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Review

A Retrospection on Mercury Contamination, Bioaccumulation, and Toxicity in Diverse Environments: Current Insights and Future Prospects

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Abstract: Owing to various industrial applications of mercury (Hg), its release into the environment at high concentration is becoming a great threat to living organisms on a global scale. Human exposure to Hg is greatly correlated with contamination in the food chain through cereal crops and sea foods. Since Hg is a non-essential component and does not possess a biological role and exhibits carcinogenic and genotoxic behaviour, biomonitoring with a focus on biomagnification of higher living animals and plants is the need of the hour. This review traces the plausible relationship between Hg concentration, chemical form, exposure, bioavailability, bioaccumulation, distribution, and ecotoxicology. The toxicity with molecular mechanisms, oxidative stress (OS), protein alteration, genomic change, and enzymatic disruptions are discussed. In addition, this review also elaborates advanced strategies for reducing Hg contamination such as algal and phytoremediation, biochar application, catalytical oxidation, and immobilization. Furthermore, there are challenges to overcome and future perspectives considering Hg concentrations, biomarkers, and identification through the nature of exposures are recommended.

Keywords: mercury fate; methyl mercury conversion; bioaccumulation; ecotoxicity; biomarkers

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1. Introduction to Mercury Pollution

According to United States (US) Government Agency for Toxic Substances and Disease Registry (ATSDR), mercury is the third most toxic substance for humans. Around 450 contaminated sites are identified and an estimated 20 million people worldwide are at risk of Hg exposure, which is a concerning health risk [1]. In recent decades, robust scientific technology has explored the different factors affecting Hg transport, fate, conversion to methyl mercury (MeHg), mechanism, and effect of Hg exposure to different biota. This information collectively helped to gather global support to stop Hg pollution for the protection of human and environmental health. As a result, the Minamata convention formed in 2017, and provides better insight on the bio-magnification and toxic effects of Hg [2–4].

Due to increased use in various industries, mercury enters into the environment and its presence affects microbial activity, seed germination, and plant morbidity and can be absorbed by human bodies through the food chain, skin contact, and direct inhalation [5,6]. Hg mainly exists in three different forms: elemental, inorganic, and organic. Elemental mercury (Hg^0) is considered as class D carcinogen by United States Environmental Protection Agency and known as metallic mercury. It oxidizes to inorganic mercury and enters the environment. Hg^0 is found in high concentration in the atmosphere and can be active from 6 to 24 months [7]. It is found that mercuric sulphide in cinnabar ore can be extracted by heating at 538 °C. Liquid metal mercury is industrially used in thermometers, dental filling, production of chloride gas, and electrical parts. Elemental mercury can be ingested by mouth but the digestive tract cannot easily absorb it [8]; however, it can be inhaled through respiratory tract where Hg vapour enters the blood from lungs and affects human body. In contrast, water soluble inorganic Hg can be absorbed by the digestive tract after oral intact or skin contact [9]. Mercury chloride, sulphur sulphide, and mercury acetate are used to extract inorganic Hg. Coal-fired power plants and other coal burning plants are also responsible for high elemental and inorganic Hg release [10]. Hg^0 is uncharged and can pass through the blood–brain barrier where it is converted into inorganic divalent mercury (Hg^{2+}) and causes brain damage [11]. Human exposure to high amounts of inorganic Hg is very rare though inorganic salts of mercury which causes skin, eye irritation, and kidney toxicity [12]. MeHg and $[(CH3_3)_2Hg]$ are examples of highly toxic and the most frequently found organic form of Hg and are considered a class C carcinogen. Aquatic microbes convert Hg^0 into their organic form which is accumulated in fatty acids of many fish and fish-consuming animals which are consumed by humans. Due to its structural resemblance with methionine, it can be found in brain tissue after crossing the blood–brain barrier [13,14]. The MeHg exists in our food chain and can be absorbed by the digestive tract (95%), blood circulation, and skin contact (remaining 5%) and readily reaches the brain and other tissues of the body. Kidney problems such as renal dysfunctions, neurological dysfunction, impaired reproductive functioning, hearing loss, and sleep disturbance are some of the toxic effects of excessive Hg accumulation in the human body [8,15].

Human activity remobilizes the released Hg and enhances their chance of methylation. Thus, aquatic and terrestrial bioaccumulation of Hg results in increased exposure of Hg to human and animals [16]. It is even found in remote regions due to long-range transport capacity. Increased artisanal and small-scale handicraft gold mining are one of the main factors in Hg pollution in the environment. Although alterations in Hg cycling, MeHg bioavailability and trophic transfer is triggered by climate change and changed land usage [17,18]. The presented manuscript is an endeavour to explore the mercury fate, bioaccumulation, and human toxicity.

1.1. Sources of Mercury Pollution

Due to varied deposition of Hg and geographical features, it is distributed unequally around the world. Industrial processes such as mining, disposal of waste and molten metals, production of chemicals, fertilizers, and natural sources are the main sources of Hg pollution. But exposure to Hg can occur through different sources and routes. Religious and cultural practices, fossil fuel combustion, and gold mining are direct sources of Hg pollution in developing countries [19]. The different sources of Hg pollution are presented in Table 1.

Waste incineration, fossil fuel combustion, cement production, artisanal gold mining, and smelting of ores are identified as anthropogenic sources of Hg emission [30]. Heating of elemental mercury is carried out in small scale gold mining and emits Hg vapour to the atmosphere and the vapours are settled as dust on water and soil surface [31]. Inorganic Hg is converted into MeHg in the aquatic ecosystems making it an essential part of Hg biogeochemical cycle. Climate change also introduces the trophic transfer and methylation of Hg [32].

Table 1. Different sources of mercury pollution.

Exposure Groups	Sources	Reference
Atmospheric exposure	Burning of fossil fuels and coal	[20]
	Metal mining	[1]
	Fertilizers, pesticides, and other chemical manufacturing	[21]
	Incineration of domestic and industrial waste	[22]
	Volcanic eruption, first fire	[23]
Hydrosphere exposure	Entry of Hg to water bodies through effluents	[24]
	Discharges from oil refineries	[25]
Food products	Consumption of food products contaminated with Hg	[26]
Cosmetic products	Skin lightening creams, toothpaste, and soap	[27]
Medical products	Dental amalgam, antiseptics and some Ayurvedic medicines.	[28]
Consumer goods	Plastic, paint, batteries, lamps, switches, thermometers, bulbs	[29]

Terrestrial vegetation act as a significant reservoir in the biogeochemical cycle of atmospheric Hg. Increasing wildfires due to drastic climate change also mobilize the Hg trapped in the terrestrial ecosystem and releases high amounts of Hg and other pollutants into the atmosphere [18]. During the year 2000, in Africa, Eurasia, and South America, the estimated Hg emissions were 43%, 31%, and 18% respectively [33]. Increased cases of wildfire is predicted in Boreal region which will increase the Hg emission and its deposition in the Arctic [34]. As Hg can be exchanged at the air–water interface, thinning out of sea ice also can introduce Hg into the atmosphere. It is reported that the Hg concentration and contamination has considerably increased in the Arctic sea and plays an Important role in transport, distribution, and transformation [35]. A study reported that Hg can be mineralized, and it can be easily accumulated. In addition, it is converted to MeHg which is persistent in soil and sediments [36]. Hg present on the surface water bio-accumulates, and methylates in the aquatic food chain very easily within a year of time. In contrast, Hg deposited on land takes several years to be bio-accumulated into the food chain [26].

Organic mercury is the most hazardous form and found in food chain, pesticides, and insecticides. Accumulation of organic Hg (methyl or ethyl mercury) in fish, poultry, and rice grain is a major health concern. Hg levels are reported to be 1.54 and 2.95 mg/Kg in raw food samples collected from the Democratic Republic of Congo and South Africa, respectively. The fish sample collected from Palestine and South Africa has also shown higher concentration (by 0.5 mg/Kg) of Hg than recommended by the European Commission [37,38]. After making entry to the human body through food, MeHg crosses the blood–brain barrier and is accumulated in brain cells. A study showed that 50–80% of total Hg found in fish muscles is MeHg [39]. Inorganic Hg is found in other foods. The estimated Hg level in human adults is 0.40 mg/Kg due to the position and bio-magnification of Hg in the food chain [40]. A study compared the concentration of MeHg in fish collected from Mediterranean and Atlantic sea and observed higher concentration of mercury in Mediterranean fish [41]. Provisional Tolerable Weekly Intake has shown the accumulation due to consumption of Mediterranean fish is 110 and 140% higher in children and adults, respectively, than the recommended level. The total Hg level in human blood, hair, and fish muscles showed the estimated weekly intake of 0.63 µg/Kg body weight/Week and over 10 days the estimated intake ranges from 0.36 to 0.97 µg/Kg body weight [42]. It is also understood that climate change plays a passive role in increased exposure and bioaccumulation of pollutants like Hg [43].

Previously, Hg was mainly used in thermometers, but recently it has gained new application in Hg vapour lamps, tube lights, and compact fluorescent lamp industries. Release of Hg from industrial installations, artisanal small scale gold mining (ASMG), metal production, and careless disposal of Hg containing products along with domestic waste represent significant sources of Hg pollution [5]. Fluorescent lamps recycling units presents a health risk for their workers due to Hg exposure. It is reported that significant concentration of Hg is released when a fluorescent lamp is destroyed [1]. An estimated 15 million people including 3 million women and children in around 70 countries are associated with ASMG where the gold is extracted using elemental Hg. They are constantly exposed to high levels of elemental Hg, not only in their workplace but also at home and during commute [44]. An approximate 50 million tons of electronic waste are disposed annually which reportedly releases Hg posing great threat to children's health [45]. Disposal of thermometers, batteries, electrical switches, and fluorescent lamps also encourage entry of Hg when exposed to humidity, rain, and wind [46]. Seepage of water from mining sites enters lakes, water streams, and rivers and accumulates Hg for many years [47]. Coal combustion is another great source of Hg release in the atmosphere accounting to a total of 71%, 46% of which comes from industrial plants and 35% from power generation plants [48]. Production of Hg, silver, and other chemicals also pose the risk of mercury contamination which varies across the countries like Asia (16%), North America (30%), and Europe (27%). During the 1970s, the commercial Hg production was at its highest, and it has decreased ever since, along with their release in air (20%), water (30%), soil (30%), and landfills (20%) [5,49]. Toxic Release Inventory and U.S Environmental Protection Agency has reported two third of all mercury released in atmosphere in USA is generated from coal-based power plants [50].

1.2. Different Routes of Mercury Incorporation

The toxic effects of Hg majorly depends on the route of entry; ingestion, inhalation, trans-dermal, and trans-placental [51]. Epidemiological and experimental studies have shown that the overall Hg body burden is influenced by sex and genetic background, while uptake and distribution of Hg is influenced by the nutritional background of the subject [52]. Exposure of Hg can result in its trans-placental movement to the uterus. Hg exposure is dangerous for the foetal brain and can cause mental retardation, vision loss, hearing loss, congenital malformation, language disorder, and delayed development [53].

Inhalation is the most direct route of Hg pollution, and the vapour can reach the brain to cause direct damage. After intake of contaminated seafood, MeHg, which is a powerful neurotoxin, is ingested by the digestive tract and accumulates in the brain for a long period [54]. Pregnant women pose a major risk as MeHg can become accumulated in foetal brain and other tissues. Many dermatologists use skin lightening products to treat hyper pigmentation disorders [1]. These products contain inorganic Hg which inhibits melanin production in epidermal melanocytes. A study has shown that these products do not mention Hg as ingredients. A case study also reported exposure of inorganic Hg can take place through breast milk and skin contact (through bed linens) [55].

A study on Indonesian gold miners has shown occupational exposure of Hg also can be severe as they develop chronic mercury intoxication. Breastfed infants possess the risk of development disorder. Major health problems reported among gold miners include headache, fatigue, mucosal irritation, tremor, memory problems, visual impairment, ataxia, muscle weakness, mood swings, persistent cough, and chest pain [8].

1.3. Patho-Physiological Mode of Action

After crossing the blood–brain barrier, MeHg is oxidized to intracellular mercury in the glial cells and remains in brain cells for a long time [14]. Exposure to ethyl mercury leads to a higher level of mercury accumulation than MeHg exposure [13]. But damage in granular layer is only observed in the case of MeHg exposure. Demethylation of Hg cannot be responsible for neurotoxicity as MeHg in glial cells can be found in neurons as well

in the symptomatic phase of the disorder [56]. Another study has shown strong affinity towards thiol groups in both organic and inorganic mercury, and is partly responsible for its toxic effects [57]. Subcellular neurotoxicity of MeHg is associated with OS, change in glutamate, and calcium homeostasis. Changed calcium homeostasis is due to the inhibition of calmodulin (calcium binding protein) and induction of neuro-inflammatory changes [39]. MeHg are reported to possess harmful effects on microtubules and Rho family proteins which causes problems in neuronal differentiation and migration [58]. The SH group in tubulin attracts methyl mercury causing depolymerisation and disintegration of cerebral microtubules. The microtubule associated protein or MAP2 functions and expression can also be affected by MeHg [59]. Decreased development in dentate gyrus neurons is due to a deficit in hippocampus-dependent spatial learning and memory loss. Astrocytes resist the harmful effects of MeHg due to the expression of glutathione or GSH [60].

2. Fate and Transport of Mercury in Terrestrial Ecosystems

The fate of Hg in the ecosystem is an intricate biogeochemical cycle that involves movement within the lithosphere, atmosphere, and hydrosphere [61]. On the local, regional, and global scale, Hg can be completely mixed vertically until the troposphere, and can be transported over great distances. Past studies in arctic fishes, collected from areas where human interventions are minimum, reported high concentrations of MeHg. It is mobilized from the lithosphere through natural geological activities. Other natural activities anthropogenic activities like mining, coal combustion, and industrial processes also led to mobilization of mercuric deposits. This has caused an immense increase of ~450% in Hg concentration in the ecosystem [62]. According to recent estimations, natural emissions of Hg (76–300 g/yr) are significantly outnumbered by anthropogenic emissions (2000 Mg/yr) [63]. The increase in Hg in the atmosphere causes its deposition throughout aquatic and terrestrial ecosystems. It is estimated that there was a fivefold increase in Hg deposition in peat and lake sediments even in remote regions in the last few decades [64]. The cycling of Hg through the ecosystem depends on its chemical form as illustrated in Figure 1.

The high inertness of gaseous Hg enables it to be dispersed along long ranges as dry depositions [17,61]. The reactive species, Hg^{2+} also is dispersed along with the inert Hg^0 as wet depositions. The dry deposition (Hg^0) of mercury is taken up by plants through stomata gas exchange. Thus, in terrestrial ecosystems the major addition of mercury to the soil happens by litterfall and its decomposition [65]. Apart from this, the Hg^{2+} is deposited directly on the canopy of trees and is washed off to the ground, adding to the soil deposits [5]. There are two possible fates for the deposited Hg^{2+}. It can either be absorbed into sediments, where it can become MeHg, or it can be reduced to Hg^0 in water [66]. The conversion of Hg^{2+} to Hg^0 is affected by different factors like water temperature, pH, total dissolved organic matter, and light intensity. The accumulated Hg^0 can cycle back into the atmosphere through volatilization, hence it is the only method in which the accumulation of Hg in life form can be limited.

Organic mercury is formed when Hg is combined with organic carbon. MeHg is one of the most common species among organic mercury, this compound exists as monomethyl mercury (CH_3Hg^+) or [$(CH3_3)_2Hg$] [67]. Other than chemical conversion in water and sediments, microbial activity and photolysis also contributes to the conversion of Hg^+ to MeHg. This organic compound is responsible for bioaccumulation of Hg in life forms. The MeHg enters the food chain commonly via aquatic life forms which thrives along the surface water. In seawater, MeHg is converted into [$(CH3_3)_2Hg$] [68].

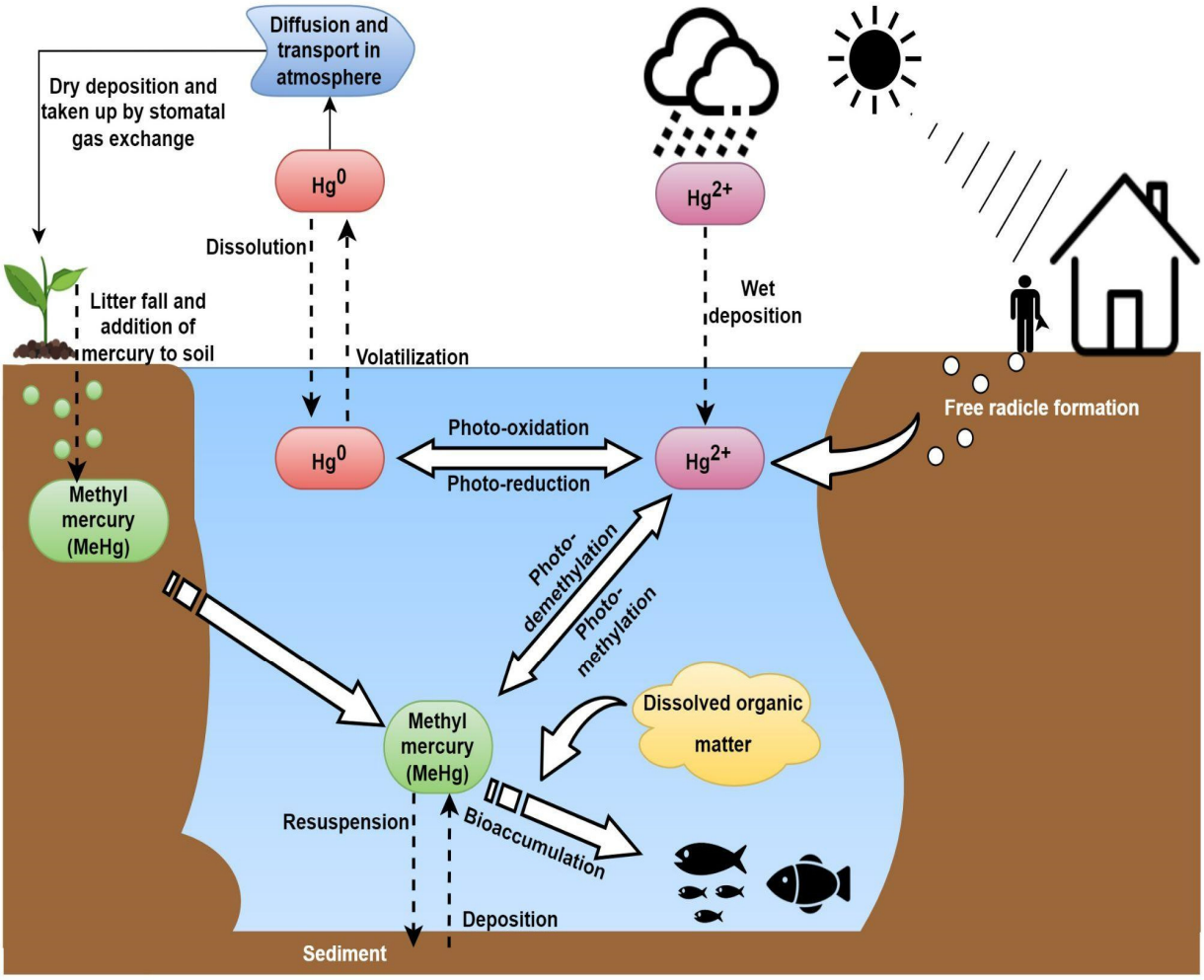

Figure 1. Cycling of mercury through terrestrial and aquatic ecosystem.

2.1. Mercury in Soil and Plant Life

It was discovered that the vegetation greenness, leaf area, soil organic matter, annual precipitation, and latitude, all affect the mercury distribution in the landscape. Compared with other ecosystems, forested watersheds and its drainage network including the river and lacustrine sediments exhibit significantly greater concentrations of Hg [17]. Soil is extensively studied for Hg concentration as it is considered as the terrestrial repository of contaminants. The deposition of mercury among the mountain regions are contributed by the local mineral depositions and anthropogenic inputs via atmospheric depositions.

The plant facilitates the addition of Hg due to the atmospheric dry deposition, which plays a key role in increasing the forest depositions four times higher than wet deposition in open sites. The forest depositions are greater than that of shrublands and deserts, which have limited vegetation cover. Although vegetation cover plays a crucial role in the accumulation of Hg in the soil, other factors like the texture, morphology and genesis, total organic matter, and pH also affect the accumulation and retention of Hg in soils [17].

Different soil types have different effects on the accumulation of Hg. The ferralitic soil retains Hg through adsorption although it lacks other nutrients. The Hg is associated with organic carbon, and later this complex compound is retained in ferralitic soil by Al/Fe oxyhydroxides [69]. Hg retention in tropical soils are controlled by the geochemical contents and texture. The ferralitic soil in the Amazon region has more Hg than that of temperate and boreal soils. Ferrelatic soil acts as the link for Hg transfer between the terrestrial and aquatic ecosystems in the humid tropical regions. The erosion and transfer

of Hg from soils would be accelerated by subsequent heavy rainfall and high discharge events, which are characteristic in humid tropical regions [70].

2.2. Mercury in Lacustrine Sediments

The settling particles scavenge the Hg that has been directly deposited on a lake surface, which are confined as sediments. The Hg that has been deposited on watersheds is transported to lakes primarily bound to organic matters and mineral matter [65]. As in soil, the Hg deposition in lacustrine sediments is also affected by many variables including physical, chemical, and biological. Some of these factors are dissolved oxygen concentration, temperature, pH, complexing agents, and nutrient distribution [66,71]. The redox reactions of Hg^{2+} and Hg^{0} occur among these sediments, which facilitate their distribution into organic as well as inorganic complexes. The methylation of Hg occurs both in sediments as well as in the water column, the methylation of Hg will be high among the sediments which have high organic matter content. The MeHg in sediments either is deposited into the lacustrine segments as further sediments accumulate on top or it leaches into the lake water which will accumulate in the life forms [71]. The concentration of Hg accumulated in various organisms collected from diverse geographical locations in the last five years is presented in Table 2.

Table 2. Accumulation of mercury in various organism collected from diverse geographical locations.

Organism	Mercuric Concentration (ppm)	Geographical Locations	Reference
Lethrinus nebulosus	0.771	Qatar	[72]
Natrix maura	0.194	Europe	[73]
Epinephelus coioides	0.55	Kuwait	[74]
Epinephelus coioides	0.045	China	[16]
Urtica dioica	21.4	France	[75]
Elateridae sp.	3.6798	France	[75]
Rastrelliger brachysoma	0.025	Malaysia	[76]
Stolephorus indicus	0.04–0.18	UAE	[77]
Scylla serrata	4.11	India	[78]
Penaeus monodon	2.25	India	[78]
Gerres oyena	0.0283	Qatar	[79]
Chiloscyllium arabicum	0.1662	Qatar	[79]
Rhizoprionodon oligolinx	0.7942	Qatar	[79]
Chaetobranchus semifasciatus	2.85	India	[80]
Liza macrolepis	1.860	India	[80]
Gazza minuta	1.97	India	[80]
Chilina sp. (snails)	0.564	Argentina	[81]
Samastacus spinifrons (crayfsh)	0.561	Argentina	[82]
Aquila chrysaetos (Scottish golden eagles)	0.0348	Scotland	[83]
Arius arius	0.977	India	[78]
Caranx affinis	3	India	[78]

2.3. Fate of Mercury in Aquatic Environments

Hg in aquatic environments usually exists in elemental form (Hg^{0}), ionic (Hg^{+}, Hg^{2+}), and as MeHg forms, constituting a major threat to aquatic life including plants and animals. The proportion of elemental mercury (Hg^{0}) is relatively less in aquatic environments compared with ionic (Hg^{+}, Hg^{2+}) and MeHg [84]. Hg^{0} is majorly present in surface water and is formed through the microbial decomposition of Hg^{2+} [85]. Reduction can also be brought about by abiotic factors present in water. Photocatalytic reduction can be a major factor in the decomposition of Hg^{2+} [24]. Among the three forms of Hg, the major concern in the aquatic habitat is posed due to the bioaccumulation and biomagnification of MeHg [86]. The transformation of Hg in the aquatic environment shares several similarities with its cycling in terrestrial environment and is broadly divided into five phases:

2.3.1. Oxidation of Hg^0

Oxidation of Hg^0 is a fast process and can be brought about by chemical agents or through photooxidation process. The major chemical agents involved in the oxidation of Hg^0 apart from oxygen includes nitrates, nitrites, iron phosphates, sulphur, and carbon dioxide [87].

2.3.2. Reduction in Hg^{2+}

Reduction in Hg^{2+} in the aquatic environment is attributed to three major factors: Major proportion reduction occurs through light-induced mechanisms (photoreduction) followed by microbe mediated enzymatic processes; A third and minor proportion of Hg^{2+} can be traced to the dark reactions that take place in aquatic ecosystems whose actual mechanism remains unclear [88].

2.3.3. Methylation of Hg^{2+}

The formation of MeHg in aquatic environments is often driven by the nature of the sediments and activity of the several microbial communities in the proximity of the aquatic ecosystems. The microbial contribution in the formation of MeHg is a thrust area of study and so far three groups of bacteria namely sulphate-reducing bacteria (SRB), iron-reducing bacteria (IRB), and methanogens were reported to aid in MeHg formation [89].

2.3.4. Degradation of MeHg

Surface water is the major site for degradation of MeHg majorly due to the photodegradation occurring at the surface due to light attenuation. The photochemical degradation of MeHg is driven by factors like the presence of dissolved organic matter (DOM), photolysis and an oxidant radical (*OH) produced as a result of photo Fenton reactions [90].

2.3.5. Demethylation of MeHg

Demethylation represents the reverse process of methylation. This process involves the removal of the methyl part of MeHg upon exposure to UV radiation. This can be carried out either by microbes, photodecomposition, or abiotic factors. Photodecomposition was observed to be accelerated in marine environments [91].

The transfer and cycling of Hg from atmosphere, terrestrial, and aquatic ecosystems is often affected by other physical factors and geographic factors which in turn determines the accumulation rate in the organism prevailing in various habitats [92].

3. Mercury Bioaccumulation and Microbial Community Changes

3.1. Source and Bioconversion of Mercury to Methylmercury

United Nations on Environmental Pollution (UNEP) classified Hg emission into two broader categories: direct emission from anthropogenic and natural activities and re-emission from the depositions. Natural direct emission includes emission from volcanoes and release from weathering of volcanic rock. After the release, Hg is transported and recycled between air, soil, and water, until it is removed from cycle as deposition as sediment in coastal and deep ocean, lakes, and soils of subsurface [93]. Atmospheric deposition contains the three principal forms of Hg, although the more abundant form is inorganic divalent mercury (Hg^{2+}) followed by MeHg and elemental mercury. Upon entering surface water, Hg enters a cycle that becomes attached to particles that settle as sediments. From sediment, it can spread through diffusion into the water column or be resuspended and sediment with other particles.

Methylation is an important step that transforms elemental mercury into a more mobile form. Anaerobic bacteria possessing the Wood–Ljungdahl pathway for carbon fixation is essential in producing MeHg. These bacteria harbour the hgcAB gene, constitutively expressed in certain anaerobes. Bacteria that process sulphate (SO_4^{2-}) is directly linked with methanogens converting inorganic Hg to MeHg. Increased natural organic content, humic acid, and algal growth are associated with increased methylation in natural reserves [94].

The conversion of inorganic mercury to MeHg is aided by *hgcA*, which transfers the methyl group from methyl-THF to inorganic mercury ion. Though the Hg^{2+} form is not a nucleophile that attacks the methyl group, theories suggest electrons in mercury undergoes a spatial conformational change that react with carbocation. HgcA, in addition to methyl transfer, also facilitates the import of elemental mercury into the cell through its transmembrane port. HgcB, an iron-sulphur protein, helps maintain the redox balance by reducing Hg during methylation. Although a common mechanism of MeHg efflux is not reported, few systems showed the presence of efflux motors which helps in MeHg depuration [95]. It enters the food chain through the phytoplankton, which absorbs the effluxed MeHg or directly to higher organisms that consume bacteria that accumulate MeHg. Ultraviolet rays from sunlight break down methyl mercury to Hg^{2+} or Hg^{0}. Elemental Hg can re-enter the atmosphere as a gas, and highly reactive ionic form can react with biomolecules and accumulate in living organisms.

3.2. Bioaccumulation of Mercury in Higher-Order Organisms

Animals accumulate MeHg at a faster rate than they eliminate it, they consume Hg accumulated in smaller organisms at each successive level of the food chain. Small concentrations of MeHg from environment can thus accumulate gradually to significant harmful levels in fish, fish-eating wildlife, and people. Even at minor deposition rates in remote locations from point sources, its biomagnification produces toxic effects on terminal consumers of food chains. The Hg undergoes vicious cycles where it transforms from inorganic forms and methylated form in environment through natural disperse and organisms (Figure 2).

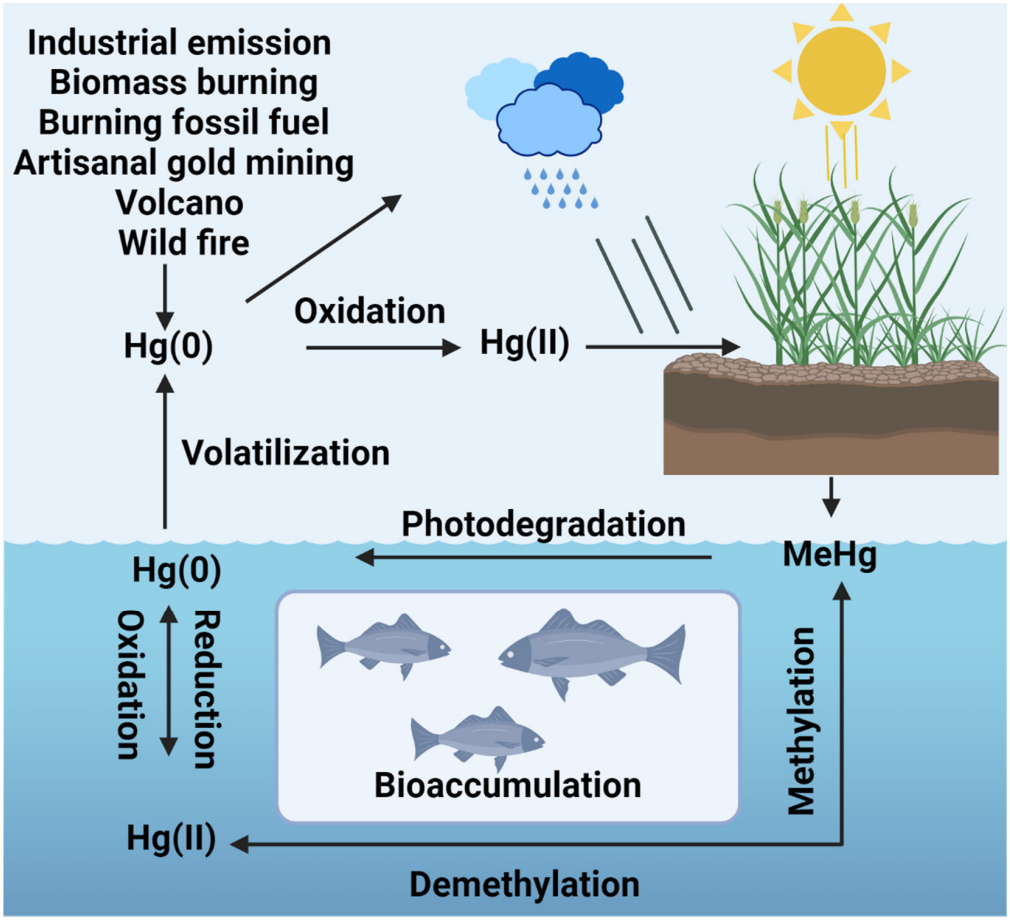

Figure 2. Vicious cycle of mercury in environment.

Dissolved organic carbon (DOC) and pH strongly affects the Hg retainment in an ecosystem. Several studies showed that for the same species of fish taken from the same region, increasing the acidity of the water (decreasing pH) with or without increasing the DOC content generally results in higher Hg levels in fish, an indicator of more significant net methylation. Similarly, higher DOC levels increased Hg accumulation until saturation level. Combined, high acidity and DOC levels enhance the mobility of Hg in the environment, thus making it more likely to enter the food chain [96].

Many marine life forms tend to accumulate Hg in less soluble inorganic form. Though Hg is toxic for bacterial species, prolonged exposure increases bioaccumulation. A study on bacterial communities in the gold mining field, which uses Hg for refining, showed all isolated accumulated Hg > 50% without losing viability, with the major being Bacillus species [97]. In fishes, *Dicentrarchus labrax* (European bass fishes), on Hg exposure, showed bioconcentration of Hg on gills and bioaccumulation of the liver. The accumulation tends to increase the OS in respective organs. In addition, the microplastics increased the rate and accumulation of Hg in the fish's liver [98]. In walruses, Hg concentration in their skeletal muscles decreases with aging. A population study on pacific walruses showed that females tend to accumulate more Hg than males, with a significant reduction in Hg levels with aging [99]. In seals, the Hg accumulation in muscle tends to transfer to the blood during the gestation period and pass on to the offspring through the placenta. The subsequent lactation period, post-delivery, showed a proportional decrease in Hg levels in offspring [100].

3.3. Mercury Toxicity and Its Effect on Microbial Community

In bacteria, the accumulation occurs in proteins where Hg binds to metallothionein through cysteine groups. *Anabaena* spp., *Pseudomonas* spp., and *Synechococcus* spp accumulate more mercury through this mechanism. In addition to intracellular metallothioneins, extracellular sequestration of Hg is captured by siderophores, oxaloacetate, phosphates, and sulphides [101].

Several reports on the microbial community in soil revealed that the abundance of microbial species remained the same. However, the abundance ratio was significantly varied. Proteobacteria, Actinomycota, Blastomonas, and Acidobacters were dominant in Hg-exposed soil, irrespective of pH. The predominant bacteria, Actinobacter, was found to dominate the post-Hg treatment though it is negatively correlated to Hg [102]. Exposure of 50 mg Hg/kg soil of mercury on agricultural soil significantly decreases pH from 7 to 6 with a decrease in total organic content and macronutrients. Due to Hg toxicity, ~36% of microbial species were lost compared with the control. Natural microbiota *Sphingobacterium* sp., *Pedobacter saltans*, and *Brevundimonas subvibrioides* were enriched by 1.42, 1.57, and 2.73 times, respectively [103].

Unlike the bacterial community, the fungal community has its detoxification system and was affected only at higher Hg concentrations. However, the fungi were susceptible to pH and hence greatly affected by pH the change caused by Hg. Basidiomycota increased to 1.61 and five times in alkaline and neutral pH, respectively, and decreased by 18% in acidic pH. On the contrary, Ascomycota showed an inverse relationship with pH, with a slight increase in population (5%) under acidic conditions [102].

3.4. Molecular Mechanism of Mercury Selection

3.4.1. Genes Involved in Mercury Resistance

The ionic form of Hg (Hg^{2+}) can readily react with the thiol group of proteins and hence cause loss or abnormality in functional proteins. Hence, the bacterial system develops resistance through mercury reductase (*merA*), which converts highly reactive Hg^{2+} to unreactive elemental mercury (Hg^0) as gas [104]. Hg resistance operon consists of genes coding for the following proteins: transmembrane proteins which transport mercury through the periplasmic membrane (*merC*, *merE*, *merF*, *merT*, and *merG*); organo mercury lyase (*merB*); and mercury reductase (*merA*). The presence of this operon increased the

resistance level compared with bacteria not harbouring the genes, and is regulated by *merR* and *merD*. A Statistical study on Hg resistance genes in bacteria showed that the abundance of merA is more than the co-existence of *merA* and *merB*. *MerT* is associated with transporting ionic mercury protein adducts, whereas merE helps transport methylmercury protein adducts. Merck is associated with transporting organic mercury, which requires *merB* and *merA* for detoxification [105]. Genes *hdcAB* is also linked with the evolution of merA, suggesting the co-evolution of Hg uptake and resistance genes [95]. The species of *Pseudomonadales* and *Xanthomonadales* were found to enrich in the presence of mercury with the help of the merA gene in the native plasmid [104].

3.4.2. Horizontal Gene Transfer on Microbial Selection

Mobile genetic elements (MGEs) are responsible for transferring resistance genes across species and affect the microbial community under extreme conditions. MGEs of *mer* operon were transferred to other species through conjugation to share Hg resistance. The conjugation was so efficient that even distant phyla Burkholderiales received Hg-resistant plasmid [104]. Hg is also linked to increased co-transfer of antibiotic genes. A common clinical carbapenemase isolates resistant *Klebsiella pneumonia* shown to co-transfer *mer* genes along with *bla* (carbapenemase-resistant gene) in the presence of Hg [106]. In addition to pesticide resistance, Hg resistance is linked with many commonly used laboratory-grade antibiotics such as chloramphenicol, ampicillin, streptomycin, sulphonamide, and trimethoprim [101]. Hg resistance from prolonged bacteria exposure in Hg-rich sites can acquire antibiotic resistance genes (ARG) through conjugation. Several studies showed that microbes from Hg-contaminated soils of mines, agricultural farms, aquaculture, and dairy farms and from the gut of animals carried microbes containing both Hg and ARG resistance genes [107].

4. Toxicokinetic and Ecotoxicology of Mercury

Hg is one of the most hazardous anthropogenic pollutants due to its extreme toxicity, and it poses a significant threat to ecosystems, especially via its accumulation and biomagnification in food webs [108]. The organic Hg compounds, especially MeHg, are the most dangerous forms of Hg due to their high solubility in lipids, which increases the likelihood of biological absorption and bioconcentration [109]. MeHg is generated when inorganic Hg undergoes a series of physiologically mediated chemical reactions in anaerobic environments [108]. The majority of Hg found in polluted fish and human bodies is found in its organic form, which is a neurotoxic that has negative effects on fish and mammal reproductive and neurological development [110].

4.1. Biomonitoring of Mercury

Biomonitoring refers to a method of measuring environmental pollution by observing the effects on live creatures. The primary value of biomonitoring is determining the relative concentrations of contaminants within a sample of organisms. Various studies have shown that the Hg level already exists globally and affects both human health and the environment. Negative health consequences from Hg exposure are evident even at low levels [1]. But the question of whether toxicity from different types of Hg builds up over time remains open and will be the focus of future research. The European Commission and the EPA/FDA have both provided guidance on acceptable Hg levels in food [1]. Unfortunately, human biomonitoring is necessary for estimating the internal dosage of Hg and associated dangers to human health. Biomonitoring in people is necessary for determining whether or not Hg were actually ingested by humans. Prospective monitoring of air, water, and other environmental samples cannot offer these data. Because it may offer quantifiable information on the authentic exposure of Hg population and aid in the assessment of preventative activities, human biomonitoring of Hg is a helpful tool to assist environment and health policymaking.

Heavy metals (HMs) may enter fish in two ways: via the water and their food, and through their semipermeable membranes. Metals may interfere with important metabolic processes in fish tissues, making fish a useful bioindicator. Using fish as a bioindicator is ideal for assessing HM contamination for two primary reasons: bioaccumulation and biomagnification. The first, is that metals may bioaccumulate in fish tissues and, in general, have very long-lasting properties. Second, biomagnification causes metals to bioaccumulate in tissues after being devoured by species at a higher trophic level, which in turn is consumed by species at a lower trophic level, further increasing the concentration of the metal in the tissues of the higher trophic level [111].

Biomarkers for Hg exposure (within human monitoring) need an understanding of exposure situations to be interpreted correctly. Hg levels in blood and urine may be used as proxies for overall Hg exposure. In both experimental and epidemiological research, sex and genetic heritage have been found to alter the total body burden of Hg. Nutritional background may influence the absorption and distribution of mercury species, and hence the human biomonitoring readings [112]. It is vital to distinguish between reference values and health-based limit values when interpreting human biomonitoring data. The reference values are arbitrary benchmarks with no clinical relevance [113].

Human hair samples from the Amazon basin near Tucuru Dam were examined for MeHg and overall mercury concentration [114]. Total Hg concentrations in hair, on average 75 g/g (about 90% of MeHg), were found to be dangerously high compared with WHO guidelines. The median total mercury content was 12.0 g/g, with 57% and 30% of the subjects having very high values (10 g/g and 20 g/g, respectively). These levels are far higher than any others seen in people living near Amazonian dams. Gold mining has not been a major contributor to Hg pollution in the studied region. However, it was recently brought to light that one of the most eaten fishes (*Cichla* sp.) can be contaminated with MeHg [114].

MeHg to total mercury ratio and particular mercury species concentrations have been reported [115]. Despite the public health and toxicologic involvement in MeHg and Hg, these Hg species have been technologically difficult to measure in large population studies, resulting in the authors conducting several multiple regression analyses to examine factors associated with MeHg concentrations. Age was a significant factor, with the peak incidence occurring between the ages of 60 and 69. MeHg levels were shown to be higher among Asians, men, the elderly, and persons with higher levels of education. MeHg share of total Hg varied by race and age, and the trend was nonlinear. MeHg reference values for the U.S. population and the ratio of MeHg to total Hg may aid in a more accurate evaluation of the public health risk associated with consuming seafood containing MeHg [115]. Absorption of ingested MeHg is thought to be high and not expected to vary much.

4.2. Mercury Toxicity to Environment

The toxic substances and disease registry under the US Government agency has ranked Hg as the third most toxic element or substance on the planet, behind arsenic and lead, which are being dumped into our waterways and soil, spilled into the atmosphere, and consumed by humans in food and water. Nearly tripling the quantity of mercury in the air is a direct result of human activities, and the atmospheric load is rising at a rate of 1.5% year. Hg in the soil or recirculated water with mercury may be ingested by animals and plants. The harmful effects of Hg on humans may bioaccumulate if it enters the food chain. Hg entry into the food chain is thought to occur in a variety of ways depending on the ecology [116].

4.2.1. Mercury Toxicity to Plants and Algae

Plants root systems are their primary access point for taking up soil nutrients, including poisonous metals. The root system has a lot of surface area, so it can take in and store nutrients the plant needs to thrive. But it may also take in and store toxins it does not need, such as HMs. To comprehend Hg's behaviour in the soil–plant system, one must first

know the Hg transfer factor between the two. Various types of plants contain Hg, which is then delivered to various organs by special proteins called transporters [117]. Even at low applied quantities, Hg is hazardous to plants, causing growth retardation, photosynthesis suppression, reactive oxygen species production, lipid membrane oxidation, DNA damage, and protein degradation. Plants have evolved several defence mechanisms to cope with the oxidative damage caused by Hg exposure. Antioxidants including glutathione, phytochelatin, salicylic acids, ascorbic acid, selenium, proline, and tocopherols are all part of these defensive systems [117].

The impact of Hg on the development and production of *Solanum lycopersicum* crops was investigated by [118]. At decreased Hg concentrations, plant growth, including germination rate, height, root length, blooming time, pollen viability, and chlorophyll content, was seen to be improved. When Hg levels were much greater, however, development was stunted, and inhibitory effects were reported. A researcher evaluated *Pisum sativum* L. for Hg-induced cytotoxicity and genotoxicity [119]. The data demonstrated cytostatic effects, including a delay in S-phase at low dosages and an arrest in G1 at high doses, as well as a reduction in mitotic index and cell proliferation index. Additionally, changes in DNA strand breakage, DNA fragmentation, the clastogenic parameter, and micronuclei were seen in roots when Hg concentrations were elevated. These findings showed that Hg may create genetic differences in plants by interfering with their cell cycle.

Halimione portulacoides, a common and widespread species in temperate saltmarshes, is well-known as a bioindicator and biomonitor of Hg pollution. *H. portulacoides* stores most of its mercury in its roots, and the element is then actively translocated to its shoots and leaves. Differences in Hg concentration can be explained by evaporation and transpiration of Hg from the plants leaves. Both thoriated and monomethyl Hg are poisonous towards this salt marsh plant. Even at low concentrations, mercury may have an effect on photochemistry with sustained exposure [120].

Numerous studies have shown that biomolecules including proteins, DNA, and lipids are susceptible to OS and damage when exposed to Hg and other HMs. Total protein concentrations in the roots and leaves of *Triticum aestivum* were found to increase by 16% and 10%, respectively, when treated with 2.5 M of $HgCl_2$ [121]. In response to Hg stress, the body may expand its total protein pool. Proteins involved in the cellular redox system are not an exception to this rule. In another study, after 40 and 60 days of Hg stress, malondialdehyde levels in plants considerably decreased from control, but malondialdehyde contents rose with increasing exposure length to Hg pollution. Exogenous Hg administration may also lead to Hg accumulation and toxicity, much as the root uptake system [122].

Photosynthesis, a crucial autotrophic metabolic activity, has been shown to be vulnerable to toxicity from metals. The oxygen-evolving enzyme, donor site, and the subunit of ATP synthase in the chloroplast are all known to be affected by Hg presence [123]. It is well-known that the production of reactive oxygen species causes the degradation of chlorophyll and membranes and limits its biosynthesis. This, in turn, destroys the structure and function of the photosynthetic apparatus and leads to an unequal distribution of energy within the cell [117]. Some researchers performed a pot experiment showing that Hg stress induces a decrease in net photosynthetic rate, stomatal conductance, and transpiration rate in the leaves of grass in response to an increase in intercellular CO_2 concentration [124]. Chlorophyll breakdown in plants responded differently depending on Hg content and exposure duration. Chlorophyll content of *Chlamydomonas reinhardtii* was reduced by 58.6% compared with the control, indicating the presence of toxic symptoms, although the photosynthetic rate was suppressed by a much smaller margin [125]. Several studies were conducted to understand the nature and toxicity of inorganic Hg and MeHg in algal systems as they represent a significant part of the food chain. Microalgae are considered to be the point of entry for Hg and MeHg to travel through the higher trophic levels in an aquatic ecosystem [126]. Exposure of microalga *Chlamydomonas reinhardtii* to inorganic Hg and MeHg was reported to cause dysregulation of cellular transport and

energy metabolism. Further Hg exposure was observed to have a negative impact on the rate of photosynthesis and oxidation-reduction directions in *C. reinhardtii* [127]. Exposure of *Chlorella vulgaris* to Hg was reported to reduce the amount of photosynthetic pigments in a concentration-dependent manner. Further, a decrease in protein content and increase in the amount of reactive oxygen species (ROS) was reported in the study [128]. Chronic exposure to Hg in *Chlorococcum dorsiventrale* Ch-UB5 collected from coastal areas of Tunisia was observed to cause reduction in biomass, altered redox activities, inhibition of photochemical activity, and appearance of oxidative stress [129]. The toxicity of Hg to microalgae is majorly due to its interaction with SH groups of the enzymes and imparting OS. Hg inside the algal cells were observed to bind to cytosolic ligands and block the functional groups of important enzymes there by altering their confirmation and functional properties [130].

4.2.2. Mercury Toxicity to Fish

Increased level of tissue mercury concentrations have been linked to point source pollution and have been shown to have obvious impacts on fish survival and development [131]. Fish with muscle Hg contents much below those resulting in overt toxicity are often found in areas that obtain Hg from atmospheric deposition rather than directly from contaminated point sources [110]. Nonetheless, increasing evidence from laboratory experiments and field observations showed that the Hg levels often seen in wild fish are adequate to elicit sublethal toxic effects such as metabolic alterations, tissue and cell damage, decreased reproduction, developmental consequences, and behavioural effects [132]. Importantly, elevated Hg concentrations have been documented in certain Arctic fish populations, especially in lakes, due to the long-range transmission of Hg from human emission sources at lower latitudes [133,134]. Variations in fish diet and trophic position cause deviations in Hg levels and the associated risk of deleterious effects. Table 3 represents the level of Hg content present in fish.

Table 3. Levels of mercury present in different fish species.

Trophic Region	Fish	Concentration of Mercury (ppm)	Portion	Reference
Omnivores	*Siganus fuscessens*	0.018	Muscle	[135]
Herbivore	*Siganus rivulatus*	0.02	Muscle	[136]
Carnivores	*Epinephelus coioides*	4.65	Liver	[137]
Herbivore	*Siganus canaliculatus*	0.032	Muscle	[138]
Carnivores	*Lethrinus nebulosus*	0.773	Liver	[72]
Carnivores	*Lethrinus nebulosus*	0.522	Muscle	[138]
Carnivores	*Gerres oyena*	0.028	Muscle	[79]
Planktivores	*Sardinella albella*	0.028	Muscle	[138]

Recently, a study reported that fish populations were considered to be in the "no danger" or "low risk" categories for muscle Hg concentrations, whereas a smaller number of populations were considered into the "high risk" category [131]. Low quantities of Hg and MeHg were found in both marine and freshwater invertebrates, indicating minimal toxicity risk to invertebrates or nutritional exposure to invertebrate-feeding fish in an initial screening. Predatory long-lived fish in the Arctic have Hg concentrations in the highest danger categories. Similar studies of screening-level evaluations need to be fine-tuned by future research that explicitly connects muscle Hg contents and impacts in Arctic species of fish at high or severe risk.

Although there is a dearth of data that would allow us to assess the hazards of fish associated with human use. Recently a study reported that Hg in organic form was accumulated in fish throughout time and was amplified up the food chain based on the fish's size, nutrition, and trophic position [139]. The muscle and liver contained about average mercury of (w/w) 268.2 ng/g and 62.3 ng/g, respectively. The levels of thymidine kinase in fish muscles and livers were positively correlated with fish size [139]. These need

to be explored well and can provide the foundation for a more accurate risk assessment of eating fish contaminated with Hg.

Fish muscle is the most examined tissue for Hg accumulations because it appears to operate as a Hg reservoir to protect other organs, and it is the major fish tissue ingested by humans. Hg also tends to concentrate in detoxifying organs like the liver and kidney [140]. For this, zebrafish (*Danio rerio*) were investigated with Hg-contaminated diet for 21 days and then monitored their Hg levels for another 28 days to determine bioaccumulation and depuration. Hg levels in fish organs were examined at different time intervals. During the absorption phase, the liver showed the greatest accumulation rates because of its central role in detoxification. However, the time it took for Hg to be eliminated from the *Danio rerio* liver was shown [140].

Exposure to Hg^{2+} at ambient concentrations may cause changes in thyroid hormone levels and the transcription of associated HPT-axis genes, disrupting the thyroid hormone metabolism process. From 2 h post-fertilization (hpf) until 168 hpf, zebrafish embryos were subjected to 1, 4, and 16 g/L Hg^{2+}. Genes associated in the hypothalamic-pituitary-thyroid (HPT) axis and thyroid hormone (TH) levels were reported by measuring their mRNA expression levels. Levels of triiodothyronine and thyroxine were reported to be higher in the entire body after exposure to 16 g/L Hg^{2+} [141]. Interestingly, Zebrafish (*Danio rerio*) intestinal tissue was sequenced for transcriptomics after being exposed to $HgCl_2$. This research created the way for further investigations into the molecular responses and intestinal defence systems of zebrafish in response to numerous harmful stimuli.

Elucidation of how long-term exposure to Hg combined with other HMs is more damaging to zebrafish oocytes than exposure to either metal alone creates interesting topic among researchers. Effects of a combination of cadmium and Hg on the ovaries of adult zebrafish were reported [142]. Female adult zebrafish were subjected to 21 days of exposure to 1 mg/L of cadmium chloride and 30 g/L of mercury chloride and a binary combination of both metals. Even though this research contributed to understanding molecular level toxicological reactions of fish to HMs and offers a new approach to evaluating environmental risk, knowledge of the links between mercury exposure and changes in gene function, biological phenotype, and toxicological reactions, among other things, is essential. Such studies need to be performed in the future.

4.3. Mercury Toxicity to Birds and Animals

Some seabirds had rising MeHg concentrations documented in many investigations [57]. Common acute neurological consequences of mercury in birds include impairments in motor skills, coordination, and motivation. Brain MeHg was also shown to have a negative connection with N-methz-d-aspartate receptor density and a positive correlation with muscarinic cholinergic receptor density in fish-eating birds [143]. Brain lesions, demyelination of neurons, changes in haematological and hormone levels, vacuolar alterations in hepatocytes, and increased liver inflammation have all been linked to sublethal exposure to Hg in birds [144].

The effects of Hg poisoning on reproduction, embryonic survival, and early development are well-documented [145]. Across the board, there is mounting evidence that exposing birds to sublethal concentrations of Hg (5 g/g MeHg) decreases reproductive output, impairs immunological function, and causes avoidance of high-energy activities [146]. According to a review of 23 different bird species, the estimated risk assessment thresholds for MeHg are between 0.2 and >1.4 mg/kg in feed, 0.05 mg/kg/d to 0.5 mg/kg/d on a dosage basis, 2.1 to >6.7 mg/kg wt in parental blood and 0.6 to 2.7 mg/kg in eggs [147]. More research is needed to clarify a consistent association between gene expression patterns and harmful consequences to comprehend the full scope of mercury's impact on birds.

In rats, Hg has a negative impact on male reproductive activities as spermatogenesis, sperm motility, and sperm head abnormalities. Exposure to Hg may lead to buildup in the ovaries of mice, which may change reproductive behaviour and contribute to infertility or ovarian failure, according to the available evidence [148]. Brain mercury levels of

5–10 mg/kg wt or higher have been linked in several studies on a wide range of mammalian species to severe toxicity and mortality. At the same time, non-marine mammal thresholds for MeHg poisoning and mortality are estimated to be between 25 and 30 mg/kg ww Hg in the liver. Brain Hg values 5 mg/kg wt have been linked to a wide range of neurochemical alterations in free-living otters and mink [149].

Symptoms of mercury poisoning in animals include a loss of coordination, sluggishness, tremors, convulsions, impaired senses, and eventually death [145]. There is no doubt that mink and otters, as adults, will perish if they are exposed to MeHg in their diets over time. Cats, dogs, mice, and primates were used in experiments to acquire evidence of motor dysfunction [150]. MeHg also has an impact on the visual, cognitive, and emotional characteristics of animals. Wild mink and Greenlandic polar bears have been shown to have a correlation between the number of muscarinic cholinergic receptors in their brains and the amount of mercury present. Neural tube anomalies, eye abnormalities, cleft palate, neurobehavioral alterations, and traditional poor birth outcomes such changes in body weight and death may all arise from MeHg exposure in utero [57].

4.4. Mercury Toxicity to Human Health

There is now no longer any doubt that Hg is neurotoxic and teratogenic, but there is less agreement on the potential effects of long-term exposure to relevant levels of Hg due to inconsistent and even contradictory observation in human studies [57]. Hg's toxicity extends to all its chemical forms. The toxicity level and the nature of the harmful effects being generated by these variants are distinct. Absorption and dispersion in the body are affected not only by the substance itself, but also by the chemical form it comes in. Hg may enter the body in a variety of ways, including inhalation, ingestion, and skin contact [151]. Whether Hg compounds are hazardous after being ingested, inhaled, absorbed via the skin, or transferred during pregnancy is dependent on the route of exposure [152].

Epidemiological and experimental investigations have demonstrated that sex and genetic background impact the total body burden of Hg. Nutritional background may also alter the intake and distribution of Hg species. Hg may enter the body by inhalation, ingestion, and skin contact and prenatal exposure is a concern as well [153]. The brain of foetus is more vulnerable through transplacental exposure. Mental retardation, congenital deformities, delayed development, visual and hearing loss, and language impairments are only a few of the neurodevelopmental impacts of mercury exposure during pregnancy.

High amounts of elemental Hg (32–160 ng/day) are readily absorbed by breathing. Lipids readily dissolve mercury at its elemental level. Once in the bloodstream, it quickly enters red blood cells, where it is concentrated at a level of 95%. In the red blood cells, the hydrogen peroxide enzyme route oxidises it even more to Hg [154]. Dental amalgams are the primary source of elemental Hg in the mouth. Elemental Hg travels via the circulatory system and into the brain. The brain is a good place for elemental Hg to build up and undergo oxidation into mercuric forms. In the metabolic process, elemental Hg undergoes oxidation, resulting in the release of mercuric ions. There is, however, a significant delay involved with this method. Hg gas is breathed out of the lungs and excreted in the faeces. In humans, the half-life of MeHg is estimated to be about 60 days (EPA, 1997). Elemental Hg toxicity is represented in Table 4.

Table 4. Mercury toxicity on human health [155].

Human System	Health Effects
Intestinal system	Effects on the gastrointestinal system include nausea, cramping, diarrhoea, and corrosiveness, disorders of the digestive tract.
Urogenital system	Short-term proteinuria with renal dysfunction, renal failure

Table 4. *Cont.*

Human System	Health Effects
Central nervous system	Erethism, amnesia, sleeplessness, impaired nerve sensation, and motor skills are all symptoms of a breakdown in the nervous system. Acrodynia, convulsions, vision and hearing loss, language difficulties, memory loss, apathy, paraesthesia, and limb and facial numbness are all symptoms of this condition. Defective brain function and stunted growth in children and unborn babies. Discomfort in the nerves, a lightening of the cerebellum and brain, erratic arm motions, difficulty swallowing, and so on.
	Skin and eye irritation, Increased blood pressure: a problem for the cardiovascular system, pain in the chest, shortness of breath, and diminished lung capacity
Genotoxicity	Abnormalities and diseases of the chromosomes, lymphocytes with chromosomal abnormalities.

High levels of Hg were linked to sterility or tubal ovulation impairment. Hg was reported to have negative impacts on semen quality measures and to cause DNA damage in sperm in males [156]. Longer Thrombotic Thrombocytopenic Purpura (TTP), spontaneous miscarriages, deformities, and higher incidence of menstruation problems were all linked to higher Hg concentrations in females. As Hg has been linked to endocrine disruption and subsequent hormonal abnormalities, which in turn have been linked to decreased ovarian and testicular function and impaired human fertility, it is conceivable that this is the cause of Hg negative impact on human reproductive health [156]. Molecular processes by which Hg affects human fertility are not understood. It is critical to learn more about the underlying molecular consequences of Hg on sperm and eggs.

Common pharmaceutical ingredients often include thimerosal, an organic Hg molecule. Haemoglobin in erythrocytes is a macromolecule that can be affected by Hg poisoning due to structural and functional alterations. The haemoglobin structure was compromised by thimerosal, resulting in the loss of its primary function as an O_2 transporter. There are less free thiol groups in haemoglobin when ethyl mercury was present [157]. There have been a lot of animal research performed on Hg, but it is not apparent whether it influences human health.

5. Advanced Techniques Involved in Mercury Remediation

Several techniques have been employed for mercury removal from the environment. As Hg cannot be broken down in the ecosystem, its remediation is mainly based on either its immobilization or removal techniques. Researchers have developed diverse techniques to reduce Hg pollution in the environment. Usually, Hg contaminants are transported to remote locations and eliminated by various physio-chemical methods, like soil purification, ion-exchange precipitation, adsorption, membrane filtration, etc. [158]. The main goal of these methods is either Hg removal from polluted sites or converting harmful mercury residues to less dangerous ones. However, these technologies can be expensive and sometimes inefficient in achieving in situ clean-ups, along with the generation of hazardous by-products [159]. Thus, developing affordable and eco-friendly methods over conventional remediation techniques are gaining popularity. Recently, several studies were conducted to create innovative mercury clean-up materials and technologies [160]. Researchers explored novel and efficient materials in these studies, particularly with more surface area, substantial porosity, and active adsorption regions. The advanced innovative techniques and their mechanisms rely on lately developed physical or chemical materials or metabolic processes of organisms, specifically plants, algae, and bacteria. Among these Hg removal techniques, advanced oxidation processes (AOPs), immobilization, reduction, biological remediation, etc., are the major ones explored. These sophisticated techniques have proven more economical and eco-friendlier than traditional treatment technologies, like thermal desorption or activated carbon adsorption. Figure 3 presents the advanced method available for Hg remediation. Some of these green and innovative approaches to Hg removal are described in the following subsection.

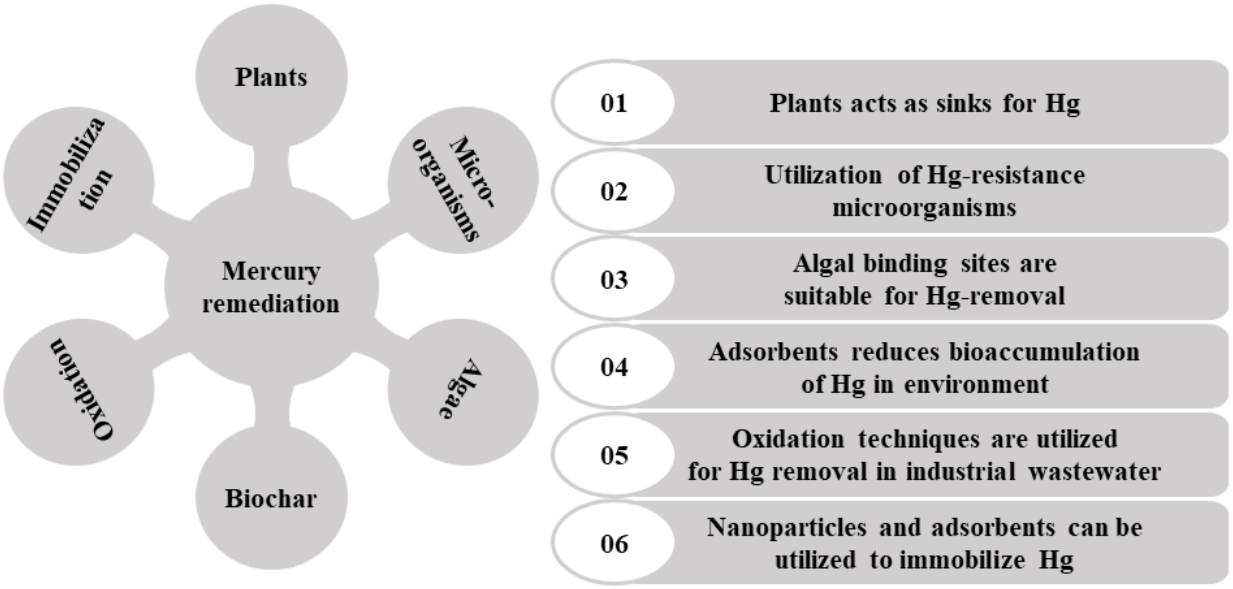

Figure 3. Advanced methods available for mercury remediation.

5.1. Phytoremediation of Mercury

Phytoremediation for environmental contaminants rely on plants that absorb HMs like Hg. Plants serve as sinks for Hg species, so selecting species with robust cellular mercury-buffering capacities, extended root systems, and higher biomass output is essential. Hg is absorbed in Hg^{2+} by roots and transported to shoots, where processes like OS-relieving enzymes, vacuolar sequestration, and chelation with thiol compounds become activated. The Hg-contaminated soil phytoremediation is comparatively more affordable and environmentally favourable than other approaches [161]. The advantage of phytoremediation lies in the fact that it permanently removes Hg from the surroundings by storing it inside plant's biomass. In general, the plant species should be able to remove substantial quantities of HMs, without exerting any stress on their biomass [162]. Some plants, referred to as hyperaccumulators, are crucial components of the remediation process, which are comparatively better performers in reducing or removing pollutants [163]. Studies on *Cyrtomium macrophyllum*, involving enriching the Hg levels to 36 mg kg^{-1}, report nil damage to leaf tissue [164]. Another study reported the ability of woody plant, Nerium oleander in enriching and effectively removing pollutants like Hg from the environment [165].

Similarly, *Festuca rubra* L., *Equisetum telmateya*, and *Leontodon taraxacoides* show promising results for removal of mercury from the mining place. In this study, Hg is found to be primarily stored in the plants' leaves, with the highest concentrations reaching 75–85 mg kg^{-1} [163]. *Oxalis corniculata* L. also show positive results; however, the majority of the Hg is localized to stems and leaves of the plant, indicating its potential as a candidate for mercury extraction. Aquatic macrophyte *Limnocharis flava* is studied to accumulate Hg from gold mining effluents [166].

In plants, the exceptional competence to store or remove Hg occurs naturally, possibly due to plant innate defence, or it might involve microorganisms around the roots. Reportedly, microorganisms play a considerable role in phytoremediation [167]. The rhizosphere bacteria are tangled in phytoremediation and have raised interest for further studies. It appears that rhizosphere bacteria can alter the soil pH, release chelators, and trigger oxidation/reduction events, which increase the metals bioavailability. The Plant Growth-Promoting Rhizobacteria (PGPRs), in particular, boost the phytoremediation efficiency of Hg by 45% [168].

Thus, it is summarized that phytoremediation of Hg-contaminated locations is promising due to the minimal adverse ecological impact and ease of usage. However, it should be kept in consideration that phytoremediation is somewhat time-consuming, and effective-

ness is constrained by various factors, like root depth and availability of the HMs. Lastly, after the harvest of plants, dealing with Hg-containing biomass remains a challenge and requires further caution; otherwise, it can negatively affect the health of lives.

5.2. Microbial Treatment of Mercury

The exploitation of microbes for removing HMs, including Hg, from contaminated wastes is promising because of its effectiveness and environmentally benign nature. Under aerobic circumstances, a variety of bacteria, such as *Pseudomonas putida*, *P. aeruginosa*, *P. stutzeri*, *Sphingomonas* sp., *Cupriavidus metallidurans*, and *Aeromonas hydrophila*, etc., reduce Hg levels efficiently [57,169,170]. In this method, Hg-resistant bacteria reportedly have the 'mer operon', which contains several Hg-tolerance genes (*merA* genes). The sulphate-reducing bacteria are also tested for cleaning Hg pollution in wetlands [171]. *Bacillus* is already investigated in Hg and lead removal [172]. Overall, *Pseudomonas*, *Bacillus*, and *Vibrio fluvialis* are proven efficient in improving mercury-contaminated soil [173,174]. In a study, *Sphingopyxis* sp. SE2 showed 44% Hg clearance rate in 6 h [175]. *Cupriavidus metallidurans* MSR33 removed mercury from contaminated water under both aerobic and anaerobic conditions, despite the fact that Hg (mercury (II)) generally damages the metabolic activity of the organism [176]. Naguib et al. tested *Stenotrophomonas maltophilia* ADW10 and showed promising results [177]. Chen et al. (2018) rather applied a two-step method, firstly performing a chemical extraction process to increase the bioavailability of Hg, followed by bacterial reduction [178]. Here, Hg was extracted from the soil into an aqueous phase for 10 h using ammonium thiosulphate (0.5 M) extraction. After that, 81% of Hg (II) removal was achieved. This procedure is more economical and environmentally friendly than the one-stage strategy, which includes only bacterial reduction [178].

5.3. Algae-Based Mercury Removal

Algae has been extensively examined for their ability to remove HMs, because they are found everywhere on Earth. Understanding of its ability to bind metals and innate adsorption mechanisms has dramatically improved in recent years. Conscious efforts were made to develop innovative algae- and seaweed-based adsorbents using various modes, i.e., extraction, nanoparticles, molecular, chemical modes, etc. [179]. The presence of functional groups, such as amino, hydroxyl, carboxyl, and sulfhydryl (SH), can serve as binding sites for metals, making the algal a suitable option for removing metals like Hg [180]. Utilizing marine macroalgae is economical for the removal of Hg. Although various types of algae can be used for the valorisation of HM wastes, but Green algae (Chlorophyta), brown algae (Phaeophyta), and red algae (Rhodophyta) are the three most studied categories of algae that show different binding preferences for different metals.

Some researchers checked six species of algae, namely *Fucus spiralis*, *F. vesiculosus*, *Ulva intestinalis*, *Ulva lactuca*, *Osmundea pinnatifida*, and *Gracilaria* sp., as mercury adsorbents. Among these, the green macroalgae (*U. intestinalis*, *U. lactuca*) displayed the best adsorption rates [181]. This investigation proved the competitive advantages of algae-based adsorbents, which display a high average of metal removal effectiveness of exceeding over 80% [181]. *Chlorella vulgaris* mercury ion biosorption is 42.1 mg g^{-1} at pH 6 [182]. Mercury uptake and accumulation by *U. lactuca (green algae)* is observed to 209 µg g^{-1} [183]. Upon accumulation, it is bound to macroalgae tissues without changing to a more hazardous form like MeHg. Therefore, investigating more algal and seaweed species, and considering novel approaches to achieving low-cost adsorbent regeneration can all encourage future research that focuses on large-scale Hg treatment.

However, there is disagreement over whether algae actually convert Hg into MeHg. According to Lei et al. (2019), algal organic material increased the MeHg content in a eutrophic lake [184]. Hence, further research is warranted to conclude whether the algae species performs this conversion.

5.4. Biochar-Based Mercury Removal

Biochar is a solid and high-carbon substance generated through the anaerobic thermal decomposition of organic biomass, at temperatures between 300 and 1000 °C in an oxygen-limited environment [185]. The biochar-based Hg elimination is a cutting-edge technique because it has excellent removal efficiency and cost-effectiveness, besides being produced from wood and agricultural wastes [186]. The presence of surface functional groups, such as -OH, C=O, link, C-O, and π bonds, facilitate biochar chemical modification by attaching thiol/amino groups and thereby increasing adsorption capabilities [187]. Studies have examined the biochar amended with copper oxides (CuO_x), cerium oxide (CeO_2), and manganese-cerium (Mn-Ce) mixed oxides, which are promising catalysts for oxidizing Hg^0 [188,189]. Lately, magnetic biochar (MBC) has received interest in treating flue gas Hg^0. However, it should be kept in consideration that biochar cannot reduce total Hg from the ecosystem, but it can significantly lower its mobility and bioavailability and thereby reduce toxicity to lives. Different independent studies conclude that biochar can lower the Hg levels in soil leachate and its bioavailability in sediments [190,191]. Further, biochar can also remove MeHg from solutions [192]. In a study, 36 biochars made from various feedstocks at various temperatures were tested for Hg removal from an aqueous solution. The study revealed that Hg is bound to sulphur (S), oxygen (O), and chlorine (Cl) in biochars and suggested that chemical interactions between mercury and different functional groups are one of the main processes of Hg removal [193]. The presence of different functional groups on the surface of amino-embedded modified biochar (AMBC) displays maximum mercury (II) adsorption capacity of 14.1 mg g^{-1} [186]. Biochar-bentonite composite (CB), prepared by the use of millet straw and bentonite, shows maximal Hg (II) adsorption capacities of ~12 mg g^{-1} much higher than adsorption values of biochar and bentonite (6.5 and 2 mg g^{-1}, respectively) [194].

Recent research suggests that biochar can lessen Hg deposition in case of crops as well. For instance, a pot study revealed that total dissolved mercury (THg) in the soil pore water is reduced by 34–44% in the rice growing season, and thus levels of THg in polished rice decrease by 58–70% [195]. In another study, similar success was observed in rice grain, where THg fell by ~82% with a 5% *w*/*w* sewage sludge charcoal amendment [196]. These innovative investigations indicate the tremendous capacity of biochar in treating Hg-polluted ecosystems. The attempts to chemically modify biochar are tested to increase its adsorbing potential. However, these techniques use caustic acids, bases, and other chemical reagents, not favourable to the environment. Therefore, research is needed to develop this technique further and streamline it for larger-scale Hg removal from the environment.

5.5. Advanced Oxidation of Mercury

The advanced oxidation process (AOP) is phase oxidation to eliminate mercury (0) from the flue gas. AOP is used in either conjunction with catalyst, ozone (O_3), or ultraviolet (UV) irradiation. AOPs is broadly divided into four categories: plasma AOPs, Titanium dioxide (TiO_2) photocatalytic AOPs, photochemical AOPs, and activated oxidant AOPs [197]. However, activated oxidant AOPs, are the most auspicious because these have strong oxidation capability with low energy usage. These cutting-edge techniques for accelerating oxidation are incredibly successful. Hydroxyl radicals (•OH) and sulphate radicals ($SO_4^{\bullet -}$) are prominently involved in the oxidation of mercury (0). Additionally, the advanced oxidation technique using hydrogen peroxide (H_2O_2) is particularly effective in the oxidation of several contaminants. Since radical-induced oxidation of mercury (0) is supposed to be effective and quick, this research will be pursued in the future for the removal of gaseous mercury (0) [198]. The vacuum ultraviolet radiation (VUV) process used in the activation of ozone/water/oxygen ($O_3/H_2O/O_2$) system displays that (•OH) and ($SO_4^{\bullet -}$) are responsible for oxidizing Hg^0 [199]. Researchers presented a manganese (Mn^{2+})-modified ferric ion Fe^{3+}/H_2O_2 wet scrubbing system, also known as Mn^{2+}-modified Fenton-like system, which is a novel oxidative absorption procedure for elemental mercury

from flue gas. The increase in Fe^{3+}, Mn^{2+}, or H_2O_2 concentration improved Hg^0 removal efficiency [200].

Catalytic oxidation of Hg^0 has attracted attention for the oxidation Hg^0 to Hg (II). Although, the current focus is on creating more reliable and affordable catalysts. Iridium oxide (IrO_2) modified Cerium-Zirconium (Ce-Zr) solid solution catalysts successfully catalyse the oxidation of Hg^0. It is noticed that surface-chemisorbed oxygen species helped the first oxidation of Hg^0 to mercuric oxide. Afterward, mercuric oxide might either react with hydrochloric acid (HCl) and release gaseous mercuric chloride ($HgCl_2$), or desorb from the catalysts [201]. Photocatalytic oxidation is another practical method to reduce Hg^0 emission. Zhang et al. developed several photo-catalysts for the photo-catalytic removal of Hg^0, including silver/silver iodide/silver carbonate ($Ag/AgI\text{-}Ag_2CO_3$) [202], silver iodide-bismuth oxyiodide/cobalt ferrite ($AgI\text{-}BiOI/CoFe_2O_4$) [203], silver/bismuth oxyiodide/zinc ferrite ($Ag/BiOI/ZnFe_2O_4$) [204], and silver@silver chloride/silver carbonate ($Ag@AgCl/Ag_2CO_3$) [202]. On the contrary, bismuth and iodide hybrid silver nanoparticles, like some other materials, might trigger the photo-catalytic oxidation of Hg^0. Here, these processes have some unsettling drawbacks, like the use of expensive chemicals, the generation of poisonous sludge, and other associated by-products, which requires more standardization.

5.6. Immobilization of Mercury

Immobilization procedures of Hg can effectively lower health hazards significantly by reducing the bioavailability of Hg. Hg is immobilized effectively, with the help of nanoparticles. In a study, iron sulphide (FeS) nanoparticles stabilized by carboxymethyl cellulose (CMC-FeS) exhibit selectivity for Hg and show higher Hg sorption capacity and faster sorption rate [159]. After applying zero valence, iron nanoparticles to the HMs-contaminated soils, a significant reduction in the availability of Hg and arsenic is observed [205]. Selenium (Se) nanoparticles are also proven successful in immobilizing Hg^0. Se nanoparticles converted 45–57% and 39–48% of the Hg^0 present in the soil into the insoluble mercuric selenide (HgSe), under aerobic and anaerobic conditions, respectively [206]. However, the fate and movement of nanoparticles in the environment and their impact on human health are poorly understood [192]. According to another study, the goethite-impregnated carbon foam also has the potential improvement for additional Hg, and/or other metal(loid)-contaminated industrial and/or abandoned mining locations without impacting the soil's electrical conductivity [207]. Researchers reported that Hg^0 immobilization capacity with molybdenum selenide ($MoSe_2$) improve by the doping of iron/copper/nickel (Fe/Co/Ni) [208]. $MoSe_2$-based adsorbents show Hg^0 removal effectiveness of 96.4–100% [208].

Although this technology exhibits remarkable efficiency in immobilizing Hg and is acknowledged as an affordable and accessible way to treat mercury-polluted soils, it is in the early stages of research, and new materials are emerging. AOPs are renowned for bridging the gap between the treatability attained by conventional physicochemical and biological methods, and the stringent day-to-day regulations set by environmental legislation. Photocatalytic methods can also be used for mercury removal [209]. Table 5 briefs merits and demerits of different remediation techniques. Among several methods, microbial treatment and biochar methods are most efficient and cost-effective.

Table 5. List of advanced and innovative techniques, and their positive and negative points in mercury removal from the environment.

Sr. No.	Name of the Technique	Positives	Negatives	References
Biological remediation				
1.	Phytoremediation	More affordable; eco-friendly; easy to usage	Time-consuming; root depth and Hg concentration limit this process	[163,164,166–168]

Table 5. *Cont.*

Sr. No.	Name of the Technique	Positives	Negatives	References
2.	Microbial treatment	High effectivity; economic; and environmentally benign Cosmopolitans in nature;	Less studied; not always appropriate for the other ecosystems	[66,174]
3.	Algae-based treatment	low-cost adsorbents; environmentally friendly; no secondary hazardous products formation	Time-consuming, less effective, some controversial where hazardous MeHg production takes place	[181,182]
4.	Biochar-based method	Excellent removal efficiency; cost-effective	Time- consuming; involve the use of caustic acids and bases and other hazardous chemical reagents	[186,194]
Physicochemical remediation				
5.	Advance oxidation processes (AOPs)	Strong oxidation capability and low energy usage	Use of expensive chemicals; generation of poisonous sludge, and other associated by-products	[198]
6.	Immobilization method	High and fast mercury sorption rate; good selectivity for Hg; affordable and accessible	Environmental unfriendly; generation of toxic secondary products	[208]

6. Challenges and Future Perspectives

Due to the persistent nature of Hg, its biomagnification is of great concern. From above discussions, it is noted that global warming, climate change from anthropological aspects, are major factors for increased Hg exposure. In addition to marine foods, recent observations demonstrated that rice consumers are subjected to MeHg exposure [210]. Researchers have established that Hg is one among the cause of infertility and the exposure is mainly through fluorescent light bulbs, broken thermometers, dental amalgams, skin lightning cream, batteries, and intake of sea foods. To establish the Hg impact on health, it is necessary to assess a dose-dependent response. However, it is challenging due to exposure source, toxicity, target organs and metabolism, which varies with each chemical form. For instance, MeHg has highest bio-absorptivity in gastrointestinal tract, hence entering central nervous system after passing blood–brain barrier. Elemental Hg exposure majorly from occupational incidents such as amalgam, and targets the kidney and brain. Although liquid Hg absorption is minimal, but after vaporization it can cause interstitial pneumonia. Over time, gaseous Hg is oxidized to Hg^{2+}, which accumulates in the kidney. The biological half-life of gaseous Hg is 2–4 days and 90% is excreted through faeces and urine [183]. In this section, challenges and future perspectives are presented, focusing on Hg fate, exposure assessment, biomarkers, reduction in toxicity and biomonitoring.

Previous studies on risk assessment often assumed Hg in fish is 100% MeHg and its complete absorption. However, few studies established that the processing method significantly changes bioaccessibility of HMs. For instance, cooking of meagre through grilling decreased bioaccessibility of Hg and MeHg to 54% and 64%, respectively [211]. In addition, the bioaccessibility of Hg and MeHg in fresh and marine fishes ranges from 21.4 to 51.7% and 19.5 to 59.2%, respectively [212]. Hence, a conservative approach of considering Hg in fish is 90–100% bioavailable can overestimate intake by 50% and additional research is necessary to characterize and categorize different species of fish with correction factors for proper risk assessment.

Hair is the preferred biomarker for MeHg exposure because sulphur containing proteins rich in hair bind to MeHg. As recommended by WHO, Hg hair to blood ratio 250 can be used to estimate Hg in blood. However, constant ratio consideration may be unreliable based on exposure type, gender, age, genetical make up, and geographical area [213]. Hence, future exposure assessments should also include blood measurement due to human

health concerns. In addition, selection of biomarkers should be categorically normalized to individual type of exposure. For instance, during pregnancy MeHg target is foetal brain and biomarkers reflecting the exposure is important to predict child development. Moreover, MeHg concentration in foetal blood reaches 2-fold higher than mothers due to active transportation across placenta. Hence, umbilical cord tissue or blood, and placenta should be preferred biomarkers to assess pre-natal exposure of Hg isomers [214,215]. In addition, Hg exposure is highly correlated with OS, hence OS markers such as glutathione can be considered to assay the extent of exposure. However, Hg can interact with SH group of glutathione, which reduces glutathione concentration and negatively correlates to Hg exposure [216]. Contradictorily, during Hg exposure, increased glutathione synthesis may be possible in response to OS induced by Hg [217]. Nevertheless, gender-dependent differences in antioxidant defence systems cannot be overruled.

In agreement with the high Hg ingestion in the Amazon, the highest levels of human exposure to Hg occur in South America. Although Hg concentration in blood has declined globally since the 1960s, [218] its emission to the atmosphere through anthropogenic sources have increased [219]. Nakamura et al., suggested high intake of selenium can provide protection against Hg toxicity [220]. As rice is the most preferred staple food in the globe and major Hg biotransporter, efforts were made to study the effect of selenium on Hg uptake by plant. It was evidenced that selenium supplementation in soil reduced inorganic Hg in brown rice: 26–74%; straw: 15–58%; and root: 0–48% [221]. In addition, increased plasma selenium and omega fatty acids decreased colour vision loss [222]. Several nutrients found in food such as selenium, iodine, and lycopene have exhibited protection against MeHg toxicity [183]. Recent studies received attention for showcasing the role of gut microbiota in excreting MeHg [223]. Hence, future studies focusing on Hg toxicity and potential reduction should consider nutritional interventions through diet and agronomic practices.

Monitoring of Hg concentration in biota is essential in reducing the risk of exposure. Many studies have characterized Hg concentrations at the organism level; however, it is necessary to define exposure nature and source. Mass independent fractionation of Hg stable isotopes in hair, blood, and urine can differentiate source of exposure, i.e., sea food (MeHg) and occupational exposure such as dental amalgam (Hg^0). Whereas mass dependent fractionation can be used to study the processes involved in chemical transformations of Hg inside the body such as demethylation of MeHg. Sherman et al., provided new insights of human mercury exposure using $\Delta^{199}Hg$, they concluded that Hg measured in hairs are majorly derived from sea foods, however, MeHg derived from fish demethylated within the body and excreted through urine along with amalgam derived inorganic Hg [224]. This disproves a generally held hypothesis that Hg in urine is originated from exposure to inorganic Hg. Hence, consideration of internal mechanisms and tissue location leading to Hg isotope change will help in better biomonitoring. In birds, MeHg conjugates with cysteine residues of protein which exhibit bidirectional exchange in blood stream. However, its toxicological risk is governed by dietary intake rather than internal demethylation, depuration to feathers and maternal transfer [225]. Hence, stable isotope technique can improve understanding of internal cycling of Hg in birds and expansion beyond seabirds to others helps in understanding environmental risk of Hg. In addition, interspecies toxicity tolerance of Hg particularly between aquatic and terrestrial mammals exists. For instance, the demethylation site for marine and terrestrial mammals is the liver and kidney, respectively [226,227]. Hence, additional Hg isotopes measurement expands deep understanding of neurological developments during exposure. In summary, it is a challenge to track Hg exposure and its cycle between the populations and sub-populations as biomarkers and its fate changes with the type of exposure. However, with harmonized experimental designs and advanced technologies in the near future, Hg can be removed at both industrial and pilot scale with low cost and biobased strategies which reduces the probability of Hg exposure.

Taking in to consideration the various levels of toxicity associated with Hg, there is a huge research gap existing in understanding the multiple routes of entry into terrestrial

and aquatic environment, transformation of Hg in various tropic levels, pattern of Hg accumulation in various organisms, interaction of Hg with cellular and subcellular components, impact of Hg pollution on physiology, biochemistry, and genetic makeup of various organism and the final fate of Hg components in various ecosystem. Most of the available analytical methods used for detecting the presence of mercury has a sensitivity range below which the concentration of Hg cannot be detected from environmental samples. Thus, development of more sensitive Hg biosensors that can detect even trace quantities of Hg from environment samples is a major concern in understanding the level of Hg contamination. Further, most of the reported studies are dealing with higher trophic levels, whereas the data related to Hg toxicity towards planktons, insects, nematodes, fungi, and bacteria are lacking in the available literature. A great deal of research studies is required in the future to understand the chemistry of Hg toxicity in these organisms.

7. Conclusions

This review highlights the Hg bioavailability, remediation, toxicity, and its distribution in ecological cycle. Human exposure to mercury through a contaminated food chain can exert a potential threat to health. Monitoring of Hg concentration in biota is essential in reducing risk of exposure. Various defence mechanisms at the cellular level, especially antioxidant generation and OS regulators monitoring, have been identified as potential biomarkers to assess extent of Hg exposure. Many studies have characterized Hg concentrations at the organism level; however, it is necessary to define exposure nature and source. In addition, various remediation strategies are emerging; however, reducing the gap between the treatability attained by conventional physicochemical and biological methods, and the stringent day-to-day regulations set by environmental legislation is highly necessary to cope with future challenges.

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