Determination of Some Metal Ions in Blood by AAS and Assessment of Their Toxicity in Exudative Lung Disorder Patients

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Abstract

Metal-induced respiratory diseases are not well documented in Bangladesh. The objective of this study is to assess metal toxicity in terms of concentration levels in exudative lung disorder patients. After acid digestion of blood collected from exudative lung disorder patients, the concentration of eight elements (Cd, Cr, Cu, Pb, Mn, Ni, Fe and Zn) was measured using AAS. The age of the exudative lung disorder patients of both genders ranged from 20 to 75 years, living in urban and rural areas from 11 districts of the Chittagong division. Patients were categorized into three groups: smokers, nonsmokers and former smokers. The role of smoking in the metal toxicity of exudative lung disorder patients was also investigated. Blood samples were collected from healthy persons aged 20-35 years. They were used as a control to compare the metal status of patients. It is shown that current smokers with lung diseases have lower Zn levels in their blood than the patients of former smokers. Linear regression analysis for Ni and Fe in the blood of smokers showed a significant correlation between Fe and Ni at $p=0.008$ and $p=0.003$. Correlation of Mn was insignificant at $p=0.371$, which clearly indicates that smoking may not be a probable factor for increasing Mn in blood. But the level of Fe and Mn in the blood of nonsmokers showed a strong and positive correlation with the coefficient value of 0.814 ($p<0.001$). The investigation showed that metal toxicity is caused more by breathing polluted air from fuel combustion in industries and vehicles than by smoking.

Keywords: *Metal toxicity, Trace elements, Lung diseases, Blood sample, AAS*

1. Introduction

The toxic impact of heavy metals is one of the major threats to the proper functioning of biological systems in the human body. Metal ions may enter the body from the environment through the body wall, respiration, and food [1], leading to various diseases. Elements like iron (Fe), copper (Cu), and zinc (Zn), in trace amounts, have significant roles in many biological systems because they take part in the development of human tissues and organs [2]. Heavy metals such as lead, cadmium, and mercury are highly poisonous. Though some others, like iron, cobalt, and zinc, are essential nutrients, they become toxic in larger amounts. The environmental exposure to Cd, Pb, Cr and Ni has both toxic effects and carcinogenic consequences. For centuries, metals have been known to cause various diseases, including pulmonary diseases [3,4].

Tobacco, cigarette paper, filters and smoke are major sources of metals. Since the gaseous phase of smoke contains these metals, smoking is considered a major source of exposure to them. Cigarette smoke contains metals, causing cancer, and it ranges from 1.1 to 2.9 μg per cigarette [5]. Various studies showed that one pack of cigarettes deposits 2.0–4.0 μg of cadmium. Smokers are considered to be at particular risk of low iron levels. The main health impact (lung damage) of cadmium is due to

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Manuscript History:
Received 23 June, 2025, Revised 8 September, 2025, Accepted 8 September, 2025, Published 31 October, 2025

https://doi.org/10.33736/jaspe.9480.2025

e-ISSN: 2289-7771

inhalation from smoking [6]. World Health Organization (WHO) reported 0.04–0.58 μg nickel per cigarette [7]. Tobacco smoke contains nickel carbonyl, $Ni(CO)_4$ in gaseous form, which is fat soluble. Another study showed that Ni-based compound $NiSO_4$ is responsible for bronchial asthma [8]. Chain smokers and nickel refinery workers may have greater Ni levels in their bodies. Nickel exposure to health from the air is dangerous because of its water solubility, which helps nickel to stay in the lungs. As a result, the concentration of Ni has been found to be higher in pulmonary tissues [9].

Combustion of fuels, petrol and gasoline is an important source of metals in the environment. Inhalation of lead is the primary route for occupational exposure. TEL (tetraethyl lead, $(C_2H_5)_4Pb$) mixed in petrol to reduce the knocking, may swiftly be absorbed by the skin, the lungs, and the gastrointestinal tract [10]. It constantly increases the amount of lead (Pb) in the air as a pollutant. Lead enters the human bloodstream through the lungs and inhibits the synthesis of haemoglobin. It may contribute to the pathogenesis of pulmonary cancers, asthma, and COPD [11].

Cr(VI) gets reduced to Cr(III) by biochemical mechanisms in the lower respiratory tract by the epithelial lining fluid and by pulmonary alveolar macrophages. The reduction in efficiency was significantly greater in smokers than in nonsmokers [12]. Therefore, tobacco smokers are at a greater risk of Cr exposure than SHS (Second Hand Smokers) or nonsmokers. It has been reported that the Cu content in tobacco leaves is approximately 156 mg/g and hence smokers are at greater risk of Cu toxicity than nonsmokers [13,14-17].

Deficiency of zinc causes carcinogenesis by increasing DNA damage and oxidative stress [18]. The lung is exposed to chronic oxidative conditions, and hence it could be sensitive to zinc depletion and cancer [19]. Too much iron absorption by the cell becomes toxic, and it can affect our lungs and airways as well. Increased mucus secretion and scarring of the lungs may result in narrowing of the airways, which makes breathing difficult [20].

Manganese has been discussed in terms of occupational metal pollution. Welding fume contains a small amount of manganese, which is able to enter the human body with inhalation. Manganese absorbed through inhalation has been linked to neurotoxicity in the human body [21]. When metal dust containing manganese is taken for a long time, it is deposited in the nasal/brain airway and in the tracheo-bronchial and alveolar or pulmonary region of the respiratory tract [22]. Manganese absorbed in the nasal/head airway can be transported to the brain via olfactory transport [23]. Manganese is known to accumulate in mitochondria of neurons as well as in the most numerous cell types within the central nervous system, astrocytes and oligodendrocytes. It is the manganese accumulation that disrupts ATP synthesis [24]. Though manganese toxicity has been reported primarily in the central nervous system, toxicity in the lungs, heart, liver, reproductive organs, and fetus is also possible [25].

Thus, regular monitoring of metal concentrations in biological fluids is critically important. This study aimed to assess metal toxicity in terms of concentration level in exudative lung disorder patients who are suffering from the most common lung diseases. In this regard, the concentrations of eight elements, including Cd, Cr, Cu, Pb, Mn, Ni, Fe and Zn, have been determined using an Atomic Absorption Spectrophotometer (AAS) after acid digestion of blood samples collected from patients with exudative lung disorder. The role of smoking in the metal toxicity of patients with exudative lung disorders was also investigated by comparing the concentration levels between smoker and non-smoker patients.

2. Materials and methods

2.1. Study design and participants

The metal toxicity in the blood of patients with different kinds of lung diseases was assessed in this cross-sectional study. Patients of the diseases e.g. asthma, pneumothorax or atelectasis (collapse of part or all of the lung), pneumonia (lung infection), lung cancer, lung abscess, COPD (chronic obstructive pulmonary disease), bronchitis (swelling and inflammation in the bronchial tubes that

carry air to the lungs), bronchial carcinoma, pulmonary tuberculosis (PTb) and airway reflux were included.

In Bangladesh, governmental statistics are limited regarding the size of the exposed population, types of industries, and the prevalence of lung diseases related to metal exposure, such as lung cancer, metal fume fever, nasal septum perforation, bronchial irritation, asthma, and bronchitis. The present study focused on the toxicological approaches to evaluate the effects of selected metals, for example, cadmium, chromium, lead, copper, iron, manganese, nickel, and zinc deposited in the lungs. The Chittagong division of Bangladesh was selected as the study area because no such research had been conducted previously.

The study population consisted of 62 individuals, divided into two groups: the study group (SG) and the control group (CG). Among them, 50 were patients with lung diseases, while the remaining participants served as control subjects of both genders. The age range of the participants was 20 to 75 years. The sampling period was September 2020 to May 2021. There were 30 patients with various pulmonary diseases diagnosed at Chattogram Medical Hospital in Ward No.13 (Medicine Department). Some patients were under treatment in a TB hospital, while a few of them were primarily diagnosed in the Bangladesh Railway Hospital, Bangladesh. Some of the patients who were previously diagnosed with and treated for various lung diseases also voluntarily donated blood. In addition, anthropometric data (body weight, age, height, previous medical history, etc) and information on personal lifestyle, including dietary habits, occupational status, were collected through face-to-face questionnaires. Each patient was asked about their smoking habits prior to sample collection. A written statement was attained along with the current address individual. Based on the questionnaires, 12 volunteers, aged 20-35 years (8 male and 4 female), were selected. Control subjects were selected from the same locality. They were alerted two months before sampling and advised not to eat any kind of seafood. They were informed two months before sampling and advised to avoid consuming any type of seafood. They were also instructed not to take any multivitamins or mineral supplements without consulting a physician. In addition, the control participants had no history of lung diseases or significant medical illness for at least three months prior to sampling. They also had no history of smoking or alcohol consumption. They were advised to have regular, balanced meals and to get adequate rest two months preceding sample collection. The whole blood analysis was used to determine Pb and Cd because these toxic elements bind preferentially to erythrocytes [26].

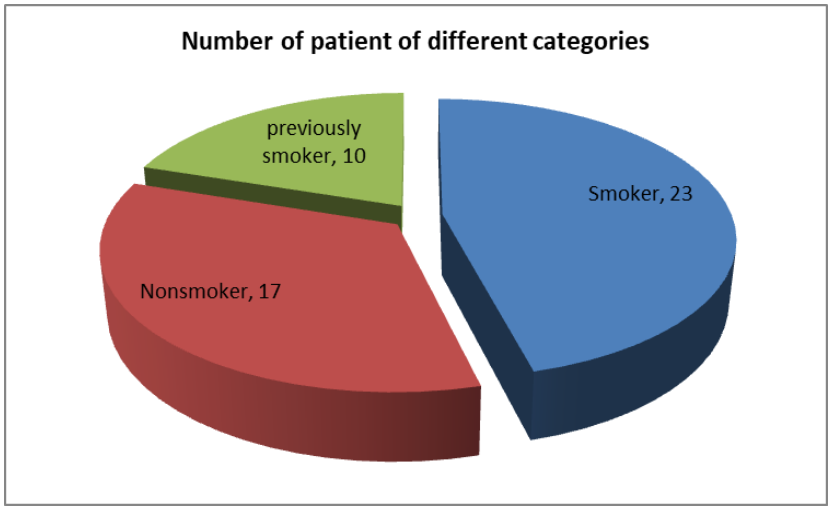

Figure 1. Number of patient of different categories

2.2. Necessary approval

Before collecting samples from the hospitals, written approval was obtained from Chittagong Medical Hospital and Epic Health Care Hospital. All the demographic data were collected from patients and healthy volunteers. The information was kept confidential during and after the completion of the research project through password-protected electronic files and securely locked hard copies.

2.3. Sample collection and storage

The venipuncture method was used for blood collection, during which approximately 3 mL of venous blood was drawn from each patient and healthy subject. Their blood samples were taken by a skilled pathologist in a hygienic way through advanced materials. Venous blood samples ~ 3 mL were collected using a metal-free safety vacutainer blood collection tube (JMI Hospital Requisite Manufacturing Ltd. (JHRML), UBUY Bangladesh) and were stored in -20°C until analysis. For the collection of blood, all the procedures, e.g. hand hygiene and gloves, were used when the venipuncture was performed. 21-gauge needles were used to collect blood from the veins of the SG and CG. 21-gauge is usually used to collect 5-25mL of blood for clinical and laboratory purposes. In that case, Heparin Lithium ASPO (green color tube) disposable blood collection tube (13*75MM, 3mL) manufactured in China was used. These tubes were coated on the inside wall with spray-dried lithium heparin in 10–30 USP units/mL blood [27]. Heparin bound to AT also inhibits FXa. Heparin activates antithrombin, thereby inhibiting coagulation, which additionally acts as an anticoagulant and blocks the clotting cascade.

2.4. Reagents and glassware

All analytical grade chemicals, reagents and the ultra pure water were used to prevent metal contamination. H_2O_2 (30%) [(M.W-34.01), Min 30.0% assay, 99.0 volumes of available oxygen] from LOBA Chemie and HNO_3 (65%) were used. Nitric Acid is a strong mineral acid and an oxidizing agent. H_2O_2 is an oxidizer too. 1 mL and 5 mL pipettes were used for pipetting blood (in 0.5 mL). 20 pieces of 50 mL volumetric flasks were used for sample dilution, and prior to use, the sample solution was diluted in AAS. Filter papers of 11 cm were used for filtering purposes. Prior to use, all the chemicals were tested for metal contamination, if any. All apparatus and plastic materials were dipped in HNO_3 (2 mol L^{-1}) for 24 hours. Ultra-pure distilled water was used for washing and rinsing.

2.5. Preparation of standard solution and preparation of calibration curves

Stock solutions of 1000 µg mL^{-1} of cadmium, chromium, lead, nickel, copper, zinc and iron were prepared with deionized water. Experimental standard solutions of six concentration levels, 1 to 6 µg mL^{-1} for each element, were prepared by dilution of a previously prepared stock solution (1000 µg mL^{-1}). A Shimadzu (Kyoto, Japan) atomic absorption spectrophotometer (Model: AA7000) equipped with a microcomputer-controlled was used for the analysis of experimental samples. By aspirating the working solutions into the acetylene flame, the absorbance of each concentration level was measured and plotted against the corresponding concentration to construct the calibration curves. The absorbance measurements were recorded after adjusting the instrument reading to zero against a blank, which was done with distilled water. Distilled water was aspirated alternatively into the flame after taking the measurement of one solution to remove possible traces of the element before proceeding to the next. The wavelengths for Cd, Cr, Pb, Ni, Mn, Cu, Fe and Zn were 228.4, 357.9, 283.3, 232.0, 279.5, 324.8, 248.3, and 213.9 nm, respectively.

2.6. Wet acid digestion of blood samples

2.6.1. Preparation of standard oxidizing solution

2:1v/v of 65% HNO_3 and 30% H_2O_2 solutions were prepared for sample digestion by the conventional wet acid method using a 10 mL measuring cylinder, and solutions were stored in a reagent bottle with a cover lid. Before the digestion of each sample, a fresh solution was prepared. Blood samples must be digested before introducing the sample into the atomizer. Chemical decomposition of sample matrices was carried out by the acid mineralization (wet digestion) method. Freshly prepared oxidizing agent consisting of a mixture of conc. nitric acid and hydrogen peroxide [HNO_3:H_2O_2, (2:1v/v)] was used to digest the samples.

2.6.2. Blood sample preparation for metal analysis

Blood samples were digested through the above procedures by adopting the method of Yahaya et al. 2013 [27]. All the samples were prepared in triplicate. Precisely 0.5 mL of whole blood was taken into separate Pyrex flasks. Then, 3 mL of freshly prepared oxidising mixture of concentrated nitric acid and hydrogen peroxide [HNO_3 - H_2O_2] (2:1v/v) was added and left for 10 minutes. The flasks were covered with a watch glass. After that, the samples were digested at 60 - 70°C for 1 - 2 hours until a clear digested solution was obtained. The excess acid mixture was evaporated to semi-dry mass. After the solution had cooled completely, it was sifted through filter paper and taken into a 50 ml volume flask. Then, 50 mL of ultra-pure water was added to it to make the solution ready to use in AAS to determine its concentration. Blank extraction without the sample using ultra-pure water was carried out following the complete procedure. The concentrations of metals (Cd, Cr, Pb, Ni, Mn, Cu, Fe and Zn) in blood solutions were determined using Atomic Absorption Spectroscopy (AAS).

2.7. Statistical Analysis

IBM SPSS Statistics Version: 28.0.0.0 (190) was used for the interpretation of data of patients as well as controls. Statistical testing was done using nonparametric tests. The Mann-Whitney *U* Test, Kruskal-Wallis Test were performed with the blood of smokers, nonsmokers and former smokers based on their smoking habit history as well as the disease type. Pearson correlation was used to interpret the data found from the AAS analysis.

2.8. Validation of the AAS method

The AAS method was validated by taking replicate measurements for all standard samples when recovery and RSD were calculated, which satisfied the analytical agreement. A matrix standard reference material, GBW09101 (National Research Centre for Certified Reference Materials, Beijing), was also analysed to check the validation of the AAS method. Recovery complied with the agreements, getting the results (Table 1) within certified levels for all elements. Quantification was performed using a calibration equation obtained from a calibration curve constructed for the six concentration levels within 1-6 µg mL^{-1}. The limit of detection of the method was set at three times the standard deviation of the reagent blank. The limit of detection (LOD) was calculated by the equation, LOD=3. SD/m where SD and m are the standard deviation of the intercept and slope of the calibration curve.

Table 1. Detection limit and analytical results of the certified standard reference material (human hair GBW09101) for all elements.

Metal ions	Detection limit (μg mL^{-1})	Certified range (μg mL^{-1})	Measured (mean±SD) (μg mL^{-1})	RSD (%)	Calibration equation	*Regression coefficient, R^2*
Cd	0.003	0.083–0.107	0.092±0.003	0.32	y=0.0155x-0.004	0.9992
Pb	0.01	6.50–7.90	7.60±0.10	0.52	y=0.053x-0.006	0.9998
Zn	0.06	181.0–197.0	185.71±1.12	0.15	y=0.63x-0.06	0.9987
Cu	0.011	21.6–24.4	21.98±0.95	0.31	y=0.015x-0.016	0.9997
Fe	0.9	64.6–77.8	69.64±0.93	0.25	y=0.23x-0.02	0.9999
Mn	0.008	2.74–3.14	2.76±0.32	0.78	y=0.043x-0.007	0.999
Ni	0.006	3.0–7.0	5.71±1.12	0.35	y=0.063x-0.003	0.9995
Cr	0.02	5.5–7.3	5.60±0.10	0.42	y=0.0055x-0.007	0.9993

3. Results and discussion

The patients with lung disease were categorized based on their smoking status. Among 40 patients, samples were collected from 16 active smokers, 14 nonsmokers and 10 former smokers suffering from different types of lung diseases. Whole blood samples collected from patients of different categories were analyzed via the AAS (Shimadzu Kyoto, Japan, Model: AA7000) and concentrations of eight elements (Cd, Cr, Pb, Ni, Mn, Cu, Fe and Zn) were determined. Results were compared with the metal status obtained from 12 control blood samples collected from healthy persons aged 20 to 40 years.

The patients were three with lung carcinoma, three with bronchogenic carcinoma, six with COPD, six with PTb, seven with asthma, five with pneumonia and ten with other lung diseases. These patients were from different kinds of occupational backgrounds. The concentrations (mean ± SD) of Cd, Cr, Pb, Ni, Mn, Cu, Fe and Zn in blood samples of smokers, nonsmokers and former smokers with types of different lung diseases has been tabulated in Table 2 and graphically presented in Fig. 2. High concentration levels of experimental metals were found in the blood of lung disorder patients across the three catagories.

Table 2**.** The mean ± SD value of Cd, Cr, Pb, Ni, Mn, Cu, Fe and Zn in the blood of the sample.

Elements	**Concentration in blood** (μg mL^{-1})		
	Smokers	**Non Smokers**	**Former Smokers**
Cd	32.548±0.368	24.640±0.327	45.194±0.376
Cr	18.879±0.248	13.014±0.177	33.084±0.303
Pb	29.964±0.245	24.732±0.241	39.975±0.326
Ni	3.654±0.042	3.509±0.033	5.259±0.061
Mn	4.548±0.072	5.986±0.081	6.156±0.064
Cu	18.648±0.148	13.309±0.124	22.834±0.242
Fe	34.744±0.37	28.038±0.304	35.734±0.393
Zn	6.6252±0.08	5.389±0.060	11.84±0.142

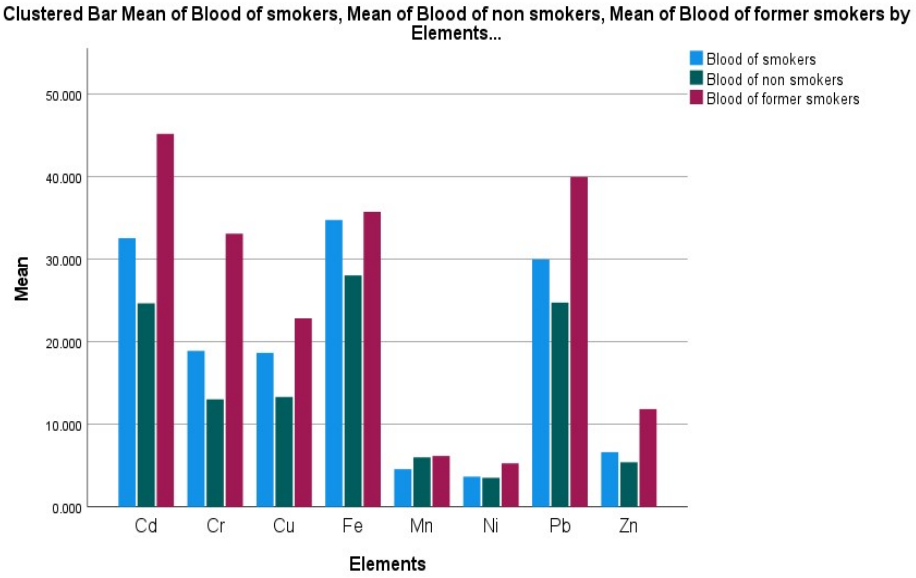

Figure 2. Target metals mean value in the blood of smokers, non-smokers and former smokers.

The mean ± SD value of the elements in the blood of the control and patient groups compared to the standard has been shown in Table 3. Concentrations of Cd, Cr, Pb, Ni, Mn, Cu, Fe and Zn were 32.942±0.356, 20.379±0.023, 30.636±0.265, 4.005±0.043, 5.454±0.073, 17.826±0.163, 32.645±0.352, 7.495±0.089 µg mL^{-1}, respectively. But the mean±SD values of Cd, Cr, Pb, Ni, Mn, Cu, Fe and Zn in control were 0.516±0.004, 1.005±0.016, 1.975±0.031, 0.752±0.010, 1.934±0.031, 1.554±0.024, 16.401±0.188 and 1.9925 ±0.025 µg mL^{-1}. Metal toxicity is evident from the analytical results. Concentration levels in smokers were higher than in nonsmokers, indicating the effect of smoking on lung diseases. However, a lower Zn level was identified in smokers with lung diseases than in nonsmokers or former smokers. Additionally, higher metal levels in control than standard were identified (Table 3, Fig. 3). It is evident that, irrespective of their smoking habit, their occupational and environmental factors might be the reason for metal toxicity in their body. Researchers reported that smoking habit increases lung cancer risk, including different types of respiratory system diseases [29].

Table 3. The mean ± SD value of the elements in the blood of the control and patient groups compared to the standard.

Elements	**Concentration in blood (µg mL^{-1})**		
	Control mean ± SD (n= 12)	**Patients mean ± SD (n=40)**	**Standard values**
Cd	0.516±0.004	32.942±0.356	0.05
Cr	1.005 ±0.016	20.379±0.023	0.025
Pb	1.975±0.031	30.636±0.265	0.05
Ni	0.752±0.010	4.005±0.043	0.02
Mn	1.934±0.031	5.454±0.073	0.06
Cu	1.554±0.024	17.826±0.163	0.85
Fe	16.401±0.188	32.645±0.352	11.50
Zn	1.9925 ±0.025	7.495±0.089	1.50

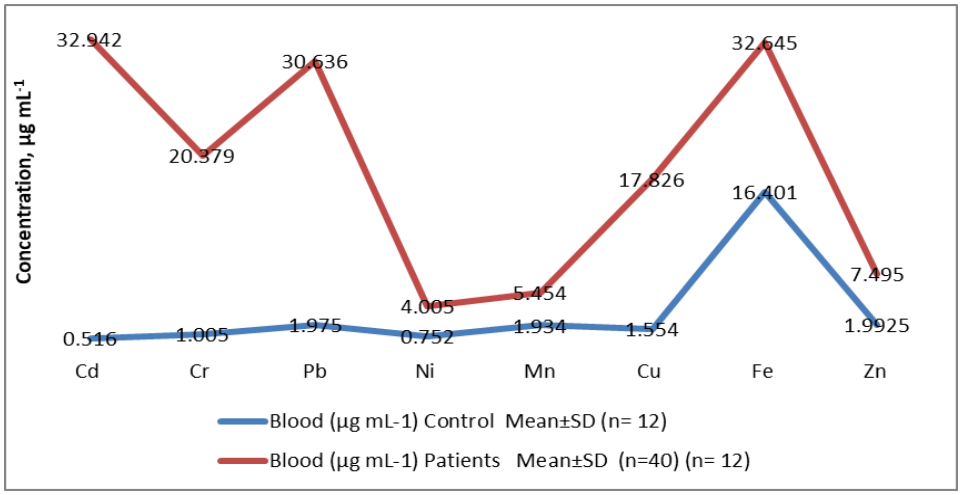

Figure 3. Target metal concentration in the blood of the control (blue) and the patient group (red)

Mean rank (µg mL^{-1}) of Cd, Cr, Pb, Ni, Mn, Cu, Fe and Zn levels in the blood of different lung diseases has been given in Table 4. The ranking of metal concentrations in blood (Table 4) clearly indicates that lung carcinoma, bronchial carcinoma, chronic obstructive pulmonary disease (COPD) and other types of lung diseases show high toxicity, whereas pulmonary tuberculosis (PTb), Asthma, and pneumonia show comparatively lower toxicity.

Table 4. Mean rank (µg mL^{-1}) Cd, Cr, Pb, Ni, Mn, Cu, Fe and Zn levels in the blood of different lung diseases.

Diseases Type	**No**	**Metal concentration in blood (µg mL^{-1})**							
		Cd	**Cr**	**Pb**	**Ni**	**Mn**	**Cu**	**Fe**	**Zn**
Lung Carcinoma	3	25.33	22.33	17.33	26.67	26.00	25.33	27.00	21.50
Bronchial Carcinoma	3	27.83	29.33	25.83	34.00	29.50	30.17	34.33	25.83
COPD	6	28.92	27.17	27.08	27.42	24.67	25.50	27.08	30.75
PTb	6	22.08	22.17	19.75	17.75	17.17	18.58	16.17	19.83
Asthma	7	14.00	14.36	14.79	14.07	17.07	14.71	12.93	15.07
Pneumonia	5	11.40	11.90	13.40	5.80	9.60	14.60	13.40	11.90
Others	10	19.95	20.90	23.90	23.95	23.50	21.30	21.90	20.95

Linear regression analysis was performed for Ni, Fe and Mn in blood with smoking habit, and the null hypotheses were rejected. Table 4 shows that there is a significant correlation between Fe and Ni with smoking habit at p=0.008 and p=0.003. But correlation of Mn is insignificant at p=0.371, which clearly indicates smoking is not a probable factor of increasing Mn in blood (Table 2).

Table 4. Linear regression analysis of Ni, Fe and Mn with smoking habit

Coefficients[a]						
		Unstandardized Coefficients		Standardized Coefficients	t	Sig. p
		B	Std. Error	Beta		
1	(Constant)	1.559	0.292		5.337	<.001
	Ni (blood)	0.336	0.120	0.761	2.801	0.008
	Mn (blood)	0.067	0.074	0.212	0.906	0.371
	Fe (blood)	-0.043	0.013	-0.765	-3.235	0.003
a. Dependent Variable: Smoking Status						

Pearson correlation of Fe and Mn in nonsmoker patients of different lung diseases is shown in Table 5. The correlation coefficient value 0.814 ($p<0.001$) indicates that the correlation is significant. A coefficient interval of 0.8-1.0 indicates a very strong correlation level. There is a strongly positive relationship between Fe absorption and Mn levels found in the human body.

Table 5. Pearson correlation of Fe and Mn in nonsmoker patients with different lung diseases.

Group	Fe in Patients Group	Mn in Patients Group
Fe in Patients Group	1.0	0.814**
Mn in Patients Group	0.814**	1.0

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

A Mann-Whitney U Test for Zn level in the blood of new and previously treated patients was performed. The value of the effect size (r) 0.49 indicates its significantly large effect. According to Cohen J. [30], effect size 0.1 = small effect, 0.3 = medium effect, 0.5 = large effect. From the data presented in Table 6, it can be concluded that previously treated or patients under treatment have higher Zn levels in their blood (U=67.0, p=0.002). The test revealed the significant difference in new patients (median= 5.5, n= 27) and previously treated patients (median= 10.50, n= 13), U=67, Z= -3.141, p= 0.002, r= 0.497. Based on this, it can be concluded that the Zn level in most previously treated patients is higher than the newly diagnosed patients. As Chattogram district is the second commercial city, the level of pollution is high here. One of the rationales behind this is industrialization.

Table 6. Mann-Whitney U Test for Zn level in the blood of new and previously treated patients

Parameters	Constants	*Effect size:* $r = \frac{Z}{\sqrt{N}}$
Mann-Whitney U	67.000	$r = \frac{3.141}{\sqrt{40}} = 0.497$
Wilcoxon W	445.000	
Z, correlation coefficient	-3.141	Here, Z= 3.141, N= 40
Asymp. Sig. (2-tailed)	0.002	
Exact Sig. [2*(1-tailed Sig.)]	0.001[b]	
a. Grouping Variable: Treatment History, b. Not corrected for ties.		

The number of factories in Chattogram city and its adjoining cities is increasing day by day where urbanization is taking place for the purpose of industrialization. Exposure to toxic elements increases in the human body through the inhalation of harmful metal compounds and particles from the air, leading to the gradual accumulation of these toxic metals in the lungs [31]. Metallic dusts deposited in the lungs may give rise to pulmonary fibrosis and functional impairment, depending on the fibrogenic potential of the agent and on poorly understood host factors. The proportion of lung

cancer due to exposure to metals attributable to occupation is around 15%. There is evidence of increased lung cancer mortality among workers involved in welding or underground mining. [32].

5. Conclusion

Since different types of lung diseases in humans are strongly correlated with smoking habits, it is important to analyse whether cigarettes and tobacco are associated with the development of various lung diseases. This investigation demonstrated the toxic effects of metallic compounds in nearly all forms of pulmonary disease. Metal concentration levels in smokers were higher than in nonsmokers, indicating the effect of smoking on lung diseases. This study indicates that former smokers have higher metal concentrations in their blood compared to patients who currently smoke. Metal levels in the control group were identified to be higher than the standard. Therefore, it is concluded that regardless of smoking habits, occupational and environmental factors might be responsible for metal toxicity. The present study provides the first data on metal toxicity in patients with lung diseases in Chattogram Division, Bangladesh. More research should be done on the toxic metal contamination and its interactions in the human body. Further pathological, clinical, functional, radiological and other investigations are needed to draw a concrete conclusion on metal toxicity and its progression.

Acknowledgements

The authors would like to acknowledge the authority of the Medicine Department of Chattogram Medical Hospital for their cooperation in collecting samples from pulmonary disease patients admitted to Ward No.13. The authors are also grateful to the Department of Chemistry, University of Chittagong for providing laboratory facilities and assistance in sample analysis, and to Berger Paints Bangladesh Ltd. for their financial support to the Department of Chemistry for research and development.

Conflict of Interest

We declare no conflict regarding the publication of the study.

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