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Trace Elements in Urban Particulate Matters: Variations in Serum Levels, Inhalation Bioaccessibility, Health and Disease Effects

Emmanuel Gbenga Olumayede, B. Babalola and I. Oghenovo

Abstract

Trace elements-bound to particulate matters are often become entrained in human respiratory airway, deposited in human nasal cavity and made available for absorption by human tracheobronchial. It has been assumed that variability and bioaccessibility of elements in the serum correlate with some health and diseases. This chapter is a summary of previous works on bioaccessibility of trace elements bound to inhale particulates using different kinds of simulated body fluids. Presented also are evidences of serum variation in some respiratory diseases, such as chronic obstructive pulmonary disease (with or without hypertension), emphysema, bronchiectasis and bronchial asthma, non-tuberculous mycobacterial (NTM) lung disease, idiopathic pulmonary fibrosis (IPF).

Keywords: trace elements, particulate matters, inhalation bioaccessibility, respiratory fluid, health effect, disease

1. Introduction

Since the industrial revolution, a considerable increase in air pollution has been noted. According to a World Health Organization air quality report [1], inhalation of trace elements bound to airborne particulates is worsening air pollution in cities of the world, thereby causing more than 2 million premature deaths annually. In urban centers, particulate matters are major pollutants in the atmosphere, as they present health risk to dwellers. Urban particulates are known for their heterogeneous mix with diverse natural and anthropogenic origins. The composition can vary depending on geographical location, resuspended soil, atmospheric deposition and sources, which include traffic related particles such as metallic components, eroded road pavement, building construction and demolition, and power generation [2, 3]. The mean daily concentration of PM of $\leq 10 \mu\text{m}$ in diameter (PM_{10}) ranges from $<10 \mu\text{g}/\text{m}^3$ to $200/\text{m}^3$ [4]. In 2002 the USEPA reported a range of maximal city concentrations of $25\text{--}534 \mu\text{g}/\text{m}^3$ [5]. These toxic contaminants originated mainly from the anthropogenic emission sources, through ubiquitous applications of elements in urban centers including automobile, industries and domestic fuels combustion [2].

Quite a lot of researchers have investigated elemental compositions of suspended particulate matters in cities worldwide [4–9]. In most of these studies, elevated levels of trace elements have been observed in atmospheric suspended dust in most cities. For example, Okunola *et al.* [8] reported the presence of Cd, Cr, Ni, Pb, Cu, and Zn in atmospheric settling dust in Kano metropolis of Nigeria. Meanwhile, Mafuyai *et al.* [9] reported that the concentrations of some trace elements were found to be far above the standard limits prescribed by WHO for respirable dust in Jos, Nigeria. Therefore, urban dwellers are exposed to considerable amounts of these elements through inhalation of airborne particulates.

Once inhaled, these particles are deposited in the lung and thereby cause serious health effects. Ruby *et al.* [10] reported that more than 80% of the binding mass of particles smaller than 2.5 μm reaches the pulmonary alveoli, where a small fraction is deposited and can stay for months to years. Zwozdziak *et al.* [11] has also observed that elements deposition in human respiratory tract decreases with increase depth. Recognizing that dissolution of inhaled particulate-bound metal in the body has been observed to depend on the ability of such metal to be solubilized in body fluids [8], therefore it is only such soluble fraction of the elements which can be taken across the cell membrane through lung pathway that have direct effects on health. Hence, it is important to assess the bioaccessibility of trace elements bound to inhaled particles over total metal concentration in particle's matrix.

In this chapter, we aimed to discuss the fates, mechanism of toxicity, and recent trends in assessment of bioaccessibility of trace elements. Attempt was made to understand influence of serum levels on trace elements in some respiratory disorders such as chronic obstructive pulmonary disease (COPD), bronchial asthma. This presentation will not consider routes of exposure other than inhalation of particulate matters.

2. Trace elements

Trace elements are elements present in natural materials at concentration of $<1000 \text{ mgkg}^{-1}$ [11]. Some of them are essential micronutrients that exist in very low concentrations in the body, forming 0.01% of the total body weight [12] while others are classified as non-essential. Generally, the major trace elements in atmospheric dust are: iron, manganese, zinc, vanadium, chromium, nickel, copper, cobalt, lead, cadmium, mercury.

2.1 The roles of trace elements in biological processes

Some trace elements are essential for human body; for cell metabolism regulation, including activation or inhibition of enzymatic reactions, and regulation of gene and membrane functions.

Many enzymes have trace elements within their structures and these trace elements act as a cofactor to them [13]. These enzymes play important roles in protection of the body by their activatory or inhibitory and antioxidant activities, with defense system molecules in diseases. For example, Iron is an important constituent of succinate dehydrogenase as well as part of heme of the haemoglobin, myoglobin and the cytochromes [14]. Zinc is involved in carbonic acid (Carbonic anhydrase) and in alcohol (alcohol dehydrogenase) formation, and in proteolysis (Carboxypeptidase, leucine, aminopeptidase etc) [15]. Copper is present in many enzymes involved in oxidation (tyrosinase, ceuloplasmin, amino oxidase, cytochrome oxidase) [16]. Changes in the levels of these trace elements decrease the

efficiency of the antioxidants systems and lead to hyper-reactivity and inflammation in the respiratory tract [17, 18].

Although, trace elements play important roles in various physiological processes and are crucial for functioning of the immune system. However, excessive accumulation or deficiency of some of these elements in human body may be associated with metabolic disturbance, tissue damage and infectious diseases.

3. Sources of particulate matters and trace elements in urban atmosphere

Human activities have been found to contribute more to environmental pollution due to the everyday manufacturing of goods to meet the demands of the large population [19]. Particulate matters in the environment emanate from two main sources: (i) Environmental sources: this include processes like forest fires, marine water sprays, and volcanic emissions, and (ii) Human-derived sources include a variety of largely industrial sources, like cement and metals manufacturing, incinerators, power plants, refineries, smelters, and vehicular exhaust and dust. Include volcanic products, minerals which occur naturally in the environment Anthropogenic activities such as Oil, natural gas production, petroleum utilization, combustion products (ie, lead in gasoline), manufacturing/industrial wastes and byproducts; commercial products (ie, lead paint in houses), or spills thereof (ie, commercial chemicals), municipal waste incinerators, landfills, sewage sludge disposal etc. **Figure 1** illustrates the cycle of trace elements in atmosphere of urban centers.

Meanwhile, trace elements in the atmosphere originate mainly from anthropogenic emission sources, through ubiquitous applications of elements in urban centers including automobile, industries and domestic fuels combustion [20]. Trace elements emitted in wind-blown dusts are mostly from industrial areas. Some important anthropogenic sources which significantly contribute to the atmospheric pollution in urban centers include automobile exhaust which releases lead; smelting which releases arsenic, copper and zinc; insecticides which release arsenic and burning of fossil fuels which release nickel, vanadium, mercury, selenium and tin. Other metals reported on the particles are iron (Fe), Zinc (Zn), and Nickel (Ni), and recently with the use of the catalytic converters an increase in the presence of Platinum (Pt), Paladium (Pd) and Rhodium (Rh) in the particles inhaled has been observed.



Figure 1. Cycling of trace elements in the urban atmosphere. Source: <http://doi.org/10.1016/j.scitotenv.2019.13447>.

4. Routes of exposure and safety limit of some trace elements

For a better understanding of the significances of trace element in human health, it is important to have some knowledge of their routes of exposure. Human are exposed to trace elements in the environment through different routes including ingestion, inhalation of dusts, gases, aerosols and dermal absorption (through skin). The main routes of exposure to trace elements bound to particulate matter (PM) in urban centers include occupational exposure through activities listed below for some specific elements such as:

4.1 Cadmium (Cd)

Cd is an environmentally widespread toxic element. It is classified as a group I carcinogen by IARC (International Agency for Research on Cancer) and has been associated with lung cancer [21]. The modes of human exposure are contamination food, drinking water, occupational or by inhalation in polluted air. Occupational exposure to cadmium primarily takes place in industrial factories such as zinc smelters, battery manufacturing and metal-recovering factories, cadmium-refining companies, production units for paint and pigment. The threshold safety cadmium exposure level has been set at 2.5 $\mu\text{g}/\text{kg}$ body weight per week [21]. Cadmium (Cd) exposure is known to induce pulmonary damage such as emphysema and lung cancer [22].

4.2 Lead (Pb)

Worldwide, lead in atmosphere originates from human activities following its uses as; gasoline additive, paints, cosmetics, ceramic glaze, etc. [23]. Lead enters the human body by ingestion or inhalation. According to the WHO-OSHA, the established safety standard for blood lead in workers is 40 $\mu\text{g}/\text{dL}$. However, it has been suggested that the criterion for elevated blood levels in children is too high in adults therefore recommended a new set of guidelines levels $>15 \mu\text{g}/\text{dL}$ [24].

4.3 Manganese (Mn)

Atmospheric Manganese originated from gasoline additive, methylcyclopentadienyl manganese tricarbonyl (MMT) is a putative modulator of dopamine biology (the primary target of Mn neurotoxicity) [25].

4.4 Chromium (Cr)

Chromium is widely used in the industry for the production of stainless steel, chromium plating, and spray-painting. According to World Health Organization (WHO) [26], the long term exposure of Cr (VI) levels of over 0.1 ppm causes respiratory problems, liver and kidney damage, and carcinogenicity. According to epidemiological studies, the hexavalent form [Cr (VI)] of this metal, appears to be drastically toxic and carcinogenic, thus it has been classified as carcinogenic to humans by the IARC [27].

4.5 Aluminum (Al)

Aluminum and its compounds [28] are released into the atmosphere during activities such as aluminum mining, processing, production and recovery. The skin,

nose, lung and gastrointestinal tract is a route for the uptake of aluminum in the body [29]. Therefore, people close to industrial areas may be exposed to aluminum through inhalation of airborne particulates.

4.6 Arsenic (As)

Elemental arsenic is a metalloid that exists in valency states; trivalent As^{III} , pentavalent As^V in the environment. The main sources of exposure to arsenic include; occupational, environmental and medicinal sources. The safety level of arsenic has been lowered from 50 ppb to 10 ppb by United State Environmental Protection Agency [30]. The presence of arsenic in airborne particulate matter is considered a risk for certain diseases. All the potential pathways of its exposure seem to have adverse effect on human health [31]. Arsenic exposure has been repeatedly associated with lung carcinogenesis [32].

4.7 Vanadium

Vanadium is a major transition element that is released primarily by the burning of fossil fuels, including petroleum, oil, coal, tar, bitumen, and asphaltite. Among Vanadium compounds, Vanadium pentoxide is highly toxic [33]. The IARC classified it as a possible carcinogen to humans (Group 2B) in 2003 [34].

4.8 Zinc

Occupational studies of workers exposed to zinc by inhalation (usually in the presence of other trace elements such as copper, lead, arsenic, and chromium) have not implicated zinc as a risk factor for cancer [35].

5. Behavior, fate, and effects of trace elements in the respiratory tract

The fate and behavior of trace elements in respiratory tract are fundamental to understanding of their health effects and in recent time has become a key aspect of potential health risk assessment.

5.1 The respiratory tract and deposition of PM in the lung

Particulate matters are inhaled during breathing. Upon inhalation, deposition of the particles in the lung may occur through five different mechanisms: sedimentation (gravity), inertial impaction, interception (particle-surface contact), electrostatic deposition, and diffusion. These mechanisms generally occur in different regions of the respiratory tract [36, 37]. Human respiratory tract can be divided into the upper respiratory region (nasal airway, pharynx and larynx), the lower respiratory region (trachea and bronchi) and the alveolar region. **Figure 2a** shows the particle size distribution in human respiratory tract. Meanwhile **Figure 2(b)** illustrates the health risk of trace elements and bioaccessibility questions. The extent of particle deposition in the lung is determined by the physicochemical properties of the particles, such as size, shape, density, and surface chemistry [38] (see **Figure 2a**). Breathing conditions, like ventilation rate, mouth or nose breathing, and airway geometry are other factors that affect particle deposition [39]. The transportation of particles into the lung can be explained by their aerodynamic diameter [40]. Meanwhile, materials with an aerodynamic diameter below $5\ \mu\text{m}$ are predominantly deposited in the alveolar regions of the airways [41].

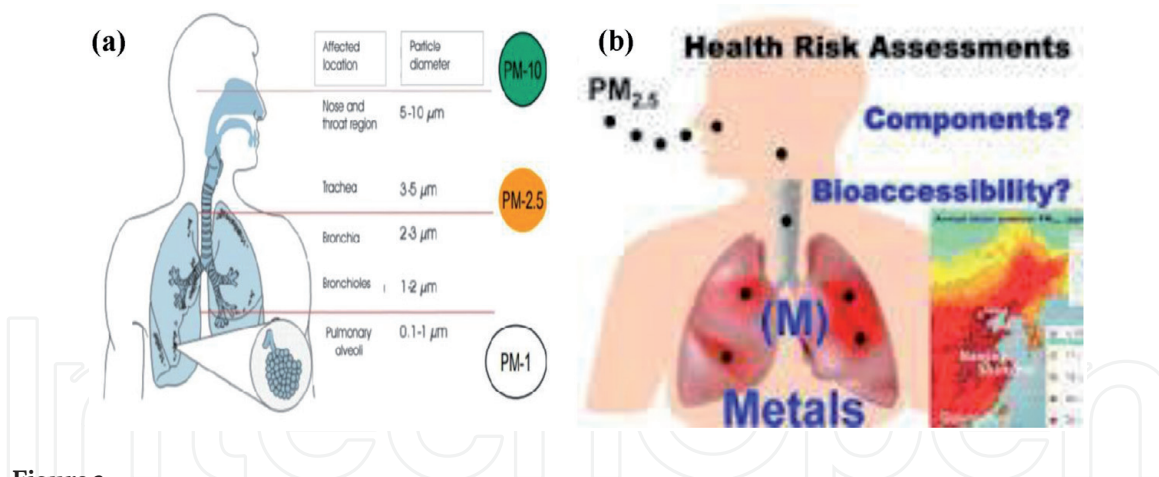


Figure 2. (a) Dust particle sizes distribution in human respiratory tract (b) human health risk and bioaccessibility questions.

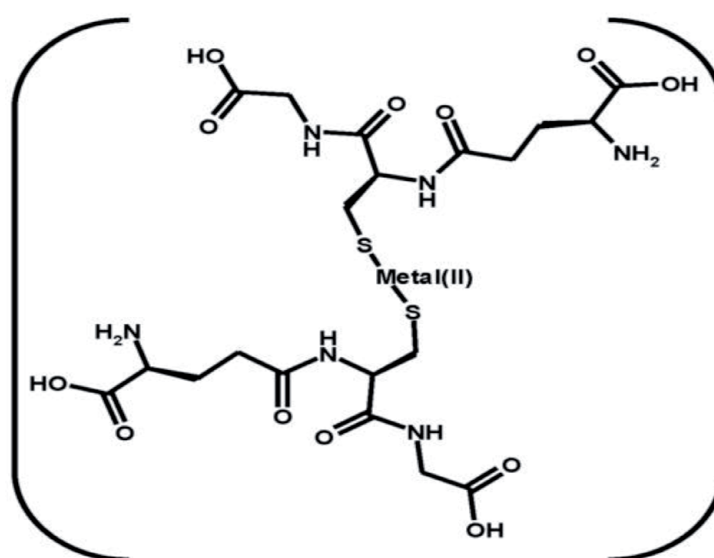


Figure 3. Glutathione-trace element complex.

When trace elements are absorbed through respiratory tract, it is transported in blood bound to metallothionein [42]. **Figure 3** shows an example of such complex, where they form complex with glutathione. This is then followed by alteration of homeostasis [43], thus directly increasing the oxidative stress and lipid peroxidation.

5.2 Mechanisms of inhaled trace elements toxicity

A primary mechanism for most trace elements toxicity is their effects on cells which has been ascribed to the oxidative stress promoting actions, as observed in *in vivo* [44] and most importantly, the inactivation of enzyme systems by binding to sulfhydryl groups [45] of proteins. The mechanisms of their actions include genetic change reactions; reactive oxygen free radicals and adduct formations, oxidative stress, and inflammation [46].

5.2.1 Reactive oxygen species (ROS) generation

Reactive oxygen species (ROS) such as superoxide, hydroxyl radical, nitric oxide radical are byproducts of metabolic processes. External substances such as smoke,

cigarette, pesticides and inhalation of trace elements-bound particulate matters can also cause the formation of free radicals in the body. Trace elements in particulate matters have been reported to cause oxidative stress. For example, pentavalent form of vanadium is reported to cause ROS generation, thus induce oxidative stress, DNA damage, and activation of hypoxia signaling [47]. Oxidation stress is a phenomenon caused by an imbalance between production and accumulation of oxygen reactive species in cell and tissues and the ability of a biological system to detoxify these reactive products [48]. Cadmium causes liver damage mainly by induction ROS inducing lipoperoxidation via Fenton reaction [49]. The increment of ROS induces DNA damage, proteins oxidation and lipid peroxidation. Copper ions are well suited to facilitate formation of ROS that can damage biomolecules, including DNA and chromatin.

5.2.2 DNA adducts formation

The genetic changes reaction of trace elements involves: formation of DNA-protein cross-links, single and double strand DNA breaks [49, 50]. The reaction of elemental ions with nucleic acid lead to a variety of dramatic effects on the nucleic acid structure e.g. crosslinking of polymer strands, degradation to oligomer and monomers, stabilization or destabilization, and the mispairing of bases. For example, Copper can directly bind with high affinity to DNA molecule; this binding can modify the conformational structure of DNA promoting carcinogenesis [51]. Cadmium also produces genotoxicity by the production of DNA single strand breaks and damage and competes for binding at sites (specifically with a zinc finger motifs that are important in gene regulation, enzyme activity, or maintenance of genomic stability [52]).

5.3 Concept of bioavailability and bioaccessibility

In toxicological study, the potential health risks of individual elements bound to inhale particulate matter depend on particle size, inhalability, bioavailability/bioaccessibility, exposure dose and deposition/retention in respiratory tract [53, 54]. Recently, it was emphasized that bio-toxicities of trace metals depend not only on the concentration as expressed by total amount, but also on their geochemical fractions and bioavailability [55]. Bioavailability is the fraction of total elements that can enter the human systemic circulation and exert toxicity on the organs [56]. Meanwhile, bioaccessibility refers to the fraction of contaminant that may become available for absorption e.g., solubilized in the respiratory tract fluid or volatilized into inhaled air and released from the matrix in a topically absorbable form. Bioaccessibility (%) can be defined as the ratio of soluble fraction of trace elements in simulated lung fluids (SLF) to the total concentrations.

5.3.1 Bioaccessibility of trace elements bound to particulate matter

The dissolution of particulate-bound metal in the body has been observed to depend on the ability of such element to be bioaccessible (solubilized) in body fluids after inhalation [57]. Different particulate-bound elemental species behaves differently in human body after inhalation and deposition, depending on their bioaccessibility in lung fluids. In general, high bioaccessible elements are easily taken up by the lung fluids and get introduced to human circulatory system. Recognizing that only soluble fraction of the metals which can be taken across the cell membrane through lung pathway has more direct effects on health. Thus, bioaccessibility of trace elements bound to inhale particles over total metal concentration in particle's matrix is being considered important for assessment of the overall health risk associated with inhalation of particulate matters.

Lung Fluids	BCR-723						
	Trace Element						
	Cd	Cr	Cu	Mn	Ni	Pb	Zn
PBS	<LD	0.8 ± 0.5	4.1 ± 1.5	0.9 ± 0.0	<LD	<LD	6.8 ± 0.8
Gamble's	<LD	0.5 ± 0.3	49.9 ± 5.6	1.7 ± 0.0	0.8 ± 0.0	7.8 ± 0.6	44.6 ± 0.8
ALF	81.4 ± 7.6	8.7 ± 0.0	65.2 ± 3.7	5.5 ± 0.1	24.1 ± 3.7	62.0 ± 3.2	76.8 ± 2.2
NIST2710							
PBS	44.2 ± 21.2	7.8 ± 0.0	8.3 ± 0.2	28.7 ± 0.4	<LD	0.04 ± 0.00	6.2 ± 0.1
Gamble's	86.0 ± 2.8	<LD	47.6 ± 1.4	40.1 ± 0.7	<LD	7.8 ± 0.4	23.7 ± 0.1
ALF	85.3 ± 8.4	<LD	59.7 ± 1.4	44.3 ± 0.2	<LD	55.0 ± 0.5	35.3 ± 0.1
NIST 1648							
PBS	24.1 ± 6.2	1.3 ± 0.4	7.3 ± 1.8	16.4 ± 1.4	<LD	<LD	4.3 ± 0.2
Gamble's	45.2 ± 4.0	2.7 ± 1.0	49.9 ± 2.7	29.6 ± 0.2	3.3 ± 1.2	9.1 ± 0.9	43.2 ± 0.2
ALF	65.6 ± 5.5	8.7 ± 0.9	55.0 ± 1.1	46.8 ± 2.6	12.2 ± 4.1	75.9 ± 2.2	66.2 ± 2.3

LD, Limit of detection.

Table 1. Bioaccessibility (%; mean ± SD; n = 3) values of trace elements in the three lung fluids (adopted from [62]).

Emerging studies [58–60] have shown risk assessment using bioaccessibility presents better understanding of the fate of trace elements upon inhalation by children and adults. However, one of the challenges for environmental toxicologist has been development of fluid with properties similar to human tracheobronchial fluids, so as to enable systematic investigation into bioaccessibility and lung deposition of particles in respiratory tracts [61]. Several fluids have been explored to mimic human respiratory tract fluids in investigation of trace elements bioaccessibility. These range from the traditional Gamble's solution to simulated artificial lung fluids (SALF), which is simply a modification of Gamble's solution.

In one of such previous study, [62] reported that pulmonary bioaccessible fraction of Pb and Cd were relatively high (69 and 74% respectively) when lung stimulating solution (artificial lysosome fluid, ALF) was used to extract fine particles. Similarly, [63–67] reported higher bioaccessibility for Cd ($88 \pm 6.4\%$ for PM_{10} and $91 \pm 6.6\%$ for $PM_{2.5}$) when ALF was used as extraction fluid compared to Gamble's solution. Tang *et al* [64] reported that As, Pb, V and Mn showed higher inhalation bioaccessibility extracted by the artificial lysosomal fluid (ALF); while V, As, Sr. and Cd showed higher inhalation bioaccessibility using the simulated lung fluid (SLF), suggesting differences in elemental inhalation bioaccessibility between ALF and SLF extraction. **Table 1** presents the bioaccessibility values of trace elements in the three lung fluids in different reference materials, as reported by [62]. In general, one of the important factors affecting bioaccessibility of trace elements is the influence of fluid's composition and pH.

6. Variation in serum trace elements levels and induced respiratory tract diseases and health problems

Inhalation exposure to trace elements can have significant health impacts on urban dwellers and nearby workers. Unlike other organs, lungs are directly and continuously exposed to high oxygen concentrations, exogenous oxidants, and pollutants: thus, they have the greatest susceptibility to oxidative stress and pollutant toxicity. The existence of concentration gradient within the lung and inter-individual concentration differences reveals the existence of two groups of elements: (i) homogeneously distributed over the lung e.g. elements Br, Cs, Cu, K, Na, Rb, Se and Zn, and (ii) heterogeneously distributed e.g. elements such as Cd, Co, Cr, Pb, Sb, Sc and V [68].

The enrichment of trace elements in the lung tissue is known to result a number of lung diseases. These diseases have been associated with disturbance of trace elements balance [69]. Here, we discussed recent observations on variation of serum levels in diseases such as chronic obstructive pulmonary disease (with or without hypertension), emphysema, bronchiectasis and bronchial asthma, non-tuberculous mycobacterial (NTM) lung disease, idiopathic pulmonary fibrosis (IPF).

6.1 Chronic obstructive pulmonary disease (COPD)

Many trace elements have activator or inhibitory roles in the antioxidants defensive mechanism in diseases. Recent study [70] showed that serum levels of Co, Cu and Fe were higher in COPD patients with pulmonary hypertension compared to COPD patients without pulmonary hypertension. Similarly, [70] reported that the serum copper (Cu) in COPD patients were higher than the control group.

6.2 Bronchial asthma

Bronchial asthma is a chronic inflammatory disease of the respiratory tract with an unknown etiology where inflammation is often associated with an increase

generation of ROS [71]. Several trace elements are known to be capable of causing bronchial asthma, such as nickel (Ni), Chromium (Cr), Cobalt (Co) etc. **Table 2** presents the variations in concentrations of some trace elements (Zn, Cu and Se) in serum of asthmatic, as observed in a study [66]. The results showed higher Cu concentration, and Cu/Zn and lower Cu/Se ratios.

6.3 Idiopathic pulmonary fibrosis (IPF)

Idiopathic pulmonary fibrosis is an interstitial lung disease with poor prognosis and an undefined etiopathogenesis [72] leading rapidly to death. It is the most common lung disease with estimated incidence of 2.8–9.3% per 100,000 per year in Europe and America [73]. Particulate matters bound trace elements deposited in the lung may give rise to more or less marked pulmonary fibrosis, depending on intrinsic properties and amount of the particulate matters. Oxidative stress by trace elements contributes to alveolar injury and fibrosis development in patients. A study [74] reported that IPF patients had significantly increased sputum levels of Cd, Cr, Cu and Pb respect to control. **Table 3** presents the variations in concentrations of some trace elements in serum in patents with NTM, TB and healthy as control, as reported by [74].

Mean ± SD	Control (n = 25)	Patient
Zn (µg/mL)	0.83 (0.14)	0.68(0.09)
Cu (µg/mL)	0.76(0.17)	1.10(0.28)
Se (µg/mL)	0.116 (0.022)	0.0057 (0.024)

Table 2.
Variation of trace elements in serum of asthmatic patients [66, 67].

Element (µg/L)	Patients with NTM (n = 95)	Patient with TB (n = 97)	Healthy control (n = 99)
Co (µg/L)	0.24(0.20–0.35)	0.54(0.22–0.83)	0.23(0.19–0.27)
Cu (µg/L)	109(97–134)	129(111–153)	91(82–102)
Cr (µg/L)	0.23(0.19–0.27)	0.23(0.18–0.27)	0.23(0.19–0.28)
Mn (µg/L)	0.90(0.81–1.07)	0.93(0.71–1.31)	0.92(0.80–1.23)
Se (µg/L)	105(95–116)	108(99–119)	115(105–123)
Zn (µg/L)	94(84–107)	84(75–93)	102(92–116)

Table 3.
Serum levels of trace elements in patents with NTM, TB and healthy [75].

Element (µg/mL)	Patient	Control
Cd (µg/mL)	110	54
Cu (µg/mL)	330	635
Pb (µg/mL)	1217	1444
Mn (µg/mL)	399	522
Se (µg/mL)	1496	1443
Zn (µg/mL)	2515	2699

Table 4.
Serum levels of trace elements in patents with Haemodialysis compare with control [76].

6.4 Non-tuberculous mycobacterial lung diseases (NTM)

Non-tuberculous mycobacterial lung diseases are emerging cause of pulmonary infection and are becoming more common in the clinical setting. A recent study [75] showed that serum concentration of copper and molybdenum (**Table 4**) were higher in patients with NTM lung disease (109 vs. 91 $\mu\text{g/dL}$, $p < 0.001$ and 1.70 vs. 0.96 $\mu\text{g/L}$, $p < 0.001$). In contrast, the media serum concentrations of Selenium and Zinc were significantly lower in patients with non-tuberculous mycobacterial lung diseases than in healthy control (105 vs. 115 $\mu\text{g/L}$, $p < 0.001$ and 94 vs. 102 $\mu\text{g/dL}$, $p < 0.001$).

6.5 Haemodialysis

Oxidants-antioxidants balance is essential for the normal lung function. Both, an increased oxidant and/or decrease antioxidant may reverse the physiologic oxidants-antioxidants balance, leading to lung injury. Available data (**Table 4**) suggested that the levels of Cd, Cr, Pb, and V were higher and the levels of Se, Zn and Mn were lower in hemodialysis patients compare with controls [76].

6.6 Parkinson disease

Parkinson disease, also known as manganism is an extrapyramidal neurological disease characterized by rigidity action tremor, bradykinesia, memory and cognitive dysfunction that occurs in workers exposed to airborne Mn. The element (Mn) in blood crosses the blood brain barrier and accumulates inside the neuron disrupting the synaptic transmission and inducing glial activation [77].

7. Conclusion

Trace elements bound to particulate matter could be trapped and deposited along the nasal cavity through inhalation of air-borne particulate matter. In this chapter, we attempted to understand influence of serum levels and bioaccessibility of trace elements in some respiratory fluids. Our investigation provides evidence that enrichment of trace elements in the lung tissue is known to result a number of lung diseases, such as chronic obstructive pulmonary disease (with or without hypertension), bronchial asthma, non-tuberculous mycobacterial (NTM) lung disease, and idiopathic pulmonary fibrosis (IPF). The findings suggest that serum Cu were higher in asthmatic patients and COPD patients than the healthy. Meanwhile, the levels of Se, Zn and Mn were lower in hemodialysis patients and non-tuberculous mycobacterial lung diseases than in healthy control.

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