Review

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Wenyu Wang , Yanfang Yang , Donglei Wang , Lihua Huang *

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Wenyu Wang, Yanfang Yang, Donglei Wang, Lihua Huang *

School of Public Health, Baotou Medical College, Baotou 014030, Inner Mongolia, China; 18503209703@163.com (W.W.); 1695958523@qq.com (Y.Y.); wang19971222@126.com (D.W.)

* Correspondence: huanglihua858@163.com

Abstract: Rare earth elements (REEs) are a type of new material resources, which have attracted significant attention in recent years. REEs have emerged as essential metals in modern-day technology due to their unique functions. Long-term large-scale mining and utilization of rare earths have caused serious environmental pollution and constitutes a global health issue, which raised concerns about human health safety. However, the toxicity profile of suspended particulate matter of REEs in the environment interacts with the human body in remain largely unknown. Studies have shown that REEs can enter the human body through a variety of pathways, leading to a variety of organ and system dysfunctions through changes in genetics, epigenetics, and signaling pathways. Through an extensive literature search and critical analysis, we provide a comprehensive overview of the available evidence, identify knowledge gaps, and make recommendations for future research directions.

Keywords: rare earth element; human health; toxicity effect; toxicity mechanism

1. Introduction

REEs refer to a series of elements, including a collective name for the lanthanides, Sc and Y, seventeen metallic elements in the periodic table [1]. According to the different structure and characteristics of REEs, REEs is usually divided into light rare earth (LREEs) and heavy rare earth (HREEs) [2]. Over the last few decades, REEs have been synthesized and used in various industries due to their characteristics [3]. However, numerous studies have shown that long-term large-scale mining of REE minerals may lead to excessive content of REEs in atmospheric particulate matter [4]. Workers in mining district are exposed to significant amounts of REEs through inhalation, ingestion, and skin contact [5]. Notably, REEs have been found in human blood, urine, and hair, implying a potential risk to human health from long-term exposure to REEs [6]. The increasing number of investigations have revealed REE pollution in ecosystems [7]. REEs can be transferred to the human body also through the food chain [8,9]. In addition, intravascular gadolinium contrast agents are used as substitutes for iodine contrast agents, iatrogenic exposure has also become an important route for REE exposure [10,11].

REEs exposure can gradually accumulate in the human body through various ways, thus affecting body health [12]. So far, there has been relatively little exploration of the toxicological effects and mechanisms of REEs effects on human health [13]. Therefore, the main purpose of this review is to focus on the current literature to provide an overview and discuss the hazardous effects of REEs exposure on human health.

2. Rare earth Exposure

Inhalation exposure is the most common exposure route of atmospheric particulates [14]. Sufficient studies have shown that long-term occupational exposure to inhaled REE particles can lead to significant REE deposition in the lungs [15,16]. The mining area is rich in REEs in the air around [17], and nearby resident daily intake of REE is much higher than the non-mining area [18,19]. And

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REEs can cross the placental barrier and affect the growth and development of the fetus [20]. Meanwhile, REEs have been detected in crops, soil, and the human gastrointestinal tract, indicating that REE exposure can also enter the body through the food chain [21,22]. Unnatural accumulation of REEs not only contaminates drinking water, but also poisons aquatic organisms [23], and directly or indirectly transfer to humans through the food chain, threatening human health [24]. In addition, studies have found that REE particles can also enter the human body through hair follicles and sweat glands, causing body damage [25]. Unlike other toxic exposure, due to the widespread use of intravascular gadolinium contrast agents, iatrogenic exposure has also become an important route of REEs exposure [11]. It indicates that the potential toxicity of REEs to human body through iatrogenic exposure cannot be ignored. In conclusion, the multiple exposure pathways of REEs and the consequent health risks have attracted our great attention.

3. Rare earth Toxicity

The increasing evidence that REEs can have a certain effect on human health hazards [26]. Due to the bioaccumulation of REEs, toxicological effects have been extensively evaluated in a large number of in vivo and in vitro models [12]. However, the relatively limited understanding and use of REEs, these studies have only simply explored the interaction between the toxic effects of some REEs and human health. The REEs associated with hazards to human health are summarized in Table 1. This section systematically discusses the toxicity studies of REEs exposure on the respiratory, cardiovascular, nervous, reproductive and other unclassified systems.

3.1. Respiratory System

Although atmospheric particles can be cleared by the immune mechanism of the human body, some REEs remain in the respiratory tract and produce toxic effects [27]. A number of observational studies on exposed populations have pointed out that workers who inhaled REE particles have a significantly increased incidence of airway and interstitial lung diseases, such as inflammation, granulomatous degeneration, pulmonary fibrosis, pneumoconiosis, and even cancer *[28–30]*, which may be caused by the accumulation and irritation of REEs. Based on epidemiological results, numerous of animal experiments simulating exposure levels of REEs have found that REEs can indeed cause severe lung damage. For example, Snow et al. showed that REE particles can be deposited in the lung through the respiration, activating oxidative stress and inducing pulmonary inflammation in mice [31]. Similarly, REEs can also cause granulomatous changes in the lung of rats [32]. Respiratory function disruption caused by long-term intratracheal instillation of REEs can lead to restricted ventilation dysfunction in mice, and eventually transformed into interstitial pulmonary fibrosis [33,34]. In addition, the vitro experiments further confirmed that REEs could enter lung cells and lead to decreased cell viability and enhanced apoptotic ability through reactive oxygen species (ROS) production and DNA damage effects [35]. Notably, the adverse effects of REEs on lung cells are influenced by environmental factors, particle size, exposure dose, and duration [15,36,37]. In particular, long-term exposure to nanoscale REEs particles can cause more serious damage to the lungs [38].

Table 1. Summary of REE-Associated Toxicological Studie.

	Element	Section Studied	Toxicity Outcome	Reference
Respiratory system	Y	Endotracheal	Dyspnea and pulmonary edema, Pleural effusions	[14]
	Ce	Environmental exposure, Skin contact	Extrapulmonary translocation, Interstitial lung disease, Pulmonary fibrosis, Pneumoconiosis, Cytotoxicity	[29] [33]
	Dy	Endotracheal instillation	Lung injury, Oxidative stress, Inflammatory response	[38]
	La	Environmental exposure	Phosphate deposition, Pulmonary fibrosis	[30]
	Nd	Occupational exposure, Environmental exposure	Cytotoxicity, Genotoxicity, Lung cancer	[35]
	Sm	Endotracheal	Lung injury, Inflammatory response, Pulmonary fibro	[57]
	Th	Environmental exposure, Skin contact	Dyspnea, Pneumoconiosis, Lung cancer	[62]
Nervous system	La	Environmental exposure, Skin contact, Food chain	Learning and memory impairment, Decreased spatial discrimination, Cytotoxicity, Memory disorders	[47] [49]
	Nd	Environmental exposure, Food chain	Fetal neural tube defects	[44]
	Gd	Iatrogenic exposure	Deposits in the brain, Brain damage	[42]
Cardiovascular system	La	Occupational exposure Food chain	Deposition in blood vessels	[57]
	Nd	Environmental exposure, Skin contact, Food chain	Abnormal cardiovascular and cerebrovascular development, DNA damage, Cytotoxicity	[58]
	Ce	Environmental exposure, Food chain	The hemoglobin level is reduced, Anemia	[53]
	Gd	Endotracheal instillation	Cytotoxicity, Hematopoietic destruction	[56]
Reproductive system	Ce	Environmental exposure, Oral administration	Oxidative stress, Placental dysfunction, Fetal abortion, Growth restriction	[64]

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	Gd	Iatrogenic exposure	Inflammatory or invasive skin diseases, Stillbirth, Neonatal death	[67]
Skeleton	Nd	Occupational exposure, Environmental exposure	Disorders of bone metabolism, Decreased bone mineral density	[72]
	La	Environmental exposure, Food chain	Abnormal metabolism of calcium and phosphorus, Decreased bone mineral density	[73]
	Gd	Iatrogenic exposure	Bone deposits, Osteoporosis	[70]
	Y	Iatrogenic exposure	Bone deposits	[71]

Note: REE, rare earth element; Y, Yttrium; Ce, cerium; Dy, Dysprosium; La, Lanthanum; Nd, Neodymium; Gd, Gadolinium; Sm, Samarium; Th, Thorium; Yb, Ytterbium.

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3.2. Nervous System

REEs can cross the blood-brain barrier and deposit in the brain, which underlies their neurotoxicity [39,40]. Epidemiological investigations of residents living in mining areas have shown that long-term exposure to REEs can cause neurological diseases, such as motor and sensory impairments, neurodegeneration or neurosis [41,42]. Observational studies in special populations, such as children and pregnant women in mining areas, have shown that REEs can lead to reduced intelligence and motor ability in children, deposit in the fetal brain, and affect neural tube development [43,44]. These studies have shown that REEs can be deposited in the brain, impair the development of the central nervous system, and even pass through the placental barrier to generate passage or transgenerational inheritance. A series of in vivo studies of neurological disorders associated with REE exposure have been reported by several groups. REEs were found to be able to deposit in the cerebral cortex and hippocampus, causing a significant reduction in plasma neurotransmitter levels and the number of neurons in mice, leading to impaired motor ability, spatial recognition, and memory [45–47]. Xu et al. found in Caenorhabditis elegans that REEs can cause neurodegenerative changes by inducing damage to dopaminergic and GABAergic neurons [48]. In addition, REEs can cause depression, anxiety, and sample behavior in mice, confirming that REE exposure can cause severe neurosis [49]. In vitro studies have also shown that REEs are deposited in human neurons and exert effects on neuron cell viability, morphology, apoptosis, and mitochondrial respiratory function [50], further revealing that REE exposure is associated with nervous system damage.

3.3. Cardiovascular System

Studies have shown that long-term exposure to REEs can cause leukopenia, increase telomerase activity in human peripheral blood monocytes, and even lead to lymphoma and leukemia [51,52]. The results of a cross-sectional study showed that children and adolescents in mining areas had lower blood levels of trace elements and hemoglobin, resulting in an increased probability of anemia [53]. These results indicate that REEs can be deposited in the blood and affect the number and classification of cells in the blood, causing harm to human health. Toxicological effects of REEs on the cardiovascular system were evaluated in animal models. REEs can be deposited in mice, reduce the number of blood cells, induce inflammatory cell aggregation and release of pro-inflammatory factors, leading to hematopoietic function and vasoconstriction disorders *[54–56]*. Zhao et al. also reported pathological changes in zebrafish after REE exposure, such as pericardial edema, cardiac contraction disorders, and myocardial hypertrophy [57], suggesting that REEs have adverse effects on the structure and function of the cardiovascular system. REEs induce abnormal vascular development in zebrafish by activating the apoptotic pathway [58]. In addition, Gojova et al. found that as markers of inflammation, intercellular cell adhesion molecule-1, interleukin-8, and monocyte chemotactic protein-1 were significantly increased in human aortic endothelial cells that internalized REE particles, suggesting that REEs can induce inflammation in vascular endothelial cells [59]. REEs can activate oxidative stress, induce inflammatory responses and damage endothelial cells, leading to atherosclerosis [60,61].

3.4. Reproductive System

The adverse effects of REEs on reproductive health have been a controversial topic. Studies have shown that the effects of REEs on male reproduction include impaired spermatogenesis, reduced sperm quality and testicular tissue damage [62,63]. Animal experiments have confirmed that there is a significant correlation between the reduction of mouse testis weight and the degree of sperm DNA damage and the exposure dose after long-term exposure to REEs [64]. This may be related to inflammation, oxidative stress, and disruption of the blood-testis barrier. Similarly, increased REE levels in women's serum may adversely affect the outcome of in vitro fertilization-embryo transfer and increase the risk of spontaneous abortion [65]. Numerous animal studies have found that REE

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can be deposited in the placental trophoblastic layer of mice exposed to REE, potentially leading to adverse pregnancy outcomes including placental dysfunction, fetal loss, or growth restriction [66]. In addition, there is an association between REE exposure and severe fetal and neonatal injury. REE exposure during pregnancy can lead to fetal cleft lip and palate and an increased risk of stillbirth or neonatal death [67,68]. Studies on pregnant mice exposed to REEs found that the number of primary follicles in newborn mice was significantly suppressed, suggesting that REEs may cause reproductive toxicity in the passage [69].

3.5. Other Systems

In addition to the above systems, the potential toxicity of REEs to the human body involves other systems. After long-term exposure to REEs, REE deposition can be detected in bone tissue, which reduces bone density and interferes with bone metabolism, leading to osteoporosis and bone and joint injury [70–72]. This is due to the influence of REEs can directly replace Ca^{2+} calcium phosphorus metabolism, or indirect regulation of osteoclast combination of Ca^{2+} receptor induced osteoporosis [73,74]. Large deposits of REE were also detected in patients with liver injury in the mining area, and there was a U-shaped relationship between serum REE levels and oral cancer risk, indicating that REE exposure can cause gastrointestinal injury [75,76]. Hao et al. pointed out that REEs can increase the burden of renal clearance of metabolites and cause damage to the urinary system [77]. REE can also induce increased thyrotropin secretion, leading to histopathological changes and thyroid dysfunction [78]. In vivo experiments further confirmed that luteinizing hormone, follicle stimulating hormone, and prolactin were significantly decreased in mice after oral administration of REEs [79], suggesting that REEs have endocrine disrupting effects. In addition, Martin-Aguilar et al found a strong association between increased number of brain MRI gadolinium enhancement lesions and multiple sclerosis recurrence, suggesting that REE exposure may lead to immune system impairment [80]. Taken together, these studies highlight the potential toxicity of REEs in various systems with adverse consequences on human health, and contribute to the further exploration of the role of REEs in toxicology to minimize the corresponding health risks.

4. Toxicity Mechanisms

Although the mechanism of REEs toxicity has been reported in several studies, research in this field still needs to be improved. This review mainly explores the mechanism of REEs toxicity from the following aspects: genetics, epigenetics and alterations in signaling pathways. Firstly, DNA damage in the form of gene mutation, chromosome damage or number change is considered to be the basic change of genetic damage, which can lead to apoptosis or necrosis. Epidemiological studies on exposed cohorts have shown that REE exposure can lead to an increase in urinary 8-OHdG, suggesting that DNA oxidative stress damage is a potential mechanism of health hazards caused by REEs [51]. A large number of in vitro and in vivo experiments have confirmed that REEs exposure can directly or indirectly activate oxidative stress, induce the cleavage of DNA repair protein Poly ADP-ribose polymerase (PARP), prevent chromosome agglutination, and lead to DNA damage *[81–84]*. In addition, cell experiments with internalized REEs particles showed an increase in DNA double-strand break marker proteins γ-H_2AX and a decrease in DNA repair proteins such as p-53 and PARP, confirming that REEs can induce genetic changes, cause DNA damage [85,86]. Secondly, with the intensive study of epigenetics in disease development, the research of non-coding RNA (ncRNA) as a molecular target has become a hot topic. High-throughput sequencing results showed that REEs induced damage was related to abnormal changes in ncRNA expression profiles [87]. For example, in a human bronchial epithelial cell model exposed to nanoparticles of neodymium oxide (NPs-Nd_2O_3), 1915 circRNAs (1025 up-regulated and 890 down-regulated) were abnormally expressed, inducing tissue dysfunction through sponge miRNAs [88]. Abnormal expression of lncRNAs can activate NF-κB and induce inflammation [89]. Moreover, Let-7a miRNA and miR-34a have also been confirmed to be abnormally increased in REE-exposed cervical cancer cells [90]. Apart from this, methylation is also crucial in epigenetic modification. Results of a recent study have shown that DNA methylation levels are reduced in human fibroblast cell lines exposed to REEs, which

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abnormally affect cell morphology and viability [91]. REE exposure can also enhance the methylation modification of histone H3, increase the binding of MLL1 complex in the NRF2 promoter region, and induce genotoxicity in cells [92]. In addition to the above mechanisms, classical pathways including inflammatory response, immune response, and endocrine signaling are significantly affected by REE exposure, such as abnormal changes in Nrf2, MAPK, and Toll-like receptor (TLR) signaling pathways *[93–95]*. A cross-sectional study of e-waste station residents showed that REE exposure caused endocrine diseases by increasing oxidative stress and altering hormone levels of the hypothalamic-pituitary-thyroid axis (HPT) [78]. Similarly, REEs deposited in animals can increase intracellular ROS levels, activate the Nrf2 endogenous antioxidant pathway, and induce vascular injury in mice [96]. In addition, in vitro and in vivo experiments have confirmed that REE particles can directly or indirectly activate the NF-κB signaling pathway, promote the synthesis and release of inflammatory chemokines, enhance immune cytotoxicity, and induce inflammation [93] [97,98]. Table 2 briefly summarizes the mechanisms of REEs toxicity based on the results of current in vitro and in vivo studies.

5. Conclusions

Due to the widespread distribution and persistence of REEs in the environment, there is an urgent need to fully understand the harmful effects and mechanisms of REE particles on human health. By comprehensively summarizing current knowledge found that REE exposure enters the human body through various pathways such as inhalation, ingestion, dermal contact and iatrogenic exposure and causes deposition, which in turn destroys the structure and function of various organs of the human body and induces multi-system diseases (e.g., respiratory, nervous, cardiovascular, reproductive and immune systems). Numerous in vitro and in vivo studies have shown that REEs exert these adverse effects mainly by affecting genetics and epigenetics, altering the activation of signaling pathways (Figure 1). Based on the evidence presented in this review, the correlation between exposure risk and potential health hazards of REEs was identified, which could contribute to their future development. However, the current information on the toxicological assessment of REEs is still insufficient, and there are still some challenges in finding the critical standard for human health hazards caused by REEs exposure.

Table 2. Summary of related toxicological mechanisms of rare earth elements.

	Type	Sample	REE Exposure	Toxicity	Reference
Genetic	In vivo	C57-ras	12.5, 25, and 50 mg/kg lanthanum nitrate for 180 d	The rare earth deposition causes direct damage	[39]
	In vitro	SH-SY5Y	10, 25, 50, and 100 μg/mL Gd_2O_3 for 24 and 48 h	Apoptosis is regulated by bcl-2/bax protein expression	[83]
	In vivo	Sprague-Dawley rat	1.5 mg/kg body weight Indium chloride for 8 weeks	Oxidative stress, Chromatin DNA damage	[81]
	In vivo	Rat	1mg / kg CeO_2 for 6 d	Oxidative stress, Inflammation, DNA damage	[96]
Epigenetic	In vitro	16HBE	10 μg/ml NPs-Nd_2O_3 for 48 h	Promote NF-κ B activation and promote cellular inflammation by negatively regulating adiponectin receptor 1 expression	[89]
	In vitro	16HBE	0, 5, 10, 20, 40 and 80 μg/ml Nd_2O_3 for 6, 12, 24, 48 and 72 h	circ_009773 regulates DNA damage	[88]
	In vitro	Human fibroblast cell	0.05 to 1.6 mg/mL of Tb-MOF for 48 h	Altered gene methylation, Induced genetic damage	[91]
Signaling pathways	In vivo	Rat	0, 1.56, 3.125, 6.25, 12.5, 25, 50, and 100 μg/mL Nd_2O_3 for 24h	Activating the NF-κ B and caspase-3 signaling pathways, promoting the synthesis and release of inflammatory chemokine	[97]
	In vivo	Rat	0, 1mg / kg CeO_2 nanoparticles for 6 d	Activation of oxidative stress, Nrf2 signaling pathways	[96]
	In vivo	C57BL/6J mice	Long-term exposure to cerium nanoparticles	Activation of NF-κ B signaling pathway can increase the cytotoxic activity of immune cells	[98]

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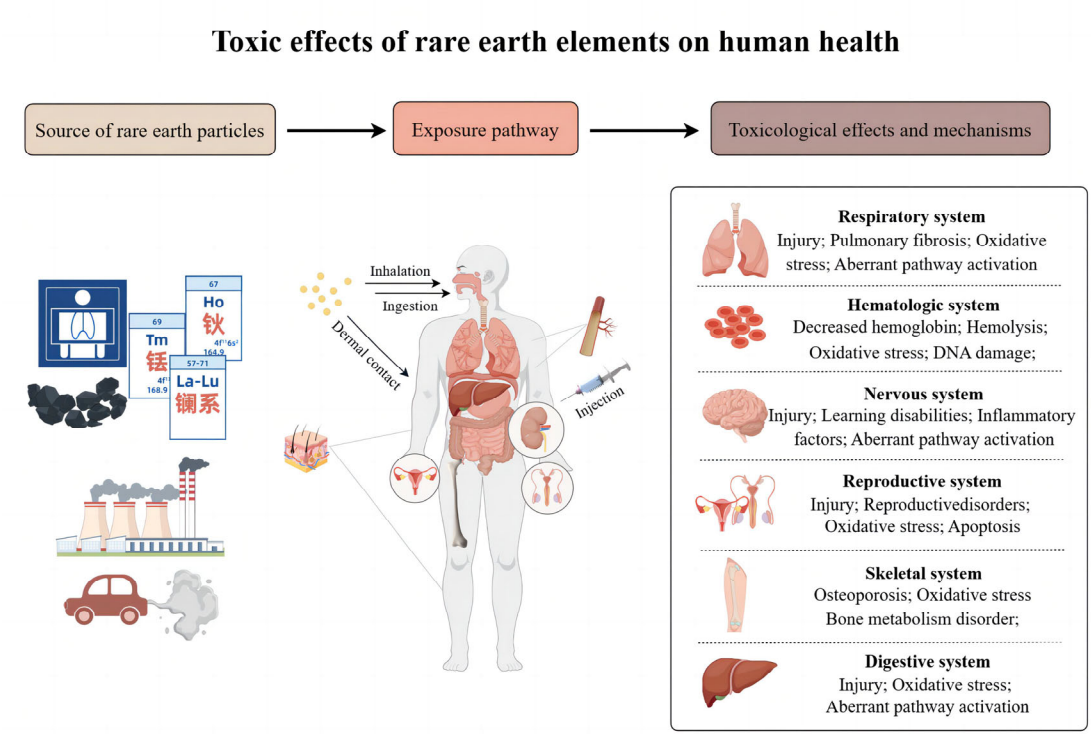

Figure 1. Toxic effects of rare earth elements on human health**.**

6. Challenges and Perspectives

Although REEs have become a hot spot in toxicology research in recent years, limited by the synergistic toxic effects of various REEs in the actual environment, imperfect detection indicators and dynamic metabolic differences in different individuals, the hazards of REEs on human health are still largely unknown. Further exploration of the interaction between them is helpful to emphasize the causal relationship between toxicant exposure and pathological state, explore the detection standard and safety limit of REE exposure, and develop new molecular markers for organ damage caused by REE. Several important issues associated with this challenge need to be addressed in this review: (1) Current studies on the interaction between REEs and health hazards are mostly limited to cell and animal models. In order to further verify the toxic effect of REE exposure on human body, long-term epidemiological cohort studies will become the next direction of close research. (2) The safety threshold of REE exposure should be established, especially the criteria for rare earth pneumoconiosis. Moreover, REE exposure dose of daily security standards are crucial. (3) The dose-response relationship between REEs and human health remains uncertain. Therefore, increasing the understanding of REE exposure, further elucidate the toxic effects and mechanisms of REEs and its compounds, and promote the development of future toxicological related research fields. Ultimately, it will contribute to the development of diagnostic and therapeutic measures for REEs related diseases, and provide regulatory guidance for hazard assessment and exposure thresholds for REEs.

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