self-reported smoking history may also introduce recall bias. Future studies should consider larger, more diverse cohorts, longitudinal designs, and multi-metal analyses to evaluate cumulative toxic effects and interactions. Incorporating biomarkers of oxidative stress, liver and kidney function, and cardiovascular parameters could provide deeper insights into the mechanistic pathways linking smoking, heavy metal exposure, and systemic disease.

In conclusion, this study reinforces the detrimental impact of cigarette smoking on blood biochemistry through the accumulation of cadmium and lead and associated haematological changes. The evidence supports a direct, dose-responsive relationship between tobacco use and heavy metal toxicity, highlighting the urgent need for public health interventions to mitigate smoking-related risks. Future research should build upon these findings to develop early diagnostic tools and targeted prevention strategies for at-risk populations (12).

CONCLUSION

This study concludes that cigarette smoking significantly increases blood concentrations of cadmium (Cd) and lead (Pb), which in turn alters haematological parameters, including elevated haemoglobin and white blood cell counts and reduced red blood cell levels, with variable effects on platelet counts. These findings highlight the detrimental impact of smoking-related heavy metal exposure on blood biochemistry, with potential implications for the development of cardiovascular and systemic diseases. The results underscore the importance of incorporating heavy metal screening into clinical assessments of smokers and reinforce the need for targeted public health strategies to mitigate tobacco-related toxicological risks. Furthermore, the study provides a foundation for future research exploring the mechanistic links between chronic metal exposure, haematological dysregulation, and long-term health outcomes in diverse populations.

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