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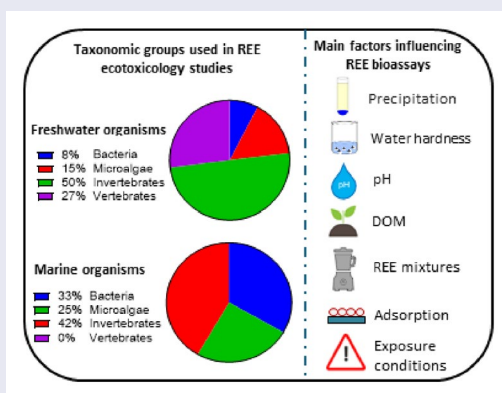
Effects of rare earth elements in the aquatic environment: Implications for ecotoxicological testing

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ABSTRACT

Rare earth elements (REE) are recognized as emerging pollutants due to their widespread use in modern society (e.g., in the production of electronics, renewable energy technologies, and advanced medical devices) which leads to anthropogenically elevated concentrations in the environment with potential consequences for ecosystem health. This article critically reviews the current scientific knowledge on aquatic bio-availability and toxicity of REE and focuses on pitfalls that could influence the outcome of ecotoxicity tests. After passing our quality criteria, we reviewed 38 papers on the ecotoxicity of REE in depth. Most studies focused on freshwater environments, indicating a need for more research on marine ecosystems, particularly on marine vertebrates. The results showed that heavy REE tend to be more toxic than light REE to aquatic organisms. Critical aspects for biotesting REE include complexation with ions such as phosphates (nutrient in algae tests) and carbonates. Carbonate complexation decreases potentially bioavailable aqueous REE species and may lower toxicity at increasing water hardness, although this may also be caused by competition of REE³⁺ and Ca²⁺ for the same binding sites in organisms. REE have a high tendency to adsorb to glass and it is recommended to use vessels made of polyethylene terephthalate or polycarbonate instead. More research is needed on chemical speciation and the interaction of REE with various organisms, also in multi-species mesocosm studies. A robust aquatic risk assessment on REE requires information on nominal and measured concentrations in both acute and chronic ecotoxicological bioassays as well as a thorough characterization of exposure.




KEYWORDS Emerging contaminants; freshwater; lanthanides; marine; sediment; speciation

Introduction

The Rare Earth Elements (REE) are a group of metals comprising the 15 elements from the lanthanide (Ln) series with atomic numbers (Z) from 57 to 71 as well as the chemically similar elements scandium (Sc) (Z=21) and yttrium (Y) (Z=39; Wall, 2021). Despite being called “rare

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earth elements,” they are not particularly rare in the Earth’s crust. Indeed, the most common REE, cerium (Ce), has a similar crustal abundance as nickel (Ni) and copper (Cu). With the exception of promethium (Pm), which has no stable isotopes, even the rarest lanthanides, thulium (Tm), and lutetium (Lu), are more common than silver (Ag) and platinum (Pt; Cotton, 2006). However, concentrated deposits are unusual.

The 17 REE show similar but not identical physical and chemical properties. Among the Ln, from lanthanum (La) to lutetium (Lu), electrons occupy the inner 4f orbitals with little effect on bonding properties. The effective nuclear charge, which is weakly shielded by the 4f electrons, causes a decrease in the atomic radius as the atomic number increases, a phenomenon known as the lanthanide contraction. This influences their chemical behavior, as heavier Ln with higher charge densities have an increased affinity for negatively charged groups. In aqueous solution, Ln as well as Y and Sc occur mostly in a trivalent (+3) oxidation state. The only other environmentally relevant oxidation states are 4+ for Ce, under oxidizing conditions, and 2+ for europium (Eu), under reducing conditions (Henderson, 1996). REE are often divided into light REE (LREE, La to samarium (Sm)) and heavy REE (HREE, Eu to Lu), with the separation of the categories being slightly different. Moreover, it is unclear whether Sc and Y should be included.

Given their low solubility in water and the assumption, that only the free trivalent ions would be (bio)available, REE have historically been considered to present little or no environmental risk. Consequently, there are no specific regulatory restrictions for REE, not even drinking water standards for human health. However, due to the unique magnetic and/or optical properties of REE and their compounds, there has been a growing demand for REE in new technologies, for example, in the automotive industry and in renewable energy generation systems over the last two decades. Intensified use goes along with increasing emissions to the environment. While REE used to serve as natural geological tracers in hydrological systems, their current anthropogenically elevated concentrations in water, soil, and sediments have prompted the question: Could REE have negative consequences for humans and the environment? This concern is reflected by the relatively rapid increase in the number of scientific publications addressing the topic of REE (eco)toxicity, along with the rise in their global production (Fig. 1).

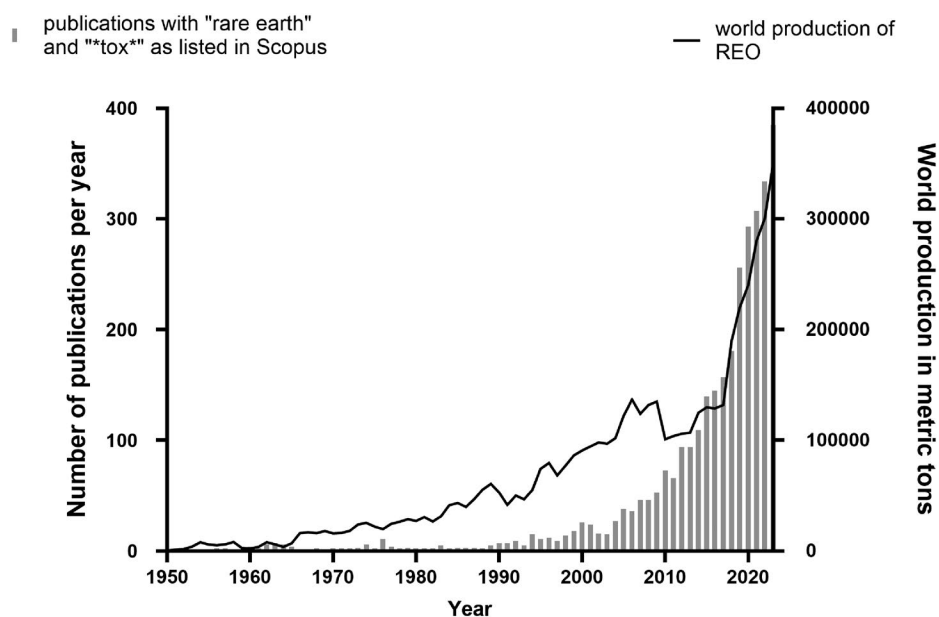


FIG. 1. World mine production of rare earth elements (REE) and the number of publications published from 1956 to 2023 containing “rare earth “AND” *tox*,” as listed in the SCOPUS database. Source for world production data: 1900–2020: U.S. Geological Survey (2014); 2021: U.S. Geological Survey (2023); 2022–2023: U.S. Geological Survey (2024).

A number of detailed experimental studies and literature reviews have been published over the last 10 years or more (Ng et al., 2011; Weltje, 2002). The more recent ecotoxicological studies have focused on specific elements such as La (Herrmann et al., 2016) and Gd (Trapasso, Chiesa, et al., 2021) or solely on marine waters (Piarulli et al., 2021). Others have provided extremely useful summaries on the current level of knowledge (Blinova et al., 2020; González et al., 2014; Kang & Kang, 2020; Malhotra et al., 2020) but have focused on individual REE, in single matrices or organisms (Herrmann et al., 2016; Malhotra et al., 2020). There are also more recently published reviews on the behavior and effects of REE in the environment (Balaram, 2019; Dushyantha et al., 2020; Neira et al., 2022). In this review, we provide and discuss the current scientific knowledge on the aquatic toxicity of the most commonly used REE, covering both marine and freshwater environments as well as sediments. We emphasize studies that have either reached a consensus or present conflicting observations. Additionally, we assess whether general conclusions can be drawn based on the current understanding of REE toxicity. We use this analysis to highlight the most critical aspects when performing biotests with REE, and to make more meaningful suggestions to improve the assessment of their environmental risk.

Study design and method

A thorough review of the literature was carried out using Google Scholar (<https://scholar.google.com>), the Scopus database (<https://scopus.com>), and PubMed (<https://ncbi.nlm.nih.gov/pubmed>), as well as by examining the reference lists of previously published reviews. The keywords used were Rare Earth Elements; REE; REY; lanthanides; Ln; the separate elements in combination with ecotoxicity, speciation, ecosystems, and toxicity in various biological species (crustaceans, bacteria, algae, vertebrates, invertebrates, and specific species (e.g., *Raphidocelis subcapitata*); and variations of these terms. This review is based on articles published until 2022, with some additions from 2023.

Studies measuring the ecotoxicity of REE-containing nanoparticles, lanthanide-modified bentonites, and contrast agents were excluded from this review because the behavior of these REE forms in water is different from that of dissolved species. All papers related to ecotoxicology were checked using the criteria of Klimisch et al. (1997). These criteria include: references to guidelines, data on the exposure period, a clear description of the test procedure, specification of the test substances, data on the test animals, the number of individuals tested, data on the measured parameters, data on the physical and chemical test conditions, the determined effect concentrations, data on the statistical evaluations, data on the dosing of the test substance, and data on feeding of the test animals (only for chronic tests). Each criterion that is included in the article is assigned a score of 1, with a maximum of 10 points for acute tests and 12 points for chronic tests. A paper was included only when the score was ≥ 8 points. This resulted in 38 REE ecotoxicity studies, of which 26 addressed freshwater and 12 marine exposure matrixes. The nematode species *Caenorhabditis elegans* was categorized within the exposure freshwater matrix.

The compiled information on the ecotoxicity of REE provided insights into the distribution of the exposure matrix type (i.e., the medium in which organisms are exposed to REE, such as freshwater or sediments) and the studied biological species across publications (Fig. 2). Most studies have focused on freshwater organisms (62%, Fig. 2b), followed by marine organisms (29%, Fig. 2c). Sediments represent the least studied exposure matrix at 9%, making them scarce and do not allow for effective concentration estimations. Most freshwater related studies have been performed on invertebrates (50%, with *Caenorhabditis elegans* and *Daphnia magna* being the most studied organisms). Only 8% of the included studies have addressed freshwater bacteria.

While only 14 freshwater biological species have been tested, most of the marine studies have been performed on the bacterium *Aliivibrio fischeri* or on different sea urchin species (invertebrates). There were no studies on marine fish that met the criteria of this review. As shown in Fig. 2, information on the effects of REE on biological species is limited. REE toxicity on certain species has been disproportionately understudied within the freshwater and especially in marine environments.

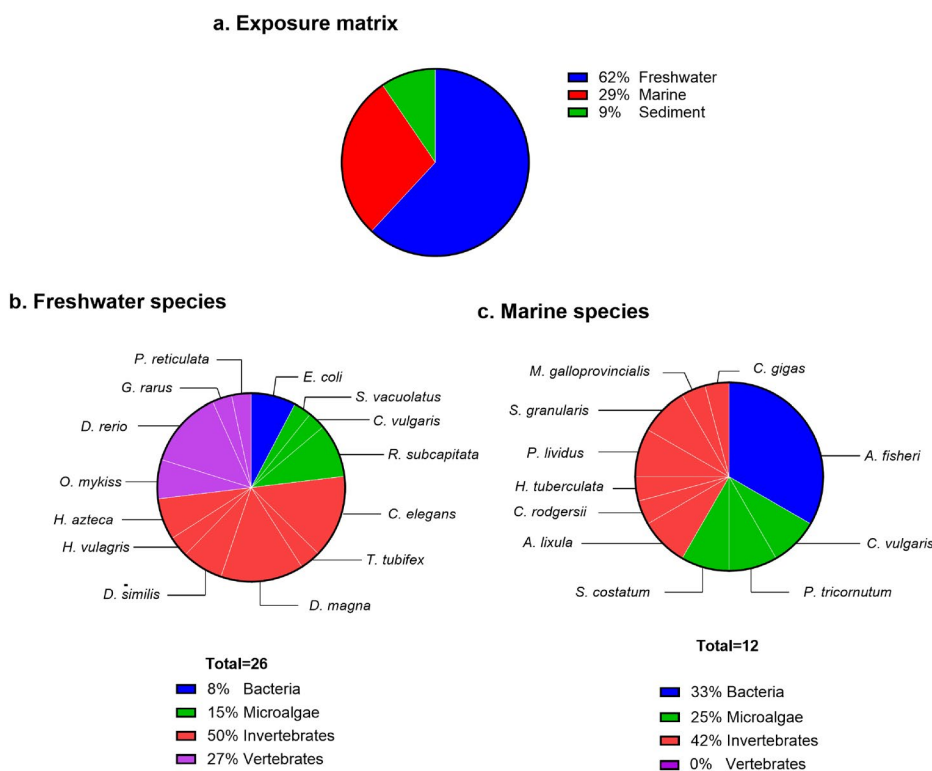


FIG. 2. Distribution of the exposure matrix type and the studied biological species used to calculate the rare earth element (REE) effect concentrations presented in this literature review. The pie charts show (a) the distribution of the exposure matrices used, (b) the distribution of the evaluated freshwater species, (c) the distribution of the evaluated marine species.

Natural occurrence and anthropogenic emissions of REE to the aquatic environment

Because of their chemical similarity, REE behave as a group and are distributed in the environment through geogenic erosion processes. Their concentrations in environmental media maintain a consistent pattern. In general, REE follow the Oddo-Harkins rule (Harkins, 1917; Oddo, 1914) with the highest concentrations being found for the first even-numbered REE (Ce), while the lowest concentrations are found for the last two odd-numbered REE (Tm and Lu), with concentrations typically two orders of magnitude lower than for Ce (Table 1 and Fig. S1).

Out of the 17 REE, only Pm does not have a stable isotope and has therefore no natural occurrence. An analysis of geochemical data prepared by the Forum of European Geological Surveys (FOREGS; Salminen et al., 2005) concluded that the overall distribution pattern over Europe is entirely due to the geological characteristics. Concentrations in water vary over two orders of magnitude, depending on the regional geology.

The absolute concentration of REE in aquatic systems depends on the rock composition in the respective catchment and the water chemistry (Elderfield et al., 1990). When normalizing dissolved REE concentrations to shale composition, the REE distribution follows the natural background level. Small anomalies in this pattern can be caused by differences in the stability of complexes or redox-sensitivities (Tepe et al., 2014), while large positive anomalies point to anthropogenic sources. For example, local and regional anomalies are derived from substantial targeted mining and intentional application in industrial products. Most extensive REE mining is carried out in China, where 62% of global production occurred in 2019 (Garside, 2021). Liang et al. (2014) compiled data from mining areas in China and showed that soil contamination in these areas reached magnitudes of mg g^{-1} (or mmol kg^{-1}), well above the world average (Fig. 3). In acidic waters from mining sites on a branch of the Yellow

Table 1. The median concentrations of rare earth elements (REE) in the upper continental crust (Rudnick & Gao, 2003) as reported in the Forum of European Geological Surveys (FOREGS database) and the median REE concentrations and ranges in stream water and stream sediment in Europe according to the FOREGS database (Salminen et al., 2005).

REE	Upper continental crust	Stream water, filtered <0.45 μm		Stream sediment, size fraction <150 μm	
	$\mu\text{mol kg}^{-1}$	Median	Range	Median	Range
		$\mu\text{mol l}^{-1}$	$\mu\text{mol l}^{-1}$	$\mu\text{mol kg}^{-1}$	$\mu\text{mol kg}^{-1}$
Sc	311	No data		No data	
Y	236	7.20×10^{-04}	$<3.37 \times 10^{-05} - 7.34 \times 10^{-02}$	289	7.63–3630
La	223	2.45×10^{-04}	$<1.44 \times 10^{-05} - 1.15 \times 10^{-07}$	230	1.33–705
Ce	450	3.93×10^{-04}	$<1.43 \times 10^{-05} - 7.21 \times 10^{-02}$	475	0.33–46.50
Pr	50.40	6.39×10^{-05}	$<1.42 \times 10^{-05} - 1.06 \times 10^{-02}$	49.70	1.27–576
Nd	187	2.77×10^{-04}	$<1.39 \times 10^{-05} - 3.99 \times 10^{-02}$	191	0.13–91.20
Sm	31.30	5.99×10^{-05}	$<1.33 \times 10^{-05} - 7.12 \times 10^{-03}$	34.60	0.68–481
Eu	6.58	3.29×10^{-05}	$<1.32 \times 10^{-05} - 1.32 \times 10^{-03}$	6.51	0.24–101
Gd	25.40	6.36×10^{-05}	$<1.27 \times 10^{-05} - 6.17 \times 10^{-03}$	31	0.42–275
Tb	4.40	1.26×10^{-05}	$<1.26 \times 10^{-05} - 8.18 \times 10^{-04}$	4.85	0.12–38.10
Dy	24	4.92×10^{-05}	$<1.23 \times 10^{-05} - 4.86 \times 10^{-03}$	27.40	0.58–247
Ho	5.03	1.21×10^{-05}	$<1.21 \times 10^{-05} - 1.03 \times 10^{-03}$	5.40	0.11–34.50
Er	13.80	3.59×10^{-05}	$<1.20 \times 10^{-05} - 2.87 \times 10^{-03}$	15.50	7.63–3630
Tm	1.78	1.18×10^{-05}	$<1.18 \times 10^{-05} - 4.03 \times 10^{-04}$	2.31	1.33–705
Yb	11.30	3.47×10^{-05}	$<1.16 \times 10^{-05} - 2.37 \times 10^{-03}$	14.30	0.33–46.50
Lu	1.77	1.14×10^{-05}	$<1.14 \times 10^{-05} - 4.00 \times 10^{-04}$	2.17	<1.27–576

REE concentrations in soil samples in Chinese mining areas

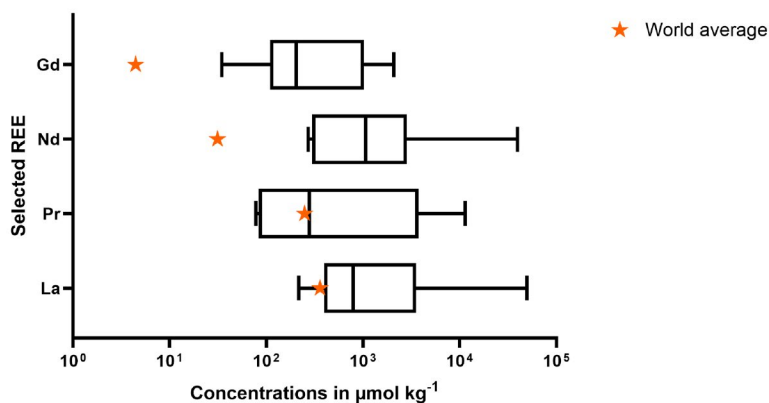


FIG. 3. The soil concentrations of select rare earth element (REE) in Chinese mining areas compared with the world average concentrations (stars), based on data from Liang et al. (2014). The box and whisker plots display the distribution of the measured concentrations, with limits of the boxes indicating the range of the central 50% of concentrations measured at seven mining areas, the central line indicating the median, and the whiskers extending to the minimum and maximum values.

River in China, REE were enriched by a factor of more than 1000 compared with the less affected main river section. In suspended particulate matter and deposited sediment, REE-concentrations exceeded average values of rivers in Northern China by up to 200-fold (Liang et al., 2014).

Outside China's mining area, product manufacturing and related technological processes can lead to elevated REE environmental concentrations. The transport pathways that link production and uses to elevated concentrations in the aquatic environment include atmospheric deposition, surface run-off, and effluents. Among these, atmospheric deposition may occur but has not been shown to contribute substantially to the contaminant load of rivers (Cidu et al., 2013), at least outside of mining areas. Substantial Gd anomalies in surface waters due to the presence of magnetic resonance imaging (MRI) contrast agents in hospital effluents have first been described by Bau and Dulski (1996). Subsequently, this phenomenon has been reported for all rivers worldwide that flow through densely populated areas (Klaver et al., 2014). For example,

anthropogenic Gd fluxes in the Garonne watershed in France increased from 203 mol year⁻¹ in 2003 to 477 mol year⁻¹ in 2017 (Lerat-Hardy et al., 2019). Moreover, production sites of fluid cracking catalysts lead to substantial increases in Ce, La, and Sm, as do mining activities (Table 2).

Industrial effluents containing REE can be discharged directly into surface waters, as REE are currently unregulated. A study by Kulaksız and Bau (2011) traced a La anomaly in the Rhine river to an upstream plant producing fluid cracking catalysts in Worms, Germany, and emitting substantial quantities of La into the river. In addition, La could be traced up to the North Sea (Klaver et al., 2014). If levels exceed the median values, they remain within the range of natural background concentrations (Table 1). With increasing emissions, these concentrations are likely to rise in the future and will specifically accumulate in sediments. However, elevated loads by themselves are not necessarily a cause for concern.

Speciation and adsorption

Speciation

In water, REE partitioning between bulk waters, pore waters, the dissolved fraction, and the colloidal fraction, is controlled by speciation and adsorption (Zhong & Mucci, 1995). The speciation of REE is characterized by the competition between stable aqueous complexes and the adsorption to or (co)precipitation of solid phases. Therefore, REE exist as free metal ions or complexed with organic or inorganic ligands and are impacted by colloidal composition and water chemistry (Verplanck, 2013).

With a similar ionic radius, REE also competes with divalent cations such as calcium (Ca²⁺) and magnesium (Mg²⁺) for binding sites of organic ligands (Du et al., 2019; Marang et al., 2008). Apart from being present in their free ionic form (REE³⁺), REE can form complexes with inorganic ligands such as carbonate, hydroxide, phosphate, and chloride (Millero et al., 2009; Moermond et al., 2001; Wood, 1990). While REE nitrates, chlorides, and sulfates are more soluble, REE carbonates, phosphates, and hydroxides have high stability constants (Table 3) and are considered practically insoluble (Wells & Wells, 2012). Metal complexation and precipitation with inorganic and organic ligands correlate positively with pH (Han, 2020; Millero et al., 2009; Moermond et al., 2001).

Organisms such as microalgae absorb carbon dioxide (CO₂) and secrete metabolites that affect the aqueous pH (Wu et al., 2022). Therefore, buffering agents are commonly employed in ecotoxicological studies to maintain a constant pH. When performing bioassays, the suitability of the pH buffer used should be considered. A study with Eu(III) indicated that the pH buffers' ability to form complexes with the metal varied significantly, with 3-(N-morpholino)propane sulfonic acid (MOPS) exhibiting the highest complex-forming capacity. In contrast, tris(hydroxymethyl)aminomethane (TRIS) had no significant interaction with Eu(III) while the other buffers did (Mandal et al., 2022). However, metal–buffer complexation should not be the only consideration when deciding which buffer is most suitable for an assay. For example, it has been demonstrated that test media buffered with sodium bicarbonate (NaHCO₃) increased the toxicity of copper (Cu) and zinc (Zn) for algae, while there were no differences in metal toxicity for the pH buffers MOPS and hydrochloric acid (HCl; De Schampelaere et al., 2004).

Dissolved organic matter (DOM, often expressed as dissolved organic carbon (DOC)) has a high complexation ability with REE. In freshwater solutions, about 95% of REE is associated with DOM, which mainly consists of humic and fulvic acids; REE bind to their carboxylic and phenolic groups (Dupré et al., 1999; Marang et al., 2008; Perdue et al., 1984; Torres & Choppin, 1984). The presence of DOM strongly affects REE speciation as it can increase the solubility of REE by counteracting precipitation and increasing the mobility of REE in the environment by serving as a transport vehicle (Choppin, 1986; McCarthy et al., 1998). However, at higher concentrations, the presence of DOM in a water system significantly reduces bioaccumulation and

Table 2. Detected anomalies of rare earth elements (REE) in aquatic systems and the reported concentrations.

REE	Concentrations	Unit	Matrix	Region/location	Country	Probable source	Reference
Ce	$1.19 \times 10^{+03}$	$\mu\text{mol kg}^{-1}$	Sediment	Anning River, Sichuan Province	China	Mining activity	Wang et al. (2021)
Ce	1.10×10^{-05} – 1.01×10^{-03}	$\mu\text{mol L}^{-1}$	Surface water	Rhine River, ca. 250 km downstream of effluent	Germany	Fluid cracking catalyst production	Kulaksiz and Bau (2013)
Ce	$1.21 \times 10^{+03}$	$\mu\text{mol kg}^{-1}$	Sediment	Rhine estuary	The Netherlands	Artificial fertilizer plant	Bakkenist and van de Wiel (1995)
Gd	1.70×10^{-05} – 1.18×10^{-04}	$\mu\text{mol L}^{-1}$	Surface water	Suzhou City (urban waters)	China	WWTP	Shilin et al. (2021)
Gd	9.79×10^{-06} – 5.51×10^{-04}	$\mu\text{mol L}^{-1}$	Surface water	Jinzhong Stream	China	WWTP from a hospital	Zhang et al. (2019)
Gd	4.58×10^{-05} – 2.09×10^{-04}	$\mu\text{mol L}^{-1}$	Surface water	Han-River, influenced by a WWTP	South Korea	WWTP from hospitals	Song et al. (2017)
Gd	1.14×10^{-05} – 4.58×10^{-05}	$\mu\text{mol L}^{-1}$	Surface water	Garonne River	France	WWTP from hospitals	Lerat-Hardy et al. (2019)
Gd	1.01×10^{-03}	$\mu\text{mol L}^{-1}$	Surface water	Havel River	Germany	WWTP from hospitals	Bau and Dulski (1996)
La	3.42×10^{-05}	$\mu\text{mol L}^{-1}$	Surface water	Anning River, Sichuan Province	China	Mining activity, industrial and agricultural activity	Wang et al. (2021)
La	2.38×10^{-03}	$\mu\text{mol L}^{-1}$	Surface water	Rhine River, ca. 50 km downstream of effluent	Germany	Fluid cracking catalyst production	Kulaksiz and Bau (2011)
La	9.07×10^{-05} – 6.41×10^{-04}	$\mu\text{mol L}^{-1}$	Surface water	Rhine River, ca. 250 km downstream of effluent	Germany	Fluid cracking catalyst production	Kulaksiz and Bau (2013)
La	$5.76 \times 10^{+02}$	$\mu\text{mol kg}^{-1}$	Sediment	Rhine estuary	The Netherlands	Artificial fertilizer plant	Bakkenist and van de Wiel (1995)
Nd	$5.55 \times 10^{+02}$	$\mu\text{mol kg}^{-1}$	Sediment	Rhine estuary	The Netherlands	Artificial fertilizer plant	Bakkenist and van de Wiel (1995)
Pr	$2.13 \times 10^{+02}$	$\mu\text{mol kg}^{-1}$	Sediment	Rhine estuary	The Netherlands	Artificial fertilizer plant	Bakkenist and van de Wiel (1995)
Sm	1.27×10^{-05} – 2.11×10^{-04}	$\mu\text{mol L}^{-1}$	Surface water	Rhine River, ca. 250 km downstream of effluent	Germany	Fluid cracking catalyst production	Kulaksiz and Bau (2013)
Sm	$1.33 \times 10^{+02}$	$\mu\text{mol kg}^{-1}$	Sediment	Rhine estuary	The Netherlands	Artificial fertilizer plant	Bakkenist and van de Wiel (1995)
Y	$2.70 \times 10^{+01}$ – $3.60 \times 10^{+02}$	$\mu\text{mol kg}^{-1}$	Sediment	Tagus estuary	Portugal	Chemical and phosphorus fertilizer industry	Brito et al. (2018)

WWTP=Wastewater treatment plant.

Table 3. The solubility constants of three selected rare-earth elements (REE) based on the increase in the atomic number (lightest = La; heaviest = Lu) (from NIST Database 46 Version 8.0).

REE species	log(K)
La(OH)	5.5
La(CO ₃)	6.98
La(NO ₃)	0.7
La(SO ₄)	3.64
LaCl	0.53
La(PO ₄)(s)	25.75
Lu(OH)	6.6
Lu(CO ₃)	8
Lu(NO ₃)	0.4
Lu(SO ₄)	3.52
LuCl	-1.97
Lu(PO ₄)(s)	24.8

toxicity of REE due to strong complexation and precipitation (El-Akl et al., 2015; Lachaux, Cossu-Leguille, et al., 2022; Tang & Johannesson, 2003; Vukov et al., 2016).

The half-maximal (50%) effective concentration (EC_{50}), which is often used to assess environmental risk, can be influenced by the solution composition and chemical speciation. So, these factors should be considered when assessing the toxicity of REE and calculating the EC_{50} (Lachaux, Catrouillet, et al., 2022). While chemical speciation is an essential consideration when investigating REE behavior and toxicity, it remains a challenging issue. Apart from analytical techniques such as voltammetry (the labile fraction including the free REE ions; Lee et al., 1997) or inductively coupled plasma mass spectrometry (ICP-MS; Haraguchi et al., 1998) that allow quantifying the dissolved metal concentration in solution, speciation modeling can be carried out by using geochemical modeling software such as PHREEQC (Parkhurst, 1995), Visual MINTEQ (Gustafsson, 2011), Geochemist's Workbench (Bethke, 2022), or WHAM (Tipping, 1994). Speciation calculations on natural waters demand an intense analytical effort to characterize the system in terms of its components but also simplification when considering the influence of DOM (Dupré et al., 1999) and other factors such as the fluctuation of abiotic parameters and/or the presence of organisms.

Surface adsorption

Sediments, and more precisely mineral phases such as iron (Fe-) and manganese (Mn-) oxyhydroxides, are efficient adsorbents for REE (Gwenzi et al., 2018). Metals associated with the Fe-containing aluminosilicate mineral fraction can undergo diagenesis after deposition in sediments (Michalopoulos & Aller, 2004; Wen et al., 2008).

Silicates (SiO_4^{4-}) are the main component of glass and are known to be a strong adsorbent of REE. Indeed, silicate materials are used to remove REE from industrial wastewater (Callura et al., 2018; Perea et al., 2018; Weltje, Brouwer, et al., 2002). The amount of lanthanum that can be adsorbed by glass (silica) is up to 25% of the total La present in the medium (Weltje, Brouwer, et al., 2002). It is therefore recommended to avoid the use of glassware apparatus in bioassays; rather, containers with a lower adsorption capacity for REE should be used.

Other material surfaces used in the apparatus in bioassays can also adsorb a certain amount of metal ions, including materials such as polyethylene, polypropylene, Teflon, stainless steel, borosilicate glass, quartz, and "soft glass" (Eichholz et al., 1965; Weltje, Brouwer, et al., 2002). In fact, Ce adsorption (measured in % adsorption per cm^2) to polypropylene is stronger than for borosilicate glass (Eichholz et al., 1965). Furthermore, a decrease in pH significantly reduces the adsorption on glass and has an even more pronounced effect on plastic. However, it has

been suggested that REE adsorption remains low enough to be neglected in bioassays (Eichholz et al., 1965). For example, Aharchaou et al. (2020) measured La and Ce concentration variation in abiotic media and found that even after 120 h, less than 20% of the initial amount had been adsorbed onto the walls of the test vessel.

The adsorption of REE on external parts (e.g., shells and cell walls) of aquatic organisms such as microorganisms, algae, invertebrates, and vertebrates has been well studied and differs among organisms (Cheng et al., 2018; Das et al., 1988, 2014; Martinez et al., 2014; Ramasamy et al., 2019; Tamjidi & Ameri, 2020). La sorption has been quantified at $1010 \mu\text{mol g}^{-1}$ for crab shells (Vijayaraghavan et al., 2009), $7200 \mu\text{mol g}^{-1}$ for the green freshwater algae *Desmodium multivariabilis* (Birungi & Chirwa, 2014) and $1080 \mu\text{mol g}^{-1}$ for the marine brown algae *Turbinana canoides* (Vijayaraghavan et al., 2010). The affinity of REE for *Bacillus subtilis* increases with the atomic number, which suggests an enrichment of HREE on the surface of these bacteria (Martinez et al., 2014). The Langmuir isotherm model points toward carboxylic groups on the cell wall of bacteria as potential REE adsorption sites.

Potential environmental impact of REE

Uptake of REE and occurrence in biota

REE are considered non-essential elements but can be (bio)accumulated in almost all organisms including microorganisms (Tsuruta, 2006), phytoplankton (Ramasamy et al., 2019), zooplankton (MacMillan et al., 2017, 2019), benthos (Merschel & Bau, 2015), and other aquatic invertebrates (Amyot et al., 2017; Pastorino et al., 2020). Bioaccumulation of REE seems to be dependent on the biological species and size (e.g., age; Hanana et al., 2017; Kazak et al., 2021; Merschel & Bau, 2015; Perrat et al., 2017) and is often related to the surface area of an organism (Palumbo, 1963) and the location (Weltje, Heidenreich, et al., 2002). These variations can be explained through site-specific REE availability and the different feeding pathways (e.g., grazing and filter feeding), as well as exposure routes (Bustamante & Miramand, 2005; Weltje, Heidenreich, et al., 2002). Accumulation of the different elements in algae and bacteria reflects the environmental concentrations, and under natural conditions follow the Oddo-Harkins rule (Weltje, Heidenreich, et al., 2002). However, this does not seem to apply to crustaceans (González et al., 2015).

Lanthanides are not equally distributed between the shells and soft tissue of bivalves (Akagi & Edanami, 2017; Tjink & Yland, 1998; Weltje, Heidenreich, et al., 2002), with REE concentrations being up to 10 times higher in tissues than in shells (Akagi & Edanami, 2017). Among the soft tissues, the digestive gland of bivalves tends to bioaccumulate the most REE (Bustamante & Miramand, 2005; Lobel et al., 1991; Perrat et al., 2017). This difference in bioaccumulation among the organs also occurs in fish. For example, La and Gd bioconcentration are known to decrease in the order of internal organs > gills > skeleton > muscle in carp (*Cyprinus carpio*; Qiang et al., 1994).

REE are also known to bioaccumulate in both micro- and macroalgae (Diniz & Volesky, 2005; Oliveira et al., 2012; Ramasamy et al., 2019; Yang & Wilkinson, 2018). The concentration of seven REE in 35 different marine species sampled in China varied from 8.69×10^{-05} to $0.27 \mu\text{mol g}^{-1}$ dry weight (Hou & Yan, 1998). This accumulation varied with algal species but also with the specific REE or the atomic number. These observations are different than what Bingler et al. (1989) reported: The marine diatom *Skeletonema costatum* showed a difference in LREE and HREE uptake. The authors concluded that LREE adsorb preferentially onto the surface of the microalgae or are taken up by the algae while HREE show a stronger affinity for the test media, meaning that metals are less taken up or absorbed by the organisms.

If a food-borne compound is bioaccumulated, it may also be biomagnified, that is, lead to concentrations in the organisms that increase with the trophic level. At present, the biomagnification potential of REE is unclear. On the level of plants and invertebrates (e.g., *Lemna minor* and several species of sea urchins and blue mussels), researchers have found limited potential

for the biomagnification of REE (MacMillan et al., 2017; Weltje, Heidenreich, et al., 2002). Others found that the opposite effect, biodilution, dominates: The metal concentrations decrease as the trophic level increases in both freshwater and marine food chains (Amyot et al., 2017; Liu et al., 2011; MacMillan et al., 2017).

La³⁺ adheres to the bacterium *Escherichia coli*, which is commonly used as food in bioassays with *C. elegans* (Zhang et al., 2010). Elemental mapping of *C. elegans* indicated dose-dependent La accumulation. This bioaccumulation might mostly be due to the ingestion of high levels of La absorbed to *E. coli*. Marked La accumulation in *C. elegans* can lead to a distinct elemental imbalance where there is a decline in concentrations of Ca²⁺, potassium (K⁺) and zinc (Zn²⁺; Zhang et al., 2010). Several studies have suggested that REE ions can bind to the Ca²⁺ binding sites in several proteins in nematodes (Burroughs et al., 1994; Fujimori & Jencks, 1990; Wakabayashi et al., 2016).

Sediment-bound REE are usually considered to be unavailable, but they may become available, when sediment particles are ingested by organisms, or when environmental conditions are changed (Mori, 1999). As such, the sediment may be considered a source for REE in water. Benthic fish have a higher concentration of REE than pelagic-feeding fish species (Mayfield & Fairbrother, 2015). Benthic fish species are in direct contact with the sediments and can ingest greater amounts of REE from benthic organisms (Mayfield & Fairbrother, 2015; Wang et al., 2019). Non-predatory microbenthic invertebrates (e.g., the genera *Caenis* and *Baetis*) have been suggested to accumulate more REE than predatory organisms because the most efficient route of assimilation is through the intake of sediment (Pastorino et al., 2020).

Further, it remains challenging to replicate identical exposure conditions between laboratory and natural environments, which could (partly) explain the discrepancies observed between field and laboratory results. REE concentrations in organisms seem to correlate negatively with the trophic level in marine ecosystems regardless of the studied conditions.

Effect assessment

The toxicity of REE to freshwater organism has been studied more widely among different levels of the aquatic food web in comparison to marine organisms. Tables 4 and 5 compile all current available lethal concentration (LC), effect concentration (EC), and inhibition concentration (IC) data on freshwater and marine organisms exposed to REE, respectively, providing a comprehensive overview of the research in this field. However, there are still knowledge gaps that require further investigation to fully understand the ecotoxicological profile of REE.

Freshwater conditions

Bacteria. Even though bacteria have been studied extensively in relation to the biological effects of common trace metals in the aquatic environment, there have been relatively few studies on the effect of REE, despite the fact that these organisms are directly exposed to REE enriched water. Moreover, bacteria have been shown to influence the mobility and adsorption of REE by producing organic matter with a high affinity for REE (Perelomov & Yoshida, 2008). This affinity results in the creation of low-dissolved REE complexes, which notably increases as the pH levels decreases (Martinez et al., 2014; Takahashi et al., 2010).

Bacteria are often more sensitive to REE than other microorganisms (e.g. fungi; Talburt & Johnson, 1967). The toxicity also varies depending on the specific REE. For example, T  cher et al. (2020) characterized the toxic effects of 16 REE on the growth kinetics of *E. coli*. The estimated EC₅₀ indicated that four HREE (Er to Lu) and Y are the most toxic metals (Table 4), with values ranging from 3.00 to 8.30 μM, followed by Sc with an EC₅₀ of 1.10 μM. There are limited data regarding the EC₅₀ of REE for freshwater bacteria, while several studies have addressed the effect of REE on marine bacteria, especially *A. fischeri* (Table 5 and the “Bacteria” subsection of “Marine conditions”; Gonz  lez et al., 2015; Kurvet et al., 2017; Weltje et al.,

Table 4. Effect concentrations and selected exposure conditions for freshwater organisms.

Species	REE	Compound	Exposure time	Effective concentration. (µM)	Effective concentration interval (µM)	Biological endpoint	Effect concentration	Concentration type	Reference
Bacteria									
<i>Escherichia coli</i>	La	LaCl ₃	7.5h	22.90	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	La	La ₂ (SO ₄) ₃	7.5h	31.00	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Ce	CeCl ₃	7.5h	24.40	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Pr	PrCl ₃	7.5h	44.60	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Nd	NdCl ₃	7.5h	24.70	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Sm	SmCl ₃	7.5h	25.80	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Eu	ErCl ₃	7.5h	27.60	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Gd	GdCl ₃	7.5h	21.40	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Tb	TbCl ₃	7.5h	26.30	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Dy	DyCl ₃	7.5h	24.40	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Ho	HoCl ₃	7.5h	24.90	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Er	ErCl ₃	7.5h	16.10	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Tm	TmCl ₃	7.5h	14.60	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Yb	YbCl ₃	7.5h	5.10	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Lu	LuCl ₃	7.5h	4.20	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Y	YCl ₃	7.5h	16.00	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	Sc	ScCl ₃	7.5h	1.50	-	Growth	EC ₅₀	m	[1]
<i>Escherichia coli</i>	La	La(NO ₃) ₃	24h	<1.00 × 10 ⁻⁰³	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Gd	GdCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Sc	ScCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Ce	CeCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Y	Y(NO ₃) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Pr	PrCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Nd	Nd(NO ₃) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Sm	Sm(CH ₃ COO) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Sm	SmCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Eu	Eu(NO ₃) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Gd	Gd(CH ₃ COO) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Tb	Tb(CH ₃ COO) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Tb	TbCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Dy	DyCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Dy	Dy(CH ₃ COO) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Ho	HoCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Ho	Ho(CH ₃ COO) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Er	ErCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Er	Er(CH ₃ COO) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Tm	Tm(CH ₃ COO) ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Tm	TmCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Yb	YbCl ₃	24h	<1.00 × 10 ⁻⁰⁴	-	Survival	IC ₅₀	n	[2]

(Continued)

Table 4. Continued.

Species	REE	Compound	Exposure time	Effective concentration. (µM)	Effective concentration interval (µM)	Biological endpoint	Effect concentration	Concentration type	Reference
<i>Escherichia coli</i>	Lu	LuCl ₃	24h	<1.00×10 ⁺⁰⁴	–	Survival	IC ₅₀	n	[2]
<i>Escherichia coli</i>	Lu	Lu(CH ₃ COO) ₃	24h	<1.00×10 ⁻⁰⁴	–	Survival	IC ₅₀	n	[2]
Microalgae									
<i>Scenedesmus vacuolatus</i> ¹	Ce	Ce(NO ₃) ₃	120h	0.57	0.49–0.67	Growth (cell density)	EC ₅₀	m	[3]
<i>Scenedesmus vacuolatus</i> ¹	Ce	Ce(NO ₃) ₃	120h	0.36	0.26–0.50	Growth (cell density)	EC ₅₀	m	[3]
<i>Scenedesmus vacuolatus</i> ¹	La	La(NO ₃) ₃	120h	0.78	0.54–1.11	Growth (cell density)	EC ₅₀	m	[3]
<i>Scenedesmus vacuolatus</i> ¹	La	La(NO ₃) ₃	120h	0.20	0.12–0.36	Growth (cell density)	EC ₅₀	m	[3]
<i>Chlorella vulgaris</i>	La	La ₂ O ₃	72h	339	326–371	Growth (cell density)	IC ₅₀	n	[4]
<i>Chlorella vulgaris</i>	Nd	Nd ₂ O ₃	72h	382	351–429	Growth (cell density)	IC ₅₀	n	[4]
<i>Chlorella vulgaris</i>	Sm	Sm ₂ O ₃	72h	171	160–180	Growth (cell density)	IC ₅₀	n	[4]
<i>Raphidocelis subcapitata</i>	La	La ₂ O ₃	72h	372	340–417	Growth (cell density)	IC ₅₀	n	[4]
<i>Raphidocelis subcapitata</i>	Nd	Nd ₂ O ₃	72h	395	354–454	Growth (cell density)	IC ₅₀	n	[4]
<i>Raphidocelis subcapitata</i>	Sm	Sm ₂ O ₃	72h	439	369–517	Growth (cell density)	IC ₅₀	n	[4]
<i>Raphidocelis subcapitata</i> ¹	Ce	CeCl ₃	72h	45.10	–	Growth (fluorescence activity)	EC ₅₀	m	[5]
<i>Raphidocelis subcapitata</i> ¹	Gd	GdCl ₃	72h	14.10	–	Growth (fluorescence activity)	EC ₅₀	m	[5]
<i>Raphidocelis subcapitata</i> ¹	Lu	LuCl ₃	72h	11.90	–	Growth (fluorescence activity)	EC ₅₀	m	[5]
<i>Raphidocelis subcapitata</i> ¹	Ce	CeCl ₃	72h	<45.70	–	Growth (fluorescence activity)	EC ₅₀	n	[5]
<i>Raphidocelis subcapitata</i> ¹	Gd	GdCl ₃	72h	19.80	–	Growth (fluorescence activity)	EC ₅₀	n	[5]
<i>Raphidocelis subcapitata</i> ¹	Lu	LuCl ₃	72h	11.80	–	Growth (fluorescence activity)	EC ₅₀	n	[5]
<i>Raphidocelis subcapitata</i> ¹	Ce	CeCl ₃	72h	0.01	–	Growth (fluorescence activity)	EC ₁₀	m	[5]
<i>Raphidocelis subcapitata</i> ¹	Gd	GdCl ₃	72h	0.01	–	Growth (fluorescence activity)	EC ₁₀	m	[5]
<i>Raphidocelis subcapitata</i> ¹	Lu	LuCl ₃	72h	0.01	–	Growth (fluorescence activity)	EC ₁₀	m	[5]
<i>Raphidocelis subcapitata</i>	La	La(NO ₃) ₃	72h	9.14	6.91–11.30	Growth (cell density)	EC ₅₀	n	[6]
<i>Raphidocelis subcapitata</i>	Ce	Ce(NO ₃) ₃	72h	8.78	7.85–9.71	Growth (cell density)	EC ₅₀	n	[6]
<i>Raphidocelis subcapitata</i>	Gd	Gd(NO ₃) ₃	72h	7.69	6.42–8.90	Growth (cell density)	EC ₅₀	n	[6]
<i>Raphidocelis subcapitata</i>	Pr	Pr(NO ₃) ₃	72h	9.65	7.24–12.10	Growth (cell density)	EC ₅₀	n	[6]
Invertebrates									
<i>Caenorhabditis elegans</i>	La	LaCl ₃	72h	14.30	–	Brood size	EC ₅₀	m	[7]
<i>Caenorhabditis elegans</i>	La	LaCl ₃	72h	>20.00	–	Body length	EC ₅₀	m	[7]
<i>Caenorhabditis elegans</i>	La	LaCl ₃	72h	>20.00	–	Nr. of eggs	EC ₅₀	m	[7]
<i>Caenorhabditis elegans</i>	Nd	NdCl ₃	48h	399	–	Mortality	LC ₅₀	n	[8]

(Continued)

Table 4. Continued.

Species	REE	Compound	Exposure time	Effective concentration, (µM)	Effective concentration interval (µM)	Biological endpoint	Effect concentration	Concentration type	Reference
<i>Caenorhabditis elegans</i>	Pr	PrCl ₃	48h	636	—	Mortality	LC ₅₀	n	[8]
<i>Caenorhabditis elegans</i>	Sc	ScCl ₃	48h	703	—	Mortality	LC ₅₀	n	[8]
<i>Caenorhabditis elegans</i>	Nd	NdCl ₃	96h	293	—	Mortality	LC ₅₀	n	[8]
<i>Caenorhabditis elegans</i>	Pr	PrCl ₃	96h	199	—	Mortality	LC ₅₀	n	[8]
<i>Caenorhabditis elegans</i>	Sc	ScCl ₃	96h	580	—	Mortality	LC ₅₀	n	[8]
<i>Caenorhabditis elegans</i>	La	La(NO ₃) ₃	24h	9.54 × 10 ⁺⁰⁵	—	Mortality	LC ₅₀	n	[9]
<i>Caenorhabditis elegans</i> (L1)	La	La(NO ₃) ₃	24h	341	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L1)	La	La(NO ₃) ₃	24h	215	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L1)	La	La(NO ₃) ₃	48h	107	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L1)	La	La(NO ₃) ₃	72h	83.80	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L1)	La	La(NO ₃) ₃	96h	78.10	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L1)	La	La(NO ₃) ₃	120h	3.90	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L4)	La	La(NO ₃) ₃	24h	1.50	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L4)	La	La(NO ₃) ₃	48h	546	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L4)	La	La(NO ₃) ₃	72h	294	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L4)	La	La(NO ₃) ₃	96h	183	—	Mortality	LC ₅₀	n	[10]
<i>Caenorhabditis elegans</i> (L4)	La	La(NO ₃) ₃	120h	7.60 × 10 ⁺⁰⁴	—	Mortality	LC ₅₀	n	[10]
<i>Tubifex tubifex</i>	La	LaCl ₃	3min	8.52 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Ce	CeCl ₃	3min	7.55 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Pr	PrCl ₃	3min	7.20 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Nd	NdCl ₃	3min	6.89 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Sm	SmCl ₃	3min	7.34 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Eu	EuCl ₃	3min	7.36 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Gd	GdCl ₃	3min	8.46 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Tb	TbCl ₃	3min	8.36 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Dy	DyCl ₃	3min	7.14 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Ho	LuCl ₃	3min	8.27 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Er	ErCl ₃	3min	8.25 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Tm	TmCl ₃	3min	8.01 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Yb	YCl ₃	3min	8.80 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Lu	LuCl ₃	3min	7.20 × 10 ⁺⁰⁴	—	Immobilization	EC ₅₀	n	[11]
<i>Tubifex tubifex</i>	Y	YCl ₃	3min	3.31	2.74 – 3.82	Immobilization	EC ₅₀	n	[11]
<i>Daphnia magna</i>	La	La(NO ₃) ₃	21d	2.14	1.64 – 2.57	Mortality	LC ₅₀	n	[12]
<i>Daphnia magna</i>	Ce	Ce(NO ₃) ₃	21d	2.06	1.49 – 2.70	Mortality	LC ₅₀	n	[12]
<i>Daphnia magna</i>	Pr	Pr(NO ₃) ₃	21d	2.15	1.46 – 3.05	Mortality	LC ₅₀	n	[12]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃	21d	3.12	2.23 – 3.31	Mortality	LC ₅₀	n	[12]
<i>Daphnia magna</i>	Gd	Gd(NO ₃) ₃	21d	224	158–289	Mortality	LC ₅₀	n	[12]
<i>Daphnia magna</i>	La	La(NO ₃) ₃	48h	144	118–171	Immobilization	EC ₅₀	n	[12]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃	48h	118	112–123	Immobilization	EC ₅₀	n	[12]
<i>Daphnia magna</i>	Gd	Gd(NO ₃) ₃	48h	188	163–213	Immobilization	EC ₅₀	n	[12]
<i>Daphnia magna</i>	Ce	Ce(NO ₃) ₃	48h	188	163–213	Immobilization	EC ₅₀	n	[12]

Table 4. Continued.

Species	REE	Compound	Exposure time	Effective concentration. (µM)	Effective concentration interval (µM)	Biological endpoint	Effect concentration	Concentration type	Reference
<i>Daphnia magna</i>	Pr	Pr(NO ₃) ₃	48h	169	167–170	Immobilization	EC ₅₀	n	[12]
<i>Daphnia magna</i>	Ce	CeCl ₃	48h	1.75	0.59–6.67	Mortality	LC ₅₀	n	[13]
<i>Daphnia magna</i>	Er	ErCl ₃	48h	1.82	0.56–7.71	Mortality	LC ₅₀	m	[13]
<i>Daphnia magna</i>	Ce	Ce(NO ₃) ₃	24h	16.40	13.20–20.80–	Immobilization	EC ₅₀	m	[14]
<i>Daphnia magna</i>	Ce	Ce(NO ₃) ₃	48h	10.70	8.60–13.40	Immobilization	EC ₅₀	n	[14]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃	48h	6.24	–	Immobilization	EC ₅	n	[15]
<i>Daphnia magna</i>	Gd	GdCl ₃	48h	1.88	–	Immobilization	EC ₅	n	[15]
<i>Daphnia magna</i>	Yb	YbCl ₃	48h	2.06	–	Immobilization	EC ₅	n	[15]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃	48h	13.90	–	Immobilization	EC ₅₀	n	[15]
<i>Daphnia magna</i>	Gd	GdCl ₃	48h	5.11	–	Immobilization	EC ₅₀	n	[15]
<i>Daphnia magna</i>	Yb	YbCl ₃	48h	4.90	–	Immobilization	EC ₅₀	n	[15]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃	48h	20.10	–	Immobilization	EC ₅₀	n	[15]
<i>Daphnia magna</i>	Gd	GdCl ₃	48h	7.53	–	Immobilization	EC ₅₀	n	[15]
<i>Daphnia magna</i>	Yb	YbCl ₃	48h	7.23	–	Immobilization	EC ₅₀	n	[15]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃	48h	25.70	–	Immobilization	EC ₆₅	n	[15]
<i>Daphnia magna</i>	Gd	GdCl ₃	48h	9.95	–	Immobilization	EC ₆₅	n	[15]
<i>Daphnia magna</i>	Yb	YbCl ₃	48h	9.29	–	Immobilization	EC ₆₅	n	[15]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃	48h	30.50	–	Immobilization	EC ₇₀	n	[15]
<i>Daphnia magna</i>	Gd	GdCl ₃	48h	12.10	–	Immobilization	EC ₇₀	n	[15]
<i>Daphnia magna</i>	Yb	YbCl ₃	48h	11.40	–	Immobilization	EC ₇₀	n	[15]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃	48h	9.71	–	Immobilization	EC ₅	n	[15]
		+ DOM							
<i>Daphnia magna</i>	Gd	8 mg C L ⁻¹ GdCl ₃	48h	4.30	–	Immobilization	EC ₅	n	[15]
		+ DOM 8 mg C L ⁻¹							
<i>Daphnia magna</i>	Yb	YbCl ₃	48h	1.29	–	Immobilization	EC ₅	n	[15]
		+ DOM 8 mg C L ⁻¹							
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃	48h	21.50	–	Immobilization	EC ₉₀	n	[15]
		DOM							
<i>Daphnia magna</i>	Gd	8 mg C L ⁻¹ GdCl ₃	48h	8.34	–	Immobilization	EC ₃₀	n	[15]
		+ DOM							
<i>Daphnia magna</i>	Yb	YbCl ₃	48h	3.87	–	Immobilization	EC ₃₀	n	[15]
		+ DOM							
<i>Daphnia magna</i>	Nd	8 mg C L ⁻¹ Nd(NO ₃) ₃	48h	29.80	–	Immobilization	EC ₅₀	n	[15]
		+ DOM							
		8 mg C L ⁻¹							

(Continued)

Table 4. Continued.

Species	REE	Compound	Exposure time	Effective concentration. (µM)	Effective concentration interval (µM)	Biological endpoint	Effect concentration	Concentration type	Reference
<i>Daphnia magna</i>	Gd	GdCl ₃ + DOM 8 mg C L ⁻¹	48h	10.80	–	Immobilization	EC ₅₀	n	[15]
<i>Daphnia magna</i>	Yb	YbCl ₃ + DOM 8 mg C L ⁻¹	48h	5.94	–	Immobilization	EC ₅₀	n	[15]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃ + DOM 8 mg C L ⁻¹	48h	37.40	–	Immobilization	EC ₆₅	n	[15]
<i>Daphnia magna</i>	Gd	GdCl ₃ + DOM 8 mg C L ⁻¹	48h	13.20	–	Immobilization	EC ₆₅	n	[15]
<i>Daphnia magna</i>	Yb	YbCl ₃ + DOM 8 mg C L ⁻¹	48h	8.26	–	Immobilization	EC ₆₅	n	[15]
<i>Daphnia magna</i>	Nd	Nd(NO ₃) ₃ + DOM 8 mg C L ⁻¹	48h	45.10	–	Immobilization	EC ₇₀	n	[15]
<i>Daphnia magna</i>	Gd	GdCl ₃ + DOM 8 mg C L ⁻¹	48h	15.30	–	Immobilization	EC ₇₀	n	[15]
<i>Daphnia magna</i>	Yb	YbCl ₃ + DOM 8 mg C L ⁻¹	48h	10.30	–	Immobilization	EC ₇₀	n	[15]
<i>Daphnia similis</i>	La	La ₂ O ₃	48h	67.70	64.40–71.30	Immobilization	EC ₅₀	n	[4]
<i>Daphnia similis</i>	Sm	Sm ₂ O ₃	48h	85.90	83.80–108	Immobilization	EC ₅₀	n	[4]
<i>Daphnia similis</i>	Sm	Sm ₂ O ₃	48h	149	–	Mortality	EC ₅₀	n	[16]
<i>Daphnia similis</i>	La	La ₂ O ₃	48h	127	–	Mortality	EC ₅₀	n	[16]
<i>Daphnia similis</i>	Nd	Nd ₂ O ₃	48h	176	–	Mortality	EC ₅₀	n	[16]
<i>Daphnia similis</i>	Sm	Sm ₂ O ₃	48h	71.80	–	Mortality	EC ₅₀	n	[16]
<i>Daphnia similis</i>	La	La ₂ O ₃	48h	90.70	–	Mortality	EC ₅₀	n	[16]
<i>Daphnia similis</i>	Nd	Nd ₂ O ₃	48h	63.70	–	Mortality	EC ₅₀	n	[16]
<i>Hydra vulgaris</i> ¹	Y	YCl ₃	96h	0.73	0.59–0.92	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	La	LaCl ₃	96h	0.57	0.51–0.62	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Ce	CeCl ₃	96h	0.89	0.64–1.21	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Pr	PrCl ₃	96h	2.26	1.66–3.07	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Nd	NdCl ₃	96h	0.86	0.70–1.09	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Sm	SmCl ₃	96h	2.11	1.78–2.52	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Gd	GdCl ₃	96h	1.40	1.16–1.69	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Tb	TbCl ₃	96h	1.87	1.53–2.36	Mortality	LC ₅₀	n	[17]

(Continued)

Table 4. Continued.

Species	REE	Compound	Exposure time	Effective concentration. (µM)	Effective concentration interval (µM)	Biological endpoint	Effect concentration	Concentration type	Reference
<i>Hydra vulgaris</i> ¹	Dy	DyCl ₃	96h	1.83	1.49–2.23	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Er	ErCl ₃	96h	1.05	0.84–1.49	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Lu	LuCl ₃	96h	0.75	0.57–0.92	Mortality	LC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Y	YCl ₃	96h	0.10	0.07–0.13	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	La	LaCl ₃	96h	0.19	0.14–0.24	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Ce	CeCl ₃	96h	0.13	0.08–0.19	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Pr	PrCl ₃	96h	0.08	0.06–0.10	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Nd	NdCl ₃	96h	0.25	0.17–0.36	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Sm	SmCl ₃	96h	0.49	0.36–0.69	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Gd	GdCl ₃	96h	0.27	0.19–0.40	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Tb	TbCl ₃	96h	0.27	0.19–0.38	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Dy	DyCl ₃	96h	0.72	0.58–0.85	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Er	ErCl ₃	96h	0.26	0.18–0.37	Morphological alterations	EC ₅₀	n	[17]
<i>Hydra vulgaris</i> ¹	Lu	LuCl ₃	96h	0.26	0.18–0.39	Morphological alterations	EC ₅₀	n	[17]
<i>Hyalella azteca</i>	Sc	–	7d	0.65	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Y	–	7d	0.74	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	La	–	7d	0.13	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Ce	–	7d	0.20	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Pr	–	7d	0.25	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Nd	–	7d	0.38	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Sm	–	7d	0.49	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Eu	–	7d	0.74	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Gd	–	7d	0.95	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Tb	–	7d	0.53	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Dy	–	7d	1.00	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Ho	–	7d	0.87	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Er	–	7d	1.14	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Tm	–	7d	<0.01	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Yb	–	7d	0.40	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Lu	–	7d	0.17	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Sc	–	7d	2.22	–	Mortality	LC ₅₀	m	[18]
<i>Hyalella azteca</i>	Y	–	7d	2.06	–	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	La	–	7d	1.65	–	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Ce	–	7d	0.83	–	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Pr	–	7d	1.30	–	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Nd	–	7d	2.34	–	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Sm	–	7d	1.97	–	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Eu	–	7d	2.67	–	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Gd	–	7d	2.86	–	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Tb	–	7d	2.30	–	Mortality	LC ₅₀	n	[18]

(Continued)

Table 4. Continued.

Species	REE	Compound	Exposure time	Effective concentration. (µM)	Effective concentration interval (µM)	Biological endpoint	Effect concentration	Concentration type	Reference
<i>Hyalella azteca</i>	Dy	-	7d	2.98	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Ho	-	7d	3.00	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Er	-	7d	3.34	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Tm	-	7d	4.27	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Yb	-	7d	1.43	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Lu	-	7d	0.69	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Sc	-	7d	3.89	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Y	-	7d	6.18	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	La	-	7d	12.00	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Ce	-	7d	4.14	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Pr	-	7d	3.13	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Nd	-	7d	3.54	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Sm	-	7d	5.63	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Eu	-	7d	4.72	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Gd	-	7d	3.81	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Tb	-	7d	4.36	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Dy	-	7d	5.52	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Ho	-	7d	4.58	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Er	-	7d	5.55	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Tm	-	7d	4.37	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Yb	-	7d	1.61	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Lu	-	7d	6.02	-	Mortality	LC ₅₀	n	[18]
<i>Hyalella azteca</i>	Tm	-	96h	3.40	-	Mortality	LC ₅₀	m	[19]
Vertebrates									
<i>Oncorhynchus mykiss</i> (j)	Gd	GdCl ₃	96h	50.90	-	Mortality	LC ₅₀	m	[20]
<i>Oncorhynchus mykiss</i> (j)	Nd	NdCl ₃	96h	416	-	Mortality	LC ₅₀	m	[20]
<i>Oncorhynchus mykiss</i> (j)	Sm	SmCl ₃	96h	10.60	-	Mortality	LC ₅₀	m	[20]
<i>Oncorhynchus mykiss</i> (j)	Ce	CeCl ₃	96h	604	-	Mortality	LC ₅₀	m	[20]
<i>Oncorhynchus mykiss</i> (j)	La	LaCl ₃	96h	864	-	Mortality	LC ₅₀	m	[20]
<i>Oncorhynchus mykiss</i> (j)	Er	ErCl ₃	96h	47.80	-	Mortality	LC ₅₀	m	[20]
<i>Oncorhynchus mykiss</i> (j)	Y	YCl ₃	96h	7.87	-	Mortality	LC ₅₀	m	[20]
<i>Oncorhynchus mykiss</i> (j)	Lu	LuCl ₃	96h	4.83	-	Mortality	LC ₅₀	m	[21]
<i>Oncorhynchus mykiss</i> (j)	Dy	DyCl ₃	96h	29.30	-	Mortality	LC ₅₀	m	[21]
<i>Danio rerio</i> (e)	La	LaCl ₃	72h	603	-	Mortality	LC ₅₀	m	[22]
<i>Danio rerio</i> (e)	Yb	YbCl ₃	72h	268	-	Mortality	LC ₅₀	m	[22]
<i>Danio rerio</i> (j)	La	LaCl ₃	144h	637	-	Mortality	LC ₅₀	n	[23]
<i>Danio rerio</i> (e)	La	LaCl ₃	144h	624	-	Mortality	LC ₅₀	n	[23]
<i>Danio rerio</i> (cl)	La	LaCl ₃	96h	864	-	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Ce	CeCl ₃	96h	760	760-969	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Pr	Pr(NO ₃) ₃	96h	793	663-857	Cell viability	EC ₅₀	n	[24]
					706-879	Cell viability	EC ₅₀	n	[24]

(Continued)

Table 4. Continued.

Species	REE	Compound	Exposure time	Effective concentration. (µM)	Effective concentration interval (µM)	Biological endpoint	Effect concentration	Concentration type	Reference
<i>Danio rerio</i> (cl)	Nd	Nd(NO ₃) ₃	96h	672	567–776	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Sm	Sm(NO ₃) ₃	96h	823	696–950	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Eu	EuCl ₃	96h	1.05 × 10 ⁺⁰³	917 – 1.18 × 10 ⁺⁰³	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Gd	GdCl ₃	96h	849	717 – 981	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Tb	TbCl ₃	96h	1.07 × 10 ⁺⁰³	865 – 1.27 × 10 ⁺⁰³	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Dy	Dy(NO ₃) ₃	96h	668	593–744	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Ho	HoCl ₃	96h	673	547 – 799	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Er	Er(NO ₃) ₃	96h	480	449–512	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Tm	Tm(NO ₃) ₃	96h	501	298–705	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Yb	YbCl ₃	96h	576	488–665	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (cl)	Lu	LuCl ₃	96h	242	79.00–404	Cell viability	EC ₅₀	n	[24]
<i>Danio rerio</i> (j)	La	LaCl ₃	72h	171	162–180	Mortality	LC ₅₀	n	[25]
<i>Danio rerio</i> (j)	Gd	GdCl ₃	72h	149	142–156	Mortality	LC ₅₀	n	[25]
<i>Gobiocypris rarus</i>	La	LaCl ₃	96h	13.80	–	Mortality	LC ₅₀	m	[26]
<i>Poecilia reticulata</i> (j)	La	LaCl ₃	144h	523	–	Mortality	LC ₅₀	n	[23]

Dash (–) indicates missing or incomplete information. e = Embryo, j = Juvenile, 1 = Adopted to current names, L1 = L1 stage of *C. elegans*, L4 = L4 stage of *C. elegans*, cl = cell lines, m = measured concentration, n = nominal concentration, ECx = effective concentration at x%, LCx = lethal concentration at x%, and ICx = Inhibitory concentration at x%. Detailed literature is available at the end of the table.

References: [1] (Técher et al., 2020), [2] (Wakabayashi et al., 2016), [3] (Aharchaou et al., 2020), [4] (Bergsten-Torralba et al., 2020), [5] (González et al., 2015), [6] (Joonas et al., 2017), [7] (Zhang et al., 2010), [8] (Xu et al., 2017), [9] (Tatara et al., 1998), [10] (Han et al., 2022), [11] (Rucki et al., 2021), [12] (Blinova, Lukjanova, et al., 2018), [13] (Galdiero et al., 2019), [14] (Ma et al., 2016), [15] (Lachaux, Catrouillet, et al., 2022), [16] (Egler et al., 2023), [17] (Blaise et al., 2018), [18] (Borgmann et al., 2005), [19] (Loveridge et al., 2021), [20] (Dubé et al., 2019), [21] (Hanana et al., 2021), [22] (Cui et al., 2012), [23] (Márcová et al., 2011), [24] (Fleurbaix et al., 2022), [25] (Kang et al., 2022), [26] (Hua et al., 2017).

Table 5. Effect concentrations and selected exposure conditions of rare earth elements (REE) for marine organisms.

Species	REE	Compound	Exposure time	Effective concentration (µM)	Effective concentration interval (µM)	Biological endpoint	Effective concentration	Concentration type	Reference
Bacteria									
<i>Aliivibrio fischeri</i> ¹	Ce	Ce(NO ₃) ₃	30min	15.40	–	Luminescence	EC ₅₀	n	[1]
<i>Aliivibrio fischeri</i> ¹	Gd	Gd(NO ₃) ₃	30min	7.82	–	Luminescence	EC ₅₀	n	[1]
<i>Aliivibrio fischeri</i> ¹	La	La(NO ₃) ₃	30min	48.30	–	Luminescence	EC ₅₀	n	[1]
<i>Aliivibrio fischeri</i> ¹	Nd	Nd(NO ₃) ₃	30min	15.70	–	Luminescence	EC ₅₀	n	[1]
<i>Aliivibrio fischeri</i> ¹	Pr	Pr(NO ₃) ₃	30min	28	–	Luminescence	EC ₅₀	n	[1]
<i>Aliivibrio fischeri</i> ¹	Ce	CeCl ₃	30min	>17.20	–	Luminescence	EC ₅₀	n	[2]
<i>Aliivibrio fischeri</i> ¹	Gd	GdCl ₃	30min	>17.20	–	Luminescence	EC ₅₀	n	[2]
<i>Aliivibrio fischeri</i> ¹	Lu	LuCl ₃	30min	8.22	–	Luminescence	EC ₅₀	n	[2]
<i>Aliivibrio fischeri</i> ¹	Lu	LuCl ₃	30min	5.24	–	Luminescence	EC ₅₀	m	[2]
<i>Aliivibrio fischeri</i> ¹	Ce	CeCl ₃	30min	9.40 × 10 ⁻⁰³	–	Luminescence	EC ₁₀	m	[2]
<i>Aliivibrio fischeri</i> ¹	Gd	Gd(NO ₃) ₃	30min	2.70 × 10 ⁻⁰³	–	Luminescence	EC ₁₀	m	[2]
<i>Aliivibrio fischeri</i> ¹	Lu	LuCl ₃	30min	2.00 × 10 ⁻⁰⁴	–	Luminescence	EC ₁₀	m	[2]
<i>Aliivibrio fischeri</i> ¹	Lu	Lu ³⁺ no ligand	7.5 min	2.16	–	Luminescence	EC ₅₀	n	[3]
<i>Aliivibrio fischeri</i> ¹	Lu	Lu ³⁺ no ligand	15min	1.57	–	Luminescence	EC ₅₀	n	[3]
<i>Aliivibrio fischeri</i> ¹	Lu	Lu ³⁺ no ligand	22.5 min	1.44	–	Luminescence	EC ₅₀	n	[3]
<i>Aliivibrio fischeri</i> ¹	Lu	Lu ³⁺ no ligand	30min	1.37	–	Luminescence	EC ₅₀	n	[3]
<i>Aliivibrio fischeri</i> ¹	La	La(NO ₃) ₃	15min	1.69 × 10 ⁻⁰³	–	Luminescence	EC ₅₀	n	[4]
Microalgae									
<i>Chlorella vulgaris</i>	La	La(NO ₃) ₃	96h	23.30	–	Growth yield	EC ₅₀	n	[5]
<i>Chlorella vulgaris</i>	La	La(NO ₃) ₃	96h	14.80	–	Growth yield	EC ₂₀	n	[5]
<i>Chlorella vulgaris</i>	La	La(NO ₃) ₃	96h	7.55	–	Growth yield	EC ₁₀	n	[5]
<i>Phaeodactylum tricornutum</i>	La	La(NO ₃) ₃	96h	13.10	–	Growth yield	EC ₅₀	n	[5]
<i>Phaeodactylum tricornutum</i>	La	La(NO ₃) ₃	96h	5.27	–	Growth yield	EC ₂₀	n	[5]
<i>Phaeodactylum tricornutum</i>	La	La(NO ₃) ₃	96h	2.91	–	Growth yield	EC ₁₀	n	[5]
<i>Phaeodactylum tricornutum</i>	La	La(NO ₃) ₃	72h	17.70	6.26 – 52.80	Growth (fluorescence activity)	EC ₅₀	m	[6]
<i>Phaeodactylum tricornutum</i>	Eu	Eu(NO ₃) ₃	72h	19.90	7.63 – 48.80	Growth (fluorescence activity)	EC ₅₀	m	[6]
<i>Phaeodactylum tricornutum</i>	Ce	Ce(NO ₃) ₃	72h	10.90	2.28 – 55.30	Growth (fluorescence activity)	EC ₅₀	m	[6]
<i>Phaeodactylum tricornutum</i>	Gd	Gd(NO ₃) ₃	72h	6.23	2.35 – 17	Growth (fluorescence activity)	EC ₅₀	m	[6]
<i>Phaeodactylum tricornutum</i>	Nd	Nd(NO ₃) ₃	72h	22.10	14.50 – 33.80	Growth (fluorescence activity)	EC ₅₀	m	[6]
<i>Phaeodactylum tricornutum</i>	Dy	Dy(NO ₃) ₃	72h	24.70	10.30 – 59.70	Growth (fluorescence activity)	EC ₅₀	m	[6]
<i>Phaeodactylum tricornutum</i>	Sm	Sm(NO ₃) ₃	72h	87.90	6.85 – 920	Growth (fluorescence activity)	EC ₅₀	m	[6]

(Continued)

Table 5. Continued.

Species	REE	Compound	Exposure time	Effective concentration (µM)	Effective concentration interval (µM)	Biological endpoint	Effective concentration	Concentration type	Reference
<i>Phaeodactylum tricornutum</i>	Er	Er(NO ₃) ₃	72h	9.27	49.00–49	Growth (fluorescence activity)	EC ₅₀	m	[6]
<i>Skeletonema costatum</i>	La	LaCl ₃	72h	29.20	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Ce	Ce(NO ₃) ₃	72h	29.70	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Nd	NdCl ₃	72h	30.30	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Sm	SmCl ₃	72h	28.70	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Eu	Eu(NO ₃) ₃	72h	29.20	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Gd	Gd(NO ₃) ₃	72h	29.80	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Tb	TbCl ₃	72h	28.50	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Dy	DyCl ₃	72h	28.30	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Ho	HoCl ₃	72h	29.30	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Er	Er(NO ₃) ₃	72h	28.70	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Tm	TmCl ₃	72h	28.80	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Yb	YbCl ₃	72h	28.50	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Lu	LuCl ₃	72h	28.60	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Sc	ScCl ₃	72h	21.90	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Y	Y(NO ₃) ₃	72h	43.20	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	La	LaCl ₃	96h	29.20	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Y	Y(NO ₃) ₃	96h	43.20	–	Growth: Cell density	EC ₅₀	n	[7]
<i>Skeletonema costatum</i>	Sc	ScCl ₃	96h	21.90	–	Growth: Cell density	EC ₅₀	n	[7]
Invertebrates									
<i>Arbacia lixula</i> (e)	Gd	Gd(CH ₃ CO ₂) ₃	48hpf	2.10	–	Embryotoxicity	EC ₅₀	n	[8]
<i>Arbacia lixula</i> (e)	Y	YCl ₃	72hpf	4.00	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Arbacia lixula</i> (e)	La	LaCl ₃	72hpf	15.50	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Arbacia lixula</i> (e)	Ce	CeCl ₃	72hpf	1.90	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Arbacia lixula</i> (e)	Nd	NdCl ₃	72hpf	1.50	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Arbacia lixula</i> (e)	Sm	SmCl ₃	72hpf	1.91	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Arbacia lixula</i> (e)	Eu	EuCl ₃	72hpf	1.47	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Arbacia lixula</i> (e)	Gd	GdCl ₃	72hpf	1.55	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Centrostephanus rodgersii</i> (e)	Gd	Gd(CH ₃ CO ₂) ₃	48hpf	132	–	Embryotoxicity	EC ₅₀	n	[8]
<i>Helicidaris tuberculata</i> (e)	Gd	Gd(CH ₃ CO ₂) ₃	48hpf	56.00	–	Embryotoxicity	EC ₅₀	n	[8]
<i>Paracentrotus lividus</i> (e)	Gd	Gd(CH ₃ CO ₂) ₃	48hpf	1.18	–	Embryotoxicity	EC ₅₀	n	[8]
<i>Paracentrotus lividus</i> (e)	Y	YCl ₃	72hpf	0.79	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Paracentrotus lividus</i> (e)	La	LaCl ₃	72hpf	0.66	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Paracentrotus lividus</i> (e)	Ce	CeCl ₃	72hpf	5.30	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Paracentrotus lividus</i> (e)	Nd	NdCl ₃	72hpf	1.51	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Paracentrotus lividus</i> (e)	Sm	SmCl ₃	72hpf	4.84 × 10 ⁻⁰⁷	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Paracentrotus lividus</i> (e)	Eu	EuCl ₃	72hpf	1.96	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Paracentrotus lividus</i> (e)	Gd	GdCl ₃	72hpf	0.20	–	Embryotoxicity	EC ₅₀	m	[9]
<i>Sphaerechinus granularis</i> (e)	Ce	CeCl ₃	48hpf	13.00	–	Embryotoxicity	EC ₅₀	n	[10]

(Continued)

Table 5. Continued.

Species	REE	Compound	Exposure time	Effective concentration (µM)	Effective concentration interval (µM)	Biological endpoint	Effective concentration	Concentration type	Reference
<i>Sphaerechinus granularis</i> (e)	Dy	DyCl ₃	48hpf	1.60	-	Embryotoxicity	EC ₅₀	n	[10]
<i>Sphaerechinus granularis</i> (e)	Er	ErCl ₃	48hpf	0.70	-	Embryotoxicity	EC ₅₀	n	[10]
<i>Sphaerechinus granularis</i> (e)	Gd	GdCl ₃	48hpf	0.90	-	Embryotoxicity	EC ₅₀	n	[10]
<i>Sphaerechinus granularis</i> (e)	Ho	HoCl ₃	48hpf	1.90	-	Embryotoxicity	EC ₅₀	n	[10]
<i>Sphaerechinus granularis</i> (e)	Lu	LuCl ₃	48hpf	1.80	-	Embryotoxicity	EC ₅₀	n	[10]
<i>Sphaerechinus granularis</i> (e)	Yb	YbCl ₃	48hpf	4.50	-	Embryotoxicity	EC ₅₀	n	[10]
<i>Sphaerechinus granularis</i> (e)	Y	YCl ₃	72hpf	0.47	-	Embryotoxicity	EC ₅₀	m	[9]
<i>Sphaerechinus granularis</i> (e)	La	LaCl ₃	72hpf	0.06	-	Embryotoxicity	EC ₅₀	m	[9]
<i>Sphaerechinus granularis</i> (e)	Ce	CeCl ₃	72hpf	1.33	-	Embryotoxicity	EC ₅₀	m	[9]
<i>Sphaerechinus granularis</i> (e)	Nd	NdCl ₃	72hpf	1.70	-	Embryotoxicity	EC ₅₀	m	[9]
<i>Sphaerechinus granularis</i> (e)	Sm	SmCl ₃	72hpf	0.41	-	Embryotoxicity	EC ₅₀	m	[9]
<i>Sphaerechinus granularis</i> (e)	Eu	EuCl ₃	72hpf	0.96	-	Embryotoxicity	EC ₅₀	m	[9]
<i>Sphaerechinus granularis</i> (e)	Gd	GdCl ₃	72hpf	5.56	-	Embryotoxicity	EC ₅₀	m	[9]
<i>Mytilus galloprovincialis</i> (e)	La	LaCl ₃	96h	0.14	-	Embryo development	EC ₅₀	n	[11]
<i>Mytilus galloprovincialis</i> (e)	Y	YCl ₃	96h	2.64	-	Embryo development	EC ₅₀	n	[11]
<i>Crassostrea gigas</i> (e)	La	La ₂ O ₃	24h	0.05	-	Embryo development	EC ₅₀	m	[12]
<i>Crassostrea gigas</i> (e)	La	La ₂ O ₃	48h	0.23	-	Embryo development	EC ₅₀	m	[12]
<i>Crassostrea gigas</i> (e)	Y	Y ₂ O ₃	24h	1.65	-	Embryo development	EC ₅₀	m	[12]
<i>Crassostrea gigas</i> (e)	Y	Y ₂ O ₃	48h	2.50	-	Embryo development	EC ₅₀	m	[12]

Dash (-) indicates missing or incomplete information. e=Embryo, j=Juvenile, 1 =Adopted to current names, m =measured concentration, n =nominal concentration, hpf= hours post-fertilization. Detailed literature is available at the end of the table. ECx=effective concentration at x%, LCx=lethal concentration at x%, and ICx=Inhibitory concentration at x%.
 References: [1] (Kurvet et al., 2017), [2] (González et al., 2015), [3] (Weltje et al., 2004), [4] (McCloskey et al., 1996), [5] (Sun et al., 2019), [6] (Siciliano et al., 2022), [7] (Tai et al., 2010), [8] (Martino, Bonaventura, et al., 2017), [9] (Trifuoggi et al., 2017), [10] (Gravina et al., 2018), [11] (Mestre et al., 2019), [12] (Moreira et al., 2020).

2004). The general consensus between the studies on freshwater bacteria (Técher et al., 2020) and marine bacteria (González et al., 2015; Kurvet et al., 2017; Weltje et al., 2004) is that REE could be toxic to microbial cells in a concentration-dependent manner. Both *E. coli* and *A. fischeri* appear to follow the pattern that LREE are less toxic than HREE, with the exception of the LREE classified Sc (Técher et al., 2020).

A study found that the LREE inorganic compound La_2O_3 shows antimicrobial activity against the gram-positive bacterium *Staphylococcus aureus*, but not against the gram-negative species *E. coli* and *Pseudomonas aeruginosa* (Balusamy et al., 2012). This is in agreement with the study of Técher et al. (2020) that LREE are not very toxic to gram-negative bacteria and that is an interaction between the gram-positive bacterial cell wall and REE. HREE appear to have different effects on bacterial activity. It has been demonstrated that Y^{3+} can inhibit the ammonium oxidation rate of bacteria commonly used in wastewater treatment systems (Su et al., 2020). However, this inhibitory effect only occurs at concentrations higher than $1120\ \mu\text{M}$, suggesting that the ammonium-oxidizing bacteria (AOB) and the specific nitrate production rate are particularly resistant to Y^{3+} . Moreover, 300 and $500\ \mu\text{M}$ Gd could temporarily reduce the nitrite production of the gram-negative nitrifying bacterium *Nitrosomonas europaea* (Fujita et al., 2020). Thus, there are indications that elevated Gd concentrations as high as $360\ \mu\text{M}$ do not negatively impact AOB. More studies are needed to determine the toxicity of HREE to different freshwater bacteria and the consequences on bacterial activity in both wastewater treatment systems and the environment.

Microalgae. The impact of REE on freshwater algae has been well studied over the last 10 years and several hypotheses have been developed to explain the toxicity to microalgae. Several studies identified a similar sensitivity for REE among different microalgae species (Table 4 and Table 5; Bergsten-Torralba et al., 2020; Joonas et al., 2017; Tai et al., 2010). While some studies have reported that REE exert similar toxicity to microalgae (Joonas et al., 2017; Tai et al., 2010), others have indicated that REE toxicity correlates positively with the atomic number (Bergsten-Torralba et al., 2020; González et al., 2015). For example, a stronger sensitivity of *C. vulgaris* to Sm (atomic number = 62, EC_{50} $170\ \mu\text{M}$) than for La and Nd (atomic number = 57 and 60, respectively, and EC_{50} 340 and $380\ \mu\text{M}$, respectively) was reported by Bergsten-Torralba et al. (2020). This difference in toxicity could be explained by a difference in the biodistribution in the microalgal cells. Indeed, Řezanka et al. (2016), using fluorescence microscopy found that La and Gd mainly biodistributed in the cytoplasm of *Desmodesmus quadricauda*, while Nd and Ce were localized in their chloroplasts. Additional studies are necessary to validate this hypothesis.

REE toxicity to algae depends on their capacity to form complexes and eventually precipitate in the presence of hydroxides, phosphates, and carbonates. REE precipitation with phosphate not only complicates the process of assaying toxicity, but also limits the availability of this nutrient for algae, leading to indirect effects on their growth (Goecke & Goecke, 2016; González et al., 2015). Sequestration of nutrients from algal growth media by REE has clearly been demonstrated in several studies (González et al., 2014, 2015; Tai et al., 2010).

REE presence exhibits intriguing dual effects due to their chemical similarities with certain elements. REE can act as competitors and cause adverse effects on algae. However, REE also have the potential to alleviate Ca deficiency by reacting with Ca^{2+} receptors, influencing algal metabolism (Goecke et al., 2015; Li et al., 2011). Moreover, the high affinity of REE for phosphate allows interactions with algal phospholipids in cell membranes, leading to metal agglomeration on the membrane surface and potentially inducing nutrient deficiencies that hinder growth and photosynthesis (Joonas et al., 2017).

Low concentrations of NdCl_3 have been shown to enhance the photosynthetic rate and to increase the total chlorophyll content, albeit with a decrease in the chlorophyll a/b ratio (Řezanka et al., 2016). Conversely, higher concentrations of NdCl_3 inhibit the photosynthetic rate, indicating potential consequences for plant growth (Goecke et al., 2015; Joonas et al., 2017).

In *Microcystis aeruginosa*, low doses of La have been observed to promote the production of microcystins (MCs), while high doses decrease this process (Liu et al., 2020; Shen et al., 2018). This modulation is accompanied by significant changes in the ratios of different MC variants. The activation of clathrin-mediated endocytosis by La enables the absorption of essential elements, stimulating algal growth, photosynthesis, and MC production (Liu et al., 2020). The ability of REE to promote growth at low concentrations has also been shown in other algal species (e.g., *D. quadricauda*) and terrestrial plants (e.g., *Glycine max* and *Oryza sativa*; de Oliveira et al., 2015; Ramírez-Olvera et al., 2018; Řezanka et al., 2016), and explains why REE are currently used as fertilizers in some countries, including China (Tommasi et al., 2020).

Invertebrates. The highest concentrations of REE are often found at lower trophic levels, especially in aquatic invertebrates (MacMillan et al., 2017). Aquatic invertebrates are more likely to be at risk from REE discharges in aquatic ecosystems because they are among the most sensitive organisms to REE at the first consumer level compared to other taxonomic groups (Bergsten-Torralba et al., 2020; Herrmann et al., 2016). However, other results indicate that crustaceans are the least sensitive species when using standardized protocols compared with cnidarians, rotifers, algae, and bacteria (González et al., 2015), they are among the most studied aquatic organisms in ecotoxicology.

The nematode *C. elegans*, even though not considered an aquatic organism, has been used very often as a model organism to assess aquatic toxicity because of its easy use, short life cycle, cellular simplicity and sensitivity (Tejeda-Benitez & Olivero-Verbel, 2016; Williams & Dusenbery, 1990). Tataru et al. (1998) reported that after exposure to La for 24 h, the half-maximal lethal concentration (LC₅₀) for *C. elegans* is 9.72 μM. Zhang et al. (2010) found that LaCl₃ has adverse effects on the growth, brood size, and number of eggs in the body of *C. elegans*, but it does not cause mortality. They reported a La EC₅₀ for brood size of 1.42 μM, making it the most sensitive endpoint for La exposure (Table 4). Several studies suggest that La, Nd, Pr, and Sc can reduce the body length and brood size of *C. elegans* without causing any mortality (Wakabayashi et al., 2016; Xu et al., 2017; Zhang et al., 2010). Sc and Lu are more toxic than the other REE (Lan et al., 2023), but less toxic than Cu (Wakabayashi et al., 2016), suggesting that these two REE may not have the same mode of action and negative effects as other REE.

Various studies have revealed that neurophysiological processes such as locomotor frequencies of body bends, head thrashes and pharyngeal pumping in *C. elegans* are affected by REE (Han et al., 2022; Xu et al., 2017). Although the life stage of *C. elegans* influences the sensitivity (Han et al., 2022), Wakabayashi et al. (2020) found that a variety of neurons are involved in the REE avoidance behavior of *C. elegans*. The nematodes use these chemosensory neurons to improve their avoidance of REE ions. Although clear avoidance of Y and all lanthanides was noted, this is not the case for Sc, perhaps due to the relative high toxicity and permeability of Sc³⁺ into the body of *C. elegans* compared with other REE ions (Wakabayashi & Nakano, 2019; Wakabayashi et al., 2016). The chemosensory system used by *C. elegans* to avoid REE ions is similar to that used to avoid heavy metal ions. However, the avoidance response for both REE and heavy metal ions only partially overlap, suggesting that the avoidance mechanisms used by *C. elegans* for REE is specific (Wakabayashi et al., 2020).

There is currently only one study that has addressed the environmental toxicity of REE salts through the oligochaete freshwater sludge worm *Tubifex tubifex* express test. Rucki et al. (2021) studied the movement inhibition (EC₅₀) of the worms by exposing the worms to all REE except Sc at five different concentrations for 3 min. The acute toxicity for *T. tubifex* shows an EC₅₀ value around 1.11 M for almost all the tested REE (Table 4), comparable to the results found for barium and cadmium salts, meaning that REE could be classified as exerting acute toxic effects on the aquatic environment.

REE toxicity to *Daphnia* spp. has been particularly well documented (Table 4). However, differences in sensitivity have been found among crustacean species. For example, *Hyalella azteca* seems to be more sensitive to REE than *Daphnia* spp. (Vukov et al., 2016). Contradictory

information can be found regarding the difference of toxicity among the REE to *Daphnia*. Several studies suggested a difference of toxicity among these elements to daphnids, with Nd^{3+} appearing to be the most toxic element (Bergsten-Torralba et al., 2020; Egler et al., 2023; Ma et al., 2016). In contrast, other studies have calculated similar toxicity to daphnids for all REE (Blinova, Lukjanova, et al., 2018; Lachaux, Catrouillet, et al., 2022). Similar EC_{50} values of $20\ \mu\text{M}$ for Nd, Gd, and Yb were obtained after 48h exposure of *D. magna* (Lachaux, Catrouillet, et al., 2022). Moreover, they reported similar toxicity when testing REE individually as well as in mixtures. In contrast, Hanana et al. (2022) reported an antagonistic effect of an REE mixture (La, Ce, Pr, Nd, and Sm) on *Hydra vulgaris*, with the mixtures showing less toxicity than the sum of the toxicity of each REE. This implies that La, Ce, Pr, Nd, and Sm compete with the same physiological targets but have different toxic outcomes. *H. vulgaris* is sensitive toward REE exposure. Furthermore, the LC_{50} calculated by Blaise et al. (2018) for CeCl_3 ($8860\ \mu\text{M}$; Table 4) is similar to what Blinova, Lukjanova, et al. (2018) reported for chronic tests with *D. magna*. It indicates that cnidarians are similar to daphnids regarding their chronic toxicity responses toward REE.

There have been very few studies evaluating the chronic toxicity of REE to crustaceans. Of the available studies, *Daphnia* are by far more sensitive to REE during chronic tests compared with acute tests (Barry & Meehan, 2000; Blinova, Lukjanova, et al., 2018a; Ma et al., 2016). Only one long-term multigenerational study is available (Galdiero et al., 2019). The authors reported that both Ce and Er reduce the survival, growth, and reproduction of *D. magna* (3.85 and $2.83\ \mu\text{M}$, respectively). They noted more pronounced negative effects of Er compared with Ce, suggesting the presence of phenotypic adaptative mechanisms (e.g., detoxification), supported by the decrease in the number of dead organisms at the end of each generation of daphnid.

The different conclusions found in the literature could be due to the use of different chemical forms of REE, which seem to have dissimilar toxicity to crustaceans (Blinova, Vija, et al., 2018). However, they could also be due to differences in REE speciation (Borgmann et al., 2005; Loveridge et al., 2021; Vukov et al., 2016). In standardized acute test (OECD, 2004), La precipitates more than Gd, leading to a difference in metal biodistribution and toxicity to *D. magna* (Revel et al., 2023). Therefore, researchers now recommend considering metal speciation and to measure dissolved concentrations when calculating the EC_{50} (Lachaux, Cossu-Leguille, et al., 2022). This is in agreement with a study that examined the toxicity to REE on *H. azteca*: Loveridge et al. (2021) demonstrated that the presence of organic matter increases the 96h LC_{50} of Tm due to stronger metal complexation.

Vertebrates. A large number of different effects have been described for fish and the toxicity of REE varies depending on the fish species, the exposed life stage, and the type of REE (Máková et al., 2011). Except when studying fish cells (Fleurbaix et al., 2022), REE toxicity to fish generally increases with the atomic number (Table 4; Cui et al., 2012; Dubé et al., 2019; Hanana et al., 2021). This toxicity can be explained by the similar ionic radius of REE and Ca^{2+} , leading to competition on the action site and perturbing different cell functions of the organism. Furthermore, REE can affect the expression of *SPARC3*, which encodes a glycoprotein that binds Ca and participates in the calcium homeostasis (Dubé et al., 2019; Hanana et al., 2021). Consistently, La can perturb Ca homeostasis by inhibiting calcium influx in killifish (Zimmer et al., 2019).

Five REE chlorides (Ce, Er, Gd, La, Nd, and Sm) affect the expression of several genes in the liver of juvenile rainbow trout (Dubé et al., 2019). These alterations lead to perturbations, such as cell growth arrest, DNA and protein damage, cell proliferation, disturbed Ca homeostasis and metabolism, as well as disruption of the detoxification pathway mediated by hemoproteins and protein chaperones. Furthermore, Hanana et al. (2021) identified that the presence of Dy or Lu activates the xenobiotic detoxification pathways of rainbow trout juveniles. GdCl_3 triggers lipid peroxidation (LPO), antioxidant gene activity, and oxidative stress in trout hepatocytes (Laville et al., 2004). On the contrary, La does not cause oxidative stress on glass eels (*Anguilla anguilla*) due to a good protection against reactive oxygen species (ROS) activity (Figueiredo et al., 2018).

REE cause severe physiological and histological alterations in a concentration- and exposure time-dependent manner (Chen et al., 2020; Correia et al., 2019; Cui et al., 2012; Gong et al., 2021; Hua et al., 2017; Su et al., 2022). For example, Nd activates apoptosis in the neurons of *Danio rerio* embryos while significant cerebrovascular arrangement structure changes and the cerebrovascular disappearance was observed which could be affected by the autophagy down-regulation flux in the cerebrovascular vessels (Chen et al., 2020). Furthermore, the activity of acetylcholinesterase (AChE) was significantly higher in La^{3+} exposed glass eels, which suggests that La reduces the acetylcholine concentration in the brain of the fish (Figueiredo et al., 2018), leading to neurotoxic effects.

The gills and liver appear to be particularly affected by REE exposure (Correia et al., 2019; Hua et al., 2017). Severe histopathological changes were observed in the gills and liver of rare minnow (*Gobiocypris rarus*) after exposure to La for 21 days, suggesting some metabolic disturbance (Hua et al., 2017). Moreover, LaCl_3 inhibits mitochondrial energy turnover in the liver of goldfish (*Carassius auratus*; (Wu et al., 2015). These alterations are accompanied by abnormal behavior, which could be due to nervous system damage (Hua et al., 2017).

A few studies have investigated the effect of REE mixtures on freshwater fish. Exposure for 96 h to binary mixtures (Nd, Gd, and Yb) resulted in a notable synergistic effect on fish cell viability, suggesting that the combined presence of these lanthanides has a greater impact than their individual effects (Fleurbaix et al., 2022). Similarly, additive effects of La and Gd mixtures were observed for *Danio rerio* (Kang et al., 2022).

Marine conditions

Research on the toxicity of REE to marine organisms is much more limited compared with freshwater environments. While REE bioaccumulation studies have been conducted, the focus on their toxic effects has been sparse, particularly regarding marine vertebrates. This knowledge gap highlights the need for further investigation to understand the potential risks and ecological implications of REE in marine ecosystems.

Bacteria. Most studies that address the effect of REE on marine bacterium have focused on the EC_{50} (González et al., 2015; Kurvet et al., 2017; Weltje et al., 2004) for the naturally luminescent gram-negative bacteria species *A. fischeri* (also known as *Vibrio fischeri* and *Photobacterium phosphoreum*). For this organism, the toxicity of REE increases with the atomic number (Table 5; González et al., 2015; Kurvet et al., 2017). For instance, González et al. (2015) calculated a nominal EC_{50} after 30 min of exposure of $18.30\ \mu\text{M}$ for Lu, $47.70\ \mu\text{M}$ for Ce, and $40.70\ \mu\text{M}$ for Gd (Table 5). The EC_{50} for Gd is slightly higher than $22.40\ \mu\text{M}$ estimated by Kurvet et al. (2017).

It was also hypothesized that the kinetics of the luminescence of the bacteria could act as a good mechanistic toxicity endpoint (Kurvet et al., 2017). Indeed, it reflects early changes in the bacterial membrane due to the exposure to REE and the high affinity of REE to phosphates. According to the acute kinetics of *A. fischeri* bioluminescence, the toxicity of REE is triggered by disturbing the integrity of the cell membrane. However, Kurvet et al. (2017) and González et al. (2015) stated that REE do not appear to have harmful effects on marine bacteria at the current environmental concentrations and forms (e.g. insoluble salts) with exception of hotspots or where peak concentrations occur.

Bacteria are particularly sensitive to REE in the presence of organic ligands. Indeed, the effect of Lu on the bioluminescence of *A. fischeri* decreases in the presence of small organic molecules such as citrate, malate, and oxalate (Weltje et al., 2004). However, the authors estimated a low elimination rate of free Lu^{3+} and calculated an EC_{50} of $1.57\ \mu\text{M}$ for Lu^{3+} after 15 min of exposure. This finding suggests that the free ion Lu^{3+} is more toxic than the free ions of bivalent metals such as Cd^{2+} or Zn^{2+} (Table 5; McCloskey et al., 1996). This hypothesis is contrary to many studies that suggest that in general REE have a lower toxicity than Cd, Cu, Pb and Zn for the marine bacterium *A. fischeri* (Newman & McCloskey, 1996).

Microalgae. REE exert varying effects on the growth of *Skeletonema costatum*, with an EC_{50} ranging from 21.90 to 43.20 μM (Tai et al., 2010). The authors also evaluated the toxicity of a mixed solution containing the same amount of the 13 REE on *S. costatum*. The EC_{50} of this mixture was not significantly different from the toxicity of single REE. Similar sensitivity to REE was found among two marine microalgae species such as *Phaeodactylum tricornerutum* (Siciliano et al., 2022) and the diatom *S. costatum* (Tai et al., 2010). In the latter study, the authors found no interactions when examining REE in mixture (Tai et al., 2010). However, Sun et al. (2019) demonstrated that the toxicity of $\text{La}(\text{NO}_3)_3$ varies with the algal species, with a nominal EC_{50} of 23.30 and 13.10 μM for *C. vulgaris* and *P. tricornerutum*, respectively (Table 5). The authors examined the mechanism of toxicity of REE by measuring fluorescence yield and antioxidant responses. The fluorescence of *P. tricornerutum* decreased rapidly compared with *C. vulgaris* at the same concentration of $\text{La}(\text{NO}_3)_3$.

Exposure to La increases in a dose-dependent manner, the activity of four enzymes involved in the antioxidant responses of *P. tricornerutum*, namely catalase (CAT), superoxide dismutase (SOD), peroxidase (POD), as well as glutathione (GSH; Sun et al., 2019). This study also demonstrated that the destruction of photosystem II (PS II) and the antioxidant system mediated by $\text{La}(\text{NO}_3)_3$ are responsible for the growth inhibition in algae. However, after 48 h photosynthesis was restored while oxidative stress remained present and affected the growth of algae in the longer term. The oxidant stress and destruction of PS II as a reason for the inhibition of algae growth, would imply the importance to study the impact of REE on essential elements for marine algae, just as it has been done for freshwater algae.

Invertebrates. Most of the marine invertebrates studied to determine REE toxicity are classified as macrozoobenthos. REE are known to strongly affect sea urchin embryos, and the effects vary depending on the REE, concentration, and sea urchin species (Table 5; Gravina et al., 2018; Martino, Bonaventura, et al., 2017; Oral et al., 2017; Pagano et al., 2016; Trifuoggi et al., 2017). Martino, Bonaventura, et al. (2017) compared the LC_{50} of Gd for four species. Of the four species, they found that *Heliocidaris tuberculata*, which naturally develops a more extensive skeleton, is the most sensitive to Gd exposure ($LC_{50} = 5.60 \times 10^{-2} \mu\text{M}$). Because Gd^{3+} has a similar ionic radius to Ca^{2+} , it can block Ca^{2+} channels (Sherry et al., 2009). If Gd^{3+} blocks Ca^{2+} channels, then it could lead to a calcification response and the development of skeletal abnormalities. Therefore, species with a high need for Ca could be more impacted compared with species with a lower need. This variable response in sea urchin species highlights the importance of evaluating the toxicity of REE on more than one species.

Studies using sea urchins have also revealed REE exposure has detrimental effects on embryogenesis, including inhibition of the mitotic activity and an increase in mitotic aberrations (Oral et al., 2017; Pagano et al., 2016), oxidative stress (Pagano et al., 2016), and/or skeletal abnormalities in embryos (Gravina, et al., 2018; Martino, Bonaventura, et al., 2017; Martino, Chiarelli, et al. 2017; Martino et al., 2018; Oral et al., 2017; Saitoh et al., 2010).

Bivalves are also macrozoobenthic organisms commonly used to study REE toxicity for marine invertebrates. Ecotoxicity tests on embryos and juveniles of oyster *Crassostrea gigas* (Moreira et al., 2020) and the mussel *Mytilus galloprovincialis* (Mestre et al., 2019) showed that La exerted significantly higher toxicity than Y. The La toxicity for *C. gigas* had an EC_{50} of $4.82 \times 10^{-2} \mu\text{M}$ after 24 h and 0.26 μM after 48 h. The EC_{50} for Y was at 24 h 1.65 μM and at 48 h 2.50 μM (Table 5). Thus, La is more toxic than Y to the *C. gigas* developing embryos (Moreira et al., 2020). On the other hand, (Mestre et al., 2019) found that La is more toxic than Y to the developing embryos of *M. galloprovincialis* than to juveniles, while Y appears to be more toxic to juveniles than to embryos. The toxic concentration of an REE can vary by a factor of 100 for the different life-cycle stages of these bivalves (Mestre et al., 2019). However, more ecotoxicological data on macrozoobenthos will be needed to further develop ecological risk assessment of REE. This should not be limited to EC_{50} based on mortality; it should also evaluate development and/or metabolic and oxidative stress.

Several studies have measured the capacity of REE to trigger oxidative stress through LPO. Compared with control organisms, the marine bivalve *M. galloprovincialis* shows significantly higher LPO when exposed to Gd (Andrade et al., 2022a; Henriques et al., 2019; Trapasso, Coppola, et al., 2021). There were similar results for this bivalve species when exposed to Nd (Freitas, Costa, et al., 2020) and Dy (Freitas, Cardoso, et al., 2020). LPO decreases significantly in *M. galloprovincialis* exposed to La (Pinto et al., 2019), although Andrade et al. (2022b) did find an increase in LPO levels at $7.20 \times 10^{-2} \mu\text{M}$ after 28 days of exposure. For Tb no cellular damages on the lipids were found in *M. galloprovincialis* regardless of the concentrations tested up to $0.25 \mu\text{M}$ for 28 days (Andrade et al., 2023).

Additional research is necessary to elucidate the effects of REE in combination with multiple stressors—for example, the rise in temperature caused by climate change. Several studies have found that La and Gd in combination with other climate-related stressors, such as increased temperature and variable salinity, depress metabolism, activate glutathione S-transferases (GSTs), inhibit antioxidant enzymes, and increase LPO in *M. galloprovincialis* and *Spisula solida* (Andrade et al., 2021, 2022a, 2022b, 2023; Figueiredo et al., 2022).

Effects from exposure to REE in sediments

Anthropogenic REE anomalies in sediments can reach high concentrations (Table 2), as ions in the water phase adsorb to the surface of particles and solid complexes are formed and deposited. In experiments with microcosms, it was found that after 12 h more than 80% of the spiked REE (La, Ce, Sm, Gd, and Y) were in the sediments (Yang et al., 1999). Metal uptake with food particles seems to be an important exposure pathway for benthic organisms and collector-gatherers, among macrobenthic invertebrates, have been suggested as good tracers of REE in freshwater systems (Pastorino et al., 2020). While bioaccumulation or REE from sediments has been studied frequently (“Uptake of REE and occurrence in biota” section), information on the toxicity of REE in sediments remains scarce.

A number of studies have demonstrated elevated toxicity in REE-contaminated environmental sediment samples (Dickman & Rygiel, 1996; Romero-Freire et al., 2018). The authors have concluded that it is even more difficult to determine the relative toxicity of REE in sediments compared with water samples due to the large number of potential contaminants in sediments with different adsorption kinetics, and the complexity of sediment geochemistry. Toxicity-testing of spiked sediments would facilitate understanding of cause-effect relationships (USEPA, 2000). In the case of REE, however, this endeavor has been restricted to three studies: They were all carried out with La on behalf of a company (Grace GmbH & Co. KG, Worms, Germany), so the results are not freely available.

Most macrozoobenthos species are exposed to REE through sediments due to the high adsorption of metal to sediment particles (Tijink & Yland, 1998; Weltje, Heidenreich, et al., 2002) and fine organic matter (Schaller, 2013). In general, the accumulation of REE is higher in benthic invertebrates compared with other species (Amyot et al., 2017; MacMillan et al., 2017). Two studies, Hanana et al. (2017) and Hanana et al. (2018), found no significant change in LPO levels in the freshwater zebra mussel *Dreissena polymorpha* exposure to Gd, Sm, and Y for 28 days. Although the levels of LPO were not significant for Gd there was still a mild increase observed for 37.90 and $0.19 \mu\text{M}$ (Hanana et al., 2017). These findings suggest that REE can induce cellular damage, which may compromise the physiological performance (e.g., growth and reproductive success) of bivalves.

Other tests have been performed with benthic organisms but with exposure to water only (e.g., Mestre et al., 2019; Rucki et al., 2021; Zhang et al., 2015). The challenges, when carrying out and interpreting sediment toxicity data, are to be aware of confounding factors that can alter the bioavailability and thus the toxicity of REE. The impact of physicochemical properties of sediments and medium composition on results of sediment contact tests was clearly demonstrated when testing sediments with naturally elevated REE concentrations (Romero-Freire et al., 2018). A study that investigated the influence of soil composition with REE and heavy metal

co-contamination. They found that shifts in microbial communities depend on both metal contamination and the physiochemical properties of soil (Luo et al., 2021). Thus, the physiochemical properties of soils and sediments in combination with REE can have major implications for ecological risk assessment.

Conclusion: Critical parameters and potential pitfalls in bioassays with REE

As metals with great technological applicability, REE are increasingly used and released into the environment. Due to the wide range of geogenic background concentrations in water and sediment (Table 1), anthropogenically elevated concentrations (Table 2) often are within the potential natural range but considerably exceed the natural median values. This gives rise to concerns regarding their potential impact on biological communities. REE are taken up by bacteria, algae, plants, invertebrates, and fish, but not enough is known yet about the species-specific toxicodynamics of the elements to relate elevated tissue concentrations to toxicity. The available evidence points to the chemical similarity of REE³⁺ and Ca²⁺ ions as an important cause for cytological effects, and La³⁺ is an efficient Ca-channel blocker. Oxidative stress and impairment of gene expression have also been demonstrated for various REE, but most of the mechanisms of action for the effects remain to be elucidated.

Experimental bioassays with REE which could serve to study cause-effect relationships have resulted in EC₅₀ values which partly differ by several orders of magnitude—even with the same biological species and for a specific REE—for example, EC₅₀ for *A. fischeri*: 48 μM of La (Kurvek et al., 2017) compared to 1690 μM of La (McCloskey et al., 1996); EC₅₀ of 9.14 μM (Joonas et al., 2017) compared to 372 μM of La (Bergsten-Torralba et al., 2020) for *R. subcapitata*. Figures 4 and 5 provide a visual overview of the ecotoxicity studies analyzed in this review.

Variations in exposure times, endpoints, and REE salts can complicate the comparison of bioassay results. Nevertheless, there are a number of challenges associated with REE bioassays and careful consideration must be given to how the data is being interpreted. A number of these aspects are covered in the sections that follow.

Complex formation in the presence of carbonates, phosphates, and hydroxides: As indicated by their high complex stability constants, REE form especially strong complexes with carbonates, phosphates, and hydroxides, leading to precipitation. Carbonates are often used as buffers in test media for bioassays with algae and daphnids. Phosphates are usually needed as inorganic nutrients in bioassays with algae. In combination with REE, complexes are formed and the concentration of free REE ions as well as nutrients is reduced. Whether this has led to an overestimation of REE toxicity in the past by confusing the effect of nutrient limitation with the effect of metals, or whether complex formation has masked the toxic potential of REE, should be investigated in the future. For instance, the toxicity of REE on microalgae could be investigated by using different media, containing various phosphate sources. With the help of speciation analysis, it may be possible to determine whether the observed toxicity is due to the direct effects of REE or due to phosphate limitation.

The impact of water hardness: Similarly to other metals, REE are affected by these confounding factors. Concentrations of Ca²⁺ and Mg²⁺ ions in water have been shown to influence the toxicity of many divalent metals (Borgmann et al., 2005; Pascoe et al., 1986), including REE³⁺. Barry and Meehan (2000) reported marked differences in La toxicity to *D. carinata* by 2 orders of magnitude based on the water hardness, with a 48h EC₅₀ of 0.31, 0.35, and 8.49 μM of La for water with a hardness of 220, 979, and 1600 μM of CaCO₃, respectively. Lower toxicity as water hardness increases may be caused by competition of REE³⁺ and Ca²⁺ for the same binding sites or by the decrease of potentially bioavailable species such as La³⁺ or LaOH²⁺ through carbonate

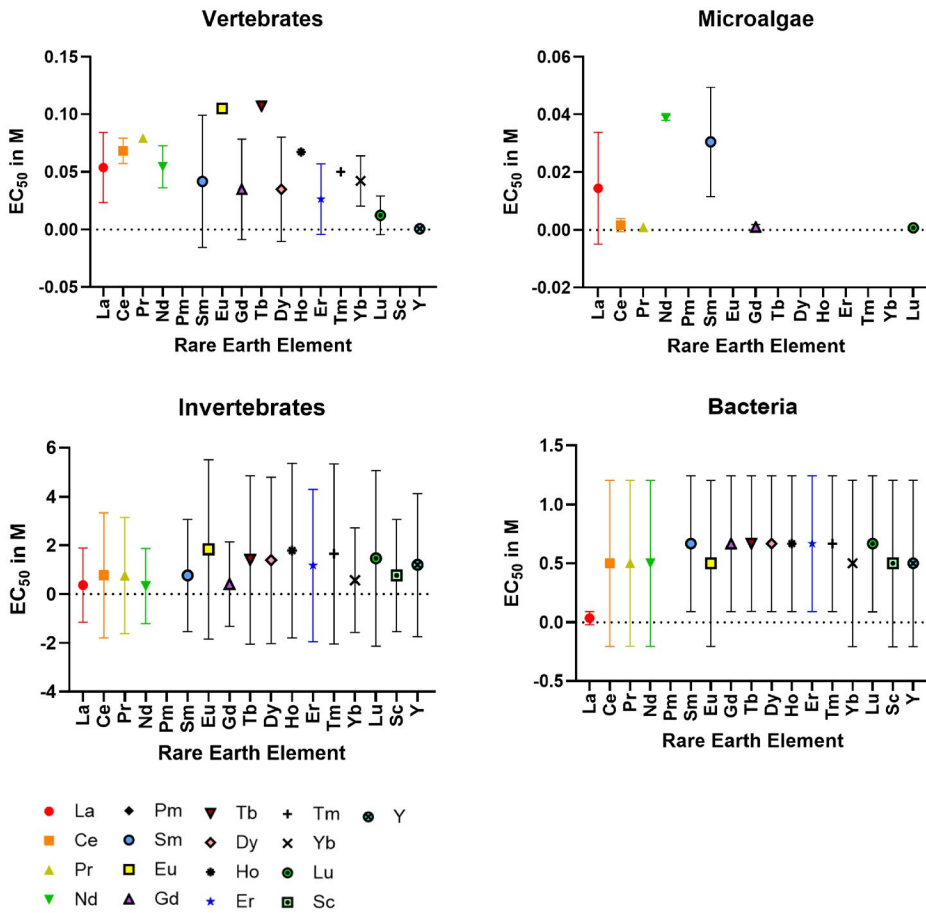


FIG. 4. Freshwater ecotoxicological data for vertebrates, microalgae, invertebrates, and bacteria, with the half maximal effective concentration (EC₅₀) presented in M. These graphs provide a highly simplified representation of Tables 4 and 5 in this review and should only be used as a visual tool and not as an actual representation of data.

complexation (Moermond et al., 2001). Linear dependence of REE toxicity to water hardness, however, has yet to be shown (Herrmann et al., 2016).

Influence of pH and use of buffer: pH strongly affects the bioavailability of REE. Consequently, accurate measurement and maintenance of pH levels become imperative in ensuring the consistency of toxicity studies. The ability of pH buffers to form complexes with REE varies greatly. For example, Eu(III) showed with MOPS (3-(N-morpholino)propane sulfonic acid) the highest complexation capacity, whilst TRIS (tris(hydroxymethyl)aminomethane) did not show a significant interaction (Mandal et al., 2022). However, metal sensitivity can also be altered if a buffer disturbs the physiology of organisms (De Schampelaere et al., 2004). Biological effects and metal-buffer complexation should both be considered when performing experiments (Ferreira et al., 2015).

Role of DOM on REE toxicity: Studying the effects of DOM on metal bioavailability and toxicity is important because organic matter is ubiquitous in natural environments. A low DOM concentration can increase the solubility of REE (Choppin, 1986; McCarthy et al., 1998), while high concentrations can reduce the bioaccumulation and toxicity of REE (El-Akl et al., 2015; Lachaux, Cossu-Leguille et al. 2022; Tang & Johannesson 2003; Vukov et al., 2016). Thus, DOM should be considered when assessing the toxicity of REE due to its influence on the bioavailability of

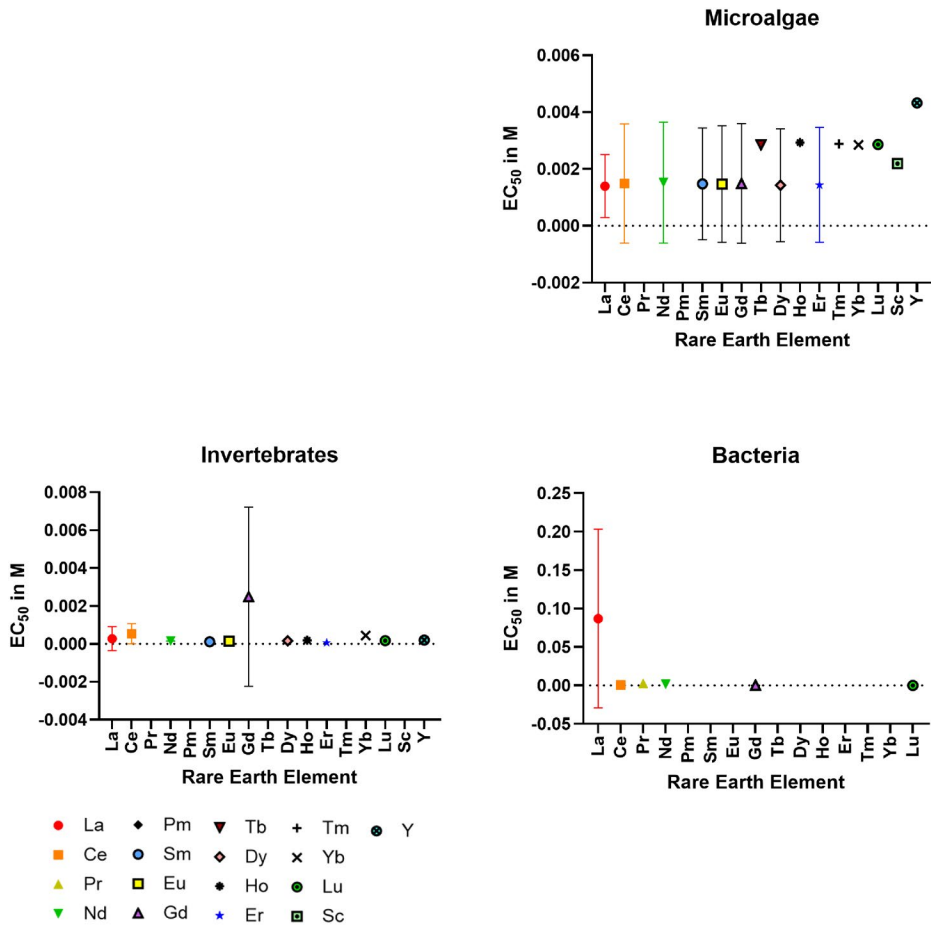


FIG. 5. Marine ecotoxicological data for microalgae, invertebrates, and bacteria, with the half maximal effective concentration (EC₅₀) presented in M. No marine vertebrate (e.g., fish) ecotoxicity data was available. These graphs provide a highly simplified representation of Tables 4 and 5 in this review and should only be used as a visual tool and not as actual representation of data.

the metals. There seems to be a large knowledge gap regarding the impact of DOM on REE. The environmental realism of bioassays could be enhanced by including DOM. Most studies do not consider biological processes such as the excretion of organisms or exudate formation by algae during biotests, and the presence of feces in natural ecosystems. There also appears to be a gap in the understanding of the effect of DOM on REE toxicity for chronic exposures, as most studies on DOM have focused on acute exposure (Lachaux et al., 2023; Lachaux, Cossu-Leguille et al., 2022; Vukov et al., 2016). To address this deficiency, researchers could perform long-term exposure experiments where organisms are exposed to REE following chronic standardized test guidelines (e.g., OECD 211 for *D. magna* or OECD TG 210 for fish) with exposure media containing natural DOM. Measuring the DOM composition and the dissolved REE concentration in the media over time, alongside conducting speciation analysis, could help to better understand the effects of organisms on REE availability and, consequently, the toxicity of REE to these organisms.

REE in mixtures: REE are rarely present as single elements in the natural environment; hence studying their behavior and toxicity in mixtures is critical. When in mixtures, REE bio-uptake

tend to decrease due to an increase of competition for the same biotic ligand (Aharchaou et al., 2020; Yang et al., 2014). Studies have obtained varying effects for REE when they are combined with additive effects (Lachaux, Cossu-Leguille, et al., 2022; Tai et al., 2010). Moreover, synergistic effect were recorded for fish cells, bacteria, and algae (Fleurbaix et al., 2022; Romero-Freire et al., 2018); and an antagonistic effect for rotifers (Romero-Freire et al., 2018) when exposed to REE mixtures. The underlying mechanisms or biological explanations for these antagonistic or synergistic effects remain to be fully elucidated.

Adsorption of REE: The adsorption of lanthanides to different surfaces has been studied in several studies (Eichholz et al., 1965; Weltje, Brouwer, et al., 2002). For La, glass surfaces can adsorb up to 25% of the total concentration (Weltje, Brouwer, et al., 2002). Thus, it is recommended to avoid glassware and to use materials such as PET (polyethylene terephthalate), polycarbonate, or nylon when performing bioassay experiments (Bau et al., 2010; Reimann et al., 2010; Weltje et al., 2003). However, there is limited knowledge regarding which materials and what specific material treatment is required to prevent REE adsorption on experimental vessels. Even when using less adsorbing REE vessels during experiments, REE can still adsorb strongly to organic surfaces such as sediments, organisms, and DOM (Birungi & Chirwa, 2014; Das et al., 1988; Gwenzi et al., 2018; Lachaux, Cossu-Leguille, et al., 2022; Martinez et al., 2018; Vijayaraghavan et al., 2009). The adsorption of REE can impact how we evaluate the toxicity of REE in our ecosystems.

Quantifying the exposure conditions: Researchers have noted the importance of considering measured concentrations over nominal concentrations. For example, the dissolved La EC_{50} (Siciliano et al., 2022) is four times higher than the nominal La EC_{50} (Sun et al., 2019) for microalgae. To use measured concentrations, we need a good understanding of the chemical speciation of REE before performing the experiments. In addition, determining the dissolved concentrations is often very time consuming and expensive, and there is a lack of understanding of the kinetics involved in precipitation. An understanding of the cause-effect relationship for the different chemical species on organisms is necessary.

With the increase in use of REE there has also been an increase in scientific scrutiny of these metals in various research fields. There is a need to have a deep understanding of the biochemical and physical processes; environmental behavior; and fate of REE in freshwater, marine, and sediment environments. Discussion on the environmental impact needs to address environmental concentrations, speciation, partitioning, and bioavailability relative to the ecotoxicity of the elements in question. Comparison and interpretation of REE toxicity data are also often complicated even more due to other confounding factors such as pH, metal precipitation, adsorption, and relevant exposure routes. The limited knowledge on chemical speciation and the interactions of REE with a wide range of biological systems, such as bacteria, microalgae, invertebrates, and vertebrates call for more systematic studies (e.g., the use of consistent and reproducible conditions to evaluate the effects of REE across different aquatic organisms). We recommend that more ecotoxicological research is performed on a wide variety of biological species across all levels of both the aquatic and marine ecosystems, while considering biochemical and physical processes, paying special attention to the pitfalls and critical parameters (i.e., pH, water hardness, or the presence of organic matter) when bio-testing REE. Not only are individual bioassays essential here but also further development across ecosystem levels and services. However, vertebrate studies should be limited as much as possible for animal welfare reasons. Most studies have focused on the nominal concentrations of La, Gd, and Ce. While there is also a need to look further into the mode of action of these lanthanides, the other REE should also be included in future studies. It will be fundamental for robust risk assessment to consider the nominal and measured REE concentrations for determining the acute and chronic ecotoxicological effects.

Authors' Contributions

Marion Revel: Conceptualization, Writing—original draft, Writing—review & editing. Chantal van Drimmelen: Conceptualization, Writing—original draft, Writing—review & editing. Lennart Weltje: Writing—original draft, Writing—review & editing. Andrew Hursthouse: Writing - review & editing. Susanne Heise: Conceptualization, Writing—original draft, Writing - review & editing.

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The authors report there are no competing interests to declare.

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