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Occupational Exposure to Cadmium and Its Compounds: A Review on Health Effects and Monitoring Techniques

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Abstract

Background: Cadmium is a hazardous metal prevalent in various industries, posing significant health risks to workers. This article examines the health dangers of cadmium in indoor and outdoor environments as well as discusses methods for sampling and analyzing cadmium-containing compounds.

Material and Method: This study involved a literature search in databases including Scopus, PubMed, Web of Science, and Google Scholar, focusing on articles published between 2015 and 2023. Keywords included "Health," "Hazards," "sampling technique," "Instruments," "Monitoring," and "Cadmium." The inclusion criteria targeted studies investigating health effects and monitoring techniques published in English or Persian.

Results: Out of 341 identified articles, 84 were selected: 51 had focused on health effects, while 33 had addressed monitoring methods. Most studies had examined cardiovascular effects, with fewer on neurological impacts. Five studies had investigated biological monitoring, and three air monitoring. Cadmium exposure can severely affect the respiratory system, kidneys, and bones, leading to conditions such as asthma, kidney failure, and osteoporosis. Many countries have set the occupational exposure limit for cadmium at 0.01 mg/m³, highlighting the need for worker protection. Filters emerged as the primary medium for cadmium adsorption.

Conclusions: High cadmium exposure is linked to acute and chronic health issues. According to studies, elevated air levels of cadmium pose serious health risks, particularly in industrial settings. Improving the accuracy of sampling methods and utilizing safety equipment such as respirators and ventilation systems are crucial in preventing exposure limits from being surpassed. Continuous monitoring of air pollution levels is also essential.

Keywords: Cadmium, Occupational Exposure, Biomonitoring, Prevention

Introduction

Cadmium is an element with the CAS registry number 7440-43-9 and atomic number 48 in the periodic table. Isotopes 114Cd and 112Cd are the most common forms found in nature. The atomic mass of cadmium is 112.4 Daltons. Classified as a transition metal in Group 12 (2-B) of the periodic table, cadmium exclusively presents an oxidation state of +2. Cadmium is a malleable metal with a silver-white appearance and a relatively low melting point of 327°C. Despite its susceptibility to corrosion, cadmium is highly resistant owing to the protective oxide layer on its surface, resulting in a standard reduction potential of -0.40 V. When cadmium compounds come into contact with mineral acids, they

dissolve and generate flammable hydrogen gas. Also, cadmium particles react with hydrogen azide, sulfur, selenium, tellurium, and oxidants, posing a fire and explosion hazard. Chemically and physically, cadmium shares many similarities with zinc and is often found in conjunction with zinc in different minerals. It is commonly derived as one of the byproducts of zinc smelting and the smelting of certain lead ores. The common compounds of cadmium are formed by reaction with acetate, sulfide, sulpho-selenide, stearate, oxygen, carbonate, sulfate, and chloride [1,2].

Cadmium metal is soft, strong, flexible and preferentially white to blue. The most common form of oxidation is cadmium 2+, but there are also some examples of 1+ oxidation. Given its structural properties, cadmium is used as an electrical conductor in batteries and plating process. This element as well as the solutions of its compounds are toxic and must be used under safe conditions [3]. Cadmium is considered a chemical pollutant in the environment and occupational settings. Today, with advances in technology and industrialization, we are witnessing growing usage of cadmium and, as a result, its widespread release into the environment. This poses a serious risks to population health and ecosystem cycles [4-6].

Cadmium is typically found in soil and minerals, such as salts, sulfides, sulfates, carbonates, chlorides, and hydroxides. It can also be present in water. With the increase in industrial activities, the level of exposure to cadmium from water, air, and soil has grown, resulting in both permanent and temporary health disorders. In addition, consumption of cadmium-contaminated food, alcohol, and smoking can elevate Cd levels in blood and urine [7-10]. Cadmium is present in varying amounts in certain plant and animal sources, such as cocoa, peanut butter, soybeans, potatoes, pomegranate, sunflower seeds, fish, and meat. It is also found in the liver and kidneys of animals, with levels reaching up to 1000 ppb [11]. Tobacco plants accumulate high concentrations of cadmium, particularly in their leaves [12]. Cadmium in tobacco could be the cause of the heightened concentration of cadmium in the blood of smokers. Batáriová et al. found that the cadmium level in the blood of smokers and non-smokers was 1.3 and o.4 µg/L, respectively [13].

The International Cadmium Association (ICdA) has published ranges for expected levels of trapped cadmium in particulate form in rural, urban, and occupational settings as 0.1-5 ng/m3, 2-15 ng/m3, and 150-15 ng/m3. It demonstrates that for workers exposed to cadmium, urine and blood cadmium levels should be checked regularly [11]. According to the world's authoritative sources, including the Joint Expert Committee for Food Additives (JECFA) and the Food and Agriculture Organization (FAO), the permissible intake of cadmium from food and other agricultural products is 25 µg/kg bw/month [14]. The International Agency for Research on Cancer (IARC) has classified cadmium as definitely carcinogenic for humans [15].

The history of environmental pollution caused by cadmium dates back to 1946. Studies have indicated that these people suffered for years from chronic poisoning from the usage of rice made from cadmium-contaminated peppers, known as Itai-Itai disease [4, 16]. Symptoms of the disease included renal failure, excruciating bone pains, and anemia [16]. Workers who were exposed to cadmium compounds experienced similar symptoms [17-20]. Great efforts were undertaken to lower the emission of cadmium into water, soil, and air once it was discovered that cadmium was the primary cause of Itai-Itai sickness [21].

According to a review of earlier studies, cadmium and its compounds are hazardous to humans and have detrimental effects on various body systems, including the skin, digestive system, respiratory system, kidneys, and bones [22]; These effects include an increase in protein excretion through the urine and a decline in bone mineral density. [23]. Epidemiological studies have revealed that even low levels of exposure to cadmium in the environment pose a serious risk to public health, causing damage to the kidneys, liver, skeletal and cardiovascular systems, as well as impaired vision and hearing [6, 24-35]. Moreover, current exposure to cadmium appears to be associated with an elevated risk of developing lung, breast, prostate, pancreatic, endometrial, and nasopharyngeal cancers [36-43]. Cadmium and its compounds are categorized by the International Agency for Research on Cancer (IARC) as Group 1, indicating that there is ample evidence to suggest that these substances can cause cancer in humans [15].

The aim of this review article is to highlight the health risks of cadmium to human health, both in non-occupational and occupational settings. It also intends to explore different methods for sampling and analyzing cadmium compounds. The review will provide objective information on cadmium exposure in various environments to determine the levels of exposure. It will also discuss strategies for reducing emissions and minimizing cadmium exposure.

Materials and Methods

Studies Search: In this study, eight reputable databases—Google Scholar, Scopus, PubMed, Embase, Web of Science, Magiran, SID, and Iran Doc—were employed to review articles related to the research. Research articles published in Persian and English in these databases from 2015 to 2023 were extracted. The keywords «Health» ،«Hazards» ،«sampling technique» ، «Instruments» ،«Monitoring» and «Cadmium» were

applied to search for articles in the mentioned databases. Studies Selection: The authors independently reviewed the search results and screened eligible articles for full-text evaluation. Inclusion criteria encompassed all studies that had specifically examined the health effects and monitoring methods of cadmium in Iran and globally from 2015 to 2023. Exclusion criteria were non-research articles such as author notes, editorials, general texts, letters to the editor, and articles not written in Persian or English.

Results

The preliminary search yielded 341 studies across five databases. Following the elimination of 112 duplicate cases, the screening process was conducted on 229 studies (Fig. 1). Ultimately, following four stages of screening, a total of 84 articles progressed to the final review of the results.

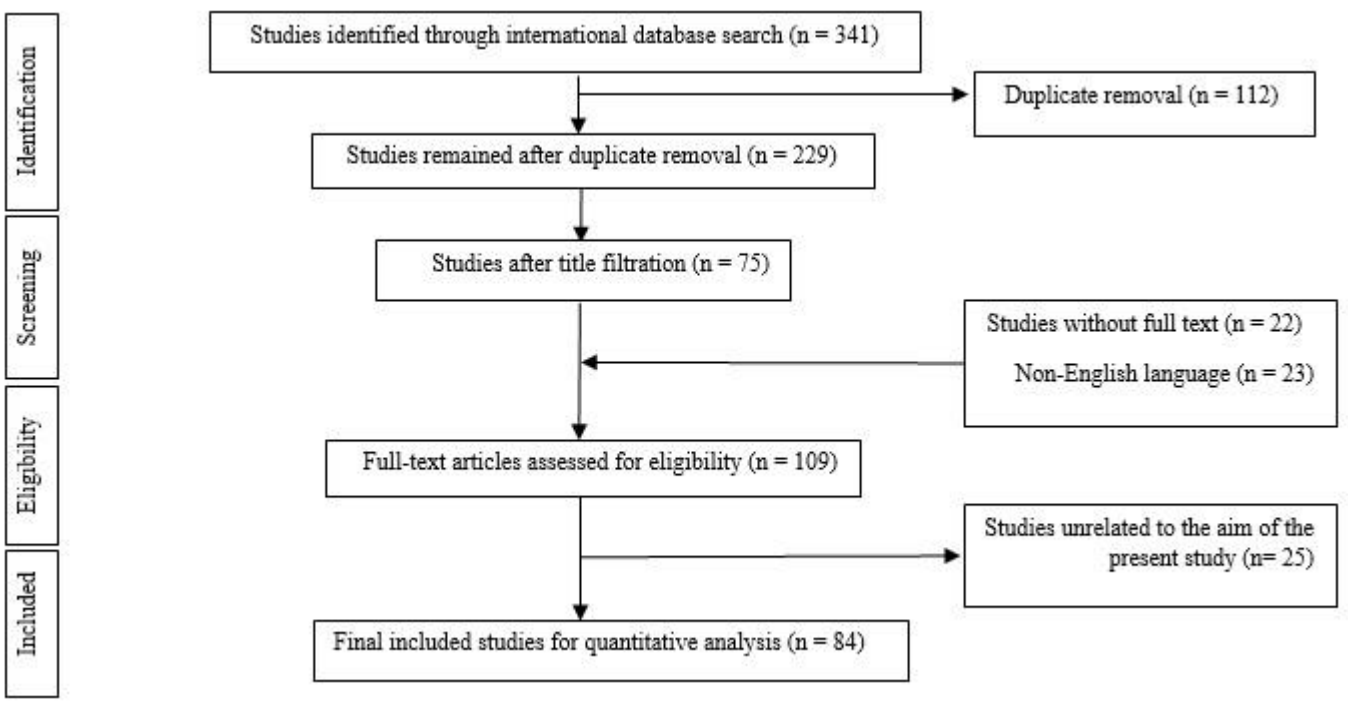

Fig. 1. Flowchart of the included studies

Occupational Exposure: The primary industrial application of cadmium is in the manufacturing of nickel-cadmium batteries, accounting for approximately 80% of cadmium consumption. Electroplating is commonly used on surfaces made of iron and steel. It is also utilized in paint pigments and as a heat stabilizer in polyvinyl chloride plastics. Further, cadmium can also be found as an impurity in cement, phosphate fertilizers, and other metals such as zinc, lead, and copper [44].

Exposure can occur through inhalation of dust, fumes, or vapor containing cadmium in various industries such as metal smelting, battery production, and paint manufacturing, as well as dermal contact with products and materials containing cadmium [37]. Contamination of surface and groundwater with cadmium owing to industrial and agricultural activities, accumulation of cadmium in polluted soils, especially in industrial and mining areas, and the subsequent transfer of cadmium into the human body also contribute to exposure. Further, consumption of food items contaminated with cadmium, such as rice, vegetables, seafood, and animal products can result in detrimental health effects in humans [39].

Cadmium chloride has several uses, including as a fungicide, a colorant for pyrotechnics, an additive in tinning solutions, along with a mordant in the dyeing and printing of textiles. It is widely used in electroplating. Production of specific photographic films and the creation of specialized mirrors as well as coatings for electronic vacuum tubes are two minor additional applications [44].

Cadmium oxide is employed as a component in various applications such as silver alloys, phosphors, semiconductors, glass-ceramic glazes, heat stabilizers for polyvinyl chloride plastics, electroplating, and phosphor production. The manufacturing of cadmium alloys, welding, and the cutting of cadmium-coated steel as well as rivets with oxyacetylene all generate cadmium vapors which can be potentially harmful. Moreover, when cadmium-containing metals are melted, refined, and smelted, cadmium is emitted. Cadmium particles can accumulate in food crops and drinking water, particularly at neutral pH levels, and ingestion of contaminated food and water can raise the likelihood of occupational exposure. Menstruating women, those with poor diets, and individuals with iron deficiency have higher rates of gut cadmium absorption and are more susceptible to cadmium toxicity [44].

In lead, zinc, and copper mines, the amount of cadmium is less than 1%, and it is present in the form of cadmium

sulfide. Cadmium metal is produced electrolytically through processing cadmium-containing deposits obtained from ore smelting. Given its lower boiling point (1040.15 K) and higher vapor pressure (400 mm Hg at 983 K) compared to its associated metals, cadmium readily evaporates upon melting and then condenses. Remaining particles from the process are released into the air, where they quickly bind with oxygen to generate respirable cadmium oxiden [45].

In the United States, nearly 4,000 tons of cadmium are used each year, with the majority of these products being pigments, batteries, plastic stabilizers, metallurgy, neutron absorption rods, nuclear reactors, and semiconductors [46]. The National Institute for Occupational Safety and Health (NIOSH) estimates that approximately 1,500,000 workers may be potentially exposed to cadmium, with over 100,000 cases being associated with exposure to specific substances or sectors that utilize cadmium [47]. This includes ore smelting, mist of cadmium-based plating, dehydration (calcination) of cadmium pigments, and transfer of cadmium oxide powder into plastic stabilizers [45].

Exposure route and intake of Cadmium: Humans may be exposed to significant amounts of cadmium because of industrial activities that elevate cadmium levels in water, air, and soil. Smoking increases the amount of cadmium in the blood and urine, which can lead to cadmium exposure. The presence of cadmium in polluted water can disrupt essential bodily mechanisms, potentially causing acute or chronic diseases [7-9]. Cadmium has been classified as a human carcinogen by IARC [15].

Work-related exposures to cadmium can occur through the manufacturing of alloys, batteries, glass, and plating industries. Given the significance of the problem, airborne cadmium levels are frequently monitored in some nations [48]. Cadmium contamination has been identified in rice, cereals, and seafood [49].

Furthermore, a small amount of cadmium is absorbed through oral consumption. The Itai-Itai disease epidemic in Japan was caused by significant levels of cadmium contamination in food and water sources. People who had been exposed experienced painful degenerative bone disease, kidney failure, as well as gastrointestinal and respiratory problems [50].

Industrial dust provides a more effective route for cadmium to reach the lungs compared to poor gastrointestinal (GI) absorption. Inhalation of cadmium, whether acute or chronic, in industrial settings, can cause damage to the lungs and renal tubules. Thus, in the vicinity of industrial areas, smokers have nearly twice the amount of cadmium in their blood compared to non-smokers. This is likely because tobacco plants naturally accumulate relatively high levels of cadmium in their tissues, particularly in their leaves [12].

For workers, cadmium is mostly absorbed through inhaling cadmium-containing fumes and mists in workplaces while for the general population, cadmium is mostly absorbed through oral route and consumption of contaminated food.

In occupational exposure to cadmium, the primary routes of exposure include inhalation of dust and fumes, as well as unintentional ingestion of cadmium-containing particles owing to hand contact with contaminated food and cigarettes [51]. Exposure to cadmium can be minimized through reducing cadmium pollution, implementing occupational health procedures, and utilizing personal protective equipment.

Cadmium particles are released into the ambient air along the extraction of cadmium and subsequent its melting. When cadmium enters the environment, it easily moves through the soil and enters the food chain. Some plants such as tobacco, rice, other grains, potatoes, and other vegetables take up cadmium from the soil. The public can be exposed to cadmium through consuming food and drinking water, inhaling cadmium-containing particles from ambient air or tobacco smoke, or soil and dust pollution. Smokers are exposed to 1.7 µg of cadmium in each cigarette. Food is the main source of cadmium intake for non-smokers. The average cadmium level in American foods is between 2 and 40 ppb. Smokers typically have more than twice as much cadmium in their blood and body as non-smokers [52]. In general, smokers have higher urinary cadmium levels than non-smokers [53].

In summary, occupational exposure to cadmium can take place through inhaling oxide fumes generated during the heating or welding of cadmium-containing materials, as well as through inhaling dust particles from metals, oxides, and pigments. Metal machining, battery manufacturing, electroplating, plastics, ceramics, paint, and welding processes are among the industries with the highest occupational exposures to cadmium [54].

Health effects of occupational exposure: The main target organs for cadmium toxicity include kidneys, lungs, bones, reproductive system, and cardiovascular system. One of the many applications of cadmium is as an anti-corrosion coating on steel. The high level of exposure to cadmium can cause severe pulmonary irritation, acute pulmonary edema, and, in some cases, death. Pulmonary and kidney dysfunction can both arise from chronic exposure to low levels of cadmium [55].

In some occupations (such as welding) after acute exposure and when inhaling cadmium fumes, workers complain of a sore throat, headache, body aches, nausea, and a metallic taste following several hours of rest. This is followed by fever, shortness of breath, and chest tightness, resulting in metal fume fever. Prolonged exposure to cadmium can damage various body organs such as the kidneys, lungs, bones, and circulatory system. Cadmium impairs the functions of the kidney ducts and can cause kidney stones. Bone pain and fractures are caused by the loss of phosphorus and

calcium from the kidneys as well as diminished vitamin D synthesis. Other effects of cadmium exposure include loss of smell, ulceration of the nasal mucosa, yellowing of teeth, and minor anemia [56].

The kidney is an important target organ for prolonged exposure to cadmium. In humans, cadmium mostly deposits in the kidneys and has a biological half-life of 10 to 35 years [57]. This accumulation can result in dysfunction of the abdominal tube, causing increased urinary excretion of proteins with a low molecular mass. While small increases in urinary excretion of these proteins are generally reversible, excessive rises can cause irreversible tubular dysfunction [58].

Excessive intake of cadmium can have negative consequences on bone health, and contribute to kidney stone formation as well as calcium metabolism. Osteoarthritis can occur in those who have been exposed to cadmium throughout their lives or who work in industrial regions. For instance, itai-itai disease, which is characterized by painful bone fractures and kidney damage, took place in lead-zinc mines where the soil in the area was contaminated with cadmium.

Previous studies have indicated that exposure to cadmium is linked to bone diseases, likely because of changes in the metabolism of cadmium, calcium, phosphorus, and vitamin D. Further, high levels of inhaled cadmium oxide fume inhalation can cause acute pneumonitis with pulmonary edema, which can be fatal. Chronic obstructive pulmonary disease (COPD), which is most commonly associated with long-term occupational exposure to high levels of cadmium, results in lung damage [2].

There is ample evidence that exposure to cadmium and its byproducts, such as mist and fumes, can result in lung cancer. Population investigations in a cadmium-contaminated area have suggested an association between cadmium exposure and lung cancer. Epidemiological studies have only provided weak support for the hypothesis that cadmium may contribute to kidney and prostate cancer. Animal studies have provided substantial evidence that cadmium compounds are carcinogenic. According to International Agency for Research on Cancer (IARC), cadmium and its compounds are categorized as Group 1, indicating that there is copious evidence to suggest that these substances can cause cancer in humans. Nevertheless, cadmium has been proven to cause genetic side effects, such as chromosomal malfunction, genetic instability, and epigenetic alterations [59, 60]. Figure 2 summarizes the detrimental effects of cadmium on body organs.

The effects of cadmium toxicity on cells are manifested through its impact on the cellular mechanism of mitochondria. By accelerating the production of reactive oxygen species, increasing lipid peroxidation, as well as depleting glutathione and protein-bound sulfhydryl groups, it has been shown to promote oxidative stress. Further, cadmium increases the production of inflammatory cytokines and inhibits the creation of nitric oxide, which has a protective effect.

Cadmium binds to the abundant protein transporter of transition metal ions and semi-metals, known as metallothionein, after entering the body through the respiratory and digestive systems. Circulating cadmium, which is bound to glutathione and metallothionein, accumulates in the body over time, where a portion of it gets permanently stored in the kidneys. Cadmium toxicity primarily affects the kidneys, specifically the renal tubules. Adsorption of cadmium results in the accumulation in bones due to its similarity to calcium. A small amount of cadmium is eliminated in the urine, primarily as a cadmium-metallothionein complex.

Exposure to cadmium through inhaling fumes causes asthmatic symptoms accompanied by fever and systemic disability, where high levels of exposure can be fatal. The organs most commonly damaged by chronic exposure to cadmium are the kidneys and skeletal system. In cases of acute exposure, the lungs are typically affected [61].

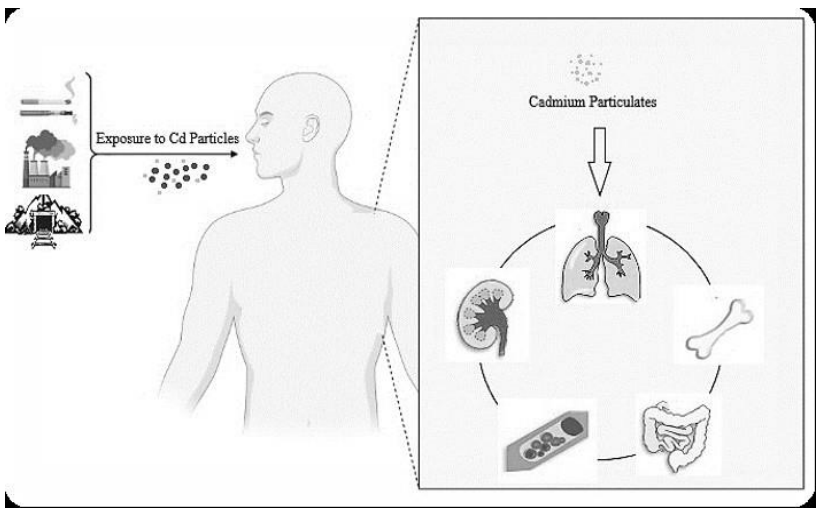

Fig. 2. Sources of cadmium emission and its toxic effects on body organs [61]

Brief description of the diseases caused by cadmium or its compounds: Subjects who are exposed to low concentrations of cadmium for a long time (chronic exposure) manifest symptoms such as stains on their teeth, weight loss, loss of sense of smell, and serious damage to body organs. The inhalation of cadmium fumes has been found to cause significant damage to the respiratory system, leasing to various conditions such as reactive airway dysfunction syndrome (RADS), rhinitis, tracheobronchitis, pneumonia, and pulmonary edema. Chills, fever, and muscle discomfort are among the flu-like symptoms which commonly appear 4 to 10 hours after exposure. Subsequent symptoms may include chest pain, coughing, and difficulty breathing. Furthermore, possible symptoms include labored breathing and coughing up blood. According to estimates, exposure to 5 mg/m3 of cadmium for 8 hours can be lethal, while 1 mg/m3 every 8 hours is considered immediately harmful to life or health (IDLH). Accidental hand-to-mouth contact with hands contaminated with cadmium or with cadmium-contaminated food can result in the ingestion of noticeable amounts of cadmium. Severe gastrointestinal damage takes place at exposure levels to cadmium of lower than 10 mg, while levels above 100 mg cause death [62].

Most cases of acute lung injury heal without causing any permanent effects. Chronic exposure to cadmium fumes and dust can culminate in long-term respiratory effects, specifically chronic obstructive pulmonary disease (COPD) and emphysema. Chronic cadmium poisoning often leads to anosmia, which is the loss of the sense of smell. This is due to the high and strong affinity of cadmium to the olfactory epithelium. Further, cadmium can bind to glutathione and metallothionein in the olfactory epithelium, penetrate the blood-brain barrier, and accumulate in the brain. Based on previous research, there is a significant correlation between olfactory disorders and the concentration of cadmium in blood as well as urine [63].

As mentioned earlier, the kidneys are the primary target organs of cadmium, which is absorbed in the outer cortex of the kidney. In the liver, cadmium binds to metallothionein before being transported to the kidney, where it is subsequently released. Increased excretion of low-molecular-weight proteins, such as retinol-binding protein, 2-microglobulin, and 1-microglobulin, is the primary detrimental effect of cadmium on the kidney. Elevated levels of excretion are thought to indicate early tubule injury since these proteins are typically absorbed by the proximal renal tubule. The critical concentration of cadmium in the renal cortex is approximately 200 ppm, corresponding to a urinary cadmium excretion of about 10 μg/g creatinine. However, individuals who are more susceptible, including those with diabetes, may experience changes in kidney biomarkers at lower levels. Renal effects can even be detected at levels as low as 2 μg/g of creatinine. At a later stage, cadmium causes glomerular injury, causing reduced filtration. Fanconi-like syndrome is a more generalized dysfunction of the proximal tubular cells, which no longer efficiently reabsorb essential soluble components such as phosphates, amino acids, urate, and glucose. This results in tubular proteinuria, as well as excretion of phosphates, glucose, amino acids, and uric acid in the urine. The primary symptom of Fanconi syndrome is bone demineralization (osteomalacia), which is caused by excessive phosphate excretion [64].

Some studies attribute a specific disorder of bone mineralization, excruciating bone pain, and the possibility of vertebral collapse resulting in nerve damage to cadmium as an underlying cause. "Itai-itai" disease, a condition prevalent in Japan over the early 20th century, mainly affected elderly females who consumed a diet primarily consisting of cadmium-contaminated rice and fish. Thus, factors such as gender, age, and inadequate nutrition are believed to contribute to the risk of developing this disease. However, since the condition has not been observed in individuals exposed to cadmium in their occupational environment, there is limited information regarding osteomalacia in the workplace. Nonetheless, in cases where tubular nephropathy is present, alterations in calcium metabolism may contribute to alterations in bone mineral density. Other mechanisms, such as stimulation of osteoclast activity, may also play a significant role. Note that bone lesions typically manifest as a late consequence of chronic environmental cadmium poisoning. These lesions are characterized by osteomalacia, osteoporosis, and spontaneous fractures [65-67]. Further, cadmium and its compounds are carcinogenic, specifically linked to the development of lung cancer. Considering the health effects of cadmium, the set of measures that can be taken to manage cadmium control include reducing emissions of cadmium, particularly into surface waters, from mining, smelting, waste incineration, sewage sludge application, as well as use of phosphate fertilizers and cadmium-containing manure. Another measure is improving working conditions in the non-ferrous smelting industry and disseminating information on the proper use of fertilizers to lower cadmium exposure. Safe disposal techniques should also be developed for cadmium-containing wastes and effluents, along with encouraging washing of fruits and vegetables as well as peeling of roots and tubers to reduce cadmium contamination. Furthermore, safe and effective measures should be promoted to increase recycling of cadmium and restrict non-recyclable uses. Elimination of the use of cadmium in products such as toys, jewelry, and plastics is another important measure. Table 1 indicates health effects of Cd on humans.

Table 1. Health effect of Cd on humans

No.	Body Target	Health Effect	Exposure	Findings	Ref
1	Kidney	Failure in organ functioning, tubular necrosis	Chronic	Linked to renal dysfunction and increased proteinuria	[68-72]
2	Cancer	including lung, prostate, pancreas, kidney, Lungs, larynx, mouth, throat, bladder, liver, stomach, cervix	Chronic	Positive Significant association with lung and prostate cancers.	[73-81]
3	Bone	Itai-Itai, osteoporosis and bone mineral density reduction	Chronic	Associated with decreased bone density and increased fracture risk	[82-87]
4	Cardiovascular and Blood	High blood pressure, cardiac Arrest, atherosclerosis, cholesterol blockage in arteries, heart attack, coronary artery disease, and stroke	Chronic	Associated with increased risk of hypertension and cardiovascular diseases.	[88-95]
5	Liver	Liver injury, fibrosis, and progression to liver cancer	Chronic	Disorders in biological functioning	[96-99]
6	Neurological	Neurological impairment	Acute	Acute exposure may result in headaches and dizziness	[100,101]
7	Reproductive	Disturbance in biological process of reproductive organs, stops of production progesterone and testosterone, reduction in sperm production, density and volume, immature gamete formation	Chronic	Linked to adverse reproductive outcomes and developmental issues.	[102-105]
8	DNA	Affecting DNA repair mechanisms, Epigenetic changes, chromosomal abnormalities, mutation in sister chromatid, breakdown of DNA strands, stops cell respiration mechanism, inhibit enzymatic activity	Chronic	Increased production of reactive oxygen species (ROS) and endothelial oxidative stress	[106-111]
9	Respiratory	Shortness of breath loss of smell ability (anosmia) and coryza and hyaline, dyspnea and wheezing	Acute	Acute exposure leads to respiratory distress and lung inflammation.	[112-116]

Air sampling and analysis techniques of Cadmium: Airborne Cd exists in the form of gas and particles along with its compounds. This section briefly describes some methods for specifying the concentration of cadmium in air.

Sampling tool: For air cadmium sampling, filters are one of the most commonly used sampling tools. The properties of certain cadmium sampling filters are outlined in Table 2; for the sampling of respirable cadmium particles, a double cellulose ester filter with an 8 µm pore size and a flow rate of 1.7 L/min, along with a nylon cyclone, should be utilized [117-119].

Sample preparation: After collecting cadmium from the ambient air and transferring the samples to the laboratory, sample preparation for analysis should be performed. In the laboratory, the filter holder has been opened and the filter has been transferred to the beaker. Nitric acid or other acids have been added in the filter and the sample heated, followed by addition of chloric acid or other acids. It is crucial to remember that the specific sample preparation methods used for cadmium particulates may vary depending on the requirements and regulations of the specific application [119-121].

Analysis of samples: Following the sample preparation, the analysis is performed based on the detection limits. Cadmium concentration in the air can be measured using methods such as atomic absorption spectrometry (AAS), X-ray fluorescence (XRF), and inductively coupled plasma (ICP). Analytical methods introduced by NIOSH and Occupational Safety and Health Administration (OSHA) include NIOH 7048, 7300, 7301, 7303 and OSHA ID121, ID125G, ID189, ID206 [120, 122]. Explanations for each method are listed in Table 2.

Measuring Cadmium in biological samples: Recent advances in sample analysis are essentially related to sample preparation and sampling methods in analyzers to lower detection limits or sample analysis time. AAS and ICP-AES are the two most popular analytical techniques for determining the level of cadmium in biological samples [123]. The samples for these techniques are prepared in different ways. Nitric acid (HNO3) digestion is the most common method [124, 125]. Graphite furnace atomic absorption spectroscopy (GFAAS) can accurately and precisely measure the concentration of cadmium in blood and plasma. The detection limit of this method for a sample is 0.4 g/L [125]. Furthermore, this technique has been reported to determine cadmium in urine and hair with 99% precision[126].

Table 2. Analytical Methods for Determining Cadmium in Air

No.	Method number	Year of publication	Principle of the method	Analysis	Flow rate (L/min)	Air volume (L)	LOQ (mg/m^3)[a]($\mu g/m^3$)[b]	LOD (µg/ sample)
1	NIOSH 7300	2003	Particulates trapped on an MCE or PVC filter in a 37mm filter cassette. Hotplate dissolution with HNO3 and HCL.	ICP-AES	1-4	13-2000	0.00005[a]	0.0075
2	NIOSH 7301	2003	Particulates trapped on an MCE or PVC filter in a 37mm filter cassette. Hotplate dissolution with HNO3 and HCL.	ICP-AES	1-4	13-2000	0.00005[a]	0.0075
3	NIOSH 7302	2014	Particulates trapped on an MCE or PVC filter in a 37mm filter cassette. Hotplate dissolution with HNO3.	ICP-AES	1-4	12-2000	-	0.1
4	NIOSH 7303	2003	Particulates trapped on an MCE filter in a 37mm filter cassette. . Hotplate dissolution with HNO3 and HCL	ICP-AES	1-4	3-500000	0.012[b]	0.0037
	NIOSH 7304	2014	Particulates trapped on an PVC filter in a 37mm filter cassette. Hotplate dissolution with HNO3 .	ICP-AES	1-4	13-2000	-	0.2
5	NIOSH 7306	2015	Internal capsule, cellulose acetate dome with inlet opening, attached to 0.8-µm pore size mixed cellulose ester (MCE) membrane filter and housed within a 2-piece, closed-face cassette (CFC) filter holder, 37-mm diameter. Hotplate dissolution with HCl and HNO3.	ICP-AES	1-4	3- >2000	-	0.0052
6	OSHA ID-121	2002	Particulates trapped on an MCE filter in a 37mm filter cassette. . Hotplate dissolution with HNO3 and HCL	FAAS	2	30-960	0.0002[a]	-
7	OSHA ID-125	2002	Particulates trapped on an MCE filter or a PVC filter (for sampling welding fume) in a 37 mm filter cassette. Hotplate dissolution: with H2SO4, HNO3, H2O2 and HCl (MCE filters) or HNO3 and HClO4 (PVC filters).	ICP-AES	2	30-460	0.001[a]	-
8	OSHA ID-189	1992	Particulates trapped on an MCE filter in a 37 mm filter cassette. Hotplate dissolution with HNO3 and HCl.	FAAS or ETAAS	2	30-960	ETAAS: 0.00005[a] FAAS: 0.0003[a]	-
9	OSHA ID-206	1991	Particulates trapped on an MCE or PVC filter in a 37 mm filter cassette. Hotplate dissolution: with H2SO4, HNO3, H2O2 and HCl (MCE filters) or HNO3 and HClO4 (PVC filters).	ICP-AES	2	480-960	0.001[a]	-

FAAS: Flame Atomic Absorption Spectroscopy, **ETAAS**: Electrothermal Atomic Absorption Method, **ICP-AES**: Inductively Coupled Plasma Atomic Emission Spectroscopy, **LOQ**: Limit of Quantification, **LOD**: Limit of Detection.

Different advances have been made in extraction, preconcentration, chelation, and complexation methods[2, 50, 55-59]. Preconcentration procedures, such as chelation and extraction, may be utilized if the concentration of cadmium in the sample solution is below the detection limit [126, 127].

The ICP method was developed to measure the cadmium content of biological materials. ICP dynamic reaction cell mass spectrometry (ICP-DRC-MS) can eliminate molybdenum polyatomic interactions that result in diminished urinary cadmium concentration [128]. Since cadmium is a ubiquitous element, meticulous laboratory procedures should be employed to minimize the risk of contamination along sampling, preparation, and analysis [129]. All glass and plastic containers should be cleaned with acid and then rinsed with distilled water when applying the procedures for detecting trace quantities of cadmium.

The cadmium concentration in biological samples can be measured by other methods, such as radiochemical neutron activation analysis (RNA) analysis. The determination of cadmium using the RNAA method involves a rapid extraction with solution in two stages [130]. Tissue cadmium concentrations are measured in the body (in vivo) [131, 132] and in the laboratory (in vitro) [133] through a neutron activation analysis (NAA). X-ray fluorescence (XRF) is also employed to measure cadmium in the kidney in vivo [131, 134-136].

Methods for analyzing cadmium in biological media include ICP-MS [137, 138], ICP-AES [139, 140] along with high performance liquid chromatography (HPLC) [141, 142]. The amount of cadmium deposited in tooth enamel was analyzed using electrothermal vaporization ICP-MS [143]. Electrochemical methods such as adsorptive cathodic stripping voltammetry (ACSV) and potentiometric stripping analysis (PSA) have also been utilized to analyze cadmium levels in hair[144], animal tissue [145] and body fluids [146]. Table 3 reports methods for measuring cadmium in biological samples.

Table 3. Analytical Methods for Determining Cadmium in Biological Materials [143-148]

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery
Blood	Digestion with nitric acid; chelation with APDC and extraction with MIBK	AAS	<1 ng/mL	99
Blood	Modification of matrix with diammonium hydrogen phosphate/Triton X-100	GFAAS	0.1 µg/L	100.8±4.3
Blood/plasma	Digestion with nitric acid; wet ashed	GFAAS	0.4 µg/L	No data
Serum	Dilution with ammonia/Triton X-100	ICP/MS	0.01 ng/mL	No data
Tissue and blood	Microwave digestion	FAAS/flow injection system	0.15 µg/L	No data
Human milk	Dilution with deionized and double distilled water	AAS	<0.01 ppb	No data
Hair	Digestion with nitric acid	AAS	0.07 µg/g	99
Kidney	None (*in vivo*)	XRF	170.1 µg/g	No data
Kidney/liver	Chelation and extraction with solvent	AAS/direct aspiration	0.01 ppm (liver) 1.9 mg (kidney)	No data
Kidney/liver	None (*in vivo*)	NAA	1.3 µg/g (liver) 1.9 mg (kidney)	No data
Muscle	Wet ashed with concentrated sulfuric acid	NAA	50 ppb	50–65
Urine	Dilution with nitric acid	ETAAS	0.045 µg/L	97–101
Urine	Modification of matrix with diammonium hydrogen	GFAAS	0.09 ng/mL	92.7–111.1
Urine	Digestion with nitric acid	AAS	5.67 ng/mL	99.4

AAS: atomic absorption spectroscopy; **APDC:** ammonium pyrrolidenedithiocarbamate; **APTH:** 1,3-bis-[1-(2-pyridyl)ethylidene] thiocarbon-hydride; **ETAAS:** electrothermal atomic absorption spectroscopy; **FAAS:** flame atomic absorption; **GFAAS:** graphite furnace atomic absorption; **ICP/AES:** inductively coupled plasma atomic emission spectroscopy; ICPIMS: inductively coupled plasma mass spectrometry; **MlBK:** methyl isobutyl ketone; **NAA:** neutron activation analysis; **PSA:** potentiometric stripping analysis; **RNAA:** radio chemical neutron activation analysis; **XRF:** x-ray fluorescence

Occupational Exposure Limits: Various international and national agencies, along with institutional databases, have established recommended limits for cadmium exposure to ensure the health of employees. These limits are based on observed concentrations of cadmium that are considered to offer sufficient protection. For instance, the ILO International Chemical Safety Cards (ICSC) database specifies limits such as 0.01 mg/m3 as an 8-hour time-weighted average (TLV-TWA) for cadmium, 0.002 mg/m3 for cadmium chloride, and 0.002 mg/m3 for cadmium oxide. Likewise, the ACGIH recommends a TLV-TWA of 0.01 mg/m3 for "total particulate" cadmium and 0.002 mg/m3 for the "respirable particulate" fraction, with the

aim of minimizing the risk of kidney dysfunction and lung cancer, respectively. The ACGIH also suggests monitoring cadmium levels in urine and blood as specific tests for chronic and recent exposure, respectively. As a biological exposure index (BEI), a cadmium concentration of 5 µg/g of creatinine is suggested for chronic exposure, while a cadmium concentration of 5 µg/L in blood is proposed for recent exposure. Furthermore, the Scientific Committee on Occupational Exposure Limits (SCOEL) of the European Commission has introduced an occupational exposure limit of 1 µg/m3 for the inhalable fraction of cadmium and a Biological Limit Value of 2 µg/g creatinine in urine [149].

According to the Carcinogens and Mutagens Directive (2004/37/EC), the occupational exposure limit (OEL) for cadmium (respirable) is 0.001 mg/m^3 [150]. Many countries have currently developed OELs related to cadmium and its mineral compounds (respirable or inhalable). Figure 3 lists Cd OELs for some countries. Nevertheless, as not all countries are included in this list, they should not be considered as a comprehensive tool.

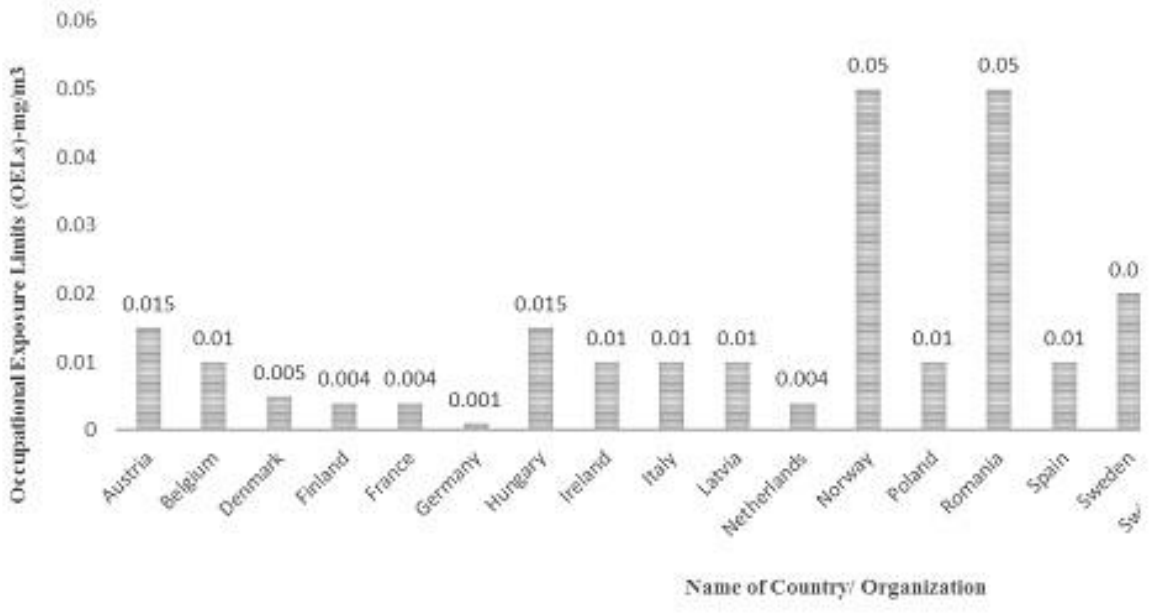

Fig. 3. Occupational Exposure Limits (OELs) at 8-h Time-Weighted Average (TWA) for Cadmium and its Compounds

Discussion

The findings of this review, derived from 84 studies (51 on health effects, 33 on monitoring), highlight systemic toxicity of cadmium and the urgent need for improved occupational safeguards. Chronic exposure, even at low concentrations, triggers irreversible damage, with cardiovascular effects being the most studied (e.g., hypertension), while neurological impacts (e.g., anosmia, blood-brain barrier penetration) remain critically understudied. This is in accordance with prior evidence linking cadmium to olfactory dysfunction, yet mechanisms such as cadmium's affinity for metallothionein in the olfactory epithelium warrant deeper exploration.

Renal toxicity, a hallmark of cadmium exposure, manifests at urinary cadmium levels as low as 2 µg/g creatinine, with Fanconi-like syndrome and osteomalacia (e.g., Itai-itai disease), underscoring irreversible risks. However, occupational bone demineralization remains underreported, suggesting a need for longitudinal studies in high-risk industries. The carcinogenicity of cadmium, particularly lung cancer, reinforces the necessity of adhering to the 0.01 mg/m^3 exposure limit and implementing controls such as fabric filters (reducing airborne cadmium to <1 mg/m^3).

Monitoring challenges persist: filters dominate air sampling, yet trace-level detection requires novel adsorbents. While NIOSH/OSHA methods (AAS, ICP-MS) are widely used, elemental interferences (e.g., molybdenum in ICP-MS) necessitate advanced techniques such as ICP-DRC-MS. Biological monitoring (urine/blood) is underutilized in spite of its value in evaluating cumulative exposure.

Cadmium is a toxic heavy metal commonly encountered in various occupational settings, especially in industries such as mining, battery production, and metal plating. Prolonged exposure to cadmium can result in severe health issues, including respiratory diseases, kidney dysfunction, and bone disorders. The inhalation of cadmium dust or fumes can cause lung damage and is associated with an elevated risk of lung cancer, making it a significant concern for workers. Moreover, cadmium can accumulate in the body over time, leading to chronic health effects that may not be immediately apparent. Regular monitoring of cadmium exposure levels and implementing strict safety measures are

crucial for protecting workers. Health surveillance programs can aid in early detection of cadmium-related health issues, ensuring timely intervention. Ultimately, addressing cadmium exposure is significant for promoting occupational health and safeguarding the well-being of workers in at-risk industries. Thanks to improvements in occupational hygiene and the introduction of more environmentally friendly forms of electric power, the level of worker exposure to cadmium along the production of nickel-cadmium batteries is diminishing. However, employees involved in cadmium smelting and recovery from iron and steel scrap, as well as waste recovery from electric and electronic equipment, should also take precautions to lower exposure [151].

There are four approaches to lowering cadmium emissions from sources: 1) Reducing the use of initial components and final products containing cadmium or using raw materials with low cadmium content; 2) Replacing cadmium-containing procedures with cadmium-free alternatives ; 3) Controlling cadmium emissions through low-emission process technologies and flue gas and waste treatment; 4) Developing substrates that do not contain cadmium [152].

Sampling and analysis methods are vital for evaluating cadmium exposure in occupational environments. Air sampling techniques, such as employing personal samplers or stationary monitors, enable the measurement of airborne cadmium concentrations, providing real-time data on exposure levels. For biological monitoring, collecting urine or blood samples allows for the assessment of cadmium accumulation in workers, offering insights into individual exposure. Analytical methods, including atomic absorption spectroscopy (AAS) and inductively coupled plasma mass spectrometry (ICP-MS), are utilized to accurately quantify cadmium levels in samples. Further, surface wipe sampling can ascertain contamination on work surfaces, helping to identify potential sources of exposure. Regular calibration and validation of equipment ensure the reliability of results. Ultimately, robust sampling and analysis methods are essential for effective occupational health management regarding cadmium exposure, facilitating timely interventions and safeguarding worker health. Effective monitoring methods are critical for managing cadmium exposure in occupational settings to protect worker health. Regular air quality assessments through personal and area sampling can help identify cadmium levels in the workplace environment. Biological monitoring, such as measuring cadmium concentrations in urine or blood, can inform individual exposure and accumulation over time. Further, health surveillance programs can track symptoms and health outcomes among workers, facilitating early intervention.

Cadmium can be emitted from processes either through fugitive emissions or via flue gas systems. Fugitive emissions refer to uncontrolled emissions caused by the release, handling, and storage of raw materials or byproducts. These emissions can be minimized by transferring these operations to completely enclosed structures equipped with ventilation systems and proper monitoring. The effectiveness of lowering fugitive emissions heavily depends on the efficiency of gas and dust exhaustion systems, such as suction hoods. The most efficient devices for controlling cadmium gases and particles are fabric filters, which can effectively remove cadmium particles from the airstream to less than 1 mg/m3. If the gas stream is passed through a system to reduce acid gases, as typically utilized in power plants and waste incinerators, the level of cadmium in the gas stream can be further reduced once dust reduction measures have been applied [153]. As observed in the literature review, there are limited methods available for sampling cadmium. The sampling media for cadmium have been restricted to filtration. In cases where there is a need to sample trace levels of cadmium, this issue should be pursued carefully by researchers to deal with the synthesis of new media with higher efficiency. The lack of adequate precision in the analytical instruments for cadmium samples is one of the weaknesses of the analytical methods. In spite of the common use of the ICP method among the analytical techniques for cadmium-containing samples, in cases where there are elemental interferences from molybdenum, it is essential to use more accurate methods such as ICP-DRC-MS.

Future research on cadmium should focus on developing new sampling and analysis techniques to accurately measure cadmium concentrations across various environmental samples, such as air, soil, and water. These techniques should be able to detect low levels of cadmium and provide precise as well as accurate results. It is also important to examine the health outcomes of low-level cadmium exposure, which is common among the general population. These studies should prioritize exploring the long-term health effects of cadmium exposure in work environments and the development of chronic diseases. Further, future research should deal with the combined effects of cadmium and other toxic substances commonly found in the environment and occupational settings. It is also necessary to identify new biomarkers of cadmium exposure and toxicity which can be used for biological monitoring and risk assessment. Finally, effective control strategies for cadmium toxicity should be developed to remove cadmium from the environment and prevent further exposure. These research efforts will enhance our understanding of cadmium toxicity and facilitate the development of effective strategies to prevent as well as minimize cadmium-related health impacts.

This review study presented several strengths and limitations. One of its key strengths was the

comprehensive overview it offered, synthesizing a wide range of studies that had examined the health impacts of cadmium exposure alongside various monitoring and measurement techniques. This diverse approach would enhance the understanding of the subject. Nevertheless, the study also faced limitations, such as potential variability in the methodologies and populations of the reviewed studies, which may affect the generalizability of the findings. Further, a lack of longitudinal studies limits insights into the long-term effects of cadmium exposure. Finally, the review could be influenced by publication bias, as significant findings are more likely to be reported.

Conclusion

Cadmium exposure in occupational settings poses serious health risks, including respiratory diseases, kidney damage, and elevated cancer risk. Workers in industries such as mining and battery manufacturing are especially vulnerable. Chronic exposure can lead to long-term health complications, underscoring the need for effective monitoring and preventive measures. To manage cadmium exposure, comprehensive air quality assessments and biological monitoring are crucial for gauging exposure levels as well as identifying at-risk individuals. Regular health surveillance programs track symptoms and health outcomes, enabling timely interventions. Fostering a safety culture through training raises awareness of cadmium risks and proper handling procedures. Advancements in monitoring techniques, such as atomic absorption spectroscopy and ICP-MS, are essential for accuracy. Control measures involve substituting cadmium with safer alternatives, implementing standard operating procedures, utilizing personal protective equipment, and conducting biomonitoring. Addressing these challenges is vital for safeguarding workers' well-being in cadmium-exposed industries.

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Conflict of interest

None declared.

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Ethical Considerations

As this study is a literature review, no new research was conducted on human or animal subjects. All cited research has been duly acknowledged, and the authors declare that they have no conflict of interest.

Code of Ethics

Ethical approval for this study was obtained from Research Ethics Committee of School of Medical Eduction-Shahid Beheshti University of Medical Sciences IR.SBMU.PHNS.REC.1401.108.

Authors' Contributions

Younes Mehrifar: Study conceptualization, methodology, data curation, writing-original draft preparation; Hamideh Pirami: Data curation, writing—original draft preparation; Somayeh Farhang Dehghan: Study conceptualization, methodology, reviewing, and editing; all authors have read and agreed to the published version of the manuscript.

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